S2313

**Esophageal Stricture Recalcitrant to Repeated Dilation in Complex Rheumatological and Bullous Disease**

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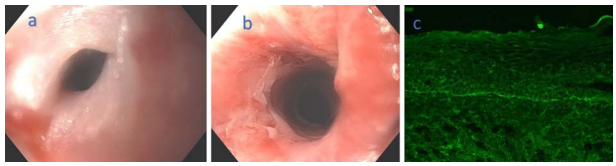
**Introduction:** Many rheumatological diseases have been well characterized to affect the esophagus. Esophageal involvement in autoimmune bullous disease is rare and almost always occurs with severe cutaneous symptoms. We report a case of severe esophageal strictures recalcitrant to repeated dilatation in a patient with complex rheumatological disease and isolated esophageal bullous disease.
**Case Description/Methods:** A 50-year-old female presented with complete inability to tolerate oral intake for several weeks. Past history included Sjogren's syndrome and esophageal strictures with many previous dilatations. Physical exam findings were relevant for poor skin turgor, dry mucous membranes, and skin hyperpigmentation on her hands and forearms. Laboratory studies showed hypokalemia, hypoalbuminemia, and anemia. The patient was admitted. Her esophagram and esophagogastroduodenoscopy (EGD) showed a proximal esophageal stricture which was dilated using a push style dilator to 14mm. The stricture recurred, requiring repeated dilatation and placement of a percutaneous gastrojejunostomy. Once oral intake was tolerated, she was discharged with outpatient follow-up. Outpatient rheumatological workup showed antibodies to ANA, RNP, SSA, CCP, and Smith indicating mixed connective tissue disease with features of systemic lupus erythematosus, rheumatoid arthritis, and scleroderma. She required multiple dilatations over several months and was started on mycophenolate mofetil with some clinical improvement. The dysphagia recurred 2 months later and EGD showed and esophageal stricture measuring 7mm requiring a neonatal upper gastroscope to facilitate balloon dilatation. Biopsies examined for immunofluorescence showed continuous linear deposition along the basement membrane zone for IgG and C3, consistent with atypical esophageal bullous systemic lupus erythematosus or atypical esophageal bullous pemphigoid. The patient declined systemic steroids. Her mycophenolate mofetil dose was increased and she received weekly dilatations to prevent Koebnerization and allow time for medical therapy effect. After 3 months of treatment, she had symptom improvement (**Figure**).
**Discussion:** In patients with systemic immune mediated disorders including rheumatological disease, bullous diseases can be considered as a cause of esophageal stricture. Bullous disease with esophageal involvement can be difficult to treat, especially in this patient who declined corticosteroids. A combination approach using both immune suppression and dilatation was effective.




[2313] **Figure 1.** a. Pre- dilatation during repeated dilation treatments. Diameter 8mm. b. Post- dilatation. Diameter 11mm. c. Immunofluorescence showing IgG deposition along the basement membrane.

S2314

**Esophageal Schwannoma: An Important Differential Diagnosis for Esophageal Subepithelial Lesions**

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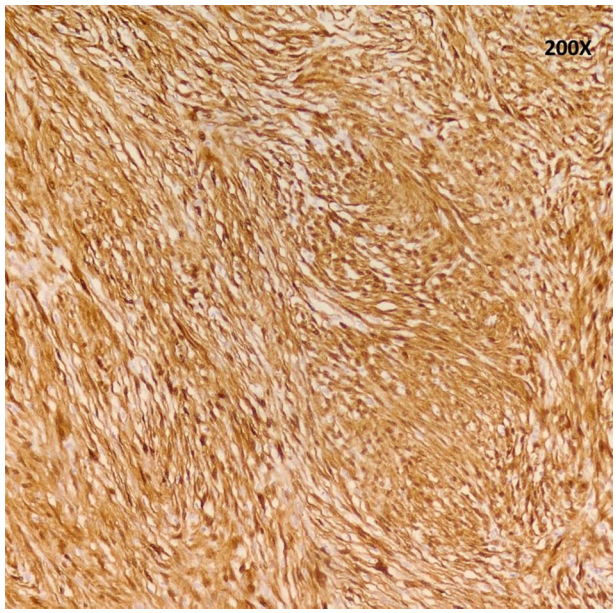
**Introduction:** Schwannomas are slow-growing, mostly benign tumors that arise from the Schwann cells of the nerve sheath. They represent about 2–6% of mesenchymal gastrointestinal (GI) tumors and are an important differential diagnosis for subepithelial lesions (SELs). Here, we present a patient who presented with dysphagia due to an esophageal SEL that was found to be a schwannoma.
**Case Description/Methods:** A 72-year-old female presented to our clinic for a complaint of dysphagia to solid food that has been progressing over a few months. We obtained an esophagogastroduodenoscopy (EGD) that showed a bulging lesion in the esophagus, causing luminal narrowing. We evaluated the mass using an endoscopic ultrasound (EUS) and it revealed a subepithelial hypoechoic heterogeneous mass originating from the 3rd and possibly the 4th layers of esophagus. EUS-guided fine needle aspiration biopsy (FNB) showed bland spindle cells proliferation with no atypia or mitosis. Cells showed S100 positivity, and negative desmin, SMA, DOG-1 and CD117. Positive staining for S100 favored schwannoma diagnosis (**Figure**). After discussion with the patient, she elected to proceed with another EGD for resection of the mass using submucosal tunnel endoscopic resection (STER) procedure. Pathologic analysis showed a well-circumscribed lesion with alternating hypercellular Antoni A with nuclear palisading around fibrillary processes (verocay bodies) and hypocellular areas. Immunohistochemical (IHC) staining showed the same profile as the FNA. Histologic and IHC profiling supported the diagnosis of schwannoma. We obtained an esophagram 1 day after the resection which was negative for any leakage. We advanced the patient's diet slowly and she was discharged home after resolution of her dysphagia.
**Discussion:** SELs of the esophagus are mostly benign at the time of diagnosis, however many of these lesions have malignant potential. EUS is the gold standard to describe SELs size, location, originating layer, echogenicity, and shape (Table). The European society of gastrointestinal endoscopy recommends EUS-FNB for all SELs ≥ 20mm. IHC is required for differentiation of lesions. Positivity for S-100 protein and negativity for actin and desmin, support a nerve sheath origin. Management is usually based on the presence of obstructive symptoms. Endoscopic full-thickness resection should be considered versus thoracoscopic enucleation if resection of the SEL was decided.




[2314] **Figure 1.** S100 positivity favors the diagnosis of Schwannoma.

**Table 1. Most common esophageal subepithelial lesions, their layers of origin, and endoscopic ultrasound (EUS) features**

| Most common esophageal subepithelial lesions | Layer of origin | EUS features/characteristics |
|---|---|---|
| Duplication cyst | Third Layer | Anechoic avascular lesions |
| GIST low risk | Second/Forth layers | Hypervascular heterogeneous hypoechoic lesions |
| Granular cell tumor | Second, Third Layers | Hypoechoic heterogeneous lesions, more echogenic compared to the surrounding muscle layer |
| Leiomyoma | Second/Forth Layers | Homogeneous hypoechoic lesions, similar to the muscle layer |
| Lipoma | Third layer | Hyperechoic homogeneous |
| Lymphangiomas | Third Layer | Anechoic avascular lesions with internal septations |
| Varices | Third Layer | Anechoic, tortious with Doppler signal |

**S2315**

**Epiglottic Calcification, a Rare a Cause of Dysphagia**

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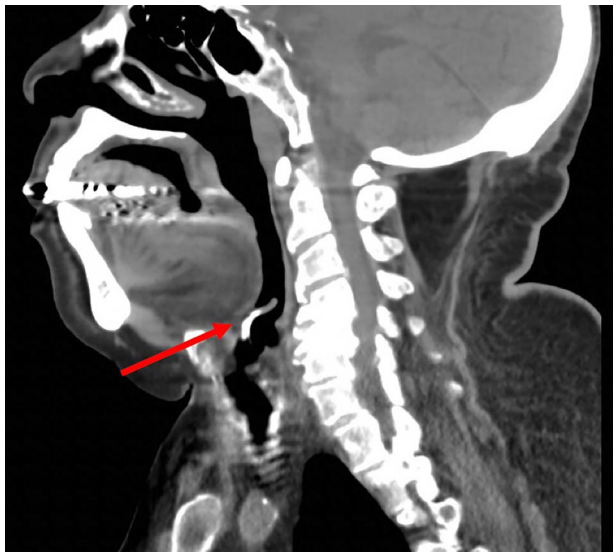
**Introduction:** In consideration of dysphagia, most cases predominate in the oropharyngeal region with the remainder caused primarily by esophageal causes. Lesser known and studied is the development of dysphagia and globus sensation from epiglottic pathology, namely epiglottic calcification. With less than a dozen published cases in literature, very little data exists on identification, diagnosis, and treatment of this known cause of morbidity. Here we present a case of oropharyngeal dysphagia arising from a rare cause, epiglottic calcification.
**Case Description/Methods:** An 81-year-old male with history of aortic stenosis and carotid artery stenosis presented with worsening dysphagia over the course of one month. The patient reported significant dysphagia initially to solids and subsequently to liquids causing a weight loss of over 50 pounds. Physical exam of oropharynx and neck was unremarkable. Bedside swallow evaluation suggested mildly decreased hyolaryngeal movement, but no other significant abnormalities were identified. A Barium swallow study revealed incomplete epiglottic excursion during the pharyngeal phase of swallowing. With gastroenterology consultation, patient underwent evaluation with contrast-enhanced esophagogram that showed severe esophageal dysmotility and reflux. CT of neck demonstrated calcification of epiglottis without epiglottal enlargement, which was ultimately found to be the source of his concern. Via ENT, the patient underwent flexible fiberoptic laryngoscopy, and via gastroenterology, patient underwent EGD with biopsy. Yet, no esophageal or gastric pathology other than epiglottic calcification were identified. As no definitive treatment modality was outlined despite evaluation, patient was changed to a modified diet with ongoing speech and swallow therapy with outpatient follow up (**Figure**).
**Discussion:** Epiglottic calcification is a rare cause of dysphagia that is poorly understood in its etiology, clinical course and outcomes. With our case, we hope to add to literature a unique presentation and imaging. Furthermore, with our case it is demonstrated that despite consultant recommendations, no clear evaluation process or treatment modality currently exists, lending to further study of this pathology. Currently, diagnosis can be accomplished with radiologic evaluation along with exclusion of other causes; however, no definitive treatments are available for this condition. Although the condition itself is rare, it adds to the differential diagnosis as a cause of significant dysphagia.



[2315] **Figure 1.** Calcification of epiglottis (red arrow) visualized on CT scan.

**S2316**

**Esophageal Pseudodiverticulosis Presenting as Chest Pain, Epigastric Pain and Nausea**

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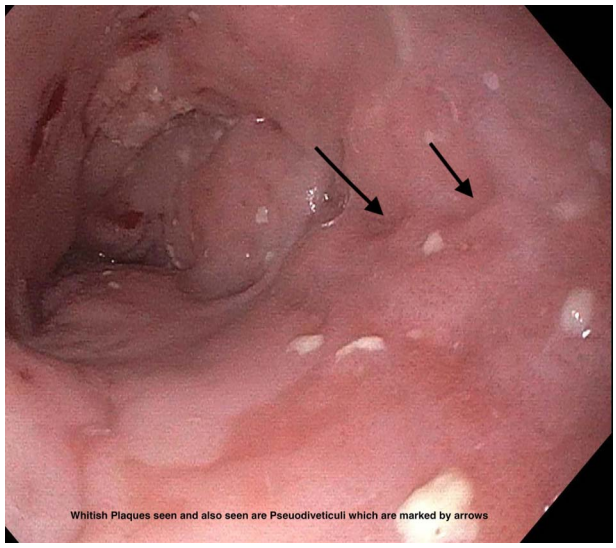
**Introduction:** Esophageal intramural Pseudodiverticulosis (EIP) is an unusual condition manifested by tiny flask-shaped out pouches in the wall of the esophagus. We describe a case of a 68-year-old woman presenting for upper endoscopy (EGD) for an abnormal CT Scan showing esophageal thickening.
**Case Description/Methods:** A 68-year-old woman presenting for EGD for an abnormal CT Scan showing esophageal thickening. This was seen as a finding on CT Angio Chest/Abdomen performed to rule aortic dissection as the patient complained of persistent chest pain. The chest pain was associated with epigastric discomfort and nausea. On EGD, the findings were consistent with the esophageal pseudodiverticulum located throughout the length but mainly in the distal esophagus. Accompanying the pseudodiverticulosis were whitish punctate plaques scattered throughout the length of the esophagus. The remainder of the exam on EGD was normal. The biopsy findings were consistent with esophagitis, in the areas where multiple esophageal psuedodiverticuli were found. The PAS stain was negative for fungal infection. The patient was prescribed Omeprazole 40 mg daily for esophagitis and was also offered treatment for Esophageal Candidiasis due to findings of white punctate plaques in the esophagus (**Figure**).
**Discussion:** Esophageal Intramural Pseudodiverticulosis is an uncommon condition first described by Mendl et al. in 1960. EIP is mainly a disease of older age with a 3:2 male to female ratio. In 90% of the cases of EIP, esophagitis is found as in our patient. Esophageal Candidiasis is commonly associated with EIP. These Intramural Pseudodiverticulosis are dilated excretory ducts of the deep esophageal mucosal glands. Treatment is required in about 90% of the patients. Treatment mainly consists of PPI therapy in cases of esophagitis and dilatation in Esophageal Strictures. Antifungal therapy is indicated in suspected Esophageal Candidiasis cases as candida infection is also associated with EIP.




[2316] **Figure 1.** In this image, whitish plaques are seen. We can also see pseudodiverticula (marked by arrows).

**S2317**

**Esophageal Candidiasis Hiding the HSV Esophagitis**

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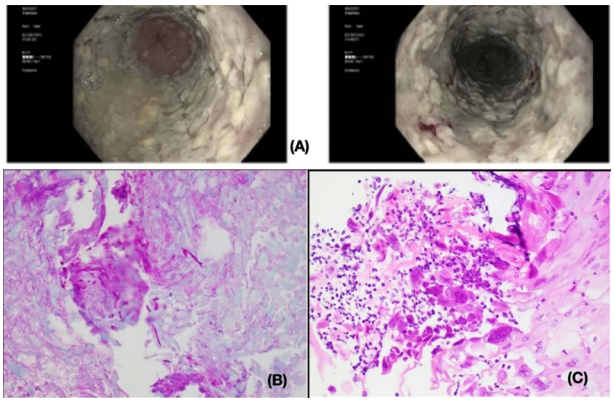
**Introduction:** Opportunistic infections are fearful complications usually observed in immunocompromised patients. Esophagitis with Candida, HSV and CMV is among those infections, but infections with 2 organisms simultaneously is uncommon and can be missed leading to complications. We are presenting a case with concomitant HSV and candida esophagitis in a patient with achalasia.
**Case Description/Methods:** A 65-year-old male with PMH of Barrett's esophagus, achalasia, GERD, stage IV prostate cancer on chemotherapy with docetaxel and on chronic steroids for metastatic bone pain. He had trouble swallowing for many years, had manometry as an outpatient and was diagnosed with type 1 achalasia. For that he underwent endoscopic dilatation in the past. He presented with worsening odynophagia and dysphagia for the last 3 days to the extent that he was unable to swallow his own saliva. He underwent EGD which showed extensive esophageal plaques consistent with candida (**Figure** A), biopsies were taken. He was started on IV Fluconazole. Pathology of biopsies with PAS and GMS stains demonstrated infiltrating hyphae and yeasts, confirming esophageal candidiasis (**Figure** B). In addition biopsies also revealed viral inclusions, multinucleation and molding, with immunohistochemistry being positive for HSV-1 (**Figure** C). After biopsy results, he was also started on IV acyclovir in addition to fluconazole with improvement in his symptoms. Treatment was extended to 21 days because of the severity of infection.
**Discussion:** Esophagitis is most often caused by non-infectious conditions such as GERD, pill-induced or eosinophilic whereas infectious esophagitis occurs predominantly in patients with imparied immunity. However, it has also been described in otherwise healthy patients. The most common causes of infectious esophagitis are Candida, followed by HSV and CMV. Concomitant infection with 2 organisms is very rare as compared to infection with a single organism. A prospective study in HIV patients showed, 20% of patients had candida with CMV and only 1.8% had HSV with candida [1]. Diagnosis is usually established with an upper endoscopy with biopsies and histopathology. It is also the best way to determine the appropriate antimicrobial therapy. In severe cases of infection with 2 organisms; therapy against both organisms is recommended.




[2317] **Figure 1.** (A, Endoscopic evaluation) (B, PAS stain showing infiltrating hyphae and yeast ) (C, H&E section showing numerous viral inclusions with margination, multinucleosis and moulding).

**REFERENCE**

1. Bonacini M, Young T, Laine L. The causes of esophageal symptoms in human immunodeficiency virus infection. A prospective study of 110 patients. Arch Intern Med. 1991 Aug;151(8):1567-72. PMID: 1651690.

**S2318**

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**Esophagitis Dissecans Superficialis After Gastric Sleeve: A Case Report**

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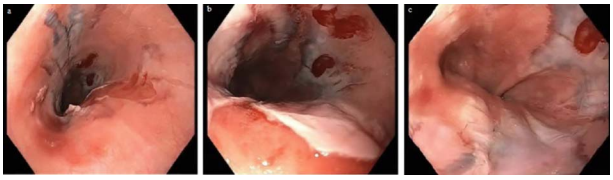
**Introduction:** Esophagitis dissecans superficialis (EDS) is a desquamative disorder of the esophagus, with little being understood behind its pathogenesis. It has been associated with medications, notably bisphosphonates, NSAIDs, hot beverages and caustic chemicals. It involves direct contact injury rather than an ischemic injury. It is often an incidental finding and represents a benign condition without complications. We present a case of NSAID-induced EDS in a patient with gastric sleeve surgery.
**Case Description/Methods:** A 63-year-old male with history of morbid obesity status post gastric sleeve surgery 10 years prior presented to the GI clinic for gastric bypass conversion. Documented medications included acetaminophen and diclofenac for arthritic pain. Physical exam revealed BMI of 50.4. EGD revealed esophageal sloughing whitish membranes adjacent to healthy mucosa. Multiple biopsies were taken and final pathology revealed features compatible with EDS with reactive epithelial atypia. The patient was started on oral PPI therapy and advised to avoid NSAIDs. Patient recovered well (**Figure**).
**Discussion:** Esophagitis dissecans superficialis has been associated with esophageal iatrogenic injury (sclerotherapy band ligation, dilation and mediastinal radiation). It has been reported in post-gastric bypass surgery patients, but to our knowledge, no cases have been reported after a gastric sleeve procedure. Our case poses the question whether the use of NSAIDs in post-gastric sleeve patients predisposes them to develop EDS.



[2318] **Figure 1.** Endoscopic views of "sloughing" esophagus dessicans.

**S2319**

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**Esophageal Squamous Cell Carcinoma: An Unfavorable Complication of Achalasia**

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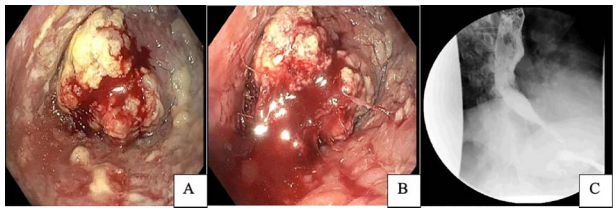
**Introduction:** Achalasia is a motility disorder of the esophagus characterized by impaired relaxation of the lower esophageal sphincter and loss of peristalsis in the distal esophagus. It is a rare condition with an annual incidence of 0.5-1.2 per 100,000 individuals. The etiology of primary achalasia is unknown, however secondary achalasia can be attributed to malignancy, infections or systemic diseases such as amyloidosis. An infrequent complication of achalasia is esophageal squamous cell carcinoma which has a prevalence of 26 in every 1,000 cases. We present a case of interval locoregionally advanced esophageal squamous cell carcinoma only 2 years after a normal upper endoscopy.
**Case Description/Methods:** A 67-year-old female with known achalasia and previous pneumatic dilation in her 30s presented to our outpatient clinic in 2019 with complaints of worsening chronic dysphagia. EGD was performed which revealed a significantly dilated esophagus with candida esophagitis. Despite completing antifungal therapy, she continued to experience dysphagia to solids and liquids. Barium swallow demonstrated absent peristalsis with pooling of contrast within the esophagus. High-Resolution Manometry testing demonstrated absent peristalsis. She opted for surgical myotomy, however due to COVID restrictions, the procedure was delayed. Repeat EGD was performed in 2022 for pre-surgical evaluation and showed a large obstructing friable esophageal mass in the lower third of the esophagus.

Pathology was consistent with invasive poorly differentiated squamous cell carcinoma. PET scan showed locoregional disease with FDG-avid esophageal and gastrohepatic node lesions. She was started on chemoradiation with Paclitaxel and Carboplatin (**Figure**).

**Discussion:** The risk of esophageal squamous cell carcinoma in achalasia has significantly increased with incidence of approximately 1 in 300 patients. The presumed mechanism of malignancy in achalasia is poor emptying resulting in food stasis, bacterial overgrowth and inflammation leading to dysplasia and development of carcinoma. Given the relatively low incidence, there are currently no guidelines on routine endoscopic screening to assess for malignancy in patients with achalasia. Survival rates are poor as patients are often diagnosed at advanced stages. This case aims to illustrate the importance and need for interval screening in individuals with long standing achalasia to improve outcomes.




[2319] **Figure 1.** Image A, B demonstrating almost completely obstructing mass, with retained food products. Lesion appears friable and is oozing blood. Image C with retained contrast and "birds beak" deformity at the gastroesophageal junction.

**S2320**

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**Esophageal Epidermoid Metaplasia: A Case of a Rare Premalignant Condition**

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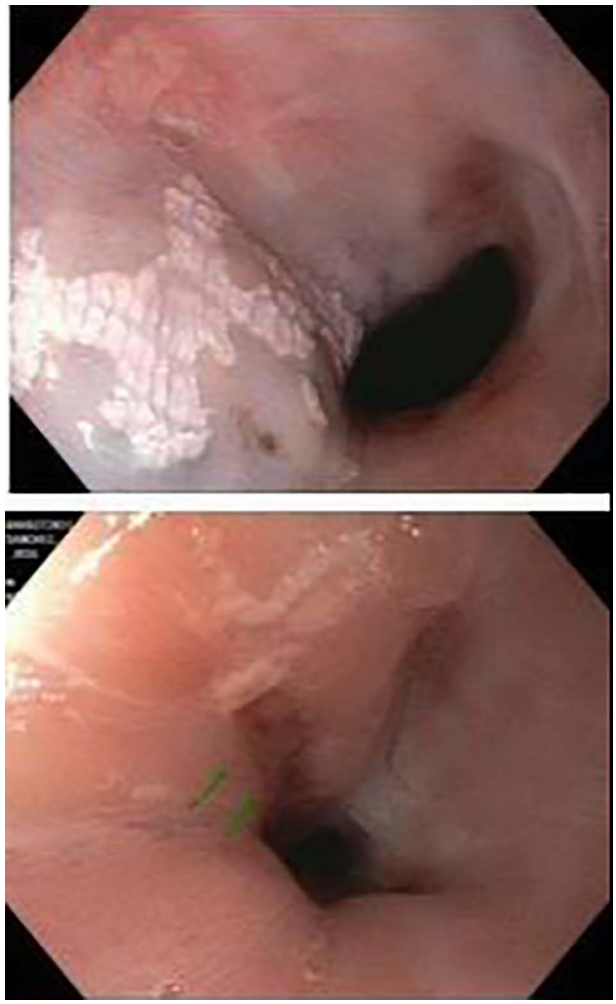
**Introduction:** Esophageal epidermoid metaplasia (EEM) also known as esophageal leukoplakia is a rare condition with potential for malignant transformation to in-situ and invasive squamous esophageal carcinoma. It can present with symptoms of dysphagia and gastroesophageal reflux or can be found incidentally on endoscopy. Due to the relative rarity of the condition the consensus on interval for screening and modality of choice for treatment remains elusive.

**Case Description/Methods:** A 63-year-old Hispanic male with history of HIV on HAART therapy was referred from HIV clinic for a few weeks history of dysphagia predominantly for solids and GERD symptoms. He denied any hematemesis, melena, weight loss, oral thrush, fevers, or systemic symptoms. Physical exam was unremarkable. He reported a history of Colon Cancer in his sister and denied a history of smoking or alcohol use. An EGD was performed which showed grade 1 esophagitis, a focal linear 2 cm segment of granular, whitish mucosa in distal esophagus, a non-obstructing schatzki ring near GE junction and mild gastritis. The pathology showed H. Pylori positive chronic gastritis and the esophageal lesion was characterized as epidermoid metaplasia on pathology. Patient was treated with H Pylori eradication therapy and PPI. Repeat Endoscopy was performed with APC of the epidermoid metaplasia. Patient reported improvement in symptoms, and he was scheduled for repeat surveillance endoscopy for the metaplasia owing to the risk of transformation to SCC and assessing adequacy of ablation with APC. Repeat endoscopy and biopsy of the area few weeks after APC showed no endoscopic or histopathologic evidence of residual metaplasia (**Figure**).

**Discussion:** EEM/Esophageal leukoplakia is a rare entity which is often missed or discovered incidentally on endoscopy. Smoking and Alcohol consumption are main risk factors which were not present in our patient. While HIV is one of the risk factors for Oral Leukoplakia, but this patient had been optimally treated for years with undetectable viral load and normal CD4 count as per HIV clinic records. There is no consensus on frequency of surveillance endoscopy and some experts suggest annual surveillance with 4-quadrant biopsies every 1–2 cm in 6 months to yearly if no dysplasia or disease progression is seen. The treatment modalities include ablation with RFA, cryotherapy, APC and EMR depending upon size of lesion and degree of dysplasia.



[2320] **Figure 1.** Esophageal Lesion (top) Pre and (bottom) Post APC Ablation.

**S2321**

**Esophageal Button Battery Retrieval: Time-In May Not Be Everything**

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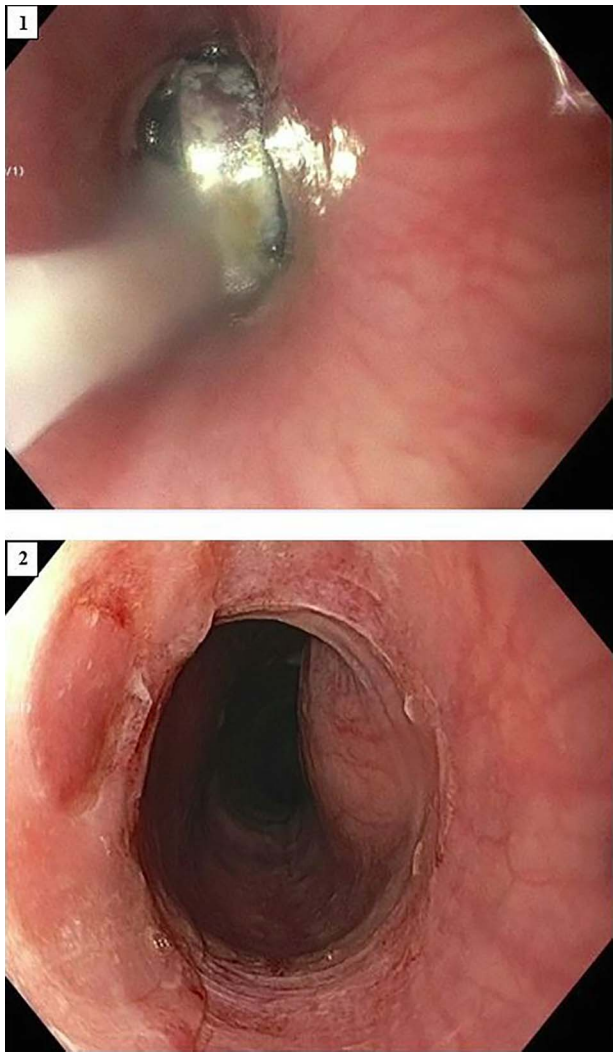
**Introduction:** Management of ingested foreign bodies is a challenging task given differences in type and presentation. Ingested button batteries cause injury via electrical discharge, corrosive contents, and toxicity. The European Society of Gastrointestinal Endoscopy recommends endoscopic retrieval within 12-24 hours for objects longer than 6 cm. Conservative monitoring can be instituted for blunt objects less than 6 cm in length and 2.5 cm in diameter, or post pyloric. Due to risks of fistulas, perforation, and vocal cord paralysis, ingested button battery cases are handled emergently. However, we present an unusual case that demonstrated significantly delayed endoscopic removal of an ingested button battery without complication.
**Case Description/Methods:** A 45-year-old male with no medical history presented 4 days after ingestion of '2 magnets'. He had dysphagia and difficulty eating solid foods, but remaining review of systems was negative. He denied tobacco or alcohol use, and no family history of gastrointestinal cancers. Vitals were normal. His abdomen was benign. Visualization of his oropharynx was unremarkable. Labs did not demonstrate leukocytosis or anemia. Imaging revealed one radiopaque body in the esophagus at T1 level and one in the rectum. Endoscopic removal of the object was delayed until day 6 given patient's initial refusal. A 20mm button battery was found in the esophagus and removed with a roth net (**Figure** panel 1). Mild localized mucosal necrosis was noted underlying the object (**Figure** panel 2). He tolerated oral intake later that evening.
**Discussion:** The management of foreign bodies is a challenge due to technical difficulties of endoscopic retrieval and unpredictable outcomes. As seen in this case, the excessive duration of time post button battery ingestion did not correlate to severity and complications expected. Despite its larger diameter of 20 mm, the button battery only caused mild mucosal necrosis. This case demonstrates that esophageal dwell time alone is not an independent risk factor and may not correlate with severity of injury. There are risks associated with performing emergent endoscopic procedures including availability of staff, abbreviated time for full preoperative evaluation, and logistic challenges. It is important to identify when procedures can be performed under controlled circumstances. Our case contributes to the fund of knowledge regarding delayed endoscopic foreign body removal of a button battery but should not be used to guide current management without additional data.




[2321] **Figure 1.** 1: Identification of a 20mm button battery in the upper esophagus. 2: Mild localized necrosis of the esophageal mucosa directly underlying the object.

**S2322**

**Esophageal Intra-Mural Pseudo-Diverticulosis With Candidiasis**

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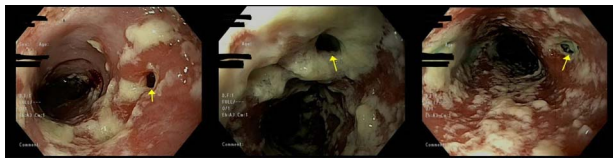
*Houston Methodist Hospital, Houston, TX.*

**Introduction:** Esophageal intramural pseudo-diverticulosis (EIPD) is a rare disorder of idiopathic etiology characterized by the presence of multiple small outpouchings protruding from the esophageal lumen. Diagnosis is typically made through radiological imaging or with esophagogastroduodenoscopy (EGD). While asymptomatic by itself, symptoms arise from associated conditions including candidiasis, esophageal stricture, and chronic esophagitis. It has a male predominance and dysphagia secondary to esophageal stricture is the most common presenting complaint. Treatment is directed at associated symptomatic conditions and whether or not the diverticula disappear post treatment has no impact on the patients' clinical outcome.
**Case Description/Methods:** This is a case of a 38-year-old male with human immunodeficiency virus (HIV) on anti-retroviral therapy. He was evaluated for a chronic history of dysphagia and odynophagia, associated with nausea, vomiting, heart burn, and weight loss. He underwent EGD which revealed pseudo-diverticula along the entirety of the esophagus with superimposed esophageal candidiasis. Patient was prescribed fluconazole 100 mg oral daily for 2 weeks and will be re-evaluated post treatment (**Figure**).
**Discussion:** Dysphagia is a common presenting symptom for numerous GI conditions. Though cases of EIPD are uncommon, it is important for clinicians to keep this diagnosis in their differential. The diagnosis of EIPD may not always be apparent through EGD, as the diverticula may be small enough to miss. If a high clinical suspicion remains, radiological imaging with contrast should be considered as it has enhanced sensitivity for detecting these smaller diverticula. EIPD has been associated with *Candidiasis*, however it is uncertain whether it causes or is a result of the infection. In our case, we were unable to confirm whether EIPD was the cause or result of a Candida infection. Additionally, patient education is important when it comes to dysphagia. Our patient had been experiencing symptoms for 6-7 months before a diagnosis was made. It is important to educate patients that dysphagia is an abnormal symptom and should be further evaluated to determine causal etiology. There is scarce literature available regarding discussion of EIPD, therefore clinicians should continue to report cases to improve our understanding of this rare pathology.



[2322] **Figure 1.** Esophagogastroduodenoscopy images demonstrating esophageal intramural diverticula indicated by yellow arrows, and showing generalized esophageal candidiasis plaques present throughout the entirety of the esophagus.

**S2323**

**First Known Case of Esophageal Parakeratosis Associated With a Severe SARS-CoV-2 Infection**

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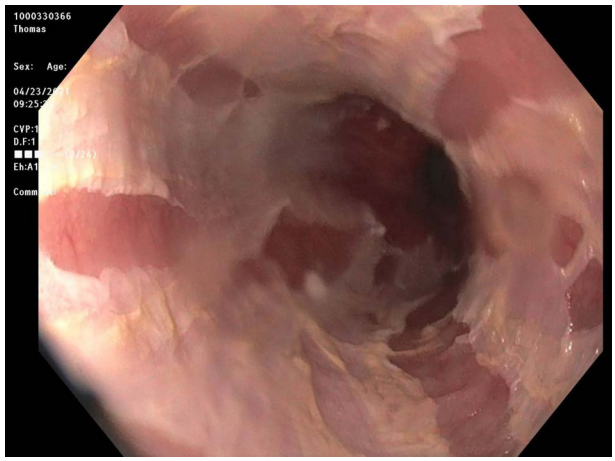
**Introduction:** Esophageal parakeratosis is a rare, benign condition that is associated with vitamin deficiencies as well as squamous cell carcinoma of the head and neck or upper gastrointestinal tract. Symptoms include dysphagia, odynophagia, and heartburn. Here we describe a patient diagnosed with esophageal parakeratosis in the setting of a recent, severe SARS-CoV-2 infection as his only associated condition.
**Case Description/Methods:** A 79-year-old man presented for an outpatient endoscopy for history of a gastric mass. He had been worked up for this presumed gastrointestinal stromal tumor (GIST) with endoscopic and surgical biopsies that were unrevealing. During that time he developed a severe case of SARS-CoV-2 with prolonged hospitalization and was started on anticoagulation for bilateral pulmonary emboli. He developed hematemesis and endoscopy revealed a large ulcer at the same site of the previous noted mass instead of the previously. He was now presenting for endoscopic workup with EGD as an outpatient for follow up. Endoscopy revealed only chronic gastritis but no evidence of mass or ulcer. However, the entire esophageal mucosa was abnormal with diffuse sloughing. Brushings were taken and he was empirically started on antifungals for presumed candida esophagitis as well as proton pump inhibitor twice daily. Histology revealed esophageal parakeratosis without dysplasia without candida. He returned for a repeat EGD and was found to have a normal esophagus at that time. All vitamin levels checked were within normal range including zinc at 78 ug/dL. There were no other known active malignancies at the time and imaging revealed no residual evidence of mass. His only other association during this time was the recent, severe SARS-CoV-2 infection (**Figure**).
**Discussion:** Esophageal parakeratosis needs to be differentiated from other conditions such as candida and eosinophilic esophagitis. It should raise suspicion for head and neck malignancies and has been found in the nearby esophageal mucosa of 90 percent of patients with esophageal cancer in one study. Other associated conditions with esophageal parakeratosis include tylosis and alcohol abuse. The temporal relationship of the severe SARS-CoV-2 infection likely causing some degree of immunosuppression in this patient could have been the precipitating factor that led to the development of his esophageal parakeratosis. More studies are needed to elucidate the relationship between the immunocompromised state during SARS-CoV-2 infection and esophageal parakeratosis.




[2323] **Figure 1.** Esophageal Parakeratosis.

**S2324**

**Epidermoid Metaplasia: An Endoscopic Surveillance Dilemma**

*Robert E. Spiller, DO, MS, Jonathan Francis, MD.*
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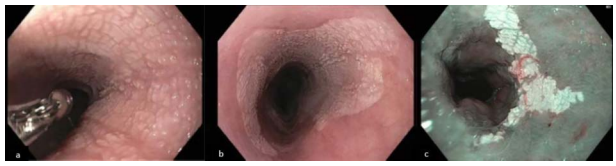
**Introduction:** Oral leukoplakia often presents as white patches on oral mucosa and carries a prevalence of 1.5-4.3% within the general population. It is a potentially malignant disorder with its own set of management principles regarding surveillance vs excision/ablation. An analogous entity within the esophagus, epidermoid metaplasia is a rare diagnosis (0.2% of 1,048 consecutive esophageal biopsies) in which the squamous epithelium of the esophagus is replaced with a layer that resembles the epidermis of the skin. It has similar risk factors to oral leukoplakia and presents in middle-aged to elderly patients with a history of smoking and/or alcohol intake. Similar to oral leukoplakia, it has been characterized as a potentially malignant disorder and has been associated with adjacent areas of high grade dysplasia and/or esophageal squamous cell carcinoma.
**Case Description/Methods:** A 69-year-old female with active tobacco use presented for GERD and globus sensation. Esophagogastroduodenoscopy (EGD) showed white plaques with an undulated appearance to the mid-to-distal esophagus (Figures 1a/1b). Mid-esophageal biopsy specimens noted a granular cell layer and hyperkeratosis, consistent with epidermoid metaplasia. EGD one year later showed similar endoscopic findings without evidence of dysplasia. Patient is scheduled to continue annual surveillance EGD. A 43-year-old-male with history of colon cancer s/p sigmoidectomy presented for refractory reflux. EGD showed Los Angeles Grade B esophagitis with an area of well-demarcated, white plaque with an irregular contour (Figure 1c). Biopsies from the gastroesophageal junction showed reactive squamous epithelial changes, including epidermoid metaplasia. His omeprazole was up-titrated to twice daily with improvement of symptoms. Patient is pending surveillance EGD in 1 year.
**Discussion:** Epidermoid metaplasia is often characterized by a well-demarcated, white plaque in the mid-to-distal esophagus. Unlike its equivalent, oral leukoplakia, there is currently no consensus on surveillance for epidermoid metaplasia. However, given its risk for malignant transformation, we recommend annual EGD with 4 quadrant biopsies.



[2324] **Figure 1.** 1a. An undulating, shaggy appearance can be seen, characteristic of epidermoid metaplasia. 1b. A clearly demarcated plaque is visualized in the mid to distal esophagus. 1c. Narrow band imaging details the white plaque in the distal esophagus.

**S2325**

**Get It Out: Hydrogel Capsules Impacting in the Esophagus**

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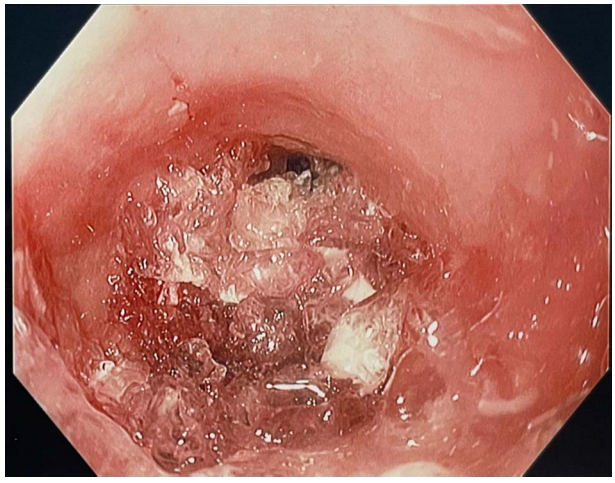
*Icahn School of Medicine at Mount Sinai, NYC Health + Hospitals/Elmhurst, Elmhurst, NY.*

**Introduction:** Hydrogel capsules are FDA-approved superabsorbent agents used for weight loss. Typically, 3 capsules are swallowed with water before eating. When in contact with water, the particles inside the capsules expand a hundredfold into an elastic gel-like structure that creates a satiated feeling in the user and promotes fullness. The capsular contents are not absorbed into the bloodstream. The most common side effects are bloating, flatulence, and abdominal pain (1-2). In this case, we discuss a patient who experienced dysphagia from pill impaction in the esophagus.
**Case Description/Methods:** A 39-year-old female with eosinophilic esophagitis (EoE) presented following impaction of 3 cellulose/citric acid hydrogel capsules taken all at once before eating as the instructions stated. She reported feeling the capsules stuck in her esophagus and she was unable to tolerate swallowing. She has had issues with dysphagia in the past, but these episodes typically improved with drinking water. At the time of presentation, she was not taking any medications for EoE. Esophagogastroduodenoscopy (EGD) was performed and demonstrated gelatinized capsules within the esophagus (**Figure**) at 20 cm and extending 5 cm distally. The impaction was broken down with graspers and retrieval devices, and the remaining material was pushed into the stomach. Due to the gelatinous content, it was extremely challenging to capture the loose material to be removed. Biopsies were taken of the esophagus which demonstrated mild to moderate eosinophilic infiltration.
**Discussion:** For some individuals, hydrogel capsules can be useful adjuncts for weight loss. However, esophageal impaction and dysphagia can occur if not swallowed properly. To reduce this risk, users should consider swallowing each of the 3 capsules individually, followed each time by a glass of water. This method of administration will likely prevent the chance of the expansion of the capsular contents into the larger gelatinous material in the esophagus and encourage it to occur in the stomach. People with dysphagia, diverticula, and other motility disorders should be extremely careful and take these gelatinous capsules individually.



[2325] **Figure 1.** Gel-like content in the esophagus from cellulose/citric acid hydrogel capsules.

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**S2326**

**Gastroesophageal Junction Adenocarcinoma With Metastases to the Dorsum of the Tongue: A Rare Case Report**

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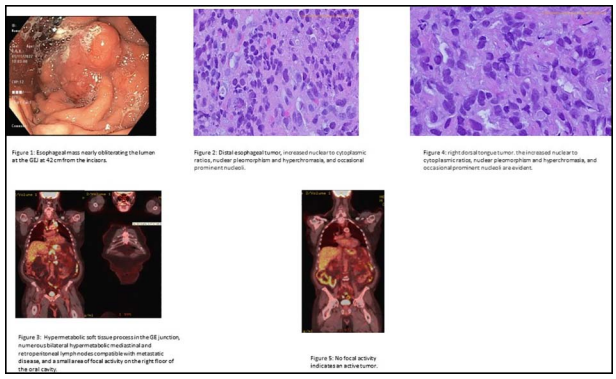
*[1]Albany Medical College, Albany, NY; [2]KHCC, Albany, NY; [3]Albany Stratton VA Hospital, Albany, NY.*

**Introduction:** Gastroesophageal Junction (GEJ) cancer is an invasive disease with a poor prognosis and commonly presents at an advanced stage. Metastases of GEJ cancer are most frequently seen in the lung, liver, and distant lymph nodes. Here, we are reporting for the first time in the literature metastatic GEJ adenocarcinoma (AC) to the oral cavity.
**Case Description/Methods:** A-72-year-old male presented with severe dysphagia to solids and liquids, and weight loss for 1 month. Evaluation with upper endoscopy revealed a tumor nearly obliterating the lumen at the GEJ, 42 cm from the incisors (**Figure** A). Biopsy confirmed the diagnosis of AC of the GEJ (**Figure** B), and genetic testing showed: MSI: Stable, PDL 1%, and HER-2: negative. The patient was evaluated further Positron emission tomography (PET) scan which showed a hypermetabolic soft tissue process in the GEJ, numerous bilateral hypermetabolic mediastinal and retroperitoneal lymph nodes, and interestingly a small area of focal activity on the right floor of the mouth (**Figure** C). Biopsy of the tongue lesion confirmed stage IV AC of GEJ origin (**Figure** D). The case was then discussed in our multidisciplinary tumor board. The decision was to proceed as per NCCN guidelines with palliative radiation therapy (RT) for severe dysphagia and sequentially FOLFOX (5-fluorouracil, leucovorin, and oxaliplatin) chemotherapy, the patient did not receive immunotherapy as his PD-L1 is 1%. PET scan after completion of radiotherapy and 5 cycles of FOLFOX showed no evidence of disease (**Figure** 5).
**Discussion:** GEJ cancer is a devastating disease that commonly presents at an advanced stage and bears a dismal prognosis. The metastatic pattern of this disease frequently involves the liver, lung, distant lymph nodes, peritoneum, and bone. Studies have shown 2 major differences in the metastatic pattern according to the histological subtype; AC tends to metastasize to the liver, peritoneum, and bone. Whereas squamous cell carcinoma most commonly metastasizes to the lungs. However, the overlap between both categories exists. To the best of our knowledge, we are reporting for the first time in the literature GEJ AC with metastasis to the oral cavity. Management of patients with advanced, unresectable GEJ cancer represents a challenging scenario. External beam radiation therapy with concurrent chemotherapy is the standard approach for patients with unresectable disease. Our patient had a wonderful response both clinically and radiologically with negative follow-up FDG PET.



[2326] **Figure 1.** A: Esophageal mass nearly obliter 1-5.

**S2327**

**Gurvits Syndrome in Setting of Uncontrolled Diabetes and Acute Gastrointestinal Bleeding Successfully Treated with Extended Duration PPI Despite Continuing DOAC**

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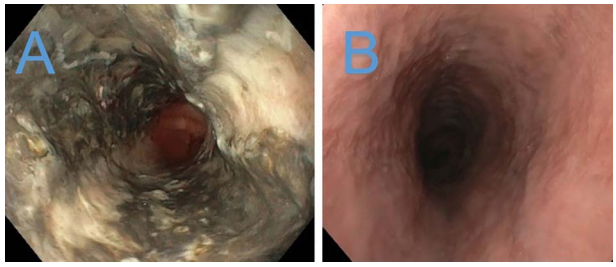
*Arnot Ogden Medical Center, Elmira, NY.*

**Introduction:** A young male with past medical history (PMH) of severely uncontrolled non-insulin dependent diabetes mellitus (NIDDM) with recurrent admissions for diabetic ketoacidosis (DKA) and hypertriglyceridemia-induced pancreatitis, both secondary to medication non-adherence, presented via emergency medical services (EMS) after being found unresponsive at home and subsequently developed acute esophageal necrosis (AEN) during a prolonged stay in the intensive care unit (ICU) for acute respiratory failure. He was successfully treated with extended duration of proton pump inhibitor (PPI) therapy and control of underlying risk factors despite being continued on anticoagulation.
**Case Description/Methods:** A 39-year-old male with PMH of uncontrolled NIDDM presented was admitted for acute respiratory failure secondary to DKA and pneumonia. He was intubated and admitted to ICU, while continued on DKA protocol including antibiotics. GI was consulted for hematemesis and melena 2 weeks into hospitalization. EGD revealed circumferential black esophageal mucosa in the distal esophagus abruptly ending at the gastroesophageal junction (GEJ) and hiatal hernia. Recommendations included Intravenous (IV) proton pump inhibitor (PPI) and strict avoidance of naso/orgastric (NG/OG) tube. Follow up EGD few months after discharge showed remarkable improvement of mucosa as noted in Figures A and B, despite continuation of DOAC as per patient wishes for DVT developed during the stay.
**Discussion:** AEN is a rare syndrome disproportionately reported in men which is characterized by circumferential black esophageal mucosa in the distal 2 thirds of the esophagus, abruptly ceasing at the GEJ. Ischemia is postulated to be an inciting event. Conditions associated with AEN are antibiotics, sepsis, gastric volvulus, hernia, DKA, malignancy, and prolonged vomiting. Symptoms of upper gastrointestinal bleeding (UGIB) and shock are common presentations. Although biopsy establishes diagnosis and rules out other causes, EGD finding is generally sufficient. Initial management consists of IV fluids and treatment of the underlying cause. IV PPI and *nil per os* (NPO) is recommended. NG/OG tubes are avoided unless vomiting or obstruction is present. Mortality is largely due to underlying disease rather than directly from AEN, hence supportive care results in resolution in most cases, as seen in our patient. Our case demonstrates the importance of addressing underlying causes, as well as PPI therapy, which can overcome continued anticoagulation.




[2327] **Figure 1.** A. Acute esophageal necrosis discovered on endoscopy while undergoing EGD for acute GI bleeding during ICU admission for DKA and acute respiratory failure requiring mechanical ventilation B. Resolution of acute esophageal necrosis with grossly normal mucosa on follow up endoscopy conducted after extended duration PPI therapy and relatively controlled diabetes.

**S2328**

**Gurvit Syndrome: An Unusual Presentation of Upper Gastrointestinal Hemorrhage in a Case of Hypovolemic Shock**

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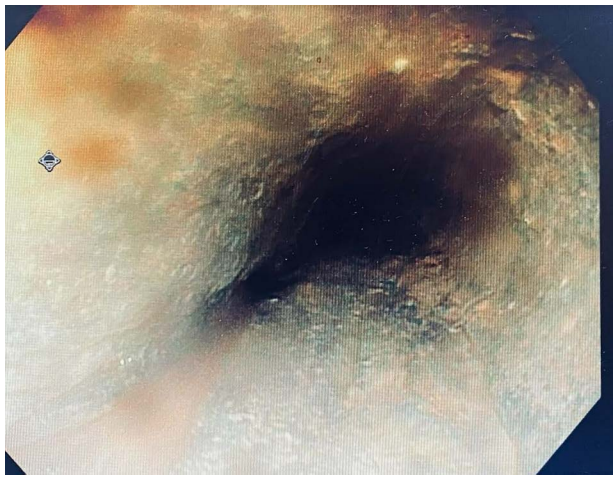
*[1]Arnot Ogden Medical Center, Ijamsville, MD; [2]Arnot Ogden Medical Center, Elmira, NY; [3]LECOM, Plainview, NY.*

**Introduction:** Acute Esophageal Necrosis (AEN) is a rare disorder with a high associated mortality often seen in the setting of ischemia is considered the most plausible etiology. Other associated conditions include coronary artery disease, gastric outlet obstruction, infection, trauma, and alcohol ingestion. Diagnosis is made endoscopically, and findings are typically characterized as circumferential, macerated mucosa appearing black. We present a case of a 69-year-old female who was admitted for diabetic ketoacidosis, who ultimately developed acute hypoxemic respiratory failure and upper gastrointestinal hemorrhage, and was found on upper endoscopy, to have distal esophageal necrosis.
**Case Description/Methods:** A 69 y/o female with a past medical history notable for poorly controlled type 2 diabetes mellitus, pulmonary hypertension, systolic heart failure (EF 40-45%) presented to the emergency room with complaints of intractable nausea and vomiting. Upon initial lab workup, she was noted to have a hemoglobin of 17.3, MCV 101, WBC 37.9, Platelets 275K, sodium 138, potassium 6.5, chloride 103, bicarbonate 17, glucose 241, BUN 97, creatinine 2.3, albumin 2.3, total bilirubin 0.4, alkaline phosphatase 73, AST 2985, ALT 1945, LDH 8074. PT 26.1, INR 2.3, lactic acid 10.3. She was sent to the ICU and managed for DKA with anion gap of 25 on arrival with concomitant hypoxemic respiratory failure. Within 24 hours the patient became hypotensive requiring maximal pressor support and was eventually intubated. Two hours later she was noted to have 1200 mL of dark black material suctioned from her NG tube, and was noted to be passing melenic stool. Repeat hemoglobin was noted to be 5.4g/dL. An EGD was performed, which showed a necrotic appearing esophagus involving the distal 2/3 of the esophagus (**Figure**). A distorted pyloric channel with large ulceration encompassing the entire duodenal bulb. The patient was continued on maximal pressor support but despite medical intervention, she ultimately expired 6 hours later.
**Discussion:** Patients with AEN often have hemodynamic compromise resulting in ischemia. Esophageal arterial supply differs based on region. The proximal and mid esophagus are supplied from inferior thyroid artery, and bronchial aa. The distal esophagus is supplied by the left gastric artery and has a relatively poor collateral supply. Management involves adequate hemodynamic support, intravenous acid suppression. Unfortunately, overall mortality of AEN remains high, estimated to be 32%.



[2328] **Figure 1.** Necrotic appearing distal esophagus

**S2329**

**Iatrogenic Cushing's Syndrome in Eosinophilic Esophagitis: A Rare Complication of Swallowed Topical Corticosteroids**

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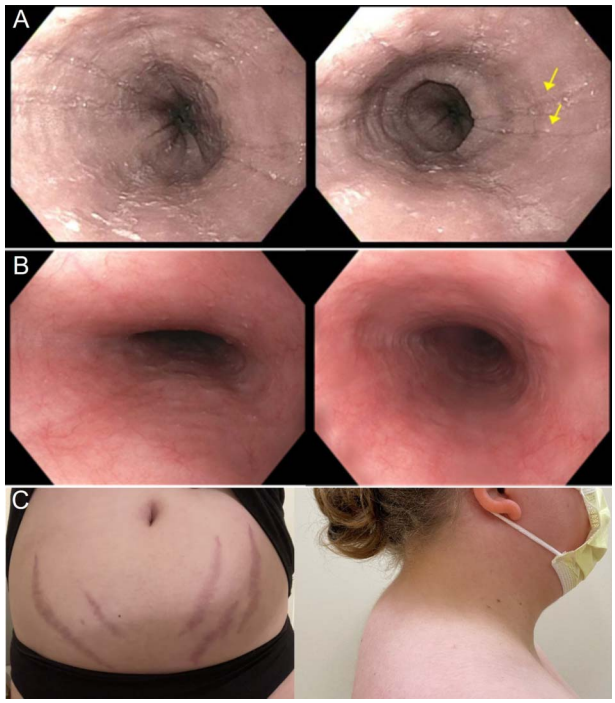
*University of Michigan School of Medicine, Ann Arbor, MI.*

**Introduction:** Topical corticosteroids (TCS) are a first-line treatment for eosinophilic esophagitis (EoE), effective at inducing and maintaining disease remission and well-tolerated with uncommon medication-related adverse events. Despite the theoretical risk of adrenal suppression (AS) with prolonged TCS use, data supporting clinically significant adrenal insufficiency in EoE are lacking and the risk of this rare complication is thought to be low. We present a case of iatrogenic Cushing's syndrome associated with chronic TCS therapy in EoE and an opportunity for steroid-sparing treatment.
**Case Description/Methods:** A 27-year-old woman with a 7-year history of EoE refractory to proton pump inhibitor, 6-food elimination diet, and swallowed fluticasone (FP) achieved disease remission with high-dose oral viscous budesonide (OVB) 4mg. Rapid recurrence of dysphagia, endoscopic findings, and esophageal eosinophilia occurred with lower OVB doses. After FP use for 5 years and transitioning to OVB for 18 months, she developed cushingoid features including prominent pathologic striae on the abdomen, breasts, and thighs, facial rounding, supraclavicular fullness, central obesity, and a dorsocervical hump. Given known exposure to exogenous steroids, features consistent with iatrogenic Cushing's syndrome, and assumed hypothalamic-pituitary-adrenal axis (HPAA) suppression, the patient began a taper of OVB and steroid-sparing treatment was initiated with dupilumab 300mg weekly. After tapering OVB to 2mg daily over 2 months, assessment (ACTH, morning cortisol, 24-hour urine cortisol) was normal, consistent with HPAA recovery (**Figure**).
**Discussion:** Topical steroids such as OVB are commonly used and recommended for long-term management of EoE with rare reports of adrenal insufficiency. As data supporting biochemical AS with TCS use in EoE is limited to observational studies with heterogenous testing for AI and lack of symptom assessment, the risk of clinically relevant AS is likely low. However, our case demonstrates that despite the favorable pharmacokinetics, systemic effects of TCS may be understated, particularly with high compliance and prolonged use of high daily doses. Clinicians should counsel patients on the non-negligible risk of AS when prescribing TCS for EoE, and consider assessment of the adrenal axis or use of steroid-sparing agents in those reliant on chronic TCS for disease control.




[2329] **Figure 1.** Endoscopic evidence of (B) active disease on FP (B) disease remission on OVB 4mg daily; (C) Cushingoid features of abdominal striae and dorsocervical hump.

S2330

**Holey Esophagus: An Indeterminate Case of a Fenestrated Esophagitis in an Immunocompromised Host**

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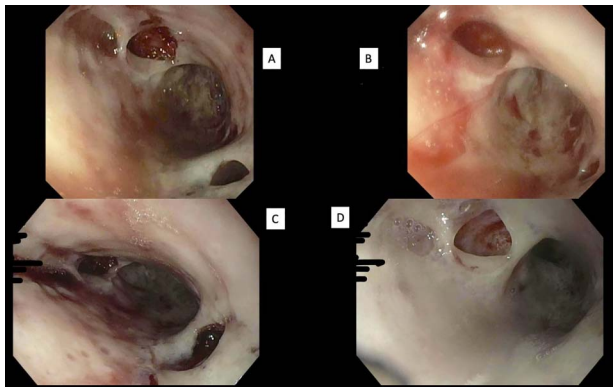
*New York Presbyterian Brooklyn Methodist Hospital, Brooklyn, NY.*

**Introduction:** Immunocompromised patients have increased risk of developing ulcerative esophagitis. The most common etiologies of ulcers are radiation induced, chemotherapy, and opportunistic infections. HSV esophagitis has yellow exudate and discrete, coalescent ulcers, while CMV esophagitis typically has large, shallow, solitary or multiple ulcers located in the mid to distal esophagus. Chemotherapy with radiation therapy and concurrent Gemcitabine and Cisplatin has been reported in up to 52% of severe esophagitis. We present a case of idiopathic esophagitis with a unique endoscopic appearance of multiple fenestrations throughout the esophagus.
**Case Description/Methods:** A 66-year-old female with past medical history significant for stage 1A Uterine Serous Carcinoma presented to the ED with 2 days of hematemesis. She reports multiple episodes of emesis with each one subsequently becoming increasingly more bloody. She had received her 5th cycle of Cisplatin and Gemcitabine 2 days prior to presentation. She underwent an esophagogastroduodenoscopy (EGD) with unique findings of severe inflammation with exudative plaques with fenestrated appearance and oozing of blood. There was initial concern for esophageal perforations, diverticulosis and fistulas however these did not match the clinical presentation. Biopsies were consistent with active esophagitis with necroinflammatory debris consistent with ulcer. Gastrografin esophagram showed no evidence of contrast extravasation ruling out fistulous connections. Stains for CMV and HSV were inconclusive despite adequate tissue sampling (**Figure**).
**Discussion:** Given the gross appearance of the esophagus on EGD and lack of CMV immunohistochemical conclusivity, the etiology of ulceration in this case is unclear. The most common endoscopic findings of CMV esophagitis are mucosal ulcerations typically located in the mid to distal esophagus. Diagnosis is made by immunohistochemical visualization of cytomegalovirus inclusion bodies within the biopsied tissue. Patients who receive chemotherapy may experience post-infusion related mucositis in addition to immunosuppression. Mucositis occurs via diffuse non-specific chemotherapeutic targeting of tissues with high cell turnover. In our case, the ulceration is focal from the mid to distal portion of the esophagus, more reflective of CMV opposed to the diffuse involvement anticipated in mucositis. We hypothesize that the atypical fenestrated appearance of esophageal mucosa may be a consequence of mucositis with superimposed CMV infection.




[2330] **Figure 1.** Image: (A, B, C, D) showing the mid to distal esophagus with multiple shallow ulcers of varying sizes. (C) appears to have oozing of blood and friability. No obvious evidence of fistula, perforation, or diverticulum noted.

S2331

**High-Potency Proton Pump Inhibitor Therapy for the Management of Chronic Hiccups**

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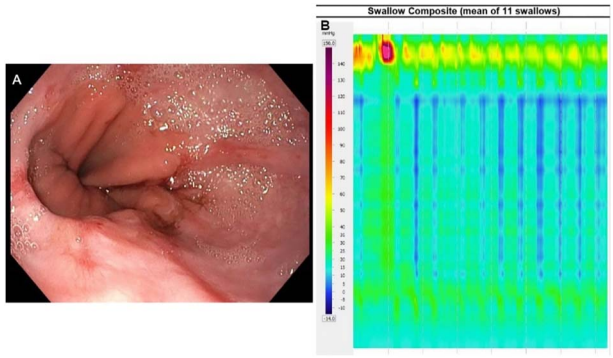
**Introduction:** Chronic hiccups can be distressing and may substantially reduce quality of life. Gastroesophageal acid reflux is in the differential for underlying non-neuroleptic and non-respiratory causes of intractable symptoms. However, proton pump inhibitor (PPI) therapy is often underdosed or mistimed, promoting medication abandonment or polypharmacy. Alternative pharmaceutical therapies are associated with side effects that may lead to eventual patient intolerance.
**Case Description/Methods:** A 67-year-old male presented to gastroenterology clinic with 2 years of hiccups and frequent nocturnal awakening. Additional symptoms included intermittent oral regurgitation without pyrosis, nausea, vomiting, or chest/abdominal pain. Prior therapies consisted of chlorpromazine and once daily pantoprazole without benefit. In addition, the patient was previously prescribed baclofen, which provided incomplete relief. Initial CBC, CMP, and thyroid function tests were unremarkable. Index radiographic imaging showed a patulous esophagus. High resolution esophageal manometry (HREM) was nondiagnostic due to hiccups occurring during the study. Ambulatory pH/impedance testing, performed off acid suppression therapy, demonstrated elevated DeMeester score of 49.8, Acid Exposure Time (AET) of 13.2%, an elevated proportion of weakly acidic refluxate, and low mean nocturnal baseline impedance, consistent with reflux. Next, the patient underwent EGD, where a 1 cm hiatal hernia, regular Z-line, and Los Angeles Grade D esophagitis were discovered. Non-bleeding erosions were found in the distal stomach, and gastric biopsies returned negative for intestinal metaplasia or Helicobacter pylori. He was recommended to continue baclofen, start colestipol 1 gm daily, and start omeprazole 40 mg twice daily for 3 months. Five months after his initial visit, the patient reported improved symptoms with resolution of nocturnal episodes. He was planned for a surveillance EGD after 3 months of twice daily PPI therapy (**Figure**).
**Discussion:** Chronic hiccups are atypical symptoms of gastroesophageal acid reflux. Important diagnostic steps include ruling out alternative etiologies, in order to tailor specific therapy. PPI are frequently prescribed but are often underdosed or lower potency options (eg. pantoprazole) are utilized. Our case suggests a limited trial of high-potency twice daily PPI therapy is effective and should be initially considered for chronic hiccups from an acid reflux etiology.




[2331] **Figure 1.** A) Endoscopic image of distal esophagus and hiatal hernia. B) High-resolution esophageal manometric composite image.

**S2332**

**HSV Esophagitis With Hematemesis and Recalcitrant Food Bolus**

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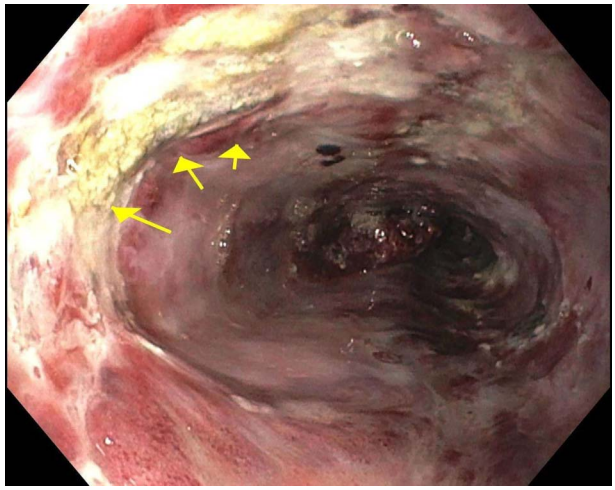
[1]University at Buffalo, Buffalo, NY; [2]Roswell Park Comprehensive Cancer Center, Buffalo, NY.

**Introduction:** HSV esophagitis (HSV-E) affects primarily immunosuppressed individuals. Common symptoms include fever, odynophagia, dysphagia and retrosternal chest pain. Rare manifestations include hematemesis and food bolus. We present a 58 year-old woman with AML refractory to hematopoietic stem cell transplants (HSCTs) who developed HSV-E with hematemesis and food bolus.
**Case Description/Methods:** A 58 year-old woman presented to an outside hospital with acute dysphagia and hematemesis. Her past medical history includes refractory AML with HSCTs in 2007, 2010 and 2019 and GI graft-versus-host-disease (GVHD) on talazoparib and gemtuzumab. EGD reported possible hematoma in the entire esophagus with superficial mucosal tears. Biopsies were negative for viral cytopathic effect or malignancy. She was transferred to our hospital for NK cell infusion for AML. She had a prolonged hospital course with neutropenic fever and sepsis and was on prophylactic acyclovir. She had dysphagia and EGD week 1 of her admission showed ulcerated, hemorrhagic esophageal mucosa with positive HSV biopsies (**Figure**). She was given IV foscarnet, acyclovir, pantoprazole and sucralfate. EGD was repeated week 3 for persistent dysphagia and odynophagia. Her esophagitis improved and biopsies were again HSV-positive. She had recurrent dysphagia and hematemesis at week 5. An updated EGD showed a massive esophageal food bolus that could not be cleared despite a 4 hour procedure. She was given a trial of Coca-Cola mixed with Creon to help dissolve the bolus. A 4th EGD was done 4 days later. This cleared the bolus after 4 more hours of procedure time. EGD was repeated at week 13 for foreign body sensation and showed mild sloughing of mucosa at 22 cm but the mucosa was otherwise healed. Biopsies showed squamous esophageal mucosa with chronic inflammation (HSV-negative). She developed recurrent colonic GVHD treated with immunosuppressants. She continued HSV prophylaxis with valganciclovir and did not develop recurrent HSV-E despite ongoing immunosuppression.
**Discussion:** HSV-E was first described in 1943 and only 1 case of HSV esophagitis presenting with food bolus has been reported. HSV-E most commonly affects immunosuppressed patients, including HSCT patients. The highest risk for HSV reactivation appears to be within 30 days of HSCT (67% of reinfections). Age >50 is associated with reactivation higher risk and HSV reactivation in the setting of cancer is associated with decreased overall survival.



[2332] **Figure 1.** Upper GI endoscopy image from week 1 showing hemorrhagic and ulcerated (arrows) esophageal mucosa.

**S2333**

**Herpes Simplex Esophagitis Masquerading as Reflux Esophagitis**

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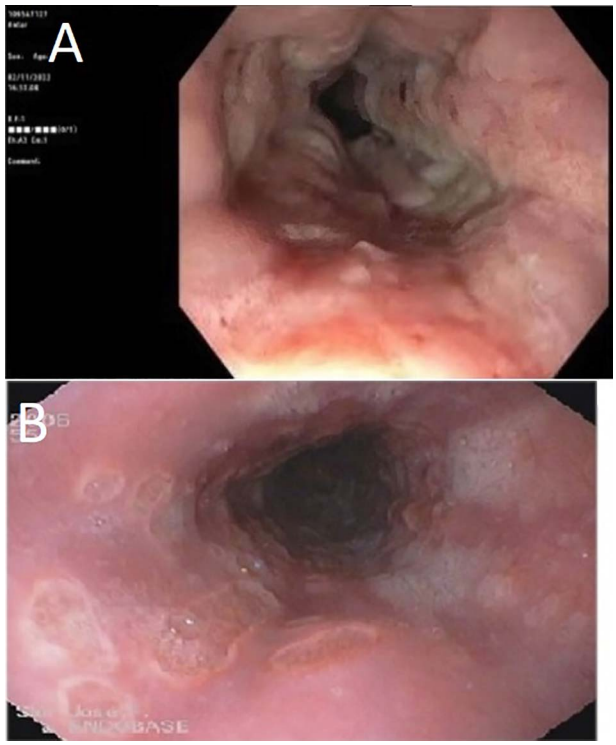
**Introduction:** Herpes simplex esophagitis (HSE), caused by the herpes simplex virus (HSV), is the 3rd most common cause of esophagitis behind GERD and candida infection. It is seen predominantly in hosts with impaired immunity. HSE may result from reactivation of HSV with spread of the virus to the esophageal mucosa by way of the vagus nerve or by direct extension of oral-pharyngeal infection into the esophagus. Most commonly caused by HSV-1 although HSV-2 has occasionally been reported. Patient's usually present with odynophagia and/or dysphagia. Lesions are typically found in the lower third of the esophagus and are well circumscribed with a volcano-like appearance.
**Case Description/Methods:** A 78-year-old male with past medical history of CLL, metastatic prostate cancer and MGUS presented with complaints of odynophagia and dysphagia. The patient's prostate cancer was actively being treated with chemotherapy (Leuprorelin and Docetaxel) so he was empirically started on fluconazole for presumed candida esophagitis. The patient's labs were not indicative of severe immunosuppression with a normal WBC of 9.3 and ANC of 6.8. He underwent EGD which did not show the characteristic HSE lesions described by Itoh et al. Instead, the patient's lesions were very typical of

reflux esophagitis (**Figure** A). However, due to his immunocompromised state, biopsies were sent to check for opportunistic infections. Histology confirmed the diagnosis of HSE and the patient was treated with acyclovir for a total of 21 days. He improved drastically and was able to tolerate diet. He was discharged and scheduled for follow up EGD in 3 months to confirm normalization of the esophagus.
**Discussion:** Endoscopic findings of HSE have been characterized into type I, II and III by Itoh et al. Types I and II show small punched-out lesions with and without raised margins, respectively (**Figure** B). Type III is defined when multiple ulcers became confluent like a map. Vesicular lesions are common in the early stages and exudative lesions are present in most cases. Mucosal necrosis can be seen in the later stages. Our case is interesting because the endoscopic findings were not typical of HSE. The patient was diagnosed with LA grade C reflux esophagitis before pathology confirmed the diagnosis of HSE. This case shows us that opportunistic infections should always be considered in immunocompromised patients even if gross endoscopic appearance does not suggest an infectious cause of esophagitis.




[2333] **Figure 1.** A - EGD images of our patient , diagnosis LA grade C reflux esophagitis. B - EGD images of typical herpes simplex esophagitis.

**S2334**

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**Incidentally Diagnosed Triple Cancers Within 2 Years: A Novel Case of Rare Metachronous Cancers, Renal Cell Carcinoma, Cholangiocarcinoma, and Squamous Cell Carcinoma of the Gastroesophageal Junction**

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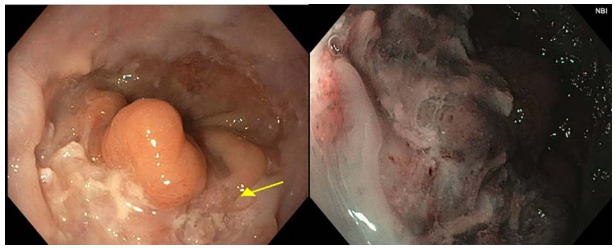
**Introduction:** The gastrointestinal (GI) tract is involved in many familial cancer syndromes usually involving the colon or stomach. We present a patient with 3 rare cancers that were detected within a span of 2 years in a single individual: clear cell renal cell carcinoma (ccRCC), cholangiocarcinoma (CCA), and esophageal squamous cell carcinoma (SCC); a combination not previously described in the literature
**Case Description/Methods:** A 50-year-old male with a history of ccRCC, GERD, and CCA was admitted for evaluation of chronic abdominal pain during chemotherapy for CCA. Regarding his cancer history, ccRCC was incidentally diagnosed 18 months prior on a CT scan for diverticulitis. He underwent a left partial nephrectomy. Surveillance CT imaging 6 months after surgery revealed new incidental liver lesions. Biopsies confirmed metastatic CCA and he started chemotherapy. He noted a 15-year history of episodic abdominal pain and vomiting with no dysphagia or weight loss. Upper endoscopy 16 months prior was normal. He was a former smoker, drank alcohol a few times per month, used marijuana daily, and used cocaine rarely. His family history was significant for multiple cancers at a young age on his maternal side of unknown type. Physical exam showed diffuse abdominal tenderness, worse in the epigastrium without guarding. Labs showed mild anemia, normal BMP/LFTs, and negative *H. pylori* stool antigen. Upper endoscopy found nodularity adjacent to the esophagogastric junction (EGJ) with abnormal pit pattern under narrow band imaging (**Figure**). Biopsies confirmed invasive SCC. Staging endoscopic ultrasound showed T3N3 EGJ SCC. He was referred to oncology to discuss treatment options. Genetic testing was positive for BAP1 tumor predisposition syndrome (BAP1-TPDS)
**Discussion:** A review of published literature did not yield a previous description of this combination of cancers in a single individual. BAP1-TPDS is an uncommon autosomal dominant syndrome caused by germline mutation in a tumor suppressor gene. Patients typically present with melanocytic tumors or malignant mesotheliomas but are at risk for RCC, hepatocellular carcinoma, meningioma and CCA. Literature has not revealed esophageal SCC to be associated. In addition to the incidental nature and timing of diagnosis of all 3 of these cancers within a short period of 2 years, the EGJ involvement is unusual. This case suggests atypical EGJ-SCC may be considered as part of the BAP1-TPDS tumor spectrum and warrant additional screening for at-risk individuals.



[2334] **Figure 1.** Left panel showing abnormal nodular mucosa adjacent to the gastro-esophageal junction under high-definition white light imaging. Right panel showing the same area under narrow band imaging.

S2335

**Large Esophageal Papilloma Causing Dysphagia Treated Successfully With Endoscopic Mucosal Resection**

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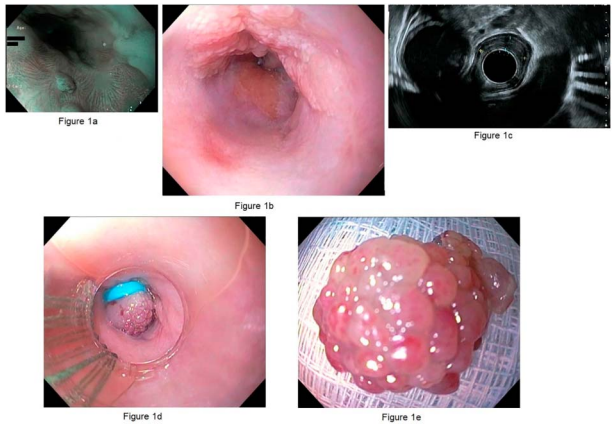
**Introduction:** Esophageal squamous papillomas (ESP) are rare, small, benign epithelial lesions most often found incidentally in asymptomatic patients. Majority of cases occur in the distal esophagus where chronic mucosal injury from the gastric refluxate of gastroesophageal disease can consistently and chemically irritate the mucosa. Diagnosis of ESP often requires biopsy although endoscopy usually demonstrates a < 6mm, solitary lesion with a triad of exophytic growth, verrucous projections, and surface crossing vessels evident on narrow-band imaging (NBI). Here we discuss a rare presentation of dysphagia secondary to a large, partially circumferential esophageal squamous papilloma.
**Case Description/Methods:** A 72-year-old woman was referred to our institution for upper endoscopic ultrasonography (EUS) with possible endoscopic mucosal resection (EMR) for suspected upper GI lesion. Previously, the patient underwent endoscopy for dysphagia and was noted to have an ulcer at 30 cm from incisors. Despite proton-pump inhibitor (PPI) use, the dysphagia did not improve. A repeat esophagogastroduodenoscopy (EGD) was performed which revealed a polypoid lesion in the lower esophagus 34 cm from incisors without an ulcer. The lesion involved 50%-60% of the esophageal lumen extending from 34 cm to 36 cm from incisor, about .5 cm above the gastroesophageal (GE) junction. Part of the lesion was suctioned and a band was deployed. Hot snare polypectomy was performed and band assisted EMR was repeated for the remainder of the lesion. The tissue was sent for histopathological examination which demonstrated squamous papilloma without dysplasia or malignancy (**Figure**).
**Discussion:** Esophageal squamous papilloma is a rare and often incidental finding on endoscopy in asymptomatic patients. Prevalence of ESPs in the general population is estimated to be less than 0.01%. Practitioners should maintain a high level of suspicion for esophageal squamous papillomas in adult patients who present with symptoms of gastroesophageal reflux disease and dysphagia who are unresponsive to proton pump inhibitors. It is critical to rule out other differentials such as squamous cell carcinoma, papillary leukoplakia, polyps, leiomyoma, malignant melanoma, and ultimately rule out ESP with biopsy of the lesion. First-line treatment has not been solidified, but removal of the lesion is preferred for symptomatic patients and to decrease the potential of malignant transformation.




[2335] **Figure 1.** 1a: Endoscopic narrow-band imaging demonstrating a solitary 2 mm lesion with crossing surface vessels in the distal esophagus **Figure** 1b: EGD revealing a verrucous, polypoid lesion at 340 mm from incisors **Figure** 1c: EUS revealing an isoechoic mass in the distal esophagus involving half of the lumen circumference without invasion into the submucosa or muscularis propria. **Figure** 1d: Band assisted endoscopic mucosal resection of the lesion **Figure** 1e: Removed verrucous lesion

S2336

**It Was There All Along**

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**Introduction:** Dysphagia Lusoria is used to describe trouble swallowing due to an aberrant right subclavian artery and its subsequent compression of the esophagus. The frequency of an anomalous subclavian artery in the general population is approximately 0.5-1.8%. It is considered a very rare anomaly and is usually diagnosed via barium swallow or chest CT.
**Case Description/Methods:** This is the case of a 78 year old male with a past medical history of hypertension and diabetes mellitus type II who was referred to the gastroenterology clinic due to dysphagia with solids and liquids for about 8 years. The only alleviating factor identified was eating smaller foods. During this same time frame, the patient had been exhibiting episodes of chest pain that required evaluation by cardiology service. However, after a thorough workup, cardiology service deemed the patient's presentation as non- cardiac in origin. Patient underwent his first EGD as part of his cardiac evaluation; this was found to be unremarkable. EGD performed later as part of dysphagia evaluation was found to be unremarkable as well. As a result, manometry was scheduled and this revealed a pulsatile high pressure zone at 33cm from incisures in the setting of normal lower esophageal sphincter pressures and normal esophageal peristaltic motility. Subsequent investigation with chest CT displayed a left-sided aortic arch with aberrant origin of the right subclavian artery and bolus dilatation of its origin as seen with a Kommerell diverticulum. These findings were re-demonstrated with barium swallow. Radiological studies and manometry findings, along with an unremarkable upper endoscopy and cardiovascular workup, suggest that etiology of chest pain and dysphagia is an aberrant origin of the right subclavian artery appropriately referred to as dysphagia lusoria.
**Discussion:** This case sheds light on a rather rare anomaly which should be considered as part of the differential diagnoses of dysphagia or atypical non cardiac chest pain, as some cases may merit vascular intervention. Also, being aware of this condition may avoid unnecessary studies and lead to more prompt diagnosis.

S2337

**Large Cell Neuroendocrine Carcinoma of the Esophagus: A Case Report**

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**Introduction:** Large cell neuro-endocrine tumors (NECs) of the esophagus are rare, high-grade tumors with poor long-term prognosis. They are aggressive and metastasize easily compared to adenocarcinomas. Here we present a case of large cell NEC of the esophagus who initially presented with respiratory failure.
**Case Description/Methods:** A 70-year-old male presented to ER with acute respiratory failure requiring intubation with mechanical ventilation. His hospital course was complicated by renal failure and gastrointestinal (GI) bleeding. To evaluate this GI bleed, patient underwent esophagogastroduodenoscopy and colonoscopy which showed a 3 cm ulcerated nonobstructive mass at the gastroesophageal junction which was biopsied. Pathology showed a poorly differentiated NEC, large cell type with a Ki-67 >80%. Immunohistochemistry showed positive CK7, synaptophysin, chromogranin and patchy CD 56+ cells. CK20 was negative. PET scan showed extensive mediastinal lymphadenopathy and right-sided eighth rib metastasis without primary lung lesion. Patient was started on carboplatin, etoposide, and atezolizumab and is currently under treatment.
**Discussion:** Neuroendocrine tumors are poorly differentiated, high-grade malignant neoplasms diffusely expressing the general markers of neuroendocrine differentiation (synaptophysin, faint or focal staining for chromogranin A) with marked nuclear atypia, multifocal necrosis and a high number of mitoses ( >20 per 10 high-power fields). Large cell NECs of the esophagus are extremely rare subtype originating from Merkel cells. These cells are mostly concentrated in the mid-esophagus but may also arise from endocrine cells in the esophageal cardiac glands present in the distal esophagus. 2017 WHO classification divides

these neoplasms into well differentiated (grade 1, 2 or 3) or poorly differentiated NEC's (small or large cell) based on tumor's Ki67% values, grading and mitotic index. Large-cell tumors also consist of cells with solid nests or acinar structures, and a low nuclear/cytoplasm ratio. Pathology is the gold standard for establishing diagnosis. Immunohistochemistry staining for NECs is usually negative for p40, p63, CK5/6 while squamous cell carcinomas express them. Compared to NECs, adenocarcinomas typically express napsin A while former do not. Though robust survival data is lacking, prognosis is usually poor, with a 2-year survival of < 20% and a median overall survival of 5 months. There is no established treatment for NEC.

**S2338**

**Incredibly Unique Case of Collagenous Esophagitis in a Long Segment Barret's Esophagus: Complication of Chronic Reflux or a New Entity?**

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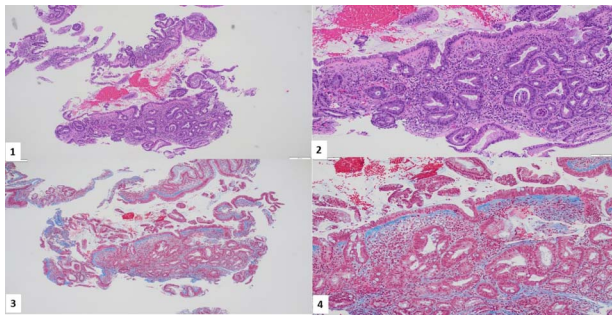
**Introduction:** Collagenous gastroenteritides including collagenous gastritis, collagenous sprue, and collagenous colitis, are all uncommon entities. Esophageal lamina propria fibrosis has been established in eosinophilic esophagitis as a long-term consequence of remodeling. The esophageal subepithelial collagen deposits can be regarded as collagenous esophagitis which is a new entity. We herein report a case of concurrent collagenous carditis and collagenous esophagitis in a patient with Barrett's esophagus (BE).
**Case Description/Methods:** A 36-year-old man with complex medical history of cerebral palsy, seizure disorder, hypothyroidism, chronic gastroesophageal reflux disease, and erosive esophagitis developed benign esophageal intrinsic stenosis at 23 cm from the incisors and BE in the lower third of the esophagus within 10-year period, which necessitated 2 endoscopic balloon dilatation procedures. Of note, he did not receive radiofrequency ablation or mucosal resection therapies at any point of time. Subsequent 3 endoscopies and esophageal and cardiac biopsies away from the stricture (between 25-33 cm from the incisors) demonstrated BE indefinite for dysplasia. The most recent one in addition to intestinal metaplasia had a new finding of lamina propria fibrosis with diffuse band-like subepithelial collagen deposits more than 10 μm in thickness, confirmed by Trichrome stains. Congo red stains were negative. Features of eosinophilic esophagitis were absent. These findings were compatible with collagenous esophagitis and collagenous carditis diagnosis (**Figure**).
**Discussion:** Collagenous gastritis is an extremely rare and poorly understood process with female and young adult predominance. The adult-onset collagenous gastritis is antrum dominant and is associated with collagenous colitis and autoimmune disorders, such as celiac disease, thyroid disorder, and others. Our patient had collagenous carditis with collagenous esophagitis in the background of BE, which has never been described before.




[2338] **Figure 1.** Biopsy stains.

**S2339**

**Incidental Acute Esophageal Necrosis in a Post-Cholecystectomy Patient**

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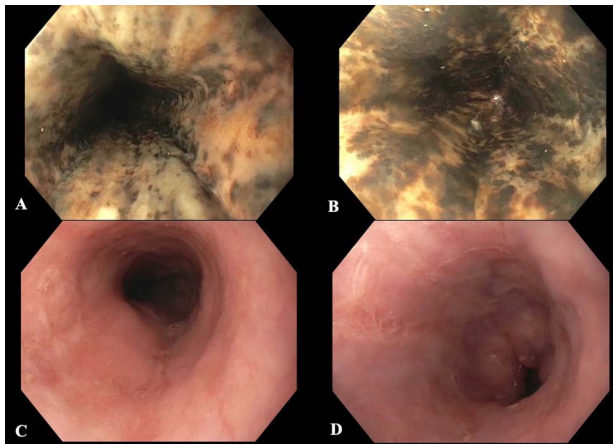
**Introduction:** Acute esophageal necrosis (AEN), or "black esophagus", is a rare clinical entity characterized by diffuse, black mucosal discoloration of the esophagus on endoscopy. Patients often present with hematemesis, shock, and melena. The condition has a relatively high mortality rate due to the comorbid conditions that are frequently present at the time of diagnosis.
**Case Description/Methods:** A 77-year-old man presented to the emergency room with post-prandial, epigastric pain. He was hemodynamically stable on presentation. A CT scan of the abdomen demonstrated cholecystitis with a dilated common bile duct (CBD) up to 10mm. Due to concerns for biliary obstruction, a magnetic resonance cholangiopancreatography (MRCP) was done and showed a hypointense filling defect within the cystic duct and CBD. The patient underwent cholecystectomy with removal of a necrotic gallbladder. An intraoperative cholangiogram showed stones that could not be flushed, but CBD exploration was deferred due to friability of the surrounding tissue. The procedure was also complicated by hypotension, requiring 15 minutes of phenylephrine infusion and hypoxia which resolved with a non-rebreather mask. The next day, an esophagogastroduodenoscopy (EGD) and endoscopic ultrasound (EUS) was done to evaluate for any retained stones. The EGD was notable for Los Angeles (LA) Grade D esophagitis with black pigmentation of the esophagus starting 20cm from the incisors down to the gastroesophageal junction (**Figure** A and B). There were also small, nonbleeding erosions seen in the duodenal bulb and second portion of the duodenum. No choledocholithiasis was seen on EUS. Biopsies of the esophagus showed necrotic tissue with fibrinopurulent debris, confirming the diagnosis of AEN. The patient was started on an oral, high-dose proton pump inhibitor twice daily for management. Repeat EGD performed 11 weeks later demonstrated only residual LA Grade B esophagitis (**Figure** C and D).
**Discussion:** AEN has been associated with ischemic injuries, trauma, vasculopathy, sepsis, and general hemodynamic instability. Prognosis can be poor, but early recognition and aggressive supportive care can improve outcomes. This patient developed AEN following a transient period of intraoperative hypotension. Despite the degree of esophageal involvement, this patient lacked any symptoms of dyspepsia or reflux. Therefore, in patients with risk factors for AEN, performing an EGD prior to EUS should be considered even if there is no clinical evidence of hematemesis.




[2339] **Figure 1.** Endoscopic images of the middle and distal esophagus demonstrating acute esophageal necrosis (A, B). Follow-up EGD 11 weeks later showed resolution of black pigmentation following high-dose PPI treatment (C, D).

**S2340**

**Massive Upper Gastrointestinal Hemorrhage Following Radiofrequency Ablation for Barrett's Esophagus**

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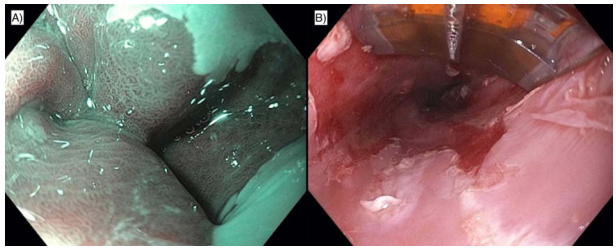
**Introduction:** Radiofrequency ablation (RFA) is an established endoscopic technique for the management of Barrett's esophagus (BE) with low grade dysplasia (LGD). Bleeding risk after RFA treatment has been reported to be around 1%. We present a case of massive upper GI bleeding following RFA treatment resulting in death as an adverse event.
**Case Description/Methods:** A 79-year-old male with history of BE with LGD underwent esophagogastroduodenoscopy (EGD) with RFA treatment. The patient had a history of coronary artery disease for which he was taking aspirin and prasugrel. His prasugrel was held 7 days prior to the procedure. During the procedure, the esophagus was carefully examined using white light and narrow band imaging. No esophageal nodules were found; however, mucosal changes secondary to established short-segment Barrett's were seen. The Barrx 90 ultra RFA catheter was used to ablate the Barrett's mucosa and the procedure was uneventful. Aspirin was resumed the following day, but prasugrel was held for 1 week post procedure. Ten days post procedure, the patient presented to an outside hospital with hematemesis and chest pain. An emergent EGD was performed that revealed massive bleeding in the esophagus with no obvious source. A mesenteric angiogram with embolization of left phrenic and gastric arteries was performed by interventional radiology. A subsequent EGD revealed multiple large, deep ulcerations in the distal esophagus. The patient stabilized and was downgraded from the ICU. However, 2 days later, the patient developed acute respiratory failure with multi-organ failure, thought to be due to aspiration. The patient was transitioned to comfort care and passed away.
**Discussion:** RFA is considered a safe and effective technique for the treatment of patients with BE with either low- or high-grade dysplasia as it is associated with an increased risk of developing esophageal adenocarcinoma. The most frequent adverse events following RFA treatment are esophageal stricture (8-10%), chest pain, bleeding (1%) and perforation (0.6%). Current guidelines for patients on dual antiplatelet therapy for secondary prevention who are undergoing elective procedures suggest temporary interruption of the P2Y12 inhibitor while continuing aspirin. These guidelines were followed in our patient. However, no clear guidance for when to resume newer anticoagulants exists. After extensive literature review, we did not find any case reports of massive upper GI bleed resulting in death following RFA treatment.




[2340] **Figure 1.** A) Barret's esophagus under narrow band imaging B) RFA treatment.

**S2341**

**Malignancy-Associated Pseudoachalasia: An Unusual Cause**

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**Introduction:** Achalasia is a rare motility disorder characterized by loss of inhibitory neurons of the myenteric plexus in the esophageal wall. Previous retrospective studies have suggested 4% of patients that present with achalasia-like symptoms and radiographic and esophageal manometric findings consistent achalasia will have causes of dysphagia besides primary achalasia, or pseudoachalasia. We describe a case of pseudoachalasia from a rare cause in a patient with dysphagia and weight loss.
**Case Description/Methods:** An 84-year-old female presented with dysphagia for solids more than liquids, regurgitation of food, and 50 lbs weight loss over 6 months. The patient underwent high-resolution esophageal manometry (HREM) that demonstrated a hypertensive lower-esophageal sphincter. HREM also demonstrated failed peristalsis, incomplete bolus clearance, and pan-esophageal pressurization. An upper GI series demonstrated minimal passage of barium into the stomach, with a tapering of the esophagus with a bird beak-like pattern. The patient was referred for POEM for achalasia. At the start of the procedure, the GE junction was noted to be very tight and would not allow passage of the gastroscope. To advance the gastroscope into the stomach, balloon dilation over a guide wire was performed using a through-the-scope balloon. Endoscopic ultrasound (EUS) was then used to further evaluate the tight GE junction. EUS demonstrated heterogeneous thickening of the esophageal wall extending to the GE junction and gastric cardia. Several enlarged lymph nodes were seen adjacent to the infiltrative process. At the GE junction there was a solid, hard hypoechoic lesion that resisted passage of the endoscope. Pathologic and immunohistochemical analysis of a core tissue biopsy demonstrated atypical mesothelial cells suggestive of mesothelioma.
**Discussion:** The epidemiology of pseudoachalasia remains uncertain given it is a rare mimicker of an already rare motility disorder. Malignancy can produce dysphagia through direct compression of the GE junction or due to submucosal invasion and disruption of the myenteric plexus. Especially in elderly patients, those with significant weight loss or a short duration of symptoms, a diagnosis of malignancy-related pseudoachalasia should be considered. As this case showed, all the other diagnostic testing in the workup of achalasia was consistent with achalasia and fooled us into missing an underlying cancer.

S2342

**Lymphocytic Esophagitis: A Rare Etiology for Persistent Heartburn**

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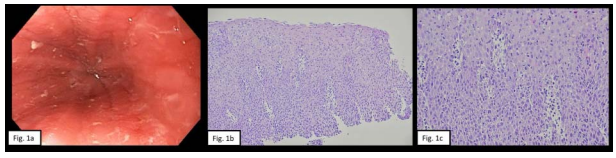
**Introduction:** Lymphocytic esophagitis (LE) is a relatively newly recognized and emerging condition. It has a higher predominance in females and is highly associated with inflammatory bowel disease and smoking. Dysphagia and heartburn are the 2 most common presentations. The esophagus usually appears normal by endoscopy; however, esophageal rings, webs, nodularities, furrows, and strictures have been described. Presented is a case with persistent heartburn and epigastric discomfort found to have LE with an unusual response to therapy.
**Case Description/Methods:** A 24-year-old female with a history of ulcerative colitis (UC) and tobacco use came for esophagogastroduodenoscopy (EGD) due to constant heartburn and epigastric abdominal discomfort that started 2 months ago. She has been prescribed Omeprazole twice daily with no adequate response. Her UC was well controlled on infliximab with normal inflammatory markers. The patient's vitals and physical exam were unremarkable. During EGD, the esophageal mucosa appeared hyperemic and congested with presence of linear furrows and white exudates suspicious of eosinophilic esophagitis (EoE) (**Figure** A). The stomach had linear erythema at the antrum, and the examined duodenum appeared normal. Biopsies were obtained from the mid-esophagus to evaluate for EoE and the stomach to rule out *Helicobacter pylori*. There was no evidence of EoE on biopsies; however, there were changes suggestive of LE (**Figure** B, C). Stomach biopsies were normal without evidence of *H. pylori*. Omeprazole was switched to Pantoprazole; however, her symptoms persisted after 8 weeks, and it was discontinued. She refused a trial of steroids, so Famotidine 20mg twice daily was started. On the subsequent visit, she stated that her symptoms had improved and did not have any more complaints.
**Discussion:** LE is not a fully understood condition yet. It is defined as peripapillary intraepithelial lymphocytosis ( >20 IEL/HPF) with spongiosis (intercellular edema) and few or no granulocytes on esophageal biopsy. A trial of a proton pump inhibitor (PPI) or topical steroids is recommended; however, there are no clear treatment and surveillance guidelines. Our patient had biopsy-proven LE and did not respond to 2 different PPIs but showed an excellent response to Histamine 2-receptor antagonist (Famotidine). This case highlights the importance of recognizing LE as a condition that can mimic EoE in symptoms and endoscopically and the need for more research to understand this entity better.




[2342] **Figure 1.** a: Endoscopic picture of mid-esophagus showing mucosal erythema, linear furrows, and white exudate. b: A low-power photomicrograph of hematoxylin and eosin stain showing squamous esophageal mucosa with dense infiltrates of intraepithelial lymphocytes (IEL). c: A medium-power photomicrograph of hematoxylin and eosin stain showing intercellular edema (spongiosis) with dense peripapillary lymphocytic infiltrates and absence of intraepithelial granulocytes (neutrophils and eosinophils).

S2343

**Long-Term Omeprazole Use Causing Critical Electrolyte Derangements**

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**Introduction:** Proton-pump inhibitors (PPI) are the mainstay therapy for acid-related gastrointestinal entities. Longstanding PPI use is associated with an increased risk of adverse effects; however, clinically significant side effects, such as metabolic derangements, are rare. Here we highlight a case of critical electrolyte derangements secondary to prolonged omeprazole use.
**Case Description/Methods:** A 61-year-old female with a history of hypertension and GERD presented with intermittent numbness for several months involving bilateral upper extremities. Exam showed no focal neurologic deficits with intact sensation and appropriate motor strength. CT head and cervical spine were unremarkable. Initial labs showed significant electrolyte derangements, including hypokalemia 3.3 mmol/L, hypocalcemia 5.7 mg/dL (0.64 mmol/L ionized), and hypomagnesemia 0.5 mg/dL. TSH, PTH, and 25-hydroxyvitamin D levels were intact (Table). EKG showed normal sinus rhythm with QTc prolongation at 542 ms. Despite aggressive intravenous repletion, her electrolytes remained suboptimal. After evaluation by endocrinology, her symptoms were thought to be secondary to PPI-induced hypomagnesemia. The patient had been taking omeprazole for nearly 20 years after evaluation by ENT for vocal cord hoarseness thought to be secondary to GERD. Patient was transitioned from PPI to an H2 blocker. She underwent EGD which showed mild gastritis with biopsies negative for Helicobacter pylori. Fortunately, her symptoms resolved after electrolyte stabilization with potassium 3.9 mmol/L, calcium 8.8 mg/dL, and magnesium 1.8 mg/dL. She was discharged on famotidine with instructions to follow up with gastroenterology.
**Discussion:** Hypomagnesemia has become an increasingly well-recognized, albeit rare side effect of PPI use. The mechanism is thought to involve interference of TRPM6 and TRPM7 located on apical membranes of enterocytes leading to intestinal malabsorption with resulting hypomagnesemia as well as hypocalcemia and hypokalemia. Interestingly, manifestations of hypocalcemia and hypokalemia can be concealed by calcium and potassium-sparing agents, which was likely the case in our patient who was taking candesartan-hydrochlorothiazide combination therapy for hypertension. Our case demonstrates the importance of limiting chronic PPI therapy given the potential for serious consequences. Increased efforts should be aimed at deprescribing PPI therapy.

**Table 1. Electrolyte derangements including hypokalemia, hypocalcemia, and hypomagnesemia, which remained suboptimal despite aggressive repletion. Electrolytes finally stabilized after switching from PPI to H2 blocker**

| | Day 0 | Day 3 | Day 5 | Day 6 |
|---|---|---|---|---|
| $K^+$ (mmol/L) | 3.3 | 3.2 | 3.8 | 3.9 |
| $Ca^{+2}$ (mg/dL) | 5.7 | 6.6 | 7.8 | 9.5 |
| $Mg^{+2}$ (mg/dL) | 0.5 | 1.6 | 1.7 | 1.6 |

S2344

**Lymphocytic Esophagitis: A Rare and Rising Cause of Dysphagia**

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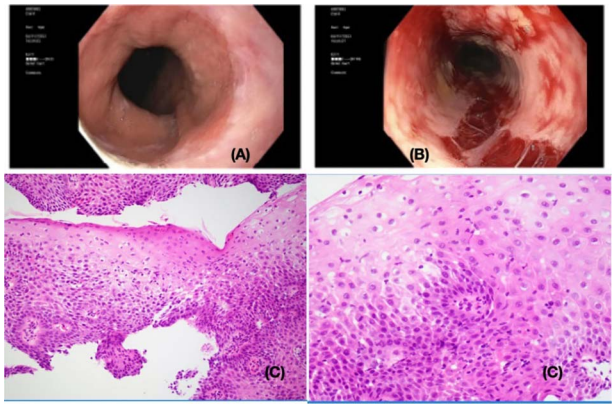
**Introduction:** Lymphocytic esophagitis (LyE) is a rare esophageal condition with an unknown pathogenesis, prognosis, and limited treatment options. It affects around 0.1% of the population but incidence has been rising. We are presenting a case of lymphocytic esophagitis as a cause of chronic dysphagia, odynophagia and dyspepsia.
**Case Description/Methods:** An 83-year-old male with a PMH of CAD, HTN, Emphysema, GERD, dysphagia and alcohol use disorder was admitted for worsening dysphagia and generalized weakness for 2 months. The patient reported to have a previous history of dysphagia and underwent esophageal dilation in the past. He described difficulty swallowing that was painful and associated with a burning sensation that recently had worsened after he had stopped taking his PPI a few months prior because of their side effects. In addition, he reported weight loss of 20 lbs over the past few weeks. He underwent an endoscopic evaluation that showed no endoscopic abnormality in the esophagus (**Figure** A). A Maloney dilator was used to dilate the entire esophagus with mild resistance at 56 Fr. Endoscopic re-insertion showed multiple moderate mucosal disruptions in the mid and distal esophagus following dilation (**Figure** B). Post dilation biopsies were obtained to rule out EoE. Histopathology of those biopsies showed squamous mucosa with increased intraepithelial and patchy peripapillary lymphocytes, along with reactive epithelial changes which are consistent with lymphocytic esophagitis (**Figure** C). After the procedure, the patient was started on PPI oral twice daily with improvement in his symptoms.
**Discussion:** LyE is a rare clinical condition, identified first time in 2006 and prevalent in around 0.1% of the population. It is still unclear if it is a distinct entity or sequelae of nonspecific inflammatory response to chronic injury from conditions such as GERD, celiac disease, IBD, motility disorders, or autoimmune disorders. The clinical presentation includes dysphagia, dyspepsia, and chest pain. Endoscopic appearance

of LyE may reveal rings, furrows, strictures, edema, or esophagitis, similar in appearance to patients with EoE. Some patients, like ours, may have a normal-appearing esophageal mucosa on endoscopy. The diagnosis of LyE relies on mucosal biopsy and histology. Hallmark findings include spongiosis or intercellular edema, and peripapillary lymphocytosis without significant presence of neutrophils or eosinophils. Current treatment options include PPI, swallowed topical corticosteroids, and endoscopic dilation.




[2344] **Figure 1.** (A, Pre dilatation esophagus) (B, Post dilatation mucosal disruption of mid & distal esophagus) (C, squamous mucosa with increased intraepithelial and patchy peripapillary lymphocytes).

**S2345**

**Metastatic, Poorly-Differentiated Neuroendocrine Tumor of the Esophagus: Case Report and Literature Review of a "Rare Rarity"!**

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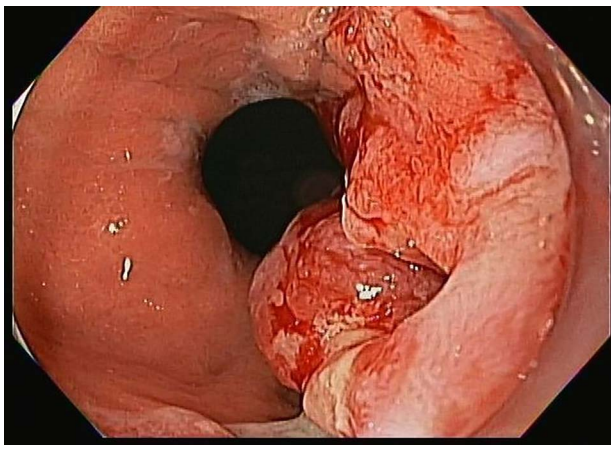
**Introduction:** Neuroendocrine tumors (NET) most commonly develop in the lungs, appendix, bowel and pancreas. Esophageal NET is extremely rare, comprising < 2% of all NETs. The most common symptom is dysphagia although patients may be asymptomatic. We present a case of metastatic esophageal NET and a literature review.
**Case Description/Methods:** A 61-year-old female with 5-month history of progressively worsening chest tightness, weight loss and diarrhea underwent abdominal CT scan which revealed enlarged gastrohepatic lymph nodes (LN). EGD showed a 3cm ulcerated lesion at the gastroesophageal junction (GEJ). Biopsies suggested high-grade NET in the background of high-grade dysplasia. PET/CT confirmed a 3.0 x 2.3cm ill-defined, hypermetabolic, soft tissue mass at the GEJ (SUV max 9.0) and several hypermetabolic, enlarged gastrohepatic LNs, largest measuring 2.8 x 2.0cm (SUV max 8). The patient was referred to our center for further management. Repeat EGD revealed a 3cm distal esophageal lesion with central ulceration extending across the GEJ into the high cardia (**Figure**). On endoscopic ultrasound (EUS), a hypoechoic mass is seen with involvement of the muscularis propria (T2). Peri-gastric lymphadenopathy was present. Transgastric fine needle aspiration (FNA) of the largest LN confirmed metastatic neuroendocrine tumor (T3N1). Repeat endoluminal biopsies from the esophageal lesion confirmed poorly-differentiated NET, CD56 and synaptophysin positive, Ki-67 > 90%. Multidisciplinary tumor board recommended chemotherapy with carboplatin and etoposide. No evidence of disease progression noted after the first 3 cycles; patient remains on treatment.
**Discussion:** Metastatic poorly-differentiated high-grade NET of the esophagus is very rare. First-line therapy is chemotherapy. Average prognosis with treatment is 12-18 months. Immunotherapy may provide additional survival benefit however data is extremely limited so this is typically reserved for second-line therapy (if progression on chemotherapy). Given the rarity and aggressiveness of these lesions, accurate pathological diagnosis and multidisciplinary discussion is critical for optimal management of these patients.



[2345] **Figure 1.** Large distal esophageal lesion with central ulceration.

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**S2346**

**Metastatic Implantation of Esophageal Adenocarcinoma to PEG Tube Site: A Case Report**

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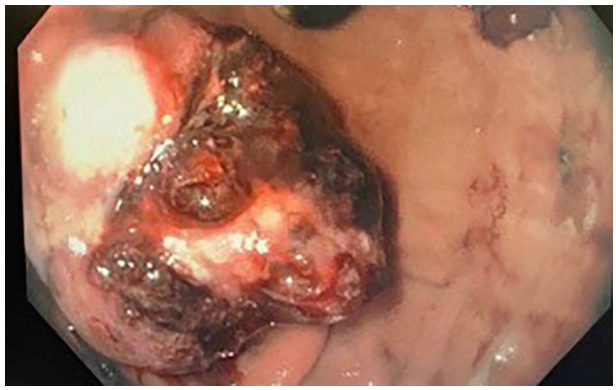
**Introduction:** Patients with esophageal cancer often need a Percutaneous Endoscopic Gastrostomy (PEG) tube to provide nutrition. However, metastasis of the original tumor to the gastrostomy site may occur as a rare, but noteworthy complication.
**Case Description/Methods:** A 64-year-old male had a PEG tube placed after he was diagnosed with Stage 2 esophageal adenocarcinoma with extension into the proximal stomach in Sep 2018. Following this, he received chemo and radiation therapy and did well. A follow up EGD demonstrated no evidence of malignancy, but a non-malignant duodenal narrowing / stenosis was noted. Subsequently, the patient suffered persistent leakage around his PEG tube; due to this and duodenal stenosis, his gastrostomy tube (G-tube) was removed and a jejunostomy tube (J-tube) was placed with surgical closure of the gastrostomy site (**Figure**). A few months later, when the patient was admitted with melena and severe anemia, an upper endoscopy performed revealed a hard, 3-4 cm malignant appearing mass with recent evidence of bleeding noted on the greater curvature of the stomach at the previous PEG tube site. Biopsies from this area showed invasive moderately differentiated adenocarcinoma with histological features similar to the previous adenocarcinoma. Though re-initiation of chemotherapy and radiation did result in improvement of the patient's stomach neoplasm, his wound healing complications at the J-tube site persisted, resulting in delay of his treatment. In October 2021, there was recurrence of his stomach cancer. Due to persistent difficulties with wound healing and recurrent illnesses, the patient elected to forgo further treatment in favor of comfort care. He subsequently passed away peacefully.
**Discussion:** Our experience with this case and review of the literature indicate that, in patients with esophageal cancer, usage of the "pull-string" technique as well as length of time of PEG tube placement are associated with a significantly higher risk of metastatic implantation. To avoid this rare complication, potential alternative techniques such as the "push" technique for PEG tube placement or radiologically / surgically placed G-tubes or J-tubes should be considered in patients with oropharyngeal or esophageal cancer for nutrition.



[2346] **Figure 1.** Endoscopic image revealing a malignant-appearing mass on the greater curvature of the stomach at initial PEG tube site.

**S2347**

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**Mixed Adenoneuroendocrine Carcinoma at the Gastroesophageal Junction: A Case Report**

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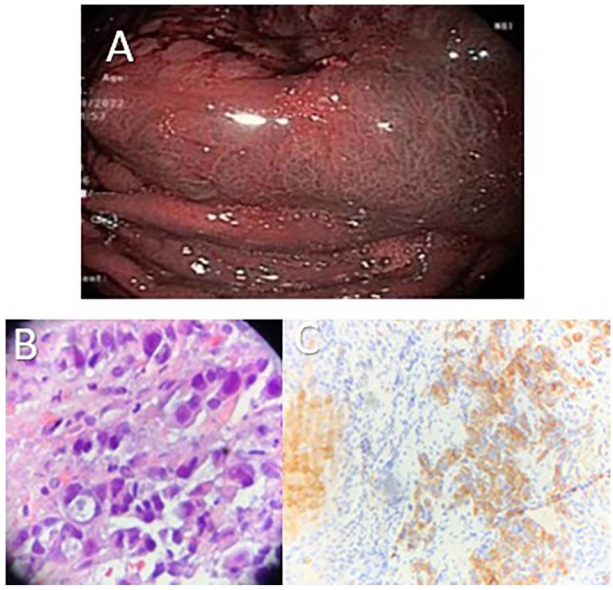
**Introduction:** Mixed adenoneuroendocrine carcinomas (MANECs) are a type of mixed neuroendocrine non-neuroendocrine neoplasm (MiNEN), which is a very rare and highly aggressive group of neoplasms found in the gastro-entero-pancreatic (GEP) tract. MANECs comprise both an adenocarcinoma and neuroendocrine carcinoma component. Currently the literature regarding these neoplasms is limited to only retrospective studies and case reports. There have been no prospective randomized trials to support any particular diagnostic algorithm or treatment plan.
**Case Description/Methods:** A 56-year-old male smoker presented to the emergency room with 2 weeks of intermittent dysphagia to solids and liquids, epigastric abdominal pain, and melena associated with a 23-pound unintentional weight loss in one month. Computed tomography (CT) revealed a narrowed and thickened distal esophagus. Subsequent esophagoduodenoscopy (EGD) revealed a malignant appearing stricture at the gastroesophageal junction (GEJ) with a large, polypoid mass bulging from the gastric cardia. Histopathological analysis revealed poorly differentiated adenocarcinoma admixed with high-grade (Grade 3) neuroendocrine differentiation. Immunohistochemistry was significant for AE1/AE3, CK7, CDX2, CK20, and synaptophysin positivity. The patient was referred to an external facility for neoadjuvant chemoradiation and local surgical resection via Ivor-Lewis esophagectomy (**Figure**).
**Discussion:** MANECs are rare tumors of the alimentary tract, composed of neuroendocrine and non-neuroendocrine carcinoma components. These tumors are highly aggressive and often fatal. We found 9 other cases of MANEC discovered at the GEJ in the English medical literature. Given this rare occurrence, we aim to add to the existing literature with another case to support diagnostic and treatment modalities for these highly aggressive mixed neoplasms in the future. This case is currently ongoing and we are following closely as the patient receives further treatment.




[2347] **Figure 1.** 1: A) Endoscopic image demonstrating polypoid mass protruding from gastroesophageal junction into gastric cardia under narrow band imaging technology. B) ) H&E stain of the specimen demonstrating poorly differentiated adenocarcinoma with signet ring features, x400. C) Immunohistochemical staining demonstrating positivity for CK7 in the neuroendocrine carcinoma component (x100).

**S2348**

**Novel Approach to Proximal Esophageal Stricture**

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**Introduction:** We present you a case of Stricture-induced severe dysphagia that was discovered to be caused by ulceration secondary to very proximal Zenker's diverticulum (ZD). This case report also describes a novel approach to esophageal endoscopy through a retrograde approach.
**Case Description/Methods:** Patient is an 80 y/o African American female with a past medical history of Gastroesophageal Reflux disease, Congestive Heart Failure, Chronic Kidney disease and Dementia who presented with Heart failure exacerbation. Patient reported weight loss and dysphagia to solid food and liquids for about 2 months. Patient then underwent a modified barium swallow study (MBSS), which showed concerns for esophageal stricture and evidence that the contrast material was not passing through the esophagus. Patient then underwent Esophagogastroduodenoscopy (EGD), which showed complete esophageal obstruction approximately 25 cm from the incisors and impacted food. The food was removed and a repeat EGD was done the next day showing a circumferential ulcer and no lumen could be identified. Biopsies were taken, which was negative for dysplasia or malignancy. After discussion with General Surgery, it was decided to proceed with Open Gastrostomy tube to allow enteral nutrition as well as access to dilate the esophagus in the retrograde fashion in the future. A repeat EGD after 4 weeks showed a healing ulcer and a blind pouch without an identifiable lumen. The ultrathin scope was advanced through the gastrostomy tract into the stomach and carefully advanced in a retrograde fashion through the lower esophageal sphincter throughout the esophagus. Narrowing was seen at the proximal end of the esophagus. The scope was advanced through this and with minimal resistance able to be advanced past the level of the stricture about 15 cm from the Incisors. Esophageal stent was deployed over the wire in an antegrade fashion with fluoroscopic guidance. This time, an area of concern was identified as a possible ZD proximal to the prior ulceration site which was later confirmed on an esophagram at the level of thoracic esophagus on the right side.
**Discussion:** This case was challenging as the diagnosis of ZD was missed despite various testing. Patient had a long history of dysphagia and the impacted food led to ulceration and esophageal strictures. The diagnosis was established after a unique approach to the stricture. This case highlights that sometimes a careful examination is required as an approach to diagnosis of dysphagia in elderly.

**S2349**

**Not Just Another Food Bolus: Large Cell Neuroendocrine Carcinoma of the Esophagus Presenting as Acute Esophageal Food Impaction**

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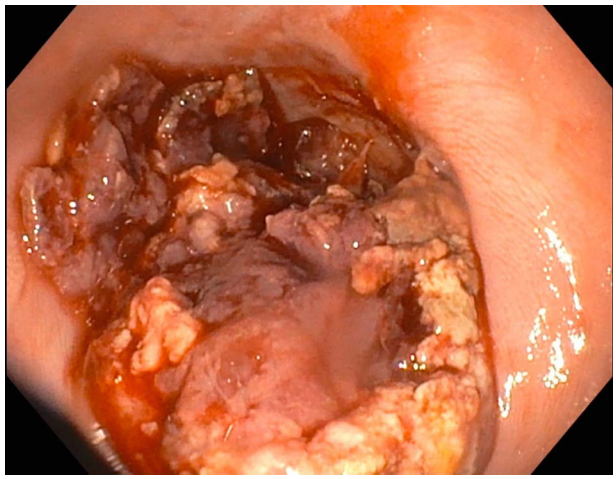
**Introduction:** Food impactions arise due to structural or functional abnormalities along the GI tract most commonly at the esophagus. The differential diagnosis is broad but importantly includes malignancy. Here we highlight a rare case of food impaction secondary to high-grade neuroendocrine carcinoma.
**Case Description/Methods:** A 71-year-old male with a history of asthma presented with trouble swallowing and epigastric pain 5 hours after eating a chicken sandwich. He had been experiencing dysphagia and odynophagia for the past 2 months, but symptoms were especially severe on day of presentation to the point that he was unable to eat or drink anything afterwards given persistent regurgitation. Urgent EGD showed a food impaction of the distal esophagus, which was successfully removed through suction cap revealing a circumferential fungating and necrotic appearing mass at 28 cm from the incisors (**Figure**). Biopsies were obtained, but the mass could not be traversed. Subsequent CT CAP showed a 6.7 cm distal esophageal mass correlating with endoscopic findings as well as nonregional lymphadenopathy concerning for metastatic disease. Pathology unfortunately revealed high-grade neuroendocrine carcinoma WHO grade 3 with small and large cell features. Patient was deemed not a surgical candidate given metastatic disease and was started on palliative chemotherapy.
**Discussion:** Neuroendocrine carcinoma is a rare type of esophageal malignancy with an incidence of 0.4-2% of cases. Prognosis is usually poor given its aggressive nature and advanced stage at initial diagnosis. Treatment depends on the extent of disease. For those with limited disease, esophagectomy with lymph node dissection +/- adjuvant chemotherapy is warranted. On the other hand, extended disease relies upon palliative chemotherapy +/- adjuvant radiotherapy. Clinicians must maintain neuroendocrine carcinoma as part of their differential for food impaction given the life-altering implications that such a diagnosis holds.



[2349] **Figure 1.** EGD showing fungating and necrotic appearing mass at distal esophagus.

**S2350**

**Mind the Steroids: Herpes Esophagitis in an Immunocompetent Patient After Steroid Therapy**

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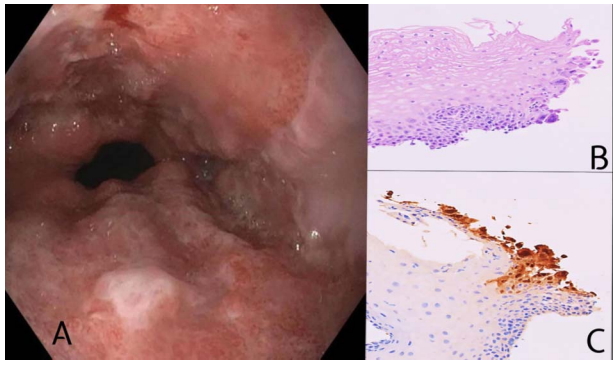
*[1]East Carolina University Brody School of Medicine, Greenville, NC; [2]ECU Health Medical Center, Greenville, NC.*

**Introduction:** Infective esophagitis (IE) is rare, but well known to complicate the immunosuppressed state; particularly the HIV infected, transplant recipient, cancer patient and those treated with chronic corticosteroids or immunomodulators. Common infections include candida, cytomegalovirus, and herpes simplex virus (HSV). Steroids have been used for symptomatic relief of an acute sore throat. Short courses are considered benign with low risk of infectious complications. We report an immunocompetent patient found to have herpes esophagitis after a short course of corticosteroids.
**Case Description/Methods:** A 41-year-old African American male with history of hypertension presented with worsening sore throat and odynophagia restricting oral intake. Prior to presentation, he had a 10-day history of sore throat. He was diagnosed with streptococcal pharyngitis and was on antibiotics and steroids 7 days ago. Given persistent symptoms he underwent laryngoscopy which demonstrated bilateral tonsillar exudates and normal pharyngeal mucosa, prompting admission and initiation of IV antibiotics and dexamethasone. He had modest improvement of pain, however 8 days later expressed worsening burning retrosternal chest pain and progressed oral intolerance. He underwent upper endoscopy revealing multiple circumferential erosions and Los Angeles grade D esophagitis of the lower third of the esophagus (**Figure** A). Biopsies demonstrated viral intranuclear inclusions that stained positive for HSV1/HSV2 (**Figure B, C**). His symptoms abated while on IV acyclovir allowing transition to oral valacyclovir.
**Discussion:** Corticosteroids are well known for their anti-inflammatory effect but immunosuppression with short courses may be underestimated. Infectious sequalae such as herpetic esophagitis may complicate steroid use even when therapy duration is short. This patient was found to have progressive odynophagia while on corticosteroids administered for analgesia and had significant improvement after antiviral therapy. It is easy to overlook IE as a cause of odynophagia in the immunocompetent, however, with history of steroid use, a diagnosis of IE, especially HSV esophagitis, should be considered in the differential. Therefore, one should have a low threshold for early endoscopy when evaluating these patients.




[2350] **Figure 1.** A: Endoscopic photograph displaying multiple circumferential erosions and Los Angeles grade D esophagitis of the lower third of the esophagus. B: (x200): Photomicrograph of hematoxylin and eosin-stained section of the esophageal biopsy, showing multiple multinucleated epithelial cells. Cowdry type A is observed. C: (x200): IHC study using antibodies against HSV-1. Numerous intranuclear viral inclusions are positive with HSV-1 within the infected cells.

**S2351**

**Not Your Average Cancer: Skip Lesions Associated With Signet Ring Cell Esophageal Adenocarcinoma, a Prognostic Indicator of Poor Outcomes**

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**Introduction:** Signet Ring Cell Adenocarcinoma (SRCC) is a rare histological subtype of esophageal adenocarcinoma (EAC). Incidence of SRCC in the proximal esophageal segment is sparsely reported in the literature. Here, we describe a case of 2 independent skip lesions of signet ring cell histology, located in proximal esophageal segment of adenocarcinoma origin, presenting as worsening dysphagia.
**Case Description/Methods:** A 65-year-old White male presented with 5-months of worsening dysphagia. His dysphagia had progressively gotten worse from liquids to solids. Past medical history was significant for gastroesophageal reflux disease, polysubstance abuse. Initial workup for dysphagia included computed tomography (CT) and esophagogastroduodenoscopy (EGD). CT demonstrated nodular wall thickening of the distal esophagus extending into the proximal stomach, innumerable hypodense lesions of the liver, and multiple enlarged lymph nodes. EGD showcased an esophageal mass in the proximal segment approximately 25 to 28cm from the oropharynx and a distal skip lesion noted to be ulcerated and fungating which was extending to the gastroesophageal junction (GEJ) Notably, through endoscopy, no gross abnormalities was detected between the 2 lesions. Biopsy of both lesions revealed well-differentiated invasive adenocarcinoma with signet ring cells. The patient deferred treatment and palliative care measures were started. The final diagnosis was metastatic signet ring cell carcinoma (SRCC) in the proximal esophageal segment. The patient passed away 7 days after diagnosis

**Discussion:** Signet ring cell adenocarcinoma is a rare histological subtype of adenocarcinoma that confers a poor prognosis. Typically, signet ring cells originate in the colon and bladder. SRCC makes up less than 3% of all cases of esophageal cancers and is commonly found at distant disease stages with poor differentiation. Uniquely, this case offers an atypical presentation of 2 separate skip lesions from a rare manifestation of adenocarcinoma in the proximal segment of the esophagus. To the best of our knowledge, the current literature demonstrates limited cases of signet ring cells in the esophagus presenting as 2 independent lesion.

**S2352**

**No Soup for You: The Case of an Intramucosal Hematoma Found During Endoscopy for an Esophageal Food Impaction**

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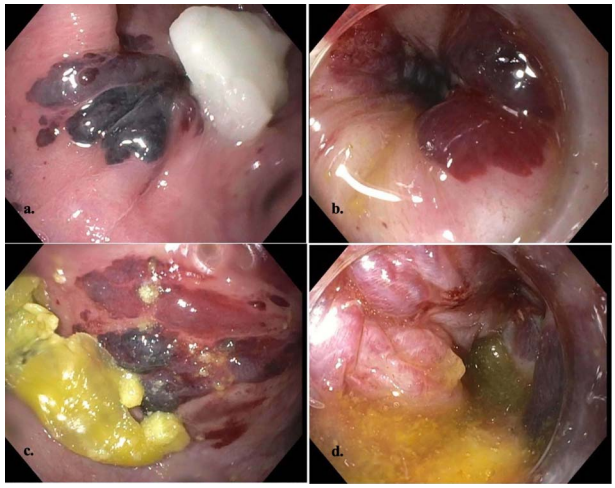
*Brody School of Medicine at East Carolina University, Greenville, NC.*

**Introduction:** Esophageal food impaction (EFI) is third most common gastrointestinal emergency, which is commonly associated with obstructive lesions such as peptic strictures and Schatzki rings, eosinophilic esophagitis and rarely motility disorders. Endoscopic therapy is the mainstay option for the management of EFI if medical therapy fails. The most commonly impacted foods include beef, chicken, pork, and undercooked vegetables with liquids rarely causing an impaction. Complications from a prolonged EFI can result in mucosal disruption to an esophageal perforation. We present an unusual case of an esophageal intramucosal hematoma found in a during endoscopic retrieval of an esophageal impaction.
**Case Description/Methods:** An 80-year-old male presented with a 2-hour history of foreign body sensation in his throat that began while eating hamburger meat and soup with potatoes. He began coughing up a large amount of mucus at symptom onset and was unable to finish his meal. He has been unable to swallow solids or liquids. History was significant for esophageal peptic strictures in the past requiring dilations. After 2 doses of glucagon failed to help pass the bolus, the patient underwent endoscopic removal of the esophageal food impaction. During the procedure, food was found in the middle and lower thirds of the esophagus and was subsequently removed with suction cap and rat toothed forceps. The esophagus was tortious and was noted to have decreased and abnormal movement throughout with a moderate stenosis found at the gastroesophageal junction. The middle and lower third of the esophagus had localized severe erythema, suspicious for the presence of intramucosal hemorrhage (**Figure**). The patient tolerated a diet after endoscopy and discharged home.
**Discussion:** Esophageal food impactions are uncommon and are commonly associated with stricture and rings. Complications from a prolonged food impaction include mucosal disruption however findings of an intramucosal hematoma are rare. Intramucosal hematoma present with chest pain, dysphagia, and hematemesis. This patient presented with dysphagia however had no other pain or episodes of hematemesis. In this patient it is unclear whether his presentation was due to an acute EFI causing an intramucosal hematoma or whether the ingestion of soup caused an intramucosal hematoma resulting in an EFI. Nevertheless, one should take caution during endoscopic evaluation for patients who presented with an EFI.




[2352] **Figure 1.** Localized severe erythema suspicious for intramucosal hemorrhage. (1a., 1b., 1c.), Lower third of the esophagus. (1d.)- Gastroesophageal junction.

**S2353**

**Esophageal Hyperkeratosis Complicated by Recurrent Dysphagia**

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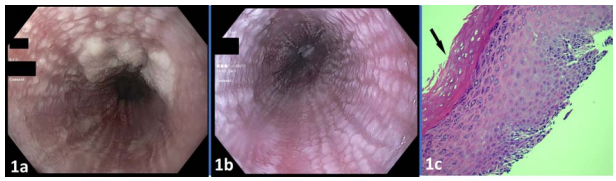
*St. Elizabeth's Medical Center, Tufts University School of Medicine, Boston, MA.*

**Introduction:** Esophageal hyperkeratosis (EH) is a rare condition characterized by keratinization of the esophageal epithelium. The pathophysiology is poorly understood, and risk factors remain unclear. We present a case of severe long segment EH leading to severe esophageal stenosis.
**Case Description/Methods:** A 70-year-old male with a long history of gastroesophageal reflux disorder (GERD) presented to the ED with acute food impaction. Prior to his presentation, he reported worsening dysphagia and ongoing heartburn for several months. He had been lost to follow-up and was not on any antacids at the time of presentation. Emergent upper endoscopy (EGD) was performed to remove the food impaction. Additional findings on EGD showed esophagitis, esophageal stenosis of 13cm in length (7mm in diameter), and white plaques. Esophageal biopsies were negative for eosinophils but confirmed candida and were suggestive of esophageal hyperkeratosis. Dilation was deferred until the esophagitis was treated with PPI and itraconazole. Repeat EGD one month later showed ongoing stenosis, healed esophagitis, as well as significantly thickened esophageal mucosa extending 13 cm proximally from the GE junction (**Figure** A, B). Balloon dilation of the stenotic lesion was performed. Repeat biopsies confirmed esophageal hyperkeratotic squamous epithelium with parakeratosis and no dysplasia (**Figure** C). Due to the severity of his stenosis, he subsequently underwent multiple EGDs with balloon dilations over the next several months to obtain a maximum esophageal lumen diameter of 19mm. Dilation of his esophagus was particularly difficult due to the length of hyperkeratosis, degree of stenosis, and loss of elasticity of his esophageal mucosa. Tearing of the tissue and bleeding were common even with small interval increases during each dilation session. In addition, healing of the tissue between sessions often led to a restricturing of his esophagus. He is currently symptom-free on pantoprazole 40mg twice a day, a soft diet, and undergoing surveillance endoscopies every 6 months with balloon dilations.
**Discussion:** Esophageal hyperkeratosis is a rare disorder with thickening of the stratum corneum and occurs in 2% of esophageal biopsies. While the prevalence and pathophysiology are unclear, it is thought to be associated with GERD, vitamin A deficiency, zinc deficiency, and a low fiber/low residue diet. There is also some evidence to suggest it may be associated with an increased risk of squamous cell carcinoma.




[2353] **Figure 1.** 1: a and b) EGD of middle and lower esophagus showing irregular mucosa with furrowing and white exudate, pre-dilation. 1c) Magnification 400x. Histopathology demonstrates esophageal squamous mucosa with dyskeratosis and extensive hyperkeratosis (black arrow).

**S2354**

**Sebaceous Glands in the Esophagus**

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**Introduction:** Sebaceous glands are small exocrine glands which secrete an oily substance called sebum. Derived from the ectoderm, they are generally found distributed over the body's skin near hair follicles and are found most commonly on the scalp and face. Occasionally, sebaceous glands can also be found in other areas, including the eyes, palms, soles, genitalia, and parotid glands. However, the presence of sebaceous glands in endodermal organs is extremely rare. Here, we report a case of ectopic sebaceous glands found in the esophagus.
**Case Description/Methods:** A 45-year-old female presented to the gastroenterology clinic for chronic reflux and cough. She reported having these symptoms for several decades and received moderate symptom control with a proton pump inhibitor. Her past medical history was significant for asthma, and she denied having any prior surgeries. Her medications included a proton pump inhibitor and metoclopramide. She denies any tobacco, alcohol, marijuana, or other recreational drug use, and family history was unknown. Her physical exam was unremarkable, and basic labs including a complete blood count and comprehensive metabolic panel were normal. To further evaluate her symptoms, she underwent an esophagogastroduodenoscopy, which revealed numerous tiny non-scrapable off-white nodules in the mid- and distal esophagus. Cytology was negative for fungal organisms; however, biopsies revealed squamous mucosa with ectopic sebaceous glands.
**Discussion:** Ectopic sebaceous glands of the esophagus are an extremely rare finding. Found incidentally on endoscopy, it can look similar to candidiasis, xanthomas, or metastatic carcinoma, and thus biopsies should be taken to make a diagnosis. Although some patients have been noted to have symptoms of gastroesophageal reflux disease, ectopic sebaceous glands are thought to not have any clinical symptoms. Furthermore, the finding is benign and there is no known risk of malignant transformation.

**S2355**

**Although Rare, a Hardware Fracture and Migration of Anterior Cervical Spine Plating Can Present With Upper Gastrointestinal Bleeding and Associated With High Mortality**

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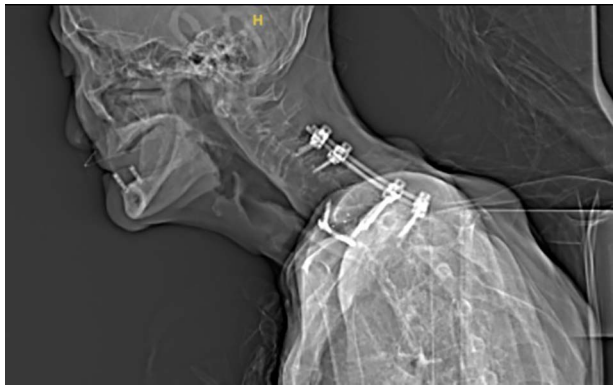
**Introduction:** Anterior cervical spine plating (ACSP) is a common surgical intervention indicated for many cervical osteopathologies. Esophageal perforation is a rare complication of ACSP that can occur shortly following surgery, due to over prominent screws or friction between the esophagus and the plate. Screw fracture and migration following the surgery is very rare but is a potentially life-threatening complication that can result in perforation of the adjacent organs like the esophagus and mediastinum, and increase the risk of infections in this area. (1-4)
**Case Description/Methods:** An 84-year-old man with cervical disc disease (C6-C7) status post ACSP (Anterior cervical spine plating ) with resultant need for a PEG tube placement secondary to newly developed severe oropharyngeal dysphagia after aforementioned spinal surgery with persistent aspiration, who presented with dark color liquid output coming out from the PEG tube.Esophagogastroduodenoscopy (EGD) showed a foreign metal hardware (looks like cervical plate) seen embedded in the wall of the hypopharynx and eroding out, but no ulceration or blood was seen around the PEG insertion site. CT cervical spine showed a fracture of the right C6 anterior fusion screw with evidence of anterior displacement of the fusion plate with 4 to 5 mm gap between the plate and the anterior margin of C6. Disc spacer material appears to extend beyond the anterior margin of the C6-7 disc space ( **Figure**).
**Discussion:** Comprehensive surgical history is important for any patient presenting with evidence of upper gastrointestinal bleeding. Patients with a history of Anterior cervical spine plating (ACSP) need urgent cervical spine imaging to assess the status of the hardware, and if there are any radiologic features of infection. Cervical spine imaging will help to triage the urgency and type of intervention, whether conservative or urgent surgical intervention.




[2355] **Figure 1.** CT scan on the cervical spine showing the hardware and screw migration to the esophagus.

**S2356**

**Black Esophagus: A Syndrome of Acute Esophageal Necrosis Associated With Diabetic Ketoacidosis**

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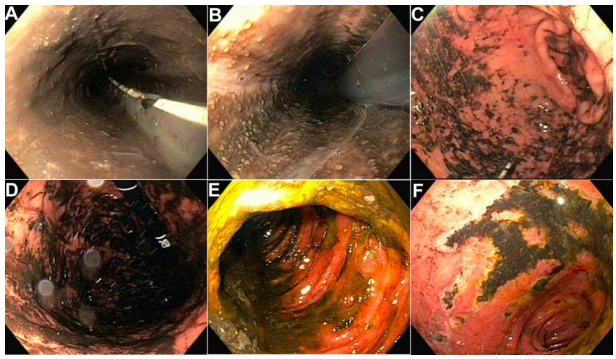
**Introduction:** Acute esophageal necrosis (AEN) also known as 'black esophagus' and Gurvitis' syndrome is a rare entity with a high mortality rate that can present with life-threatening upper gastrointestinal bleeding (UGIB). We aim to illustrate this rare presentation of diabetic ketoacidosis which has a high mortality rate and life-threatening complications that must be managed promptly to improve survival.
**Case Description/Methods:** A 25-year-old man with insulin-dependent type I DM and amphetamine use disorder was brought to the ER after several days of nausea, emesis, and non-adherence to insulin therapy causing obtundation. Nasogastric aspiration revealed coffee-ground fluid. Laboratory work-up was significant for hyperglycemia, hyperkalemia, hyperglycemia-induced hyponatremia, and acute kidney injury. Urine analysis and drug screen were positive for ketones (20 mg/dL) and amphetamines respectively. An abdominal CT scan showed gastrointestinal mucosal hyperenhancement and ascites. After

resuscitation with IV fluids, insulin drip, proton pump inhibitor (PPI), and vasopressors, an esophagogastroduodenoscopy (EGD) was performed which revealed diffuse black discoloration, mucosal ulcers starting from the upper esophagus extending to the gastro-esophageal junction, scattered areas of necrosis in the stomach, as well as large black demarcated ulcers in the bulb and D2 segment of the duodenum (**Figure**). With continued supportive care, he was discharged after 12 days of hospital care without any further complications and a plan to repeat EGD in 8 weeks.

**Discussion:** AEN should be considered in critically ill, diabetic patients who present with DKA and UGIB as DKA can trigger ischemic injury to the esophageal mucosa due to hemodynamic instability and acid reflux from the stomach. Rapid initial resuscitation with IV fluids, gastric acid suppression, and treatment of the underlying diabetic ketoacidosis with insulin is the mainstay of management along with continued supportive care. A high index of clinical suspicion is required for the early identification and management of AEN. Surgical intervention is required for patients with AEN complications such as esophageal perforation, and mediastinal infections.




[2356] **Figure 1.** Black necrotic mucosa of the esophagus (A, B). Scattered areas of ischemic necrosis in the gastric antrum (C) and fundus (D). Ulcerated duodenal mucosa (E, F).

**S2357**

***Candida*-Necrotizing Esophagitis as a Cause of Upper GI Bleed in a Patient With Poorly Controlled Insulin-Dependent Diabetes Mellitus**

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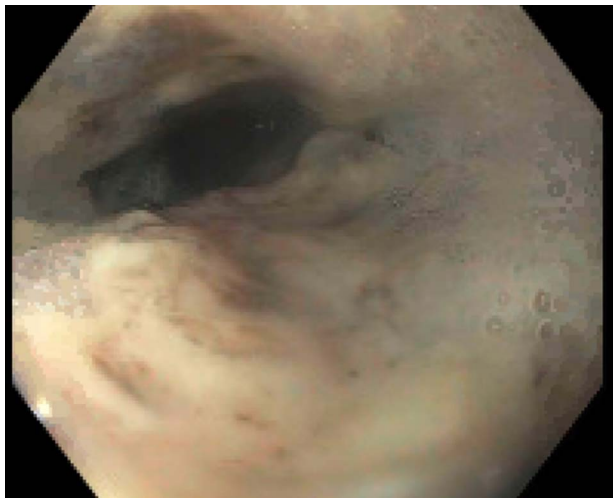
**Introduction:** Necrotizing esophagitis, also known as Black Esophagus, is a rare cause of upper gastrointestinal bleeding (GI). Patients can present with hematochezia or melena. The diagnosis is made on endoscopic evaluation with good prognosis if caught early.

**Case Description/Methods:** A 27-year-old-woman with poorly controlled insulin dependent type I diabetes mellitus (hemoglobin A1c 15) requiring multiple admissions for diabetic ketoacidosis (DKA) presented to the emergency department with nausea and vomiting. She was found to have a hemoglobin of 9.2 g/dL down from 11.2 g.dL. CT scan showed diffuse esophageal wall thickening. She left the emergency department AMA without further treatment but re-presented 4 days later with persistent symptoms. She reported chronic, repeated self-induced emesis to alleviate symptoms of early satiety. Initial emesis was bilious but subsequently changed to dark red after repeat bouts of emesis. Repeat labs showed significant metabolic acidosis with pH 7.04, anion gap >20, hyperglycemia >600, and hemoglobin now of 4.5 g/dL. She was admitted to the medical intensive care unit for management of DKA and bloody emesis. She received 4 units packed red blood cells with appropriate hemoglobin response to 8.0 g/dL. The gastroenterology service was consulted and performed an esophagogastroduodenoscopy. It revealed severe, diffuse ulceration throughout the entire esophagus with stigmata of recent bleeding (**Figure**). Biopsies showed numerous fungal forms consistent with candidiasis as well as features of necrotizing esophagitis. A diagnosis of candida and necrotizing esophagitis was made. She was started on fluconazole daily and twice daily proton pump inhibitor. Unfortunately, she was seen in clinic once and then lost to follow up.

**Discussion:** Necrotizing esophagitis is a rare cause of acute GI bleed with unknown etiology. This disease is associated with DKA (likely due to volume depletion), hypoxia, carcinoma and ischemic events. Management includes fluid and blood product resuscitation, and treatment of the underlying disease. Complications include the development of esophageal strictures or perforation. Overall mortality is low, with the underlying disease being the cause of death in most instances. In this patient, endoscopic evaluation was key to establish the cause of her bleeding. Treatment of her candida and strict glycemic control were key for medical optimization that led to successful discharge and no re-bleeding episodes for the 3 weeks she remained for follow up.



[2357] **Figure 1.** Endoscopic view of the lower esophagus revealing diffuse ulceration.

**S2358**

**Black Esophagus: A Case of Acute Esophageal Necrosis Secondary to Binge Drinking**

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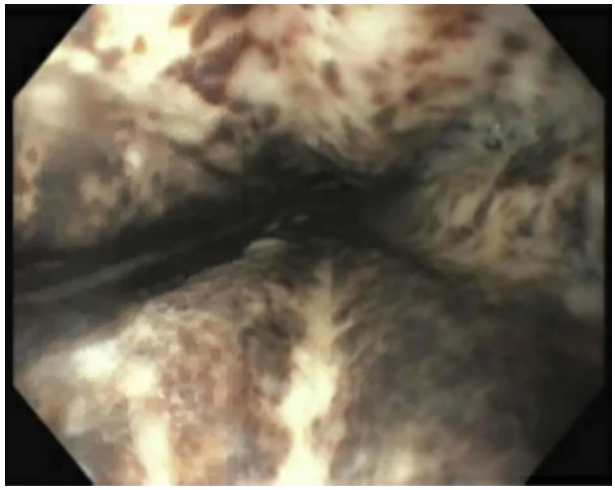
**Introduction:** Acute esophageal necrosis (AEN) is a rare, potentially life threating and under-diagnosed syndrome that presents most commonly with upper GI bleeding, hematemesis, and abdominal pain. Here we present a case of an otherwise healthy, middle-aged man with coffee ground emesis who had classic findings of black, necrotic, ulcerative esophageal mucosa on esophagogastroduodenoscopy (EGD) after presenting in the setting of binge drinking.

**Case Description/Methods:** A 47-year-old man with a history of alcohol use disorder presented to the hospital with multiple episodes of coffee ground emesis and epigastric pain. He recently restarted drinking alcohol. He denied any blood thinner use. He was hypertensive and tachycardic on arrival. His physical exam was overall non focal. His relevant lab work included a BUN 24, lactate of 8.3, lipase 11, Hgb 14.5 g/dL; AST, ALT, platelets, and INR were all normal. The patient was treated with IV fluids and started on a PPI drip. A CT scan of the abdomen/pelvis was performed and it was remarkable for nonspecific wall thickening of the thoracic esophagus. The GI service was consulted and an EGD was performed showing a 4 cm hiatal hernia and black mucosal necrosis in the mid and distal esophagus (**Figure**). The patient was made NPO, continued PPI therapy, and cardiothoracic surgery (CTS) was consulted. After gradual symptomatic improvement, he was discharged on lansoprazole, sucralfate, and morphine. He was maintained on a clear liquid diet until outpatient follow up with CTS, at which point his diet was gradually advanced without complications.

**Discussion:** AEN is a rare condition with an incidence of 0.01-0.28% of patients undergoing EGD. The pathophysiology is usually multifactorial due to ischemic injury, gastric acid exposure and impaired mucosal repair mechanisms. Multiple medical comorbidities such as diabetes, hypotension, and alcohol abuse have been associated with an increased risk of developing AEN. In our case, binge drinking, and emesis likely contributed to AEN in an otherwise healthy patient. Management is largely supportive with hydration and acid suppression but can include surgery or balloon dilation if the course is complicated by perforation or stricture formation. Prompt performance of an EGD with timely referral to CTS is recommended for risk stratification and to guide follow up management. Repeat EGD is warranted after a month to document healing.



[2358] **Figure 1.** Esophagogastroduodenoscopy of the mid and distal esophagus.

**S2359**

**Pancreatic Heterotopia at Gastroesophageal Junction**

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**Introduction:** Pancreatic heterotopia is defined as pancreatic tissue found outside the normal anatomical location. It is often an incidental finding but becomes clinically evident when complicated by pathologic changes such as inflammation, bleeding, obstruction, and malignant transformation. We present a case of a 57 years old female who presented with dyspepsia and was found to have ectopic pancreatic tissue at the gastroesophageal junction on esophagogastroduodenoscopy (EGD).
**Case Description/Methods:** A 57-year-old female with a past medical history of Cecal polyp presents to the gastroenterology clinic for epigastric pain under the rib cage ongoing for several months; pain worsens if she bends over. She denies nausea, vomiting, fever, chills, dysphagia, or weight loss. She used to take pantoprazole but stopped taking it 2 months ago. CT chest was significant for small hiatal hernia. EGD significant normal esophagus but irregular Z-line (Figure 1A), small hiatal hernia, and gastric erythema. Z-line was mildly irregular and was biopsied, which shows junctional mucosa showing focal pancreatic metaplasia/heterotopia with no malignant changes. Negative for intestinal metaplasia or dysplasia (**Figure** 1B)
**Discussion:** Heterotopic pancreas in the stomach is usually located within 5 cm of the pylorus and is more common along the greater curve, but the involvement of the GE junction is very rare [1]. Symptoms vary depending on the affected location and size of the mass. Patients with ectopic pancreatic tissue at the GE junction can be asymptomatic or present with epigastric pain, reflux or heartburn. These patients can be managed conservatively with medical treatment [2]. The risk of malignancy arising in the heterotopic pancreas is exceedingly rare, but several documented cases have appeared in the literature. Pancreatic heterotopia should be considered as a source of a potentially malignant lesion, and early treatment or close monitoring for aberrant pancreas is recommended.

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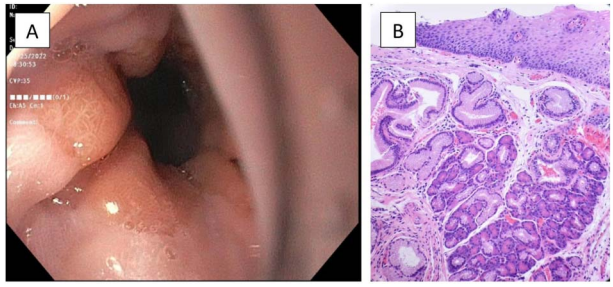


[2359] **Figure 1.** A: Endoscopy showing irregular Z- line. B: Biopsy showing junctional mucosa with focal pancreatic metaplasia or heterotopia.

**S2360**

**Plasmablastic Lymphoma Arising in Barrett's Esophagus: A Case Report**

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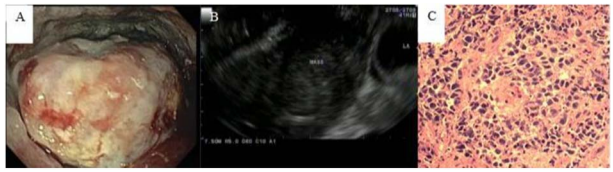
*Gundersen Health System, La Crosse, WI.*

**Introduction:** Plasmablastic lymphoma occurring in the gastrointestinal tract represents a rare malignancy in gastroenterology and has an unknown prevalence. There are only a handful of cases in the literature in which this rare malignancy presents in the GI tract, typically in patients who have preexisting immunosuppression. Here we present a patient with known Barrett's esophagus who was found to have plasmablastic lymphoma of the distal esophagus.
**Case Description/Methods:** A 54-year-old White male with history of Barrett's esophagus, presbyesophagus, GERD with hiatal hernia, adenomatous polyps, cerebral palsy, Factor V Leiden heterozygous, and seizure disorder presented to his primary care clinic for several months of progressive dysphagia, nausea, emesis, and functional decline. His Barrett's esophagus had been present for at least 9 years undergoing regular surveillance without evidence of dysplasia. His labs showed a normocytic anemia and significantly elevated ESR and CRP. A CT scan of the chest, abdomen, pelvis was performed which revealed thickening of distal third of the esophagus, 2.7 cm necrotic paraesophageal lymph node, and multiple enlarged lymph nodes in the gastric hepatic ligament. The patient underwent EGD with EUS which revealed a tumor in the distal third of esophagus (**Figure**). Multiple biopsies were taken which showed necrotic plasmablastic lymphoma, non-dysplastic Barrett's mucosa positive for CD10, CD138, MIB-1, epithelial membrane antigen, and weakly positive for PAX-5, CD45, CD56. The patient underwent additional evaluation by oncology and was started on EPOCH chemotherapy. HIV testing returned negative. Unfortunately, the malignancy progressed despite chemotherapy and radiation and the patient died about 5 months after initial diagnosis.
**Discussion:** Plasmablastic lymphoma represents a rare, aggressive subtype of diffuse large B-cell lymphomas. Typical organs involved include the GI tract, lymph nodes, oral mucosa, and skin. Unfortunately the disease course relapses frequently and is refractory to chemotherapy. It is more prevalent in patients with immunocompromised states including patients with inflammatory bowel disease, HIV, or EBV. However, in our patient this malignancy arose in the setting of Barrett's esophagus previously without dysplasia or prior HIV, EBV infections. We suggest this rare diagnosis be in the differential in patients undergoing evaluation of esophageal masses.




[2360] **Figure 1.** A – EGD image of large distal esophageal mass B – EUS image of distal esophageal mass C – Biopsy pathology with H&E stain.

**S2361**

**Pancreatic Protrusion: A Rare Case of Grade IV Hiatal Hernia With Complete Pancreatic Herniation**

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**Introduction:** Hiatal hernias are commonly described as herniation of abdominal contents through the esophageal hiatus of the diaphragm. With this, classification systems exist to define hernias as either sliding or type one, or to consider types 2, 3 and 4 as paraesophageal which also present with other sequelae. While most commonly, hiatal hernias are type one and less severe in nature, 5 percent of cases

become more involved and severe. The current prevalence of hernias ranges between 10 to 80 percent of North Americans. In this case, we shed light on the diagnosis and imaging of a rare sequelae of hiatal hernia which included not only small and large protrusion, but also herniation of the pancreas.
**Case Description/Methods:** The patient is a 95 year old woman with a past medical history of abdominal aortic aneurysm as well as a large hiatal hernia presenting with a chief complaint of poor oral intake over the last several days prior to presentation. The patient was found to be significantly lethargic compared to baseline and reported decreased oral intake due to discomfort after eating. The patient's initial vitals demonstrated a mild tachycardia with initial lab findings within normal ranges. A CT chest and abdomen was performed which demonstrated a large hiatal hernia, which now contained loops of small and large bowel as well as the pancreas. During the hospital course, interdisciplinary rounds were conducted in regards to the patient case with surgical and gastroenterology recommendations. The uniqueness of presentation of pancreatic protrusion was discussed as well as the current treatment and monitoring modalities of a Grade 4 hiatal hernia, now presenting with complications. Ultimately, the discussions were conducted with the patient who reported she was most amenable to comfort focused care with a pleasure based diet in an attempt to avoid any medical and surgical intervention.
**Discussion:** In emergent cases, such as with respiratory distress and bowel strangulation, emergent surgical intervention is considered in cases of hiatal hernia. However, lesser defined and established are the findings and management as well as treatment modalities of more severe hernias involving protrusion of pancreatic contents. Here, we add to imaging the unique presentation of a common diagnosis with rare sequelae. We also hope to add to a growing body of literature on the imaging, and potential stepwise approach to management of type 4 hiatal hernias with severe and emergent treatment considerations.

**S2362**

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**Paraneoplastic Mucous Membrane Pemphigoid: Not Your Typical Cause of Odynophagia**

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**Introduction:** Paraneoplastic Mucous Membrane Pemphigoid (MMP) is a rare inflammatory autoimmune disorder that primarily affects the oral, ocular and aerodigestive mucous membranes and is associated with an underlying malignancy. IgG, IgA, and C3 autoantibodies target basal membrane zones causing subepithelial blisters with erosive lesions in the mucosa. It is rarely seen in the clinical setting, with an incidence of 2-10 cases in 100,000 individuals. High clinical suspicion is needed in order to diagnose Paraneoplastic MMP presenting as odynophagia and dysphagia.
**Case Description/Methods:** A 88 year old female presented with a 5 month history of worsening odynophagia, dysphagia to solids and liquids and a fifty pound weight loss. Physical examination was remarkable for diffuse erosive lesions of the oral mucosa, tense bullae on the soft palate, and bilateral temporal symblepharon along with inflammation of upper and lower eyelids. Laboratory workup demonstrated elevated Cancer Antigen-125 (574.20 U/mL). Immunofluorescence study of biopsies taken from esophageal mucosa, right lateral tongue lesions and the left inferior conjunctiva were remarkable for IgG, IgM, IgA interkeratinocytic deposits at lower level of the epidermis and C3 linear deposits at the dermoepidermal junction consistent with Paraneoplastic Mucous Membrane Pemphigoid. Chest-abdominal-pelvic computed tomography scan demonstrated an irregular enhancing right adnexal mass, intra-abdominal lymphadenopathy, left hemiabdomen omental metastatic deposits and bilateral pulmonary nodules. Computed tomography guided core biopsy of a left omentum deposit was remarkable for primary ovarian metastatic high grade serous carcinoma. Due to refractory odynophagia and dysphagia, intravenous Solumedrol was initiated with subsequent improvement of symptoms. Patient was discharged with multidisciplinary team follow up for chemotherapy initiation and Rituxan induction.
**Discussion:** Paraneoplastic MMP is an uncommon autoimmune mucocutaneous disease diagnosed when there is presence of stomatitis, histologic features of acantholysis or interface dermatitis, demonstration of anti-plakin autoantibodies on biopsy, with an underlying neoplasm. Less than 500 cases have been reported where clinical features, presentation, and autoantibodies profiles have varied widely between patients highlighting the challenge of diagnosis. Early recognition of Paraneoplastic MMP presenting as odynophagia is difficult due to the overlap of symptoms in more prevalent conditions.

**S2363**

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**Pagetoid Spread of Esophageal Adenocarcinoma**

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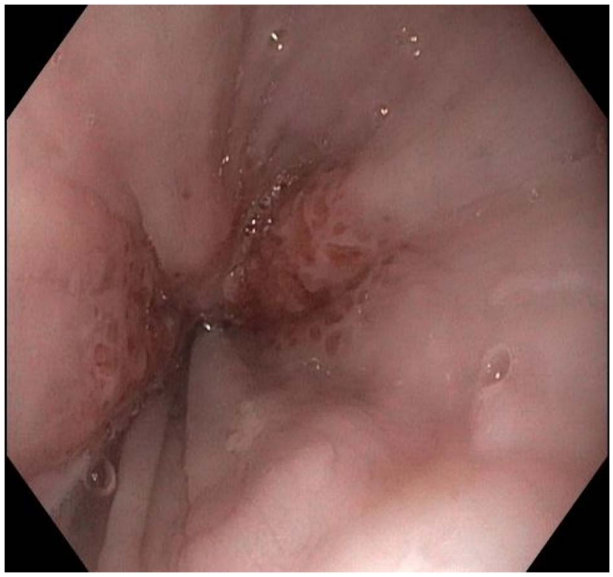
**Introduction:** Pagetoid spread refers to the "upward spreading" of cancer cells into the upper dermis from below. In the esophagus, it is a rare finding seen with poorly differentiated adenocarcinomas and is associated with deeper invasion.
**Case Description/Methods:** A 75-year-old male with history of diabetes, hypertension, and hyperlipidemia presented to the gastroenterology clinic for dysphagia to solids. He reported that his symptoms started intermittently approximately 3 weeks prior and had gradually progressed. He denied a history of acid reflux, heartburn, or prior episodes of dysphagia. He also reported an unintentional loss of 10 pounds over the past 5 months. His medications included lisinopril, metformin, pioglitazone, and simvastatin. He denied a history of tobacco or alcohol use and had no family history of gastrointestinal malignancy. Physical exam was unremarkable. To further evaluate his dysphagia, an esophagogastroduodenoscopy was performed, revealing a small fungating mass in the distal esophagus near the gastroesophageal junction. Superior to this in the distal and mid-esophagus, there was a long segment of circumferential induration with irregular, reticulated erosions (Figure). Biopsies of the lesion revealed esophageal adenocarcinoma with pagetoid spread into overlying squamous epithelium, arising in Barrett mucosa with high-grade dysplasia. Endoscopic ultrasound was subsequently performed, revealing a mass measuring 12 mm in thickness with sonographic evidence of invasion into the adventitia and one malignant appearing lymph node in the lower paraoesophageal mediastinum (staged T3, N1).
**Discussion:** Upper endoscopy is highly sensitive and specific for diagnosing esophageal cancer. However, in cases where there may not be an obvious fungating tumor, the presence of indurated mucosa with reticular erosions should alert the endoscopist to the presence of malignancy, possibly with pagetoid spread and deep invasion, as was seen in this patient.



[2363] **Figure 1.** Circumferential induration with irregular, reticulated erosions found in the mid esophagus during upper endoscopy.

S2364

**Primary Esophageal Extra-Gonadal Yolk Sac Tumor Metastasized to the Liver**

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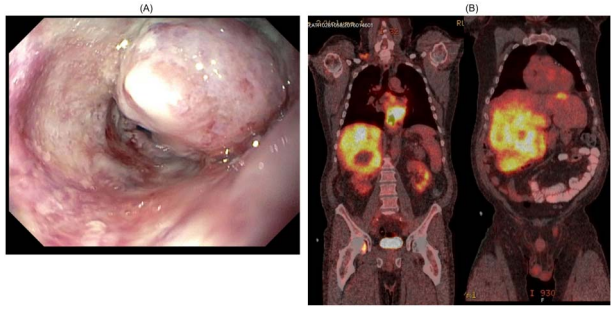
[1]University of Toledo, Toledo, OH; [2]The University of Toledo, Toledo, OH.

**Introduction:** Extra-gonadal germ cell tumors (EGCTs) usually arise from midline structures such as the retroperitoneum, mediastinum, and sacrococcygeal region. EGCTs originating from the gastrointestinal system such as the stomach and esophagus are rarely reported. Very few cases of extra-gonadal choriocarcinoma arising from the esophagus have been reported in the literature. However, no reported case of primary esophageal yolk sac tumor (YST) has been published yet in the literature. Herein, we report a rare case of a metastasized primary esophageal YST that presented with dysphagia.
**Case Description/Methods:** A 62-year-old male with a past medical history of diabetes mellitus and hypertension presented with difficulty swallowing and feeling of food stuck in the middle of his chest for 2 months. There were associated right upper-quadrant abdominal pain, early satiety, and weight loss (25 pounds) in the last 3 months. Abdominal CT demonstrated abnormal thickening in the distal esophagus and metastatic disease in the liver adjacent to the distal esophagus. Biopsy of the liver lesions showed poorly differentiated carcinoma with features consistent with YST (positive isochromosome 12p FISH). EGD showed partially obstructing tumor in the lower third of the esophagus (**Figure** A). Biopsy of the esophageal mass also showed findings consistent with the YST. PET scan showed increased activity in the lower esophagus but did not identify testicular activity (**Figure** B). Blood tests revealed AST 51, ALT 49, ALP 358, total bilirubin 0.5, AFP of 12,752, HCG of 11, and LDH of 1039. Brain MRI and testicular ultrasound findings were unremarkable. Eventually, he was diagnosed with stage IIIc M1b (liver metastasis) primary esophageal YSK. The patient was started on a chemotherapy regimen with etoposide, ifosfamide, and cisplatin. However, he died because of his esophageal yolk sac tumor 3 months after starting the chemotherapy.
**Discussion:** Primary gastrointestinal germ cell tumors have been very rarely reported in the literature. To the best of our knowledge, our case is the first extra-gonadal yolk sac tumor that originated from the esophagus and metastasized to the liver. Our case adds to the literature and provides an example of an unusual location and clinical presentation of an extra-gonadal YSK. EGCTs should be kept in mind in the differential diagnosis of middle-aged male patients with malignant evidence presenting with dysphagia and esophageal mass.




[2364] **Figure 1. A:** EGD showing partially obstructing tumor in the lower third of the esophagus. **B:** PET scan showing increased activity in the lower esophagus.

S2365

**Rare Case of Immune-Mediated Necrotizing Myopathy With Isolated Dysphagia as Presenting Symptom**

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**Introduction:** Immune mediated necrotizing myopathy (IMNM) is a rare, but progressive disease that accounts for about 19% of all inflammatory myopathies. Dysphagia occurs in 20-30% of IMNM patients. It often follows proximal muscle weakness and ensues in the later stages of the disease. We report a rare case of IMNM, presenting with dysphagia as the initial symptom, followed by proximal muscle weakness.
**Case Description/Methods:** A 74-year-old male with a past medical history of coronary artery disease, hypertension, and hyperlipidemia presented to the ED with 2-3 weeks of intractable nausea, vomiting, and dysphagia for solids and liquids. Vital signs were stable, and initial labs displayed an AST of 188 U/L and ALT of 64 U/L with a normal bilirubin. Computed tomogram of the chest, abdomen, and pelvis were negative. An esophagram showed moderate to severe tertiary contraction, no mass or stricture, and a 13 mm barium tablet passed without difficulty. Esophagogastroduodenoscopy exhibited a spastic lower esophageal sphincter. Botox injections provided no significant relief. He then developed symmetrical proximal motor weakness and repeat labs demonstrated an elevated creatine kinase (CK) level of 6,357 U/L and aldolase of 43.4 U/L. Serology revealed positive PL-7 autoxantibodies, but negative JO-1, PL-12, KU, MI-2, EJ, SRP, anti-smooth muscle, and anti-mitochondrial antibodies. Muscle biopsy did not unveil endomysial inflammation or MHC-1 sarcolemmal upregulation. The diagnosis of IMNM was suspected. A percutaneous endoscopic gastrostomy feeding tube was placed as a mean of an alternative route of nutrition. He was started on steroids and recommended to follow up with outpatient rheumatology. He expired a month later after complications from an unrelated COVID-19 infection.
**Discussion:** The typical presentation of IMNM includes painful proximal muscle weakness, elevated CK, presence of myositis-associated autoantibodies, and necrotic muscle fibers without mononuclear cell infiltrates on histology. Dysphagia occurs due to immune-mediated inflammation occurring in the skeletal muscle of the esophagus, resulting in incoordination of swallowing. Immunotherapy and intravenous immunoglobulin are often the mainstay of treatment. Our patient was unique in presentation with dysphagia as an initial presenting symptom of IMNM, as well as elevated enzymes from muscle breakdown. It is critical as clinicians to have a high degree of suspicion for IMNM due to the aggressive nature of the disease and refractoriness to treatment.

S2366

**Rare Case of 27-Year-Old With Esophageal Cancer**

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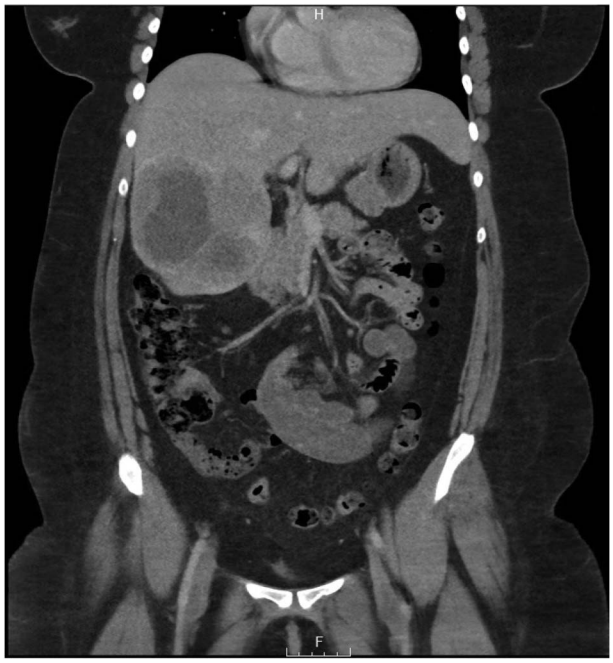
**Introduction:** Amongst esophageal cancers, neuroendocrine tumors represent 0.5 to 2.0% of cancers with a median age occurrence of 69 years old.[1] Our case represents a 27-year-old patient with neuroendocrine esophageal cancer.
**Case Description/Methods:** 27 year old female with history of obesity presented to clinic for acid reflux with globus sensation and stated symptoms are worse after eating. The patient started on famotidine 20mg daily with no improvement, patient developed dysphagia to solids daily. Esophagogastroduodenoscopy (EGD) demonstrated a 4cm mass at distal esophagus bridging the gastroesophageal junction with a 2cm cratered ulceration at the distal end of the mass. Computed tomography of the abdomen showed abdominal adenopathy and 11cm metastatic lesion at the liver as seen in **Figure** 1. Pathology was chromogranin A positive representing a neuroendocrine tumor. Patient started on carboplatin and VP-16. Currently, the patient continuing to receive chemotherapy and will receive PET scan within the next month.
**Discussion:** As neuroendocrine tumors of the esophagus compromise a small portion of esophageal cancers with a female to male ratio of 1 to 3.7 emphasizing the importance of our case report.[1] Clinical manifestations include weight loss, anemia, and dysphagia. Endoscopic biopsy will confirm biopsy. Visualization on biopsy provides guidance on staging; early esophageal cancer presents as superficial plaques, nodules or ulcerations. Advanced lesions will appear as strictures, ulcerated masses, circumferential mass, or a large ulceration. Regardless of visualization on endoscopy, diagnosis is confirmed by biopsy. Biopsy confirms with an endocrine marker, example chromogranin A, synaptophysin, and CD 56. Staging then assists in guiding treatment. Endoscopic ultrasound is one of the most accurate techniques to assess with locoregional staging. Most common sites of metastasis are liver, lungs, bones and adrenal glands. There is controversy regarding the use of diagnostic laparoscopy but procedure generally reserved for patients who have potentially resectable tumor, T3 or T4 staging, Siewert II or III adenocarcinomas of the esophageal gastroduodenal junction, or when there is a suspicion for an intraperitoneal metastatic disease that cannot be confirmed otherwise.




[2366] **Figure 1.** 1: large heterogeneous lesion within the right hepatic lobe, measuring 10.5 x 11.5 cm which appears to demonstrate central necrosis.

**REFERENCE**
1. Egashira A, Morita M, Kumagai R, et al. Neuroendocrine carcinoma of the esophagus: Clinicopathological and immunohistochemical features of 14 cases. PLoS One 2017;12(3):e0173501.

**S2367**

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**Pseudoachalasia due to Metastatic Prostate Adenocarcinoma**

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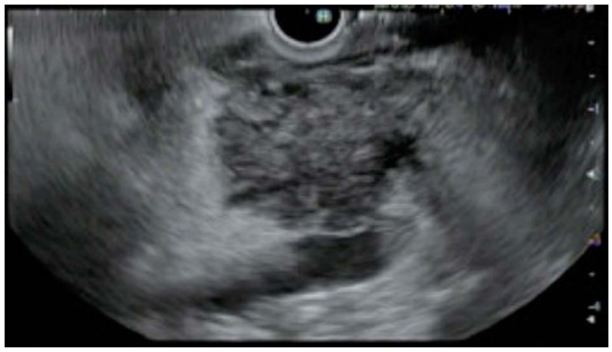
**Introduction:** Pseudoachalasia (also known as "secondary achalasia") is a condition in which esophageal dysmotility and impaired relaxation of the lower esophageal sphincter (LES) occur due to a secondary etiology such as tumor invasion or a paraneoplastic syndrome. The literature shows that only 4% of all cases of achalasia are diagnosed as pseudoachalasia and of those cases, up to 70% are due to esophageal adenocarcinoma. The early distinction between primary and secondary achalasia is important for patient outcomes as their treatments differ greatly and delayed diagnosis can cause progression of an underlying malignancy. This is especially important in cases in which the underlying malignancy is related to metastases from a distant site as the presentation of disease can be atypical.
**Case Description/Methods:** Here we describe a case of a 76-year-old man presenting with unintentional weight loss and new onset dysphagia to solids and eventually liquids. Esophagogastroduodenoscopy (EGD) was notable for a hypertonic LES with mild resistance to scope passage. CT abdomen & pelvis showed a 2.4cm x 1.5cm mediastinal mass with enlarged mediastinal lymph nodes. Barium swallow showed no intraluminal defect while manometry results were consistent with type II achalasia per Chicago Classification. Endoscopic ultrasound with biopsy revealed metastatic prostate adenocarcinoma and patient was referred to Oncology clinic for further treatment (**Figure**).
**Discussion:** This patient presented with classic symptoms of achalasia with dysphagia and weight loss and after thorough diagnostic evaluation was found to have pseudoachalasia due to mass effect from an underlying malignancy. Although esophageal adenocarcinoma is the culprit malignancy 70% of the time, this patient was diagnosed with distant metastases from prostate adenocarcinoma prior to having any urologic symptoms. Timely differentiation of achalasia from pseudoachalasia presents a diagnostic challenge as the 2 conditions are often clinically homogenous. This case presents an opportunity to highlight the clinical and endoscopic findings more often found in pseudoachalasia compared to achalasia. When compared to primary achalasia, pseudoachalasia is associated with a much shorter duration of symptoms, a greater severity of weight loss lower integration relaxation pressure on esophageal manometry and inducible relaxation of the LES with amyl nitrate. Knowledge of these relatively more predictive signs can lead to a faster diagnosis and ultimately better patient outcomes.



[2367] **Figure 1.** Hypoechogenic mass seen on endoscopic ultrasonography (EUS) in the middle mediastinum.

S2368

**Progressive Dysphagia in Patient With Cervical Plate Complicated With Posterior Pharyngeal Wall Erosion**

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**Introduction:** The incidence of esophageal perforation following anterior cervical spine surgery is reported to be between 0.02% and 1.49% with mortality rate around 6 percent. Although most esophageal erosions occur in intraoperative or immediately following surgical intervention, few cases reported with delayed presentation. Diagnosis can be made with cervical radiographs, however negative imaging does not rule out esophageal injury and further evaluation with surgical exploration warranted in the presence of high clinical suspicion.
**Case Description/Methods:** A 58-year-old male patient with past medical history significant for Parkinson's disease, and solitary cervical spinal sarcoma who underwent corpectomy, fusion of C3-C6 with cervical fixation plate placement and stereotactic body radiation therapy, presented with 3 weeks history of dysphagia, concomitant with weakness, diplopia, and weakness. Initial work up, revealed aerodigestive tract soft tissue enhancement in the cervical magnetic resonance imaging (MRI). Dysphagia progressed during the course of hospitalization and complicated with aspiration pneumonia and respiratory failure requiring intubation and mechanical ventilation. Patient subsequently underwent endoscopic gastroesophageal duodenoscopy (EGD) for further evaluation and PEG placement, which revealed posterior pharyngeal wall, and upper cervical esophageal erosion and presence of cervical fixation plate in the hypopharynx. Patient underwent surgical exploration of cervical spine, and the anterior cervical fixation plate removed with flap reconstruction and cervical dural tear repaired with resolution of his symptoms.
**Discussion:** Our patient presented with progressive dysphagia as delayed manifestation of posterior pharyngeal wall erosion. EGD revealed the diagnosis and patient underwent surgical exploration of cervical spine, and the anterior cervical fixation plate removed with flap reconstruction and cervical dural tear repaired with resolution of his symptoms. There are few cases reported in the literature with esophageal perforation following anterior cervical spine surgery. Although most esophageal erosions occur in intraoperative or immediately following surgical intervention, few cases reported with delayed presentation.

S2369

**Rare Chest Pain Presentation of Esophageal Intramural Pseudodiverticulosis in AIDS Patient**

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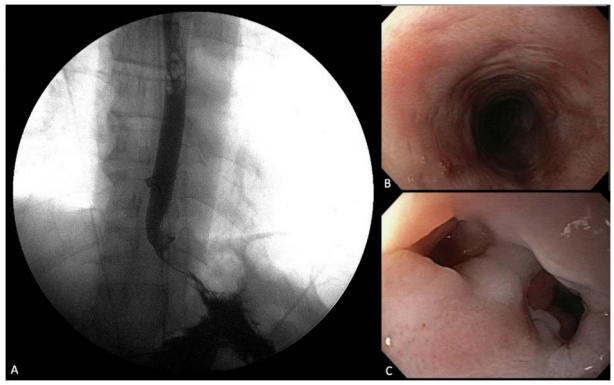
**Introduction:** Esophageal intramural pseudodiverticulosis (EIP) is an extremely rare condition. It is commonly associated with esophageal candidiasis, gastroesophageal reflux disease or esophageal carcinoma. It has a bimodal age distribution among teenagers and patients in their 50s. With the pathophysiology of EIP being poorly understood, it becomes crucial to make note of unique presentations such as this case of chest pain in a patient with AIDS.
**Case Description/Methods:** A 58-year-old male with a medical history of HIV/AIDS, presented to ED with a 5-day history of progressive worsening of chest pain and shortness of breath. He denied dysphagia, odynophagia, nausea and vomiting. Patient is nonadherent to his HIV medications. His vitals were stable but was febrile at 39.5 C. Physical exam revealed a cachectic, ill-appearing man with the rest of the exam being unremarkable. Labs revealed an absolute neutrophil count of 800 cells/mm, CD4 count of 53 cells/mm and negative blood cultures. Acute coronary syndrome was ruled out. He was treated with cefepime for his neutropenic fever. CT of the chest and abdomen showed distal esophagitis with a small hiatal hernia, no pneumonia or pulmonary embolism. Esophagogastroduodenoscopy (EGD) showed multiple small pseudodiverticula throughout the esophagus and a large diverticulum noted just proximal to the gastroesophageal junction (GEJ). No endoscopic evidence of esophagitis was noted and the gastroscope easily passed the GEJ with no resistance. The area adjacent to the distal diverticulum was biopsied and showed chronic and focal acute inflammation. A subsequent esophogram showed a small contrast-filled focal outpouching at the distal aspect of the esophagus correlating with esophageal diverticulum. After a negative cardiopulmonary evaluation for the etiology of chest pain it was attributed to an uncommon symptom of EIP. Chronic inflammation from chronic esophagitis likely contributed to the progression of EIP. He was started on PPIs, anti-retroviral medications, prophylactic fluconazole which improved his symptoms (**Figure**).
**Discussion:** Chest pain without dysphagia is an extremely uncommon presenting symptom of EIP. Once cardiac etiology of chest pain is excluded, an EGD should follow to further assess the etiology of symptoms and if EIP is noted it is important to rule out complications such as a mediastinal fistula formation and perforation. Treatment should include management of the underlying inflammatory/infectious pathology such as HIV in this patient.




[2369] **Figure 1.** 1. A. Esophagogram showing a large esophageal diverticulum adjacent to the LES. B. Multiple small pseudodiverticula in the upper esophagus. C. Large esophageal diverticula adjacent to LES.

S2370

**Rapid Sequence Induction-Intubation Facilitated Passage of Impacted Food in Esophagus: A Single Institutional Case Series**

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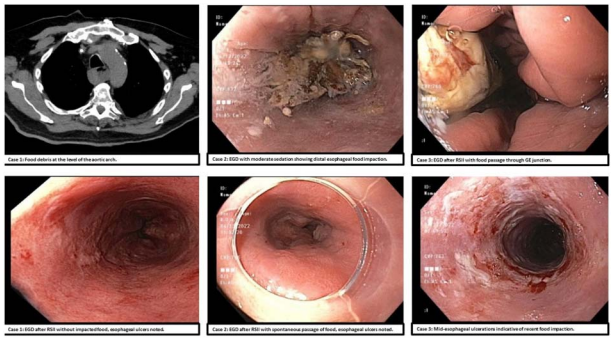
**Introduction:** Complete esophageal food impaction (EFI) if not relieved may progress to perforation. Existing literature does not support use of pharmacologic interventions alone and urgent endoscopic intervention is recommended to relieve the obstruction. We present a case-series of 3 EFI resolved without any endoscopic interventions after rapid sequence induction-intubation (RSII).
**Case Description/Methods:** Case 1: A 77-year-old man with history of EFI from "distal esophageal spasm" presented 4 days after the onset of symptoms suspicious for EFI. CT chest showed esophageal dilation and food debris up to level of the aortic arch. Case 2: An 87-year-old man with history of EFI from a massive goiter presented 3 days after the onset of solid food dysphagia and inability to swallow saliva. Esophagogastroduodenoscopy (EGD) with moderate sedation showed EFI at distal esophagus which was not able to be manipulated further. Case 3: A 65-year-old woman with history of chronic dysphagia and EFI relieved with glucagon in the past presented one day after the onset of severe dysphagia and inability to tolerate oral secretions. All 3 patients underwent RSII using fentanyl and rocuronium with subsequent EGD. In the first 2 cases EGD did not reveal impacted food despite recent CT/endoscopic evidence of showing such. In the third case distal EFI was noted, but it was now able to be pushed into the stomach. All 3 patients had severe esophageal ulceration due to their EFI (Figure).
**Discussion:** Esophageal foreign body impaction is the third leading cause of non-biliary gastrointestinal (GI) emergencies following upper and lower GI bleeding. Current guidelines recommend urgent EGD for complete EFI. Airway protection with intubation is recommended only for patients who are at increased risk of aspiration. All 3 of our patients presented with delayed EFI (more than 24 hours from symptom onset) and were thus at higher risk of perforation. RSII not only reduced risk of aspiration but also reduced need for endoscopic intervention and shortened sedation time. RSII involves rapid onset of deep

sedation which is hypothesized to relax smooth muscle fibers in distal esophagus followed by paralysis which relaxes skeletal muscles fibers in the diaphragm and proximal esophagus allowing passage of impacted food. We propose RSII prior to EGD for patients presenting with delayed EFI to minimize need for prolonged endoscopic interventions, thus reducing risk of esophageal perforation.



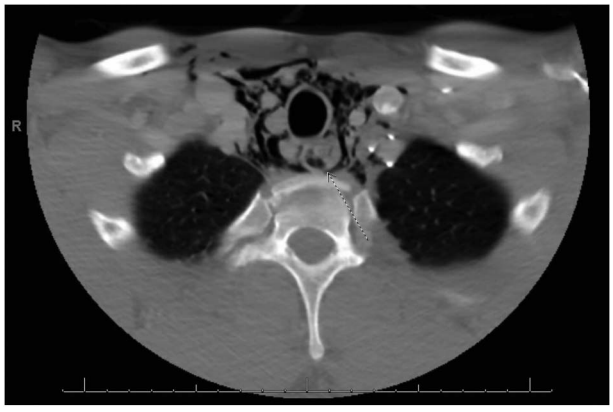
[2370] **Figure 1.** Radiographic and endoscopic findings before and after rapid sequence induction-intubation for Cases 1, 2 and 3.

**S2371**

**Ripped by Chips: A Case Report of Esophageal Perforation After Consumption of Chips**

*Diana Jomaa, MD, Taha Ashraf, MD, Jeffery Tang, MD.*
*Henry Ford Health, Detroit, MI.*

**Introduction:** Eosinophilic esophagitis (EoE) is an uncommon condition defined as a chronic, immune-mediated, esophageal disease characterized clinically by symptoms related to esophageal dysfunction and histologically by eosinophil-predominant inflammation.[1] Patients typically present with esophageal dysphagia and food impaction. We present a unique and rare case presentation of a young male patient who presents with pneumomediastinum in the setting of undiagnosed EoE.
**Case Description/Methods:** A 31-year-old male with no significant past medical history presents to the emergency department with lower neck pain. He reports sudden onset of pain after consuming a sandwich containing potato chips. This was followed by 2 immediate episodes of hematemesis. Vital signs were within normal limits. Physical exam was significant for bilateral crepitus on exam. CT Chest with contrast was significant for extensive pneumomediastinum around the esophagus suggestive of an esophageal perforation. CT neck was significant for extensive emphysema in the neck and pneumomediastinum with esophageal wall emphysema suggestive of esophageal rupture (**Figure**). An esophagram revealed no evidence of gross esophageal leak suggesting a contained micro perforation, so he was managed conservatively with IV pain medications, initial period of nil by mouth followed by slow escalation to a liquid diet. His diet was slowly escalated, and he was discharged home in stable condition. Two months later at an outpatient gastroenterology follow up, an esophagogastroduodenoscopy was significant for ringed esophagus, longitudinal furrows, white plaques and white specks throughout the entire esophagus. Biopsies were consistent with eosinophilic esophagitis with increase eosinophil count in the proximal esophagus. He was started on a daily proton pump inhibitor with ongoing follow up.
**Discussion:** The incidence of EoE appears to be increasing, in part due to rising recognition of the disorder. It is important for physicians to identify its features early to prevent further complications. Esophageal perforation is a rare but serious complication of eosinophilic esophagitis, occurring in ~2% of cases.[2] Ongoing inflammation from longstanding untreated EoE has previously been reported to have led to esophageal perforation but rarely in such a dramatic fashion.[3]




[2371] **Figure 1.** 1. Significant emphysema in the neck and pneumomediastinum with thinning of the posterolateral aspect of esophagus concerning for esophageal rupture.

**S2372**

**Severe Dysphasia With Eosinophilic Esophagitis Pattern of Injury and Autoimmune Gastritis Related to Pembrolizumab Therapy**

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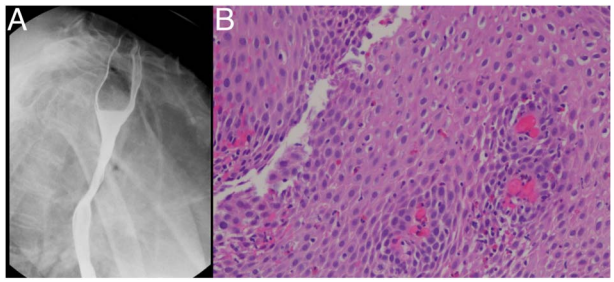
*[1]UT HSC - Houston, Houston, TX; [2]UT Health Sciences Center at Houston, Houston, TX; [3]UTH HS - Houston, Houston, TX.*

**Introduction:** Immune checkpoint inhibitor (ICI) therapies are effective treatments of many cancer types, including non-small cell lung carcinoma of the lung (NSCLC). While efficacious, ICI therapy can be associated with immune-related adverse effects (irAEs). We present a 67-year-old man with NSCLC who developed severe dysphasia with an eosinophilic esophagitis (EoE) pattern of injury on histopathology and concomitant autoimmune gastritis while on pembrolizumab maintenance therapy.
**Case Description/Methods:** A 67-year-old man with NSCLC on 3 years of maintenance pembrolizumab with stable disease presented to GI clinic for evaluation of progressively worsening solid and liquid food dysphagia with regurgitation. Surveillance CT scans prior to advent of symptoms showed stable mediastinal lymphadenopathy with stable external compression of the mid-esophagus. A barium swallow study showed narrowing of the mid-esophagus. An EGD was performed and showed mid-esophageal lumen narrowing (Figure). Biopsies were obtained from the distal esophagus and showed up to 42 intraepithelial eosinophils (Eo) per high power field, consistent with an EoE pattern of injury. A subsequent EGD with stomach mapping prior to initiation of therapy showed concurrent autoimmune gastritis with no increase of Eo infiltrates. PPI therapy twice a day was initiated but the symptoms did not improve after 2 months. Topical glucocorticoid therapy with budesonide was then started. Despite dual therapy, his dysphagia progressed. Pembrolizumab was discontinued and docetaxel with ramucirumab was started, which resulted in a complete resolution of his dysphagia symptoms. Patient was maintained on PPI therapy with a plan to repeat EGD.

**Discussion:** While GI irAEs are increasingly recognized, ICI-EoE is a rare entity. In the literature, only one other case of ICI-EoE has been reported. This patient's dysphagia in the context of >15 intraepithelial Eo per high power field without other identifiable etiologies of increased esophageal Eo, and the resolution of symptoms with the discontinuation of pembrolizumab is consistent with ICI-EoE. In addition, the patient had concomitant autoimmune gastritis, which is also a rare but increasingly recognized irAE. Our case demonstrated both entities presenting in one patient. Treatment of irAEs often involves discontinuation of the ICI. Refractory cases may require systemic immunosuppression with corticosteroids or biologic therapy.




[2372] **Figure 1.** 1A – Mid-esophageal narrowing on barium swallow study Figure 1B – Intraepithelial eosinophilia consistent with EoE.

**S2373**

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**Russell Body Esophagitis: A Possible Indication to Screen for Hematologic Malignancy**

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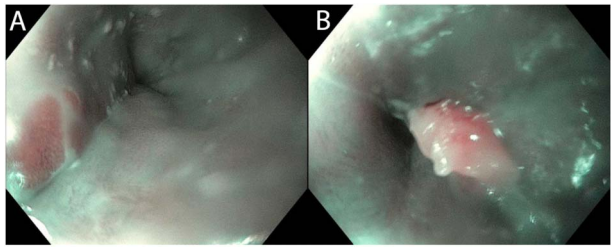
**Introduction:** Russell Body Esophagitis (RBE) was first described in 2005. Diagnosis is made via identification of Russell Bodies (RBs) within plasma cells on biopsies of esophageal mucosa. RBs are cytoplasmic inclusions of immunoglobulins found in plasma cells. RBE is believed to share its pathogenesis with other gastrointestinal RBs. RBE is associated with Barrett's Esophagus. Russell Body Gastritis (RBG) and Russell Body Duodenitis have more published cases and an association with Helicobacter pylori.
**Case Description/Methods:** An elderly man with lymphoplasmacytic lymphoma (LPL) presented with a chief complaint of weakness. The patient was found to be anemic, so Gastroenterology was consulted for further management. Esophagogastroduodenoscopy was performed. Islands of salmon-colored mucosa were present at 35 cm. At the GE junction (approximately 40 cm), an 8 mm nodule was present (Figure B). No source of upper gastrointestinal bleeding was identified. Biopsies were negative for Barrett's Esophagus and H. pylori. The esophageal nodule was diagnosed as Herpes Esophagitis. H&E staining demonstrated pink globules within B Cells in the lamina propria (Figure C). The CD138 and CD79a stains confirmed the presence of lymphocytes. In situ hybridization for Kappa light chain showed clonal B lymphocytes. Additionally, clonal immunoglobulin heavy chain (IGH) and Kappa light chain (IGK) gene rearrangements were detected by PCR. Overall, the findings were compatible with RBE.
**Discussion:** The lesions appeared to be Barrett's Esophagus; however, pathology did not show any Barrett's metaplasia, making this case unique. RBG has been associated malignancy, especially in the absence of H. pylori, but there are no reported cases of RBE and malignancy. LPL has been detected in the stomach and ileum; LPL is classically known to cause the formation of RBs in bone marrow. The presence of RBs in the esophagus is a possible manifestation of this patient's LPL. This case adds to the growing body of evidence that RBs (in the absence of H. pylori and Barrett's Esophagus) herald malignancy. This case describes a new presentation of RBE – in the absence of Barrett's Esophagus. Our case of RBE comes in a patient with LPL and Herpes Esophagitis. Unlike LPL, Herpes infection has never been reported to be associated with RBs in the gastrointestinal tract. The herpetic nodule are noteworthy given herpesviruses are a known source of Russell Body Cervicitis.




[2373] **Figure 1.** A (Left): Salmon-colored mucosa visualized at 35cm. Figure B (right): 8 mm herpetic nodule observed at gastroesophageal junction.

**S2374**

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**Recurrent Strictures in a Patient With Missed Diagnosis of Eosinophilic Esophagitis**

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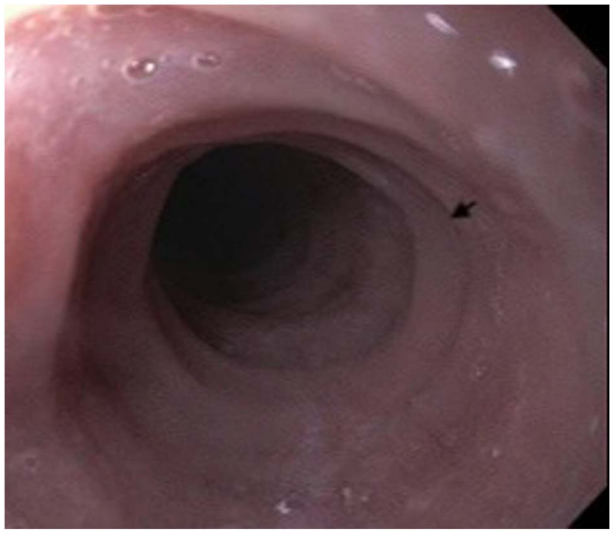
**Introduction:** EoE is an antigen-mediated inflammatory disorder of the esophagus that affects both children and adults. Diagnosis requires symptoms related to esophageal dysfunction and esophageal biopsy showing >15 eosinophils/hpf is confirmatory for diagnosis. Proton pump inhibitors (PPIs) are considered the first line agents for the treatment of EoE, together with elimination diet and topical corticosteroids. Nonadherence to therapy and undiagnosed disease can increase the risk of fibro-stricturing disease and food impactions. Practicing evidence-based guidelines in diagnosing and managing patients with EoE can allow standardization of care and prevent complications in patients with EoE.
**Case Description/Methods:** A 34-year-old man presented with a longstanding history of intermittent dysphagia to solids. He had an emergent esophagogastroduodenoscopy (EGD) for food impaction about 3 years prior to presentation. No biopsies were obtained at that time, and he was prescribed a short course of PPI before discharge. On current presentation, the patient underwent an EGD which showed multiple untraversable strictures requiring dilation (**Figure**). Biopsy from multiple sites showed >200 eosinophils/hpf, consistent with a diagnosis of eosinophilic esophagitis (EoE). Thereafter, the patient was initiated on PPIs but had to undergo further dilations for recurrent strictures. Topical steroids were subsequently added to the therapy with satisfactory response.
**Discussion:** Once considered rare, EoE is now a well-known entity, with an estimated prevalence of 51.6/100000 in the US. However, variability and deviations from standard therapy continues to persist among gastroenterologists. According to a national survey conducted in 2020 among 240 gastroenterologists, only 24 % were "very familiar" with the EoE guidelines and only 12 % followed EoE guidelines in their clinical practice. The current case underscores the need to standardize clinical practice related to the care of patients with EoE in order to provide quality and evidence-based care and improve overall outcomes.



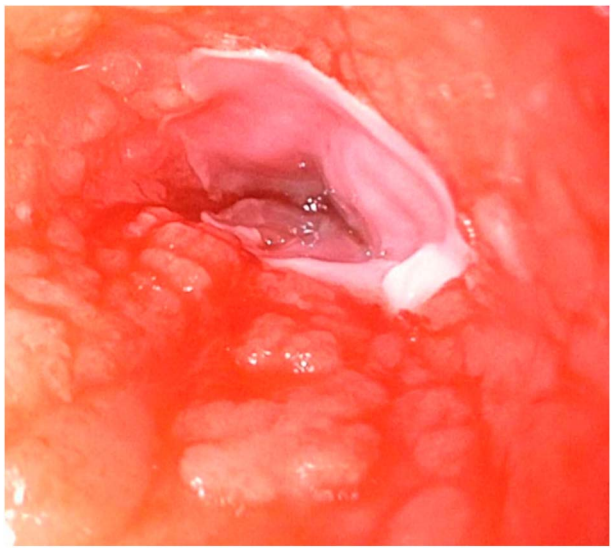
[2374] **Figure 1.** EGD showed trachealization of the esophagus.

**S2375**

**Steroid-Resistant Esophagitis Dissecans Superficialis With Recurrent Stricturing**

*Thomas King, MD, Richard A. Manfready, MD, AM, Michael Brown, MD.*
*Rush University Medical Center, Chicago, IL.*

**Introduction:** Esophagitis dissecans superficialis (EDS) is a rare endoscopic finding characterized by sloughing of esophageal mucosa. Dysphagia, regurgitation of casts, and vomiting can result from superficial mucosal stripping. Histologically, the epithelium is split above the basal layer. Cases may be associated with desquamating dermatologic conditions such as pemphigus vulgaris, or due to bisphosphonates, chemical irritants, or autoimmune conditions. EDS is a benign condition, and most cases respond to a short course of steroids with rapid symptomatic and histologic improvement. Chronic cases are very rare. We present a rare case of structuring EDS that did not respond to steroids or serial dilations.
**Case Description/Methods:** A 79-year-old man with a history of short-segment nondysplastic Barrett's esophagus presented with progressive dysphagia to solids, vomiting, and weight loss. Index upper endoscopy revealed severe esophagitis with peeling white strips of mucosa and a 7 mm lumen in the distal esophagus. Histology showed necrotic fragments of epithelium with minimal focal inflammation and granulation tissue without intestinal metaplasia, eosinophilia, bacterial colonies, or fungal organisms on special staining. Findings were consistent with EDS. When symptoms failed to respond to acid suppression alone, he was started on 20 mg of prednisone daily. The stricture was balloon dilated with temporary relief. Over the course of the next year, the patient presented monthly with severe dysphagia requiring repeat stricture dilation and triamcinolone injection. Stricturing and sloughing persisted despite an empiric trail of 40 mg of prednisone daily, pantoprazole, and high-dose famotidine. Repeat biopsies remained consistent with EDS. Workup was negative for inflammatory and autoimmune conditions, including IgG4. Esophageal motility on high-resolution manometry was normal apart from a high-pressure zone at the stricture. The patient eventually required a gastrostomy tube for nutrition (Figure).
**Discussion:** EDS, a form of desquamative esophagitis, is typically self-limiting or responds readily to steroids. Stricturing EDS is a rare entity described infrequently in case reports, which have identified etiologies such as esophageal IgG4 disease. Our patient did not have any of the common risk factors given the absence of dermatologic and autoimmune conditions and the lack of culprit medications. The presence of recurrent debilitating strictures may necessitate alternative therapies such as biologic therapy or surgery.



[2375] **Figure 1.** Distal esophageal desquamation and stricturing.

S2376

**Squamous Cell Carcinoma Arising From Zenker's Diverticulum**

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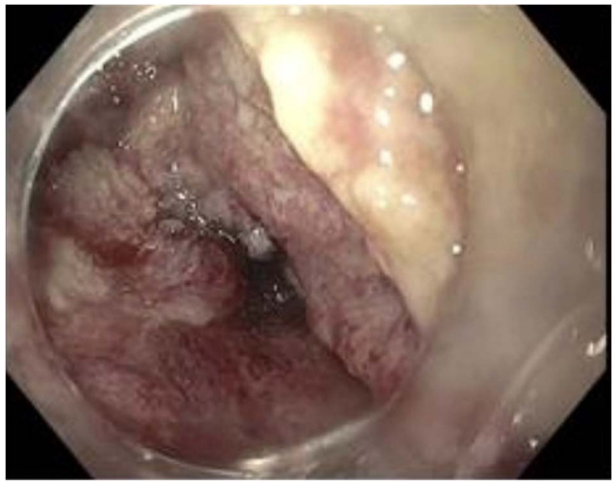
**Introduction:** The development of esophageal squamous cell carcinoma (ESCC) from a Zenker's diverticulum (ZD) is a rare event. Recurrence of ZD after a surgical or procedural intervention can be a warning sign for development of malignancy. We present a case of an 83-year-old male with 20-year history of symptomatic ZD, who had undergone multiple surgical interventions and ultimately found to have ESCC arising from his ZD.
**Case Description/Methods:** An 83-year-old male with symptomatic ZD for 20 years presented with worsening dysphagia. 4-years prior, the patient had undergone an endoscopic cricopharyngeal myotomy with resolution of ZD both clinically and on post-operative imaging. He did well for 50-months but had recurrence of dysphagia. Barium esophagram confirmed recurrent ZD. Another cricopharyngeal myotomy was performed. Post-operative modified-barium swallow showed persistent ZD and evidence of significant aspiration. A transcervical diverticulectomy was then performed; however, the surgery was technically difficult and only a partial diverticulectomy was performed. Pathology did not demonstrate SCC. He had limited improvement in symptoms and was readmitted 4-months later with worsening dysphagia, odynophagia, hemoptysis, and unintentional weight loss. A repeat barium esophagram demonstrated a large diverticulum. At this time the gastroenterology service was consulted for for consideration of a peroral endoscopic myotomy of the ZD (Z-POEM). Advancement of the endoscope into the cervical esophagus revealed a circumferential ulceration with firm friable tissue measuring 2-3 cm which extended from the distal rim of the ZD. The mucosa at the base of the ZD pouch was normal. Biopsies revealed invasive, moderately differentiated ESCC. The patient has since been discharged and is undergoing evaluation for continued management (**Figure**).
**Discussion:** Development of ESCC from a pharyngoesophageal diverticulum is a rare event with reports in the literature ranging from 0.3-7%. Malignancy is thought to develop in part to chronic inflammation of the diverticulum but otherwise the pathogenesis is not well defined. Patients who appear to be at higher risk for development include those with large ZD, advanced age and longstanding history of ZD. A distinct worsening of dysphagia may indicate development of malignancy. Recurrence of ZD in a patient with a history of prior surgical or procedural treatment should also be concerning. Development of such symptoms should prompt urgent investigation.



[2376] **Figure 1.** 1: Upper endoscopy revealed a circumferential ulceration with firm friable tissue measuring 2-3 cm and extending from the distal inner rim of the ZD.

S2377

**Successful Treatment of a T2 Esophageal Adenocarcinoma With Endoscopic Mucosal Resection**

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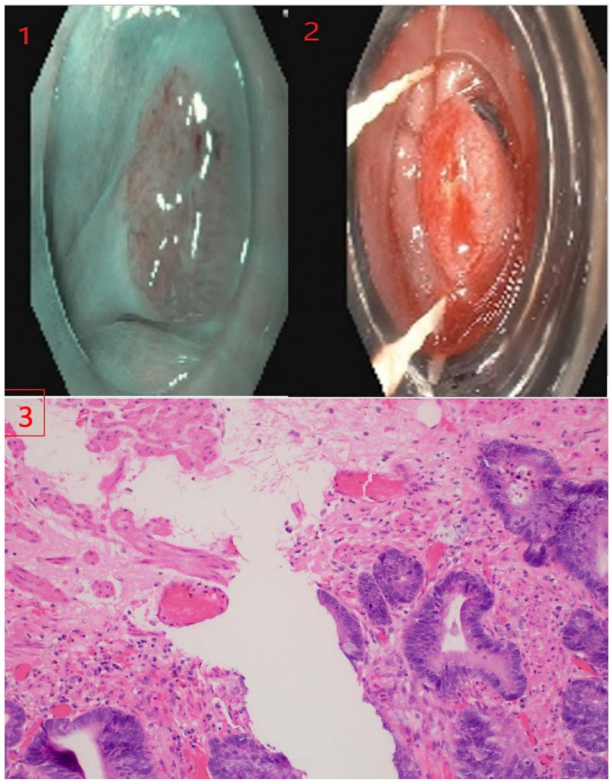
**Introduction:** The incidence of esophageal adenocarcinoma (EAC) continues to rise in the US with a 16-fold increase over the last 50 years. The prognosis of the disease is very poor with a 5-year survival rate of only 14%. The standard of care for EAC is dependent on the TNM stage of the cancer. Typically, patients with T1a stage cancer are treated with Endoscopic Mucosal Resection (EMR) while T2 stage and above are treated with esophagectomy. We present a patient with nodular BE with dysplasia who was successfully treated with EMR, later identified histologically as T2 stage.
**Case Description/Methods:** A 77-yr old male with past medical history significant for cirrhosis secondary to chronic hepatitis C, GERD, and remote tobacco use was referred for management of his short segment Barrett's Esophagus with high grade dysplasia with focal radio frequency ablation (RFA). His vitals and physical exam were unremarkable. 6-months following the initial session of RFA, patient returned for an EGD and was noted to have 10mm nodular lesion in the distal esophagus at 36cm from the incisors. The lesion was removed via band-EMR (**Figure**). Pathology came back as EAC with the tumor extending into the muscularis propria signifying a T2 grade tumor. All margins and edges (at least 3mm) were uninvolved, and no lymph node involvement was noted. CT abdomen and chest showed no evidence of metastatic disease. The tumor was graded as a T2N0M0. An endoscopic ultrasound (EUS) was performed which showed no suspicious mucosal abnormalities. The patient subsequently underwent 2 more follow up EGDs with biopsies and a EUS to evaluate for evidence of residual lesion. There was no evidence of dysplasia or carcinoma noted on imaging and biopsies.
**Discussion:** The treatment protocol for T2N0M0 disease is not well defined as it falls into an intermediate group. Typically, patients undergo esophagectomy with some institutions opting to use neoadjuvant chemotherapy prior to surgical resection. Of note, EMR of T2 esophageal adenocarcinoma is not the mainstay of treatment. However, in our case, EMR was successful in treating T2 EAC. With high morbidity and mortality associated with esophagectomy especially in the presence of other co-morbidities like cirrhosis, our case highlights the importance of possibly considering EMR for selected T2 tumors as an alternative option especially in high risk surgical groups.




[2377] **Figure 1.** (1.1): Esophageal Nodule. (1.2): Banding of Esophageal Nodule with EMR. (1.3) Histopathology of esophageal adenocarcinoma.

**S2378**

**Small Cell Neuroendocrine Esophageal Carcinoma: A Rare Cause of Dysphagia**

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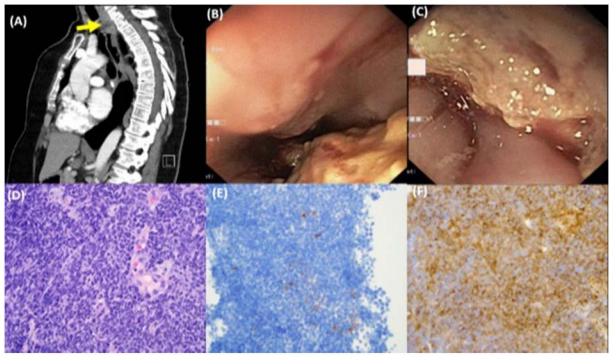
**Introduction:** New onset dysphagia warrants endoscopic evaluation to rule out structural etiologies, including malignancy. The vast majority ( >95%) of esophageal cancers are adenocarcinoma or squamous cell carcinoma. We report a rare case of esophageal small-cell neuroendocrine carcinoma (NEC) presenting with dysphagia.

**Case Description/Methods:** A 76-year-old African American female with a history of left lung non-small cell neoplasm status post left lower lobectomy and adjuvant chemo-radiation presented to an outside hospital with dysphagia to solids and unintentional weight loss over the previous month. She denied history of smoking. Her BP was 163/81, pulse 81, temperature 36.7 and respiratory rate 16. Rest of the physical exam was normal. Laboratory tests showed mild normocytic anemia (hemoglobin 11.2 g/dL). Chest CT scan revealed a soft tissue mass at junction of the cervical and thoracic esophagus (Figure 1A). Due to the proximity of the mass to the airway, she was transferred to our hospital for evaluation. EGD confirmed an esophageal mass located at 16 cm from incisors, just distal to the upper esophageal sphincter, causing near complete esophageal obstruction (Figures 1B-C). Using a pediatric gastroscope, the mass was traversed and noted to extend to 25 cm from the incisors. Multiple biopsies were obtained with histology showing small cell NEC (Figures 1D-F). Positron emission tomography revealed lymph nodes metastases above and below the diaphragm, liver and multiple bones. Brain magnetic resonance imaging was negative for metastases. Patient was not a candidate for surgical resection or stenting given the proximal esophageal location of the tumor and required gastrostomy tube placement for nutrition by interventional radiology. Palliative chemoradiation (carboplatin, etoposide, atezolizumab) has started and the patient is currently tolerating therapy for 2 months since diagnosis.

**Discussion:** Esophageal small-cell NEC is a rare esophageal malignancy, accounting for 0.4-2.0% of esophageal cancers. Esophageal small-cell NEC are typically highly aggressive, frequently have metastasized by the time of diagnosis, and associated with poor prognosis. While surgical resection may be attempted for limited disease, those with metastases are typically managed with chemotherapy, with or without radiotherapy. Gastroenterologists should include rare esophageal malignancies, including small cell NEC, on their differential for new onset dysphagia to solids.




[2378] **Figure 1.** 1A: Computerized tomography scan of the chest (sagittal view) showing a soft tissue mass at the junction of the cervical and thoracic esophagus (arrow). Figure 1B-C: Esophagogastroduodenoscopy showing an esophageal mass located at 16 cm from incisors causing near complete obstruction of the esophagus. Figure 1D: A high power (400x, H&E) view shows a highly cellular tumor comprised of basophilic cells, with scant cytoplasm, high nuclear/cytoplasmic ratio, salt-and pepper chromatin, and nuclear molding with crush artifact. There are abundant

apoptotic cells and frequent mitotic figures, indicating a highly proliferative tumor. Figure 1E: 400x, chromogranin immunostaining, showing focally positive cells, consistent with neuroendocrine differentiation. Figure 1F: 400x, synaptophysin immunostaining, showing diffuse positivity in tumor cells, consistent with neuroendocrine differentiation.

**S2379**

**Squamous Cell Carcinoma of the Gastroesophageal Junction as an Unusual Underlying Cause of Pseudoachalasia**

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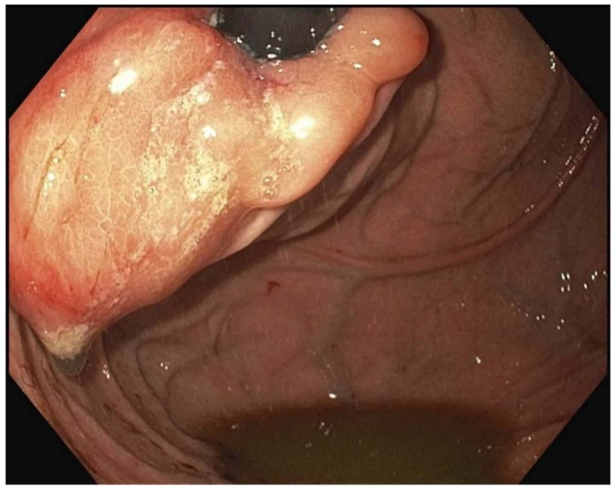
**Introduction:** Achalasia is a rare esophageal motility disorder characterized by progressive dysphagia to solids and eventually liquids[1-2]. Pseudoachalasia refers to an achalasia secondary to an underlying etiology, such as masses, post-operative complications, or paraneoplastic syndromes[1-2]. The most common cause of pseudoachalasia is adenocarcinoma of the cardia or esophagus [1] however, we discuss a rare case that deviates from the typical etiology of esophageal cancer.
**Case Description/Methods:** This is a 71-year-old male presenting with intermittent dysphagia for years with rapid progression for a month including intolerance of oral intake and 6-pound weight loss. On examination he was hemodynamically stable with a soft, nontender abdomen. His initial barium esophagram revealed decreased esophageal peristalsis, esophageal distention, and narrowing at the gastro-esophageal junction (GEJ) with "bird beak" appearance consistent with possible achalasia. Further imaging studies were performed with computed tomography (CT), esophagogastroduodenoscopy (EGD) with dilation, and endoscopic ultrasound with biopsies revealing an invasive squamous cell carcinoma of the GEJ with submucosal involvement extending into the gastric cardia (**Figure**). We present our patient as the first reported case of pseudoachalasia due to SCC of the GEJ. The patient underwent esophageal stenting and mediport placement with plans for neoadjuvant chemotherapy with concurrent radiotherapy followed by definitive resection.
**Discussion:** Pseudoachalasia is often misdiagnosed for primary achalasia because of its rarity, indistinguishable symptomatology, and normal findings on CT, EGD, and biopsy[1-2]. Nearly 75% of pseudoachalasia cases are due to adenocarcinoma of the gastric cardia[1]. Only one case of pseudoachalasia has been known to be caused by SCC in the distal esophagus[3]. SCC of the GEJ is an extremely rare esophageal malignancy and has never previously been reported as an underlying cause of pseudoachalasia.



[2379] **Figure 1.** Esophagogastroduodenoscopy revealed severe stricture at the GEJ with a 4 cm submucosal mass at the GEJ with extension to the gastric cardia.

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**S2380**

**Small Cell Neuroendocrine Carcinoma of the Esophagus Presenting as Acute Hematemesis**

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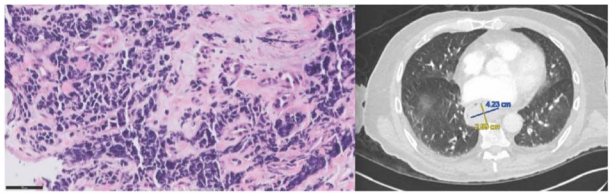
**Introduction:** Esophageal cancer often presents as dysphagia and weight loss and is diagnosed with the help of imaging and endoscopy. Patients with esophageal carcinoma are most commonly found to have either squamous cell carcinoma or esophageal adenocarcinoma. Gastrointestinal small cell neuroendocrine carcinomas account for 0.1% to 1% of all gastrointestinal malignancies and can be seen throughout the GI tract, most commonly noted in the small intestine, appendix, pancreas, and rectum. We present a case of an 85-year-old male with decreasing appetite and 1 day of hematemesis found to have high grade small cell neuroendocrine carcinoma of the esophagus with necrosis.
**Case Description/Methods:** An 85-year-old male with a medical history of hypertension, dementia, and gastrointestinal bleed 5 years ago in the setting of NSAID use with an unremarkable EGD, presented to the hospital with 4 episodes of coughing up blood. Patient had a productive cough with white sputum for 1 week with decreased appetite and progressive weakness. On admission, the patient was hemodynamically stable with labs significant for down-trending hemoglobin from 12.4 to 10.7 on repeat labs. CT pulmonary angiography and CT chest for concern for hemoptysis showed soft tissue thickening and a mass involving the proximal stomach and the gastroesophageal junction with thickening of the distal esophagus and large amount of fluid and debris (**Figure**). Furthermore, enlarged surrounding lymph nodes and thickening of the distal aspect of the stomach were concerning for malignancy and metastatic disease. Subsequent endoscopy showed a large partially obstructing bleeding esophageal mass with food debris proximal to the mass. Biopsy revealed high grade small cell neuroendocrine carcinoma with necrosis involving fibromuscular tissue (**Figure**). Given the obstructive nature of presentation and poor prognosis, the family deferred feeding tube placement and elected for inpatient hospice.
**Discussion:** High grade neuroendocrine tumors can be subdivided into small cell and large cell types and occur throughout the body, more commonly seen in the lungs, appendix, and small intestine. They can present as hemoptysis in patients with pulmonary involvement. Hematemesis is an unusual presentation of esophageal cancer, and esophageal small cell neuroendocrine carcinomas are extremely rare with a poor prognosis given the aggressive nature of the disease. Treatment modalities including chemotherapy, surgery, and radiation are selected based on staging of the disease.



[2380] **Figure 1.** Image 1: Left: H&E stain of esophageal mass biopsy revealing tumor cells that are small with scant cytoplasm, salt and pepper chromatin, inconspicuous nucleoli, nuclear molding and smudging. Right: CT chest revealing for 4.2 x 3.0 cm distal esophageal mass. There is circumferential wall thickening involving the mid and lower esophagus.

**S2381**

**Tracheoesophageal Fistula Presenting With Gastrostomy Tube Pressurization**

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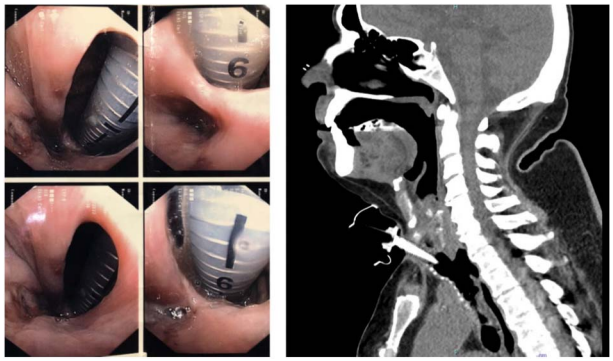
**Introduction:** Patients with COVID pneumonia who require intubation and prolonged mechanical ventilation are at risk for complications such as recurrent infection, tracheomalacia, tracheal stenosis, and the development of tracheoesophageal fistula (TEF). TEF is a devastating complication where the trachea and esophagus develop an abnormal connection in the lower airway that dramatically increases the mortality of critically ill patients by recurrent aspiration and pneumonias. Though commonly associated with neoplasms another risk is pressure induced ischemia of the common wall between the trachea and esophagus. This can occur due to overinflation of the endotracheal (ET) cuff, especially with concomitant use of a nasogastric tube (NGT). Definitive management requires surgical repair.
**Case Description/Methods:** A 69-year-old male patient presented with acute hypoxemic respiratory failure secondary to COVID pneumonia requiring intubation and insertion of an NGT. On day 29 the patient underwent percutaneous enterogastrostomy (PEG) placement and tracheostomy; it was noted intraoperatively that the tracheal mucosa was inflamed and friable. On day 36 bronchoscopy was performed through the tracheostomy tube due to concerns for mucus plugging. Friable mucosa with granulation tissue was seen at the distal end of the tube, so an extra-long tracheostomy tube was exchanged to bypass the granulation tissue. Later that night the ventilator measured a 50% discrepancy between the delivered and exhaled tidal volumes, triggering an alarm. Exam noted distension of the PEG-bag with a fluid meniscus in the tubing moving in sync with each respiration. TEF was considered and bronchoscopic evaluation confirmed a 1-centimeter TEF. The patient underwent successful TEF repair and is slowly recovering (**Figure**).
**Discussion:** Critically ill patients who require prolonged support are at high risk of complications and device related injury. With each device-day there is an increased risk of complications, such as infection, dislodgement, and pressure-related injuries. This case highlights the importance of serial physical examinations as well as understanding possible device related complications. An unexpected finding, such as a persistent air leak, air in a PEG bag, or a fluctuating meniscus should raise suspicion for the development of a serious complication and would warrant prompt confirmatory testing. Our literature review revealed no reports of a PEG tube abnormalities as a presenting finding for TEF.



[2381] **Figure 1.** Left: TEF as visualized from the esophagus during endoscopy. ET tube visible through TEF. Right: Neck CT showing the connection between the Esophagus and Trachea.

**S2382**

**Tracheal Compression as an Initial Presentation of Achalasia**

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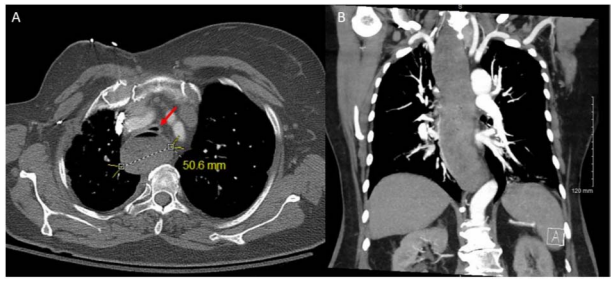
**Introduction:** Achalasia is due to esophageal dysmotility and impaired lower esophageal sphincter relaxation. It is often insidious in presentation with gradual disease progression. We present a case of acute respiratory failure due to external compression of the trachea by a dilated esophagus as an initial presentation of achalasia.
**Case Description/Methods:** An 80-year-old female with degenerative cerebellar ataxia and prior breast cancer treated with mastectomy and chemoradiation presents with acute respiratory failure due to esophageal compression of the trachea. The week prior to her admission, the patient had mild coughing following meals, but never had symptoms of dysphagia or reflux. She experienced sudden onset dyspnea with rapid progression prompting her to present to an outside hospital where she was found to be in severe respiratory failure with audible stridor. She was intubated and computed tomography showed a dilated food-filled esophagus with external compression of the intrathoracic trachea. She was transferred to our institution for a higher level of care. On arrival, the patient was already intubated and sedated. Review of external imaging showed a dilated food-filled esophagus to around 5cm with external compression of the intrathoracic trachea. Esophagogastroduodenoscopy (EGD) showed retained food throughout the esophagus with a hypertonic lower esophageal sphincter. The sphincter was dilated to 10mm using balloon dilation and the food was passed into the stomach. Subsequent EGD was performed with Botox injection into the lower esophageal sphincter. It was determined that the underlying cause of the patient's esophageal dilation was due to undiagnosed achalasia likely related to her underlying cerebellar ataxia. The patient was subsequently extubated and tolerated an oral diet. She was discharged with plan for outpatient follow up (**Figure**).
**Discussion:** Achalasia is typically an insidious disease most commonly presenting as dysphagia initially. In this case, the initial presentation of achalasia was tracheal compression due to a dilated esophagus from previously undiagnosed achalasia likely secondary to long standing cerebellar ataxia. Tracheal compression is an uncommon complication of achalasia and is rarely the initial presentation. Early recognition of achalasia and its spectrum of illness severity is important to prevent serious complications of the disease.




[2382] **Figure 1.** Thoracic computed tomography with intravenous contrast. (A) Axial and (B) coronal views demonstrate a dilated esophagus to 5cm with associated compression of the trachea demonstrated by the red arrow.

**S2383**

**Take My Breath Away: A Rare Cause of Dysphagia**

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**Introduction:** Esophageal adenoid cystic carcinoma (EACC) is an extremely rare malignancy, with very poor prognosis with distant metastases, accounting for only 0.1% of esophageal malignancies. It is most common in the parotid and salivary glands with low locoregional metastasis, hence dysphagia is rarely associated with EACC. Here we report a case that debuted with dysphagia.
**Case Description/Methods:** An 81-year-old man with a history of smoking, chronic obstructive pulmonary disease, and acute promyelocytic leukemia in remission presented with 4 years of intermittent dysphagia to solids that improved with drinking water. He denied weight loss, chest pain, vomiting, regurgitation of food contents, odynophagia or abdominal pain. Physical exam and laboratories were unremarkable. Barium swallow showed a corkscrew appearance suggestive of esophageal spasm. GI service was consulted and recommended an EGD for direct visualization as symptoms were not consistent with esophageal spams. A chest/neck CT was ordered by Pneumology service due to associated symptoms of persistent cough and dysphonia. The CT revealed a lesion in the subcarinal compartment measuring approximately 2.7×2.1cm proximal to the left mainstem bronchus, inseparable from the esophagus. An endobronchial ultrasound (EBUS) with fine-needle aspiration (FNA) was performed on the mediastinal mass, and pathology reported an adenoid cystic carcinoma. Positron emission tomography–computed tomography showed abnormal metabolic activity of posterior mediastinum retrocarinal space, with no lymphadenopathy or evidence of distant metastases. EGD with normal esophageal mucosa, fixed extramural compression against the esophageal wall. An endoscopic ultrasound (EUS) with FNA confirmed the presence of EACC. Pathology showed cohesive clusters and aggregates of atypical epithelial cells in a tubular and cribriform pattern.
**Discussion:** The most common endoscopic finding of EACC is an esophageal endoluminal mass. Histologic findings of this tumor may show 3 growth patterns: cribriform, tubular, or solid, the latter being associated with a worse prognosis. Treatment of choice for EACC is radical excision. Left main bronchus involvement require a tracheal and bronchus reconstruction. The reported 5-year survival rate of EACC is around 35%. This highlights the importance of expedited evaluation in cases of dysphagia and the awareness of rare causes such as EACC, as the diagnosis may be confused with other entities if direct visualization and biopsy are not performed.

**S2384**

**The First Case of Water Immersion Endoscopy in the Definitive Diagnosis of Atrio: Esophageal Fistula**

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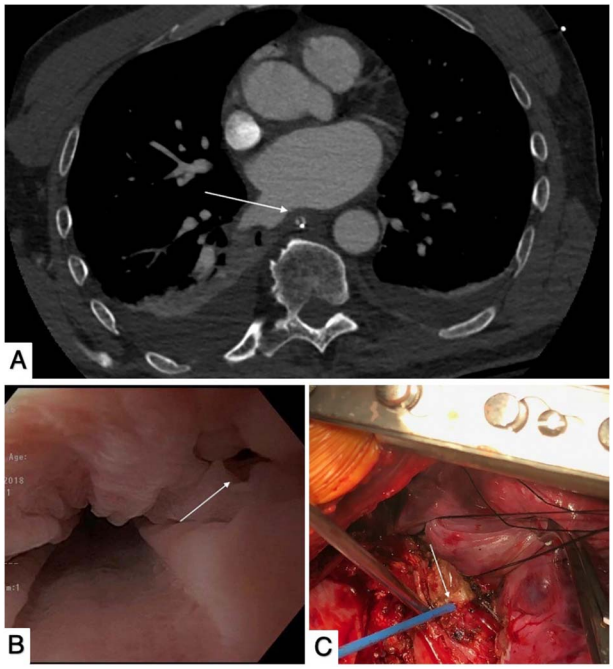
**Introduction:** Atrial fibrillation (AF) is the most common heart rhythm disorder with estimated global prevalence of 33.5 million. Catheter ablation is the treatment modality of choice for refractory AF. Atrio-esophageal fistula (AEF) is a serious complication of catheter ablation. Diagnosis is critical to the management of AEF, and includes computed tomography of the chest, esophagram and esophagogastroduodenoscopy (EGD). Prompt diagnosis of AEF is essential for survival.
**Case Description/Methods:** A 56-year-old male with AF presented with fever, fatigue and bilateral leg weakness. Twenty days prior to presentation he underwent pulmonary vein isolation radiofrequency ablation and discharged the following day. Upon re-presentation his vitals were notable for fever and tachycardia. Labs revealed a normal white count and hemoglobin, and elevated lactate and troponin. One hour after arrival he developed complex focal seizure and AF with rapid ventricular rate followed by respiratory decompensation requiring intubation. His blood cultures revealed Streptococcus mitis and Gemella haemolysans. His MRI disclosed numerous bilateral emboli concerning for endocarditis, however TEE did not reveal any valvular vegetation, thus proposing AEF as a possible complication accounting for the emboli. CT chest angiogram revealed subtle irregularity along the posterior wall of the left atrium (figure A), but did not provide conclusive evidence to warrant high-risk cardiothoracic surgery. Water immersion EGD was performed to diagnose and localize the fistula, revealing esophageal defect in the proximal esophagus with movement of non-bloody debris through the tract (figure B). One hemostatic clip was placed near the tract to assist with localization for surgical closure. The following day the patient underwent right thoracotomy with placement of intercostal muscle flap to fistula (Figure C).
**Discussion:** AEF is a rare complication of radiofrequency ablation therapy for AF. High mortality rate is primarily attributed to major cerebrovascular morbidity from septic thrombi. Currently there is no effective modality to visualize AEF safely and reliably. Water immersion colonoscopy allows for completion of difficult colonoscopies, decreased insertion pain, and increased adenoma-detection rates. This technique was applied to identify the AEF. Water immersion EGD is an important modality that can confidently be considered within the diagnostic armamentarium of gastroenterologists to ensure safe and rapid diagnosis and treatment of AEF.




[2384] **Figure 1.** 1: (A) Illustrates a subtle contour abnormality involving the posterior wall of the left atrium (arrow) concerning for AEF (B) Water immersion endoscopy illustrating 3mm defect (arrow) in the wall of the esophagus (C) Underwent right thoracotomy to access AEF. This image highlights 3mm fistula site (arrow) that is approximated to be at the midpoint of the atrium.

**S2385**

**The Great Mimicker: Esophageal Sarcoidosis Masquerading as Malignancy**

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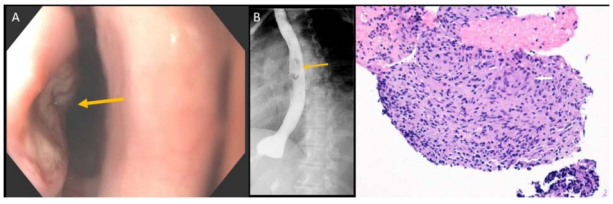
**Introduction:** Sarcoidosis is a chronic, multi-system disorder that most commonly affects the lungs. Luminal gastrointestinal involvement including the esophagus is rare, occurring in less than 2% of patients. We describe a case not previously reported of esophageal sarcoidosis presenting as a cratered, esophageal ulcer concerning for malignancy.
**Case Description/Methods:** A 43-year-old woman presented to clinic with 3 months of solid food dysphagia and epigastric pain. Her medical history was notable for laparoscopic Roux-en-Y gastric bypass surgery 2 years earlier. Esophagogastroduodenoscopy (EGD) demonstrated a large, cratered ulcer with irregular borders in the mid esophagus concerning for malignancy (**Figure**). The ulcer was deep with concern for an underlying fistula, and esophageal biopsies were deferred until further imaging was performed. Esophagram showed a 3 cm ovoid filling defect in the anterolateral mid-thoracic esophagus with surrounding edema. Computed tomography scan of the chest demonstrated a subcarinal soft tissue mass adjacent to the right esophageal wall measuring 21x27x47 mm, difficult to distinguish from surrounding posterior mediastinal lymph nodes. There was prominent mediastinal lymphadenopathy. Esophagogastroduodenoscopy with endoscopic ultrasound showed a 17x40 mm non-circumferential, hypoechoic, and heterogeneous mass with invasion of the esophageal wall from the adventitia through the mucosa, which appeared consistent with a large lymph node. Pathology showed multiple non-necrotizing granulomas consistent with sarcoidosis. The patient was referred to Rheumatology and due to prior intolerance to prednisone (severe psychosis), treated with mycophenolate mofetil and close follow-up in clinic.
**Discussion:** This is a rare presentation of esophageal sarcoidosis presenting as a large, cratered esophageal ulcer concerning for malignancy. Esophageal sarcoidosis most often presents with dysphagia that can occur secondary to mucosal involvement with stricture or mass, infiltration of the myenteric plexus and associated dysmotility, or external compression from lymphadenopathy. Corticosteroids are the primary treatment, and the duration of therapy depends on the clinical response. In patients with atypical endoscopic findings including mucosal lesions, strictures, or ulcers, sarcoidosis should be considered.




[2385] **Figure 1.** A: Image from esophagogastroduodenoscopy of a large, cratered ulcer (arrow) in the middle esophagus. B: Fluoroscopic images from esophagram demonstrating an irregular, ovoid shaped filling defect consistent with ulcer (arrow) in the mid esophagus. C: Microscopic image from endoscopic ultrasound-guided fine needle biopsy of the mediastinal lesion demonstrating a non-necrotizing granuloma with a multinucleated giant cell (arrow). Hematoxylin and eosin stain; original magnification x 200.

**S2386**

**The Esophagus and Beyond: A Case of Refractory Chest Pain**

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**Introduction:** Esophageal disorders are a common etiology for noncardiac chest pain, including esophagogastric junction outlet obstruction(EGJOO). Herein, we describe a case of a patient with EGJOO with refractory chest pain who was ultimately found to have Hereditary alpha-tryptasemia(HaT). This rare presentation allows clinicians to broaden the differential when investigating noncardiac chest pain.
**Case Description/Methods:** The case is an 80-year-old female with 2 decades of unresolved chest pain. For years, she was treated for reflux and functional chest pain. Subsequent testing revealed EGJOO and hypercontractile peristalsis. Despite treatment with various endoscopic (botulinum toxin, celiac plexus block and peroral endoscopic myotomy) and medical therapies (nitrates, calcium channel blockers, antispasmodics, neuromodulators, PPIs, H2 blockers, viscous lidocaine, cannabinoids, gut-directed hypnosis, cognitive behavioral therapy) her symptoms progressed to frequent and severe chest pain with nausea and abdominal pain. She became intolerant to all foods with symptoms now triggered by exercise. With a history of 48 allergies, a tryptase level was found to be elevated (tryptase 21.4 ng/ml [nl < 10.9]).

Genetic testing revealed 2 copies of the *TPSAB1* gene and negative KIT mutation. These findings, with her clinical syndrome, met diagnostic criteria for HaT (1, 2). Her symptoms improved with cromolyn but she could not tolerate antihistamines.

**Discussion:** HaT is an autosomal dominant disorder with elevated tryptase levels due to an increased copy number in the *TPSAB1* gene (1). It is found in up to 6% of individuals with European ancestry. The clinical presentation varies ranging from asymptomatic to idiopathic anaphylaxis or dysautonomia (1). GI manifestations of HaT are still unclear due to the under recognition of this disorder and overlap with mast cell activation syndrome. Increased duodenal mast cells have been described in patients with HaT and symptoms of abdominal pain, diarrhea, constipation and nausea (2). The objective of this case is to bring awareness of HaT to the GI community to promote early diagnosis, especially in patients presenting with multiple allergies and refractory symptoms. Further studies are needed to elucidate the role of HaT in GI motility.

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**S2387**

**The Connections You Don't Want to Make: A Rare Case of Atrio-Esophageal Fistula Formation After Thermal Ablation for Atrial Fibrillation**

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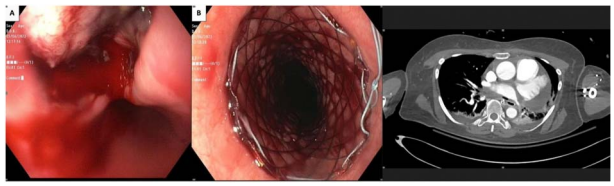
**Introduction:** Atrio-esophageal fistula (AEF) is a rare yet devastating complication of ablation therapy for atrial fibrillation, with a prevalence of 0.015%-0.04%. Unfortunately, AEF tends to be rapidly fatal without swift recognition and emergent intervention. We present a unique case of AEF in a patient presenting with focal weakness and hemodynamic instability.

**Case Description/Methods:** A 65-year-old female with a history of Hypertrophic Cardiomyopathy, Atrial fibrillation on Eliquis who had undergone elective thermal ablation 2 weeks ago presented to the emergency department with left-sided weakness (NIHSS score of 6). A non-contrast CT head revealed multiple infarcts in the right frontal and cerebellar region. Her hospital course was complicated by recurrent episodes of painless large volume, bright red blood per rectum, fever of 105.7F, and hypotension. A massive transfusion protocol was initiated, and she was intubated for airway protection. An emergent upper endoscopy demonstrated a blood-filled esophagus that was adequately suctioned. A 3cm tear gushing blood in a pulsatile manner was unveiled at the 10 o'clock position, 28cm from the incisors (**Figure**). A 20x120mm Through-The-Scope fully covered self-expandable metallic stent was deployed and secured under direct vision with a stent fixation device. After successful hemostasis, she underwent a CT Angiogram of the chest and abdomen, which unveiled air within the left atrium and a defect in the wall suggestive of an AEF (**Figure**). Given her rapid deterioration requiring 3 pressors and persistent bacteremia, she was not a candidate for surgical intervention, and the family opted for palliative extubation and comfort measures.

**Discussion:** Our case demonstrates the rare yet devastating complication of thermal injury via catheter ablation with the formation of an Atrio-esophageal fistula, which has a mortality rate of up to 80% and usually presents within 6 weeks of the procedure. Therefore, clinicians should have a high index of suspicion for patients presenting with hematochezia, bacteremia, and neurological symptoms in the setting of ablation therapy. Early recognition and management with antibiotics and surgical repair is the key. In cases where immediate surgery is not feasible, an esophageal stent provides immediate hemostasis.



[2387] **Figure 1.** Upper endoscopy demonstrating 3 cm esophageal tear at the 10 o'clock position in the esophagus, 28 cm from the incisors (Left, A), and status post 20x120mm TTS stent placement covering the tear with successful hemostasis (Center, B). Coronal view of a CT angiogram showing air within the left atrium and a small defect in the esophageal wall suggestive of an atrial-esophageal fistula with esophageal stent placement. Additionally, air seen within the pericardium (Right).

**S2388**

**Time for a Rendezvous: Multi-Disciplinary Management of Complete Esophageal Obstruction in a Patient Following Radiation to Post-Surgical Anatomy**

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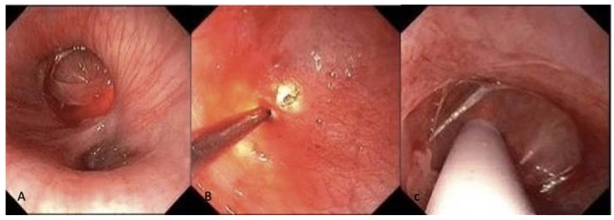
**Introduction:** Esophageal stricture is a common complication of radiation exposure. Balloon dilation restores luminal patency for partial narrowing, though complete obstruction can require recanalization through combined anterograde and retrograde endoscopic dilation (CARD), also known as the rendezvous technique. We describe a case of successful CARD recanalization following multimodal cancer treatment.

**Case Description/Methods:** A 65-year-old male with right tonsillar cancer underwent resection of the right tonsil, tongue base and palate with pectoralis major reconstruction, followed by radiation. One year later, he reported progressive dysphagia to solids and liquids that required gastrostomy tube placement to maintain adequate nutrition. Laryngoscopy revealed radiation changes but no evidence of malignancy, and upper endoscopy revealed complete luminal obstruction. Retrograde endoscopy via gastrostomy initially was unsuccessful due to scarring, so wire-guided Savary dilation was performed until an ultra-thin endoscope could pass into the stomach. Retrograde inspection revealed a benign-appearing, complete stenosis in the proximal esophagus. Concurrent antegrade laryngoscopy visualized a thin tissue membrane with distal endoscopic illumination. The membrane was pierced retrograde under direct visualization with the sharp end of a Savary wire, after which it was advanced out the mouth. Stepwise antegrade Savary dilation then was performed over this wire to 45 French under visualization with the retrograde endoscope. Post-dilation inspection showed moderate mucosal disruption without luminal perforation, significant improvement in luminal narrowing, and minimal bleeding. A 12 French nasogastric tube was placed to maintain luminal patency, and the patient was discharged home. While he regained the ability to swallow liquids, a second dilation session was required to achieve complete dysphagia relief (**Figure**).

**Discussion:** Most CARD procedures are performed by gastroenterologists to treat post-radiation luminal obstruction. However, this case demonstrates CARD can be performed collaboratively with surgeons, which can be helpful in the setting of post-operative anatomy. CARD is effective, safe, and well-tolerated, with a lower complication risk (esophageal perforation, pneumomediastinum) compared to blind antegrade dilation. High technical success rates (83%) and frequent dysphagia resolution (44%) make CARD a preferred approach to restore luminal patency in patients with severe radiation-induced dysphagia.



[2388] **Figure 1.** A- Stricture in the upper third of the esophagus; B- Piercing of the membrane in the upper third of the esophagus; C- Savary dilation of the upper third of the esophagus.

S2389

**The Odd Couple: Active *H. pylori* Infection Within Long Segment Barrett's Esophagus**

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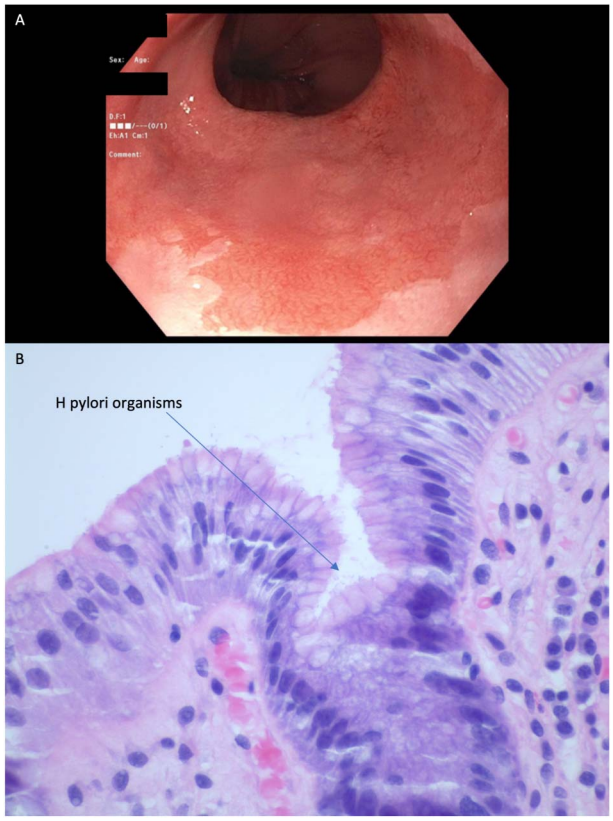
**Introduction:** Barrett's esophagus (BE) is a consequence of poorly-controlled GERD leading to intestinal metaplasia, which predisposes to esophageal cancer. Similarly, there is a well-established link between *Helicobacter pylori* and development of gastric cancer. However, there is a negative association between *H. pylori* and risk of BE. Here, we present a rare case of *H. pylori* infection within Barrett's mucosa.
**Case Description/Methods:** A 54-year-old woman was referred for one month of dysphagia to solids. She described food sticking in her mid-esophagus without difficulty swallowing pills or liquids. She also endorsed significant heartburn and belching but denied systemic symptoms, regurgitation, abdominal pain, or weight loss. EGD demonstrated a 5 cm segment of circumferential salmon-colored mucosa suspicious for Barrett's esophagus, a small esophageal ulcer, and a normal stomach and duodenum. Gastric biopsies revealed mild gastritis with *H. pylori* organisms. Esophageal biopsies showed intestinal metaplasia consistent with Barrett's esophagus as well as *H. pylori* organisms. Quadruple therapy was initiated with metronidazole, tetracycline, omeprazole, and bismuth (**Figure**).
**Discussion:** We describe a rare case of *H. pylori* discovered within long-segment Barrett's esophagus. Evidence suggests an inverse association between *H. pylori* and BE. However, a recent meta-analysis demonstrated that this association disappeared in patients with GERD. One proposed mechanism for *H. pylori's* protectiveness against BE is decreased gastric acid production. Together, this suggests *H. pylori* protects against BE by preventing development of GERD but once GERD is present, *H. pylori* is no longer protective. Thus, our simultaneous diagnosis of BE and *H. pylori* could be explained by long-standing GERD and BE prior to H pylori infection. While it is uncommon to diagnose *H. pylori* and BE together, it is even rarer to see *H. pylori* within Barrett's mucosa, given the organism's proclivity for gastric epithelium. One potential explanation is that this patient's significant GERD allowed reflux of *H. pylori* organisms into the esophagus, leading to colonization of the metaplastic epithelium. Furthermore, there may be microscopic satellites of gastric mucosa within the segment of BE, providing a friendlier environment in which the organisms could grow. More observation is needed to determine the significance and long-term effects of active *H. pylori* infection in Barrett's mucosa and if this facilitates progression to dysplasia or carcinoma.




[2389] **Figure 1.** 1. A. Esophagoduodenoscopy (EGD) image of 5 cm circumferential salmon mucosa consistent with Barrett's esophagus. B. Hematoxylin and eosin (H&E) stain of esophageal mucosa demonstrating intestinal metaplasia and H pylori organisms.

S2390

**The Dark Side of Drugs: Black Esophagus - A Case Report of Acute Esophageal Necrosis in a Methamphetamine User**

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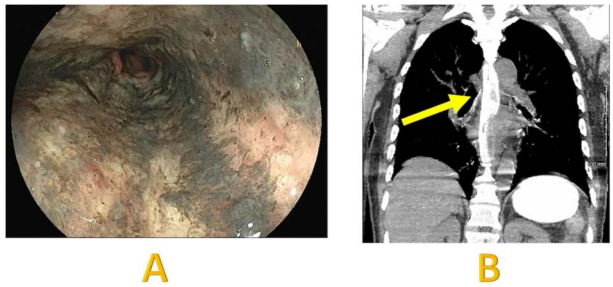
**Introduction:** Acute esophageal necrosis (AEN) is a severe form of acute esophagitis, visualised endoscopically as dark, black-appearing distal esophageal mucosa. It is a rare finding, with prevalence of 0.01-0.28% of all upper GI endoscopic procedures. Etiology is multifactorial, with tissue hypoperfusion and compromised local protective barriers contributing to necrosis. Patients commonly present with upper GI bleeding. Perforation and stricture formation are important complications. Though direct mortality related to esophageal disease is $< 6\%$, around 1/3rd of patients succumb to the underlying illness.
**Case Description/Methods:** A 60-year-old male with history of polysubstance abuse presented with coffee ground emesis and melena for 10 days. He was found to be in septic shock, respiratory and renal failure. With ongoing hematemesis and agitation, he was intubated emergently, needing 2 pressors and CRRT. Transaminitis, hypoalbuminemia and coagulopathy were consistent with active alcohol use. UDS was positive for methamphetamine and HCV antibody was detected. With 1.5 L of coffee ground aspirated overnight in the orogastric tube, ocreotide and PPI infusion were initiated with concerns for a variceal bleed. CT chest without contrast showed patulous esophagus with high attenuation enteric contrast material throughout the esophagus. Subsequent EGD revealed circumferential black mucosal discoloration in the esophagus which abruptly stopped at the GE junction, consistent with AEN (**Figure**). A melanotic clot was seen in the fundus but no specific bleeding site was identified. OGT was pulled out. With supportive care, the patient's improved clinically with time. He was weaned off pressors and extubated on day 7. Blood cultures grew Candida, which was suspected to have translocated from the necrotic esophagus. Micafungin was initiated for candidemia. Patient was discharged on day 16, with referral to substance rehab and hepatology for HCV treatment.
**Discussion:** Though esophageal necrosis is rare owing to the rich network of collaterals, hemodynamic changes in shock can make the mucosa more vulnerable to ischemia. Mechanical ventilation and vasoactive agents can compound these changes. Though an association has not yet been reported, methamphetamine, a known vasoconstrictor, could have precipitated AEN in our patient. Most cases resolve with standard of care for upper GI bleeding. It is important to avoid nasogastric tubes and maintain NPO status for at least 24 hours to avoid perforation and ensure mucosal healing.




[2390] **Figure 1.** Image A: EGD showing diffuse circumferential black mucosal discoloration, consistent with AEN. Image B: CT Chest without contrast showing patulous esophagus with high attenuation enteric contrast material throughout the esophagus (arrow).

**S2391**

**Time Needed for Development of an Epiphrenic Diverticula?**

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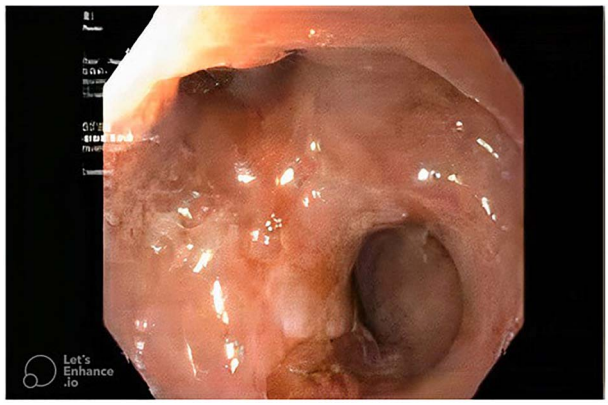
*Parkview Medical Center, Pueblo, CO.*

**Introduction:** Esophageal diverticulum are a relatively rare clinical entity with an increasing prevalence due to the greater availability of radiographic and endoscopic examination. These diverticula can be divided into proximal or Zenker diverticulum and distal or epiphrenic diverticula. Epiphrenic diverticula comprise approximately 20%, and due to their rarity etiology is unclear. They are thought to be secondary to motility disorders of the esophagus but obtaining diagnostic information before and after development is exceedingly difficult as most are asymptomatic. An undiagnosed diverticulum can lead to regurgitation, weight loss, aspiration, and gastrointestinal bleeding. Here we present a case of an epiphrenic diverticulum that developed over a period of 7 months.
**Case Description/Methods:** A 79-year-old male with a past medical history of hyperlipidemia and hypertension was admitted to the hospital for a stroke. As a result, he developed moderate oropharyngeal dysphagia diagnosed by modified barium swallow (MBS). There was no contrast extravasation to indicate the presence of a diverticulum at that time. He was discharged home on ground solids and a liquid diet. Over the next 7 months, he had worsening dysphagia and weight loss, ultimately resulting in endoscopic evaluation. He was found to have an epiphrenic diverticulum present in the middle third of the esophagus at 34 cm (**Figure**).
**Discussion:** Epiphrenic diverticulum continues to be a rare condition despite increased availability in diagnostic modalities. Our patient developed a diverticulum over 7 months after developing persistent dysphagia secondary to a stroke, as shown by its absence on MBS. This further supports dysmotility as the etiology and also helps to establish a timeline required for the development. It is vital to remember epiphrenic diverticulum as a consequence of compromised esophageal motility as it can impact morbidity as it did in our patient. Further reports are needed to determine causality and the long-term effects of epiphrenic diverticulum.




[2391] **Figure 1.** Endoscopic image of an epiphrenic diverticula that developed over a period of 7 months.

**S2392**

**The Marching Band: Pseudoachalasia Secondary to Gastric Band Slippage**

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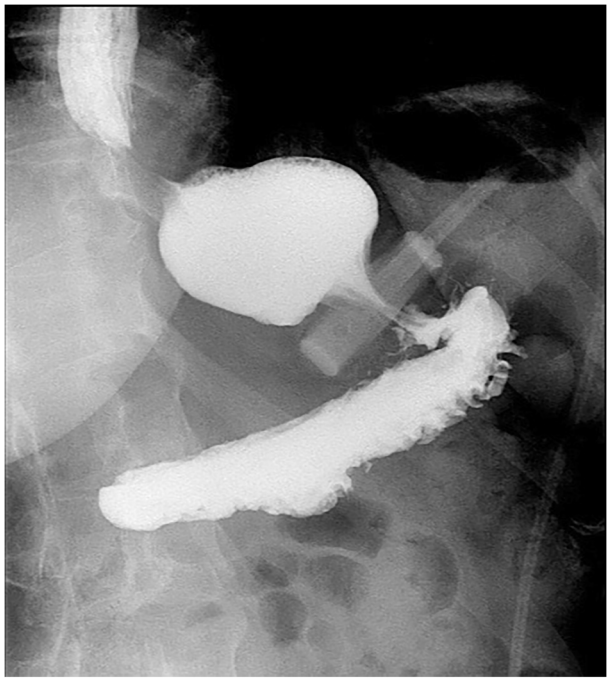
*[1]Icahn School of Medicine at Mount Sinai Morningside-West, New York, NY; [2]Mount Sinai Beth Israel, New York, NY; [3]Mount Sinai, New York, NY.*

**Introduction:** Gastric band slippage is a late complication of laparoscopic adjustable gastric banding surgery (LAGBS) with an incidence of less than 5%. We present a case of pseudoachalasia caused by gastric band slippage in a patient with a history of LAGBS.
**Case Description/Methods:** A 53-year-old female with a history of LAGBS for obesity 10 years ago presented to our motility clinic with complaints of recurrent emesis for the last 10 years. Episodes were associated with minimal retching, often self-induced to relieve chest discomfort. She appeared tearful while explaining the debility from her symptoms, severely affecting her quality of life. Due to severe, progressive worsening of symptoms for 3 months prior to presentation, a gastric emptying study performed by her gastroenterologist did not reveal any pathology. A recent esophagogastroduodenoscopy showed mild esophagitis. Proton pump inhibitors provided no relief. Given the incongruence between symptoms and workup, we decided to obtain an upper gastrointestinal (UGI) series. UGI series revealed tilting of the laparoscopic band, collimation around the thoracic esophagus immediately on barium swallow followed by passage of short amount of contrast along the narrowed diameter at the location of the band. Over the course of the study, significant stasis was noted in the thoracic esophagus causing severe restriction of passage confirming the diagnosis of gastric band slippage. She was referred to bariatric surgery for adjustment of the gastric band (**Figure**).
**Discussion:** The complication rate from LAGBS increases by 3-4% each year that the band is left in-vivo and rises to 40% at 10 years. Complications such as slippage and obstruction at the band site present with nonspecific symptoms such as vomiting and regurgitation. In our patient's case, pseudoachalasia was one of the differential diagnoses given the long-standing dysphagia, regurgitation, and chest discomfort. Although majority of pseudoachalasia is cancer related, benign causes also exist. LAGB can lead to formation of scar tissue from tight fundic band can also cause pseudoachalasia by creating persistent high-pressure and low flow resistance, leading to impaired LES relaxation, weakening of the esophageal body. This case highlights the importance of primary care providers and gastroenterologists familiarizing themselves with presentation, diagnosis, and management of late complications of bariatric procedures as delayed diagnosis can severely affect quality of life as demonstrated by our case.



[2392] **Figure 1.** UGI series demonstrating tilting of the laparoscopic band, collimation around the thoracic esophagus immediately on barium swallow along the narrowed diameter at the location of the band.

**S2393**

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**The Black Esophagus: A Rare Complication of Diabetic Ketoacidosis**

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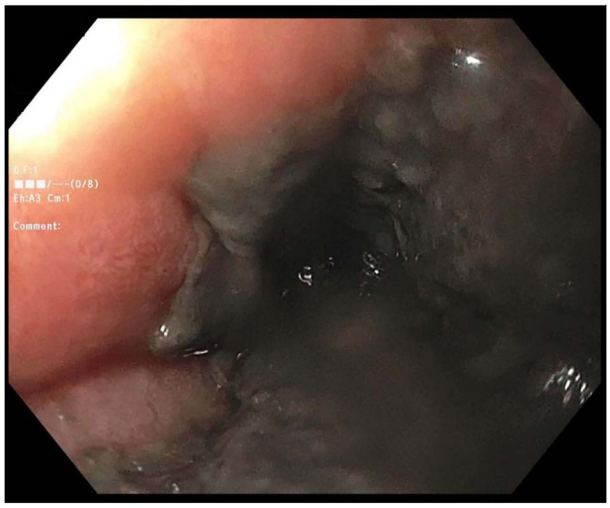
*University/Kent Hospital, Providence, RI.*

**Introduction:** Acute esophageal necrosis (AEN), which is also known as “black esophagus,” “acute necrotizing esophagitis,” or “Gurvits syndrome,” is a very rare condition with uncertain etiology and was first described in 1990. Its prevalence is less than 0.2%, the specific mortality is 6%, while the overall mortality rate due to comorbidities can go up to 32%. The patient is usually a male, old (average age of 67 years), and has chronic comorbidities, including cardiovascular disorders, diabetes mellitus (DM), malignancies, malnutrition, cirrhosis, gastric outlet obstruction, alcohol abuse, chronic respiratory disease, hypercoagulable state, and renal disease.
**Case Description/Methods:** Our patient is a 58-year-old gentleman with a past medical history of uncontrolled DM, untreated hepatitis C, and EtOH abuse disorder who was admitted to a hospital for generalized weakness and was found to have DKA. He was treated in MICU with subsequent closure of his anion gap. Later, he developed a cold foot concerning for ischemia. Computed tomography (CT) demonstrated left common femoral and popliteal artery thrombosis. He was started on heparin and underwent 4-compartment fasciotomy, popliteal arteriotomy, and subsequent below-knee amputation. Later, he developed melena and hematemesis and became hemodynamically unstable with an Hgb drop down to 5.8. He had a cardiac arrest, underwent cardiopulmonary resuscitation, was intubated, and achieved a return of spontaneous circulation (ROSC). After stabilization, He underwent an EGD which showed a black esophagus consistent with the severely necrotic esophagus. He underwent bronchoscopy on the same day with no signs of tracheoesophageal fistula but was started on Micafungin due to the high risk of esophageal perforation. Sedation was weaned but remained unresponsive or able to follow commands. He was taken for a pan-scan due to worsening shock and abdominal distention, but while in the scanner, he aspirated and died (**Figure**).
**Discussion:** Researchers have proposed a "2-hit" hypothesis for AEN while the initial event is usually an ischemic state on top of an altered mucosal barrier system in a malnourished and debilitated patient. It has also been linked to backflow injury from the chemical contents of gastric secretions and thromboembolic events. Early detection is vital to preventing life-threatening complications of AEN, such as esophageal perforation and mediastinitis.



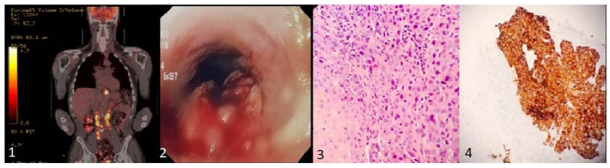
[2393] **Figure 1.** Acute esophageal necrosis.

**S2394**

**The Conundrum of a Metastatic Retroperitoneal Lymph Node: Where Is the Primary Tumor?**

Sneha Adidam, MD[1], Naga Sai Shravan Turaga, MD[2], Anish Konde, MD[3].
[1]Howard University Hospital, Washington, DC; [2]University of Arkansas for Medical Sciences, Little Rock, AR; [3]Providence Regional Cancer System, Lacey, WA.

**Introduction:** Cancer of Unknown Primary (CUP) is defined as biopsy proven cancer thought to be metastatic in nature, without an identifiable primary site of origin, despite a thorough history and physical examination and standard imaging. We present an unusual case of squamous cell carcinoma (SCC) with unknown primary involving a retroperitoneal lymph node.
**Case Description/Methods:** A 55-year-old man with a history of hypertension, diabetes and left cerebral ischemic stroke presented with complaints of diffuse abdominal pain and constipation for 2 days. He had a weight loss of 30-40 pounds in 10 months. Initial vitals and labs were normal. Physical exam was significant for mild diffuse abdominal tenderness. Abdominal Computerized Tomography (CT) revealed diffuse retroperitoneal lymphadenopathy with a 1.5 cm celiac axis node with central attenuation suggestive of necrosis, a 2cm left periaortic lymph node. CT guided fine needle aspiration and core biopsy of right retrocaval lymph node was performed and histology revealed medium to large pleomorphic sheets of cells with surrounding large necrotic regions suggestive of moderate to poorly differentiated SCC. Immunohistochemical staining showed positive staining for p63 and CK 5/6 positive, negative staining for TTF1 and RCC. Skull base-mid thigh positron emission tomography (PET) CT revealed an area of hypermetabolic activity involving distal esophagus with standardized uptake value (SUV) of 6.3 and extensive retroperitoneal adenopathy with SUV of 4.5. Subsequently, given the high suspicion for esophageal primary, upper gastrointestinal endoscopy was done which showed a 38cm mass concerning for malignancy. Histological examination revealed moderately differentiated squamous cell carcinoma. He was started on chemoradiation for esophageal cancer (**Figure**).
**Discussion:** National Comprehensive Cancer Network guidelines discuss about workup for unknown primary with metastatic adenocarcinoma in retroperitoneal lymph nodes with but there is no description about workup for metastatic SCC. Furthermore, distal esophageal cancers are usually adenomatous in origin and metastasize to cervical lymph nodes. Hence, a thorough workup including PET-CT scan should be done in cases metastatic SCC involving retroperitoneal lymph nodes and distal esophageal SCC should be considered a differential in such cases.




[2394] **Figure 1.** Image 1. shows skull base-mid thigh Positron Emission Technology CT scan which revealed an area of hypermetabolic activity involving distal esophagus with standard uptake value of 6.3. There was also extensive adenopathy peripancreatic, aortocaval space as well as periaortic space with FDG avid with SUV value of 4.5. Image 2. shows 38 cm mass in the mid-lower esophagus concerning for malignancy. Image 3. shows squamous cell carcinoma, invasive nests of squamous epithelial cells with moderate cytologic atypia. Image 4. shows immunostaining for cytokeratin shows diffuse positive staining supportive of squamous cell origin.

**S2395**

**Weight Goes Up, Esophagus Pops: A Case of Boerhaave's Syndrome in a Weightlifter Complicated by Empyema and Shock That Was Managed by Esophageal Endoluminal Vacuum-Assisted Therapy**

Amit Sah, MD[1], Lesley-Ann G. McCook, MD[2], Tiffany Chomko, MD[1], Akiva Marcus, MD[3], Kayode Olowe, MD[3].
[1]University of Miami/JFK Medical Center, Atlantis, FL; [2]University of Miami/ JFK Medical Center Palm Beach Regional GME Consortium, Atlantis, FL; [3]JFK Medical Center, Atlantis, FL.

**Introduction:** Boerhaave's Syndrome (BS) is a transmural rupture of esophagus from sudden increase in intraluminal pressure. It classically presents with vomiting, chest pain and subcutaneous emphysema, which is not always present. It is potentially lethal if not diagnosed in a timely manner.
**Case Description/Methods:** A 25 years-old-man was transferred with a concern for esophageal rupture from an outside facility where he presented after loss of consciousness in the gym associated with dyspnea. CXR revealed tension pneumothorax relieved by chest tube placement. Given further deterioration in clinical status with empyema and new complain of epigastric pain, CT scan was performed that revealed possible fistula vs esophageal tear. He was then transferred to us for management of esophageal rupture complicated by empyema. Barium esophagram revealed esophageal perforation at left posterior margin proximal to GE junction. He underwent left thoracotomy with closure of perforation with pleural flap. Repeat esophagram was negative for leak. His post operative course was complicated by hemorrhagic shock and he had an urgent EGD that was limited due to hematoma. Exploratory laparotomy with oversewing of the mucosal ulceration were done urgently. Due to poor esophageal healing and continued acute blood loss anemia requiring multiple transfusion, an esophageal endoluminal vacuum (Endo-Vac) was placed. After multiple Endo-Vac exchanges, the perforation started to heal. After almost 3 months, he survived this complicated case of BS and was discharged home.
**Discussion:** BS management ranges from conservative to endoscopic to surgical interventions. Its nonspecific symptoms could delay diagnosis leading to mortality as high as 40%. Our patient diagnosis was delayed due to frame bias of pneumothorax leading to a complicated course. Our case highlights the importance of multidisciplinary management that involved surgery, gastroenterology, infectious disease, ICU and radiology. It shines light on the Endo-Vac therapy which was first reported in 2008 for anastomotic leak management. It has since been used for multiple GI pathologies. It is minimally invasive and has reported success rate as high as 80-90%. It is one of the promising intervention for management of esophageal perforation and fistula. Research with large number of patients is lacking to evaluate the efficacy and success of this promising alternative therapy. Our case highlights its successful role in management of a complicated esophageal rupture case.

S2396

**Uphill and Downhill Esophageal Varices Secondary to Pulmonary Hypertension**

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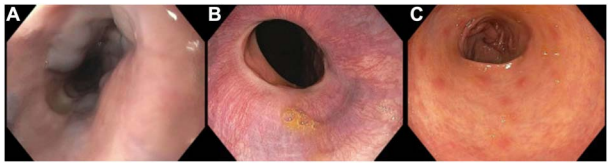
**Introduction:** Proximal downhill esophageal varices (EV) are rare, accounting for 0.5% of EV. They develop from superior vena cava (SVC) obstruction and retrograde blood flow through upper and middle peri-esophageal collaterals. Historically, downhill EV are associated with malignancy, thrombosis, or iatrogenic causes. Few cases of benign obstruction, as in pulmonary hypertension (PH) with tricuspid regurgitation (TR), have been reported.
**Case Description/Methods:** A 67-year-old female with PH and atrial flutter presented with 3-weeks of melena. She denied vomiting, hematochezia, or history of malignancy. Her warfarin dose was stable, and she reported decreased vegetable intake for months. She endorsed chronic alcohol use but no NSAID or tobacco use. She was hemodynamically stable on admission. Physical exam revealed a LLSB holosystolic murmur. Notable labs included hgb 7.5g/dL, iron deficiency and INR 11.3. She received IV vitamin K, IV PPI BID and warfarin was held. EGD revealed large proximal EV, grade 1 distal EV and nodular gastric antral vascular ectasia, but no bleeding. CT chest and abdomen ruled out malignancy or SVC obstruction but revealed cirrhosis and portal hypertension. TTE showed severe TR and PH. She had no further episodes of melena. She was discharged with a MELD-Na of 24 and plans for liver biopsy and right heart catheterization for further workup (**Figure**).
**Discussion:** Of the few cases of downhill EV due to PH, both TR and PH were present, allowing high filling pressure and SVC congestion. Hemorrhage is infrequent, accounting for 0.1% of EV bleeds, likely from low gastric acid exposure, submucosal location, and lack of coagulopathy in cirrhosis. Studies found higher rates of bleeding in benign obstruction with only 14% related to malignancy. Anticoagulant and antiplatelet agents increase risk. Treatment focuses on correcting the underlying cause. Endoscopic band ligation or sclerotherapy are reserved for emergent bleeding due to risk of esophageal perforation, stricture, or infarction. Studies of repeat EGD 5 years post diagnosis revealed stable size providing evidence against routine surveillance. Our patient's melena was likely from gastric mucosal oozing due to supratherapeutic INR and vitamin K deficiency. In summary, downhill EV are often incidental and associated with severe comorbidities. Further workup is crucial for ruling out malignancy and preventing hemorrhage. Additional cases are needed to evaluate the safety and efficacy of endoscopic intervention and utility of surveillance screening.




[WC] **Figure 1.** EGD revealing both proximal "downhill" and distal "uphill" esophageal varices (EV) and nodular gastric antral vascular ectasia (GAVE). (A) Prominent proximal EV with no stigmata of recent bleeding. (B) Non-bleeding, grade 1 distal EV. (C) Nodular GAVE located in the antrum of the stomach.

S2397

**When Chronic Medical Conditions Mask Acute Symptoms: A Case of Esophageal Perforation With Neck Pain**

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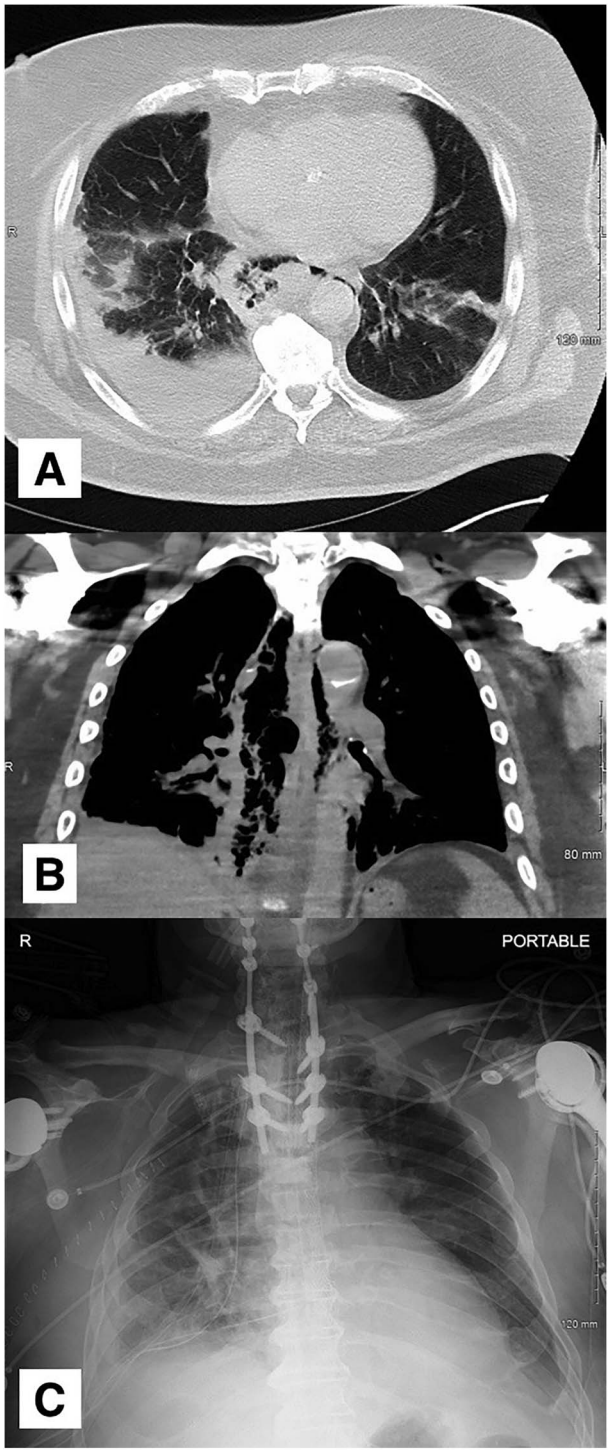
**Introduction:** Esophageal perforation is a rare and potentially life threatening condition, with mortality greater than 20%. Classic symptoms include chest pain, vomiting, and subcutaneous emphysema, known as the Mackler triad. However, patients can present with a variety of symptoms including neck pain, epigastric pain, hoarse voice, dysphagia, tachycardia, and dyspnea. Early clinical suspicion, diagnosis, and treatment are necessary to decrease chances of a poor outcome. Here, we present a patient whose symptoms were masked by his chronic medical conditions.
**Case Description/Methods:** This is an 81 year old White male with a past medical history of cervical and upper thoracic spinal fusion secondary to advanced arthritis who presented to the emergency department after a low velocity motor vehicle collision (MCV). His only complaint at that time was neck pain, which he describes as chronic, for which he takes daily opioid medication. He became frustrated and left the emergency department against medical advice after initial chest x-ray was obtained, which did not reveal acute findings. He represented to the emergency department after 48 hours, stating that he had run out of his opioid medications and is having continued neck pain. He described episodes of coughing while eating and drinking at home. He was found to have an oxygen saturation of 98% on room air. Knowing that the patient was recently in an MCV, a CT chest without contrast was ordered, demonstrating extensive pneumomediastinum centered around a thick walled esophagus, raising concern for esophageal perforation [Figure A, B]. A Gastrografin esophagram was then completed, which revealed visualization of extraluminal contrast. The patient was initiated on broad spectrum antibiotics with vancomycin and piperacillin-tazobactam. He then underwent thoracotomy with washout, decortication, and esophageal repair [Figure C]. Unfortunately, the patient had a complicated 60 day hospital stay, where he had to undergo multiple surgical procedures for complications related to the esophageal perforation. He was ultimately discharged to an inpatient skilled nursing and rehabilitation facility, where he stayed for an additional 60 days. At the time of discharge, the patient was fully independent for most activities of daily living and was able to ambulate without assistive devices.
**Discussion:** This case demonstrates the importance of maintaining a high index of suspicion for esophageal perforation in patients involved in trauma, even in the absence of classical symptoms.




[2397] **Figure 1.** A) Transverse slice of CT chest demonstrating esophageal perforation with right sided pleural effusion. B) Coronal slice of CT chest demonstrating esophageal perforation. C) Portable chest x-ray demonstrating post-operative changes, with esophageal stent in place.

**S2398**

**Under Pressure: An Uncommon Case of Extraluminal Esophageal Air**

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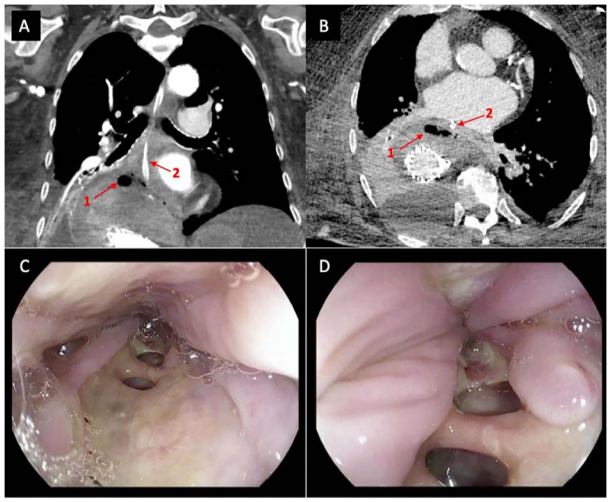
**Introduction:** The presence of air adjacent to the esophagus on imaging can be an emergent finding warranting surgical or endoscopic evaluation, and the etiology can be esophageal, pulmonary, or vascular in nature. We report a case of an aortoesophageal fistula (AEF) in a critically ill patient after a recent thoracoabdominal endovascular aortic aneurysm repair (TEVAR).
**Case Description/Methods:** A 70-year-old woman with hypertension and atrial fibrillation was in intensive care after a recent complicated TEVAR performed for aortic rupture. She was noted to have tachycardia and hypotension; she denied any nausea, melena, or hematochezia. Vital signs revealed a heart rate of 123 beats per minute and a blood pressure of 85/51 mm Hg. Physical exam showed a critically-ill

appearing woman in no distress, a clear oropharynx, a non-tender abdomen, and a rectal exam without blood. Labs noted a leukocytosis of 19.0 x$10^9$/L, a Hgb of 6.8 g/dL, and a lactate of 2.1 mmol/L. Labs drawn 12 hours prior noted a Hgb of 8.2 g/dL and a lactate of 0.9 mmol/L. CT imaging of the chest (**Figure** A-B) showed interval air in the excluded thoracic aneurysmal sac adjacent to the esophagus (arrow 1), which was intubated by an enteric tube (arrow 2). EGD (**Figure** C-D) revealed 2 large non-bleeding perforations in the middle esophagus with possible fistulation to the mediastinum. The clinical presentation suggested post-TEVAR AEF, likely due to compressive ischemia and necrosis of the esophagus from the aneurysmal sac compounded by prolonged shock. Given high operative risk, an esophageal stent and gastrostomy tube were placed by cardiothoracic surgery.

**Discussion:** Post-TEVAR AEF formation is a rare but serious complication occurring in 1.7–1.9% of patients after TEVAR, and can be due to graft infection, aortic pressure necrosis, or esophageal erosion related to aortic expansion. Chiari's triad, the hallmark symptoms of AEF, include mid-thoracic pain and sentinel hematemesis followed by massive hematemesis. However, massive hemorrhage may be observed soon after a sentinel episode of hematemesis or can be delayed by years. Imaging findings include air within the aortic thrombus or esophageal wall, expanding fluid collection around the graft, or extraluminal aortic contrast extravasation. Endoscopic findings include esophageal submucosal protrusions, ulcerative lesions, or fistula formation. In stabilized patients, management options include subtotal esophagectomy and aortic debridement and reconstruction.




[2398] **Figure 1.** Endoscopic and Imaging Investigations for Aortoesophageal Fistula. Panels A-B. Coronal and axial, respectively, CT images of the chest with contrast. The development of air was noted in the excluded aortic aneurysmal sac adjacent to the esophagus after TEVAR (arrow 1); the esophagus is intubated by an enteric tube (arrow 2). Panels C-D. EGD revealed 2 large non-bleeding perforations in the middle esophagus with surrounding ulceration and possible fistulation to the mediastinum.

**S2399**

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**When All You See Is Black: A Case of Acute Esophageal Necrosis**

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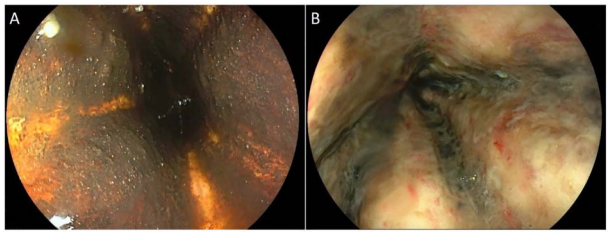
**Introduction:** Acute esophageal necrosis (AEN), sometimes called "black esophagus" given its endoscopic appearance, is a rare condition with an incidence of around 0.01% of all patients undergoing EGD. The pathophysiology of AEN is hypothesized to be multifactorial involving hemodynamic compromise, backflow of stomach acid, any state where esophageal mucosal repair mechanisms are diminished, or thromboembolic phenomena. The primary associated symptoms are hematemesis, melena, and shock. AEN is a life-threatening condition with a mortality rate of approximately 32%.

**Case Description/Methods:** A 73-year-old male with a medical history of NASH cirrhosis, hypertension, coronary artery disease, abdominal aortic aneurysm, and diabetes mellitus type 2 presented from home with hematemesis. He was discharged 24 hours before presentation after spending 5 days in the hospital for hepatic encephalopathy. On physical examination he was afebrile, normotensive with a slight tachycardia. Hemoglobin and hematocrit were 11g/dL and 31%, respectively. He was started on an octreotide drip, given IV Protonix 80 mg, and 1 gram of Ceftriaxone. EGD later that day revealed black material coating the esophagus unable to be washed away, portal hypertensive gastropathy, a large amount of altered blood mixed with food in the stomach, duodenitis, and superficial duodenal ulcers. A repeat EGD 24 hours later revealed ulceration of the mid and distal esophagus consistent with acute esophageal necrosis. A CTA aorta was negative for stenosis or thromboembolic phenomena. He remained hemodynamically stable, did not develop esophageal pain, and was started on a Sucralfate slurry. He was able to progress his diet quickly. Six days after admission his labs normalized and was discharged home (**Figure**).

**Discussion:** Our patient had multiple factors likely leading to the development of AEN due to his numerous comorbidities. It is important to monitor for complications of AEN, including perforation and microbial superinfection during the initial recovery period, and stenosis or strictures as a late sequala. Due to the patient's prompt triage and intervention with appropriate therapy, a good outcome was achieved. This case highlights the importance of quickly triaging patients and intervening appropriately to combat the high mortality rate of AEN.




[2399] **Figure 1.** 1: Endoscopic appearance of the esophagus on day of presentation (A) showing black necrotic appearance of esophagus and 24 hours later on repeat examination (B) demonstrating ulcerated esophageal mucosa.

**S2400**

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**Granular Cell Tumor: A Rare Disease of Esophagus**

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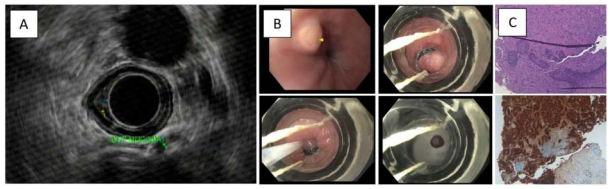
**Introduction:** Granular cell tumors (GCTs) are rare, often benign tumors of neurogenic origin. GCTs are usually found incidentally but can present with dysphagia in symptomatic patients. They are diagnosed via biopsy and histology following an esophagogastroduodenoscopy( EGD). We present a case of a 46-year-old female who presented with dysphagia, GERD, and weight loss diagnosed with esophageal granular cell tumor following an EGD.
**Case Description/Methods:** A 46 years old female with a past medical history of H. pylori gastritis presents with complaints of dysphagia for 3 months and GERD associated with weight loss. EGD reviewed a single 4 mm nodule found in the lower third of the esophagus with the appearance of a lipoma (**Figure**). Biopsy of esophagus nodule resulted in esophageal squamous epithelium with focal granular cell tumor; Immunohistochemistry tumor cells are positive for S100, CD56, CD68, negative for calretinin and AE1/3 supporting rare esophageal granular cell tumor. EUS demonstrated a 4.7 mm x 2.6 mm intramural (subepithelial) lesion appeared to originate from within the superficial mucosa (Layer 1), Patient underwent repeat EGD with en-bloc cap and band-assisted endoscopic mucosal resection (EMR) followed by 3 hemoclips placed for hemostasis. The patient was discharged home on a proton pump inhibitor. The patient reports clinical improvement on follow-up after 4 weeks.
**Discussion:** GCTs are thought to originate from Schwann cells; the prevalence of GCTs is challenging to determine as there have been roughly 300 documented cases of GCTs discussed in the literature. Conversely, patients with larger diameter lesions typically present with indolent dysphagia and other less common symptoms such as gastro-esophageal reflux disease, dyspepsia, chest pain, cough, or nausea. GCTs on EGD appear sessile, yellow-white in color, and firm, with a negative pillow sign. After performing EGD, an endoscopic ultrasound (EUS) is diagnostically utilized to ascertain tumor invasion and provide a biopsy through fine needle aspiration (FNA). Histopathological diagnosis, including immunohistological staining, is essential for confirmation of GCT. Immunohistological staining is positive for S-100, PAS (Periodic Acid Schiff), neuron-specific enolase, nestin, and vimentin. Patients whose neoplasms are less than 1 cm in diameter are treated conservatively with endoscopic follow-up, with tumors greater than 1 cm in diameter, or having clinical symptoms that need endoscopic or surgical resection.




[2400] **Figure 1.** A: EUS showing 4.7 mm x 2.6 mm hypoechoic intramural (subepithelial) lesion was found in the lower third of the esophagus. It appeared to originate from within the superficial mucosa (Layer 1). A tissue diagnosis was obtained prior to this exam. B: EGD significant for sessile, yellow white in color, and firm mass followed by EMR. C: Histopathology with H&E showing clusters of polygonal cells with granular eosinophilic cytoplasm (top) and S100 stain (below).

**S2401**

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**A Rare Case of Lichenoid Esophagitis Causing Refractory GERD**

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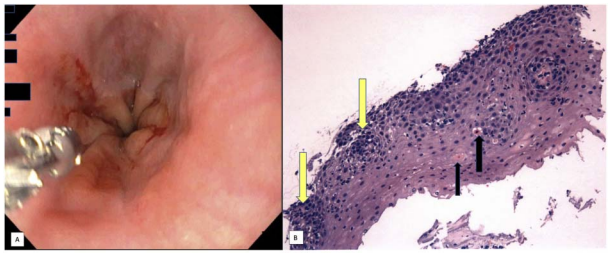
**Introduction:** Lichenoid esophagitis (LE) refers to a rarely seen lichenoid pattern of inflammation in the esophagus. We report a unique case of LE in a patient with refractory gastroesophageal reflux disease (GERD) in the absence of any known risk factors that have been associated with LE.
**Case Description/Methods:** A 74-year-old female with history of type 2 diabetes mellitus, hypertension, and GERD presented for evaluation of persistent reflux symptoms despite being on appropriate therapy with a proton pump inhibitor (PPI) for several months. Patient underwent upper endoscopy for further evaluation which showed LA grade A esophagitis without any evidence of bleeding. Esophageal biopsy showed lichenoid esophagitis pattern without any dysplasia or neoplasia. There was no evidence of CMV, HSV, or fungal infections. Patient did not have any history of cutaneous lichen planus or any rheumatological disorder. She tested negative for HIV and viral hepatitis. Her PPI dose was increased and a repeat endoscopy was scheduled in 6 months for surveillance (**Figure**).
**Discussion:** LE involves esophageal mucosal infiltration with inflammatory lymphocytes and dyskeratotic epithelial cells. It can be seen in medication-induced injury (polypharmacy), rheumatological disorders, esophageal involvement by lichen planus, viral hepatitis and HIV infections. It typically presents with esophageal strictures and dysphagia. However, it is rarely associated with refractory GERD as seen in our patient. It is prudent to recognize this histopathological entity as it has significant risk of further progression to dysplasia and neoplasia. Treatment of the underlying cause, if known may help in clinical resolution. While there are no defined guidelines currently in place, surveillance endoscopy can be valuable in the prevention of future clinical complications.




[2401] **Figure 1.** 1A: Lower third of the esophagus during endoscopy showing esophagitis without any evidence of bleeding. Biopsy specimen taken and described in next image. 1B: Esophageal biopsy: squamous mucosa with a predominantly basal lymphocytic inflammatory infiltrate (yellow arrows) and associated scattered dyskeratotic epithelial cells (black arrows). No intraepithelial eosinophils are seen, no acute neutrophil-mediated inflammation is identified. No fungal organisms identified, immunohistochemistry for CMV and HSV negative. No intestinal metaplasia seen.

**S2402**

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**A Case of Heterotropic Pancreatic Tissue at the Gastroesophageal Junction**

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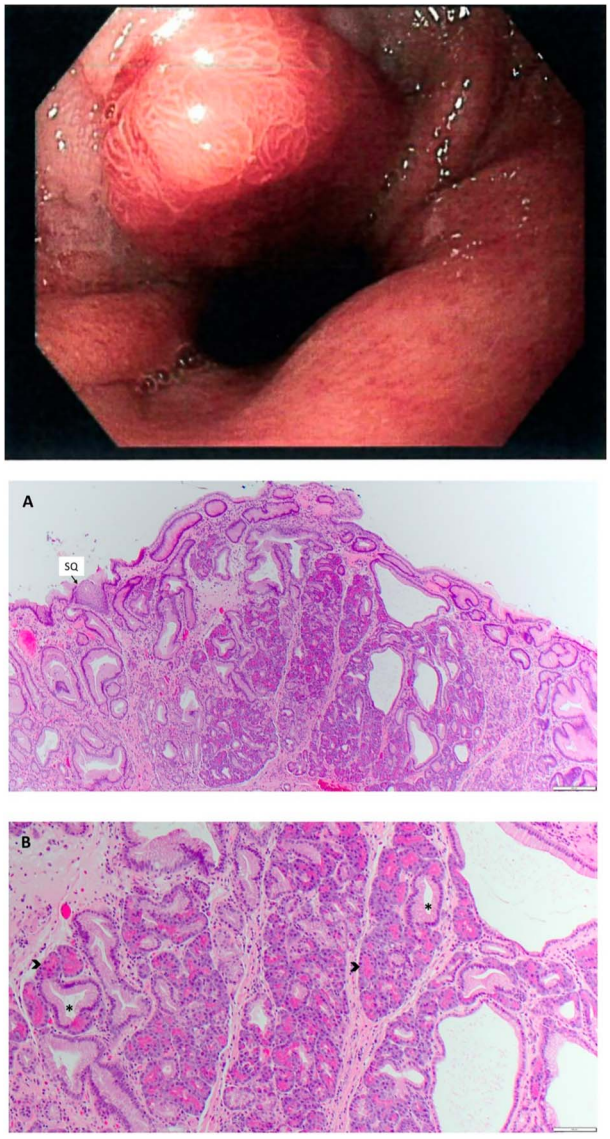
*UCLA, Los Angeles, CA.*

**Introduction:** We report a rare case of heterotropic pancreas located at the GE junction which was incidentally discovered during esophagogastroduodenoscopy (EGD). Heterotropic pancreas (HP) (pancreatic rest or ectopic pancreas) is a congenital anomaly in which there is ectopic pancreatic tissue indicating that it is separate from the main pancreatic gland, and does not have any continuity (anatomic, vascular, or ductal) with the main pancreatic gland. HP is generally found in the stomach, duodenum, or proximal jejunum. The prevalence of HP is believed to be 0.55 to 13.7% on autopsy. It is uncommon to find HP in the esophagus with nineteen cases being reported in literature it is rare to find HP at the GE junction with only 6 cases being reported in literature.
**Case Description/Methods:** A 45-year-old male presented for chronic heartburn. On EGD, distal to the gastroesophageal (GE) junction there was a 2 cm nodule that was biopsied and removed by a hot snare. In the gastric antrum, there was gastritis characterized by erythema. The duodenum appeared normal. Pathology from the GE junction nodule was consistent with heterotropic pancreatic tissue. The gastric biopsies were consistent with chronic active gastritis (**Figure**).
**Discussion:** Pancreatic rest is a submucosal nodule where pancreatic tissue is developed and commonly found in the upper small intestine or stomach. The pathogenesis for HP is not known. Nearly all pancreatic rests have no symptoms. It can be incidentally found through surgery or endoscopy. HP may still function in a similar manner as the main pancreatic gland and may secrete enzyme rich serous fluid, proteolytic enzymes causing a local inflammatory response, in which case there may be symptoms as a result. When present at the GE junction, symptoms that have been reported in literature include dysphagia due to mass effect, symptoms of heartburn, or epigastric pain. The endoscopic appears is usually a well-circumscribed submucosal lesion commonly with central "umbilication" covered by normal mucosa. Surface biopsies may be non-diagnostic due to sampling of overlying mucosa. The final diagnosis is based on histology of the endoscopically or surgically resected specimen. Endoscopic ultrasound with fine needle aspiration of the lesion may also be utilized for diagnosis. The symptoms of heartburn present in our patient may have been contributed to by the incidentally found HP.




[2402] **Figure 1.** Gross Image: EGD showing GE junction with 2-cm nodule. Pathology Image A: Pancreatic heterotopia/metaplasia is the lobular structure within the lamina propria; a small focus of esophageal squamous epithelium (SQ) is seen (hematoxylin and eosin stain, (x40). Pathology Image B: Pancreatic acinar cells (arrowhead) have deeply eosinophilic granules and ductal cells have pale cytoplasm (x100).

**S2403**

**A Rare Case of Lymphocytic Esophagitis Presenting With Food Impaction**

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**Introduction:** Lymphocytic esophagitis (LyE) was first described in the literature in 2006 and remains a very rare diagnosis, with diagnostic criteria being met in only 0.1% of esophageal biopsies. LyE shares features with GERD and eosinophilic esophagitis and is often misdiagnosed. It is most common in middle-aged women and presents with very nonspecific symptoms, most commonly dysphagia. Esophageal biopsy is the gold standard for diagnosis and LyE is currently defined solely on histological criteria. Treatment options are limited, with proton-pump inhibitors (PPIs) being the first line treatment.
**Case Description/Methods:** A 71-year-old male with a past medical history of hypertension and tobacco and alcohol use disorder presented after experiencing an episode of acute dysphagia to solids and liquids. Urgent EGD was performed, which showed a large food bolus at the distal esophagus/gastroesophageal junction. This was fragmented and pushed distally into the stomach by an endoscopic retrieval device. Several ulcers were present in the lower esophagus and biopsies taken showed mild erosive gastropathy and duodenitis. Repeat EGD was performed 8 weeks later, and a distal esophageal stricture was noted and was dilated with a balloon dilator to 15mm. Biopsies taken of the proximal and distal esophagus showed squamous mucosa with >20 intraepithelial lymphocytes per high-power field with rare neutrophils and rare eosinophils with associated patchy, peripapillary lymphocytic infiltrates, consistent with LyE. Patient was placed on a proton-pump inhibitor twice daily with reported improvement of symptoms and resolution of dysphagia at follow-up.
**Discussion:** Lymphocytic esophagitis (LyE) is a very rare cause of esophagitis and is often misdiagnosed due to its variable symptoms and nonspecific gross appearance on endoscopy. Diagnosis can only be made with biopsy, specifically the presence of >20 intraepithelial lymphocytes per high-power field. Food impaction is a rare presentation of LyE and has only been shown in around 9% of cases. Although the pathogenesis remains unknown, inflammation has been associated with the development of this condition. LyE was diagnosed in this male patient after endoscopic removal of food impaction, resulting in stricture. This could support the theory that inflammation due esophageal trauma/injury could be associated with the development of LyE. This diagnosis is important to keep on the differential for causes of esophagitis.

S2404

**Managing Complications Following Endoscopic Myotomy as a Treatment for Upper Esophageal Sphincter Achalasia: A Case Report**

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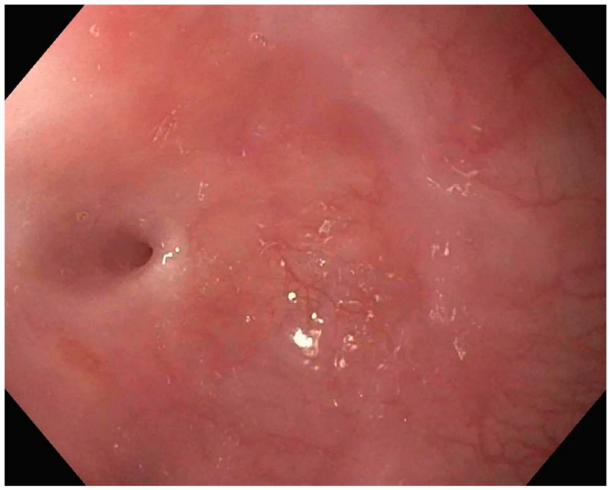
**Introduction:** Achalasia is a chronic gastrointestinal disorder characterized by increased esophageal sphincter tone that is initially asymptomatic but eventually causes dysphagia. While this condition usually affects the lower esophageal sphincter, we present a rare case of upper esophageal sphincter (UES) achalasia of unknown etiology in a female in her sixth decade of life. The patient underwent a UES endoscopic myotomy by otorhinolaryngology that was complicated by perforation and subsequent severe esophageal stenosis. Consequently, she was referred to an advanced endoscopy-trained gastroenterologist (GI) for further evaluation. She was treated over the course of 3 months with 6 endoscopic dilatations and glucocorticoid injections. Few cases of idiopathic UES achalasia have been described to date.
**Case Description/Methods:** Upon presentation, the patient was profoundly dysphagic to solids and liquids, noting a 50-pound weight loss in the past 2 months since the UES endoscopic myotomy. During the first esophagogastroduodenoscopy (EGD), the esophagus was found to be severely stenotic (15cm distal to the upper incisors at the level of the UES) reducing the lumen to 2mm in diameter at its narrowest point (**Figure**). Under fluoroscopic guidance, a guidewire was passed through the UES, and a 6mm balloon biliary dilator was inflated. A 24 French Savary dilator was then passed, successfully enlarging the lumen to 8mm without perforation. Due to the patient's poor nutrition status and necessity of additional dilations, she was referred to interventional radiology for percutaneous endogastric (PEG) tube placement. During subsequent EGDs, through-the-scope balloon and Savary dilators were used to increase the luminal diameter to a desirable size of 20mm. Local triamcinolone injections (2mL at 40mg/mL) were administered at the level of the UES during each EGD to reduce this risk of fibrosis and consequential stricture formation. After the sixth and final dilation, the patient was non-dysphagic, so the PEG tube was removed. She was instructed to resume her usual diet and contact GI as needed.
**Discussion:** This case of idiopathic UES achalasia represents an uncommon cause of severe dysphagia in an otherwise healthy adult without a history of stroke, radiation therapy, or a known congenital predisposition for pharyngoesophageal dysfunction. Complications following UES endoscopic myotomy must be diagnosed and treated urgently, as esophageal stenosis can increase the risk of aspiration and cause profound weight loss.



[2404] **Figure 1.** Anterograde view of the esophageal lumen proximal to the upper esophageal sphincter. Circumferential constriction at 15 cm distal to the upper incisors reduced the luminal diameter to 2 mm at the narrowest point. The image was taken with an esophagogastroduodenoscope during the patient's first visit to the advanced endoscopy gastroenterology clinic.

S2405

**Pseudoachalasia in Lung Cancer**

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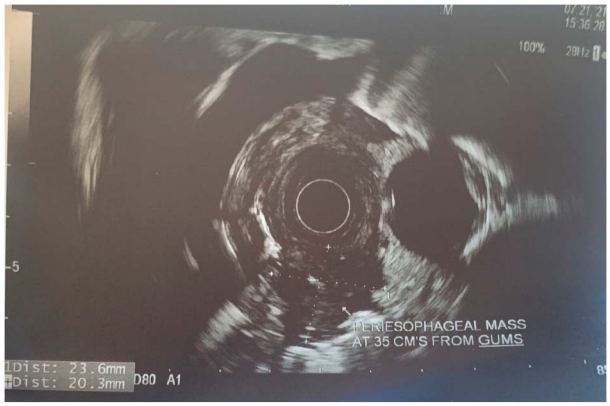
**Introduction:** Although not common, lung tumor-induced achalasia will occasionally be encountered by gastroenterologists in patients with dysphagia and/or achalasia. Clinical features suggesting the possibility of malignancy as a cause of achalasia include: abrupt development of the dysphagia, significant unintentional weight loss in a short period of time ( >15 pounds), age > 55 years; and extensive smoking history ( >20 years).
**Case Description/Methods:** A 67-year-old male with extensive smoking history presented to the hospital with complaints of difficulty tolerating foods and liquids with intermittent nausea and vomiting. Patient has to cut food into pieces to be able to go down. At presentation he had lost 25lbs in 2 weeks. CT abdomen showed abnormality at the gastroesophageal junction. EGD which showed a severely ulcerated esophagus with a stenotic mass effect in the distal third of the esophagus preventing the scope from advancing into the stomach (**Figure**).
**Discussion:** Differentiating between idiopathic achalasia and pseudoachalasia is difficult and is often only possible when there is a diagnosis of other illnesses observed to have caused patients to experience achalasia symptoms with malignancy accounting for about 5% of these cases (Campo, 2013). Esophageal and gastric cancers tend to be the most common malignancies that cause pseudoachalasia, accounting for up to 70% of cases (Gockel et al., 2005). It is quite rare to come across a case of lung cancer causing pseudoachalasia and for that reason very little literature exists about the association between the 2 and its clinical expressions. In this particular case the ability for food to go down albeit in extreme difficulty indicated that there was some peristalsis which is seen often in pseudoachalasia but not in idiopathic achalasia (Kim et al., 2015). Another factor which helped in our diagnosis was the inability of the scope to advance past the mass unless moderate pressure was applied. In achalasia patients, the scope should be able to advance past the gastroesophageal junction with no more than light pressure applied (Eckardt & Eckardt, 2009). Existing literature indicates that in many cases, the presentation of achalasia symptoms precedes the diagnosis of cancer in pseudoachalasia sufferers. Therefore, testing and retesting may need to be done to rule out malignancy in patients who present with achalasia symptoms who are over the age of 60 years, have a history of smoking, and have experienced rapid weight loss in a short period of time (Tucker, 1978).




[2405] **Figure 1.** EUS showing the periesophageal mass.

**S2406**

**A Case of Cervical Esophageal Adenocarcinoma Arising From Gastric Inlet Patch: A Benign Lesion With Malignant Potential**

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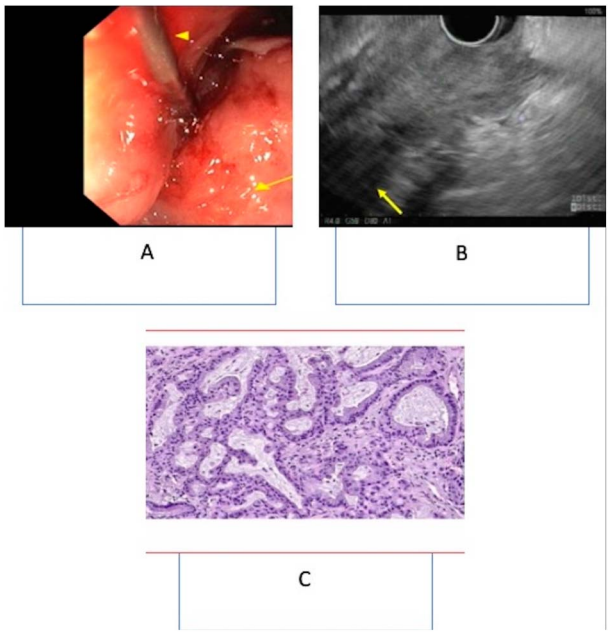
*[1]UTMB, Galveston, TX; [2]University of Texas Medical Branch, Galveston, TX.*

**Introduction:** Esophageal adenocarcinoma is typically localized to the distal third of the esophagus. It is associated with long-standing acid reflux and resultant Barrett's esophagus. In contrast, adenocarcinoma in the proximal esophagus without Barrett's metaplasia is extremely rare. A gastric inlet patch (GIP) is a lesion of ectopic gastric mucosa usually found in the cervical esophagus and is considered a benign lesion. However, albeit rare, malignant transformation of GIP can occur. Here, we present a case of cervical esophageal adenocarcinoma arising from gastric inlet patch.
**Case Description/Methods:** A 50-year-old male with GERD presented with a 6-month history of progressive dysphagia to solids and liquids and 20-pound weight loss. He reported a 1 pack-year smoking history but quit 30 years prior and consumed alcohol socially. Family history was negative for esophageal cancer. Upper endoscopy (HQ190F) showed a malignant stricture (2 cm long, 6mm diameter) at 18 cm from the incisors and a gastric inlet patch adjacent to the stricture. The endoscope was downsized (XP190N), and the stricture was dilated (24F) and traversed. The Z-line was at 40 cm with Barrett's esophagus extending from 36 cm to 40 cm. Pathology of biopsies from the stricture showed acute and chronic inflammation with increased intraepithelial eosinophils suggestive of acid reflux. CT scan of the neck with contrast showed a heterogeneously enhancing ill-defined mass involving the cervical esophagus below the cricoid cartilage indenting the posterior margin of the trachea. Bronchoscopy was negative for bronchogenic cancer. Endoscopic ultrasound showed a non-circumferential hypoechoic mass in the cervical esophagus 18 cm from the incisors extending to 20 cm. The mass was predominantly extrinsic but was also noted to have a luminal component and poorly defined endosonographic borders. Pathology of FNAB showed moderately differentiated adenocarcinoma (**Figure**).
**Discussion:** Proximal esophageal adenocarcinoma is extremely rare, making up less than 1% of esophageal cancers. Case studies have been published associating GIP and esophageal adenocarcinoma, but the pathogenesis of malignant transformation remains unclear. There are no established screening guidelines for ectopic gastric mucosa and routine biopsies are not recommended as dysplasia within GIP is rare. GIP may be overlooked during EGD and NBI exam may improve detection rates. Our case re-emphasizes careful examination of GIP and to strongly consider biopsy if abnormal.




[2406] **Figure 1.** A) Endoscopic view of malignant stricture 18 cm from incisors. Gastric inlet patch (arrow) adjacent to stricture. B) Endoscopic ultrasound image at the level of esophageal stricture showing irregular hypodensity suggestive of malignancy. C) Pathology of biopsy performed through fine needle aspiration (EUS) of upper esophageal mass demonstrating moderately differentiated adenocarcinoma, cribriform pattern, with mild nuclear pleomorphism, abundant mitoses, and apoptosis (H&E, 10x).

S2407

***Pylori*in the Patch**

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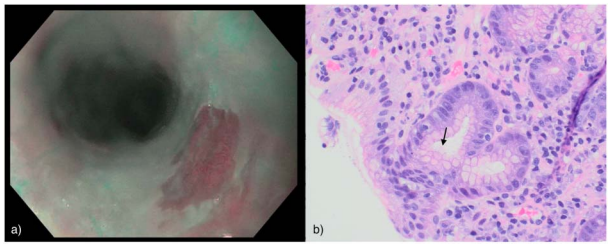
**Introduction:** *Helicobacter pylori* is the most common chronic bacterial infection found in humans. *H. pylori* is restricted in its ability to colonize only gastric mucosa; however, this can occur anywhere in the gastrointestinal tract where gastric mucosa is found. Heterotopic gastric mucosa of the upper esophagus (inlet patch) is thus a potential reservoir for *H. pylori*. We present a case of *H. pylori* infection in an inlet patch.
**Case Description/Methods:** A 44-year-old Hispanic male presented to the gastroenterology clinic for chronic dyspepsia. The patient endorsed a year-long history of uncontrolled acid reflux, nausea with vomiting, loss of appetite and diffuse abdominal pain. He reported being diagnosed with H. Pylori infection through a stool test performed 3 months prior to the visit. He completed treatment with amoxicillin, clarithromycin, and pantoprazole, but had not yet been tested for eradication. The patient underwent an esophagogastroduodenoscopy (EGD) that revealed 2 areas of esophageal inlet patch in the upper third of the esophagus suggestive of ectopic gastric mucosa, with associated nodularity. The biopsy from the esophageal lesion confirmed gastric heterotopia with chronic active gastritis and a positive hematoxylin and eosin (H&E) stain for *H. pylori*. Random gastric biopsies were also positive for *H. pylori* (**Figure**). The patient was treated with quadruple therapy (pantoprazole, bismuth, metronidazole, and tetracycline) for 14 days with subsequent improvement of his symptoms during follow up visit.
**Discussion:** Inlet patches are mostly found in the upper third of the esophagus, typically adjacent to the upper esophageal sphincter. Clinical symptoms of inlet patches can include chronic cough, heartburn, sore throat, globus sensation, dysphagia, and regurgitation. These symptoms likely occur due to the acid production in the heterotopic gastric mucosa. The incidence of H. Pylori in a patch has been found to closely correlate with the density of the bacteria in the stomach. However, H. Pylori colonization of heterotopic gastric mucosa is not seen in all cases of H. Pylori gastritis, which points towards reflux as the inciting event. Isolated H. Pylori infection in an inlet patch without concurrent H. Pylori gastritis and cases of primary adenocarcinoma arising from the heterotopic gastric mucosa have been reported. Biopsies of the inlet patch should be considered in all patients presenting with esophageal and extraesophageal symptoms, especially if it is associated with nodularity.




[2407] **Figure 1.** a) Endoscopy demonstrating esophageal inlet patch with increased nodularity b) Helicobacter Pylori organisms in lumen (H&E, 400x).

S2408

**A Case of Advanced Esophageal Carcinosarcoma**

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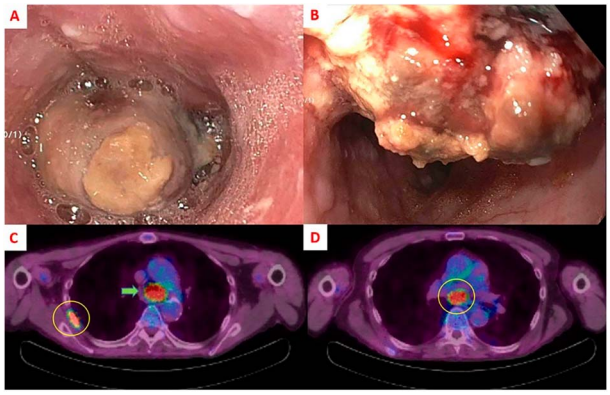
**Introduction:** The majority of esophageal cancers are classified histologically as squamous cell carcinomas or adenocarcinomas. We present a case of a 69-year-old man, presented with progressive dysphagia, found to have an esophageal carcinosarcoma, a rare etiology of esophageal cancer.
**Case Description/Methods:** A 69-year-old man, an everyday smoker with a history of ongoing alcohol use, presented for the evaluation of unintentional weight loss and progressive dysphagia of 2 months duration. Upper endoscopy revealed a large, 6 cm in diameter, partially obstructing ulcerating mass in the mid esophagus. PET/CT revealed intense FDG uptake in the esophageal mass, mediastinal lymph nodes, T6 and T10 ribs suggestive of metastatic disease. Surgical pathology of the tumor was consistent with a high grade squamous cell carcinoma mixed with a high grade spindle cell sarcoma (carcinosarcoma). The patient was started on chemoradiation including Paclitaxel, Carboplatin. Improvement of symptoms and CT findings was documented with treatment (**Figure**).
**Discussion:** Esophageal cancer ranks seventh in incidence and sixth in cancer-related mortality. The majority of esophageal cancers are classified histologically as squamous cell carcinomas or adenocarcinomas. When 2 distinct malignant components, both carcinomatous and sarcomatous are present histologically, the tumor is classified as esophageal carcinosarcoma (ECS). The pathogenesis is debatable, with some arguing that it is an initial carcinoma, facilitating a simultaneous secondary development of sarcoma, while others hypothesize a single cell origin. Carcinosarcomas were documented in tumors of the uterus, breast, thyroid, lung and gastrointestinal tract. ECSs have a rare incidence, accounting for approximately 1.5% of all esophageal tumors. It has been noted that ECS tumors have different clinical behaviors when compared to squamous cell carcinomas or adenocarcinomas. ECS tumors were noted to have a calculated doubling time of 2.2 months, with predominantly exogenous polypoid growth, perhaps explaining the earlier symptom of dysphagia, earlier diagnosis and more favorable 5 year survival rate. Given their rarity, no standard protocol has been developed to treat ECS tumors. Resection, regional lymph node dissection, radiotherapy and chemotherapy have all been utilized. Our case presentation further enriches the literature regarding rare, advanced esophageal adenocarcinoma tumors responding to systemic therapy.




[2408] **Figure 1.** A, B - Endoscopic findings. A large protruding, 6 cm in diameter partially obstructing ulcerating mass in the mid esophagus. C - Intense FDG uptake by the esophageal tumor in the esophagus (green arrow) and intense FDG uptake in the posterior/lateral aspect of the right T6 rib (yellow circle). D - Intensely avid subcarinal lymph node (yellow circle).

S2409

**A Case of Esophago-Pulmonary Fistula From Esophageal Candidiasis Diagnosed on Endoscopy**

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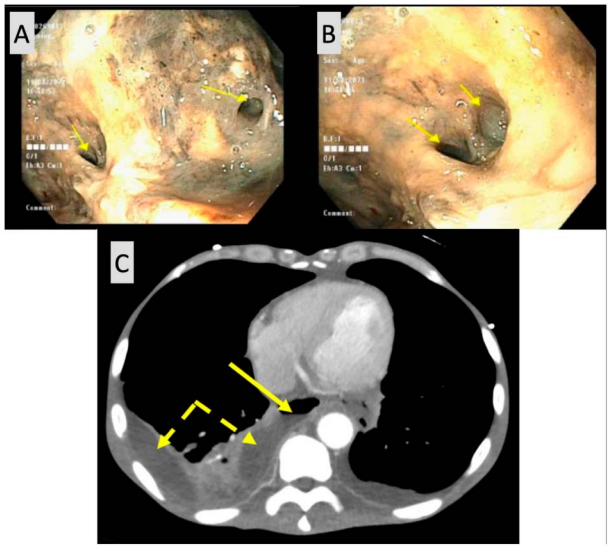
**Introduction:** Esophago-pulmonary fistulas (EPFs) are abnormal connections between the esophagus and lungs that are rarely seen despite the proximity of the 2 structures. They are associated with high morbidity and mortality. Fistulization between the esophagus and lungs is a known complication of pneumonectomy procedures, but other causes include malignancies and infection.
**Case Description/Methods:** A 61-year-old male with a history of tobacco and alcohol use disorder, diabetes mellitus, and esophageal candidiasis, with both *Candida glabrata* and *Candida albicans* isolated on biopsies from an otherwise normal endoscopy 4 months prior presented with presumed septic shock and melena. Blood cultures were positive for *C. glabrata*. Esophagogastroduodenoscopy (EGD) was done and revealed a fistulous tract leading to a bronchial bifurcation consistent with an esophago-pulmonary fistula with necrotic and friable tissue surrounding it (Figure A and B), in addition to grade D esophagitis. Subsequent CT chest showed definite right and possible left esophago-pleural fistulization with associated right loculated pleural effusion and small left pleural effusion (Figure C). No evidence of pulmonary malignancy or pulmonary tuberculosis was noted on imaging. Serum interferon gamma release assay and 3 sets of sputum acid fast bacillus testing for tuberculosis testing were negative. Thoracic Surgery were consulted but the procedure offered was multistage and did not align with patient's goals of care. The patient continued to decline despite antifungal and antibiotic treatment and was ultimately transitioned to hospice care.
**Discussion:** While formal tissue diagnosis was not obtained in this case due to patient's tenuous condition, the etiology of EPF was presumed to be invasive esophageal candidiasis infection, especially in the absence of malignancy on imaging and recent EGD. Interestingly, fistulas related to empyema were commonly reported in the pre-antibiotic era. A CT scan of the chest is useful for early diagnosis of EPF. While different modalities help establish the diagnosis of EPF, clinical suspicion remains key in initiating appropriate investigation.




[2409] **Figure 1.** A and B: Lower third of esophagus with necrotic tissue surrounding esophago-pleural fistula (arrows). C: (Axial CT showing an air-fluid level within the distal esophagus with loss of the fat plane between the esophagus (arrow) and the loculated right pleural effusion (dashed arrows), compatible with an esophago-pleural fistula).

S2410

**A Case of ADPKD Leading to Extrinsic Compression of the Stomach and Dysphagia**

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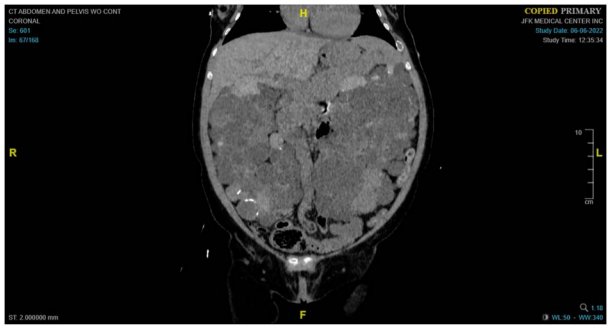
**Introduction:** Autosomal dominant polycystic kidney disease (ADPKD) is a common inherited disease with incidence of 1 in a 1000 live births. Disease symptoms usually present after 30 years of age and progresses to end-stage renal disease by the fifth decade of life. In addition to development of renal cysts which enlarge the kidneys, these can also form in the liver, pancreas and spleen. In the setting of renal enlargement, serious complications can develop such as extrinsic compression of internal structures. We present a case of massively enlarged polycystic kidneys leading to progressive dysphagia mimicking esophageal dysmotility disorder secondary to extrinsic compression of the stomach.
**Case Description/Methods:** A 43-year-old female diagnosed with ADPKD 4 years prior, presented with progressive dysphagia to solids and liquids for 1 month. Patient suffered from ESRD on hemodialysis. CT abdomen and pelvis showed massively large polycystic kidneys occupying most of the abdominal space, polycystic liver and extrinsic compression of the stomach. EGD from a recent prior hospitalization showed esophagitis and biopsy confirmed gastritis. GI series showed physiologic pattern of swallowing, no strictures or intrinsic obstruction seen in the esophagus, stomach and duodenum. The patient was started on a dysphagia diet which partially controlled her symptoms. Urology was consulted for bilateral nephrectomy (**Figure**).
**Discussion:** This case reports a rare complication of ADPKD and to the best of our knowledge, this is the first case of massive renal cysts presenting as dysphagia in ADPKD. Mass effect symptoms described in literature were leg edema, abdominal fullness or pain, emesis, early satiety, heart failure, arrhythmia, intestinal obstruction and IVC compression. Dysphagia due to extrinsic compression should be considered as a complication of this disease. Renal cyst size correlates with degree of renal function and mass effect. In such cases, unilateral or bilateral nephrectomy should be strongly considered to decrease morbidity and mortality.




[2410] **Figure 1.** Massive polycystic kidneys.

**S2411**

**A Case of Esophagitis in a Patient With Poorly Controlled Systemic Lupus Erythematosus**

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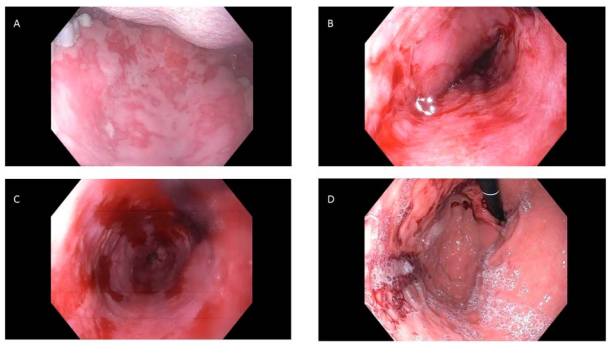
**Introduction:** Primary Systemic Lupus Erythematosus (SLE) esophagitis is a rare entity that most commonly presents with odynophagia and dysphagia. Though esophageal involvement with SLE is common, it typically manifests as a motility disorder or secondary infectious or pill esophagitis rather than a primary SLE esophagitis. While its underlying pathologic mechanisms may be similar to other manifestations of SLE, SLE esophagitis remains poorly understood.
**Case Description/Methods:** A 33-year-old man with history of juvenile discoid SLE complicated by arthritis, cutaneous involvement, oral ulcers, cerebritis, and 4-limb avascular necrosis secondary to chronic steroid use presented with a week of worsening oral ulcers, new-onset odynophagia, stable diffuse arthralgias, and active discoid lupus with ear and scalp involvement. His oral ulcers were more numerous and painful than was typical for his flares, and he had not previously experienced odynophagia during flares. Esophagogastroduodenoscopy (EGD) revealed diffuse erythematous, exudative, sloughed mucosa throughout the entire esophagus and stomach, while initial pathology revealed nonspecific findings with concern for possible vesiculobullous disorder. The patient was managed with increased immunosuppression for presumed SLE flare and his symptoms improved. Repeat EGD was performed to rule out an autoimmune blistering process, and immunofluorescence demonstrated granular deposits of IgG, IgM, and complement within the epithelial basement membrane, suggestive of SLE as the basis for the esophageal inflammation (**Figure**).
**Discussion:** SLE esophagitis is an uncommon condition that results from granular deposition of immune complexes in the basement membrane of the esophageal epithelium, representing a form of SLE mucositis in an unusual location. In patients with a history of SLE who present with odynophagia or dysphagia this diagnosis should be considered as a possibility, especially in those with many previous SLE-related complications. Diagnosis requires EGD with biopsy to differentiate between this and more common causes of esophagitis seen in SLE patients. Management of SLE esophagitis should mirror treatment of other SLE flares with appropriate immunosuppressive therapy but warrants follow-up with a gastroenterologist to assess need for future EGD monitoring.




[2411] **Figure 1.** A: Ulcers on palate of mouth; B: Middle third of the esophagus; C: Lower third of the esophagus; D: Antrum of the stomach.

**S2412**

**A Case of Idiopathic Hypereosinophilic Syndrome Involving the Gastrointestinal Tract**

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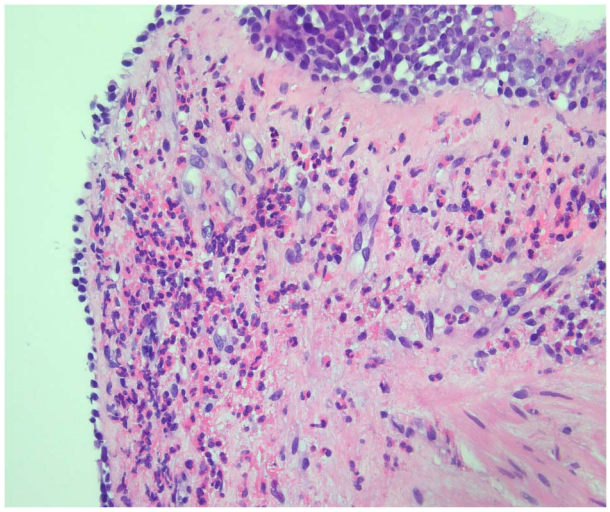
*[1]Creighton University School of Medicine, Omaha, NE; [2]CHI Creighton University Medical Center, Omaha, NE.*

**Introduction:** Hypereosinophilia (HE) is defined by an absolute eosinophil count greater than 1.5 k/mcL taken one month apart or by histologic confirmation of eosinophilic infiltration of the tissues. Hypereosinophilic syndrome (HES) occurs when HE causes organ damage. Complications from eosinophilic gastrointestinal (GI) disease include perforation, ischemic colitis, and portal vein thrombosis. Here we present a case of idiopathic HES involving multiple organs including the GI tract.
**Case Description/Methods:** A 39-year-old male presented to the hospital with 3 weeks of burning epigastric pain and diarrhea associated with fever; chills; fatigue; shortness of breath, and macular rash to the lower extremities. This began days after initiating a 12-day prednisone taper starting at 20 mg twice daily for presumed insect bite reaction. Eosinophils were 6.0 k/mcL on admission. Skin biopsy performed on lower extremities showed diffuse eosinophilic infiltration without evidence of vasculitis. Bronchoscopy revealed eosinophilic infiltration (92% eosinophils) without infection. ANA, anti-dsDNA, SSA, SSB, scleroderma antibody, C3, C4, RF, IgM, IgE, urine histoplasma antigen, serum coccidioides antibody, and antiphospholipid antibodies were within normal limits. With eosinophilic tissue infiltration associated with organ dysfunction and absolute eosinophil count >1.5 k/mcL, the patient met criteria for HES. Two doses of intravenous methylprednisolone 125 mg were administered before starting oral prednisone 60 mg AM and 20 mg PM for 90 days. The patient's epigastric pain, shortness of breath, and pruritus resolved. Eosinophilia levels trended down from 9.3 k/mcL to 0.4 k/mcL within 3 days.
**Discussion:** While HES is primarily an immunological condition, it is important for physicians to be aware of eosinophilic manifestations of the GI tract. The patient's severe epigastric pain and diarrhea were suggestive of eosinophilic gastroenteritis. He had skin and bronchial biopsies (**Figure**) along with peripheral eosinophilia and multiorgan involvement meeting criteria for idiopathic HES. Esophagogastroduodenoscopy and tissue sampling from the GI tract was not obtained as the patient responded well to treatment, hence adding an extra procedure wouldn't have altered the course of management. Early recognition of our patient's symptoms led to a minimally invasive patient experience and a rapid response to therapy.



[2412] **Figure 1.** Biopsy of bronchial mucosa showing dense infiltration of eosinophils beneath the bronchial epithelium (greater than 50 per high power field). The granular pink cells are the eosinophils, largely on the left-hand side of the image (400X magnification, H&E stain).

**S2413**

**A Case of Esophageal Intramural Pseudodiverticulosis**

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[1]Larkin Community Hospital, Doral, FL; [2]Larkin Community Hospital, Hialeah, FL; [3]Palmetto General Hospital, Hialeah, FL.

**Introduction:** Esophageal intramural pseudodiverticulosis (EIP) is a rare benign condition, presenting in < 1% of endoscopy cases. Its trademark appearance is that of a flask-shaped outpouching lesions along the esophageal wall. It is known to coexist with gastroesophageal reflux disease (GERD), esophageal stricture, diabetes, alcohol dependence syndrome, and achalasia. They are not considered true diverticula due to them morphologically consisting of widened secretory ducts of the submucosal glands. Patients with EIP present most commonly complaining of dysphagia. Patients generally are diagnosed by barium swallow study, computed tomography, and endoscopy; with endoscopic ultrasound being used for further determination of the layers involved. Here we present a case of a patient with a previously treated esophageal stricture who suffered from chest pain and was diagnosed with EIP by esophago-gastro-duodenoscopy (EGD).
**Case Description/Methods:** A 68-year-old Hispanic female with a history of hypertension, hypothyroidism, and untreated GERD presented to the emergency department complaining of chest pain for 1 week. Initial chest pain evaluation for cardiac results were negative, further investigation revealed a history of esophageal stricture with pneumatic dilation 30 years prior to presentation. Patient underwent a barium swallow study demonstrating pockets of residual barium. An EGD was performed, discovering >20 pseudodiverticula, gastritis, and multiple duodenal ulcers. The patient admitted to a long history of dysphagia with her most recent EGD being performed in Cuba 10 years prior. No diagnosis or discovery of esophageal diverticula was mentioned, but was found to have gastritis and ulcers at that time. Proton pump inhibitor therapy was initiated with further evaluation plans to consist of endoscopic ultrasonography.
**Discussion:** The risk of developing esophageal diverticula is increased by the presence of GERD and esophageal motility disorder, primarily in the thoracic portion of the esophagus. In this case, our patient had a history of a pneumatic dilation as well as untreated GERD which greatly increased her risk of developing diverticula. This may be a unique presentation of EIPD after prior esophageal stricture treatment. Though the definitive cause of EIPD remains uncertain at this time, it is common to treat these patients with PPIs due to its association with GERD. It may be beneficial for future studies to further identify potential high risk factors.

**S2414**

**A Classic Case of Black Esophagus**

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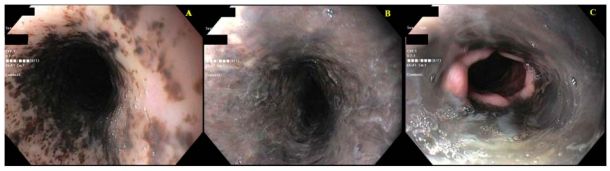
Mount Carmel Health Systems, Grove City, OH.

**Introduction:** Acute esophageal necrosis (AEN) is a rare syndrome also known as black esophagus. Aptly named, it is described as a circumferential, diffuse, black appearing esophageal mucosal lesion affecting variable lengths of the esophagus. Classically, the discoloration ends sharply at the gastroesophageal junction.
**Case Description/Methods:** A 50-year-old woman presented to the emergency department via ambulance for altered mental status. She had undergone 2 renal transplantations and was on chronic immunosuppression. She was intubated and treated for septic shock. CT scan demonstrated ureteral obstruction of her lone functioning transplant kidney with pyelonephritis and severe gastric distention suggestive of outlet obstruction. Nephrostomy tube was placed, and the patient was admitted to the critical care unit. Two days later, significant amount of coffee ground enteric contents was noted via nasogastric tube. Gastroenterology was consulted and twice daily intravenous proton pump inhibitor (PPI) was started. Urgent esophagogastroduodenoscopy was performed. Severe, circumferential black esophagitis was seen without ulceration. Intravenous PPI therapy and supportive care was continued. The patient was discharged 8 days from admission in stable condition with persisting odynophagia (**Figure**).
**Discussion:** This case represents the classical, 2-insult presentation of AEN: septic shock and gastric outlet obstruction. Once diagnosis is established, careful surveillance is required. However, there are no established guidelines for surveillance. The most important and life-threatening complication is perforation and should be suspected if rapid clinical worsening is seen. Per expert opinion, PPI therapy should be transitioned to oral therapy once improvement is noted and continued until repeat endoscopy in approximately 8 weeks. As of 2006, there were 88 cases documented in medical literature. AEN is thought to require 2 insults due to the significant collateral and segmental blood supply of the esophagus. First, global hypoperfusion is expected. Second, profound mucosal injury from reflux of gastric contents. Treatment includes managing the underlying medical conditions, supportive care, and PPI therapy. Complications following AEN are strictures, abscesses, and perforation. Associated mortality is approximately 30%. This is likely due to the underlying medical conditions that cause AEN. Awareness and understanding of the condition and its complications is important to manage and surveil survivors appropriately.




[2414] **Figure 1.** A- Upper third of the esophagus. B- Middle third of the esophagus C- Lower esophageal sphincter.

S2415

**A Common Procedure Leads to the Diagnosis of an Uncommon Cause of Dysphagia**

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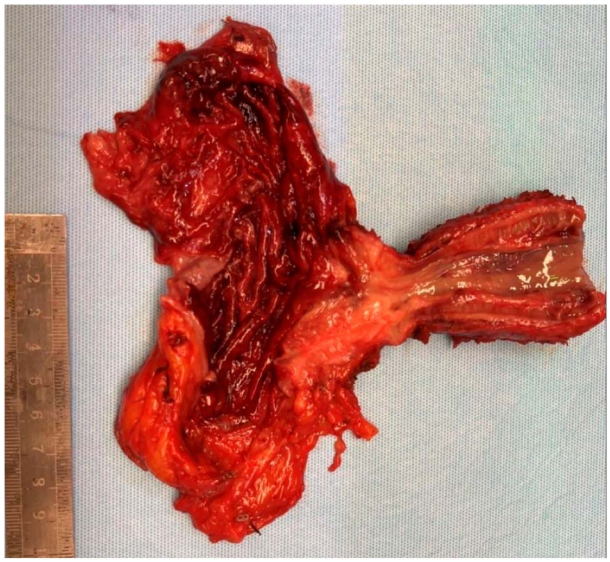
*Kaiser Permanente Los Angeles Medical Center, Los Angeles, CA.*

**Introduction:** Epithelioid angiosarcoma is a rare and aggressive soft-tissue endothelial cell neoplasm that accounts for less than 1% of sarcomas and rarely occurs in the gastrointestinal tract. We report a case of a 41-year-old female without prior medical history who presented with progressive dysphagia and unintentional weight loss.
**Case Description/Methods:** Initial upper endoscopy found to have a narrowed gastroesophageal junction (GEJ) that required balloon dilation to 10mm and the use of a pediatric upper endoscope to traverse. Only thickened gastric folds at the cardia were seen, which were biopsied. Histology revealed chronic active moderate gastritis with *Helicobacter pylori* and patient was treated with triple therapy. However, due to ongoing dysphagia a contrast enhanced computed tomography (CT) of the chest was obtained and demonstrated thickening of the distal esophageal wall. Next, an endoscopic ultrasound (EUS) was performed and demonstrated unusual thickening of the distal esophageal wall. Fine needle biopsy (FNB) revealed high-grade esophageal epithelioid angiosarcoma of the distal esophagus. She was treated with neoadjuvant chemo- and radiotherapy followed by a robotic laparoscopic Ivor Lewis esophagectomy. The patient has been disease free after 1 month of follow up (**Figure**).
**Discussion:** This case highlights the importance of timely recognition and diagnosis of dysphagia caused by pseudo-obstruction. Timely CT and EUS/FNB should be performed to evaluate for less common entities.



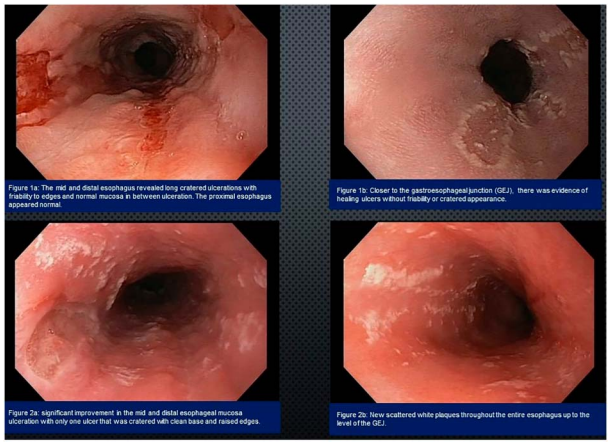
[2415] **Figure 1.** Case image.

S2416

**A Case of Severe Undifferentiated Ulcerative Esophagitis as an AIDS-Defining Illness in Acute HIV Infection**

Clemence White, DO[1], Christina Steinhauser, DO[2].
[1]*Walter Reed National Military Medical Center, Bethesda, MD;* [2]*Mike O'Callaghan Military Medical Center, Las Vegas, NV.*

**Introduction:** The esophagus is a common target organ in acute HIV infection (fungal or viral agents). Idiopathic esophageal ulcers are less common and we present a case of severe idiopathic ulcerative esophagitis in acute HIV infection.
**Case Description/Methods:** 29-year-old male with no prior medical history presented with symptoms of odynophagia, dysphagia and limited oral intake for 2 weeks. Due to his symptoms, he lost 25lbs at presentation. Esophagogastroduodenoscopy (EGD) revealed long cratered ulcerations with friability to the edges in the mid and distal esophagus (Figure 1a). The mucosa between the ulcers appeared normal. Closer to the gastroesophageal junction (GEJ), there was evidence of healing ulcers without friability or cratered appearance (Figure 1b). These lesions were biopsied (edges and base). The mucosa of the proximal esophagus, stomach and duodenum appeared normal. Due to endoscopic assessment concerning for viral infection, serology (HSV, CMV and HIV) were ordered. The patient was treated with empiric Valgancyclovir for presumed CMV esophagitis. The biopsy stained positive for undifferentiated hyphae type mold elements and viral stains were negative. His viral serology were normal but HIV RNA testing was positive. With infectious disease engagement, additional tests were done to rule out any opportunistic infection and results were negative. He was started on HAART therapy. Two weeks after initiating Valgancyclovir and HAART therapy, a repeat EGD showed significant improvement in the mid and distal esophageal mucosa ulceration with only one clean base cratered ulcer noted (Fig 2a). There were new and enlarged scattered white plaques throughout the entire esophagus up to the level of the GEJ (Fig. 2b). Several biopsies obtained from the ulcer and the white plaques were consistent with candida esophagitis, see Figure 2b. He was treated with a course of fluconazole. Currently, patient is well controlled on HAART therapy with excellent immunologic and virologic response.
**Discussion:** Idiopathic esophageal ulcers occur in about 10% of patients with acute HIV infection or AIDS. Prior reports have described giant (greater than 5cm) ulcers with profound depth usually located in the mid esophagus. The exact pathophysiology is unknown. Gastroenterologists should maintain a low index suspicion for an immunocompromised state in all patients presenting with odynophagia or dysphagia. Upon diagnosis, compliance with HAART therapy is key for treatment of HIV related esophageal ulcers.




[2416] **Figure 1.**

**S2417**

**A Diagnosis That Is Difficult to Swallow: Digging Deeper for Answers**

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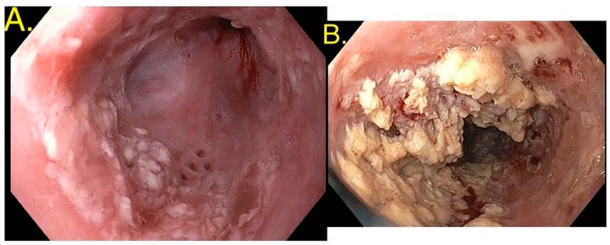
**Introduction:** Esophageal parakeratosis (EP) is an uncommon finding with largely unknown clinical implications and malignant potential. Here, we report a case of severe esophageal parakeratosis.
**Case Description/Methods:** Patient is a 69-year-old African American male with history significant alcohol and cocaine abuse, prior tobacco abuse, keloids, gastroesophageal reflux, food impaction, and progressive dysphagia for 20 years with 40-pound weight loss. Patient's first esophagogastroduodenoscopy (EGD) in 2000 revealed moderate esophagitis and a distal esophageal stricture with 9 mm lumen bougie dilated to 12 mm, but dysphagia persisted. Repeat EGDs between 2006 and 2016 showed whitish plaques with no evidence of fungal organisms. Given classic appearance of EOE, he was trialed on empiric fluticasone and budesonide with no improvement in dysphagia. In September 2021, he was hospitalized with severe dysphagia. EGD (**Figure**) at this time showed gray, yellow frondlike lesion replacing esophageal lumen from mid to distal esophagus with mucosal friability. His dysphagia transiently responded to repeat dilations, but continued to recur and worsen. Unfortunately, patient continued to have weight loss and dysphagia unresponsive to dilations. The patient was then started on gastric tube feedings. He was offered surgery due to the severity of his symptoms, but he declined. Most recently, the patient underwent endoscopic mucosal resection with deeper tissue samples revealing superficial fragments of laterally spreading squamous cell carcinoma of the esophagus.
**Discussion:** In this case, there was a high clinical suspicion for malignancy, leading the endoscopist to obtain deeper tissue samples via endoscopic mucosal resection as superficial fragments of the esophagus only revealed parakeratosis. It is important to trust clinical judgment, especially if there is a high suspicion for malignancy. Tylosis also has a known association with EP and often presents with areas of thickened skin plaques on palms and feet. Tylosis has a strong association with head, neck, and esophageal squamous cell carcinoma. The patient had no family history of tylosis and no cutaneous features. Despite this, a careful inspection of the entire EP area with targeted biopsies should be obtained to evaluate for dysplasia. This case represents EP with years of no evidence of squamous cell cancer despite aggressively searching for it, which at least anecdotally suggests a precancerous potential for EP.




[2417] **Figure 1.** A. Initial EGD B. Recent EGD.

**S2418**

**A Middle-Aged Patient With an Unusual Cause of Dysphagia**

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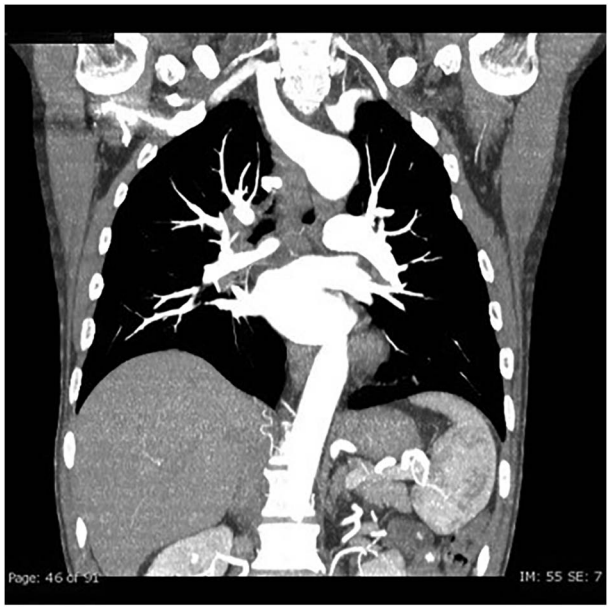
**Introduction:** Dysphagia is a common bothersome symptom affecting 3% of the US at any given time [1]. It comes with a wide range of potential causes that can be categorized into anatomic, obstructive, and neuromuscular. The most common pathophysiologic origin is from gastroesophageal reflux disease. Other causes include stroke, malignancy, and anaphylaxis. Due to the broad nature of this symptom, the rarer causes of dysphagia go unrecognized and under-diagnosed. Here we present a rare case of dysphagia in a middle-aged woman diagnosed from radiologic imaging.
**Case Description/Methods:** A 65-year-old male patient presented to the emergency department with a 2-day history of progressive dysphagia. Past medical history was significant for squamous cell carcinoma involving the base of the tongue, pharynx, and larynx, which was treated in 2018 with definitive chemoradiation therapy. A chest computed tomography scan with intravenous contrast revealed an anomalous prominent right subclavian artery (ARSA) coursing posterior to the esophagus (**Figure**). Arrangements were made for the patient to be transferred to a nearby tertiary medical center for a right carotid-subclavian bypass with proximal ligation of the ARSA.
**Discussion:** ARSA is an anatomical anomaly derived from the abnormal origin of the right subclavian artery directly from the aortic arch as opposed to from the brachiocephalic artery. It courses through the right arm, crossing the midline of the chest, passing behind the esophagus. ARSA has the potential to compress the esophagus, causing dysphagia. According to Natsis et al., ARSA was found to have a prevalence of 0.16%-4.4% of the general population and has a relative high incidence in females and people of Greek origin [2]. In approximately 93% of these patient's the anomaly is asymptomatic however the remaining may experience dysphagia, stridor, dyspnea, or chest pain due to esophageal compression. Recognizing this condition prior to any thoracic surgery is essential as unintentional injury to this artery during surgical procedures may be life-threatening [3].




[2418] **Figure 1.** Coronal View Reconstructed coronal section of computerized chest tomography with maximum intensity projection demonstrating the rise of the right subclavian artery directly from the aortic arch rather than the brachiocephalic artery.

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**S2419**

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**A Motility Study in a Patient With Isolated Diaphragmatic Agenesis: An Interesting Study in a Very Rare Entity!**

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**Introduction:** Diaphragmatic agenesis is a very rare congenital defect. Diagnosis is typically made in infancy and it is commonly associated with other congenital malformations and high mortality. However, it has also been described in the literature as a distinct clinical entity with potentially later in life diagnosis, as in the case reported here.
**Case Description/Methods:** Our subject is a 57-year-old male who was diagnosed to have diaphragmatic agenesis at the age of 45 during a thoracotomy for a suspected diaphragmatic hernia. He presented to the gastroenterology clinic with a chief complaint of life-long intermittent but progressive dysphagia to both solids and liquids. Chest x-ray showed bowel loops and air-fluid level in the left-side of the chest with a cardiac and a mediastinal shift to the right side. Barium swallow was done and was negative for any abnormalities. A motility study was also done and with details showing in Tables 1-4 (**Figure**). Findings of the motility study are summarized as the following: 1) Normal resting pressure at lower esophageal sphincter with incomplete relaxation during swallows (high IRP), 2) Normal amplitude peristaltic contractions in esophageal body (Normal DCI), 3) Complete bolus transition, 4) Esophagogastric morphology I, and 5) Normal upper esophageal sphincter resting pressure. A chest X-ray obtained and attached in figure A shows the position of the probe used in the esophageal manometry with the gastroesophageal junction in the thoracic cavity. Figure B shows our patient's manometric study evaluating pressure changes that occurred during liquid swallowing. The diagnosis of esophagogastric junction (EGJ) outflow obstruction was made based on study results.
**Discussion:** Esophagogastric junction outflow obstruction is a major motility disorder based on the Chicago Classification of esophageal motility disorders. This entity involves a heterogenous group of underlying etiologies with diaphragmatic agenesis being one potential but rare cause evidenced by our reported case. Management of (EGJ) outflow obstruction is dependent on the underlying etiology.

Table (1) Lower esophageal sphincter:

| | | |
|---|---|---|
| LESP (mid respiration) | 34 mmHg | Normal 10-45 mmHg |
| Total length | 3.9 cm | |
| Median IRP | 21 mmHg | Normal <=20 mmHg |
| EGJ Morphology | Type I | |

Table (2) Lower esophageal body (Liquid: 10 swallows), analyzed according to Chicago Classification Metrics

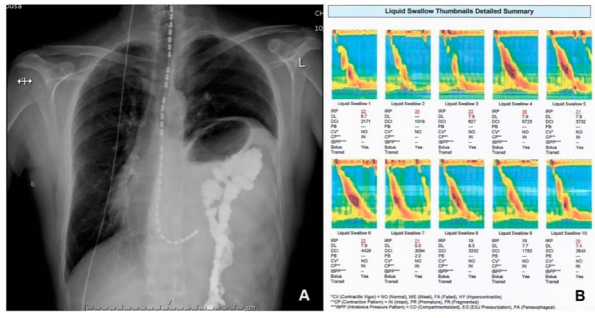
| | | |
|---|---|---|
| Normal DCI (Distal contractile integral) | 100 % | Normal >= 50.0 % |
| Mean DCI | 2855 mmHg-s-cm | Normal 450-8000 mmHg-s-cm |
| Ineffective | 0.0 % | Normal < 50.0 % |
| Hypercontractile | 0.0 % | Normal < 20.0 % |
| Premature (DL <4.5) | 0.0 % | Normal < 20.0 % |
| Panesophageal | 0.0 % | Normal <= 0.0 % |

Table (3) Lower esophageal body analyzed according to conventional metrics

| | | |
|---|---|---|
| DEA (Distal esophageal amplitude) | 134 mmHg | Normal <= 220 mmHg |
| Peristaltic | 100.0 % | Normal >= 80.0 % |
| Ineffective | 0.0 % | Normal < 50.0 % |
| Simultaneous | 0.0 % | Normal < 20.0 % |
| Retrograde | 0.0 % | |
| Non-Transmitted | 0.0 % | Normal < 20.0 % |

Table (4) Impedance

| | | |
|---|---|---|
| Distal baseline impedance | 841 ohms | |
| Complete Transient (liquid: 10 swallows) | 100.0 % | Normal >= 80 % |




[2419] **Figure 1.** (Top) Motility study details. A) Chest Xray showing the position of the probe used in the esophageal manometry. B) Patient's manometric study evaluating pressure changes that occurred during liquid swallowing.

S2420

**A Case of Type II Achalasia Presenting With Markedly Elevated Troponins**

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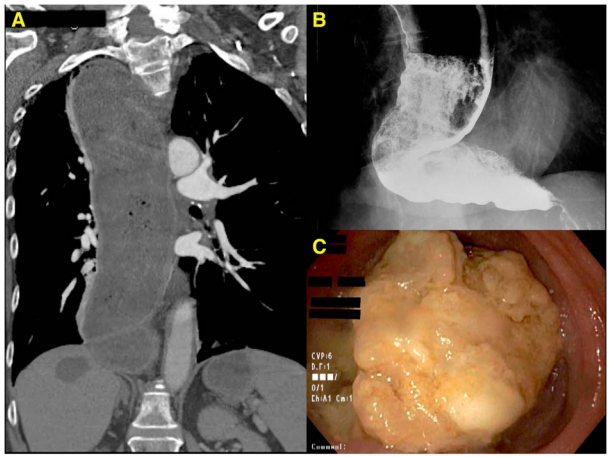
**Introduction:** Achalasia is an esophageal motility disorder that most commonly presents with dysphagia to solids and liquids, regurgitation, and heartburn over several years. In rare instances, it can mimic the presentation of acute coronary syndrome resulting in delayed diagnosis and unnecessary interventions. We report a case of achalasia that presented with markedly elevated troponins, prompting extensive cardiac workup.
**Case Description/Methods:** A 74-year-old female with a past history of hyperlipidemia presented with diffuse chest pain and shortness of breath for 1 day. Physical exam revealed tachypnea and diffuse chest tenderness. Chest X-ray showed a widened mediastinum. Laboratory studies revealed markedly elevated serial troponin values of 0.06 ng/ml, 0.09 ng/ml, and 1.14 ng/ml, respectively. This prompted an emergent cardiac evaluation with an electrocardiogram showing junctional ST depression, followed by a transthoracic echocardiogram that was unremarkable. CT angiogram of chest and abdomen revealed mega-esophagus measuring 6 cm in diameter containing a large amount of debris extending to the cervical esophagus (Figure A). Barium esophagram showed a dilated esophagus with pooling at the distal esophagus (Figure B). Esophagogastroduodenoscopy revealed hypertonic lower esophageal sphincter (LES), severely dilated esophagus with large amounts of undigested food (Figure C). Botulinum toxin was injected at the LES followed by balloon dilation. Esophageal manometry confirmed the diagnosis of Type II Achalasia. The patient underwent an uncomplicated Heller myotomy with fundoplication with gradual improvement in symptoms.
**Discussion:** Cardiac troponins (cTn) are sensitive biomarkers of myocardial ischemia (MI) and markedly elevated levels suggest acute thrombotic MI. Infrequently cTn elevation may be observed in gastrointestinal disorders such as achalasia that result in myocardial injury without ischemia. The likely mechanism is postulated to be significant myocardial compression secondary to the enlarged esophagus resulting in myocardial damage and troponin release. This case highlights the importance of forming a broad differential when evaluating patients presenting with elevated troponins and the importance of considering gastrointestinal causes to avoid misdiagnosis and delayed intervention once acute coronary syndrome has been ruled out.




[2420] **Figure 1.** A. Computed tomography angiogram of chest and abdomen showing megaesophagus in coronal view. B. Barium esophagram showing a dilated esophagus with pooling at the distal esophagus. C. Esophagogastroduodenoscopy showing severely dilated esophagus with large amounts of undigested food.

S2421

**A Dark Omen: Acute Esophageal Necrosis Associated With COVID-19**

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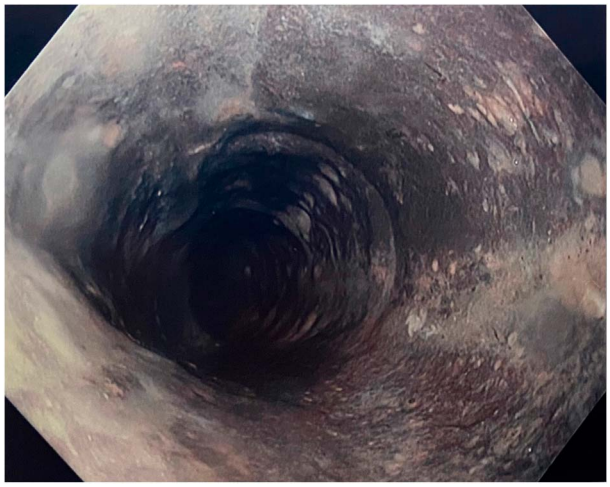
**Introduction:** Black esophagus, also known as acute esophageal necrosis (AEN), is a rare clinical entity with an estimated prevalence of 0.01 to 0.28 percent of cases in endoscopy series. It can present with striking endoscopic findings characterized by diffuse circumferential black discoloration of the esophagus terminating at the gastroesophageal (GE) junction. It is mostly associated with tissue hypoxia due to low flow vascular states and corrosive injury in the setting of severe illness. Patients typically present with gastrointestinal bleeding. We present a rare case of a black esophagus in the setting of severe Coronavirus-19 (COVID-19) infection.
**Case Description/Methods:** A 75-year-old male with diabetes and chronic kidney disease presented after a mechanical fall with a prolonged down time of 13 hours. He was found to have acute on chronic renal failure with rhabdomyolysis. He was initially stable however later developed respiratory failure from COVID-19 requiring steroids and ultimately ventilator support. His course was further complicated by new onset atrial fibrillation for which he received anticoagulation, worsening renal function requiring hemodialysis and significant hypotension requiring vasopressor support. In the intensive care unit, patient was noted to have melena with a significant drop in hemoglobin, he underwent an esophagogastroduodenoscopy which showed diffuse continuous black discoloration of the esophageal mucosa with abrupt transition at the GE junction consistent with black esophagus with multiple ulcers in stomach and duodenum (**Figure**). Patient was kept on IV proton pump inhibitors and anticoagulation was discontinued. Given worsening multiorgan failure, he was transitioned to comfort measures only and passed away the next day.
**Discussion:** Black esophagus or AEN is a rare but concerning finding on endoscopy which must be recognized early and managed aggressively in order to improve clinical outcomes. In our patient AEN was likely due to a combination of hypoperfusion and hypoxia in the setting of severe COVID-19 infection with multiorgan failure. AEN could be seen more frequently in patients with severe COVID-19 infections who are frequently anticoagulated due to the associated prothrombotic state with increased risk of GI bleeding. It is important for physicians to be aware of AEN as a possible etiology of GI bleeds in these patients.



[2421] **Figure 1.** 'Black esophagus' - Acute esophageal necrosis.

**S2422**

**A Lichen for the Esophagus: A Rare Case of Esophageal Lichen Planus**

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**Introduction:** Lichen planus is an inflammatory condition of the skin and mucosal membranes. Sites of disease are variable and esophageal involvement is uncommon. We highlight a case of esophageal lichen planus masquerading as eosinophilic esophagitis.
**Case Description/Methods:** A 59-year-old female with a past medical history of hypothyroidism status post thyroidectomy presents with progressive dysphagia and odynophagia to solids for 3 years. Symptoms initially began after thyroidectomy. Evaluation by ENT was notable for inflammatory changes of the oropharynx for which PPI trial was given for clinical features of silent reflux. Despite a 2-month course of therapy, symptoms persisted. Videofluoroscopic swallow study was unremarkable without evidence of aspiration or penetration. Subsequent EGD was notable for exudates, linear furrowing, and rings to suggest the diagnosis of eosinophilic esophagitis. Esophageal biopsy pathology features, however, showed band-like lymphocytic infiltration of the lamina propria and civatte bodies within the squamous epithelium consistent with esophageal lichen planus. Unfortunately, the patient was lost to follow up and subsequent treatment was unable to be initiated.
**Discussion:** Esophageal lichen planus is an exceedingly rare entity with less than 100 documented case reports. It is likely underdiagnosed given that it can mimic other esophageal entities, such as eosinophilic esophagitis as seen in our patient. As such, histologic examination is necessary to identify key features of esophageal lichen planus which include lymphocytic infiltrates of the lamina propria as well as civatte bodies, which represent necrotic keratinocytes. Esophageal lichen planus has a tendency to be chronic and require topical or systemic therapy with steroids. Stricture development will typically require dilatation. Clinicians should consider esophageal lichen planus as part of their differential for dysphagia especially given the potential for malignant transformation to squamous cell carcinoma, which has been reported in 1-2% of cases.

**S2423**

**A Case Report of Iatrogenic Esophageal Perforation: Complication of Orogastric Tube Placement**

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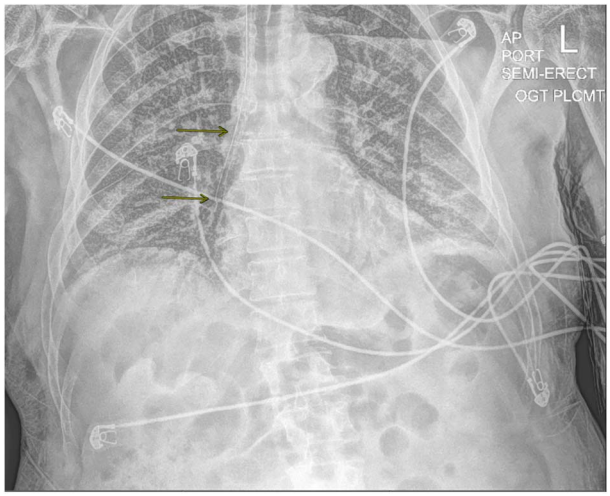
MedStar Health, Baltimore, MD.

**Introduction:** Orogastric tube insertion is a routine procedure in medical care. However, misplacement of the tube can cause a variety of complications, which can be life threatening in some instances.
**Case Description/Methods:** 71-year-old male presented with dyspnea, fever, chills, cough, and myalgia for 2 weeks. He had tachycardia, tachypnea, and was hypoxic to 66% in room air. He was found to have acute hypoxic respiratory failure secondary to COVID-19 Pneumonia and was admitted to ICU. But, he continued to be hypoxic and was started on BiPAP. He eventually became altered, and was intubated. Post intubation orogastric tube (OGT) placement was unsuccessful on the first attempt due to resistance. On the second attempt, the nurse was able to advance partially (**Figure**). But, a chest XR showed OGT in the mediastinum, and OGT was removed. CT of neck and chest revealed pneumomediastinum with possible mid-thoracic esophageal perforation. The patient was started on broad-spectrum antibiotics and thoracic surgery was consulted. Given his mechanical ventilation requirement, surgery deemed him unfit to tolerate thoracotomy and the endoscopic procedure was not available in the hospital. So, recommendation was to manage conservatively. His hospital course was complicated by hypotension requiring vasopressors and metabolic acidosis in setting of acute renal failure requiring CRRT. Code status was changed by the family to Do Not Resuscitate due to his deteriorating condition. Eventually, he had a PEA arrest and was expired.
**Discussion:** OGT intubation is performed at hospitals for feeding, medication administration or gastric decompression. Although it is considered a safe procedure, complications can arise due to OGT misplacement or trauma caused by the OGT itself or the intubation process. OGT misplacement is typically endotracheal or intracranial. Misplacement within the upper GI lumen is usually detected by a kink in the oropharynx or esophagus. The subsequent complications are identified by the structure that is perforated (e.g., mediastinitis or pneumothorax). Regardless of whether counteraction is perceived, the physician must be careful not to apply excessive force. The location of the OGT tip should be determined by a chest radiograph; visualization of the tip below the diaphragm verifies appropriate placement. Complications of OGT insertion are uncommon; however, the consequences are potentially serious, and the anatomy of the upper GI tract should be understood by all who are involved in the care.




[2423] **Figure 1.** Orogastric tube lies to the right of the trachea and has its tip adjacent to the right side of the heart. Orogastric tube may have perforated the esophagus and is positioned in the right side of the mediastinum.

**S2423A**

**Not a Regular Gastric Inlet Patch: A Unique Patient With an Ulcerated Heterotopic Gastric Mucosa of the Entire Middle Esophagus**

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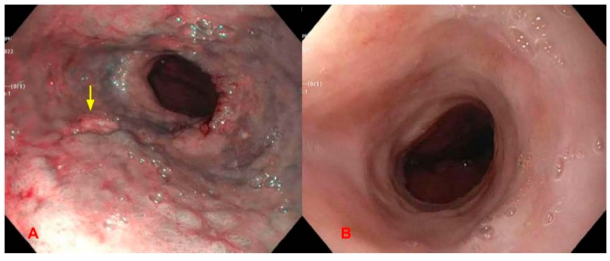
**Introduction:** Heterotopic Gastric Mucosa (HGM), the presence of columnar epithelium of the stomach, can be detected in any part of the gastrointestinal (GI) tract. Inlet patch is a relatively common endoscopic finding where HGM is seen in the cervical esophagus. Mid esophageal HGM is an exceedingly rare entity. Finding of HGM in distal esophagus can be challenging since it needs to be differentiated from Barrett's esophagus (BE), a lesion with high malignant potential. We report a unique patient with a large (10 cm) patch of mid esophageal HGM who presented with upper GI bleeding.
**Case Description/Methods:** A 82 year old female with history of hypertension and breast cancer presented with shortness of breath and syncope. Patient reported that since 3 weeks she was noticing intermittent liquid black stools. Patient denied taking any non-steroidal anti-inflammatory drugs. Patient also denied any surgical intervention in the abdominal or thoracic cavity. Vitals were unremarkable. Physical exam demonstrated black-tarry liquid stools on rectal exam consistent with melena. Blood work was significant for hemoglobin of 5.9 g/dL with a baseline of 12 g/dL , BUN/Creatinine 68 mg/dL/1.66 mg/dL and with a baseline of 18 mg/dL/1.30 mg/dL. Subsequently patient had an esophagogastroduodenoscopy (EGD) which revealed diffuse salmon-colored mucosa present at 19 to 29 cm from incisors that was erythematous, nodular, friable, and with ulcerations [Fig 1A]. Mucosa was biopsied in a targeted manner in 4 quadrants at intervals of 2 cm. These biopsies demonstrated gastric mucosa with moderate chronic inflammation without any squamous epithelium present. These biopsies were also negative for H. pylori, intestinal metaplasia and dysplasia. Patient was started on Proton pump inhibitor and is planned for a repeat EGD in 3 months to assess for mucosal healing.
**Discussion:** Mid esophageal heterotopic gastric mucosa is an extremely rare entity which if ulcerated, can lead to life threatening bleeding. Majority of the patients are asymptomatic and diagnosed incidentally when it is complicated with esophageal webs, rings, strictures, fistula, ulcers as in our case, and malignancy. Biopsy plays a key role in differentiating HGM from BE by finding columnar epithelium without intestinal metaplasia or Goblet cells. Treatment is mainly focused on complications seen during endoscopic examination and histopathology. Unlike BE, there are no clear guidelines for the surveillance of HGM since malignant transformation is exceedingly rare.




[2423A] **Figure 1.** A) 10cm salmon-colored mucosa in mid esophagus consistent with gastric mucosa without any squamous cells or intestinal metaplasia.The mucosa was erythematous, nodular, and friable with ulcerations (arrow) B) Normal appearing lower esophagus.

**S2424**

**A Hard Pill to Swallow: Unique Presentation of Massive Esophageal Food Bolus Impaction**

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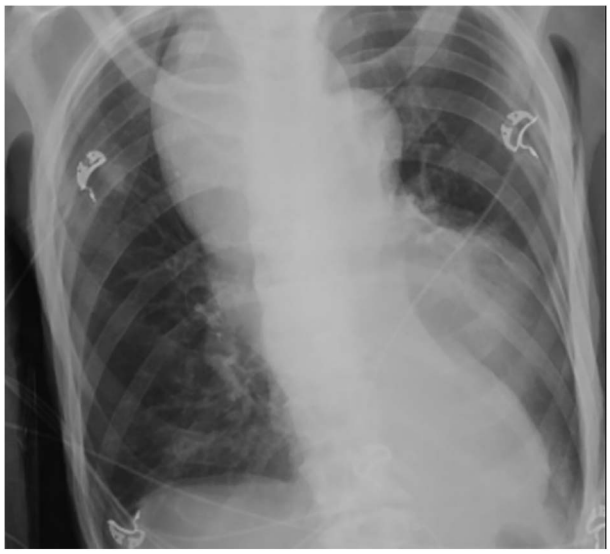
**Introduction:** Foreign body ingestion and esophageal food bolus impactions (EFBI) are common Gastroenterology emergencies, with an annual incidence of 13:100,000. While the vast majority of cases resolve on their own, about 10-20% of cases require endoscopic evaluation and disimpaction. In most cases, the composition of the food bolus, clinical condition of the esophagus, and underlying esophageal pathology contribute to the impaction. Imaging is usually difficult to obtain given the radiolucent nature of food. In this case, we encounter a presentation of esophageal food impaction that eventually contributes to the patient's demise.
**Case Description/Methods:** An 86-year-old male with a past medical history of achalasia complicated by small distal esophageal perforation status post laparoscopic Heller myotomy in 2013 was admitted after presenting with complaints of chest pain and an inability to tolerate a solid diet. On admission, the patient noted that he used to weigh approximately 180 lbs about a year ago compared to his admission weight of 90 lbs. Initial imaging in the ED showed an air-filled, dilated esophagus decreased in caliber from prior imaging. Eight days into the patient's admission, his family presented to bedside to assist in 1-to-1 feeds at both the patient's and family's request. Thereafter, A CT chest following demonstrated a markedly dilated appearance of the patient's esophagus with internal food material without a definite large obstructing lesion, markedly progressed from prior imaging. The patient was subsequently made NPO, transitioned to TPN, and plans were made for a follow-up disimpaction via EGD. The patient, however, was too unstable to undergo the procedure and expired 9 days after a cardiac arrest despite the efforts of a multidisciplinary team (**Figure**).

**Discussion:** Through literature review, a majority of cases of food bolus impaction are self limited. In the cases described, boluses pass on their own or with the assistance of an EGD. In most cases, underlying esophageal or motility dysfunction is known. We illustrate a common presentation to gastroenterologists and physicians of a food bolus impaction. Though, due to the profound radiographic presentation and severe morbidity of our clinical scenario, we hope to bring attention to the need for rapid evaluation, treatment, and consideration of adverse outcomes in patients presenting with food boluses as well as the severity and life-threatening outcomes that may preside with the previously trivially described pathology.



[2424] **Figure 1.** Manifestation of Esophageal Food Bolus Impaction on Initial Imaging.

**S2425**

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**A Case Report of Volatile Ingestion-Induced Acute Esophageal Necrosis**

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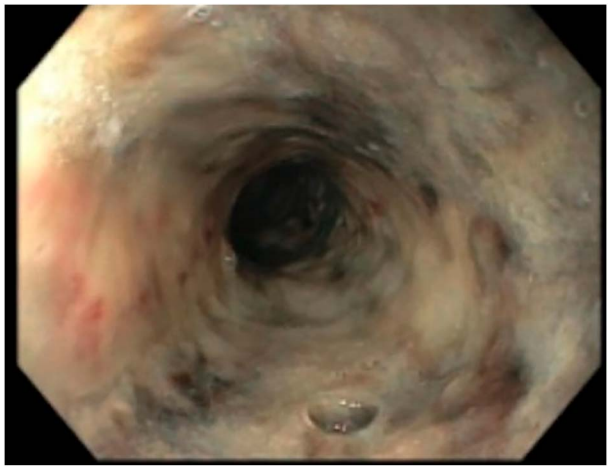
**Introduction:** Acute esophageal necrosis (AEN), also known as black esophagus or acute necrotizing esophagitis, is a rare syndrome with diffuse circumferential black discoloration often of distal third esophageal mucosa due to relative hypovascularity and termination at the gastro-esophageal junction. It usually presents with upper GI hemorrhage. Despite controversial pathogenesis, there is strong consideration for a 2-hit hypothesis where an initial low-flow vascular state predisposes the mucosa to a severe topical injury. Chemical injury by gastric contents, toxin ingestion, severe infectious diseases, diabetic ketoacidosis and shock are associated with AEN. Advanced age, male gender and comorbidities like diabetes mellitus (DM), hypertension, cardiovascular disease, chronic kidney disease, malnutrition, chronic alcohol abuse, post-irradiation are predisposing risk factors for AEN. We present a patient with a favorable outcome of a generally rare, life-threatening condition due to early recognition and treatment.
**Case Description/Methods:** A 74-year-old male presented to the hospital with shock and 3-day history of hematemesis. He had history of coronary artery disease, Type II Diabetes Mellitus, chronic obstructive pulmonary disease, esophageal adenocarcinoma post chemotherapy and radiation complicated by erosive esophagitis and alcoholism. Labs were significant for acute kidney injury (AKI), high anion gap metabolic acidosis (HAGMA), osmolar gap of 59 mOsm/kg and hemoglobin drop from 14 to 9 g/dL. Volatile panel not sent; drug-screen benign, infectious work-up negative. CT abdomen had distended stomach; thus, NG tube placed for decompression, yielding 700cc of coffee ground emesis. Upper endoscopy performed 2-days later showed Los Angeles Grade D esophagitis with entire esophagus with black appearance, gastric body erosions and superficial clean-based duodenal ulcers. Patient was continued on PPI twice daily for 8 weeks with planned repeat EGD (**Figure**).
**Discussion:** This patient likely had volatile ingestion chemical injury given shock, hematemesis, AKI with HAGMA and osmolar gap. Despite AEN's rarity with prevalence of 0.2 percent in autopsy states, given overall 32 percent mortality rate and 6 percent mortality rate directly attributed to complications of AEN, early recognition and prompt medical management is imperative for favorable outcomes. Treatment focuses on supportive therapy: namely resuscitation, PPIs, sucralfate, keeping the patient NPO and avoiding nasogastric tube to avoid esophageal perforation.



[2425] **Figure 1.** Proximal Acute Esophageal Necrosis.

S2426

**A Rare Case of Esophageal Verrucous Carcinoma**

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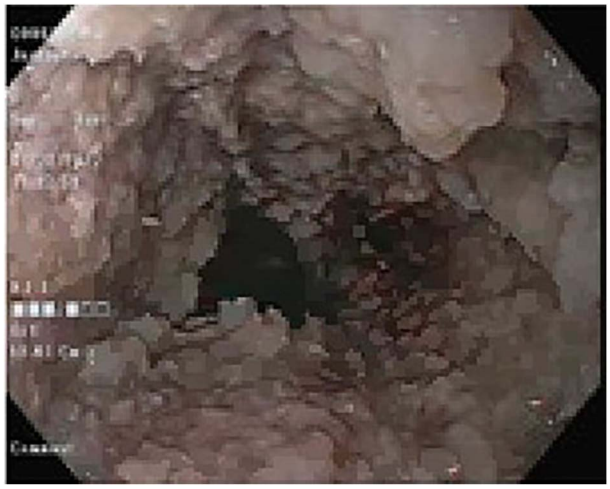
**Introduction:** Verrucous carcinomas are a slow-growing variant of squamous cell carcinoma that can rarely be seen in the esophagus. Risk factors are thought to be chronic mucosal irritation, tobacco use, alcohol use, achalasia, and human papilloma virus. The disease can go undiagnosed as superficial biopsies are nonspecific and often deep mucosal biopsies are needed to make a true diagnosis. Here we have a case of a patient that had slow-growing verrucous carcinoma that took 3 upper endoscopies with biopsies to finally make the diagnosis.
**Case Description/Methods:** This is a case of an 81-year-old man who presented to clinic with a history of progressive dysphagia. He had a history of candida esophagitis and strictures for which he had been dilated in the past. He underwent repeat upper endoscopy that revealed esophageal mucosa with fungating lesions and white exudate. Due to a recent history of progressive dysphagia with weight loss, jumbo forceps and bite-on-bite biopsies were taken for deeper examination. The biopsies revealed verrucous carcinoma (**Figure**). He underwent imaging and paratracheal lymph node biopsy that also returned positive for squamous cell carcinoma. He underwent chemotherapy and radiation with great improvement in his symptoms.
**Discussion:** Verrucous carcinoma is a rare form of slow-growing squamous cell carcinoma that is rarely seen in the esophagus, and when it is, can spread locally. It is often treated with either surgery or chemo-radiation. It is important to recall this diagnosis as it can be difficult to diagnose on superficial biopsies but rather takes clinical suspicion based on endoscopic findings to recognize and evaluate with deeper mucosal biopsies or endoscopic ultrasonography with biopsy. Oftentimes, patients will require percutaneous gastrostomy tubes placed for feedings while they undergo therapy for the verrucous carcinoma.



[2426] **Figure 1.** Hyperkeratotic appearing esophageal mucosa consistent with verrucous carcinoma.

S2427

**A Rare Case of Malignant Transformation of Esophageal Squamous Papillomatosis**

*Adam Spandorfer, MD, Vaidehi Avadhani, MD, Manjusha Das, MD, Jason M. Brown, MD.*
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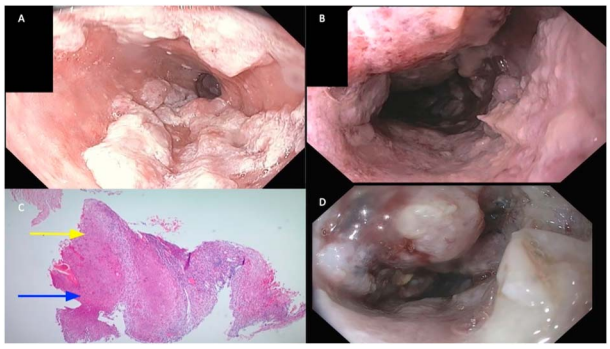
**Introduction:** Development of extensive esophageal papillomas, also known as esophageal squamous papillomatosis (ESP), is extremely rare. Because there is a growing association between ESP and the development of squamous cell carcinoma (SCC), this disease can have significant clinical implications. We describe a case of ESP that develops into SCC.
**Case Description/Methods:** A 68-year-old man presented to clinic with a one-month history of progressive dysphagia and 10 lb weight loss. He has a history of chronic active *Helicobater pylori*-associated gastritis, invasive sigmoid colon adenocarcinoma status post resection without recurrence, tobacco use disorder and chronic esophagitis. He was seen 2 years prior for dysphagia to solids with endoscopy demonstrating numerous lesions (Figure 1, A) and benign inflammatory histology. Several months before his current presentation he had an upper endoscopy which revealed worsening of these lesions (Figure 1, B). Biopsies showed squamous epithelium with acanthosis, superficial hyperkeratosis, parakeratosis (Figure 1, C, yellow arrow) and cores of fibrovascular connective tissue (Figure 1, C, blue arrow) that were negative for Human Papilloma virus (HPV) or malignancy, overall suggestive of ESP. Subsequent upper endoscopy revealed a large, ulcerated esophageal mass (Figure 1, D). Biopsy of the mass demonstrated squamous cell carcinoma.
**Discussion:** Esophageal squamous papillomatosis is an extremely rare but important endoscopic finding. Grossly, they are described as small, pearly, wart-like projections, and histologically, as finger-like projections of hyperplastic squamous epithelium with a core of fibrovascular connective tissue. Pathogenesis is thought to be due to chronic inflammation from either long-standing acid reflux, pathologies associated with chronic esophagitis, HPV infection, or recurrent exposure to irritants. Surveillance guidelines and management of ESP have not been established due to its rarity, and little is known about the natural history of ESP. There are an increasing number of case reports of its association with the development of SCC as seen in our patient. It is important that providers identify esophageal squamous papillomatosis early and are aware of the risk of malignant transformation to squamous cell carcinoma.




[2427] **Figure 1.** Endoscopic and histologic findings of esophageal papillomatosis (panel A, B, and C) and squamous cell carcinoma (panel D).

S2428

### A Rare Case of Diffuse Esophageal Ulceration in a Patient With a Recent Viral Illness

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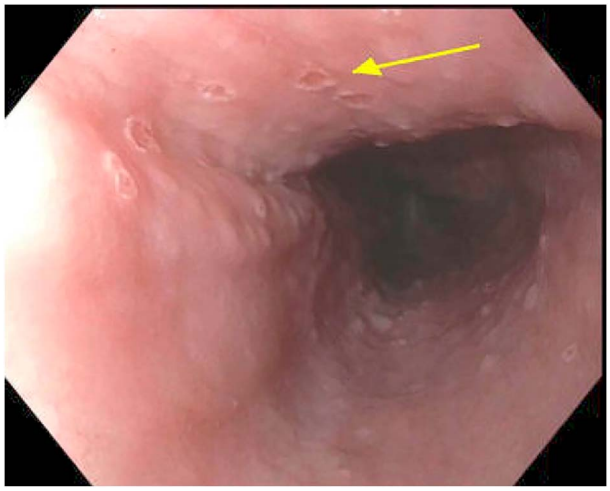
**Introduction:** Pemphigus vulgarism (PV) is an autoimmune bullous disorder involving skin and mucous membranes. PV is rare, especially when it primarily involves the oropharynx and esophagus. Per the literature review, it is an under-diagnosed etiology of odynophagia and can progress to severe ulceration, upper gastrointestinal bleeding, strictures, and increased morbidity and mortality. We present a case of PV manifesting with oropharyngeal and esophageal disease.
**Case Description/Methods:** A 47-year-old male with a history of hypertension presented to the emergency department (ED) for painful swallowing for the past 3 weeks. The patient had previously presented to a dentist for a tooth abscess and was prescribed amoxicillin 1,000 milligrams (mg) or 20-milliliter solution every 12 hours for 8 days. The patient then presented to his primary care doctor for oral ulcers and was prescribed acyclovir 400 mg 3 times a day for 5 days empirically for herpes simplex virus infection. In the ED, the patient was afebrile and hemodynamically stable. The exam was notable for multiple friable erythematous macules on the buccal mucosa and oropharynx. Labs were significant for mild leukocytosis and acute kidney injury. Microbiology was significant for influenza B. Imaging with CT soft tissue neck without contrast was negative for abnormalities. Upper endoscopy was significant for diffuse ulceration throughout the larynx and esophagus with clean-based circular ulcers, gastritis, and duodenitis. Pathology was significant for acantholysis and *Helicobacter pylori* but negative for HSV and CMV. Dermatology recommended repeat biopsy with Michel's solution for examination under direct immunofluorescence. Repeat biopsies were positive for granular immunoglobulin G at interspinous processes and serology was positive for desmoglein 1 and 3. The patient was initiated on prednisone and Rituxan for PV with esophageal involvement (**Figure**).
**Discussion:** PV with primarily esophageal involvement is exceedingly rare and is associated with increased morbidity and mortality due to delay in diagnosis. It can affect patients that are not immunocompromised, as in this case. Despite multiple evaluations by providers and initial endoscopy, a second endoscopy and consultation with dermatology were required to confirm the diagnosis. This case helps to illustrate the importance of considering PV in the differential for patients with acute odynophagia with ulceration after a viral illness and the use of Michel's solution for examination under immunofluorescence.



[2428] **Figure 1.** Well circumscribed clean-based ulcers throughout the esophagus, measuring 2-3 mm with slightly raised borders.

S2429

### A Rare Case of Esophageal Adenocarcinoma Presenting as an Isolated Brain Lesion 6 Years After Treatment

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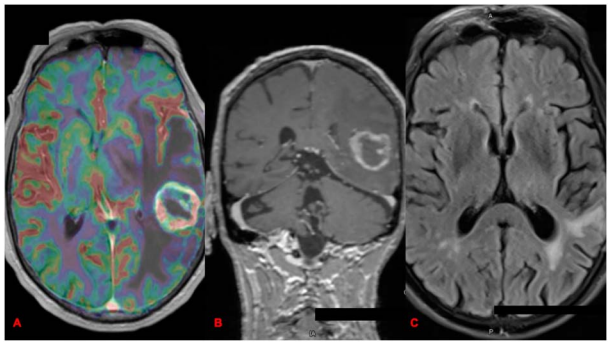
**Introduction:** Esophageal adenocarcinomas account for ~1% of all cancers in the U.S. The prevalence of central nervous system (CNS) metastases associated with these cancers is around 1.8%. Response rates to standard of care are poor and prognosis is dismal. Often CNS involvement is associated with widely metastatic disease, herein, we present a case of a solitary brain lesion from esophageal adenocarcinoma at first recurrence.
**Case Description/Methods:** A 72-year-old-male with a 40 pack-year smoking presented with progressive dysphagia in March 2015. Contrast enhanced CT of the chest, abdomen and pelvis revealed a constricting mass at the esophagogastric junction that led to endoscopy guided biopsy revealing esophageal adenocarcinoma. He underwent neoadjuvant chemotherapy (cisplatin/5-fluorouracil) and radiation therapy followed by esophagogastrectomy in July of 2015 where diagnosis of stage IIIBT3N2cM0 esophageal adenocarcinoma was confirmed. Clinical and imaging surveillance for 6 years had not shown any progression of cancer. However, in June of 2021, he presented with altered mental status that led to contrast enhanced brain MRI showing a T2 hyperintense left parietal lobe mass with an 8 mm midline shift and surrounding vasogenic edema (**Figure**). FDG PET-CT head to toes did not show extracranial recurrence. He underwent left posterior temporal craniotomy for resection of tumor confirming the diagnosis of metastatic esophageal adenocarcinoma. IHC showed positivity of cytokeratin7, CDX2 and SATB2. Whole exome sequencing revealed mutations in ARID1A, FH, and TP53 with variants of unknown significance of ATM and CDH. Tumor displayed proficient mismatch repair protein, a combined positive score of 15 for PD-L1, and negative Her2/Neu. Further sequencing showed tumor mutational burden of 4, no genomic loss of heterozygosity, and stable microsatellite repeats. He received gamma-knife radiation to the solitary left temporal lobe lesion. Patient continues to maintain his quality of life with no evidence of clinical and radiographic recurrence.
**Discussion:** Isolated brain metastasis from esophageal adenocarcinoma is a very rare phenomenon. Limited data suggests that the survival of patients with isolated CNS lesions is $< 1$ year, however, achieving pathologic complete response after neoadjuvant chemo-radiation therapy has shown to improve survival. These esophageal cancers with isolated CNS metastases may share a unique genomic profile and more research is required to understand their alterations.




[2429] **Figure 1.** MRI of the brain. 1A- Pre-op axial OLEA of L. temporal lobe tumor (06/2021), 1B- Pre-op axial coronal T1 (06/2021), 1C- Follow-up T2 MRI.

**S2430**

**A Rare Case of Acute Esophageal Necrosis Precipitated by *Klebsiella pneumoniae***

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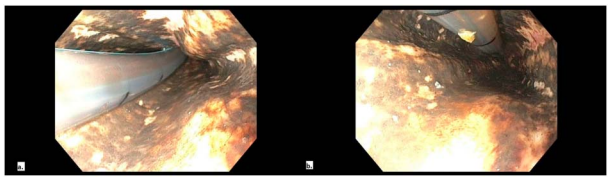
*The Wright Center for GME, Scranton, PA.*

**Introduction:** Acute esophageal necrosis (AEN) is a rare ischemic condition diagnosed on endoscopy (EGD) by black appearance of the esophagus. Its pathophysiology is multifactorial and often associated with underlying comorbidities such as age, malnourishment, ischemia and debilitated state. Early identification, appropriate management are associated with better outcomes. Treatment is primarily conservative therapy. In our case report we will discuss a patient who was found to have AEN in the presence of Klebsiella pneumonia.
**Case Description/Methods:** 88-year-old active male with no significant medical history presented to the ED with syncope and neck pain. He was tachycardic, found to have bilateral cervical C6 spine fracture and epidural hematoma at the level of C2-C3 with posterior CSF leak on imaging. Xray showed airspace opacities in the left lower lobe. Shortly afterwards he developed hyponatremia, altered mental status and coffee ground emesis. He underwent emergent EGD with biopsy which revealed multiple plaques in the proximal esophagus 25 cm from the gastroesophageal junction with circumferential "black esophagus" (Figure 1). Biopsy revealed necrotic esophageal squamous mucosa with associated acute inflammation and necrotic debri. He was started on pantoprazole and fluid infusion. Bronchial aspirate was positive for Klebsiella pneumoniae and he was treated with ceftriaxone. He was found to have worsening posterior epidural hematoma with severe spinal cord and canal compression and underwent emergent laminectomy however his condition continued to deteriorate and he eventually passed away.
**Discussion:** AEN must be considered as a cause of upper gastrointestinal bleeding especially in patients with the risk factors of advanced vasculopathy including male gender, advanced age, diabetes mellitus, dyslipidemia and ischemic cardiomyopathy and a debilitated physical state consisting of malnutrition and/or recent history of malignancy. The pathophysiology is likely due to hypoperfusion, followed by weakened protective barrier and gastric reflux. If there is any underlying infection, there is a high possibility of dissemination of the infection to precipitate AEN. Hence, it must be recognized early, with aggressive management to improve clinical outcomes. Management entails treating the underlying pathology, early PPI infusion and maintaining hydration. NG tube should be avoided due to risk of perforation and surgical intervention are reserved for complex AEN or complications.



[2430] **Figure 1.** Endoscopy images of lower third (a) and middle third (b) of the esophagus showing "black esophagus."

**S2431**

**A Rare Case of Esophageal Kaposi Sarcoma Causing Dysphagia**

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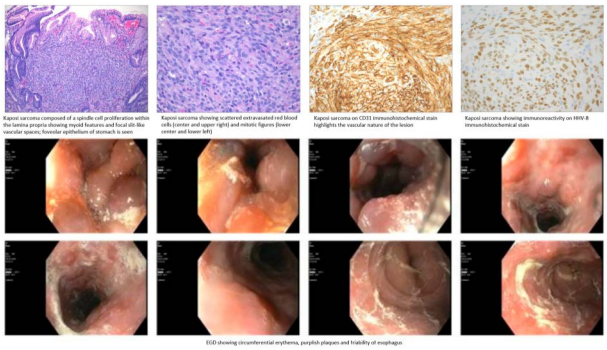
*University of Arkansas for Medical Sciences, Little Rock, AR.*

**Introduction:** Kaposi sarcoma (KS) is an angioproliferative disorder(low grade vascular tumor) caused by human herpesvirus-8 in immunocompromised patients. The disease is multifocal, ranging from indolent skin manifestations to extensive visceral involvement. We are describing a rare case of esophageal KS causing dysphagia.
**Case Description/Methods:** A 57-year-old bisexual male with a medical history of hypertension, syphilis and recent diagnosis of HIV (untreated) admitted for hypotension, tachycardia, nausea, vomiting,hemoptysis and dysphagia to soilds. He was noted to have oropharyngeal lesions and biopsy of the lesions were obtained. An EGD performed for evaluation of dysphagia (denied odynophagia) showed circumferential erythema, purplish plaques and friability of the esophagus. Biopsies were obtained from esophagus. Pathology from tongue, gingiva, proximal and distal esophagus returned positive for Kaposi sarcoma with positive staining for HHV-8 and CD31. CD4 count was 31/uL and HSV8 PCR showed 1,000,000 copies. Patient was treated with Bictegravir, emtricitabine and tenofovir alafenamide. Hematology was consulted for potential pulmonary KS and patient was started on liposomal doxorubicin. He developed Ventricular fibrillation and cardiac arrest. He was revived and intubated. Patient improved initially but developed neutropenic fever. Infectious disease treated him with empirical antibiotics and antivirals. Patient further developed Castleman's disease, respiratory failure, acute kidney failure requiring CRRT, and altered mental status. Despite aggressive measures patient did not survive (**Figure**).
**Discussion:** AIDS-related KS varies in clinical progression and occurs in 20% of patients with AIDS. The CD4 count will typically be less than 150 cells per cubic millimeter with a high viral load ranging from greater than 10,000 copies per millimeter. Gastrointestinal manifestation is the most common extra-cutaneous site of KS in AIDS-related cases though rarely does it occur without cutaneous involvement. The majority of the patients are asymptomatic, roughly 75%. Symptomatic patients present with nonspecific findings such as abdominal discomfort, cramps, nausea, vomiting, diarrhea, and upper or lower GI bleed. KS lesions causing dysphagia are extremely rare. The mainstay of therapy is combined antiretroviral therapy (ART). Local intralesional chemotherapy can be utilized to manage limited lesions. Systemic therapy with liposomal anthracyclines is recommended for patients with advanced or rapidly progressive disease.



[2431] **Figure 1.** Kaposi Sarcoma Lesions.

**S2432**

**A Rare Case of Dysphagia Megalatriensis Presenting With Acute Decompensated Heart Failure**

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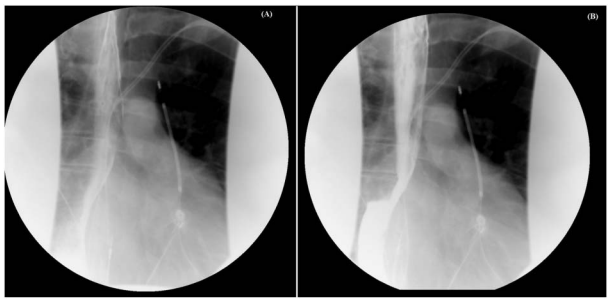
*[1]Albany Medical Center, Albany, NY; [2]King Edward Medical University, Albany, NY.*

**Introduction:** Dysphagia is a common complaint that can manifest in a large variety of conditions. Dysphagia megalatriensis is a rare cause of esophageal dysphagia and is caused by extrinsic compression of the esophagus by a dilated left atrium. This results in solid food dysphagia in the mid-distal esophagus. Symptoms can be minimal to debilitating.
**Case Description/Methods:** A 52-year-old male with a past medical history of dilated cardiomyopathy and heart failure with ejection fraction less than 25% presented with acute decompensated heart failure and NSTEMI. The patient reported that for the past few months he had a sensation of solid food getting stuck in his mid-chest. He denied any alcohol or tobacco use, hematemesis, nausea, or vomiting. On physical examination, the patient had no abdominal pain or distention. He denied any weight loss or family history of cancers. Vital signs were stable on admission and laboratory testing revealed an elevated troponin of 0.23 ng/mL, creatinine of 1.43 mg/dL, and INR of 2.4. He was started on 40 mg pantoprazole twice a day, upper GI series was ordered which showed smoothly marginated extrinsic compression on the esophagus related to cardiomegaly. The narrowing was positional and most pronounced in the antero-posterior (AP) plane. This was worse during atrial dilation in the cardiac cycle and resulted in delayed emptying of the upper and middle esophagus. Figures A and B show extrinsic compression in diastole and systole respectively. The patient was advised to have small frequent meals and aggressive diuretic therapy was performed. These measures resulted in the improvement of his symptoms.
**Discussion:** This case highlights the importance of the recognition of dysphagia secondary to cardiomegaly. Patients typically have mitral valve stenosis (cause of atrial dilation) which was not present in our case. Although rare, dysphagia megalatriensis can be an early sign of impending decompensation in heart failure patients [1]. In our case, the patient's dysphagia developed weeks before decompensated heart failure. Hence this condition should be identified sooner than later. Barium esophagram yields the best diagnostic results while diet and diuretics are the cornerstones of therapy. The most severe complication is fatal atrioesophageal fistula formation.




[2432] **Figure 1.** A: Mid and distal extrinsic esophageal compression during cardiac diastole. B: Mild resolution of extrinsic compression during cardiac systole.

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1. Matta A, Elenizi K, Carrié D, Roncalli J. Dysphagia as an early sign of cardiac decompensation in elderly: case report. Eur Heart J Case Rep. 2020 Jun 17;4(4):1-5.

**S2433**

**A Rare Case of Esophageal Perforation in Liposarcoma After Cryoablation**

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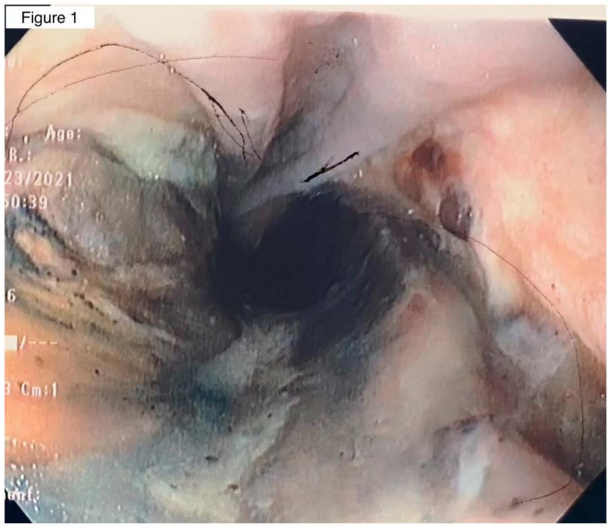
*[1]Yale-Waterbury Internal Medicine Residency Program, Waterbury, CT; [2]Waterbury Hospital, Waterbury, CT; [3]Yale New Haven Hospital, New Haven, CT.*

**Introduction:** Liposarcoma is a locally aggressive solid tumor where mainstay treatments typically include surgical resection or debulking. Recently, cryoablation in liposarcomas has been utilized as a safe and well-studied modality for limiting tumor growth and subsequent symptoms of mass effect. Cryoablation is considered palliative for solid tumors and can prolong patient survival in advanced or recurrent cancer. Cryoablation can cause a variety of adverse events, including fevers, skin frostbite, and local nerve injury secondary to localized edema. Our case presentation showcases the only reported case demonstrating esophageal perforation following cryoablation thought to be related to local nerve injury.
**Case Description/Methods:** Our patient is a 64-year-old male with an extensive mediastinal sclerosing liposarcoma who underwent debulking surgery in 2018 with subsequent tumor enlargement. Given the precarious location of the enlarging mediastinal mass with major compression of the carina, mainstem bronchi, left atrium, and encasement of the esophagus and descending aorta, a complicated resection was deferred, and instead, extensive palliative treatment modalities were explored where cryoablation to the subcarinal area was selected as being the safest and most effective. He initially did well post-procedure, but 13 days later, developed worsening stridor which progressed to hematemesis, prompting the patient to present to the ED. He underwent emergent bronchoscopy demonstrating external carinal compression of mainstem bronchi and EGD confirmed a large contained full-thickness esophageal perforation (Figure 1) He did not develop of sepsis or mediastinitis. For additional airway protection, he underwent a Y-stent with eventual improvement and healing of the esophagus and resumed a normal oral diet.
**Discussion:** The large mediastinal liposarcoma extending to the carina caused several compressive symptoms, including difficulty breathing and stridor. Although palliative CT-guided cryoablation accurately targeted the carina, while avoiding large, important structures, the patient developed severe coughing. This was believed to be related to local edema affecting the vagus nerve which may have led to a Mallory Weiss tear, resulting in hematemesis. Although local nerve injury is a known possible side effect of cryoablation, our case is one of the first to highlight such severe injury to the extent of esophageal perforation.



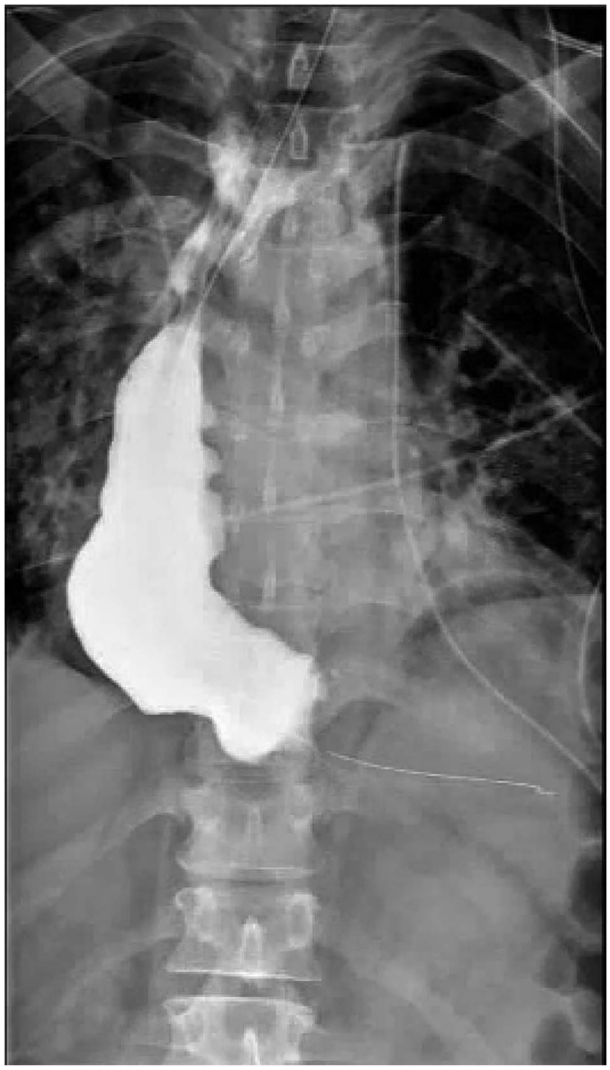

[2433] **Figure 1.** EGD revealing contained full-thickness esophageal perforation.

**S2434**

**A Rare Case of Epiphrenic Diverticulum as a Complication of Esophageal Motility Disorders**

*Alaa Taha, MD[1], Ranim Chamseddin, MD[2], Vatsal Khanna, MD[2], Tripti Nagar, MD[2], Hanan Hannoodee, MD[2].*
*[1]Wayne State University School of Medicine, Rochester, MI; [2]Wayne State University, Rochester, MI.*

**Introduction:** Esophageal diverticula are a rare finding with an estimated incidence of 1:500 000 per year and a prevalence of 0.015–2%. It is usually associated with esophageal motility disorders, particularly Achalasia. The suspected mechanism is secondary to increased intraluminal pressure from the primary esophageal motility disorder, which leads to herniation of the mucosa and submucosa through the muscular layer. It is usually asymptomatic but can present with dysphagia, regurgitation, nausea, vomiting, aspiration, heartburn, weight loss, and retrosternal pain. Here we offer a case of a large esophageal diverticulum in a 52-year-old male who has been having symptoms for years. Given the symptomatic nature of his diverticulum, he underwent surgical repair.
**Case Description/Methods:** A 52-year-old patient with no significant past medical history presented with complaints of dysphagia that was worsening over several years, associated with halitosis and food regurgitation. The patient was having a routine screening colonoscopy when he had an episode of coffee-ground emesis. He subsequently underwent an esophagogastroduodenoscopy which revealed a large epiphrenic esophageal diverticulum. He then had an esophagogram and Computer Tomography (CT) of chest and abdomen, which confirmed the presence of the diverticulum and significant narrowing of the GE junction. Esophageal manometry was not pursued, given the difficulty of placing the probe and the risk of perforation. He subsequently underwent left thoracotomy with resection of the large esophageal diverticulum, long myotomy (11 cm), and Besley fundoplication. Esophagram following surgery was negative for any leak (**Figure**). He was started on a clear liquid diet that was later advanced to full liquid, with the patient tolerating it well. He was subsequently discharged home.
**Discussion:** Esophageal diverticula are rare findings with an estimated incidence of 1:500 000 per year and a prevalence of 0.015–2%. Most esophageal diverticula are acquired and present in older adults. Surgical repair is associated with mortality of 0 to 9% and morbidity of 20%. This case serves as a reminder to keep a broad differential diagnosis when approaching a patient with symptoms of a motility disorder. Early detection can provide timely management and prevent further complications.



[2434] **Figure 1.** Esophagram after surgical repair of epiphrenic esophageal diverticulum.

**S2435**

**A Rare Case of Plummer-Vinson Syndrome Treated With Pneumatic Dilations and Iron Supplementation**

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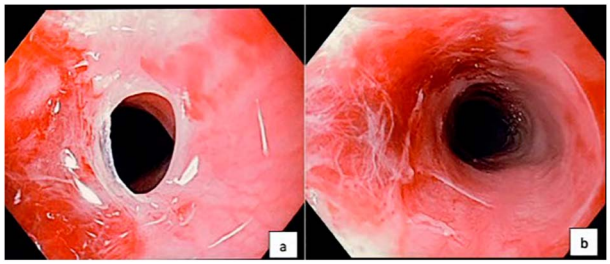
[1]Jefferson Health Northeast, Philadelphia, PA; [2]Trinity Health Mid Atlantic, Darby, PA; [3]Jefferson Health New Jersey, Sewell, NJ.

**Introduction:** Plummer-Vinson Syndrome (PVS), also known as Paterson-Kelly syndrome, presents with a classic triad of dysphagia, iron-deficiency anemia and esophageal webs. Although this triad of disease is increasingly less prevalent, early detection and intervention are imperative to reduce mortality given increased risk of malignant transformation.[1]
**Case Description/Methods:** A 22-year-old female with a history of iron deficiency anemia presented to the emergency department with syncope in the setting of palpitations and dyspnea. The patient endorsed alarm symptoms including hematochezia over the past 6 weeks, 20-lb unintentional weight loss and dysphagia. Initial labs were remarkable for a hemoglobin of 11.1 g/dL, and microcytic indices, with an MCV of 72.4 fL. The patient underwent esophagogastroduodenoscopy (EGD) to investigate the etiology of iron deficiency anemia. EGD revealed a prominent esophageal web (Figure A) in the proximal esophagus with associated erythema and mucosal friability. Significant narrowing impeded safe passage of the endoscope and thus serial pneumatic dilations to 10 mm were performed. Following pneumatic dilation, the endoscope was successfully advanced beyond the web (Figure B) to the gastroesophageal junction. The patient tolerated the procedure well and had immediate resolution of dysphagia following EGD with pneumatic dilation. She was then continued on iron supplementation as previously prescribed at discharge.
**Discussion:** Plummer-Vinson Syndrome comprises the pathological triad of dysphagia, esophageal web, and iron deficiency anemia. Other signs and symptoms can include glossitis, angular cheilitis, and koilonychia. The decreased incidence of this syndrome parallels the decreased prevalence of iron deficiency in the developed world. Iron deficiency induces an iron-dependent enzyme dysfunction which causes oxidative stress, mucosal DNA damage and ultimately potentiates the formation of esophageal webs. [2] Without intervention, patients with this syndrome may develop absolute dysphagia, aspiration pneumonia and other complications. Due to increased risk of malignancy, surveillance EGDs are necessary to screen for esophageal carcinoma in patients with PVS.




[2435] **Figure 1.** Esophageal Web pre dilation (a) and post dilation (b).

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**S2436**

**A Rare Case of a Large Esophageal Gastrointestinal Stromal Tumor**

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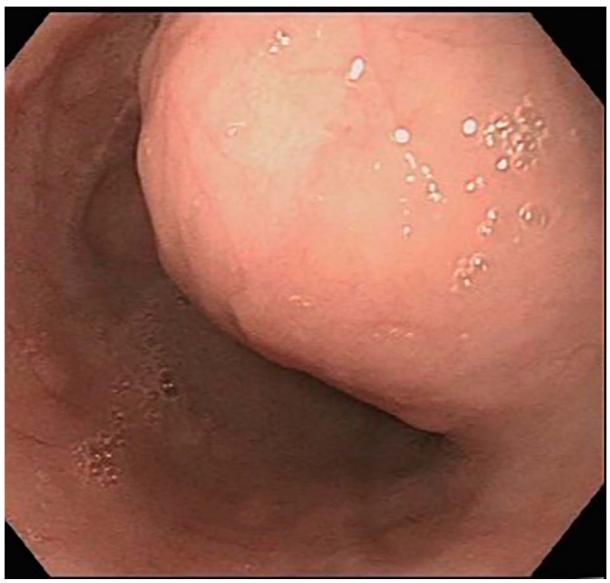
**Introduction:** Gastrointestinal stromal tumors (GISTs) are rare mesenchymal neoplasms, accounting for approximately 1 to 2 percent of primary gastrointestinal (GI) cancers. Presenting symptoms include abdominal pain, nausea, vomiting, early satiety, bleeding, and dysphagia, but vary depending on primary tumor location. Most tumors are discovered incidentally with the majority found in the stomach (40 to 60 percent) followed by small bowel (25-30 percent), colon and rectum (5-15 percent). We present a case of an elderly patient diagnosed with esophageal GIST after he was found to have an incidental esophageal mass on imaging.
**Case Description/Methods:** 80-year-old male with PMH of HTN, HLD, DM2, CKD Stage III, COPD, former tobacco user was referred for an upper endoscopy for evaluation of an incidental finding on CT scan. CT demonstrated a mass arising from the distal esophagus, measuring 6.5 x 7.5 cm. Patient denied any dysphagia, abdominal pain, nausea, vomiting, weight loss, hematemesis, or melena. Endoscopy showed submucosal mass in the distal esophagus (**Figure**). Biopsy was inconclusive and subsequent EUS-guided fine needle biopsy was performed. Upper endoscopic ultrasound (EUS) showed an intramural lesion in the distal esophagus arising from the muscularis propria. Pathology results confirmed GIST, positive for CD117 and DOG-1. Further testing revealed a mutation in the KIT gene exon 11. Patient is currently undergoing neoadjuvant therapy with imatinib mesylate with anticipated surgical resection.
**Discussion:** This is a rare case of a large esophageal GIST. Most mesenchymal esophageal tumors are leiomyomas, and GIST only accounts for 0.7% of them. EUS with fine need aspiration biopsy is the most accurate method for diagnosis and definite diagnosis is made histologically. Majority of GISTs, expresses the CD117 antigen, a KIT receptor tyrosine kinase. Another commonly found mutation is the platelet derived growth factor (PDGFRA) tyrosine kinase gene. Tumor size and mitotic rate are used for prognostication, with increased malignancy risk seen in tumor size $>$ 3 cm with irregular margins. Tumor location is also a key factor in risk stratification; non gastric primary location has higher rates of metastases. Liver and peritoneum are the most common sites of metastatic GISTs. Treatment focuses on surgical resection and neoadjuvant therapy with imatinib. Given significant implication for its management, esophageal GIST should be considered in differential diagnosis and distinguished from more common esophageal leiomyomas.



[2436] **Figure 1.** Submucosal mass in distal esophagus.

**S2437**

**A Rare Presentation of Esophageal Squamous Cell Carcinoma Presenting as a Paraneoplastic Syndrome**

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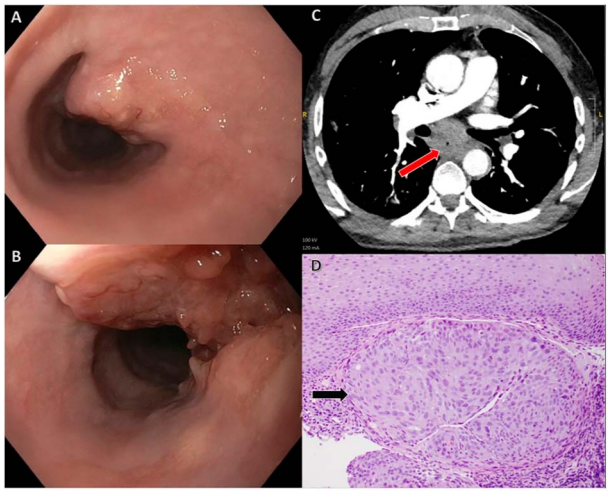
**Introduction:** Lambert Eaton myasthenic syndrome (LEMS) is a rare disease of the neuromuscular junction caused by formation of antibodies against voltage-gated calcium channels leading to a constellation of symptoms including muscle weakness, paresthesia, and autonomic dysfunction. LEMS can present as either a paraneoplastic condition or secondary to an autoimmune disorder. Research shows that 60% of

cases have an associated malignancy of which more than half are small cell lung cancer. Although LEMS has been associated with other malignancies, no prior cases have been reported with esophageal malignancy. We present a patient with symptoms of LEMS due to esophageal squamous cell carcinoma (SCC).

**Case Description/Methods:** A 59-year-old male with a history of GERD and hyperlipidemia presented with bilateral paresthesia and progressive weakness of his distal extremities for approximately one month, as well as new onset shortness of breath and a left-sided facial droop. After CVA was ruled out, he was admitted for possible Miller-Fischer variant of Guillain-Barre Syndrome and was started on treatment with IVIG. However, a CTA of the chest obtained on presentation demonstrated short-segment wall thickening of the mid-esophagus suggestive of esophagitis or possible underlying mass. Based on this finding, a paraneoplastic panel was ordered, and the patient underwent endoscopic evaluation. EGD revealed a single fungating ulcerated mass in the mid-esophagus covering one-third of the circumference. Biopsies demonstrated moderately differentiated squamous cell carcinoma (**Figure**). Results of the paraneoplastic panel were positive for P/Q-type calcium channel binding antibody. These findings indicated that the patient's symptoms were due to a paraneoplastic condition consistent with LEMS in the context of a newly diagnosed esophageal SCC.

**Discussion:** An extensive review of the literature found no previously reported cases of esophageal SCC associated with LEMS. A comprehensive evaluation of our patient found no other synchronous malignancies that would account for the paraneoplastic process. Although exceedingly rare, this case indicates a potential correlation between esophageal squamous cell carcinoma and P/Q-type VGCC antibodies resulting in LEMS. Current research suggests that up to 80% of patients present with paraneoplastic symptoms prior to any indication of cancer. Therefore, early recognition of possible paraneoplastic syndrome in patients is paramount to facilitate a timely evaluation for underlying malignancy.




[2437] **Figure 1.** Upper endoscopy demonstrates single fungating ulcerated mass in mid-esophagus (A,B). CT imaging shows thickening of mid esophagus concerning for malignancy (arrow) (C). Esophageal biopsy significant for benign squamous mucosa (top) with underlying squamous cell carcinoma (arrow) (20X, H&E) (D).

**S2438**

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**A Rare Cause of Esophageal Varices**

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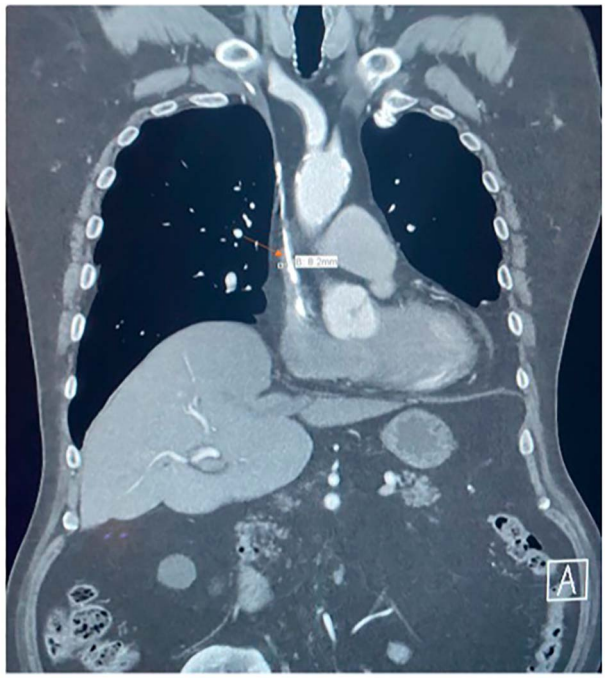
**Introduction:** Esophageal varices (EV) are classified as downhill or uphill varices. Uphill varices are common and found in the distal esophagus that develop secondary to portal hypertension. Downhill esophageal varices (DEV) are rare and found in the proximal and mid esophagus and most commonly develop from obstructed venous blood flow in the superior vena cava (SVC).

**Case Description/Methods:** A 74-year-old White male with past medical history of ESRD who was on hemodialysis for 5 years before a renal transplant was performed, COPD Gold Stage II, and obstructive sleep apnea presented to clinic with weight loss and dysphagia for the past year. The patient had no history of liver disease, physical examination was unremarkable, and his labs were normal. An esophagogastroduodenoscopy (EGD) revealed one large non-bleeding esophageal varix (EV) in the mid-esophagus and whitish plaques later confirmed by pathology to be Candida esophagitis. The patient was placed on IV micafungin and his dysphagia resolved. A CT Abdomen showed a normal liver and spleen. To further assess the DEV, a CT angiogram of the chest was obtained and revealed significant narrowing of SVC. (**Figure**). To evaluate the cause of SVC stenosis, an echocardiogram was ordered which revealed moderate to severe dilation of the right ventricle and evidence of pulmonary hypertension deemed to be likely secondary to his COPD and OSA. Of note, the patient had a prior chronic hemodialysis (HD) indwelling catheter in his SVC. After discussion with cardiology and CT surgery, the patient was referred to cardiology for an SVC venogram and right heart catheterization to measure pulmonary pressures.

**Discussion:** DEV are formed when there is an obstruction in the SVC causing retrograde blood flow into the right atrium through collateral channels. The proximal and mid esophageal veins drain into the collaterals and the increased pressure result in DEV. The most common etiology is thrombosis of the SVC, but DEV can also be caused by severe pulmonary hypertension, thyroid tumors, and complications with HD catheters. In our case, the patient's prior chronic HD catheter likely caused narrowing of the SVC lumen, along with long-standing pulmonary hypertension. Currently, there are no guidelines for screening or management of DEV. Physicians should be aware of unusual causes of varices and the management of DEV involves a multidisciplinary approach to treat the underlying cause of the SVC obstruction.




[2438] **Figure 1.** Significant narrowing of SVC (red arrow) on CT chest.

**S2439**

**A Rare Entity: Esophageal Dieulafoy Lesion**

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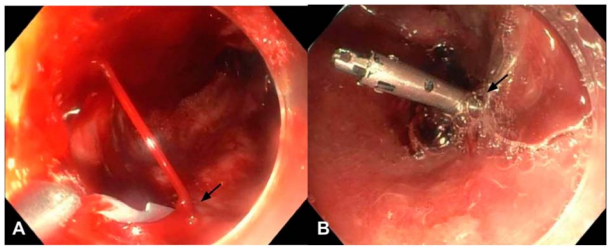
**Introduction:** Dieulafoy lesions (DL) consist of dilated aberrant submucosal arteries which may potentially lead to life-threatening gastrointestinal (GI) bleeding upon erosion of overlying mucosa. They are most commonly located in the lesser curvature of the stomach followed by duodenum and colon. The esophagus is an exceedingly rare location for DL. We report a hemodynamically unstable patient with distal esophageal DL who presented with massive hematemesis.
**Case Description/Methods:** A 57-year-old man was brought to the hospital with massive hematemesis and unresponsiveness requiring intubation in the field. His medical history was significant for alcohol-induced cirrhosis decompensated with ascites. Previous upper endoscopy revealed LA class D esophagitis but no evidence of varices. Upon presentation, the patient was tachycardic to 130 bpm and hypotensive to 89/56 mmHg. Physical exam was remarkable for distended abdomen and positive fluid wave. Initial blood work showed hemoglobin of 4.3 g/dL and platelet of 36/uL. Nasogastric tube suction aspirated 2000 ml of coffee ground content and frank blood. The patient was emergently resuscitated with intravenous fluid and massive transfusion protocol. Subsequently, bedside esophagogastroduodenoscopy revealed active pulsatile bleeding from an exposed vessel in the distal portion of the esophagus (Figure A). Two hemostatic clips were successfully deployed and hemostasis was achieved (Figure B). The patient was then transferred to the critical care unit and maintained on proton pump inhibitor infusion. Hospital course was further complicated by hepatic encephalopathy and hepatorenal syndrome. Given MELD-Na score of 40, the patient was eventually transferred to an advanced center for liver transplantation.
**Discussion:** DL in the esophagus is an extremely rare entity with few cases published to date. Lesions are due to persistently dilated caliber of submucosal artery, as opposed to normal narrowing, as the vessel approaches overlying mucosa. Continuous arterial pulsation may damage the mucosa with a stream of arterial bleeding, as in our case. Upper endoscopy is the mainstay of diagnosis and treatment for DL. Band ligation, electrocautery or hemoclips can be deployed through endoscopy, as with our case. Due to the intermittent nature of bleeding, endoscopy is not always diagnostic and angiography with embolization is an alternative option. Surgery is always a last resort.




[2439] **Figure 1.** A) Active pulsatile bleeding from an exposed vessel in the distal portion of the esophagus. B) Hemostatic clip successfully deployed with complete hemostasis.

**S2440**

**A Rare Cause of Dysphagia: Esophageal Stricture Due to Lichen Planus**

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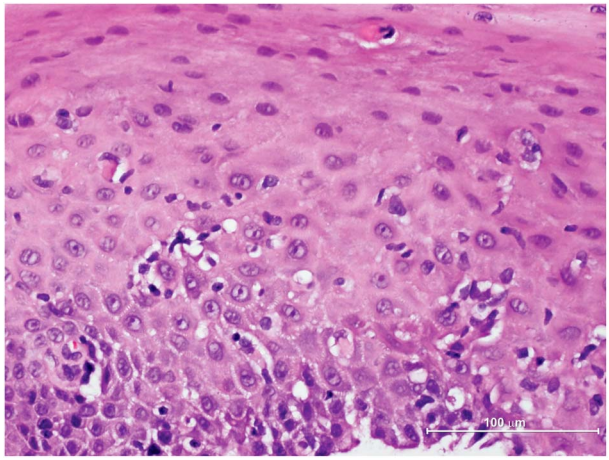
**Introduction:** Esophageal lichen planus (ELP) is an unusual and under-recognized cause of dysphagia. Timely diagnosis is crucial for effective treatment as ELP has been associated with increased risk for development of squamous cell carcinoma.
**Case Description/Methods:** A 72-year-old man presented with esophageal dysphagia to solids. Past medical history included oral lichen planus (LP) and osteoporosis with use of alendronate. He underwent endoscopy (EGD) which showed a proximal esophageal stricture for which balloon dilation ranging from 8 to 15 mm was done. Biopsy of the stricture showed esophageal mucosa with increased intraepithelial

eosinophils (**Figure**). The working diagnosis was peptic stricture with possible eosinophilic esophagitis (EoE). He was started on pantoprazole 40 mg twice daily. He underwent 2 more EGDs with dilation for recurrent stricture and on the 2nd EGD, biopsy of the stricture showed numerous intraepithelial lymphocytes and focal apoptotic squamous cells (Civatte bodies) compatible with ELP. Budesonide slurry 1 mg twice daily was instituted and an EGD 6 months later showed near resolution of the stricture. The patient no longer requires dilations or antiacid therapy and is asymptomatic currently. He had Candida esophagitis 1 month after initiation of budesonide which was treated with fluconazole.

**Discussion:** ELP is prevalent in patients with mucocutaneous forms of LP, most often oral LP. It is rarely seen in males with a case series by Franco et al describing the female to male ratio as 5:1. The endoscopic differential diagnosis for ELP should distinguish findings of esophagitis and stricture secondary to EoE, reflux esophagitis or esophageal Candidiasis. A low threshold should be kept for suspecting ELP in a patient with dysphagia and mucocutaneous LP. If histology is inconclusive but a high index of suspicion remains, particularly in patients with mucosal LP, repeating biopsies may be warranted for diagnosis as is highlighted in this case. A co-relation between longer duration of disease with severity of inflammation has been proposed with case series reporting strictures. Treatment includes serial dilations, topical and/or systemic steroids. Recurrence has been described despite topical steroid therapy with requirement of additional dilations. Successful use of immunomodulators such as cyclosporine, mycophenolate has been described. There is paucity of data regarding surveillance and optimal treatment for ELP, and thus further studies are needed for better understanding of this disease.




[2440] **Figure 1.** Esophageal biopsy showing esophageal squamous mucosa with basal cell hyperplasia, intraepithelial lymphocytes, and 2 apoptotic keratinocytes (Civatte or colloid bodies). Findings are compatible with esophageal lichen planus.

**S2441**

**A Rare Presentation of Esophageal Lichen Planus in a Patient With Collagenous Gastritis and Duodenitis: A Case Study**

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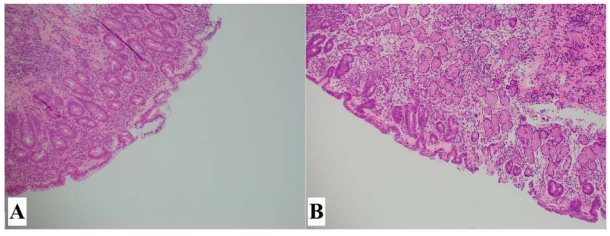
**Introduction:** Lichen planus (LP) is an idiopathic, immune-mediated, chronic inflammatory condition that can involve the skin, hair, nails and mucus membranes. Cutaneous and oral LP are relatively common and present with violaceous pruritic papules or white reticulated striae. Esophageal lichen planus (ELP) is an atypical and underdiagnosed cause of esophagitis and stricture, most common in middle to elderly aged women. We report a case of isolated ELP with concomitant collagenous gastritis (CG) and duodenitis (CD). This case supplements the literature on ELP and details an uncommon constellation of diseases that may support further evaluation in ELP patients to investigate the presence of other associated conditions.

**Case Description/Methods:** An 80-year-old White female was referred for further evaluation after having multiple endoscopies with dilation and a gastrostomy tube placement for a refractory esophageal stenosis that led to significant dysphagia, loss of appetite and a 30-pound weight loss. She denied altered bowel habits or skin lesions. Endoscopy revealed sloughing mucosa lining a narrowed esophagus with healthy mucosa in the distal third. The stomach and small bowel mucosa appeared atrophic and pale. Biopsies of the esophagus were suggestive of ELP and the duodenum and stomach biopsies revealed CG and CD. A swallowed budesonide slurry markedly improved her dysphagia and helped regain weight (**Figure**).

**Discussion:** There should be a high index of suspicion for ELP in patients with the correct demographics presenting with dysphagia, proton pump inhibitor (PPI) refractory esophagitis and stenosis. Endoscopic evaluation should be considered in LP patients to define disease extent. Additional evaluation and biopsies should also be considered if refractory symptoms or those consistent with another disease process are present. Our patient, in addition to ELP, was diagnosed with isolated CG and CD, a rarity when presenting without colonic involvement. Proximal collagenous gastroenteritides–which also show a slight female predominance and autoimmune association–are diagnosed endoscopically and histologically by subepithelial collagen deposition, nodular changes and inflammatory infiltrate.[1] The lack of literature and scant incidence of both conditions suggests that further analysis of ELP patients should be considered to determine if there is an association with CG or CD, as this may also shed light on their pathogenesis.




[2441] **Figure 1.** Histological findings of collagenous gastritis and duodenitis. (A) Duodenum depicting signs of villous atrophy and subepithelial collagen deposition with background chronic inflammation; (B) Gastric antrum depicting subepithelial collagen deposition with background chronic inflammation.

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**S2442**

**A Rare Finding of a Common Disorder**

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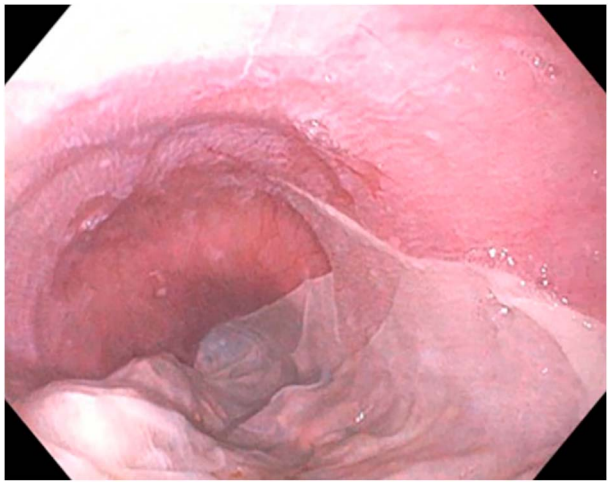
*[1]University of Illinois at Chicago, Chicago, IL; [2]University of Ilinois at Chicago, Chicago, IL.*

**Introduction:** Esophagitis dissecans superficialis (EDS) is a rare desquamative disorder of the esophagus, characterized by sloughing of the esophageal squamous mucosa. Most cases of EDS have been associated with autoimmune bullous dermatoses, medications like bisphosphonates, chemical irritants, iatrogenic injury, and celiac disease. To our knowledge, EDS has not previously been reported with achalasia.
**Case Description/Methods:** Our patient is a 42-year-old woman with Hashimoto's thyroiditis and untreated achalasia type II diagnosed on high-resolution manometry 4 years ago who presented to the hospital with dysphagia to both solids and liquids and oral intake intolerance for one week. She also endorsed weight loss, nausea, and vomiting clear secretions mixed with thick white cord-like material. Physical exam was remarkable for dry mucous membranes. Laboratory tests were notable for mild leukocytosis. Given her history of achalasia and progressive symptoms, esophagogastroduodenoscopy (EGD) was performed and showed large fragments of mucosal sloughing in the distal esophagus or "tissue paper esophagus" consistent with EDS and hypertonic lower esophageal sphincter (Figure 1). Pneumatic dilation (PD) to 30 mm was successfully performed with improvement in symptoms. She was continued on a daily proton pump inhibitor and was doing well on her 4-month follow-up.
**Discussion:** EDS can be an incidental finding on endoscopy or can present with a variety of symptoms, including dysphagia, odynophagia, coughing, or vomiting the sloughed esophageal mucosa like in our patient. The pathogenesis remains unclear. It might represent a mucosal reaction to various insults (iatrogenic, chemical, thermal, and immunological). Although EDS has been reported in association with certain dermatologic and gastrointestinal autoimmune diseases like celiac disease, it has not been reported in the setting of achalasia. Perhaps the possible autoimmune pathogenesis of achalasia related to antibodies against the enteric neurons contributes to its occurrence. Biopsies are recommended to rule out other etiologies such as candidiasis and squamous cell carcinoma (SCC). There is no specific treatment for EDS; a combination of acid suppression and discontinuation of the offending agent if present can result in healing. Prognosis is benign without lasting esophageal complications. In conclusion, endoscopists need to be aware of EDS in order not to mistake it for other conditions like esophagitis and SCC and to recognize its association with achalasia.



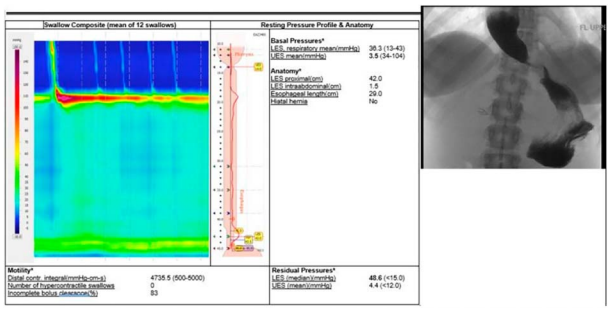
[2442] **Figure 1.** Sloughing of the esophageal mucosa described as "tissue paper esophagus" in esophagitis dissecans superficialis.

**S2443**

**Achalasia in a 16-Year-Old: Diagnostic Dilemma**

*Syed Musa Raza, MD[1], Daniyal Raza, MD[2], Nazar Hafiz, MD[1], Maryam Mubashir, MD[1], Shazia Rashid, MD[1], Anush Vasikaran, MD[3], Sudha Pandit, MD[1].*
*[1]Louisiana State University Health Sciences Center, Shreveport, LA; [2]Louisiana State University, Shreveport, LA; [3]Ochsner LSU Health Shreveport, Shreveport, LA.*

**Introduction:** Achalasia is a disorder, particularly rare in children, with an incidence of 0.11/100000 in the pediatric population. It is a motility disorder of the esophagus characterized by inadequate esophageal peristalsis and failure of the esophageal sphincter to relax. Symptoms include but are not limited to dysphagia, vomiting, and weight loss. Barium esophagogram and manometry are the tools used for diagnosis. Treatment options among children include laparoscopic Heller myotomy and peroral endoscopic myotomy.
**Case Description/Methods:** Our patient was a 16-year-old child who presented with persistent vomiting and weight loss for 6 to 8 months. The patient underwent an EGD at an outside hospital which was inconclusive. Initially, there were concerns about an eating disorder, but it was ruled out. The patient had episodes of hypokalemia and syncope, requiring admissions to the hospitals on numerous occasions. The patient had to be started on TPN for nutrition. During our hospital admission, the patient underwent an EGD, which showed dilated esophagus suspected of a motility disorder. Esophagram showed findings consistent with an early stage of achalasia (**Figure**). Subsequently patient had endoscopy-guided placement of a manometry probe, and she was diagnosed with Achalasia type 1. The patient underwent POEM, which resulted in significant improvement of her symptoms. Her electrolyte abnormalities resolved as she started to tolerate a diet without emesis.
**Discussion:** Achalasia is a rare disorder that usually presents between the age of 25 and 40. Establishing an early diagnosis in children can be challenging since more than half of the cases are diagnosed at an older age creating a diagnostic dilemma for pediatricians and gastroenterologists. Nonspecific symptoms can lead to a misdiagnosis posing significant health risks to the patient if left untreated. Our patient had 50 pounds of weight loss and was started on TPN for nutrition even before the diagnosis. Given the rarity of the case, it helps increase awareness among pediatricians and gastroenterologists to consider motility disorders early on, with esophageal manometry considered the gold standard for diagnosis.




[2443] **Figure 1.** Manometry and esophagram.

**S2444**

**Acute Esophageal Necrosis and Stricture Formation: A Case Report and Literature Review**

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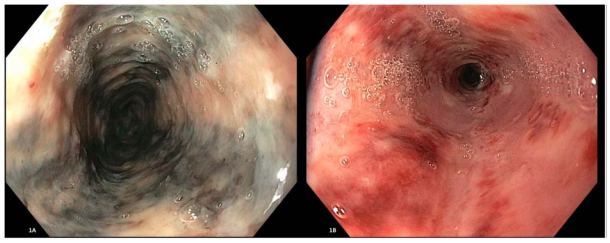
*[1]Yale New Haven Hospital, New Haven, CT; [2]Yale University/Yale-New Haven Hospital, New Haven, CT; [3]Yale University School of Medicine, New Haven, CT.*

**Introduction:** Acute esophageal necrosis, or 'black esophagus', is an uncommon endoscopic finding characterized by dark discoloration of the esophageal mucosa that occurs in the context of ischemia, often in combination with other risk factors such as esophagitis, sepsis, or alcohol use. Although diagnostic and management considerations are well-described, limited data exist on frequency and risk factors for long-term complications, including stricture formation. We present a case of acute esophageal necrosis with stricture formation and provide a brief literature review relating to this complication.

**Case Description/Methods:** A 59-year-old man with history of alcohol use disorder, gastroesophageal reflux disease, and cardiovascular disease presented with epigastric pain and emesis and was found to be hypotensive. He was diagnosed with acute pancreatitis and managed conservatively with clinical improvement. During his hospitalization, he reported new-onset dysphagia and odynophagia. Barium esophagram demonstrated narrowing of the distal esophagus, and an endoscopy showed circumferential esophagitis with black eschar spanning the entire esophagus (Figure 1A). He was started on a proton pump inhibitor and sucralfate. Endoscopy 8 weeks later revealed Los Angeles grade D esophagitis without bleeding and a lower esophageal stricture measuring 1 cm in diameter x 12 cm in length (Figure 1B). In the context of severe esophagitis, dilation was deferred in favor of continued observation. The patient died 3 months later due to unrelated complications. Methods: A literature review was conducted using the PubMed database for cases of acute esophageal necrosis and stricture. Risk factors, treatments, and outcomes associated with stricture formation were extracted from articles. Median, percentage, and counts were calculated for variables of interest. Results: Out of 69 screened articles, 17 articles were included. Patients with strictures were older (median age 61.5 years), male (70%), or with shock or severe hypotension (80%) (Table 1). Perforation and death occurred in 10% of cases.

**Discussion:** Male sex, hypotension, older age are risk factors for stricture formation in the setting of acute esophageal necrosis. It is unclear if cardiac disease, diabetes, or alcohol/nonsteroidal anti-inflammatory drug use are associated with stricture formation. Perforation and death were rare complications. Further studies are needed, however, this analysis may help identify higher risk patients.



[2444] **Figure 1.** 1A: Acute esophageal necrosis with black eschar. 1B: Severe esophagitis with stricture formation at follow-up endoscopy.

**Table 1. Published Case Reports of Acute Esophageal Necrosis with Stricture Formation Legend: 'Y': Present; 'N': Not present; '?': Article does not mention Characteristics of the 19 previously published cases with the addition of our own case are detailed above**

| | | | Risk Factors | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Case | Sex | Age | HTN | HLD | CAD/ PAD | Diabetes | Cirrhosis | GERD | NSAID Use | AUD | Shock (including pre/post surgery) | Sepsis (or confirmed infection) | PPI | Sucralfate | Antimicrobial (antibiotic or antifungal) | TPN | Dilation | Stenting | Esophagectomy | Perforation | Death | Citation |
| 1 | M | 62 | Y | Y | ? | Y | N | ? | ? | ? | Y | N | Y | Y | N | ? | Y | N | N | N | N | Sandu, S. JGH Open. 2021 Mar 1;5(4):528-530. doi: 10.1002/jgh3.12520. |
| 2 | F | 85 | Y | Y | ? | Y | N | ? | ? | ? | Y | Y | Y | N | Y | Y | Y | N | N | N | N | Laredo, V. Gastroenterol Hepatol. 2020 Apr;43(4): 201-202. doi: 10.1016/j. gastrohep.2019.10.005. |
| 3 | M | 61 | Y | ? | ? | N | N | Y | ? | ? | Y | Y | Y | N | Y | ? | Y | N | Y | N | N | Ota, I. Clin J Gastroenterol. 2021 Aug;14(4):975-979. doi: 10.1007/s12328-021-01410-w. |
| 4 | M | 82 | N | N | Y | N | N | N | N | N | Y | N | N | Y | Y | Y | Y | N | Y | N | Y | Goldenberg, SP. Gastroenterology. 1990 Feb; 98(2):493-6. doi: 10.1016/ 0016-5085(9090844-q). |
| 5 | F | 62 | N | N | N | N | N | ? | N | Y | Y | Y | Y | N | Y | Y | Y | N | N | N | N | Pereira, O. BMJ Case Rep. 2013 Jan 29;2013: bcr2012008188. doi: 10. 1136/bcr-2012-008188. |
| 6 | F | 51 | ? | ? | ? | ? | Y | ? | ? | Y | Y | N | ? | ? | ? | ? | Y | Y | Y | Y | N | Kim, DB. Semin Thorac Cardiovasc Surg. 2017 Summer;29(2):256-259. doi: 10.1053/j.semtcvs. 2017.01.006 |
| 7 | F | 78 | Y | Y | Y | N | N | ? | N | ? | Y | Y | Y | ? | Y | Y | Y | Y | N | N | N | Argueta, EA. R I Med J (2013). 2019 Aug 1;102(6): 41-43. |
| 8 | M | 66 | ? | ? | ? | Y | Y | Y | ? | Y | Y | N | Y | ? | Y | Y | N | Y | N | N | Y | Planchard, JA. Cureus. 2019 Feb 19;11(2):e4090. doi: 10.7759/cureus.4090. |
| 9 | M | 56 | N | N | N | N | Y | ? | N | Y | ? | N | Y | N | ? | Y | Y | N | Y | N | N | Imaoka, K. Clin J Gastroenterol. 2021 Apr; 14(2):415-421. doi: 10. 1007/s12328-020-01326-x. |
| 10 | F | 81 | Y | N | N | Y | N | ? | N | Y | Y | N | Y | N | ? | Y | Y | N | Y | N | N | Same as above |
| 11 | M | 84 | ? | ? | ? | ? | Y | ? | ? | ? | ? | ? | ? | ? | Y | ? | N | N | N | N | N | Worrell, SG. Ann Thorac Surg. 2014 Jul;98(1):341-2. doi: 10.1016/j.athoracsur. 2013.09.023. |
| 12 | M | 84 | Y | ? | ? | Y | N | ? | ? | ? | ? | ? | ? | ? | ? | Y | Y | N | N | N | N | Same as above |
| 13 | M | 75 | Y | Y | Y | Y | N | ? | ? | ? | Y | N | Y | Y | Y | ? | Y | N | N | N | N | Gurvits, GE. Dig Dis Sci. 2015 Feb;60(2):444-53. doi: 10.1007/s10620-014-3382-1. |
| 14 | M | 55 | ? | ? | ? | ? | ? | Y | ? | Y | Y | Y | Y | Y | ? | ? | Y | N | N | N | N | Same as above |
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| 16 | M | 41 | N | N | N | N | Y | ? | N | Y | Y | Y | Y | N | Y | Y | Y | N | N | N | N | Endo, T. World J Gastroenterol. 2005 Sep 21; 11(35):5568-70. doi: 10. 3748/wjg.v11.i35.5568. |
| 17 | M | 58 | ? | ? | ? | ? | ? | ? | ? | Y | N | N | Y | ? | ? | ? | Y | N | N | N | N | Jinushi, R. DEN open. 2021 Sep 1;2(1):e43. doi: 10. 1002/deo2.43. |
| 18 | M | 34 | ? | ? | ? | Y | N | ? | ? | ? | Y | N | Y | N | Y | Y | Y | N | Y | N | N | Kim, YH. World J Gastroenterol. 2007 Nov 14; 13(42):5662-3. doi: 10. 3748/wjg.v13.i42.5662. |

Table 1. (*continued*)

| Case | Sex | Age | Risk Factors | | | | | | | | | | PPI | Sucralfate | Antimicrobial (antibiotic or antifungal) | TPN | Dilation | Stenting | Esophagectomy | Perforation | Death | Citation |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | HTN | HLD | CAD/ PAD | Diabetes | Cirrhosis | GERD | NSAID Use | AUD | Shock (including pre/post surgery) | Sepsis (or confirmed infection) | | | | | | | | | | |
| 19 | M | 58 | ? | ? | ? | ? | ? | Y | ? | Y | Y | N | Y | N | ? | Y | Y | Y | Y | Y | N | Tse, A. J Surg Case Rep. 2015 Jul 4;2015(7):rjv072. doi: 10.1093/jscr/rjv072. |
| Our Case | M | 59 | Y | Y | Y | N | N | Y | N | Y | Y | N | Y | Y | Y | N | N | N | N | N | N | N/A |
| # Cases with available data | | | 12 | 10 | 8 | 14 | 17 | 6 | 7 | 11 | 17 | 18 | 17 | 14 | 13 | 12 | 20 | 20 | 20 | 20 | 20 | |
| n (%) | Male: 14 (70%) | Median 61.5 | 8 (67%) | 5 (50%) | 4 (50%) | 7 (50%) | 5 (29%) | 5 (83%) | 0 (0%) | 10 (91%) | 16 (94%) | 6 (33%) | 16 (94%) | 5 (36%) | 11 (85%) | 11 (92%) | 17 (85%) | 4 (20%) | 7 (35%) | 2 (10%) | 2 (10%) | |

**S2445**

**Acute Esophageal Necrosis Due to Posterior Inferior Cerebellar Artery Stroke**

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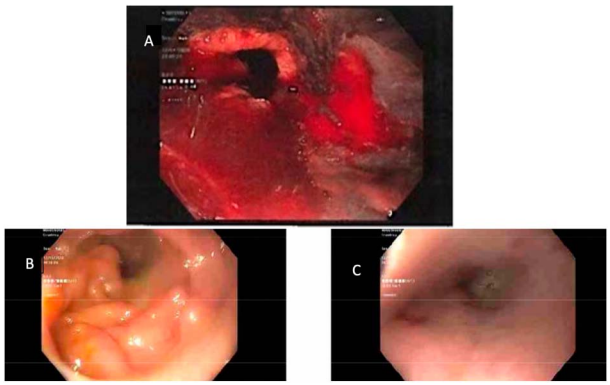
**Introduction:** Strokes involving the cerebellum can cause severe vomiting and lead to significant morbidity. Intractable vomiting after cerebellar stroke may be a precursor for the development of acute esophageal necrosis (AEN). Acute esophageal necrosis is a rare syndrome in which presumed hypoperfusion predisposes the esophagus to severe injury via reflux of acid and pepsin. It can be seen in association with protracted vomiting. In this case, we report the development of AEN related to vomiting from posterior inferior cerebellar artery (PICA) stroke.
**Case Description/Methods:** A 59-year-old male with past medical history of diabetes, hypertension, and GERD presented with intractable vomiting and dizziness for one week. Imaging revealed a subacute stroke of the right inferior cerebellum in the distribution of the right PICA. His nausea and vomiting were initially difficult to control with first line antiemetics, but after 4 days oral intake and nausea improved enough for safe discharge home with plans to continue vestibular therapy. Two days later, he returned to the hospital with more severe vomiting, dizziness, and dehydration. Multiple anti-emetics were tried unsuccessfully. Despite these efforts at symptom control, he continued to have retching and nausea. On hospital day 2, he developed brisk hematemesis requiring emergent upper endoscopy. He was found to have severe esophagitis with an area of circumferential necrosis in the distal esophagus, consistent with AEN (Figure A). Bleeding was treated with hemoclips (Resolution Clips, Boston Scientific) and epinephrine. In order to avoid further emesis and esophageal necrosis, the patient was left under deep sedation with intubation and was monitored in the ICU. The following day, a J tube was placed surgically. On day 6, surveillance EGD revealed complete resolution of necrosis with only white exudate remaining (Figure B,C). After improvement in mental status, the patient was extubated on hospital day 12 and returned to baseline mentation. Oral feedings were reintroduced after extubation. The patient fully recovered.
**Discussion:** This case demonstrates a previously undescribed presentation of acute esophageal necrosis as a complication of an acute right PICA infarct associated with intractable vomiting. Our approach using prolonged deep sedation, intubation, and jejunal feelings avoided further retching and allowed healing of necrotic esophageal mucosa. This case report may serve as a model for care of this rare syndrome and may stimulate further study.




[2445] **Figure 1.** A: Evidence of acute esophageal necrosis of the distal esophagus. B and C: Endoscopic findings of white exudate and recovery of prior necrotic esophagus.

**S2446**

**A Unique Endoscopic Finding in an HIV/AIDS Patient With Dysphagia**

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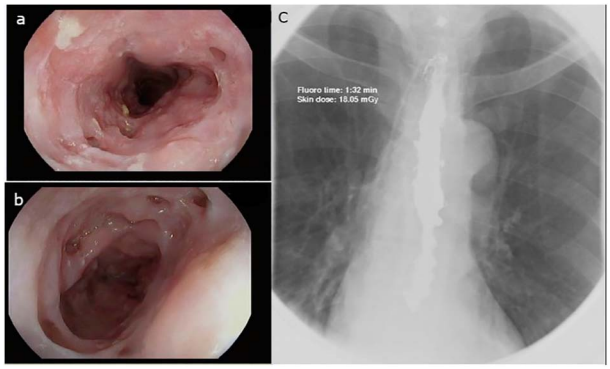
**Introduction:** There are many etiologies of dysphagia in patients with HIV/AIDS, including infection, structural issues, or medications. A broad differential is needed to properly diagnose and treat these patients. Here, we present a case of dysphagia in an HIV/AIDS patient related to esophageal intramural pseudodiverticulosis (EIPD).
**Case Description/Methods:** A 58-year-old man with HIV/AIDS presented with a 2-week history of dysphagia. He had been non-compliant with highly active antiretroviral therapy (HAART), and his CD4 count at presentation was noted to be $< 20$. His past medical history was notable for hypertension, chronic kidney disease, *Candida* esophagitis, and Mallory-Weiss syndrome. He underwent an esophagogastroduodenoscopy (EGD), which revealed numerous outpouchings and white plaques in the mid esophagus (Figures 1a and b). Esophageal brushings confirmed *Candida* infection, and biopsies were positive for cytomegalovirus (CMV). A barium esophagram showed outpouchings throughout the esophagus, without any evidence of perforation (Figure 1c). No stricture was evident on EGD or barium esophagram. The patient was diagnosed with EIPD and treated with fluconazole, a proton pump inhibitor, and valganciclovir, with improvement in symptoms.
**Discussion:** EIPD is characterized by the development of multiple small flask-shaped outpouchings in the esophagus. Patients with reflux esophagitis, *Candida* esophagitis, diabetes mellitus, HIV, and eosinophilic esophagitis are at higher risk for developing EIPD. There are approximately 250 published cases of EIPD in the literature, and the prevalence of EIPD is estimated to be 5-50 per 100,000 cases. Development of pseudodiverticula in the esophagus is suggested to be secondary to chronic inflammation, obstruction of the submucosal glands due to debris, or increased intraluminal pressure proximal to a luminal obstruction. The diagnosis is primarily made with EGD, barium esophagram, or computerized tomography. The treatment for EIPD includes treatment of underlying conditions and endoscopic dilation of strictures when they are present [1]. Our patient had several risk factors for development of EIPD such as *Candida* esophagitis, Mallory-Weiss syndrome, and HIV. However, a prior association between EIPD and CMV esophagitis has not been reported. Here, we highlight CMV esophagitis as a potential risk factor for development of EIPD.




[2446] **Figure 1.** EGD (a and b) and barium esophagram (c) findings.

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**S2447**

**"Outpouchings in the Esophagus": A Rare Case of HIV-Associated Esophageal Intramural Pseudodiverticulosis in the Absence of Esophageal Candidiasis**

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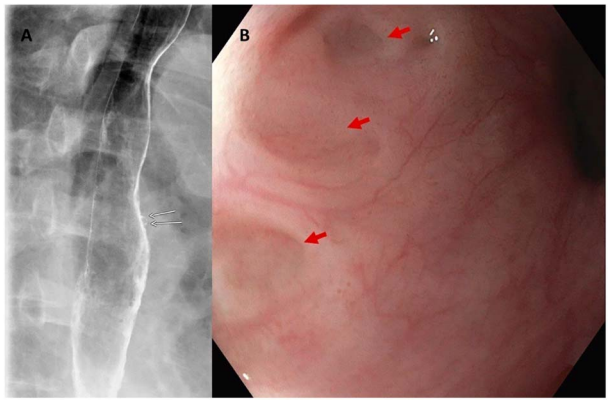
**Introduction:** Esophageal Intramural Pseudodiverticulosis (EIP) is a benign condition that presents with dysphagia. EIP with HIV AIDS is rare, especially in the absence of concomitant esophageal candidiasis. We present a case of dysphagia in a patient with HIV, found to have EIP.
**Case Description/Methods:** 51-year-old African American female with AIDS and noncompliance to Highly Active Antiretroviral Therapy (HAART) and a CD4 count of 0.5 cells/ mm3, presented with a 3-month history of progressively worsening dysphagia, to solids and liquids, odynophagia, and retrosternal burning. A barium esophagogram demonstrated an intramural pseudodiverticula. On upper endoscopy, multiple diminutive pseudo diverticula were visualized with no other gross abnormalities, including ulcers or stenoses (Figure B). Pathology from esophageal biopsies showed chronic inflammatory changes with no fungal or viral elements identified. The patient was re-initiated on HAART and high dose proton pump inhibitors with complete resolution of dysphagia on follow up.
**Discussion:** The pathogenesis of EIP involves esophageal excretory ductal obstruction and submucosal fibrosis from chronic inflammation and duct dilation from dysmotility. Risk factors include HIV, diabetes mellitus, esophageal candidiasis, chronic alcohol abuse, Mallory-Weiss syndrome, Crohn's disease, GERD, corrosive esophageal injury and esophageal malignancies. Single contrast barium esophagogram remains the study of choice and demonstrated numerous 1–4 mm flask-shaped diverticula. An upper endoscopy can be performed to corroborate imaging findings and diagnose concomitant structural and infectious etiologies. Management includes PPI therapy, treatment of underlying etiology, and dilation of any stenoses. Surveillance endoscopy can be considered due to an association with esophageal malignancies. The association of HIV with EIP, especially in the absence of esophageal candidiasis, is rarely reported. Clinicians should consider EIP as part of the differential diagnosis for HIV patients presenting with dysphagia.




[2447] **Figure 1.** A. Barium esophagogram shows psuedodiverticula (white arrows) B. Pseudodiverticula seen on upper endoscopy (red arrows).

**S2448**

**Acute Esophageal Necrosis From Trimethoprim-Sulfamethoxazole Use**

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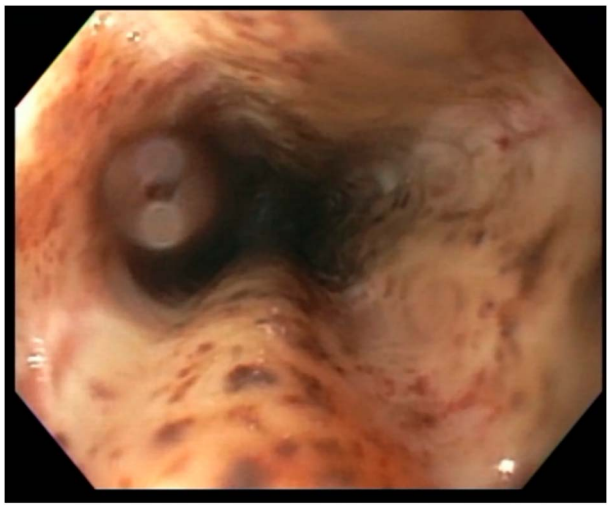
**Introduction:** We present a case of acute esophageal necrosis (AEN) secondary to Trimethoprim-Sulfamethoxazole (TMP-SMX). There have been reports of other drugs implicated in the cause of AEN, however, to our knowledge this is the first case reporting AEN after TMP-SMX use.
**Case Description/Methods:** A 70-year-old male with past medical history of type 2 diabetes mellitus, chronic kidney disease stage IV, hypertension, presented with epigastric pain, nausea, and vomiting. Four days prior he started taking TMP-SMX for a foot ulcer. Subsequently, he developed burning epigastric pain and multiple episodes of coffee ground emesis. On physical exam, he was tachycardic, pale, and had epigastric tenderness. Laboratory findings revealed white blood cell count of 19,100 /μL. He received a bolus of intravenous (IV) pantoprazole, followed by a continuous infusion. Esophagogastroduodenoscopy (EGD) revealed severe necrotic esophagitis with circumferential black eschar and severe friability, no linear tracking to suggest reflux at the beginning of the gastroesophageal junction, gastric mucosa was normal (**Figure**). Biopsy revealed ulcerative esophagitis with necrosis. Stains were negative for any viral or fungal organisms. Patient was discharged on pantoprazole and sucralfate.
**Discussion:** Acute esophageal necrosis (AEN) is a rare condition in which patients typically present with hematemesis or melena with endoscopy characterized by diffusely spread necrotizing lesions of the distal esophageal mucosa abruptly ending at the gastroesophageal junction. Several proposed mechanisms of AEN include hypoperfusion leading to ischemic injury, gastric acid damage, and suppressed protecting mechanisms [1]. AEN is more commonly found in older men in their seventh decade, with other co-morbidities such as diabetes, renal insufficiency, cardiac disease, hypertension, or history of alcohol abuse

[3,4]. There are several drugs including NSAIDs, antihypertensive agents, cefazolin, terlipressin, clozapine to name a few which have caused AEN, however TMP-SMX has not been reported so far. Most of the drugs cause either a direct mucosal injury or induce changes in vascular perfusion [2]. Management of AEN includes discontinuing the offending agent and is mainly supportive care along with aggressive IV proton pump inhibitor (PPI) therapy initially until symptoms improve, then transitioning to oral PPI. Repeat EGD is recommended in one month to confirm healing of esophageal mucosa and to survey for strictures which is a possible complication [3].



[2448] **Figure 1.** EGD showing severe esophagitis with circumferential black eschar and severe friability.

**S2449**

**A Unique Case of Simultaneous Anal Squamous Cell Carcinoma and Esophageal Squamous Cell Carcinoma**

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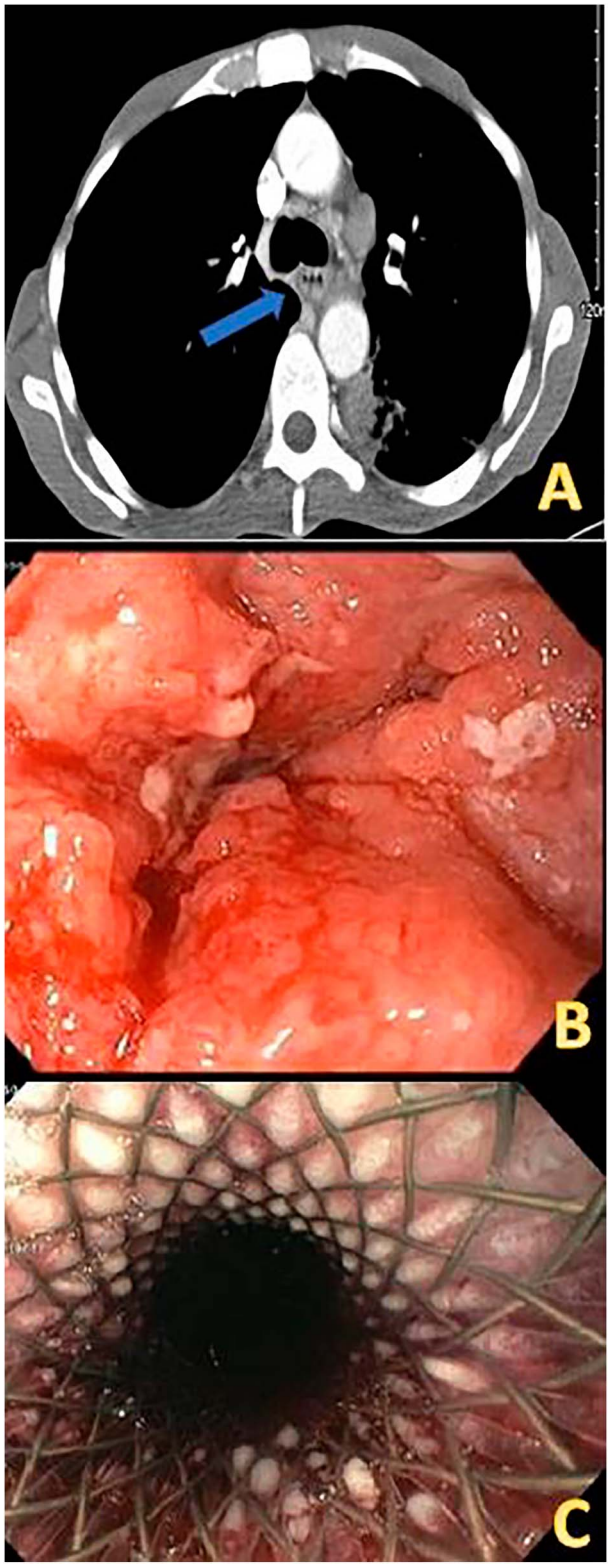
**Introduction:** Anal cancer (AC) is uncommon and comprises only 3% of all digestive system malignancies. It is associated with HPV, tobacco use, and HIV. Esophageal cancer (EC) is the 8$^{th}$ most common cancer and 6$^{th}$ most common cause of death worldwide. Among other associations, HPV and tobacco use has been strongly linked to development of esophageal SCC. Though they have similar risk factors, the literature is limited when it comes to cases of simultaneous anal SCC and esophageal SCC.
**Case Description/Methods:** A 50-year-old male with history of tobacco use presented with dysphagia, dyspnea, and weight loss. His symptoms were progressive with 20 lbs. of weight loss in 3 months. He initially had solid food dysphagia which progressed to liquids and constant regurgitation of food. His cough and dyspnea were acute and worsened 1 week prior to his presentation. He endorsed a bulging growth around his anus that grew over several months. He was found to have a left lower lobe necrotizing pneumonia (NP) on CT scan as well as thickening of the esophagus with liver lesions suspicious for metastatic disease. Labs showed leukocytosis, macrocytic anemia, and a mild transaminitis. He was tested for tuberculosis and HIV which was negative. He underwent an EGD which showed a large, friable, fungating esophageal mass whose biopsies showed SCC. For his anal lesion, he underwent an excisional biopsy which was positive for SCC as well. The patient was treated with antibiotics for his NP with a plan of 6 weeks of therapy followed by repeat imaging to determine if any surgical interventions would be needed. Oncology planned for palliative chemoimmunotherapy once his NP resolved. To improve his dysphagia, a palliative esophageal stent was placed (**Figure**).
**Discussion:** This patient had both a primary anal SCC and esophageal SCC with his only risk factor being tobacco use. Though both AC and EC commonly metastasizes to the liver, EC is more likely to metastasize. For esophageal SCC, local lymph node invasion typically occurs early since the lymphatics are in the lamina propria rather than beneath the muscularis mucosa. In this case, the patient presented with a NP and though no definitive fistula was noted on imaging, local invasion of esophageal SCC can present with fistulizing disease. Unfortunately, Stage 4 esophageal SCC is incurable though systemic therapies may provide a palliative effect and survival benefit. In this case, an endoscopic esophageal stent was placed for palliation of his dysphagia while awaiting chemoimmunotherapy.




[2449] **Figure 1.** Image A demonstrates the CT scan finding of esophageal wall thickening. Image B is the EGD finding of a large, friable, fungating, circumferential esophageal mass. Image C shows the successful placement of an esophageal stent for palliative treatment of the patient's dysphagia.

S2450

**Acute Esophageal Necrosis: Low Flow Leading to Darkness**

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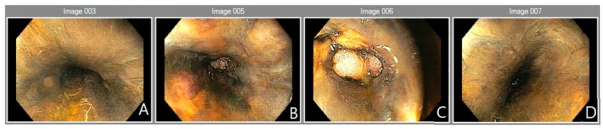
**Introduction:** Acute esophageal necrosis (AEN), commonly referred to as black esophagus due to the striking circumferential blackening of the esophageal mucosa, is a rare clinical condition with a prevalence of up to 0.2%. The etiology of this condition is unclear but thought to involve the interplay of ischemia and gastric outlet obstruction with gastroesophageal reflux. As this condition carries with it a mortality rate of nearly 40%, prompt recognition and aggressive management is of the utmost importance to prevent perforation or stricture. Considering this, AEN should be recognized as a possible outcome of an ischemic event and considered a potential cause of spontaneous esophageal perforation.
**Case Description/Methods:** A 43-year-old female with a history of epithelioid hemangioendothelioma status post resection, Crohn's colitis in remission, and recent hospitalization for a complicated urinary tract infection and abdominopelvic ascites concerning for malignancy with hospitalization complicated by cardiopulmonary arrest presented to the emergency department 8 days after discharge following a near syncopal episode. She was found to be hypotensive with tachycardia concerning for shock. She was started on broad spectrum antibiotics, 3 vasopressors, intubated, and admitted to the ICU. A CT scan of her chest, abdomen, and pelvis showed extensive pneumomediastinum concerning for esophageal perforation and pneumatosis intestinalis. Perforation was confirmed utilizing serial chest x-rays with contrast demonstrating a right-sided esophageal leak at the gastroesophageal junction. Both cardiothoracic surgery and gastroenterology were consulted, she was not deemed a surgical candidate and endoscopy with covered stent placement was planned. Endoscopy revealed a black esophagus consistent with an ischemic process throughout the entire esophagus and pneumatosis intestinalis suggesting small bowel ischemia. Stenting was aborted and the findings were discussed with the patient's family who decided to pursue comfort measures. The patient died the following day (**Figure**).
**Discussion:** It has been demonstrated that critically ill patients often experience low flow states, poor nutritional status, and disruption of intrinsic repair mechanisms. Coupled together, these insults increase the risk of developing AEN and must be acknowledged as a possible complication in this patient population. Although this is a rare condition, it should be recognized as a potential outcome of an ischemic event.




[2450] **Figure 1.** Images A-D depicts a diffusely abnormal mucosa throughout the esophagus, characterized by black/brown tissue consistent with ischemia.

S2451

**Acute Esophageal Necrosis: A Rarely Encountered Clinical Entity**

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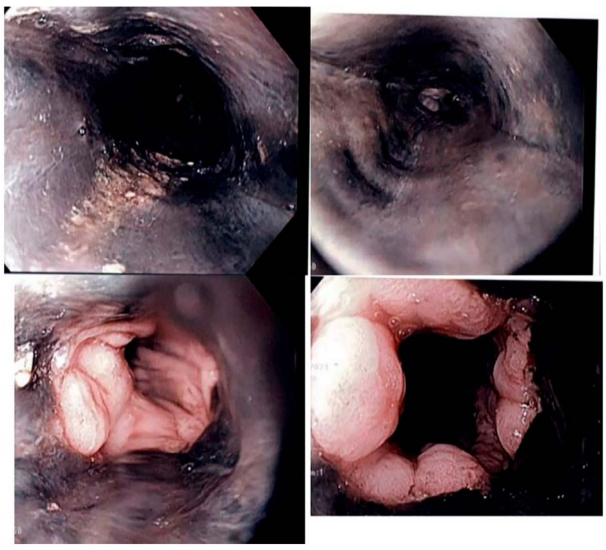
**Introduction:** Acute Esophageal Necrosis (AEN), otherwise known as black esophagus for its remarkable appearance during endoscopy, is a rather rare entity in the gastroenterology field.
**Case Description/Methods:** A 51-year-old male with a past medical history of diabetes complicated by peripheral neuropathy presented to the hospital with left lower extremity swelling and pain after a fall. On admission he underwent lower extremity x-ray with subcutaneous air concerning for necrotizing fasciitis. He was taken emergently to the operating room where he underwent 3 compartment exploration with irrigation and drain placement. On day 7 of hospitalization he developed persistent nausea and vomiting for which we were consulted. The following morning he was taken to the endoscopy suite. He was noted to have circumferential black appearance of the esophagus from 28 to 40 cm with abrupt transition to normal-appearing gastric mucosa at the GE junction. This was endoscopically consistent with acute esophageal necrosis He was placed on high-dose intravenous proton pump inhibitor (PPI) therapy as well as sucralfate suspension. Transfer was facilitated to a tertiary care center where repeat endoscopy confirmed the diagnosis. He had a gastrostomy tube placed and ultimately underwent repeat endoscopy 8 weeks later at the same tertiary care center which revealed severe benign appearing esophageal stricturing with inability to pass adult endoscope. It was recommended to continue on PPI therapy with repeat endoscopic evaluation with possible dilation the following month. Unfortunately, in the meantime the patient expired due to unknown causes (**Figure**).
**Discussion:** AEN is defined by a circumferential, necrotic appearance that almost always affects the distal esophagus with abrupt cessation of the black contour at the GE junction. Incidence is 0.28%, affects men over 4 times more often than women with a mortality near 32%. AEN is usually multifactorial and affects people with many comorbidities. Presenting symptoms varied greatly, with the most prevalent being hematemesis (34%). Injury is related to esophageal ischemia and topical injury. AEN is almost always diagnosed by endoscopy. Biopsy can rule out other similarly appearing pathologies. Management of AEN typically involves treating the underlying cause. Empirical treatment includes IV fluids, PPI, sucralfate, NPO, and TPN in patients with poor nutritional status after 24 hours.



[2451] **Figure 1.** Multiple images showing diffuse esophageal necrosis with abrupt transition to normal mucosa at the GE junction.

S2452

**An Unusual Case of Esophageal Perforation**

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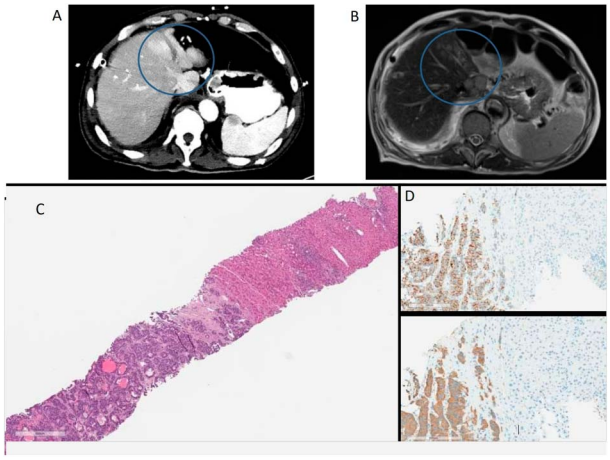
**Introduction:** Zollinger Ellison syndrome (ZES) is characterized by gastric hyperacidity, resulting from increased gastrin production. This leads to symptoms such as abdominal pain, heartburn, and diarrhea. Given the overlap in the symptoms with idiopathic Peptic Ulcer Disease (PUD) and Gastro-Esophageal Reflux Disease (GERD), the diagnosis can be delayed and complications such as gastric obstruction, hemorrhage, or even perforation can be the initial presentation. Here we present an unusual case of esophageal perforation that was ultimately diagnosed with ZES.
**Case Description/Methods:** A 65-year-old male with a history of perforated gastric ulcer s/p Graham patch repair 2 months prior presented with worsening abdominal pain, vomiting, and inability to swallow secretions. He had a positive H. pylori stool antigen test during the prior hospitalization s/p course of eradication therapy. CT chest showed pneumomediastinum and an esophageal perforation. An upper gastrointestinal endoscopy, done to place an esophageal stent, revealed multiple ulcers in the first portion of the duodenum. Robotic-assisted drainage of the posterior mediastinum was then pursued. Abdominal imaging (CT/MRI) demonstrated enhancing masses in multiple liver segments as well as periportal adenopathy (Figure A, B). FDG PET/CT showed increased uptake by the left liver lesion and multiple porta hepatis adenopathy, concerning for metastatic disease. The primary tumor could not be clearly delineated. A percutaneous liver biopsy revealed a metastatic, grade 1 well-differentiated neuroendocrine tumor (Figure C, D). Serum gastrin levels were elevated to 433 pg/mL. Serum ionized calcium and parathyroid hormone were within normal limits. Family history was negative for multiple endocrine neoplasia 1 (MEN1). A diagnosis of metastatic, sporadic ZES with an unknown primary site was made. The patient was discharged on high-dose lansoprazole and somatostatin analog with outpatient follow-up for DOTATATE scan and definitive treatment.
**Discussion:** Peptic ulcer perforation is seen in 4-6 % of the patients with ZES. Most perforations involved the duodenum with one report of jejunal involvement and no reports of gastric perforation to date. There is evidence of an increase in esophageal involvement, with several cases of esophagitis and Barrett's esophagus; however, very few cases of esophageal perforation were reported. This case is unique in that our patient developed complications rarely described in literature i.e. gastric perforation and esophageal perforation.




[2452] **Figure 1.** A: CT abdomen showing 3.5 cm mass inseparable from segment II of the liver. B : MRI abdomen showing multiple enhancing masses in segments I, II, and IVA with subtle washout and associated periportal adenopathy most compatible with metastatic disease process. C: This image of a liver core needle biopsy shows the transition from liver parenchyma (upper right) to the tumor (lower left). The nested pattern of the tumor is suggestive of possible neuroendocrine origin. D: Chromogranin (left) and synaptophysin (right) immunohistochemical stains positively highlight the tumor cells (brown) and do not stain the liver parenchyma, confirming neuroendocrine tumor. The lack of pleomorphism, mitotic figures, and low Ki-67 proliferation index (~25, not shown) identify this as a well-differentiated neuroendocrine tumor.

S2453

**An Uncommon Cause of Dysphagia in a Patient With Thyroid Disease**

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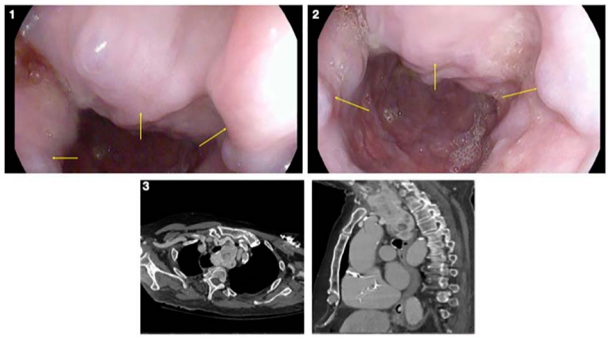
**Introduction:** An 88-year-old man was seen for evaluation of an abnormal barium swallow in the setting of yearlong dysphagia to solids with globus sensation.
**Case Description/Methods:** His medical history included atrial fibrillation and hyperthyroidism. He had undergone transesophageal echocardiography, notable for difficulty in passing the probe. Consequently, we performed an upper endoscopy that showed large, non-bleeding varices extending 11 cm (proximal third) to 32 cm from the incisors (middle third) (Figure, panels 1-). The gastroesophageal junction was noted at 40 cm from the incisors. A subsequent computed tomography scan with angiography of his chest showed substernal extension of a large goiter into the middle mediastinum causing mass effect and collateralization arising from the left brachiocephalic vein creating upper esophageal varicosities (Figure, panel 3). The patient was referred to surgical endocrinology for further management.
**Discussion:** Proximal esophageal varices, termed downhill varices, are usually seen in the setting of benign or malignant superior vena cava obstruction (SVCO). The upper and mid esophageal veins drain into the azygous and innominate veins. The presence of a SVCO leads to the collateralization of the azygous and innominate veins to divert venous return from the head and mediastinum. This collateralization and increased pressure lead to the development of downhill varices. Treatment of downhill varices is aimed at correcting the cause of the obstruction.




[2453] **Figure 1.** Panels 1 and 2: 3 columns of large varices extending from the proximal third to the middle third of the esophagus. Panel 3: 2 representative computed tomography scan slices in the axial and sagittal planes highlighting the mass effect leading to compression of the right brachiocephalic vein with collateralization. This also shows the mass effect compressing the left brachiocephalic vein with collateralization.

S2454

**An Incidental Finding of Multiple Esophageal Intramural Pseudo-Diverticuli**

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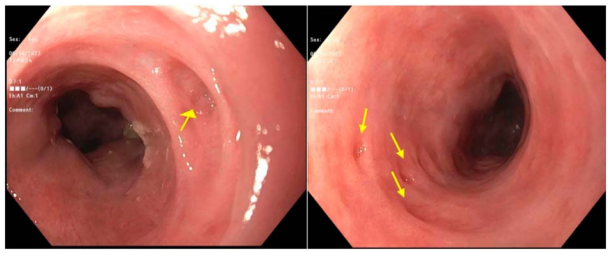
**Introduction:** Esophageal intramural pseudo-diverticulosis (EIP) is characterized by multiple small outpouches protruding from the esophageal lumen. It is a rare disorder, found in 0.15% of esophagrams. Clinical manifestations include progressive dysphagia, food impaction, and occasionally bleeding. Esophageal strictures are the most common complication of EIP. We present a case of EIP thought to be due to multiple etiologies causing chronic esophageal inflammation.
**Case Description/Methods:** A 55-year old male with a past medical history of alcohol use disorder, recurrent pancreatitis, tobacco use disorder, and hypertension with a recent admission for likely alcohol induced acute pancreatitis presented for a follow up to the clinic. During admission, a CT scan revealed a cystic lesion concerning for an intraductal papillary mucinous neoplasm. A follow up MRI showed a growing cystic pancreatic head lesion, measuring 1.6 cm from 7mm several years prior. He was referred to GI for further evaluation of the pancreatic cyst with endoscopic ultrasound/fine needle aspiration (EUS/FNA). During his EUS, he was noted to have multiple small pseudo-diverticula in the esophagus and a narrow caliber esophagus so EUS was not performed due to concern for perforation risk (Figure 1). He had no history of dysphagia, odynophagia, weight loss or bleeding. He was also found to have oral candidiasis and started on nystatin swish and spit solution. He was started on a proton-pump inhibitor (PPI) for suspected EIP despite absence of symptoms to potentially prevent progression to stricturing disease.
**Discussion:** Although rare, it is important to identify and closely monitor patients with EIP. The exact etiology is unknown but findings of EIP are linked to chronic inflammatory states. In our patient, chronic alcohol and tobacco use as well as oral candidiasis can contribute to development of EIP. EIP is linked to formation of esophageal strictures causing dysphagia symptoms prompting investigation and treatment with esophageal dilation. Our patient was asymptomatic and EIP was an incidental finding. However, prior studies have shown an increased prevalence of EIP in patients with esophageal carcinoma so close monitoring of patients with findings of EIP is essential. The role of therapies such as PPIs for preventing progression of stricturing disease in EIP is not clear. Further studies are needed to help us better understand this disease.



[2454] **Figure 1.** Multiple esophageal intramural psuedo-diverticuli noted on esophagoduodenoscopy (EGD) (yellow arrows).

S2455

**An Esophagus as Dark as the Night: Acute Esophageal Necrosis**

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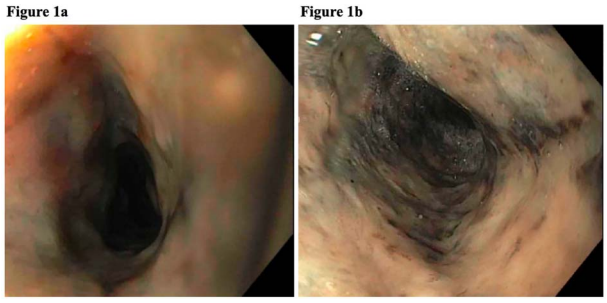
**Introduction:** In one of its most severe forms, esophagitis may present as acute esophageal necrosis (AEN), which is a condition characterized by a diffusely black esophageal mucosa on upper endoscopy. AEN is a rare disorder, with a prevalence of only 0.2%. Since the first endoscopic characterization by Goldenberg et al. (1999), the underlying pathophysiology remains elusive. This case demonstrates the more recently identified correlation between diabetic ketoacidosis (DKA) and AEN.
**Case Description/Methods:** A 46-year-old male with a medical history of cocaine use disorder, type 2 diabetes, and hypertension presented to our hospital with hematemesis and altered mental status. He reported no melena or hematochezia and had no prior history of endoscopic evaluation. On arrival, he was hypotensive, tachycardic, and tachypneic. Physical exam revealed diffuse abdominal tenderness. He was noted to be in DKA and urine toxicology was positive for cocaine. He was resuscitated and started on a Protonix drip. Insulin drip was also initiated per DKA protocol. Gastroenterology was consulted for coffee ground emesis and down trending hemoglobin. Upper endoscopy showed severe active esophagitis with ischemic appearing mucosa in the entire esophagus (Figure 1a and Figure 1b). Biopsies revealed severe esophagitis with ulceration and immunohistochemical testing for CMV and HSV was negative. The patient was managed medically with pantoprazole twice daily and he recovered gradually with plans to repeat EGD in 6-8 weeks to assess healing.
**Discussion:** AEN is an uncommon syndrome characterized by diffuse, circumferential black esophageal mucosa with an abrupt transition at the gastroesophageal junction. The underlying pathophysiology is thought to be multifactorial: tissue hypoperfusion, poor local defense barriers, and an insult to esophageal mucosa from reflux of gastric contents. Underlying clinical conditions such as diabetes mellitus, cardiovascular diseases, cirrhosis, sepsis, and renal insufficiency are likely predisposing factors. As in the case above, there is also a strong link between DKA and AEN. Yasuda et al. (2006) described 4 cases of AEN in patients who initially presented with DKA. AEN diagnosis requires a high index of suspicion as it generally carries a poor prognosis with one-third of patients succumbing to their underlying illness. The goal of therapy is prompt treatment of coexisting, underlying medical diseases, adequate fluid resuscitation, hemodynamic stabilization, and nutrition.




[2455] **Figure 1.** a: Circumferential necrosis in the upper third of the esophagus. b: Circumferential necrosis in the middle third of the esophagus.

S2456

**An Unexpected Finding of a Black Esophagus: Atypical Presentation of Acute Esophageal Necrosis**

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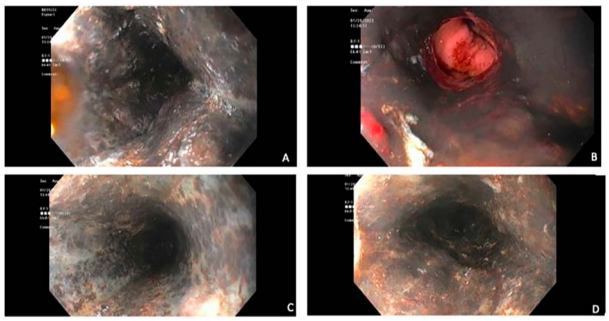
**Introduction:** Acute esophageal necrosis (AEN), or black esophagus, is a rare condition that manifests as black mucosa of the distal esophagus with an abrupt transition at the gastroesophageal junction. AEN typically presents as an upper gastrointestinal bleed. Risk factors include male sex, advanced age, diabetes, coronary artery disease, pulmonary disease, gastroesophageal reflux, malignancy, and malnutrition in the setting of hemodynamic instability. Treatment includes management of underlying conditions, intravenous proton pump inhibitor therapy, nutrition, fluid resuscitation, and bowel rest. High mortality rates have been demonstrated but many patients can recover with supportive care.
**Case Description/Methods:** A 67-year-old male with chronic obstructive pulmonary disease, peripheral vascular disease, and hypertension presented as a transfer for escalation of care of acute on chronic respiratory failure and triple vessel coronary artery disease. After 7 days of mechanical ventilation, the patient experienced oropharyngeal dysphagia and failed a swallow evaluation. Enteral access with dobhoff tube placement was attempted without success, prompting gastroenterology consult for endoscopic placement. He was tachycardic, normotensive, afebrile, and saturating well on 4 L nasal cannula. Labs showed a hemoglobin of 12 g/dL, blood urea nitrogen 12 mg/dL, and creatinine 0.64 mg/dL. He denied abdominal pain, vomiting, melena, or hematochezia. Endoscopy showed a normal upper third of the esophagus with distal black mucosa, and an abrupt transition at the gastroesophageal mucosa. Biopsy showed ulcer exudate and granulation tissue with acute inflammation, negative for CMV, HSV, or fungal organisms. Patient remained with nothing by mouth and on total parenteral nutrition. A do not resuscitate order was placed and the patient unfortunately expired from a ventricular arrythmia 4 days later (**Figure**).
**Discussion:** This case demonstrates an unexpected finding of AEN in a patient presenting atypically with oropharyngeal dysphagia in the absence of obvious upper gastrointestinal bleeding. This patient's critical illness, age, and multiple medical comorbidities most likely contributed to his AEN. This case highlights the importance of maintaining a broad differential diagnosis for dysphagia in critically ill patients. It also highlights the need to evaluate the acute onset of dysphagia in this patient population prior to enteral tube placement, given high risk of perforation.




[2456] **Figure 1.** A, C, D) black, necrotic appearing distal esophagus B) black, necrotic appearing distal esophagus with abrupt transition at the gastroesophageal junction.

**S2457**

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**An Unusual Case of Dysphagia Secondary to Cervical Osteophytes**

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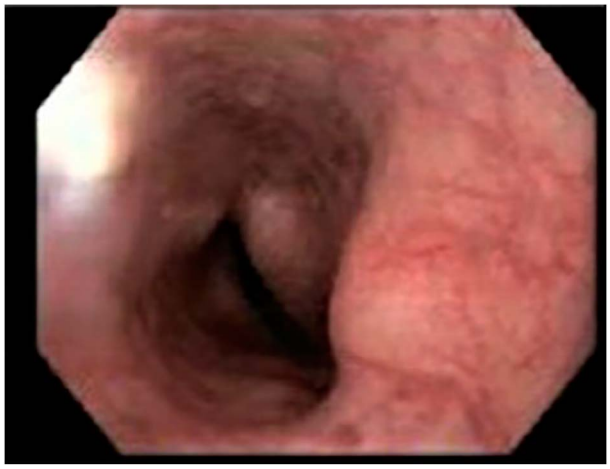
*University of Massachusetts Chan Medical School - Baystate, Springfield, MA.*

**Introduction:** Leading causes of dysphagia include esophageal stricture, Schatzki ring, eosinophilic esophagitis, esophageal spasm, and achalasia. Cervical osteophytes are an uncommon cause of dysphagia. We report a woman who presented with dysphagia and was found by esophagogastroduodenoscopy (EGD) to have protrusions in the upper and mid esophagus consistent with cervical osteophytes.
**Case Description/Methods:** A 66-year-old woman with past medical history of chronic gastroesophageal reflux disease (GERD), Barrett's esophagus, and systolic heart failure was admitted to the hospital for IV diuresis due to heart failure exacerbation. On hospital day 3, she reported years of difficulty swallowing associated with globus sensation near her sternum. A barium swallow showed a filling defect in the thoracic esophagus associated with significant dysmotility proximal to the defect. She was taken for EGD, which demonstrated 2 protrusions in the upper to mid esophagus that were hard in texture, consistent with a diagnosis of cervical osteophytes (**Figure**). She was instructed to take small bites of food with sips of water to facilitate food passage. Once she was stable from a heart failure perspective, neurosurgical intervention could be considered for definitive treatment.
**Discussion:** Cervical osteophytes can cause dysphagia, odynophagia, and globus sensation when they protrude from the anterior cervical vertebrae into the esophagus. This can cause mechanical compression leading to obstruction as well as spasm inflammation and spasm. Dysphagia caused by cervical osteophytes is usually managed with dietary modifications, swallowing therapy, and non-steroidal anti-inflammatory drugs. When conservative management fails, or osteophytes become large, surgical intervention is recommended. A review by Verlaan et al. showed that 35 of 169 cases were managed conservatively; the remainder were treated with surgery, the most common being resection of the osteophyte without spinal fusion. Complications of osteophyte resection include vocal cord palsy, esophageal perforation or fistula formation, and Horner syndrome. Clinicians should consider this rare cause of dysphagia in the differential for dysphagia patients and consider the risks and benefits of surgical intervention should conservative management fail.



[2457] **Figure 1.** EGD revealing 2 protrusions in the upper to mid esophagus with hard texture.

S2458

**Rosacea Improved Following Treatment With Combination Antibiotics and Fecal Microbiota Transplantation: Two Case Reports**

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*Centre for Digestive Diseases, Five Dock, New South Wales, Australia.*

**Introduction:** Rosacea is a chronic skin condition affecting nearly 18% of the population worldwide. It is characterised by flushing or redness of the cheeks, nose, chin and/or forehead. In severe cases visible blood vessels, inflammatory acne or skin thickening may be present. The cause of rosacea remains unknown and treatments are limited. Emerging evidence suggests a link between gastrointestinal (GI) microbiome dysbiosis and rosacea. Here, we report 2 cases who received fecal microbiota transplantation (FMT) for irritable bowel syndrome (IBS) and reported an improvement in concomitant rosacea.
**Case Description/Methods:** Case 1: A female (65yrs) presented with known IBS characterised by bloating, abdominal pain and diarrhoea. Medical history included subtotal hysterectomy and optical rosacea, treated with hourly eye drops and cyclosporine eye gel. The patient was subsequently treated with vancomycin and rifaximin, with reduction in abdominal pain, bloating and bowel frequency. The patient also reported reduced eye irritation from optical rosacea. The patient then underwent FMT for 10 days (1 colonoscopic infusion, 9 rectal enemas). At 6 months, the patient reported a 75% improvement in GI symptoms and reported ongoing improvement in optical rosacea although still reliant on cyclosporine eye gel. Follow up at one year post FMT demonstrated recurrence of GI symptoms and optical rosacea. Case 2: A female (45yrs) presented with abdominal cramps, bloating, flatulence and alternating diarrhoea and constipation. Concomitant medical history was endometriosis and severe acne rosacea treated with twice daily application of topical mupirocin ointment. Investigations excluded organic pathology and IBS was diagnosed. The patient was subsequently treated with vancomycin and rifaximin followed by FMT for 10 days (1 colonoscopic infusion, 9 rectal enemas). At 6 months, the patient had no ongoing GI symptoms and continued to report ongoing improvement in acne rosacea with some flushing persisting. Follow up at 6 years post FMT demonstrated ongoing clinical improvement with occasional abdominal pain and diarrhoea. The patient no longer experienced acne rosacea and had ceased all medications.
**Discussion:** To our knowledge this is the first report of antibiotics followed by FMT to treat rosacea, with improvements reported in both the short and long term. Future prospective trials are required to confirm FMT as a treatment for rosacea.

## FUNCTIONAL BOWEL DISEASE

**S2459**

### Bad News Gummy Bears: A Case of Gastrointestinal-Isolated Alpha-Gal Allergy

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**Introduction:** Galactose-α-1,3-galactose (alpha-gal) allergy is an IgE-mediated reaction to mammalian meat and dairy, with symptoms typically developing 3 to 6 hours after ingestion. Alpha-gal allergy with isolated gastrointestinal (GI) symptoms (abdominal pain, diarrhea, vomiting) without urticaria or anaphylaxis has been described.
**Case Description/Methods:** A 49-year-old male presented to GI clinic with years of episodic cramping abdominal pain and 4-6 diarrheal stools daily. Colonoscopy, EGD, CT abdomen, and hydrogen breath testing were unrevealing. We diagnosed irritable bowel syndrome. Rifaximin briefly relieved symptoms. He did not tolerate tricyclic antidepressants. Serum alpha-gal IgE level was found to be elevated at 0.27 kU/L (reference range, < 0.1 kU/L) after the patient reported past tick bites. The patient stopped eating mammalian meat and symptoms improved but continued. He further excluded gelatin in the form of gummy bears, dairy, and carrageenan, which led to near resolution of symptoms. At a visit 2 years following allergy diagnosis, he reported one formed stool daily and no chronic abdominal pain. Self-challenging with foods that have small amounts of alpha gal, like a handful of gummy bears, had resulted in severe abdominal cramping.
**Discussion:** The diagnosis of GI alpha-gal syndrome can be challenging because of delayed onset of symptoms after eating mammalian products, nonspecific GI complaints on presentation, and overlap with preexisting GI diseases. While sensitization is believed to be caused by a bite from the Lone Star tick, not all patients report a history of tick bites. Further studies are needed to better characterize GI alpha gal, but 2 cohort studies found that ~75% of patients improved on a diet free of mammalian products. These diets are somewhat ill-defined in how strict they are, particularly whether they are just free of mammalian meat or all products containing alpha-gal. A stepwise approach is recommended, starting with mammalian meat, then dairy, and then gelatin, gelatin capsules, and mammalian food additives if symptoms persist. Our patient experienced improvement in symptoms with elimination of mammalian meat from his diet, but no resolution until he eliminated all alpha-gal, particularly gelatin and carrageenan. This case highlights the importance of a thorough history, increased awareness of foods containing alpha-gal, and the need for future prospective studies for improving early recognition and treatment of GI alpha gal (**Figure**).


[2459] **Figure 1.** Recommended order of alpha-gal elimination from the diet: (i) mammalian meat with or without dairy; (ii) dairy; and (iii) gelatin, gelatin capsules, and mammalian food additives. Figure used with permission from McGill SK, Richards RD Jr, Commins SP. Suddenly Steakless: A Gastroenterologist's Guide to Managing Alpha-Gal Allergy. Am J Gastroenterol. 2022;117(6):822-826.

**S2460**

### Autoimmune Enteropathy (AIE) in a Patient Diagnosed with Thymoma: A Case Report

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*[1]Creighton University, Omaha, NE; [2]Creighton University School of Medicine, Omaha, NE.*

**Introduction:** Autoimmune enteropathy (AIE) is a very rare immune disorder that mainly attacks the gastrointestinal tract by T-cell. The full pathology mechanism is not clear. Typically, characterized by intractable diarrhea and nutritional malabsorption with extra-intestinal manifestations. The proposed diagnostic criteria include small bowel villous atrophy not responding to diet restriction, circulating gut epithelial cell autoantibodies (GECA), and lack of immunodeficiency. We describe a case of AIE with extensive GI involvement, presenting in a 60-year-old patient diagnosed with Type AB thymoma.
**Case Description/Methods:** Our gentleman with a history of Covid-19 complicated with pulmonary embolism and an incidental finding of malignant thymoma. A CT-guided biopsy was consistent with undifferentiated malignant thymoma supported by immunohistochemistry staining. Subsequently, complicated severe diarrhea erupted with significant weight loss. Conservative management, antibiotics, and diet restriction were ineffective. Diagnostic work-up was unremarkable except for anti-enterocytes antibodies (AEA) and anti-goblet cells antibodies (AGA). Bowel biopsy revealed villous blunting, loss of Paneth cells, and minimal intraepithelial lymphocytosis with no evidence of crypt abscesses. Corticosteroid and Octreotide have helped the patient's diarrhea. Thoracoscopy thymectomy performed with radiation therapy due to local and lymphovascular invasion.
**Discussion:** AIE characterized by severe villous blunting with the absence of goblet cells and Paneth cells, intraepithelial lymphocytosis, and increased crypt apoptosis. In comparison, graft vs host disease lack crypt abscesses, celiac disease shows increase in the intraepithelial lymphocytosis with intact goblet and Paneth cells, whereas inflammatory bowel disease has intact goblet and Paneth cells, and CVID characterized by absence of plasma cell in the lamina propria. The presences of the GECA are nonspecific but it may help in confirming the diagnosis or may predict the prognosis and recurrence. Only AGA has been reported in IBD. Neither has been observed in celiac disease. The low incidence of AIE and the limited existing literature available on the optimal guidance in management. Oral nutritional supplementation as well as total parenteral nutrition is helpful. The target is to control diarrhea and optimize the nutritional status before surgery. The main treatment is thymectomy.

**S2461**

### Chronic Diarrhea: A Rare Presentation of Eosinophilic Granulomatosis With Polyangiitis

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*[1]Jersey Shore University Medical Center, Neptune, NJ; [2]Saint George's University School of Medicine, Neptune, NJ; [3]Jersey Shore University Medical Center, Apex, NC; [4]Monmouth Gastroenterology, Freehold, NJ.*

**Introduction:** Diarrhea is a common occurrence with a significant healthcare burden, responsible for nearly 1 million emergency department (ED) visits per year. Chronic diarrhea affects up to 5% of adults and is defined by greater than 4 weeks of symptoms. In resource-abundant countries, the most common etiologies include irritable bowel syndrome, inflammatory bowel disease, malabsorption syndromes, chronic infections, and drug-induced. We describe a case of chronic diarrhea with an unusual etiology, eosinophilic granulomatosis with polyangiitis (EGPA).
**Case Description/Methods:** A 60-year-old man with a history of asthma and nasal polyps presented to the ED with nonbloody diarrhea starting 5 weeks prior. Associated symptoms included a dry cough. Of note, the patient was prescribed montelukast for asthma roughly one year prior to admission. Vital signs on presentation were normal and physical exam was unremarkable. A complete infectious workup was negative including autoimmune testing for antineutrophil cytoplasmic antibody (ANCA) panel. However, the patient had profound eosinophilia. A computed tomography scan of the chest and abdomen revealed scattered ground-glass opacities in bilateral lungs and a moderate amount of fluid throughout the colon. Upper and lower endoscopy with biopsy demonstrated chronic inflammation with marked eosinophilia in lamina propria of the gastroesophageal junction, gastric antrum, and duodenum. Nasal polyp biopsy results were obtained and consistent with vasculitis. The patient was diagnosed with chronic diarrhea secondary to montelukast induced EGPA. Montelukast was discontinued and his symptoms resolved with corticosteroids.
**Discussion:** EGPA is the rarest ANCA-associated vasculitis and affects small to medium-size vessels. It is characterized by eosinophilic granulomatous inflammation and an association with asthma and eosinophilia. Typical organs involved include peripheral nerves, paranasal sinuses, and lungs. Gastrointestinal manifestations are rare, and some studies have shown increased mortality in these patients. Numerous case reports have shown an association with montelukast and the development of EGPA. Glucocorticoid therapy leads to remission in a majority of patients, however, a relapsing disease course is common. Immunosuppressant maintenance therapy is controversial, and some studies have shown rituximab and mepolizumab to be effective in treating ANCA-positive and ANCA-negative EGPA, respectively.

S2462

**Hereditary Alpha Tryptasemia Syndrome (HαTS): An Autobiographical Case Report and Literature Review of an Under-Recognized Clinical Entity Emulating Irritable Bowel Syndrome (IBS) and Inflammatory Bowel Disease (IBD)**

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[1]Miami Miller School of Medicine, Miami, FL; [2]University of Miami, Miller School of Medicine, Miami, FL.

**Introduction:** HαTS is an autosomal dominant disease first characterized in 2014 by Lyons JJ. et al. and present in ~5% of the White population. HαTS is responsible for ~90% of patients in the western world with elevated basal serum tryptase (eBST). It modifies clonal and nonclonal mast cell (MC) disorders with increased prevalence and/or severity of anaphylaxis and MC mediator-related symptoms. We aim to present an autobiographical case report and peer literature review of HαTS.
**Case Description/Methods:** We conducted a review of peer literature selected using search terms: HαTS, Tryptase, IBS, IBD. Case: A 26-year-old male with a cc of change in bowel habits for 3 years, going from 1-2 formed stools to up to 6 loose bowel movements (BMs) per day. These are associated with excessive flatulence and crampy abdominal pain relieved by passage of BMs. GERD, nausea, and generalized pruritus worse at night which frequently awakens him from sleep. He also has chest and facial flushing with alcohol intake. No symptom relief from low-FODMAP or low-histamine diets, with relief of symptoms on a very low carbohydrate diet. Negative work up included allergy testing, celiac serology and HLA testing, brush border disaccharidases, CBC and CMP, thyroid panel, fecal elastase. VIP slightly elevated at 65 pg/mL (nl < 58.8 pg/mL). Endoscopy revealed small esophageal ulcer that resolved. Duodenal biopsies showed increased MC density at 24 per hpf (nl < 15 per hpf) and increased intraepithelial lymphocytosis. Colonoscopy with biopsies nl. Initial serum tryptase was 5.6 ng/mL, repeated 2 years was elevated to 11.4 ng/ml (nl < 6.5 ng/mL). Diagnosis: genetic PCR testing of buccal swab revealed extra-allelic copy of alpha tryptase gene on TPSAB1 gene locus, consistent with HαTS. Literature Review: HαTS presents with variable multisystemic symptoms with 1/3rd of patients asymptomatic, 1/3rd have mild disease, and 1/3rd have severe disease. Flushing, pruritis, dysautonomia, and symptoms emulating IBS and IBD with diarrhea present in 30-50%. Other symptoms may include food intolerances, IgE-mediated food allergies, neuropsychiatric symptoms, and joint hypermobility (**Table**).
**Discussion:** HαTS should be considered in the differential for symptoms of IBS and IBD—especially when flushing, pruritis, or dysautonomia are present. This will prevent delay of diagnosis and reduce total costs. Duodenal biopsies showing increased density of MCs and elevated serum tryptase ( >6.5 ng/mL) are suspicious for HαTS. Genetic testing is confirmatory.

**Table 1. Clinical features of HαTS, IBS, and IBD patients**

| Clinical Feature | HαTS | IBS | IBD |
|---|---|---|---|
| Age of Onset | Unknown | 20-30 y/o | Major peak 15-25 y/o, minor peak 50-70 |
| Male:Female | Male = Female | Female > Male | Male = Female |
| Western Prevalence | ~5% of Whites | 10-20% | 1.3% |
| Common Symptoms and Signs | Diarrhea predominant, crampy abdominal, pain, GERD, flushing, and pruritus | Diarrhea and/or constipation, crampy abdominal pain, GERD | Diarrhea, crampy abdominal pain, bloody stools, bowel fistulas, intestinal strictures/fibrosis, weight loss, anemia |
| Serum Lab Values | Elevated Basal Serum Tryptase (Suspicion Mild-Moderate 6.2-7.9 ng/mL; Suspicion High >8.0 ng/mL) | No significant findings | Elevated C-reactive protein, erythrocyte sedimentation rate, fecal calprotectin, and Iron deficiency |
| Genetic Characterization | Extra allelic copy of alpha tryptase encoding gene on TPSAB1 gene locus[2] | Normal | +/- Positive |
| Small Bowel Histology | Increased density of Mast Cells forming 2-15 cell clusters | No significant findings | Granulomas, Inflammation, crypt abscesses |
| Current Treatments | H1 and H2 antihistamines, Oral Cromolyn, Carbohydrate Restriction*, Compounded Oral Ketotifen**, Sub Q Omalizumab**, Other Biologics** | See 2019 ACG Clinical Guidelines for IBS | Immunomodulation Therapies |

*Treatment used successfully by patient in case study, but not described in literature.
**Treatments reported in literature with variable responses and not used by patient in case study).

S2463

**Immunosuppression in Chronic Total Parenteral Nutrition Patient Causing Invasive Pulmonary Candidiasis**

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**Introduction:** The complications of total parenteral nutrition (TPN) are well established in the literature, including a higher risk of fungal infections via immune system dysregulation, translocation, seeding, etc. We present a 42-year-old female requiring chronic TPN who was found to have cavitary pulmonary candidiasis.
**Case Description/Methods:** A 42-year-old female with ileocolonic Crohn's disease complicated by multiple bowel resections and Roux-en-Y bypass was evaluated inpatient for a 2-week history of daily fevers and nonproductive cough. The patient had a significant history of nutritional replacement therapy with failed percutaneous endoscopic gastrostomy requiring Mediport catheter placement for TPN feedings. Due to short gut syndrome, she was dependent on TPN. She had no history of malignancy or cardiac conditions, including valve abnormalities. CT scan revealed a 2x2 cm cavitary consolidation in the lingula and a 1.9x1.7 cm cavitating lesion in the left upper lung lobe. Voriconazole was initiated, but the patient continued to spike high fevers without improvement. The Mediport catheter was removed and sent for cultures. Bronchoscopy was also done. Mediport and bronchial wash cultures both grew Torulopsis glabrata. Fungal blood cultures were negative. After 4 weeks of appropriate antifungal therapy, the patient's fevers resolved and she was discharged home in stable condition.
**Discussion:** TPN predisposes to fungal infections by varied mechanisms. In our patient, given the anatomical path of the catheter, septic fungal emboli from the Mediport is the most plausible mechanism of invasive candidiasis. However, the vulnerability of our patients to fungal infections is likely because of gut disruption in the setting of short gut syndrome and long-term TPN dependence. The risk of infection with TPN occurs at both tissue and cellular levels. Loss of gut barrier function due to epithelial disruption and decreased IgA production is seen in chronic TPN users. Studies also show that TPN causes shifts in the gut microbiome resulting in decreased regulatory T-cells and dysregulation of toll-like receptors. Furthermore, some fungi, such as Candida species, can multiply in parenteral nutrition solutions in which even bacteria cannot grow. Some care centers prophylactically start antifungals in critically ill patients who require TPN, but data on the efficacy of prophylactic antifungal therapy for those requiring long-term TPN is needed.

S2464

**Misnomer Leading to Delayed Diagnosis of an Autoinflammatory Syndrome in a Latino Male**

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**Introduction:** Familial Mediterranean Fever (FMF) is a rare auto-inflammatory disease ordinarily found in patients of Mediterranean and Middle Eastern descent, however it can affect any ethnic group. Symptoms of FMF are non-specific and can mimic many common diseases, resulting in unnecessary workup, surgeries, and delayed diagnosis. The following case report demonstrates the ease of overlooking a classic presentation due to implicit bias, racial disparities, and a disease misnomer.
**Case Description/Methods:** We present a case of a 30-year-old Latino male who was evaluated for a 15 year history of episodic peritoneal type abdominal pain associated with constipation. The patient reported that his first attacks began at age 9, during which he had severe abdominal pain associated with bloating, constipation, chills and night sweats. The pain was so severe that he was unable to get out of bed or walk without hunching over. These attacks occurred twice a year, lasted 3-4 days, and severity was significantly exacerbated by stress. The patient had no relief from laxatives and prune juice taken for constipation. He stated that many family members in Mexico were diagnosed with irritable bowel syndrome (IBS) or "colitis nerviosa." He was also told that he had IBS-C, even though his constipation and pain resolved between attacks. He reported a normal colonoscopy at age 10, in addition to an appendectomy. During one attack, his inflammatory markers were found to be markedly elevated. The patient denied any associated chest pain, joint pain, or rashes. His parents were originally from Jalisco, Mexico, an area that was colonized heavily by the Spanish. His lab work and physical exam in between attacks were unremarkable. As the patient did not meet Rome criteria for IBS, and due to the suspicion of recurrent peritonitis, MEFV mutation testing was ordered. Genetic testing revealed that the patient was heterozygous for the M694I mutation. Due to classic presentation and supportive genetic testing, he was prescribed 1.2 mg of Colchicine daily. At 6 months follow up, he has had no further attacks.
**Discussion:** This case illustrates the implications of a misnomer as well as how racial disparities and implicit bias delayed diagnosis in a patient with classic presentation of FMF. The patient was untreated for over 2 decades and may have undergone an unnecessary appendectomy. It is important to remember that despite the name, FMF can affect any ethnic group and awareness should be raised about this treatable disease.

**S2465**

**Neurogastroenterology & Motility Manifestations After COVID-19 Infection: A Case Series**

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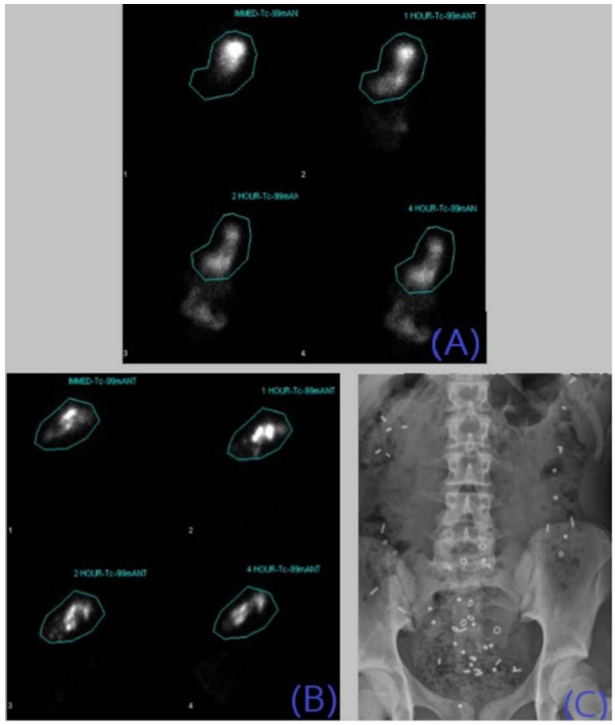
**Introduction:** In a subset of Covid19-convalescent patients, a multitude of long-term sequelae are increasingly being reported. We report 4 cases with varying neuro-GI and motility manifestations after recent COVID-19 infection.
**Case Description/Methods:** Case 1: A 23-year-old man contracted COVID-19 and had a protracted course of respiratory illness. Despite resolution of respiratory symptoms and dysgeusia, he continued to experience early satiety, postprandial nausea, vomiting and unintentional weight loss. Gastric Emptying Scan (GES) revealed gastroparesis (**Figure** A). Dietary modification and metoclopramide led to symptomatic improvement. Case 2: A 39-year-old woman with migraines, suffered from Covid-19 infection where anosmia and respiratory symptoms lasted for 2 weeks. Despite resolution of initial symptoms, she started experiencing nausea and vomiting, and reported stereotypical symptoms with complete absence of vomiting between episodes. Endoscopic examination, CT head and GES were normal. Urine tox screen was negative for cannabinoids. She responded favorably to amitriptyline and ondansetron. Case 3: A 47-year-old man started experiencing severe constipation associated with abdominal pain and bloating soon after being diagnosed with COVID-19. Three months after resolution of respiratory symptoms, in addition to constipation, he began reporting postprandial fullness, early satiation and epigastric pain. GES showed gastroparesis ( **figure** B) and a Sitzmarks® Study revealed delayed colonic transit (**Figure** C). Prucalopride was started, leading to improvement in symptoms. Case 4: A 74-year-old woman with obesity and diabetes, was hospitalized and intubated for severe respiratory distress due to COVID-19. After discharge, she had persistent symptoms of brain fog, fatigue, dyspnea as well as diarrhea and abdominal cramping, persisting despite loperamide and dicyclomine. *C. difficile* toxin, random colonic biopsies and $H_2$ breath test were unremarkable. Her symptoms eventually improved with rifaximin.
**Discussion:** We report 4 cases with post-COVID gastroparesis, cyclical vomiting syndrome, pan-gut dysmotility, and post-infectious IBS phenotypes.The pathophysiology of post-infectious-gut–brain disorders is still obscure. The current conceptual framework implicates acquired neuropathy, altered motility, intestinal barrier disruption and persistent intestinal inflammation. Similar pathophysiology may be involved in COVID-19 infection leading to sustained neurogastroenterological dysfunction and gut dysmotility.




[2465] **Figure 1.** Image (A) showing delayed gastric emptying with 64% retention at 4 hours. Images (B) and (C) both from Case-3 showing markedly delayed gastric emptying with 86% retention at 4 hours and abnormal colonic transit study with X-ray showing large amount of stool and retention of markers throughout the entire colon, in the same patient.

**S2466**

**Pelvic Floor Dysfunction as a Cause of Chronic Intestinal Pseudo Obstruction**

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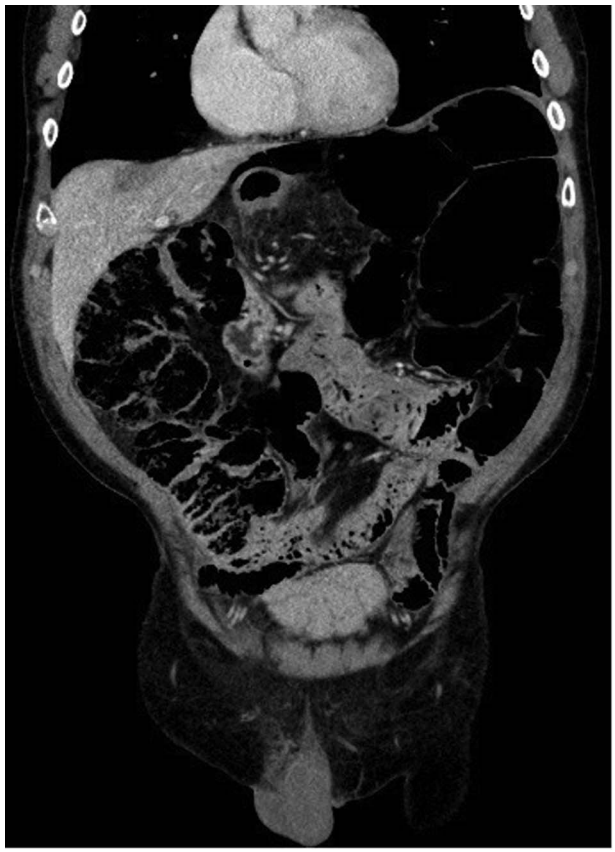
**Introduction:** Chronic intestinal pseudo-obstruction (CIPO) is a rare motility disorder with a variety of secondary causes, including genetics, metabolic disease, paraneoplastic syndromes, inflammatory disorders, and neurologic disorders. We present a case of CIPO secondary to pelvic floor dyssynergia.

**Case Description/Methods:** A 64 year-old male with a past medical history of hypertension, atrial tachycardia, and heavy alcohol use was referred for further GI workup by his primary care physician following a 5 month history of worsening constipation after a viral infection. Prior to the infection, he had a bowel movement 2-3 times per week. However, he now describes one Bristol 4 or 7 bowel movement with straining per month. Symptoms were refractory to Dulcolax and Colace. Initial workup included a normal extended electrolyte panel, normal TSH, and negative Celiac disease serologies. A CT abdomen and pelvis revealed extensive dilation of proximal colon, compression of descending and sigmoid colon, and small bowel fecalization. A representative coronal image is shown in **Figure** 1. Subsequent colonoscopy did not reveal any evidence of obstruction. Anorectal manometry (ARM) findings included elevated resting pressure (74.94 mmHg), decreased sensation, paradoxical contraction with Valsalva, and inability to pass the balloon catheter after 2.5 minutes. Normal rectal capacity (280 mL) and excellent squeeze pressure but paradoxical contraction with Valsalva is consistent with pelvic floor dyssynergia. Biofeedback therapy was recommended.
**Discussion:** Evaluating for secondary causes of CIPO (i.e., obstruction, metabolic disturbances, autoimmune disorders, neurologic disorders, and musculoskeletal disorders) is an important diagnostic step. Key tests include imaging, colonoscopy, electrolytes and autoimmune panels, and anorectal manometry. This case highlights long-standing pelvic floor dyssynergia as an underlying cause of CIPO.



[2466] **Figure 1.** CT abdomen and pelvis with extensive dilation of proximal colon and small bowel fecalization.

**S2467**

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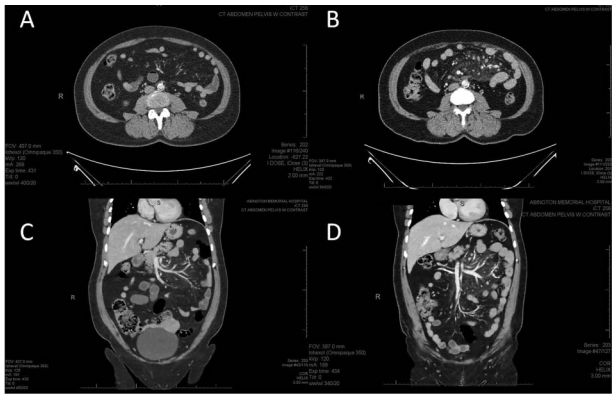
**Sclerosing Mesenteritis: A Case Report in a Young Female**

*Kyle L. Marrache, DO[1], Stephanie Tzarnas, MD[2].*
*[1]Abington-Jefferson Health, Abington, PA; [2]Abington-Jefferson Hospital, Abington, PA.*

**Introduction:** Sclerosing Mesenteritis (SM) is a rare fibrotic inflammatory condition of mesenteric adipose tissue. It is of clinical significance since it appears to carry a 5 times higher risk of malignancy. In this case report we present a female with both clinical symptoms and radiographic findings suggestive of SM.
**Case Description/Methods:** A 58-year-old female presented to the resident clinic with the complaint of epigastric pain. On exam she had abdominal distention and tenderness over the epigastrium. Shortly after evaluation in the clinic the patient presented to the ED twice in 2 weeks with worsening abdominal pain where she got a CT scan both times demonstrating generalized haziness and interval enlargement of mesenteric lymphadenopathy. Colonoscopy and EGD for her ongoing abdominal pain which were both unremarkable. Duodenal and stomach biopsies were normal. AST, ALT, CRP, ESR, and CBC were all unremarkable. MRI of the pelvis as well as MR enterography was performed and this showed unchanged haziness of the mesentery but found the lymph nodes were not enlarged. The patient was seen by oncology who agreed that her lymphadenopathy had largely resolved, and it was unlikely she had an underlying malignancy. Surgery was consulted to evaluate for biopsy but felt given the clinical picture of SM was clear it was not warranted. The patient was seen in the clinic again and started on a regiment of colchicine. She currently has follow-up scheduled.
**Discussion:** Risk factors for SM are abnormal post-surgical healing, autoimmune mediated, and malignancy. Our patient did have prior abdominal surgery but no other risk factor. Studies have shown a connection with a variety of malignancies, but no statistically significant correlation. The interval enlargement in lymph nodes noted on our patient was concerning but this had resolved on MRI. SM is frequently found incidentally on CT scan. Findings suggestive of SM on CT are a "fat ring sign" or "pseudo-capsule" formation. More rarely "misty mesentery" can be seen which is the generalized haziness seen in our patient (**Figure**). Confirmation of SM is by biopsy. In our patient it was felt that the imaging was consistent enough to not warrant a biopsy. Treatment includes prednisone or colchicine. We will continue to follow our patient and see if she has a response to the colchicine with the possibility of starting prednisone in the future if there is no response.



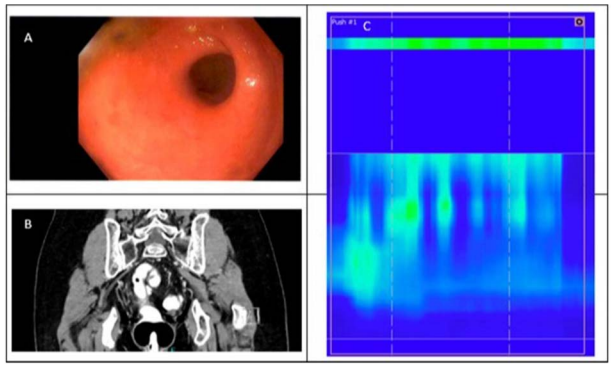

[2467] **Figure 1.** A: Axial cut from Aug. B: Axial cut from Sep. C: Coronal cut from Aug. D: Coronal cut from Sep.

**S2468**

**Stop, Drop, and Roll: One Woman's Pelvic Floor Disease Journey**

*David Lehoang, MD, MBA, Sean Dewberry, Niharika Mallepally, MD, MPH, Christine Hsieh, MD, Sonia Sharma, MD.*
*Keck School of Medicine, University of Southern California, Los Angeles, CA.*

**Introduction:** Pelvic floor disease includes symptoms of the anterior (urinary, vaginal) and posterior (anorectal) pelvic floor. This case describes a woman who developed dyssynergic defecation after surgical management of urinary incontinence. The diagnostic route taken to reveal the unusual cause of her symptoms is a lesson in how to evaluate ambiguous gastrointestinal(GI)/genitourinary symptoms.
**Case Description/Methods:** A 69-year-old woman presents with bloating and difficulty defecating for 2 years. She senses incomplete evacuation, passing 1-2 thin stools per day despite an extensive bowel regimen (e.g. posturing, straining). Two years ago, she underwent sacrocolpopexy and mesh placement to treat pelvic organ prolapse. Her urinary incontinence improved but constipation worsened. She was thus evaluated by GI- many CTs showed nonspecific sigmoid (SC) thickening. A colonoscopy noted SC narrowing; mucosa pathology was normal. She came to our clinic to investigate further. Digital rectal exam had a normal tone with widening of the vault with strain. Anorectal manometry (ARM) showed dyssynergic defecation and unsuccessful balloon expulsion, consistent with mechanical obstruction. A flexible sigmoidoscopy showed benign-appearing extrinsic compression at the rectosigmoid junction. The SC also had a large stool burden despite 2 pre-procedure enemas. Given a concern for mass effect of the mesh into the colon, an exploratory laparoscopy was performed, showing a redundant SC with scarred, edematous mesentery. A partial sigmoidectomy was performed and adhesions at the anterior rectal wall (near the sacrocolpopexy mesh) were lysed. In just 3 months, the patient's symptoms resolved. Repeat ARM had a normal defecation pattern and balloon expulsion (**Figure**).
**Discussion:** Postoperative adhesions are present after 63-97% of open abdominal surgeries. The most common complication of such adhesions is bowel obstruction, occurring in 15% of patients within 1 month of surgery. Our patient had an even more unique complication- constipation and dyssynergic defecation. Literature on post-op adhesions and defecation dysfunction mainly describes surgery of the rectal sphincter. In contrast, the sacrocolpopexy did not involve the rectum or surrounding peritoneum, but caused adhesions with a mass effect on the rectum. Given the morbidity associated with lower abdominal adhesions, suspicion must remain high in populations with a history of complicated abdominal surgery- especially for treatment of anterior pelvic floor disease.




[2468] **Figure 1.** A. Flexible sigmoidoscopy: Very narrowed lumen in the sigmoid colon. B. CT abdomen and pelvis: Coronal view showing sharp angulation in the sigmoid colon (due to the mass effect of the mesh and adhesions) C.Anorectal manometry: The Y-axis notes length (cm) from anal sphincter (0cm) into sigmoid. The X-axis shows time (in seconds). The color of the data points denotes pressure exerted. Here, the top horizontal line in green shows rectal pressure. The same amount of pressure is shows in the bottom "green" areas, indicating that anal tone faily to appropriately relax. This increase in anal pressure with a good push, indicating dyssynergic defecation pattern.

**S2469**

**The Big Stretch: A Report of Three Cases of Gastroparesis as an Uncommon Complication of Hypermobile Ehlers-Danlos Syndrome**

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**Introduction:** Ehlers-Danlos syndrome (EDS) is a rare autosomal dominant connective tissue disorder that affects the gastrointestinal system, skin, joints, and vasculature. Hypermobile EDS (hEDS) is a subset of EDS with a prevalence of 1 in 5000 people. Major GI features of hEDS include abdominal pain, nausea, constipation, heartburn, and irritable bowel syndrome-like symptoms. Gastroparesis is a debilitating disorder defined as delayed gastric emptying in the absence of a mechanical obstruction. However, hEDS as a cause of gastroparesis is not well established in the literature. Here we report 3 cases of gastroparesis in patients with hEDS to demonstrate its uncommon and varying presentations.
**Case Description/Methods:** Our first patient is a 20-year-old woman who was diagnosed with hEDS at a young age and then developed severe epigastric postprandial abdominal pain with bloating, intractable nausea, early satiety, and unintentional weight loss. She had multiple ER admissions over the past few years for similar symptoms. Physical examination revealed hyperflexible joints, normal bowel sounds with nontender abdomen. Lab values were within normal limits. Esophagogastroduodenoscopy (EGD) and gastric emptying studies both confirmed Grade III gastroparesis. She was initially started on Metoclopramide and Ondansetron without improvement. Patient ultimately achieved symptom control with Azithromycin, Ondansetron, Promethazine as needed and diet modifications. The second patient is a 24-year-old man who was also diagnosed with hEDS at an early age and presented with recurrent postprandial abdominal pain with bloating and heartburn, but no weight loss. He underwent EGD and was found to have gastritis, hiatal hernia and reflux esophagitis. Subsequent gastric emptying scintigraphy later confirmed Grade II gastroparesis and his symptoms improved with diet modifications as well as

Metoclopramide. Lastly, our third patient is a 26-year-old woman also with known history of hEDS who presented with mild intermittent postprandial abdominal pain and abdominal bloating. Prior work-up with EGD and gastric emptying scans confirmed the diagnosis of Grade I gastroparesis. Fortunately, her symptoms clinically resolved with regular cannabis use.
**Discussion:** Gastroparesis should be considered in patients with hEDS complaining of severe postprandial abdominal pain. Due to its uncommon presentation and multi-system organ involvement, EDS should be considered in patients with idiopathic gastric dysmotility and joint hypermobility.

**S2470**

**Unusual Suspect: Diarrhea and Shock Secondary to Pheochromocytoma and VIPoma**

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*Coney Island Hospital, Brooklyn, NY.*

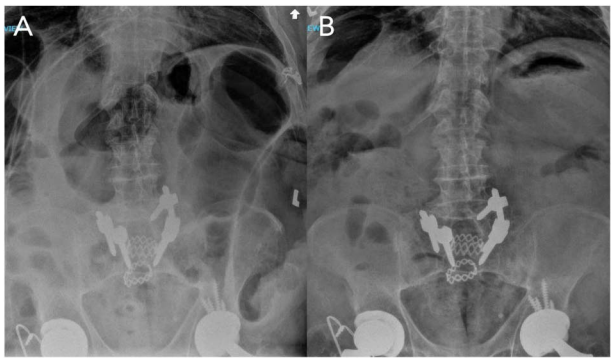
**Introduction:** Pheochromocytoma is a catecholamine secreting tumor that is found at a rate of 0.0008% annually in the general population. Vasoactive intestinal peptide, also known as vasoactive intestinal polypeptide or VIP, is a peptide hormone that enhances cardiac contractility, produces vasodilation, increases glycogenolysis, reduces arterial blood pressure, and relaxes the smooth muscle of the trachea, stomach, and gallbladder. Moreover, VIP may lead to the secretion of water and electrolytes, hypokalemia, and flushing. This case presentation involved a patient with an atypical pheochromocytoma, suspected of producing VIP.
**Case Description/Methods:** A 51-year-old male presented with palpitations, diaphoresis, nausea, and intermittent substernal chest pain that exacerbated when he lied on his right side. Hypertension and left adrenal mass are among his medical history. He developed emesis, stomach pain, distention, and profuse secretory diarrhea. On day 4 of his hospital stay, this resulted in hypovolemic shock due to a 5L/day output. He further developed metabolic acidosis, hypokalemia, and his EKG revealed intermittent ventricular tachycardia and T wave inversion. Plasma metanephrines were 5679 pg/mL, VIP was 239 pg/mL, and gastrin was 313 pg/mL, according to an endocrine examination. CT scan revealed a 5 cm adrenal mass in the gastro-entero-pancreatic area; pheochromocytoma was then confirmed with the positive metanephrines. Patient finally underwent adrenalectomy.
**Discussion:** Pheochromocytoma is a rarely identified and diagnosed tumor in the majority of patients. The classical presentation of pheochromocytoma is typically an episodic headache, diaphoresis, headache, and tachycardia. On rare occasions, these tumors may also secrete Vasoactive intestinal peptide (VIP). VIP can result in the relaxation of smooth muscles of the GI tract along with peripheral vasodilation and hypovolemia. This patient presented with atypical pheochromocytoma, suspected of secreting VIP. Due to the VIP secretion, the patient developed copious secretory diarrhea, leading to hypovolemia and in turn leading to hypokalemia. The hypokalemia resulted in the development of Ventricular tachycardia. This presentation leads to the conclusion that it is imperative to test metanephrines and image catecholamine-secreting-tumors if suspected in a patient. Pancreatic VIPomas must also be ruled out as elevated VIP levels may be due to pheochromocytomas and be detrimental to the patient's health.

**S2471**

**Using Prokinetic Agents in Chronic Intestinal Pseudo-Obstruction (CIPO)**

*Yushan Wang, MD[1], Saleh Alghsoon, MBBS[1], Suma Gondi, MD[1], John Roberts[2], Raffi Karagozian, MD[1].*
*[1]Tufts Medical Center, Boston, MA; [2]Tufts School of Medicine, Boston, MA.*

**Introduction:** Chronic intestinal pseudo-obstruction (CIPO) is a rare disease. Dilatation may involve the colon or small bowel and is usually due to an underlying neuropathic disorder. Clinical manifestations of CIPO include abdominal distention, bloating, and pain, which can be acute, chronic, or recurrent. Diagnosis of CIPO should be suspected in patients with these symptoms for at least 3 months in the absence of a mechanical cause. Diagnosis requires exclusion of mechanical obstruction and other causes of dysmotility (**Table**).
**Case Description/Methods:** This is a case of a 62-year-old male presenting with 3 weeks of worsening abdominal pain and distention. History includes colon cancer with sigmoid resection/colostomy reversal, alcohol use disorder, and L5-S1 fusion on chronic opioids. He had 2 recent hospitalizations for similar symptoms thought to be due to non-obstructive ileus. At that time, colonoscopy showed significant colonic dilation with no masses or strictures. He was endoscopically decompressed and started on methylnaltrexone with relative improvement during a prior hospitalization. On this presentation, he had a massively distended abdomen with minimal tenderness. He was initially managed conservatively with bowel rest, rectal tube, and avoidance of opioids. However, serial abdominal X-rays showed no improvement in dilation (**Figure**). He was started on pyridostigmine with significant improvement in dilation and had normal bowel movements prior to discharge.
**Discussion:** This patient had recurrent CIPO with multiple admissions without complete resolution of previously suspected non-obstructive ileus despite conservative management and methylnaltrexone. Imaging was consistent with severe colonic dilation. As the patient had no improvement with conservative management, he was started on a trial of pyridostigmine after which he had significant improvement of bowel dilation on X-ray with normal bowel movements, illustrating the role of prokinetics in treating suspected CIPO. Knowledge of CIPO is important to prevent delays in diagnosis. Intervention focuses on diet and treatment of the underlying disease. For patients with symptoms despite dietary modifications, prokinetics such as prucalopride or pyridostigmine can be used for symptomatic relief. Pyridostigmine has demonstrated efficacy in the chronic phase of CIPO in small observational studies and is more commonly used in pediatric CIPO. Through this case, prokinetic agents show promise for broader use in adult CIPO cases.




[2471] **Figure 1.** Abdominal X-Rays of patient before (A) and after (B) use of pyridostigmine showing improvement of colonic pseudo-obstruction.

**Table 1. Acute Intestinal Pseudo-Obstruction vs Chronic Intestinal Pseudo-Obstruction (CIPO)**

| | Acute Intestinal Pseudo-Obstruction | Chronic Intestinal Pseudo-Obstruction |
|---|---|---|
| Prevalence | 100 per 100,000* | 0.80-1.00 per 100,000 |
| Course | Acute | Chronic |
| Presentation | Abdominal distention<br>Cramping pain<br>Nausea/vomiting | Abdominal distention<br>Abdominal pain<br>Bloating |
| Anatomic involvement | Colonic dilatation, usually cecum, right colon | Colonic or small bowel dilatation |
| Pathophysiology | Multifactorial<br>Autonomic dysfunction strongly implicated | Neuropathic disorder of enteric or extrinsic nervous system<br>Myopathic disorder of smooth muscle<br>Malfunction of interstitial cells of Cajal |
| Management | Fluid resuscitation, correction of electrolyte abnormalities, avoidance of opioids/ anticholinergics<br>Ambulation, bowel rest<br>Decompression with nasogastric or rectal tubes<br>Pharmacologic treatment with neostigmine<br>Operative intervention in cases of colonic perforation or ischemia | Dietary modification, treatment of underlying disease<br>Prokinetics, such as Prucalopride, for symptomatic relief (Grade 2C)<br>Pyridostigmine in chronic phase of CIPO |

*Inpatient admissions.

**S2472**

### A Case of Functional Bowel Disease Misdiagnosed as Carcinoid Syndrome

*Shane Quo, DO[1], Wisam Zakko, MD[2].*
*[1]University of Miami Holy Cross Hospital, Fort Lauderdale, FL; [2]Broward Health, Fort Lauderdale, FL.*

**Introduction:** Carcinoid tumor is a well-differentiated neuroendocrine tumor (NET) that arises from enterochromaffin cells. NETs produce serotonin which is metabolized to the inactive 5-hydroxyindoleacetic acid (5-HIAA) by the liver and the lungs. Carcinoid syndrome results from the secretion of serotonin into the systemic circulation by a metastatic NET that originates from the midgut. The primary screening method for carcinoid syndrome is 24-hour urinary 5-HIAA (U-5HIAA) which has a 90 percent sensitivity and specificity in diagnosing carcinoid syndrome. This case illustrates a unique scenario of a markedly elevated U-5HIAA not due to NET.
**Case Description/Methods:** A 29-year-old female presented to our clinic with abdominal pain, bloating, diarrhea, and weight loss associated with postprandial facial flushing. Medications included Dextroamphetamine and Fremanezumab. Based on her symptoms, U-5HIAA was ordered and was elevated at 148 mg (normal < 6). Carcinoid Syndrome was suspected based on symptoms and markedly elevated U-5HIAA. Imaging studies were ordered to identify primary or metastatic NET. These included negative CT abdomen/pelvis and a negative Ga68 Dotatate NETSPOT PET scan. Upper endoscopy and Colonoscopy were also negative. The patient expressed difficulty in stopping dextroamphetamine for the urine collection but due to the negative evaluation for NET she finally agreed to hold it for 3 days. Repeat U-5HIAA decreased down to 10.4 mg. Her symptoms were then believed to be secondary to functional bowel disease. The patient's symptoms improved with mirtazapine and dicyclomine, and she regained the weight.
**Discussion:** This is a unique case with a misleading U-5HIAA. While mildly elevated levels of U-5HIAA can be seen with tryptophan rich foods and certain drugs, including amphetamines, marked elevation, like in our case is more specific and usually seen with the carcinoid syndrome. Our patient had symptoms suspicious of carcinoid syndrome and a U-5HIAA of 148 mg, which is 25 times the upper normal. However, there was no evidence for NET on extensive work up, and the levels rapidly decreased with stopping dextroamphetamine. To our knowledge, this is the highest falsely positive U-5HIAA reported. This case shows the importance of a complete drug history, including over-the-counter medications. When the 5-HIAA is elevated, the test needs to be repeated after discontinuing all potentially interfering medications.

**S2473**

### A Case of Familial Mediterranean Fever Diagnosed by Abdominal CT Scan in a Patient Previously Diagnosed With Functional Abdominal Pain

*Eduardo Villa, MD[1], Wisam Zakko, MD[2], Mary Parianos, MD[3], Shane Quo, DO[3].*
*[1]University of Miami, Fort Lauderdale, FL; [2]Broward Health, Fort Lauderdale, FL; [3]University of Miami Holy Cross Hospital, Fort Lauderdale, FL.*

**Introduction:** Familial Mediterranean fever (FMF) is a hereditary autoimmune disease in which patients present with periodic, self-limited episodes of fever and serositis. It is prevalent in individuals of Middle Eastern origin. Since no definitive diagnostic test is available, FMF is suspected based on clinical symptoms; there are major and minor criteria to aid in making the diagnosis. Imaging modalities are not included in these criteria. We present a patient whose only symptom was recurrent abdominal with no family history of FMF. At the time he was diagnosed with Functional Abdominal Pain. FMF was suspected based on unexplained focal peritonitis on computerized tomography (CT).
**Case Description/Methods:** A 45-year-old male of Middle Eastern descent was seen in clinic complaining of episodic right upper quadrant (RUQ) abdominal pain since he was 17 years old. The pain occurred once or twice per month. Over the years he had multiple visits to the emergency department and extensive work up including endoscopic evaluation and imaging studies. He underwent cholecystectomy, and bilateral inguinal hernia repair without benefit. He was diagnosed with Functional Abdominal Pain then. When evaluated in our clinic, lab work showed mild iron deficiency anemia; otherwise, unremarkable. Upper endoscopy, colonoscopy, small bowel series and capsule endoscopy were normal. 3 months later he presented to our ER with an acute episode of severe RUQ pain. He was afebrile; abdominal exam was remarkable for localized tenderness in the RUQ. Lab work was remarkable for an elevated erythrocyte sedimentation rate of 31. Abdominal CT was only remarkable for fat stranding around the hepatic flexure and the proximal half of the transverse colon. Colonoscopy was normal with no evidence for segmental colitis to explain the inflammatory changes. Given the history of recurrent abdominal pain, ethnicity, and focal unexplained peritonitis on CT scan, FMF was suspected, and treatment with colchicine was initiated without recurrence of symptoms for now several years since the diagnosis, meeting diagnostic criteria for FMF.
**Discussion:** If not considered, FMF can remain misdiagnosed for many years leading to unnecessary procedures and surgeries. This was an unusual presentation with afebrile episodes of intermittent RUQ pain with the diagnosis ultimately suspected by CT imaging and confirmed by response to colchicine. Clinicians should consider FMF in patients of high-risk ethnic origin presenting with unexplained focal peritonitis on CT scan.

**S2474**

### Ankylosing Spondylitis Improved Following Faecal Microbiota Transplantation: Two Case Reports

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**Introduction:** Ankylosing spondylitis (AS) is an autoimmune disease which causes inflammatory arthritis in the spine and sacroiliac joints. Symptoms of AS include back pain, stiffness and reduced mobility in the spine. Patients with AS are known to have subclinical gut wall inflammation and dysbiosis in the gut. There is no known treatment to cure AS. Here, we report incidental improvement in AS in 2 patients with irritable bowel syndrome predominant diarrhoea (IBS-D) who received faecal microbiota transplant (FMT).
**Case Description/Methods: Case one**: Female, 47 years of age, with known IBS-D, presented with recurrent abdominal pain, explosive diarrhoea 5 time a day, nocturnal bowel motions and urgency. At the same time, she was on salazopyrin and arava for her AS with limited improvement in symptoms. Stool testing was for positive *Clostridium difficile* toxin and she received 2 fresh FMT's (one via colonoscopy, one via enema) in 2016. Testing 1 month after FMT treatment was negative to *Clostridium difficile* toxin. The patient had no ongoing gastrointestinal (GI) symptoms. She also reported significant improvement in AS symptoms (specifically reduction in excruciating pain in lower back and hip). After 5 years, her IBS-D symptoms relapsed and she had another 2 FMTs (one via colonoscopy, one via enema) which resulted in improvements in GI symptoms and more improvement in AS than previous FMTs. Her CRP and ESR levels were normal pre and post treatment.
**Case 2:** Male, 68 years of age, with known IBS-D received fresh 2 FMTs (one via colonoscopy, one via enema) in 2005 for treatment of IBS-D. Prior to treatment he had abdominal pain, cramps and loose motion and AS symptoms including excruciating pain in the sacroiliac joint. He was on infliximab and analgesics for his AS. After FMT treatment, he reported resolution of all GI symptoms and AS symptoms. His pre and post CRP levels were 100 and 2.6 and ESR levels were 18 and 7 respectively. In 2009, he had antibiotic treatment for sinusitis and his GI symptoms relapsed. He had a further 5 FMTs, again with complete recovery of GI symptoms and continued AS remission.
**Discussion:** We observed short-term and long-term improvement in AS symptoms after treating patients with FMT. Future prospective trials are required to confirm FMT as a treatment for AS.

## GENERAL ENDOSCOPY

**S2475 Presidential Poster Award**

### Outside In: Endometriosis of the Appendix, Cecum, and Ileum Masquerading as Suspected Crohn's Disease

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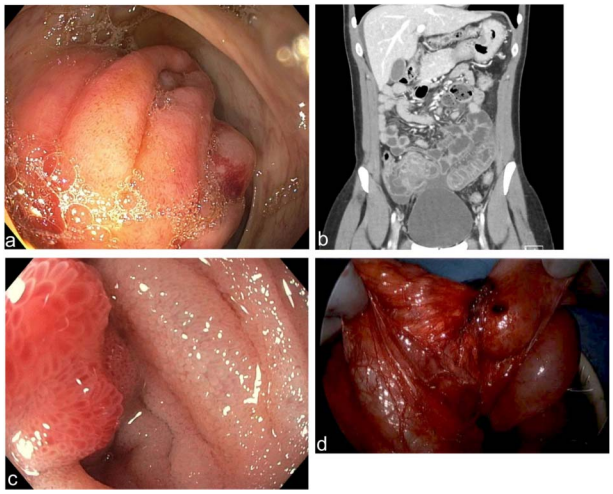
**Introduction:** Extra-pelvic endometriosis is a rare condition that may present with nonspecific abdominal pain, diarrhea, and/or hematochezia, which can mimic symptoms associated with inflammatory bowel disease. We present a case of suspected Crohn's Disease (CD) in a patient who subsequently was found to have extra-pelvic endometriosis of the appendix, cecum, and terminal ileum (TI).
**Case Description/Methods:** A 34-year-old female presented with abdominal pain and intermittent hematochezia that was sporadically associated with her menstrual cycles. Her fecal calprotectin (FC) was 510 mcg/g but her other inflammatory markers and labs were normal. Abdominal and pelvic computed tomography (CT) showed ileitis. Index colonoscopy showed inflammation of the appendiceal orifice and focal erythema in the TI. Biopsies of those areas were entirely normal. Magnetic resonance enterography (MRE) revealed an inflammatory conglomeration of the appendix, cecum, and TI without a definitive fistula. Repeat colonoscopy showed similar findings with mild architectural distortion of the appendiceal orifice. She then received an empiric antibiotic course for possible chronic appendicitis without alleviation of symptoms and persistent findings on repeat MRE. Subsequent exploratory laparoscopy revealed chocolate-colored lesions deposited throughout the pelvis and at the confluence of the terminal ileum and cecum consistent with endometriosis. After intraoperative consultation with gynecology, a decision was reached for definitive management with ileocecectomy. She reported improvement of symptoms after surgery (**Figure**).
**Discussion:** Extra-pelvic endometriosis accounts for 9% of endometriosis. Endometriosis of the appendix as a cause of acute appendicitis is rare and constitutes less than 1% of pathologies mimicking a clinical presentation of acute appendicitis. MRE can be useful in the diagnostic evaluation of endometriosis of the appendix, cecum, and TI but may be limited due to peristaltic artifacts and bowel contents. Laparoscopic intervention of endometriosis has been shown to improve symptoms. Our patient's presentation of abdominal pain with hematochezia, elevated FC, and ileitis on CT, was concerning for CD. Although endoscopic findings were suspicious for Crohn's disease, histological assessment was not, which contributed to the diagnostic dilemma in this case. Endometriosis should be on the differential in female patients presenting with intermittent abdominal pain, especially when the pain is associated with menstrual cycles.




[2475] **Figure 1.** Endometriosis of the appendix, cecum, and terminal ileum. a, Erythema and edema of the appendiceal orifice on first colonoscopy. b, Computed tomography of abdomen and pelvis with contrast showing ileitis. c, Focal area of edematous and erythematous mucosa within the terminal ileum on second colonoscopy. d, Terminal ileum and cecum with endometriotic cysts during laparotomy.

**S2476**

### COWden Syndrome: A Rare Spotting

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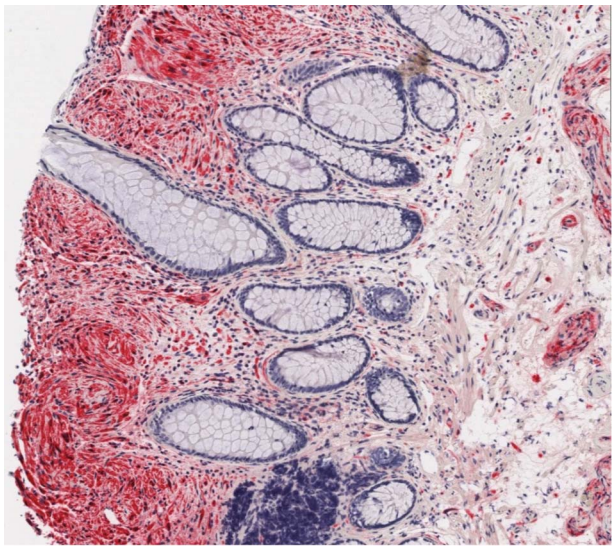
**Introduction:** Cowden syndrome (CS) is a rare autosomal dominant disorder characterized by multiple hamartomas in any organ throughout the body. Patients with CS are at an increased risk of developing various other cancers. Below, we describe a unique case of diffuse ganglioneuromas found in the duodenum of a patient with a PTEN mutation and established diagnosis of CS.
**Case Description/Methods:** A 40-year-old man was referred to the gastroenterology clinic for upper endoscopy and colonoscopy after recently being diagnosed with CS based on the PTEN gene mutation. Physical exam findings were significant for frontal macrocephaly and hyper-extendable joints. Laboratory markers were unremarkable. Colonoscopy revealed multiple small and large polyps; upper endoscopy showed numerous gastroduodenal polyps, which were removed.Polypoid-appearing duodenal mucosa was biopsied and pathology findings confirmed ganglioneuroma via positive S-100 stain (**Figure**).
**Discussion:** The mutated PTEN gene is responsible for the multiple hamartomas and possible neoplasm formation seen in CS. Classic manifestations of CS include variable expression of dermatologic manifestations. These include: oral papillomas, trichilemmomas on the face, sclerotic fibromas of the skin (found in 90% of patients). GI involvement of CS may include asymptomatic hamartomas, lipomas, and adenoma polyps (found in 80% of patients). GI ganglioneuromas are rare, well-differentiated benign tumors of the enteric nervous system that are predominantly found in the colon. Not commonly found, GI ganglioneuromas have been associated with CS and can remain asymptomatic until growing large enough to produce a mass effect. This cancause GI bleeding secondary to ulceration of the mucosa. Thus, when found, it is crucial these tumors are resected for excellent prognosis. Duodenal ganglioneuromas are very uncommon and there has been no previously reported association of them with CS. Limited case reports are available on duodenal ganglioneuromas, however, one case described treating the tumor similarly to other GI ganglioneuromas.This proved surgical resection is successful in eliminating recurrence of the tumor based on surveillance with monthly stool occult blood tests in the first year after diagnosis?, followed by annual endoscopies. In conclusion, the discovery of intestinal ganglioneuromas should make clinicians consider PTEN genetic testing. Endoscopists should consider performing random duodenal biopsies for patients with established Cowden's syndrome.



[2476] **Figure 1.** Histological Slide: S-100 immunohistochemistry of the duodenal polyp showing expansion of the lamina propria with the presence of diffusely positively stained ganglioneuronal cells.

**S2477**

**Gastric Xanthelasma: A Rare Endoscopic Finding**

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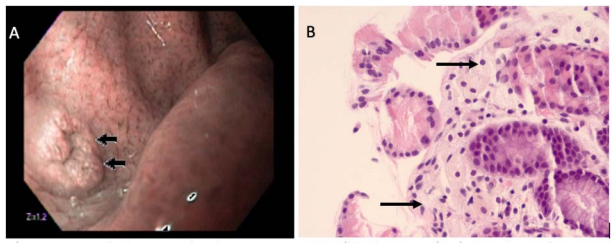
**Introduction:** Xanthelasmas are benign lesions which are most commonly found on the skin but can also be found in the gastrointestinal (GI) tract. The stomach is the most common site within the GI tract for xanthelasmas with a prevalence of approximately 0.2-0.8%. Endoscopically, they appear as plaque like yellowish-white lesions, and on histology are characterized by foamy lipid laden histiocytes. Xanthelasmas are generally asymptomatic, and thus are usually incidental findings on esophagoduodenoscopies (EGDs) performed for variety of other indications. While they are themselves benign, gastric xanthelasmas are associated with H. pylori associated gastritis, chronic gastritis, as well as gastric dysplasia, emphasizing the need to identify and diagnose this otherwise rare condition.
**Case Description/Methods:** We describe the case of a 43-year old male with medical history of hypertension and psoriatic arthritis who was referred for evaluation of dyspepsia and chronic heartburn. He was taking ibuprofen, celecoxib, hydrocodone-acetaminophen, losartan and was started on omeprazole prior to GI referral. Family history was notable for father with stomach cancer at age 48 and death at age 50. His vitals were within normal limits and his abdomen was soft, non-tender, non-distended. He underwent an upper endoscopy which showed a polypoid lesion in the fundus, but was otherwise unremarkable (**Figure** 1A). The polyp was biopsied and was suggestive of a xanthelasma, without evidence of metaplasia or dysplasia (**Figure** 1B). Given improvements in his symptoms, he was told to decrease the omeprazole dose and avoid NSAIDs. Follow up EGD at 6 months and 7 years showed no evidence of recurrence and screening colonoscopy at age 50 also showed no signs in the lower GI tract.
**Discussion:** Gastrointestinal xanthelasmas are rare, and tend to be more commonly found in women. They are usually incidental endoscopic findings and are generally benign. However, they can be associated with pre-cancerous states including gastritis and gastric dysplasia. A prior study has shown that the presence of a gastric xanthelasma was independently associated with gastric cancer with an odds ratio of 6.19. The location of a xanthelasma in the upper region of the stomach was also significantly associated with gastric cancer. This highlights the importance of identification of these lesions on endoscopy with subsequent biopsy for identification and closer monitoring.




[2477] **Figure 1.** A) Gastric xanthelasma in fundus on endoscopy (black arrows). B) Histology of gastric xanthelasma showing foamy lipid laden macrophages (black arrow).

**S2478**

**Blanching Gastric Mucosa: An Endoscopic Finding in a Patient With Celiac Artery Occlusion Leading to Vascular Insufficiency**

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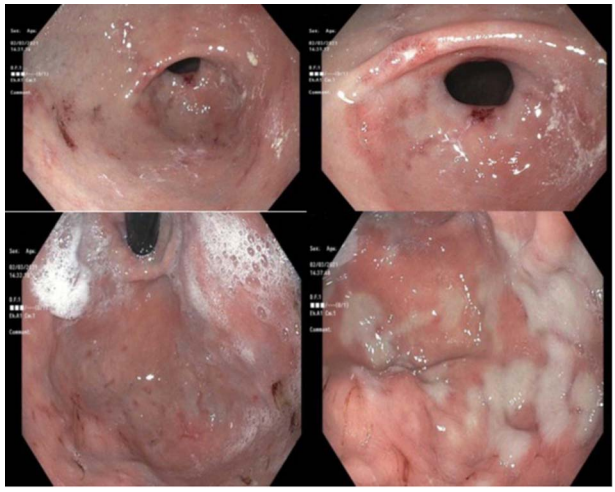
**Introduction:** Celiac artery occlusion is a significant and debilitating disorder usually characterized by abdominal pain and diagnosed via radiological imaging modalities. Endoscopic findings in this disease have not been clearly depicted. We present a case with significant endoscopic findings of patchy blanching gastric mucosa induced by insufflation during esophagogastroduodenoscopy (EGD).
**Case Description/Methods:** A 40-year-old woman with past medical history significant for hypertension, intracranial aneurysm with open clipping, coronary artery disease, polyarteritis nodosa presented to the emergency department (ED) complaining of intractable epigastric abdominal pain which had become progressively worse over the past month. Initially, the pain was described as intermittent and crampy which then became constant, 10/10 in intensity, associated with poor oral tolerance, nausea and non-bloody, non-bilious vomiting. Upon presentation vital signs were T 97.9 F, HR 96 bpm, BP 162/117 mmHg, SpO2 99% on room air. On physical exam, patient was ill appearing with diffuse abdominal tenderness with guarding and rebound tenderness, soft, non-distended, with bowel sounds appreciated. Computed tomography angiography (CTA) of the abdomen/pelvis revealed complete occlusion of the celiac artery at the origin and stenosis at the SMA origin with retrograde filling of the celiac territory via an enlarged IPDA collateral. The Gastroenterology service was consulted and an EGD was performed. Upon insufflation of air into the stomach, patchy blanching of the gastric mucosa was noted which continued to worsen, this immediately improved upon deflation. Superficial erosions were also seen in the entire stomach, otherwise the remainder of the exam was normal. Based on the patient's recent findings on CTA and gastric blanching observed during the EGD, she was assumed to have vascular insufficiency likely secondary to vasculitis in light of positive ANCA and lupus anticoagulant. After appropriate treatment the patient was discharged with rheumatologic follow up (**Figure**).

**Discussion:** This case is presented to educate physicians on a possible manifestation of vascular insufficiency on endoscopy and to always keep it in mind when evaluating a patient presenting with severe abdominal pain. Vascular insufficiency can manifest itself in many ways, but in our case, different clues led us to the diagnosis from her physical exam, to the findings seen on CTA and to the results appreciated on EGD.



[2478] **Figure 1.** Patchy blanching of the gastric mucosa with superficial erosions as demonstrated on EGD.

**S2479**

**Breast Cancer With Synchronous Endoluminal Upper and Lower GI Metastases**

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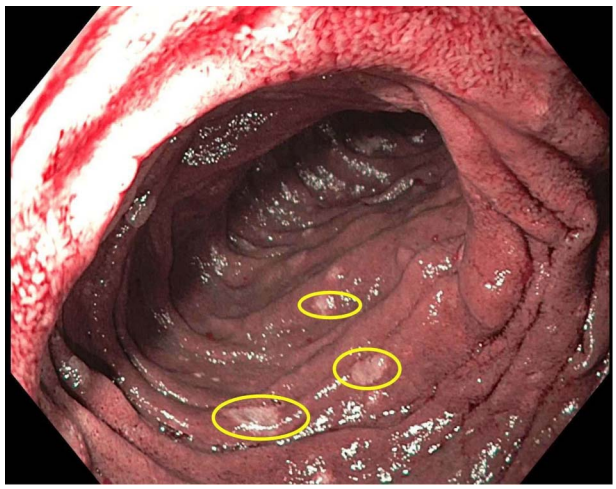
**Introduction:** Breast cancer is the most common cancer of women and spreads commonly to organs such as the lungs, bones, brain and liver. We present a case of a 61 year-old woman who had isolated upper and lower GI endoluminal metastases from a primary breast lobular carcinoma in situ (LCIS) lesion.
**Case Description/Methods:** A 61-year-old woman underwent screening mammogram that demonstrated focal asymmetry in the right upper outer quadrant. Breast US showed a BIRADS-4 lesion. Nodal biopsy was positive for Pleiomorphic LCIS Nuclear Grade 2 without invasive component. She then had lumpectomy. The LCIS was 0.1cm from the margin without invasive carcinoma present. She started raloxifene and underwent surveillance MRI and mammogram at regular intervals. Two years after diagnosis, she had a screening colonoscopy that showed a single hepatic flexure polyp. Pathology showed poorly-differentiated metastatic adenocarcinoma (GATA3+, CK+, ER weakly positive 2-5%, CK20-, TTF1-, PAX8- and CD56-), suggesting breast cancer origin. CT chest/abdomen/pelvis, bone scan, CT brain, breast MRI and PET scan were all negative for apparent metastatic disease. She then started letrozole and pabloclib. Repeat colonoscopy after 6 months did not show any change in the lesion. EGD was performed 3 years after first diagnosis and discovered white patches in the duodenum. Biopsies showed metastatic adenocarcinoma of breast origin. She continued letrozole and pabloclib. Serial CT scans and bone scans remained negative for metastatic disease. Surveillance EGD was repeated every 6 months and showed gastric and duodenal lesions that were biopsy-proven metastatic breast cancer. The lesions remained stable on treatment over a follow-up period of more than 3 years (**Figure**).
**Discussion:** Breast cancer is the most common cancer affecting women and is also the second most common cause of cancer death in women. Metastases to the gastrointestinal tract are rarely seen in clinical practice and when these are present, the stomach is the most common site of metastasis. Most patients (81%) with endoluminal GI metastases usually have other apparent metastatic disease. Isolated synchronous endoluminal GI metastases of both the small and large bowel are very rare with only one other case identified in the literature. Breast cancer with gastric metastases has a median survival of 24-36 months. Our patient survived and had stable disease for more than 3 years.



[2479] **Figure 1.** Duodenum examined using Narrow-Band Imaging (NBI) showing metastatic deposits (circled in yellow).

**S2480**

**Black Speckled Villi**

*Paul Aguilera, MD[1], Akshaj Pole, DO[1], Gregory Brennan, MD[2].*
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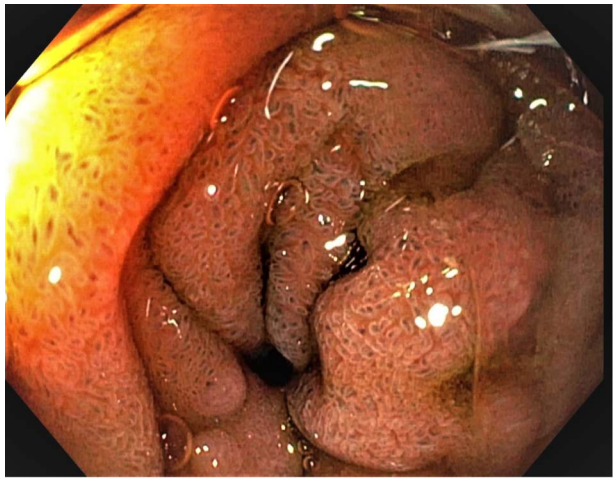
**Introduction:** Pseudomelanosis duodeni (PD) is a rare incidental finding characterized by brownish black pigmentation seen primarily in the first and second portions of the duodenum. The exact cause is unknown but it is associated with certain medications and chronic medical conditions. Here we present a case to help gastroenterologists recognize this rare endoscopic finding.
**Case Description/Methods:** A 49-year-old man with a past medical history of GERD, obesity, obstructive sleep apnea, type 2 diabetes and atrial fibrillation was referred for evaluation of rectal bleeding and iron deficiency anemia. His medications included iron supplementation, amlodipine, aspirin, atorvastatin, carvedilol, furosemide, and hydralazine. The cause of the patients rectal bleeding was determined to be from hemorrhoids found during colonoscopy. Esophagogastroduodenoscopy demonstrated diffuse mucosal changes characterized by speckled black pigmentation extending along the villi (**Figure**). Biopsies were taken and showed benign black pigmented macrophages in the lamina propria.
**Discussion:** Pseudomelanosis duodeni is a rare finding of unknown etiology. It is associated with chronic medical conditions including iron deficiency anemia, hypertension, diabetes, chronic kidney disease and common medications such as ferrous sulfate, hydralazine, propranolol, hydrochlorothiazide and furosemide. Despite the association with these common conditions and medications, PD remains a rare endoscopic finding. Why certain patients develop this finding is unknown. Interestingly, there is no association with anthraquinones as seen in pseudomelanosis coli. Histology typically shows black granular pigment inside macrophages within the tips of the villi. Although the endoscopic findings are striking, the condition has no clinical consequence and requires no treatment.



[2480] **Figure 1.** Pseudomelanosis duodeni.

**S2481**

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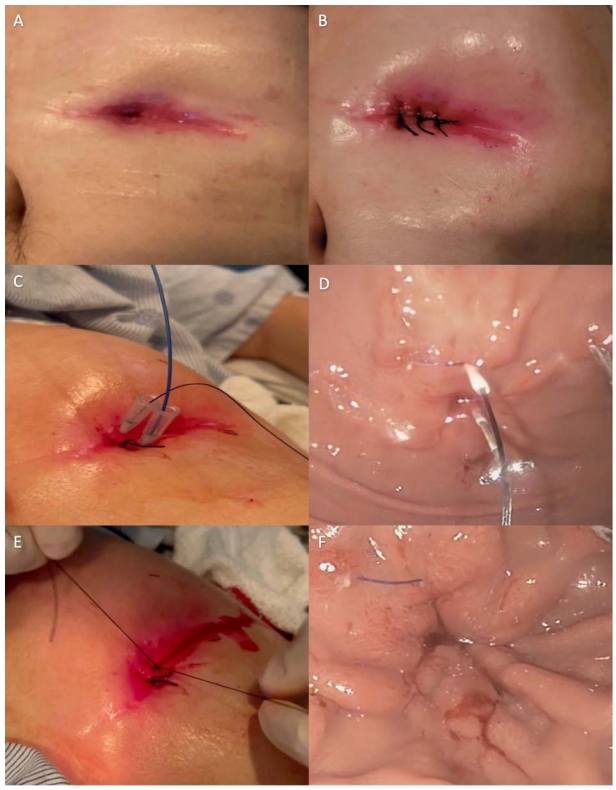
**Closure of Refractory Gastrocutaneous Fistula With Endoscopically Guided Percutaneous Suturing With the Use of SpyBite Biopsy Forceps**

*Rogelio Garcia, MD[1], Juan D. Gomez Cifuentes, MD[1], Tara Keihanian, MD[1], Marvin Ryou, MD[2], Wasif Abidi, MD, PhD[1].*
*[1]Baylor College of Medicine, Houston, TX; [2]Brigham and Women's Hospital, Boston, MA.*

**Introduction:** Persistent gastrocutaneous fistula (GCF) is a rare but well-known complication of long-term Percutaneous Endoscopic Gastrostomy (PEG) tube use. To avoid invasive surgery, endoscopic closure has been used as an initial step for treatment but is not always successful. We report a case of successful GCF closure with a novel endoscopically guided percutaneous suturing technique using the SpyBite biopsy forceps.
**Case Description/Methods:** Our case is a 28-year-old male with a history of cystic fibrosis (CF) complicated by malnutrition, requiring PEG tube placement since childhood. After starting CF therapy with elexacaftor/tezacaftor/ivacaftor and achieving optimal nutritional status, his PEG tube was removed. Unfortunately, he developed a persistent GCF. Initial attempts at closure with over-the-scope-clip and endoscopic suturing failed. The decision was made to proceed with GCF closure by endoscopically guided percutaneous suturing using the SpyBite forceps. Under endoscopic guidance, 2 16G long angiocaths were advanced into the gastric lumen, one caudal and one cranial to the fistula tract in a sterile fashion. A 2-0 silk suture was advanced through one angiocath and externalized using SpyBite biopsy forceps through the other angiocath. The angiocaths were then removed over the suture and the loop was tied down using a surgical knot. This process was repeated 2 more times along the fistula tract, 5mm from each other. Internal closure of the GCF was then performed using endoscopic suturing. One interrupted and 2 running sutures were placed along the border and cinched to reinforce the site. There were no immediate adverse events or delayed skin inflammation. The patient had no further leakage from the GCF site at follow-up 2 weeks later (**Figure**).
**Discussion:** With the emergence of novel CF therapies, the dependence on feeding tubes has decreased. Unfortunately, these patients are at high risk of GCF formation after PEG tube removal. Given the difficulty in closing GCF, we advocate a multimodality approach, as described here, using transcutaneous and endoscopic suturing. In previously described endoscopically guided percutaneous suturing, the suture loop is externalized through the GCF tract or the mouth. Our technique differs in using SpyBite forceps to externalize the suture through a second catheter. This method is simple and provides a safe and effective alternative for the closure of refractory GCFs.




[2481] **Figure 1.** A-B: Gastrocutaneous fistula before (A) and after closure (B). C-D: 2-0 silk suture inserted through the angiocath and SpyBite forceps inserted through the second angiocath. The suture was grasped with SpyBite forceps and pulled through the second angiocath to form a loop (C: external view, D: endoscopic view) E-F: The suture was pulled externally, and a surgical knot was performed (E: external view, F: endoscopic view).

**S2482**

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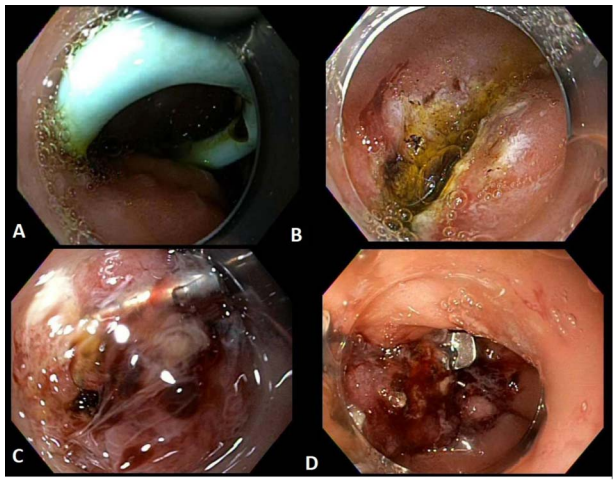
**Closure of Bilo-Entetric Fistula Using Combination of Endoscopic Suturing and Over-the-Scope Clip Placement**

*Taiwo Ajose, MD[1], Krystal Mills, MD[2], Nicholas McDonald, MD[3], Idowu Ajose, MBBS[3], Bryant Megna, MD[3], Mohammad Bilal, MD[4].*
*[1]University of Minnesota Medical School, Minneapolis, MN; [2]Morehouse School of Medicine, Atlanta, GA; [3]University of Minnesota, Minneapolis, MN; [4]University of Minnesota, Minneapolis VA Medical Center, Minneapolis, MN.*

**Introduction:** Bilo-enteric fistulas are an uncommon complication after placement of biliary drains. Most reports describe surgical management for bilo-enteric fistulas and reports of these fistulas managed endoscopically are rare. Here, we report a case of a bilo-enteric fistula which was managed by combination therapy with endoscopic suturing and over-the-scope-clip.
**Case Description/Methods:** A 75-year-old man underwent laparoscopic cholecystectomy for acute cholecystitis, which was complicated by injury to the common bile duct and required conversion to an open cholecystectomy and Roux-en-Y hepaticojejunostomy. After discharge, he returned with an ongoing bile leak and interventional radiology (IR) placed an internal-external biliary drain across the hepaticojejunostomy anastomosis. Following IR drainage, he had persistent fever and leukocytosis. Imaging revealed a perihepatic abscess and biloma. He underwent an additional IR-guided percutaneous drain placement into the biloma. Subsequently, cholangiogram revealed bile leak at the hepaticojejunostomy site with extension of contrast into the proximal duodenum concerning for a fistulous communication between the duodenum and biloma. After multidisciplinary discussion, the decision was made to evaluate the fistulous communication with esophagogastroduodenoscopy (EGD). An EGD was performed and the previously placed external drain was seen fistulized into the duodenal bulb [**Figure** 1A]. The external drain was pulled back outside the duodenal lumen. The fistulous opening was 15 mm in size. Argon plasma coagulation of the tract edges was performed to promote tissue healing and closure followed by endoscopic suturing to close the tract [**Figure** 1B]. Endoscopic suturing was performed using the OverSitch device and one suture was placed in a figure of 8 fashion. However, a 3-mm opening was seen immediately distal to the pylorus even after cinch placement [**Figure** 1C]. To close the remaining defect, a 12-mm in diameter over-the-scope clip was placed [**Figure** 1D]. Following closure, there was no residual contrast leakage confirming successful closure of the fistula. The external drain was subsequently removed without evidence of biloma re-accumulation on follow-up imaging.
**Discussion:** Our report highlights that a combination of endoscopic suturing and over-the-scope clip closure approach can be a potential endoscopic option for managing bilo-enteric fistulas.




[2482] **Figure 1.** A- External drain seen fistulized into the duodenal bulb. B- Endoscopic suturing to close the fistula tract. C- Pylorus opening seen after endoscopic suturing. D- 12-mm over-the-scope clip was placed to close the defect.

**S2483**

**Combined Endoscopic Ultrasound and Endobronchial Ultrasound to Stage and Diagnose Non-Small Cell Lung Cancer in the Setting of CLL/SLL**

*William C. Green, MD[1], Kathleen Herring, MD[2], Cynthia Ezedike, MD[3], Jason Conway, MD[3].*
*[1]Wake Forest University School of Medicine, Winston-Salem, NC; [2]Internal Medicine Residency Program, Wake Forest University School of Medicine, Winston-Salem, NC; [3]Wake Forest Baptist Health, Winston-Salem, NC.*

**Introduction:** Chronic lymphocytic leukemia and small lymphocytic lymphoma is an indolent leukemia with a variable course affecting 4-5 per 100,000 population in the US/year. Patients with CLL are at an increased risk of other cancers, including lung cancer. Endosonography can be used to diagnose and stage suspected lung cancer. EUS-FNA can be used not only for cancers of the gastrointestinal tract, but can also be utilized as an alternative to surgical staging for lung cancer, such as mediastinoscopy. We present a case of lung cancer that was staged and diagnosed via combined endoscopic and endobronchial ultrasound with biopsies.
**Case Description/Methods:** A 78-year-old man with a history of CAD, prostate cancer in remission and chronic lymphocytic leukemia (CLL) presented to the ED from his scheduled bronchoscopy for a right hilar mass with pleural effusion and 2 weeks of progressive dyspnea. During hospitalization, gastroenterology was consulted for a EUS-FNA for the patient's subcarinal node seen on imaging and pulmonology consulted for an EBUS due to this left hilar mass with mediastinal lymph nodes. EBUS revealed an extrinsic compression of the RLL and RUL and EBNA performed on 3 mediastinal lymph nodes. EUS-FNA showed 3-4 cm conglomeration of well-defined, round and hypoechoic lymph nodes in the subcarinal space with fine needle biopsy. Final pathology of these specimens diagnosed stage III non-small cell lung cancer.
**Discussion:** EUS can be utilized to reach the left and lower paraesophageal structures as well as structures below the diaphragm. EUS-FNA added tissue samples from his subcarinal lymph nodes that supported the correct diagnosis of non-small cell lung cancer. Due to the diagnosis of non-small cell lung cancer from his combined EBUS/EUS-FNA, the patient did not require surgical staging. Our case demonstrates the need use of a combined EUS/EBUS approach to diagnose a patient with CLL with non-small cell lung cancer. Second malignancies in CLL cases are rare and approximately 2% of patients with CLL develop lung carcinoma. Recent studies showed that patients with CLL less than 55 years old have a greater risk of developing secondary solid tumors, not like our patient who was 78 years old. Tissue samples from the EUS-FNA/EBUS are very important to help guide therapy due to the high resistance to conventional chemotherapy in patients with CLL and a second malignancy. A multidisciplinary approach can be used to diagnose and stage lung cancers with EBUS/EUS-FNA in the setting of CLL.

**S2484**

**ColoWrap Device Facilitates Successful Colonoscopy in Patient With Prior Incomplete Colonoscopy Due to Looping and Adhesions**

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**Introduction:** Two primary causes of incomplete colonoscopy related to patient anatomy include redundancy and post-surgical adhesions. This case report details on the successful completion of a previously failed colonoscopy in a 70-year-old obese male patient with a redundant colon and prior abdominal trauma (gunshot wound) using a colonoscopy compression device (ColoWrap).
**Case Description/Methods:** A 70-year-old obese male patient with a history of abdominal trauma (gunshot wound) underwent surveillance colonoscopy in November 2021. During the procedure, significant looping occurred once the scope was advanced past the distal colon, inhibiting progress beyond the hepatic flexure. All standard measures including torquing, manual abdominal pressure, and patient repositioning were employed yet were unsuccessful and after one hour the procedure was aborted. The patient thereafter underwent virtual colonoscopy which indicated a lesion in the ascending colon. The patient was scheduled for a repeat colonoscopy in January of 2022 with the same experienced endoscopist. Due to prior difficulties, the physician decided to perform the patient's repeat attempt at colonoscopy using a ColoWrap. ColoWrap is a single-use lower abdominal compression device designed to help mitigate looping during colonoscopy. The device applies broad, sustained compression across the lower abdomen and features 2 secondary straps that can be used to direct additional, location-specific pressure. The ColoWrap was applied just prior to the exam per manufacturer instructions. Looping was once again encountered past the distal colon. In response, the ColoWrap secondary straps were adjusted to apply directed compression to the areas of presumed looping in the left and the transverse colon. This enabled successful advancement past the hepatic flexure and to the cecum. The lesion that had been indicated on virtual colonoscopy was identified and biopsied. The biopsies revealed a large, high-risk adenoma not amenable to endoscopic removal and surgery was performed (**Figure**).
**Discussion:** In this case, use of ColoWrap enabled successful cecal intubation in a patient with a previous incomplete colonoscopy who had a known proximal colonic lesion identified by virtual colonoscopy. ColoWrap's intra-procedural adjustability and features delivering targeted compression to specific regions of the abdomen were instrumental in overcoming the anatomical difficulties that had resulted in the failure of the prior colonoscopy performed by the same, experienced endoscopist.


[2484] **Figure 1.** A. ColoWrap Anti-Looping Colonoscopy Compression Device B. ColoWrap in-situ (previous, single-strap version).

**S2485**

**Clinically Significant Upper Gastrointestinal Bleeding Post Esophagogastroduodenoscopy Cold Biopsy: A Rare Case Report**

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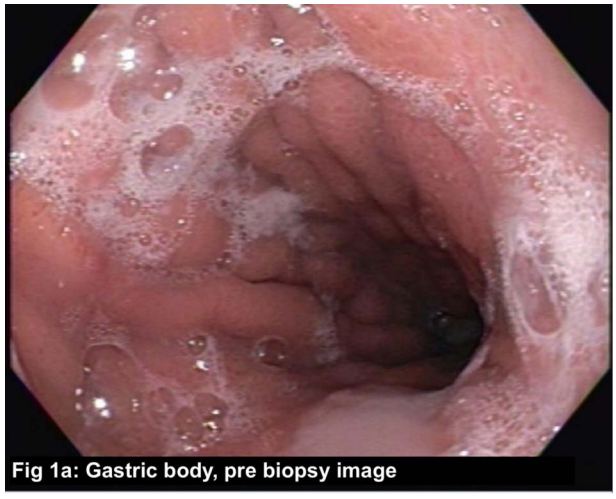
**Introduction:** Bleeding after cold forceps biopsy of the gastrointestinal tract is an extremely rare phenomenon, estimated incidence < 0.1%. Clinically significant bleeding is even rarer. However, in patients who have evidence of gastrointestinal bleeding (GIB) after endoscopic biopsy, it is an important cause to consider.
**Case Description/Methods:** A 42-year-old female with a past medical history of kidney transplant for end stage renal disease of unknown etiology (with allograft rejection 3 months prior to admission), on intermittent hemodialysis and chronic normocytic anemia presented with acute on chronic abdominal pain, diarrhea, nausea, and emesis. Infectious evaluation was unrevealing. CT abdomen and pelvis showed moderate colitis and moderate to severe enteritis of the mid small bowel. Esophagogastroduodenoscopy (EGD) showed scalloping of the mucosa with a few scattered submucosal hemorrhages in the mid-esophagus and pseudomelanosis duodeni. Colonoscopy showed erythema and submucosal hemorrhages throughout the colon (sigmoid and rectum spared). Cold forceps biopsies were taken from the esophagus, stomach, duodenum, and colon. Several hours after endoscopy, she had hematochezia, hematemesis, and new severe epigastric abdominal pain along with tachycardia and relative hypotension. Labwork revealed a hemoglobin/hematocrit of 4.3g/dL/14% (down from 8g/dL/24%), INR 1.4, and platelets 177thou/uL. There was no evidence of disseminated intravascular coagulation or hemolysis. CT angiogram did not show active bleeding. After resuscitation, repeat EGD revealed oozing from gastric and duodenal biopsy sites. Eight hemoclips were placed for hemostasis at the sites of bleeding. Biopsies from initial EGD revealed esophagitis, lymphocytic gastritis (LG), negative helicobacter pylori stain, and normal duodenal and colonic mucosa (**Figure**).
**Discussion:** The risk of bleeding after endoscopic cold biopsy is rare, ranging from 0.004% to 0.07 %; hemodynamically significant luminal bleeding is even rarer. The association of LG with this phenomenon, in the absence of other histologic features, remains understudied. There are no reports that suggest LG increases the risk of bleeding, however this might be a novel presentation. LG has many causes, including medications like angiotensin receptor blockers (ARBs). There are some reports that ARBs can lead to platelet disregulation, but this has not be linked to any cases of GIB. Endoscopic evaluation is warranted in this setting for diagnostic and therapeutic purposes.




[2485] **Figure 1.** Pre and Post Biopsy Endoscopic Images.

**S2486**

**Chilaiditi's Sign: Should You Give Up or Persist?**

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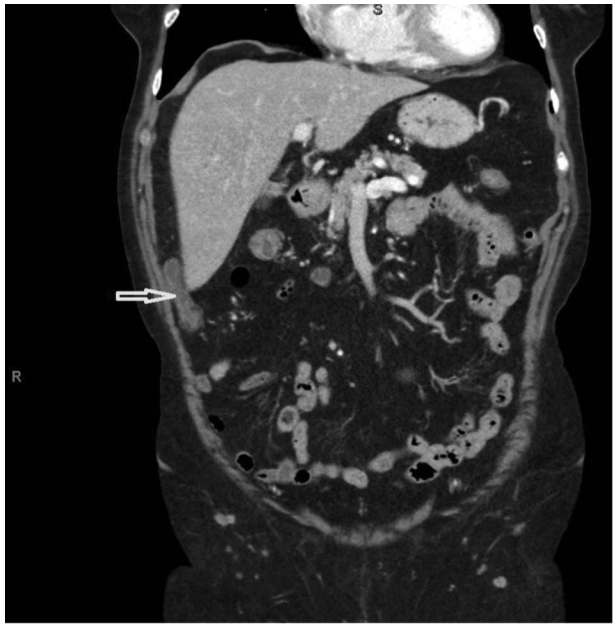
**Introduction:** Chilaiditi's sign is an incidental radiographic finding of the interposition of the bowel loop between the liver and the diaphragm. With an estimated worldwide prevalence of 0.028 - 0.25 %, this is commonly associated with older age and men. Below, we describe an unusual case of Chilaiditi's sign incidentally found during a routine colonoscopy in a female patient
**Case Description/Methods:** A 73-year-old asymptomatic woman was referred to the Endoscopy Unit for a surveillance colonoscopy. On physical examination, the patient had no abdominal pain or distension. CBC and CMP were unremarkable. Pediatric colonoscope was inserted via the rectum and reached the hepatic flexure. However, due to an acute angulation, the scope was not able to be advanced further and the procedure was therefore aborted prematurely. A CT scan of the abdomen and pelvis performed the same day identified interposition of the distal transverse colon and proximal ascending colon between the liver and the anterior abdominal wall, which explained the difficulty encountered in advancing the colonoscope beyond the hepatic flexure (**Figure**). She is being rescheduled for repeat colonoscopy.
**Discussion:** Acute angulation of the bowel and technical difficulty in completing a colonoscopy may be indicative of Chilaiditi's sign. Predisposing factors include: cirrhosis, COPD, or presence of ascites. When this anatomical distortion becomes symptomatic, it is referred to as Chilaiditi's syndrome. These symptoms are secondary to elevation of the diaphragm and/or bowel obstruction, perforation, or ischemia. It can be confirmed with CT imaging, which typically demonstrates an interposition of the transverse colon between the liver and the abdominal wall. While no intervention is usually required for asymptomatic patients with Chilaiditi's sign, awareness of the condition is important for physicians. Forceful pressure against the interposed bowel can trap air in the colon segment and increase intra-luminal pressure,

potentially causing perforation. To decrease the risk of perforation, carbon dioxide can be administered in patients with known Chilaiditi' sign during colonoscopy as an insufflating agent. Limited case reports are available on the management protocol for patients with Chilaiditi's sign; however, according to a previous case report, carbon dioxide is safe and effective in the GI tract. It is imperative that endoscopists are aware of this condition and exercise extreme caution while performing a difficult colonoscopy.




[2486] **Figure 1.** CT scan of abdomen showing the interposition of the distal transverse colon and proximal ascending colon between the liver and the anterior abdominal wall.

**S2487**

**CMV-Induced Gastritis, Duodenitis and Colitis in an Immunosuppressed Patient**

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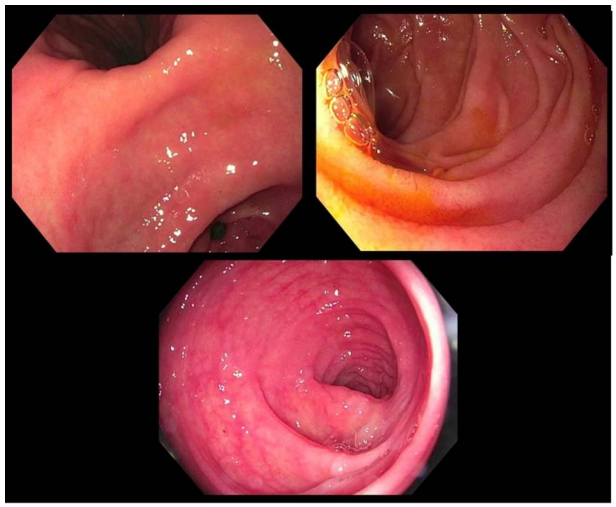
**Introduction:** Cytomegalovirus (CMV) is a common cause of morbidity and mortality in immunocompromised patients. CMV can infect any part of the Gastrointestinal tract, with the colon being the most affected. There are isolated reports of gastric, small intestine, and esophagus involvement, however, we report a case of CMV-induced gastritis, duodenitis, and colitis which occurred concurrently in an immunosuppressed patient.
**Case Description/Methods:** A 65-year-old woman with a history of deceased donor kidney transplant presented with fatigue, generalized weakness, and a 2-week history of black-colored bowel movements. Her social history was negative for smoking, alcohol ingestion, and illicit drug use. Surgical history and review of systems were otherwise insignificant. On admission, vitals and physical exam were not significant. On laboratory analysis, serum creatinine of 2 mg/dL (baseline of 1.4-1.6 mg/dL) and a hemoglobin level of 7 g/dL was present; complete blood count and chemistry panel were otherwise normal. The patient was CMV positive with a viral load of PCR 309000 IU/mL. Due to her history of transplant and anemia an esophagogastroduodenoscopy (EGD) and colonoscopy were performed and revealed erythematous mucosa in the antrum, duodenum, ascending colon, transverse colon, descending colon, and ileocecal valve. Biopsies of these sites revealed cytopathic changes and positive immunostaining for CMV. The patient received IV ganciclovir followed by oral valganciclovir until 2 weekly CMV viral load had been negative. Subsequent outpatient Follow-up visit showed resolution of symptoms and CMV viral load (**Figure**).
**Discussion:** CMV gastroenteritis in acute settings can present with a wide variety of symptoms including nausea, vomiting, abdominal pain, and bloody diarrhea. CMV gastroenteritis is often an isolated finding in one site and therefore can be easily missed if there is not a significant clinical suspicion. Upon review of the literature, no other case reports were found involving all the above-mentioned sites in a single patient. It is important to note that patients who are immunocompromised may present with vague symptoms, which should be investigated further, as was the case with our patient. Our case emphasizes that physicians should be aware of various clinical presentations and that although rare, CMV can affect multiple areas in the same patient.



[2487] **Figure 1.** Diffuse erythema can be observed on the gastric mucosa (top left), the second portion of the duodenum (top right), and throughout the colon (bottom center).

S2488

**Diffuse Mesenteric Lymphadenopathy and Duodenal Polyposis Secondary to Mantle Cell Lymphoma: A Rare Case Report**

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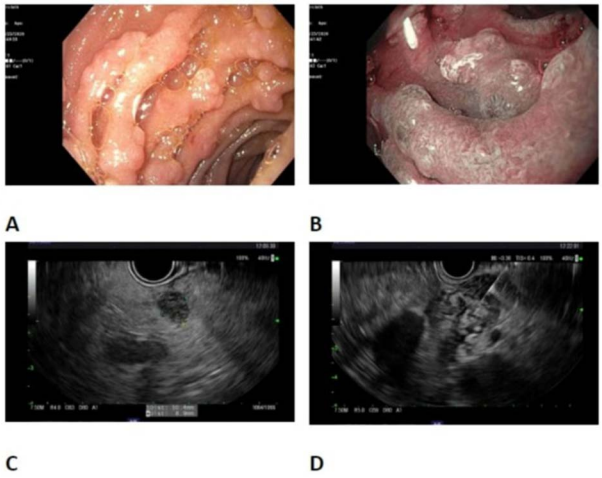
**Introduction:** Mantle cell lymphoma (MCL) is a rare type of B-cell non-Hodgkin lymphoma characterized by atypical small lymphoid cells within the mantle zone of germinal center follicles. Immunohistochemistry shows cyclin D1 overexpression associated with CD5+ and CD20+ expression and a t(11;34)(q13;q32) translocation. Despite being classified as low-grade, it is an aggressive lymphoma because it is usually discovered at a late stage with splenomegaly, lymphadenopathy, and blood and bone marrow involvement. Gastrointestinal involvement is common as an extranodal site of the MCL; however, primary GI lymphoma is very rare (about 1-4% of all GI malignancies).
**Case Description/Methods:** A 58-year-old male presented with epigastric abdominal pain, nausea, and vomiting for a few weeks. He denied unintentional weight loss, night sweats, or anorexia. The physical exam and essential laboratory work were unremarkable. Computed tomography (CT) scan showed proximal small bowel wall thickening, with moderate epigastric, retroperitoneal, and mesenteric lymphadenopathy concerning lymphoma. Esophagogastroduodenoscopy (EGD) showed diffuse polyposis in the proximal duodenum (**Figure** A,B). Endoscopic ultrasound (EUS) showed multiple enlarged peri duodenal and peripancreatic lymph nodes and abnormal duodenal wall thickening. A fine needle biopsy of the duodenal wall and lymphadenopathy was obtained using a 22-gauge needle (**Figure** C,D). Histology was consistent with mantle cell lymphoma, and molecular testing was positive for monoclonal IgH gene rearrangement. The patient was referred to oncology and started on chemotherapy per protocol.
**Discussion:** Mantle cell lymphoma (MCL) is a rare type of B-cell non-Hodgkin lymphoma. Because of poor detection by radiological imaging, multiple organ systems are involved by the time of diagnosis leading to a poor prognosis with very low median survival. Only a few case reports are available in the literature about primary gastrointestinal mantle cell lymphoma; thus, little is known about the outcome, the response to treatment, and the duration of remission in primary GI MCL patients.




[2488] **Figure 1.** (A) EGD image shows diffuse polyposis in the proximal duodenum, (B) EGD image with narrow-band imaging shows diffuse duodenal polyposis, (C) Endoscopic ultrasound shows peripancreatic lymphadenopathy, (D) Endoscopic ultrasound with fine-needle biopsy of the thickened duodenal wall.

S2489

**Differentiating CMV Colitis From GVHD in Post-Transplant Patients: A Case Study**

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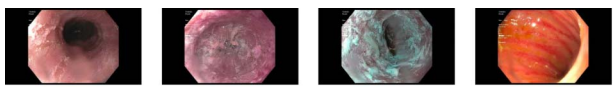
**Introduction:** Cytomegalovirus (CMV) infection can complicate the clinical course of immunocompromised patients including allogeneic hematopoietic stem cell transplant (allo-HSCT) recipients, where the infection can be serious. CMV can involve different organs including lungs, brain, eyes, and the GI tract. Signs and symptoms of CMV infection may overlap with other conditions including graft-versus-host disease (GVHD), a multisystem disorder commonly seen in allo-HSCT recipients.
**Case Description/Methods:** A 57-year-old female presented with abdominal pain and diarrhea. Past medical history included relapsed acute myeloid leukemia, treated with azacitidine and venetoclax followed by allo-HSCT. She was recently hospitalized for suspected GVHD with skin, liver, and gastrointestinal involvement. She was also found to have a CMV viral load of 920 IU/mL. Endoscopy showed diffuse inflammation across the GI tract, with biopsies revealing apoptotic bodies consistent with acute GVHD. She was treated with ruxolitinib and prednisone for GVHD, and valganciclovir prophylactically for CMV viremia. At this presentation, labs were notable for lymphocytes of 39 x$10^3$/uL and platelets of 22 x $10^3$/uL, and CMV viral load of 1,822,242 IU/mL. CT imaging revealed thickened distal small bowel loops, cecum, and right colon, and edematous gastric walls with submucosal enhancement. Infectious work-up, including stool culture, ova/parasite, and clostridium, was negative. She underwent endoscopic evaluation; EGD showed white discoloration that washed off in the esophagus and diffuse erythematous mucosa in the stomach (**Figure**). Biopsies from gastric mucosa and right colon mucosa revealed focal nuclear changes. Immunohistochemical stains were positive for CMV. She was diagnosed with refractory CMV colitis and treated with maribavir with improvement of symptoms and reduction of viral load, and prior course of steroids was tapered.
**Discussion:** Immunosuppression for GVHD has been identified as an important risk factor for CMV reactivation. Differentiating CMV colitis and GI-GVHD poses a diagnostic challenge, and endoscopic evaluation is needed to address this. Endoscopy can identify cecal ulcers, a reliable finding for CMV colitis in patients with GVHD after allo-HSCT.[1] It is important to timely identify CMV colitis to prevent post-transplant morbidity and mortality. This case also demonstrates effectiveness of maribavir, a novel antiviral, for refractory post-transplant CMV colitis.



[2489] **Figure 1.** EGD images showing white discoloration in the entire esophagus and diffuse erythematous mucosa in the entire examined stomach.

**REFERENCE**

1. Matsuda K, Ono S, Ishikawa M, et al. Cecum ulcer is a reliable endoscopic finding in cytomegalovirus colitis concomitant with graft-versus-host disease after allogeneic hematopoietic stem cell transplantation. Ann Hematol 2018;97(5):877-883.

S2490

**Discovery of Gastric Adenocarcinoma During PEG Tube Placement in Patient With Epiglottic Squamous Cell Carcinoma**

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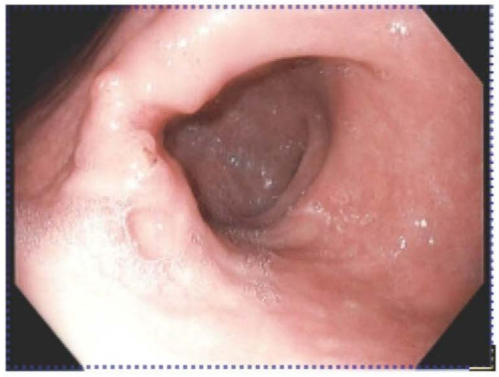
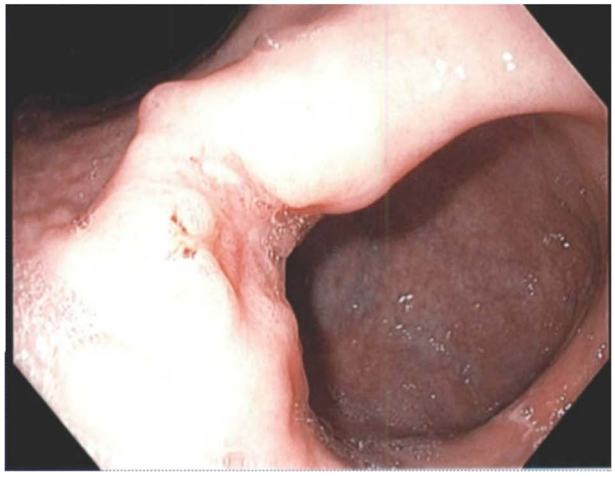
**Introduction:** Multiple primary neoplasms constitute up to 2-17% of cancer diagnoses. Etiological factors such as genetics, lifestyle choices, and immune status have a significant impact on the likelihood of patients developing synchronous multiple primary tumors. The timing of diagnosis for the different cancers has a major impact on disease management, treatment plans, and patient outcomes. We report a patient who was diagnosed with gastric adenocarcinoma at the time of percutaneous endoscopic gastrostomy (PEG) tube placement for dysphagia secondary to squamous cell carcinoma of the anterior epiglottis.
**Case Description/Methods:** A 77-year-old male presented to the outpatient surgery center for upper endoscopy with PEG tube placement. Two months prior, the patient was diagnosed with p16 negative invasive squamous cell carcinoma of the anterior epiglottis. He was referred for PEG tube placement for nutrition supplementation due to 5 months of progressive dysphagia and protein calorie malnutrition with an unintentional weight loss of 50 lbs. Past social history was significant for tobacco dependence with 52 pack years and alcohol dependence. At the time of PEG tube placement, endoscopy revealed a 1.5 cm excavated lesion at the gastric incisura. Biopsy was performed to rule out malignancy. PEG tube was successfully placed. The gastric biopsy was consistent with diffuse type signet ring gastric adenocarcinoma. PET scan 1 month prior to PEG tube placement did not show any foci of abnormal FDG uptake outside of the primary lesion in the epiglottis. Patient is currently undergoing treatment for laryngeal carcinoma with chemotherapy and radiation. Assessment and treatment for gastric cancer diagnosis will be deferred until completion of treatment for laryngeal carcinoma (**Figure**).
**Discussion:** Concurrent laryngeal and gastric cancer is a unique diagnosis that has not been well reported in the literature. However, given the strong association for both malignancies with chronic alcohol and tobacco use, it is not unreasonable for both to occur in the same patient in an independent manner. The literature has shown that signet ring cell carcinomas have significantly lower $^{18}$F-FDG uptake than other forms of gastric cancer. These findings highlight the importance of completing a full endoscopic evaluation in all patients undergoing endoscopy even for procedures as straightforward as PEG tube placement.





[2490] **Figure 1.** Esophagogastroduodenoscopy showing gastric adenocarcinoma at the gastric incisura.

S2491

**Conservative Management of a Gastrosplenic Fistula Due to Splenic Abscess: A Success Story**

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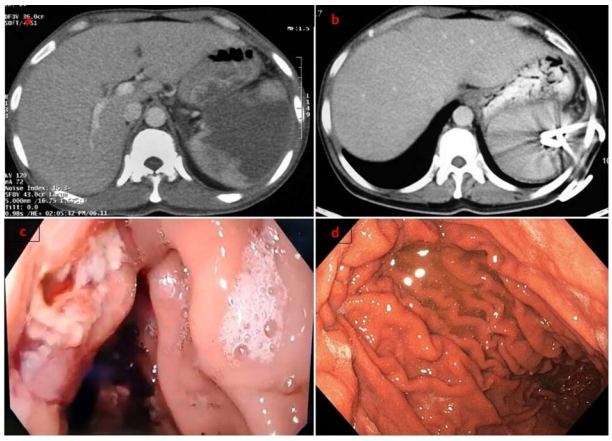
**Introduction:** Splenic abscess in itself is a rare clinical scenario with data being limited to case reports and case series. Gastrosplenic fistula is a known complication and only few gastrosplenic fistula have been described with benign etiologies. In all these cases, the patient underwent surgical management. Historically, splenectomy was the gold standard of management for splenic abscess but recent case series have established efficacy of conservative approach for splenic abscess.
**Case Description/Methods:** A 35-year-old male with no known comorbidity presented with fever and recurrent malena to our emergency. He was found to have a large splenic abscess with a gastrosplenic fistula [**Figure** 1a] showing active ooze of pus and blood in gastric cavity on oesophagoduodenoscopy (EGD)[**Figure** 1c]. He was managed with broad spectrum antibiotics and multiple transfusions.A 10 french(fr) percutaneous drain (PCD) was inserted in the splenic abscess as a bridging modality to surgery. A multidisciplinary team comprising of intervention radiologist, surgeon and the treating gastroenterologist decided to try for upgrading the PCD before taking the patient for surgery due to high surgical morbidity in presence of gastrosplenic fistula and poor nutritional status. The drain was later upgraded with 2 14Fr PCD in the abscess [**Figure** 1b]. Pus cultures were sterile and no trophozoites were seen but the amoebic serology(IgG) was positive suggesting an amoebic etiology. On adding metronidazole, there was rapid clinical response with resolution of fever and malena. On regular follow up over 6 weeks, there was complete resolution of gastrosplenic fistula on EGD **Figure** 1d] with resolution of abscess.
**Discussion:** Our case is the first case to be reported where complete healing of gastrosplenic fistula has been demonstrated without requiring surgery. There are no set guidelines for management of splenic abscess. Historically, surgical management was considered as the gold standard of management but was associated with significant morbidity and mortality of upto 17%. Another recent review on management of

gastrosplenic fistulas showed similar survival of 82% in all cases of gastrosplenic fistulas. However, in recent times safety and efficacy of PCD has been well established. PCD has been attempted in prior reports with gastrosplenic fistula but required surgery for definitive management. The choice of surgery is generally open splenectomy with partial gastric resection but laparoscopic techniques have been described.




[2491] **Figure 1.** (a) Large splenic abscess with loss of fat planes with stomach (b) Resolution of abscess with double pigtail catheters (c) Gastrosplenic fistula opening seen in body of stomach on endoscopy (d) Healed gastric wall after 6 weeks of initial presentation.

**S2492**

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**Double Pylorus Secondary to Peptic Ulcer Disease**

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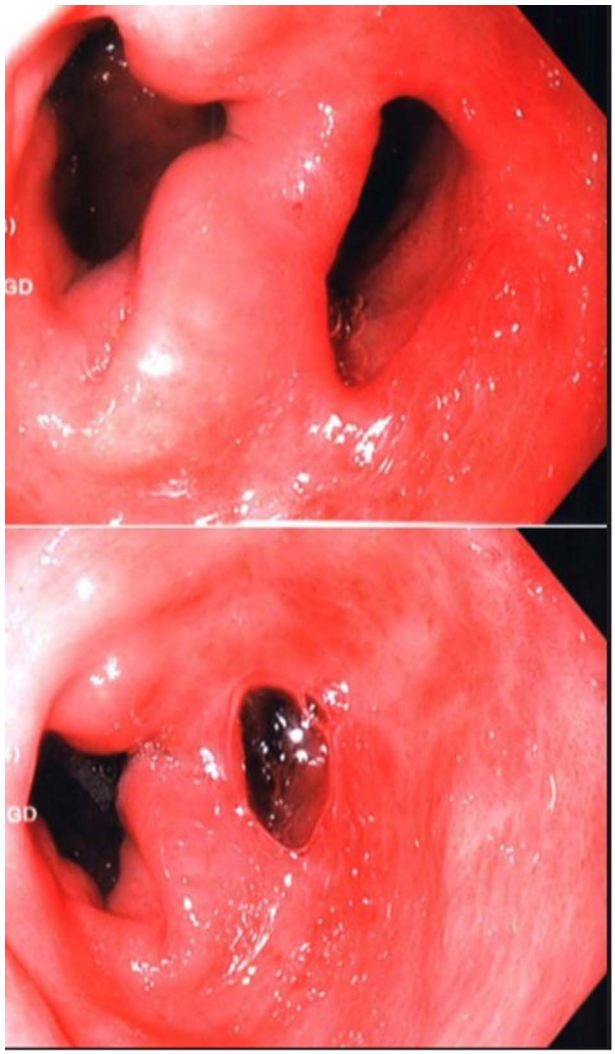
*[1]Indiana University Internal Medicine Residency - Southwest, Evansville, IN; [2]Indiana University Internal Medicine Residency - Southwest, Vincennes, IN; [3]Good Samaritan Hospital, Vincennes, IN.*

**Introduction:** Double pylorus is an unusual endoscopic finding viewed as a fistula between the gastric antrum and duodenal bulb. The presence of double pylorus occurs in 0.001 to 0.4% of upper endoscopy procedures and is more frequent in men and those with peptic ulcer disease. We describe a 74-year-old female with prior *H. pylori* infection and gastroduodenal ulcerations resulting in a double pylorus.
**Case Description/Methods:** A 74-year-old female was referred to our clinic for evaluation of dyspnea and persistent iron deficiency anemia over several months with a history of alcoholic cirrhosis, right hemicolectomy due to diverticulitis, and gastroduodenal ulcer disease in the setting of prior *H. pylori* infection. Her dyspnea was associated with dull pleuritic epigastric pain radiating to her back but she denied nausea, melena, hematochezia, dysphagia, odynophagia, and weight loss. She was compliant with oral iron supplementation and reported 3 years of sobriety. Vital signs and physical examination were unremarkable. CBC displayed macrocytic anemia with iron deficiency on iron panel. Abdominal CT revealed a large paraesophageal hernia and a lobular liver contour compatible with cirrhosis. Given her prior antral and duodenal ulceration, EGD was performed, which revealed antral erythema, an 11 cm hiatal hernia, and a double pylorus (**Figure**). Both ostia at the antrum were intubated separately and appeared endoscopically normal. *H. pylori* biopsies were negative. The patient continued proton pump inhibitor therapy without further procedural intervention.
**Discussion:** Most cases of double pylorus are acquired in the setting of peripyloric ulceration with resulting mucosal perforation from repetitive damage and impaired healing. After perforation occurs, a fistula can form between the gastric antrum and duodenal bulb. Common symptoms include dyspepsia, emesis, and chronic abdominal pain. Occult bleeding is common and diagnosis requires upper endoscopy to visualize each ostia of the double pylorus along the lesser curvature. Management typically consists of risk factor reduction (NSAID and corticosteroid use, *H. pylori* infection) and pharmacologic promotion of mucosal healing (proton pump inhibitors, H2 receptor antagonists, and antacids). Utilization of these strategies was shown in one study to close the double pylorus false lumen in 9% of cases and cause the 2 pylori to fuse in 27% of cases. However, 64% of patients in the same study had persistent double pylorus despite treatment.



[2492] **Figure 1.** Endoscopic view of the pylorus from the antrum of the stomach.

**S2493**

**Endoluminal Vacuum-Assisted Wound Closure Therapy for Anastomotic Leak Following Robotic-Assisted Colorectal Cancer Surgery**

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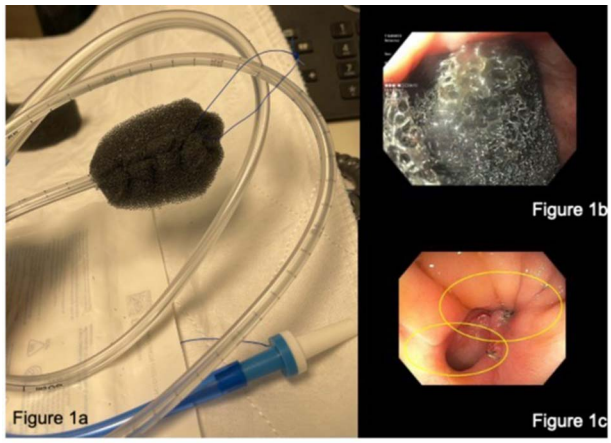
*Saint Mary's Hospital, Waterbury, CT.*

**Introduction:** Anastomotic leak is one of the most feared and potentially life-threatening complications of colorectal surgery, with leak rates ranging from 3% to 10%. Endoluminal vacuum-assisted wound closure (EVAC) therapy is well known for treating esophageal perforations. However, there are few reports of its use in treating colonic perforations.
**Case Description/Methods:** A 60-year-old woman presented to the emergency department with a one-week history of severe rectal pain worsening with defecation after undergoing robotic-assisted surgery one month prior for colonic adenocarcinoma. Computed tomography (CT) scan of the abdomen revealed a pre-sacral ill-defined collection measuring up to 3.6 centimeters (cm) of fat-stranding extraluminal gas and fluid posterior to the rectal surgical suture line concerning for anastomotic leak. Endoscopic repair was attempted using X-tack, which was unsuccessful. An endoluminal vacuum suction system was built using a GranuFoam sponge of approximately 5 cm x 3 cm, sutured to an NG tube, and placed partially into the cavity under endoscopic guidance. The system was renewed approximately every 72 hours 3 times, with complete closure of the leak one month later on follow-up endoscopy (**Figure**).
**Discussion:** Our case illustrates that endoluminal wound VAC therapy may be a good therapeutic option in the appropriate setting. Further studies are needed.




[2493] **Figure 1.** a: Endoluminal vacuum device put together using nasogastric tube and granufoam. b: Endoluminal vacuum set up at the anastomotic leak site via colonoscopy. c: Follow-up colposcopy demonstrating healing of the anastomotic leak site.

**S2494**

**Elusive Intussuscepting Intra-Appendiceal Carcinoid Tumor Diagnosed on Colonoscopy**

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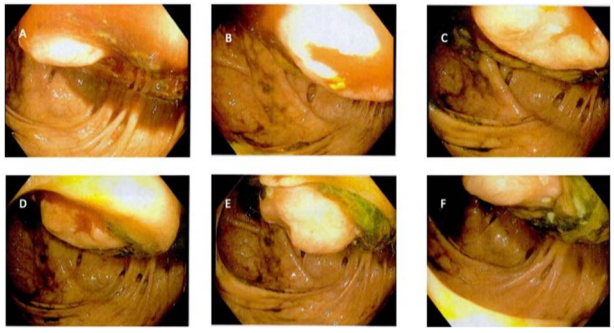
**Introduction:** Appendiceal neoplasms are asymptomatic and non-obstructive that make up ~1% of appendectomy specimens and ~0.5% of intestinal neoplasms. We present an incidental finding of appendiceal neoplasm in a patient with an acute GI bleed.
**Case Description/Methods:** A 61-year old male with PUD and diverticulosis on colonoscopy (CLN) 3 years ago presented with painless hematochezia associated with a 3-month 30-lb weight loss. On presentation, vital signs were stable. Physical exam noted for mild abdominal distention. Laboratory values showed WBC of 17.2 K/mcL, hgb of 10.9 g/dL (from 15.1), MCV of 75 fL and platelet count of 1,126 K/mcL (normal one year ago). Computed tomography (CT) of the abdomen and pelvis with contrast revealed a 3.3 x 2.5 cm hyperenhancing mass near the ileocecal valve. GI was consulted for a CLN which showed diverticular bleeding, but no mass. He remained asymptomatic. A bone marrow biopsy showed hypercellularity with atypia and fibrosis most consistent with myeloproliferative neoplasm (MPN). Cytology for JAK-2 V617 mutation was positive and initiated on hydroxyurea for essential thrombocythemia (ET). Meanwhile his hgb continued to downtrend presumed from an acquired von Willebrand syndrome (VWD) and repeat CT scan on hospital day (HD) 5 showed no evidence of acute GI bleed but again showed the mass. On HD7 the patient had BRBPR and a hgb drop to 7.8. On repeat CLN, a mass was discovered intussuscepting in and out of the appendix. A I-123 MIBG Scan was positive in the RLQ with elevated chromogranin A level. Biopsies revealed a low-grade carcinoid tumor. He was discharged with a plan for future right hemicolectomy once platelets stabilized (**Figure, Table**).
**Discussion:** Neuroendocrine tumors (NET), aka carcinoid tumors, are typically detected in the 5th decade of life. Around one-fifth of NETs are found to have an associated non-carcinoid tumor for which colorectal cancer is 25% to 50% of the cases. In our patient, it was discovered during CLN and imaging prompted by rectal bleeding. It is likely that his carcinoid tumor could have gone undiagnosed if not for his rectal bleeding. We hypothesized this was due in part to his bleeding disorder (acquired VWD) which is very prevalent in those with ET. The patient's coexisting carcinoid tumor and MPN was also of interest as myelodysplastic syndrome can develop in 2% of patients with metastatic NET who were treated with peptide receptor radionuclide therapy thought to be due to myelotoxicity. Treatment of localized >2 cm appendiceal NET is right hemicolectomy.




[2494] **Figure 1.** After careful observation of the cecum, a mass inside the appendiceal orifice was seen intussuscepting in and out. A to F shows time lapse of events.

**Table 1. Differential Diagnosis for Terminal Ileitis**

| Infectious | Drug-related | Vasculitides | Small Bowel neoplasms | Infiltrative | Other |
|---|---|---|---|---|---|
| Yersinia | NSAID | Systemic lupus erythematosus | Adenocarcinoma | Eosinophilic enteritis | Crohn's disease |
| Nontyphoidal Salmonella | Mycophenolate mofetil | Polyarteritis Nodosa | Lymphoma | Sarcoidosis | Appendicitis |
| Typhlitis | Potassium chloride | Henöch-Schönlein purpura | Carcinoid tumor | Amyloidosis | Ischemia |
| Mycobacterium tuberculosis and avium | | | Familial Adenomatous Polyposis Coli | | Radiation enteritis |
| Actinomycosis | | | Hereditary Nonpolyposis Colorectal Cancer | | Cryptogenic Multifocal Ulcerating Stenosing Enteritis |

Table 1. (*continued*)

| Infectious | Drug-related | Vasculitides | Small Bowel neoplasms | Infiltrative | Other |
|---|---|---|---|---|---|
| Anisakiasis | | | Peutz–Jeghers Syndrome | | Spondyloarthropathies |
| Cytomegalovirus | | | | | Backwash ileitis |
| Clostridium difficile | | | | | Endometriosis |

**S2495**

**Early Esophageal Necrosis: A Food Impaction in an Elderly Patient With Multiple Comorbidities**

*Joseph Cook, MD, Cassandra Craig, MD.*
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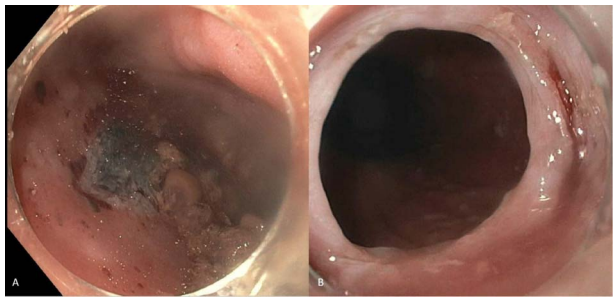
**Introduction:** Food impactions represent the most common esophageal foreign bodies in adults. Patients can be stratified into emergent, urgent and non-urgent, and in urgent cases upper endoscopy (EGD) is recommended within 24 hours. We present a patient who developed esophageal necrosis well before 24 hours.
**Case Description/Methods:** An 87-year-old womane with multiple comorbidities presented to the emergency department complaining of chest pain that she attributed to eating salmon and mashed potatoes ~6 hours prior. Physical exam had no crepitus, labs revealed a mild leukocytosis and radiographs were unremarkable. The gastroenterology service obtained a CT scan to rule out esophageal perforation but alerted the operating room (OR) to set up for an EGD. Imaging was notable for findings concerning for a distal esophageal mass with proximal esophageal distention and impacted contents, but no esophageal perforation. EGD was performed 14 hours following the initial ingestion. Patient was tolerating secretions and vitals remained stable. On EGD a large food bolus in the mid esophagus was partially removed with a cap and net retrieval device unearthing a 2x2 cm patch of necrotic tissue. An 18F nasogastric tube (NGT) was placed proximal to necrotic area to facilitate water soluble contrast administration for a CT esophagram which did not suggest perforation. Intravenous antibiotics were initiated, and patient was kept intubated. Repeat EGD the next day was notable for migration of food bolus and interval improvement of necrotic patch. A 10F NGT was placed endoscopically and proton pump inhibitor was initiated. Over the next several days patient was extubated and diet was advanced. Repeat EGD after discharge was notable for a ring which was sequentially dilated to 20mm and at follow up 4 weeks later patient was denying all dysphagia. (Figure)
**Discussion:** Food impactions represent the most common esophageal foreign bodies in adults. Timing of EGD is widely debated. Guidelines recommend urgent cases to undergo EGD within 24 hours to minimize the risk. However, as highlighted in this case EGD within 24 hours in patients with comorbidities might not be sufficient to prevent complications. This case highlights the need for increased granularity in the stratification of patients with food impactions. We propose that advanced age and/or presence of atherosclerotic disease may warrant earlier EGD.




[2495] **Figure 1.** A: Esophageal necrosis. B: Esophageal ring.

**S2496**

**Endoscopic Source Control of Gram-Negative Bacteremia Secondary to Foreign Body Ingestion - A Rare Case**

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**Introduction:** Foreign body (FB) ingestion is usually observed in the pediatric population; however, adult cases of FB ingestion are not uncommon. Usual FB culprits seen in adults include fish bones, chicken bones, and dentures. Possible complications include dysphagia, complete occlusion with risk of aspiration, and perforation. Such complications warrant retrieval of FB via endoscopic removal or surgical on case-to-case basis. We present a very rare case of FB ingestion resulting in bacteremia necessitating endoscopic removal leading to source control.
**Case Description/Methods:** The patient is a 58-year-old male with a past medical history of diabetes mellitus, hypertension, and chronic venous stasis. He was initially admitted for sepsis secondary to cellulitis and had to be readmitted due to cultures from the initial admission being positive for slow-growing gram-negative anaerobic rods (GNAR). The most common sources of GNAR are recognized to be genitourinary and gastrointestinal in origin. The patient had no history of benign prostatic hyperplasia, and denied any urinary complaints, with negative urine cultures from the initial admission. The patient did not have any abdominal complaints either; however, cross-sectional abdominal imaging was obtained to assess for any intra- abdominal collections/abscess, which surprisingly showed a 5.3 cm tubular radiopaque focus in the cecum suggestive of a foreign body. Blood cultures from the second admission also grew GNAR, which speciated into capnocytophaga. Gastroenterology was consulted to assist with endoscopic evaluation of this finding with possible retrieval of the foreign body. Colonoscopy was attempted and a chicken bone was found in the cecum which was successfully removed with a snare. Possible inflammation of this bowel segment from cecal trauma could have resulted in transient translocation of the bacteria from the cecum with resultant bacteremia. Subsequent blood cultures after FB removal were negative suggesting adequate source control.
**Discussion:** GNAR bacteremia warrants workup for genitourinary and gastrointestinal sources. Our case highlights a rare clinical scenario where FB ingestion led to GNAR bacteremia. Though this is a very rare phenomenon, clinicians investigating the causes of GNAR bacteremia should have a broad differential in cases where the culprit is not obvious. Timely identification of the GNAR source and its control is very crucial in treating such population.

**S2497**

**Duodenal Hematoma With Partial SBO After Routine EGD in a Young Female Patient With No History of Hematologic Disorder**

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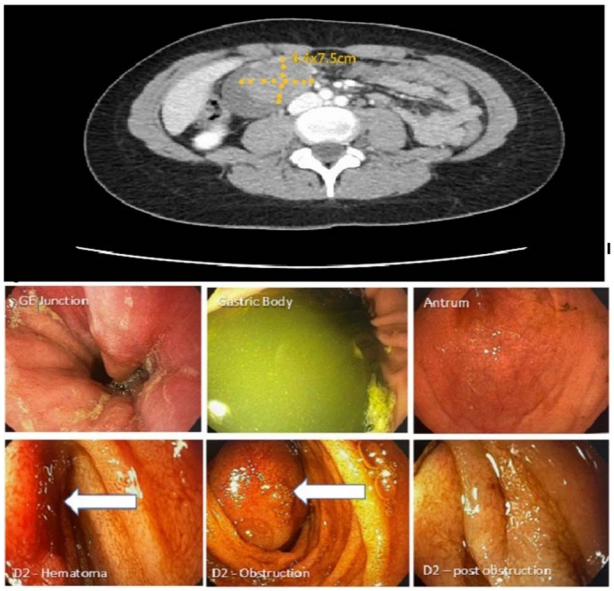
*[1]Trinity Health Livonia, Livonia, MI; [2]A.T. Stills University, Livonia, MI; [3]St. Mary Mercy Hospital, Livonia, MI.*

**Introduction:** Duodenal hematomas are a rare complication of endoscopic biopsy most often seen in children and young adults. Common factors include blunt trauma, anticoagulation, Henoch-Schonlein purpura and blood dyscrasias. They can cause significant morbidity and mortality, including duodenal obstruction, hospitalization and need for intravenous nutrition. Intramural hematomas are more commonly associated with complications. Treatment is generally conservative and consists of electrolyte replacement, decompression, and total parenteral nutrition. If conservative management fails, endoscopic or surgical interventions to relieve the hematoma are required.
**Case Description/Methods:** A 22-year-old female with past medical history of chronic abdominal pain presented to the emergency department 7 hours after an uncomplicated esophagogastroduodenoscopy (EGD). She complained of abdominal pain, nausea, and vomiting. In the ED her vital signs were stable. Labs were remarkable for a mild leukocytosis and elevated total bilirubin. CT abdomen/pelvis was

concerning for duodenal hematoma (**Figure**, **Top**). Repeat EGD showed intramural duodenal hematoma at the biopsy site with no active bleeding (**Figure, Bottom**). Patient was managed conservatively and discharged home with GI follow up. She returned to the ED at another hospital 4 days later for ongoing nausea, vomiting and abdominal pain. There was interval enlargement of the hematoma with increasing mass effect on the duodenum with no signs of infection or active bleeding. Consideration was given to endoscopic evacuation or percutaneous drainage. No invasive therapy was required, and she was discharged with GI follow up.

**Discussion:** Hemorrhages such as this one can lead to intramural accumulation of blood resulting in a hematoma that pulls fluid from the surrounding area by osmotic fluid shift. This forms an intraluminal bulge which can lead to duodenal occlusion causing compression on the pancreatic and biliary ducts. Typical symptoms include abdominal pain, nausea, and vomiting. Our patient was successfully managed conservatively. If conservative management fails, next step is endoscopic or surgical evacuation of the hematoma which is typically considered after 7-10 days of conservative therapy or if there are worsening complications. Newer therapies include ultrasonically guided drainage and balloon dilation. Our case illustrates the rare complication of intramural hematoma in a young adult, with no history of hematologic pathology, following routine EGD with duodenal biopsy.




[2497] **Figure 1.** Top - CT abdomen/pelvis showing duodenal hematoma. Bottom - EGD images showing GE junction, gastric contents, antrum, intramural hematoma in the 2nd portion of the duodenum and 2nd portion of duodenum distal to obstruction.

**S2498**

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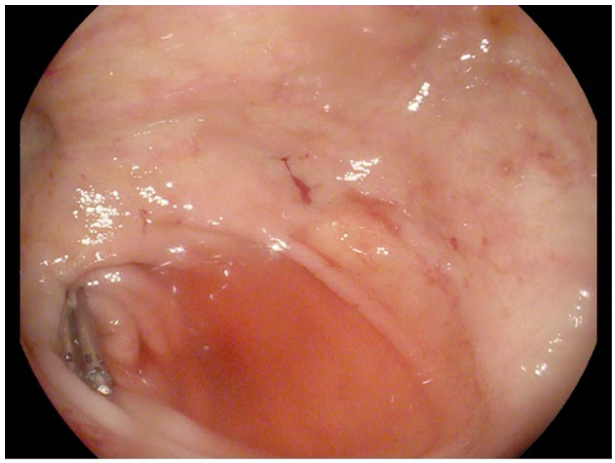
**Endoscopic Treatment of a Dieulafoy's Lesion in the Appendiceal Orifice**

*Justin P. Canakis, DO[1], Justin Ong, BS[2], Danial Nadeem, MD[3], Leen Raddaoui, MD[2], Samuel A. Schueler, MD[1].*
*[1]George Washington University, Washington, DC; [2]The George Washington University School of Medicine and Health Sciences, Washington, DC; [3]MedStar Washington Hospital Center, Washington, DC.*

**Introduction:** A Dieulafoy's lesion, typically a large caliber submucosal artery that erodes gastrointestinal (GI) mucosa, is a less common cause of GI bleeding. While these vascular abnormalities may occur throughout the GI tract, appendiceal Dieulafoy's lesions are exceedingly rare, with described cases often resulting in surgical intervention. We present a case of a Dieulafoy's lesion in the appendiceal orifice leading to massive GI bleeding which was successfully diagnosed and treated endoscopically.

**Case Description/Methods:** A 75-year-old female with end stage renal disease, hypertension, hyperlipidemia, and heart failure with preserved ejection fraction presented with abdominal pain and rectal bleeding for 2 weeks duration. At onset, she was admitted to an outside hospital where computed tomography (CT) angiography showed aortoiliac and mesenteric atherosclerosis without evidence of large vessel occlusion. Esophagogastroduodenoscopy did not reveal a bleeding source. She reported infraumbilical abdominal pain and continued rectal bleeding and was transferred to our medical center. Upon arrival, the patient was hemodynamically stable with active rectal bleeding on exam. Laboratory analysis revealed hemoglobin of 6.9 grams per deciliter, platelet count of 101 per milliliter, blood urea nitrogen of 6.6 milligrams/deciliter (mg/dl), and creatinine of 6 mg/dl. She was treated with 3 units of packed red blood cell and a proton pump inhibitor. Colonoscopy showed a Dieulafoy's lesion at the appendiceal orifice. Hemostasis was achieved with placement of 2 hemoclips (**Figure**). She was discharged 6 days after colonoscopy without recurrence of bleeding.

**Discussion:** Only 6 cases of appendiceal Dieulafoy's lesions have been reported in the literature and all were treated with laparoscopic appendectomy (**Table** 1). To our knowledge, this is the first reported case of an appendiceal Dieulafoy's lesion that was successfully treated with endoscopic placement of hemoclips. There is no data comparing the efficacy of endoscopic intervention versus laparoscopic appendectomy in treating appendiceal Dieulafoy's lesions; however, this case highlights that therapeutic endoscopy may be both safe and effective. Further reports are needed to inform recognition and optimal approach to appendiceal Dieulafoy's lesions. Furthermore, in cases where hemostasis is achieved endoscopically, longer term follow-up may inform if appendectomy can be safely avoided.



[2498] **Figure 1.** Successful hemostasis of a Dieulafoy's lesion at the appendiceal orifice after placement of 2 hemoclips.

**Table 1. Previously reported Appendiceal Dieulafoy's Lesions% and Outcomes**

| Case Report | Patient | Clinical presentation and course | Endoscopic hemostasis attempted? | Management? |
|---|---|---|---|---|
| Xue et al, 2020 | 21 F | Massive hematochezia with lower abdominal pain and LOC* | N | Laparoscopic appendectomy |
| Choi et al, 2016 | 72 M | Hematochezia with associated mild abdominal pain and bloating sensation, previous melena from duodenal ulcer | N | Laparoscopic appendectomy and cecum wedge resection |
| Johnson et al, 2014 | 51 M | RLQ# pain, Acute appendicitis with incidental finding of Dieulafoy's lesion on mid-distal appendiceal wall | N | Laparoscopic appendectomy |
| Reynolds et al, 2013 | 68 M | Massive hematochezia with perfusion requirement | N | Laparoscopic appendectomy |
| Lee et al, 2011 | 22 M | Severe lower GI† bleed | N | Laparoscopic appendectomy |
| So et al, 1995 | 42 M | Melena and dizziness | N | Laparoscopic appendectomy |

%In all previously reported cases, a Dieulafoy's lesion was diagnosed using colonoscopy to visualize blood emerging from the appendiceal orifice, then subsequent resection revealing an ulcerated appendiceal mucosal lesion and microscopy and histopathology demonstrating tortuous vasculature penetrating the circumferential and longitudinal muscular wall of the appendix. Abbreviations: *Loss of consciousness, #Right lower quadrant, †Gastrointestinal.

**S2499**

**Gastric Adenocarcinoma After Sleeve Gastrectomy**

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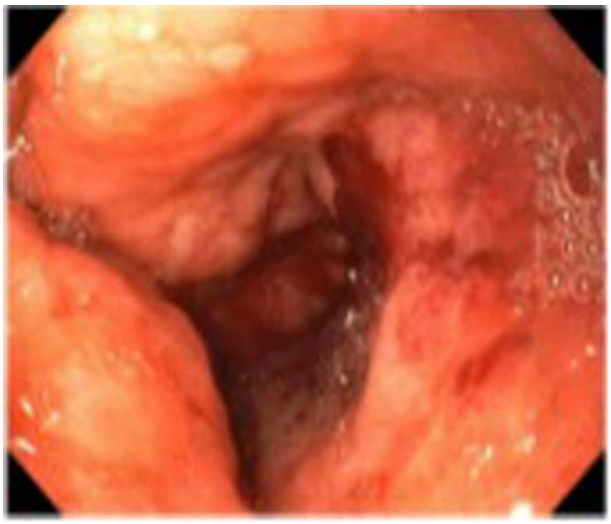
*[1]Saint Peter's University Hospital, New Brunswick, NJ; [2]Rutgers Medical School of Robert Wood Johnson, Saint Peter's University Hospital, New Brunswick, NJ.*

**Introduction:** Gastric cancer remains the fourth most common cancer worldwide. Unfortunately, it is usually diagnosed in advanced stage. Risk factors include *Helicobacter pylori*, gastroesophageal reflux disease, and obesity. Reported cases of gastric cancer after bariatric surgery have been known to be anecdotal. Laparascopic sleeve gastrectomy for obesity is increasingly preferred by surgeons due to its easy technique combined with excellent weight loss results. However, recently there has been a rising incidence of gastric-esophageal cancers seen post bariatric surgery. Cancer in the distal stomach after gastric bypass was first described in 1991 in a female patient 5 years after the original procedure. We present a case who was found to have gastric adenocarcinoma 7 years post sleeve gastrectomy.
**Case Description/Methods:** A 65-year-old Hispanic male presented to the emergency department with a complaint of vomiting for 10 days in duration. This was associated with epigastric pain and weight loss. His past medical history was significant for morbid obesity for which he underwent a gastric sleeve 7 years prior to presentation. He had GERD and *H. pylori* treated and eradicated with quadruple therapy. He did not report any family history of GI malignancies. On examination, the vital signs and physical examination were unremarkable. His laboratory values were normal as well. Abdominal radiography, right upper quadrant ultrasound and computed tomography did not show evidence of intra-abdominal pathology. Upper endoscopy showed a circumferential mass in the fundus of the stomach. A biopsy confirmed gastric adenocarcinoma with focal signet cell features. He was started on neoadjuvant therapy and had a J tube put in place in order to tolerate feeds.
**Discussion:** The aim of this report is to show a case of de-novo gastric adenocarcinoma after bariatric surgery with sleeve gastrectomy. Although, obesity, a known risk factor for gastric carcinoma, increasing trends have been noted with rise in carcinoma after sleeve gastrectomy. It is not known whether bariatric surgery is the cause or the fact that patient had history of *H. pylori* treated that predisposed him to develop the malignancy. The relationship between bariatric surgery and subsequent gastric carcinoma needs further investigation. Patients might be at higher risk of developing gastric adenocarcinoma post sleeve gastrectomy especially if they have history of *H. pylori* infection in the past and this high-risk group might benefit from surveillance EGD.



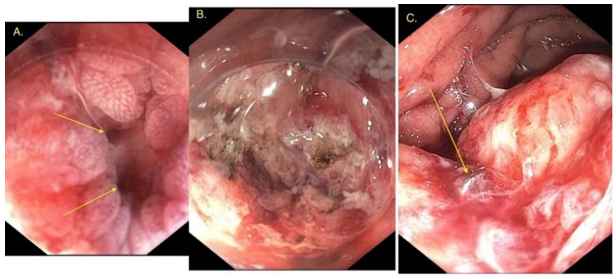
[2499] **Figure 1.** Endoscopy: Circumferential mass in gastric body.

**S2500**

**Gastrosplenic Fistula - An Open and Closed Case**

*Thomas J. Mathews, MD, Mitesh Patel, MD.*
*University of Kansas Medical Center, Kansas City, KS.*

**Introduction:** In this case, we will discuss endoscopic treatment for immunocompromised patients with gastrosplenic fistulas.
**Case Description/Methods:** A 36-year-old White female with history of diffuse large B cell lymphoma (DLBCL) on rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone (R-CHOP) chemotherapy presented with neutropenic fever, persistent cough with deep inspiration, and burning pain in her left shoulder that had worsened since chemotherapy 2 weeks prior. On admission, she was tachycardic and afebrile. White blood cell count was 1,200/mm$^3$, absolute neutrophil count 120/mm$^3$, and blood cultures were negative. CT demonstrated improved splenomegaly measuring 6.9 x 7.0 cm (previously 13 x 11 cm) and moderate gas and fluid within the spleen with suspected fistulous communication to the adjacent greater curvature of the stomach. An upper GI series confirmed gastrosplenic fistula (GSF) and fluid collection in the spleen. Gastroenterology and interventional radiology (IR) were consulted. Endoscopy revealed diffuse, severely congested mucosa in the gastric fundus and body, causing difficulty visualizing the fistula. An endocap was attached to the endoscope to assist with visualization. A 5 mm fistula with ulceration was found on the greater curvature of the gastric body. Argon plasma coagulation was performed for tissue devitalization in and around the fistula. The scope was then outfitted with an over-the-scope clip, which successfully closed the fistula (**Figure**). IR then placed an abdominal drain, and fluid cultures grew Streptococcus constellatus, Streptococcus anginosus, Lactobacillus rhamnosus, and Parvimonas micra. Empiric antibiotics were changed to intravenous ertapenem. The patient was able to tolerate a regular diet. Three weeks later, CT abdomen revealed significant decrease in size of splenic gas and fluid collections.
**Discussion:** GSFs are a rare complication in patients with lymphoma and occur almost exclusively in patients with DLBCL. This patient did not present with typical features such as melena, hematochezia, severe sepsis, severe abdominal pain, nausea, vomiting, or hematemesis. It is important to consider GSF in patients with DLBCL presenting with GI issues. The overwhelming majority of GSF cases are surgical emergencies, but not every case will require surgical intervention. This can be important in patients at increased risk of surgical morbidity and mortality. A GI consult could save patients from unnecessary risk and financial burden.




[2500] **Figure 1.** A. Gastrosplenic fistula (gastric body) B. After APC C. After Clip Deployment.

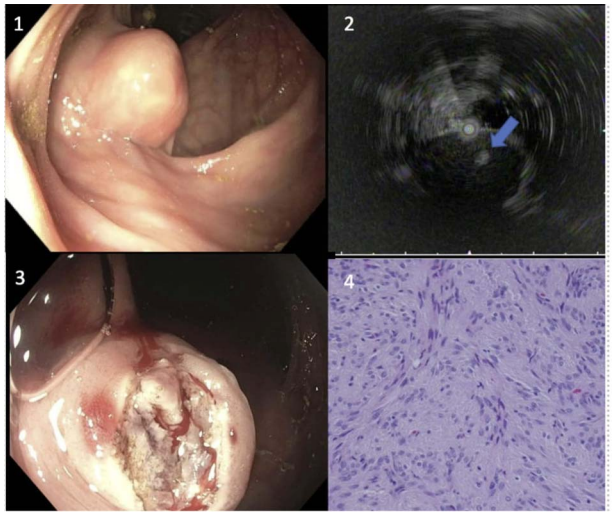
**S2501**

**Getting to the GIST of It: Rare Case of GIST in Ascending Colon**

*Deepa Kumarjiguda, DO[1], Shreya Vatsala, MD[2], Ruchit Shah, DO[1], Bradley D. Confer, DO[2].*
*[1]Geisinger Health System, Danville, PA; [2]Geisinger Medical Center, Danville, PA.*

**Introduction:** Gastrointestinal stromal tumors (GIST) are the most common mesenchymal tumor of the gastrointestinal (GI) tract. GIST is most localized in the stomach (50 – 60%), followed by the small intestine (30 – 40%), and the colon (5-10%). Herein we describe a unique case of an ascending colon GIST requiring advanced endoscopic dissection to confirm the diagnosis.
**Case Description/Methods:** A 78-year-old male presented with generalized abdominal discomfort associated with heartburn and for colon cancer screening. The upper endoscopy was unremarkable. On colonoscopy, a 4 cm subepithelial lesion was found in the ascending colon (Panel 1). Traditional endoscopic ultrasound (EUS) using a linear or forward viewing scope was not possible given the redundant sigmoid colon, leading to significant scope-looping. The lumen was filled with water and the miniprobe ultrasound catheter was advanced through the working channel of the colonoscope to assess the lesion. Ultrasound revealed a heterogenous, hypoechoic 4 by 3 cm lesion arising from the muscularis propria, suggestive of a mesenchymal tumor (Panel 2). Dissection of the middle portion of the lesion was completed using needle-knife to the muscularis propria and biopsy forceps were used to obtain deep biopsy (Panel 3). A clip was placed due to the small increased risk of delayed bleeding or perforation. Histopathology was positive for CD117, DOG-1, CD34, and h-caldesmon and stains negative for S100, GFAP, and SMA, consistent with GIST (Panel 4). Further imaging of chest, abdomen, pelvis did not show metastatic disease. Surgical evaluation was recommended due to size, heterogeneous appearance on EUS and colonic origin. The patient underwent a right hemicolectomy. Pathology confirmed a low mitotic GIST. The post operative period was uneventful, and he was discharged home. He did not require adjuvant imatinib.

**Discussion:** Tumor size, mitotic rate, and location have been studied and used as prognostication factors to predict degree of malignancy potential in GIST but is only validated in gastric and small intestinal tumors. Knowledge is limited regarding colonic tumors versus antral tumors since occurrence is rare. Current data suggest colonic tumors are more malignant. Due to the unusual location of the subepithelial lesion and redundant colon, traditional EUS would not work to biopsy the lesion. Needle knife dissection assists in identifying and finding cancerous lesions in challenging situations.




[2501] **Figure 1.** Panel 1. Endoscopic image of colonic subepithelial lesion. Panel 2. EUS demonstrates hypoechoic lesion with small hyperechoic area (blue arrow) in the center of the lesion. Panel 3. Post needle-knife dissection and biopsy. Panel 4. Histopathology reveals spindle cell morphology confirming GIST along with correlating diagnostic stains on H&E stain.

**S2502**

**Gastrointestinal Kaposi Sarcoma: A Case Report**

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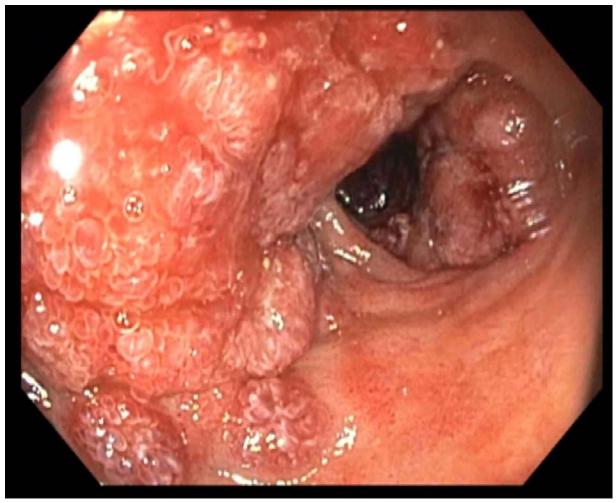
*[1]William Beaumont Hospital, Royal Oak, MI; [2]Beaumont Health, Royal Oak, MI*

**Introduction:** Kaposi sarcoma (KS) in the gastrointestinal (GI) tract is an uncommon entity specially without any cutaneous manifestation since the introduction of HAART therapy in HIV patients. Most of the gastrointestinal KS, remains undiagnosed as majority of KS is asymptomatic. Here we present a case of GI- KS in a HIV-AIDS patient.
Case Description/Methods: A 21-year-old male with history of HIV-AIDS came in for nausea, vomiting and hematochezia for 1 month. Lab showed; total CD4 of 10, elevated ALP 435 U/L, AST, ALT, total and direct bilirubin, lipase was normal. There was periodic increase in ALP hence MRCP was done, which revealed multiple hepatic and intrabiliary lesions. On EGD and colonoscopy, a large infiltrative mass, with recent stigmata of bleeding was found in the duodenum (Figure) to the ileum causing a partial obstruction. The biopsy showed extensive spindle cell proliferation with immunostains positive for HHV8 , ERG which was consistent with Kaposi Sarcoma. A liver biopsy was also done which showed non caseating granuloma with lymphocytic infiltrate with portal ductal proliferation and edema. He underwent ERCP revealing multiple segmental moderate biliary strictures in the common bile duct (CBD), left and right intrahepatic branches. A biliary sphincterotomy and a plastic stent in CBD was performed. He was on HAART therapy and anthracycline based chemotherapy was considered, but the patient passed away due to multiorgan failure from sepsis.
**Discussion:** GI- KS can present as multiple GI symptoms including nausea, vomiting, GI bleeding as in our case or abdominal pain, anemia. (1) However, most of the time gastrointestinal KS remains asymptomatic. (1) Clinician should have high suspicion for GI-KS especially in patients with HIV- AIDS, not in therapy. This will help in the earlier diagnosis and management of GI-KS.



[2502] **Figure 1.** Large infiltrative mass of Kaposi Sarcoma found in duodenum.

**S2503**

**Heads or Tails? A Quarter Impacted Proximal to the Ileocecal Valve Is Endoscopically Retrieved Revealing the Answer**

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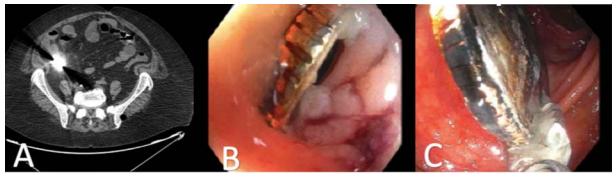
**Introduction:** Persistent foreign body impaction in adults is rare, as the majority of ingested foreign bodies pass spontaneously. Only 10-20% require endoscopic intervention, and about 1% require surgery. Foreign body ingestion is usually seen in children, psychiatric, or elderly patients. Impaction at the ileocecal valve poses a particular challenge as endoscopic retrieval is technically difficult and may carry an increased risk of perforation.
**Case Description/Methods:** A 73-year-old female with atrial fibrillation and heart failure presented to the hospital with a 2-week history of right-sided abdominal pain. She was tolerating a regular diet, passing stool and flatus regularly, and had no nausea or vomiting. A CT scan was performed and showed a rounded metallic foreign body within the terminal ileum. The patient denied any known ingestion of a foreign body. She was observed in the hospital for 2 days with no passage of the foreign body on serial abdominal X-rays. On the third day, endoscopic retrieval was attempted. The terminal ileum was successfully intubated, and a metallic foreign body was visualized behind an ulcerated stricture in the terminal ileum. Removal with a rat-tooth forceps was attempted but was unsuccessful due to the presence of an ileal stricture. Surgery was consulted, but ultimately deferred intervention due to the patient's extensive comorbidities. A repeat colonoscopy was performed. The terminal ileum was intubated and was found to be strictured 10cm proximal to the ileocecal valve. An 0.035 inch guidewire was placed across the stricture using endoscopic and fluoroscopic guidance. A 8-10 mmCRE Balloon Dilator was passed over the guidewire. The terminal ileum was dilated, and the foreign body was retrieved with rat-tooth forceps. The metallic object was found to be a US quarter. The following day, the patient's pain fully resolved and she was discharged from the hospital (Figure).
**Discussion:** Ingested coins can become impacted in the gastrointestinal tract causing obstruction, pain, and rarely perforation. Persistence of an impacted foreign body can cause ulceration, intestinal stricture, and fistula formation. The appropriate management for impacted foreign bodies distal to the ligament of Treitz is close observation as the majority of the impacted objects pass spontaneously. However, if symptoms persist and the foreign body does not pass on serial imaging, endoscopic removal can be performed for symptomatic relief and to avoid further complications such as perforation.




[2503] **Figure 1.** A. CT scan showing coin-like opacity in the terminal ileum. B. Coin impacted in the terminal ileum with surrounding ulceration and stricture. C. Endoscopic retrieval of a US quarter.

**S2504**

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**Giant Gastric Cardia Lipoma**

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**Introduction:** Lipomas are slow-growing benign tumors which are rarely found in the gastrointestinal tract. They are more common in the colon, but can also be found in the stomach. Gastric lipomas represent < 3% of benign gastric tumors, and giant gastric lipomas ( >4cm) are even less common. Most lesions remain asymptomatic, but pain, dyspepsia, intussusception, obstruction and bleeding occur as their size increases. There are few reports of giant gastric lipomas in the literature. We present an illustrative case.
**Case Description/Methods:** A 52-year-old Asian male presented to our outpatient clinic with pain and burning in the stomach for about 6 to 8 weeks. He denied nausea, vomiting, early satiety, or weight loss. His symptoms were somewhat improved after eating and with OTC omeprazole. He reported consuming 3-4 drinks of alcohol every few days as well as smoking 1/2 pack per day for the last 2 years. On physical exam vitals were normal and he had mild epigastric tenderness. The only abnormal lab was a mildly elevated ALT of 47. Upper endoscopy revealed a large ulcerated mass with normal overlying mucosa in the gastric cardia. This was biopsied and pathology was reported as chronic active inflammation, ulceration, and granulation tissue. Staining for Helicobacter pylori returned negative. EUS confirmed an intramural, subepithelial lipoma at the lesser gastric curvature. FNA was negative for malignant cells; only lymphocytes and neutrophils were seen. The patient underwent robotic wedge gastrectomy and complete resection of the giant 8 cm gastric lipoma with complete resolution of symptoms 4 weeks post-operatively.
**Discussion:** Giant gastric lipomas are sparsely reported in the literature. One review suggested that these lesions frequently present with life-threatening UGI bleeding. Diagnostic workup generally includes EGD and abdominopelvic CT. FNA via EUS is pursued when initial biopsies are inconclusive. There is no universally accepted standardization of therapy once lipoma is confirmed. Endoscopic or surgical resection result in a highly favorable prognosis. Our patient was found to have an 8cm lipoma that caused only vague symptoms of dyspepsia and epigastric pain, and proceeded for a totally robotic approach for resection with complete resolution of his presenting complaints. His case illustrates the heterogeneity in possible presentations of this infrequently described scenario. His favorable outcome highlights the success of minimally invasive surgery as a therapeutic modality.

**S2505**

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**Holy Cow! When Cecal "Polypectomy" Specimen Shows Cow's Liver**

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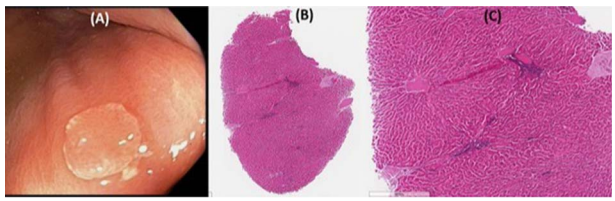
**Introduction:** Patients with history of colon tubular adenomas need surveillance colonoscopies to decrease the risk of colon neoplasia. Sometimes, contamination of polypectomy specimen with ingested food can create confusion while interpreting the results. We present an interesting case of a cecal "polypectomy" specimen that showed liver tissue, that on further investigation was attributed to a cow's liver that the patient ingested the day before his procedure.
**Case Description/Methods:** A 72-year-old White male with past medical history of colon tubular adenomas, chronic constipation, hypertension, obstructive sleep apnea and class III obesity was referred for surveillance colonoscopy. His constipation was well-controlled with linaclotide 72 mcg daily. He did not report any hematochezia or weight loss. His physical examination including vital signs and abdominal examination was unremarkable. Colonoscopy was performed after a 4-liter polyethylene glycol bowel preparation which revealed 3 polyps. These included a 5 mm Paris 0-1s polyp which was removed via cold snare polypectomy (Figure A), and 2 2 mm sessile polyps in the ascending and descending colon (removed via biopsy forceps). The ascending and descending colon polyps were tubular adenomas on histological examination. We were notified by the reading pathologist that the cecal "polyp" showed liver tissue (Figure B, C) with the concern for iatrogenic perforation of the colon during colonoscopy and accidental biopsy of patient's liver. The patient denied any symptoms suggestive of colonic perforation. Upon further discussion, he informed us that he ate fried cow's liver the day before the procedure which was inadvertently suctioned in the trap after cold snare polypectomy of his cecal polyp.
**Discussion:** Identification of liver tissue in colonoscopy polypectomy specimens should raise possibility of catastrophic complication such as a perforation or simply a possibility of contamination of specimen with ingested food. Following-up with the patient to clarify the situation is important. A careful review of patient's dietary intake before the colonoscopy procedure can help correctly interpret the unexpected pathology results.




[2505] **Figure 1.** 1A: Paris 0-1s 5 mm cecal polyp during surveillance colonoscopy. Figure 1B: At low power the section shows an eosinophilic homogenous tissue without surface epithelium. Figure 1C: At higher power: sheets of polygonal cells with round, centrally located nuclei, and abundant eosinophilic cytoplasm compatible with hepatocytes. The hepatocytes arranged in plates extend from central vein to portal triad.

S2506

**Hit Me With Your Best Shot: Splenic Laceration Post Colonoscopy**

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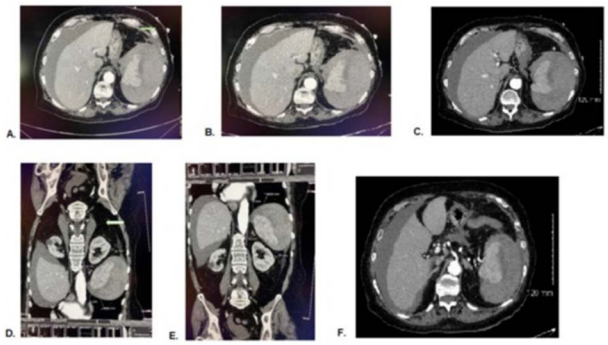
**Introduction:** With more than 16 million colonoscopies conducted each year in the United States, colonoscopy is the gold standard for detecting any colonic pathology. The most common complications described are intraluminal hemorrhage (0.3–2.1%) and colonic (micro)perforation (0.1–2.5%). Splenic injury during colonoscopy was initially identified by Wherry and Zehner as a rare but life-threatening event. Here, we discuss a case of post colonoscopy splenic laceration associated with multiple polypectomies.
**Case Description/Methods:** 74-year-old female with hypertension, hyperlipidemia, on no blood thinners, presented 3 days after screening colonoscopy (16 polyps removed) to the hospital with complaints of left upper quadrant abdominal pain, weakness and fatigue without any GI bleeding. Of note, a previous colonoscopy 3 years ago with 18 removed, all benign, was uneventful. All her polyps were sub centimeter, required only hot and cold snare, no endoscopic mucosal resection. Labs demonstrated hemoglobin of 7gm/dl, WBC 15.5K, creatinine 1.7, lactic acid 2.2, and CT the abdomen revealed 3 cm splenic laceration grade 3 with subcapsular hematoma without bleeding (Figure 1). She was transfused 2 units of packed cells with improvement of her hemoglobin to 10.0 after which her blood count remained stable, she was monitored and treated for pain, and discharged successfully home after 2 days with instruction to avoid high-impact activities which could result in further trauma to the spleen.
**Discussion:** Splenic injury has an incidence ranging from 1 in 100,000 to 1 in 6,387 colonoscopies . The mortality rate for a traumatic splenic injury requiring splenectomy has been reported to be 25%. Advanced age, with an average age of 63.0 years, is thought to be a risk factor for splenic damage, with female patients accounting for 72–75 percent of splenic injuries after colonoscopy. The most likely causes being excessive manipulation done to advance the scope beyond the splenic flexure or traction on the splenocolic ligament. A higher incidence has been observed when biopsies or polypectomies were performed during therapeutic colonoscopies, with polypectomy accounting for a 7-fold increase risk of adverse events. This complication may be avoided with a left lateral position and minimizing the external pressure. Henceforth, we would like to draw your attention to the fact that a clinician should be extra cautious while doing these maneuvers since, while it is a rare complication, it can be life threatening.




[2506] **Figure 1.** A-F - Coronal view of linear lucencies within the spleen suggestive of lacerations. The largest measures 3 cm. There is also an 11 cm subcapsular splenic hematoma. There is no evidence of acute contrast blush. Findings are consistent with grade 3 splenic injury.

S2507

**Hepatic Abscess and Septicemia From *Bacteroides Pyogenes* in a Patient Undergoing Endoscopic Retrograde CholangioPancreatography (ERCP)**

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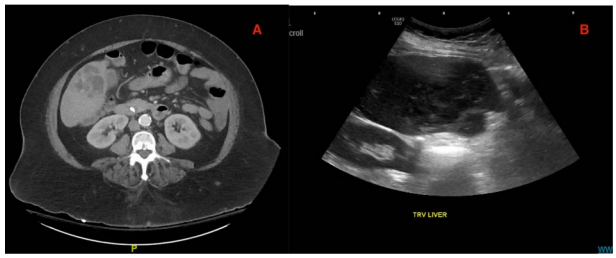
**Introduction:** Liver abscess is a rare but reported complication of ERCP. Typical organisms are members of the *Enterobacteriaceae* family, such as *Klebsiella* and *Escherichia coli*. We report the first case of a liver abscess and bacteremia caused by *Bacteroides pyogenes,* an anaerobic Gram-negative rod, in a patient who underwent ERCP.
**Case Description/Methods:** A 73-year-old woman presented to the emergency department at a large academic medical center 11 days after ERCP for obstructive choledocholithiasis with 3 days of severe dyspnea, sore throat, weakness, anorexia, and right upper abdominal discomfort. She had 2 other ERCPs in the past month for work-up of possible extrahepatic cholangiocarcinoma. In the ED, her temperature was 101.3F. Her Blood Pressure was 86/48. She responded to 3L of IV fluids. Labs showed a leukocytosis of 13.0 and AST/ALT of 149/116. A CT of the abdomen showed a 7.2x5.3 cm abscess in the right hepatic lobe. She was started on ceftriaxone and metronidazole. A hepatic drain was inserted into the abscess, and it continued to drain throughout the hospitalization. Cultures of the drain and blood grew *Bacteroides pyogenes*. The patient was discharged on hospital day 6 with a PICC line to complete a 28-day course of ceftriaxone 2g daily and metronidazole 500mg 3 times daily (Figure).
**Discussion:** Hepatic abscesses are rare, serious complications of ERCP, with mortality ranging from 6-15%. Most hepatic abscesses in the US are caused by native aerobic flora, such as *Enterobacter, Streptococci, Staphylococci, or Enterococci* spp. This patient presented with a hepatic abscess and septicemia from *B. pyogenes,* an anaerobic Gram-negative rod found in the oral flora of dogs and cats that may causes wound infections and can rarely cause septicemia in persons with dog bites. This happened 11 days after her third ERCP within 2 months. She had no traumatic dog exposure but did admit to having a dog that gave her frequent "kisses." There has been one other case of hepatic abscess and septicemia from *B. pyogenes* reported, and 13 cases of *B. pyogenes* septicemia reported in total, with only 2 cases not having had a traumatic animal bite. This is the first case of *B. pyogenes* causing hepatic abscess and septicemia after ERCPs. This highlights the need to ensure empiric antibiotic therapy for liver abscess covers anaerobes and indicates that more studies are needed to understand the pathologic potential of anaerobes such as *B. pyogenes,* which appear to be increasing in systemic disease incidence.




[2507] **Figure 1.** A. Axial CT demonstrating a loculated rim-enhancing fluid collection in the right hepatic lobe adjacent to the gallbladder fossa consistent with hepatic abscess. B. The hypoechoic abscess from a transverse hepatic view.

S2508

**Ischemic Gastritis With Gastric Pneumatosis and Portal Venous Gas**

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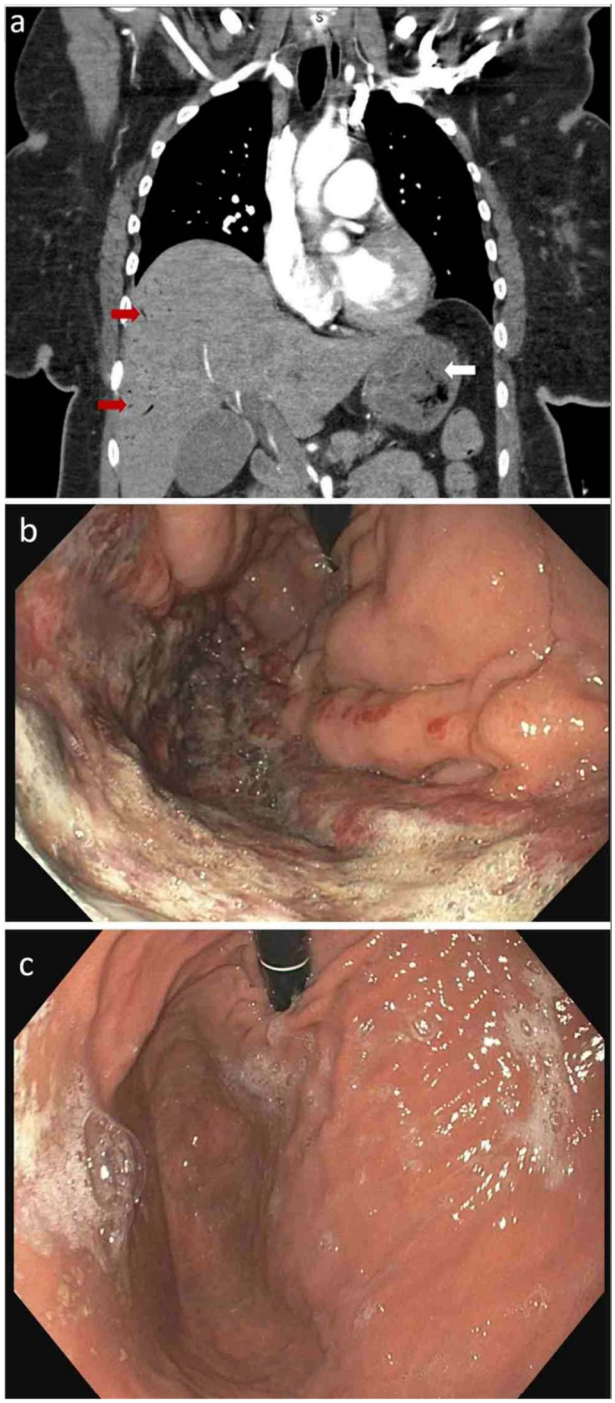
**Introduction:** Gastric ischemia is rare due to extensive collateral blood supply to the stomach, but if it occurs is associated with poor prognosis. We present the case of acute severe gastric ischemia in the context of severe hypotension with demonstration of portal venous gas (PVG) and gastric wall pneumatosis. The inciting event was a suspected gastric volvulus which had spontaneously resolved by the time of radiologic and endoscopic evaluation. This case highlights the successful conservative management of this dreaded clinical condition.
**Case Description/Methods:** A 50-year-old African American woman presented to the emergency department with 1-day history of severe lower retrosternal and epigastric pain with persistent nausea, coffee ground emesis and retching. Examination revealed an ill-appearing, tachycardic, hypotensive (blood pressure 70/41mmHg) female with epigastric tenderness. The rest of vitals, physical exam, EKG and chest x-ray were normal. Basic labs after initial fluid resuscitation noted WBC of $12.5x10^9$/L. Troponin, electrolytes, liver enzymes and lactic acid were normal. Chest CT angiogram showed peripheral hepatic PVG (Figure A, red arrows) and a thickened gastric wall with pneumatosis (Figure A, white arrow) suggestive of gastric ischemia. No evidence of pulmonary embolism, bowel perforation or vascular abnormality was found. Esophagogastroduodenoscopy (EGD) revealed extensive necrotic ulcerative changes along gastric greater curvature from fundus to body consistent with gastric ischemia (Figure B). Treatment included intravenous fluids and proton pump inhibitors, analgesics, IV antibiotics (ceftriaxone and metronidazole) and bowel rest. Patient responded well to treatment and was discharged home after 6 days. Patient remained asymptomatic and a repeat EGD after 12 weeks revealed completely normal appearance of stomach (Figure C).
**Discussion:** Gastric ischemia may be caused by local vascular abnormalities, systemic hypoperfusion and mechanical obstruction. Typical symptoms include abdominal pain, vomiting and gastric bleeding. The mechanism for developing PVG in bowel ischemia is not fully understood but usually suggests an ominous pathology which may require a prompt surgical intervention. Transient gastric volvulus was suspected as a possible initial trigger, causing hypotension and gastric ischemia as no other etiology was identified. Enhanced provider cognizance of gastric ischemia and appropriate management tailored to each patient's needs should improve clinical outcomes.




[2508] **Figure 1.** Chest CT angiogram showed peripheral hepatic PVG (Figure A, red arrows) and a thickened gastric wall with pneumatosis (Figure A, white arrow). EGD showing extensive necrotic ulcerative changes along gastric greater curvature from fundus to body (Figure B). EGD after 12 weeks showing normal stomach (Figure C).

**S2509**

**Mantle Cell Lymphoma Presenting as Multiple Lymphomatous Polyposis: A Case Report**

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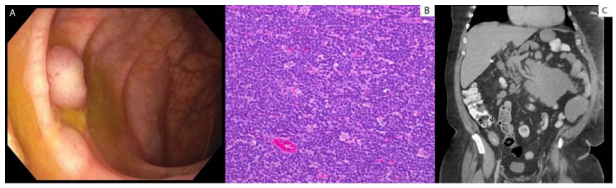
**Introduction:** Mantle Cell Lymphoma (MCL) typically presents with extensive lymphadenopathy, fevers, night sweats and unintentional weight loss. However, Multiple Lymphomatous Polyposis (MLP), MCL arising from the gastrointestinal tract, is an aggressive malignancy and is infrequently described.
**Case Description/Methods:** A 68-year-old White man with atrial fibrillation and hypertension presented to the hospital with watery diarrhea, fatigue and intermittent epigastric pain for the past several weeks. He denied melena, hematochezia, heartburn, dysphagia, odynophagia, weight loss, loss of appetite, NSAID use, recent travel or sick contacts. There was no family history of gastrointestinal malignancy. No history of prior endoscopic evaluation. Physical exam was notable for brown stool on rectal exam. Further evaluation revealed Hgb 6.4, MCV 60.9, iron 43, TIBC 442 and iron saturation 10% consistent with iron deficiency anemia. Upper endoscopy was normal. Colonoscopy showed 8 6-16 mm polyps scattered throughout the colon, all of which were resected completely. Analysis showed a range of histology – 3 tubular

adenomas, 1 serrated and 2 hyperplastic polyps. However, histology of 4 polypoid appearing lesions (Figure 1A) showed prominent nodular atypical submucosal lymphoid aggregates (Figure 1B) that were positive for CD20, cyclin D1, weak CD5, and negative for CD10 and CD23. This was consistent with the diagnosis of MCL. CT imaging showed diffuse lymphadenopathy, including bulky disease with a 16.6 x 10.6 cm small bowel mesenteric mass (Figure 1C), along with proximal small bowel and terminal ileal thickening. He was induced with 6 cycles of bendamustine-rituximab combination therapy and maintained on rituximab. A re-staging scan at 27 months showed no evidence of disease progression.
**Discussion:** We present a case of MCL presenting as MLP with symptoms of anemia, diarrhea and epigastric pain. MCL is a rare B-cell non-Hodgkin's lymphoma that portends a poor prognosis, making early identification and diagnosis critical. Obstruction, GI bleeding, and perforation are common complications for MCL presenting as MLP. It is important to keep MLP on the differential when multiple small nodular or polypoid lesions are identified on colonoscopy. All different types of polyps should be resected or sampled during colonoscopy, and each evaluated by the pathologist to avoid missing clinically significant conditions. Early diagnosis is key to prevent morbidity and mortality in MLP as described in our case.




[2509] **Figure 1.** (A) polypoid lesion (B) nodular atypical submucosal lymphoid aggregates (C) 16.6 x 10.6 cm small bowel mesenteric mass.

**S2510**

**Limitations of Endoscopic Tools for Foreign Body Retrieval: A Case of Massive Ferromagnet Ingestion**

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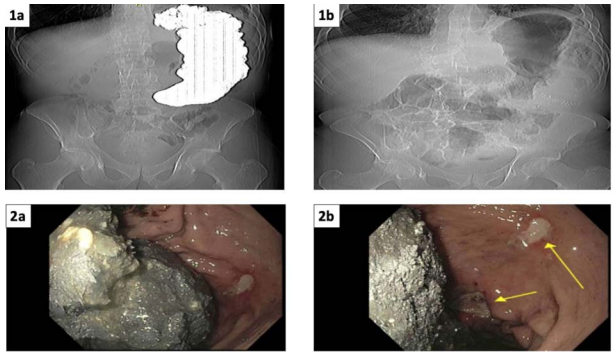
*University of Texas Health Science Center, San Antonio, TX.*

**Introduction:** Esophagogastroduodenoscopy (EGD) remains the standard of care for foreign body (FB) retrieval. The decision to pursue endoscopic intervention is based on multiple considerations including the probability of passage of the FB, the risk of potential injury caused by ingestion of the foreign body, and the risk of potential injury caused by performing the intervention. We present a case of FB ingestion resulting in practically impossible endoscopic retrieval. We also highlight endoscopic cues that should prompt a gastroenterologist to consider surgical evaluation in high-risk cases.
**Case Description/Methods:** A 49-year-old man with schizoaffective disorder and a history of multiple ingestions of foreign bodies presented to the hospital for vomiting and diarrhea for 4 days. An abdominal x-ray revealed a large radiopaque structure that conformed to the shape of the stomach in the gastric region [Figure 1a]. EGD revealed multiple large consolidated ovoid masses of gray metal in the gastric body and antrum [Figure 2a]. Endoscopic retrieval was attempted using a large cold snare, which could not be wrapped around or secured around the FB without slipping upon closure. A Roth net was tried with the same unsuccessful result. Bites of the FB using rat-tooth forceps slightly deteriorated the mass and the foreign body material impeded the full-closing of the jaws of the forceps so that the forceps could not be retrieved through the working channel. The malleable material then had to be irrigated and scraped off to allow for reuse. Due to the large size of the FB and small space in the stomach for gastroscope maneuverability and the presence of multiple scattered large and deeply cratered ulcers along the gastric wall, the procedure was aborted to avoid perforation [Figure 2b]. The patient was referred for surgical intervention and soon thereafter underwent FB removal via partial gastrectomy with gastric reconstruction [Figure 1b]. The recovered FB weighed 2,885 grams and measured 32 x 31 x 1.5 cm
**Discussion:** Care should be practiced when planning and executing the retrieval of foreign bodies. The type, size, and location of the FB, the patient's anatomy, the presence of deep ulceration and the risk of organ perforation are important considerations for the endoscopist. Surgical intervention should be considered when the risk of perforation with endoscopic intervention is high and/or endoscopic attempts at retrieval have been unsuccessful after practically exhausting the endoscopic tools available.




[2510] **Figure 1.** 1a: CT scan without contrast on admission Figure 1b: CT scan without contrast post-surgery. Figure 2a: Foreign body in gastric body Figure 2b: Foreign body with ulcers in gastric body.

**S2511**

**Loose Screws: Removal of Foreign Bodies From the Lower Gastrointestinal Tract**

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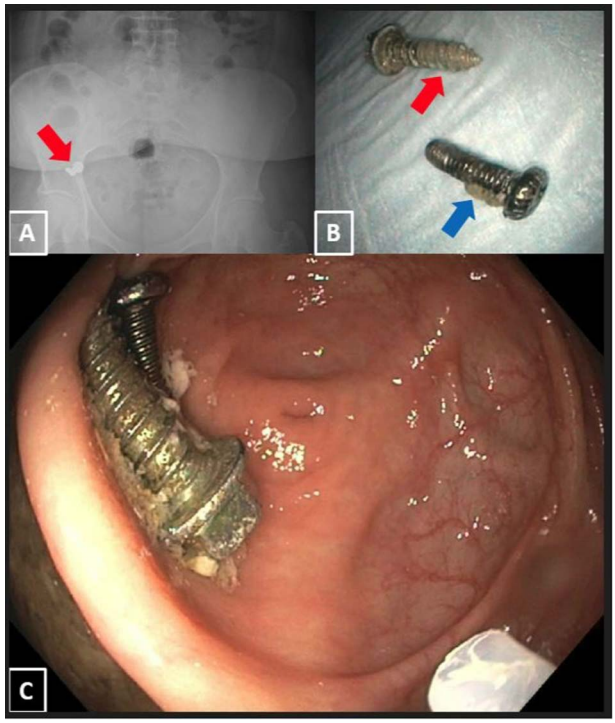
**Introduction:** The American Society for Gastrointestinal Endoscopy and European Society of Gastrointestinal Endoscopy have both released guidelines on the management of ingested foreign bodies in the upper GI tract, but neither has addressed how to approach foreign bodies that are in the lower gastrointestinal (GI) tract. This is perhaps due to the high likelihood of foreign body passage ( >80%) without any interventions, especially once the foreign body is in the lower GI.
**Case Description/Methods:** A 45-year-old female with a significant psychiatric history presented with a chief complaint of abdominal pain and nausea. She reports visiting her Rastafarian, who made her a "Tack Shake" to help with her symptoms of anxiety and depression. Physical exam was overall unremarkable, and she was persistently hemodynamically stable and afebrile. Laboratory findings were also unremarkable and demonstrated no evidence of other toxic ingestions or infection. Initial imaging showed 4 nails in the colon and 2 adjacent screws in the small bowel, and she was admitted for serial abdominal imaging and monitoring. After 5 days and 8 liters of bowel preparation, the patient had experienced passage of all nails, but the screws had not changed position (Figure A). As this presentation raised concern for failure to pass the ileocecal valve, an ileocolonoscopy was performed 6 days after the initial ingestion of foreign bodies. The screws were located in the cecum and appeared to be intertwined, although subsequent manipulation separated them (Figure C). One screw was removed utilizing a Roth Retrieval Net, while the second screw was removed using a cold snare (Figure B). During retraction, care was taken to orient the sharp end of the screws away from the colonoscope, in an attempt to minimize the risk of injury and perforation. The patient had an uncomplicated postprocedural recovery and was discharged to an inpatient psychiatric facility.

**Discussion:** This case increases the body of evidence needed to formulate guidelines on the appropriate timeline and indications for endoscopic removal of ingested foreign bodies in the lower GI tract. It also demonstrates 2 successful separate methods for removal of foreign objects from the lower GI tract. Additionally, it highlights a potentially new complementary and alternative medicine practice not previously reported in the literature: a drink/mixture called a "Tack Shake," which contains screws and nails and is purported to assist with anxiety.




[2511] **Figure 1.** 1A: Plain radiograph depicting persistent foreign objects 1B: Removed screws with blunt (blue arrow) and sharp (red arrow) tips 1C: Positioning of the screws in the cecum.

**S2512**

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**Multiple Synchronous Gastrointestinal Stromal Tumors: Mediastinal and Gastric**

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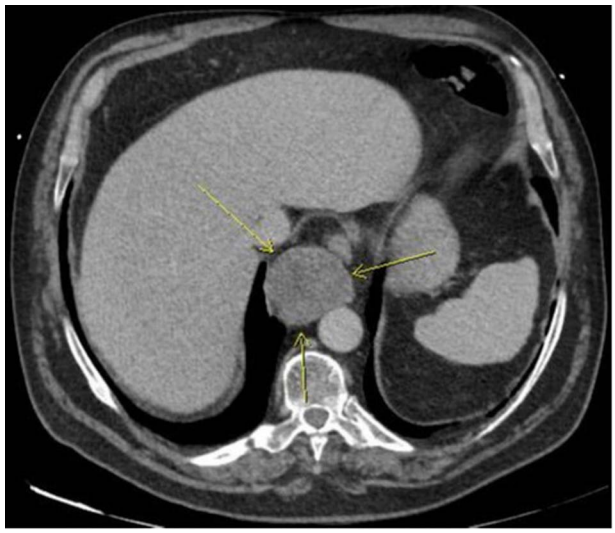
**Introduction:** Gastrointestinal stromal tumors (GISTs) make up the more common of the 2 groups of mesenchymal GI neoplasms, typically of the subepithelium. GISTs are unique as they are thought to arise from the pacemaker cells of the GI tract, the interstitial cells of Cajal, that give them CD34 positivity and are typically associated with mutation of KIT gene, identified via CD117 positivity. GISTs typically occur in the stomach (40-60%) or jejunum/ileum (25-30%) but can occur anywhere in the gastrointestinal tract, including mediastinum and accessory structures like mesentery, peritoneum or omentum.
**Case Description/Methods:** A 62-year-old male with a past medical history of type 2 diabetes mellitus presented to the emergency department for abdominal pain. He had one day of epigastric pain radiating to his back, worse with oral intake. He endorsed a 10-pound unintentional weight loss over the previous 2 months. Computed Tomography (CT) of the abdomen with intravenous (IV) contrast demonstrated inflammation in the porta hepatis with accompanying porta lymphadenopathy and a large paraesophageal, retrocrural mass (Figure). Esophagogastroduodenoscopy (EGD) with endoscopic ultrasound (EUS) was performed revealing 5.6 cm paraesophageal mass with central anechoic degeneration and a 1.8 cm fourth layer gastric cardia subepithelial lesion, both suspicious for GIST. Both were biopsied and were CD34 and CD117 positive, consistent with GISTs. The patient was later discharged home with GI follow but was lost to follow up.
**Discussion:** Although GISTs are the most common type of stromal tumor in the GI tract, they only make up approximately 1% of primary GI malignancies. GISTs have an incidence of 0.68 cases per 100,000 population. Mediastinal GISTs are more unique, with only twelve cases reported. Furthermore, multiple synchronous GISTs have only been reported in 3 other cases when not associated with GIST syndromes. CT is the imaging modality of choice for GISTs. GISTs usually appear as smooth, solid masses easily seen with CT scan. EGD is especially useful for diagnosing GISTs if located in the esophagus, stomach or duodenum appearing as smooth, submucosal masses with possible central ulceration and/or protrusion into the lumen. On EUS, GISTs appear hypoechoic, homogenous with clear margins arising most commonly from the fourth layer (muscularis propria) and less likely from the second layer (muscularis mucosa). GIST management often consists of surgery and/or imatinib depending on the characteristics of the GIST.



[2512] **Figure 1.** Transverse view computed tomography of the abdomen demonstrating paraesophageal gastrointestinal stromal tumor in the mediastinum.

**S2513**

**Not All That Appears Like Oral Thrush Is Candidiasis: A Case of Oral Actinomycosis**

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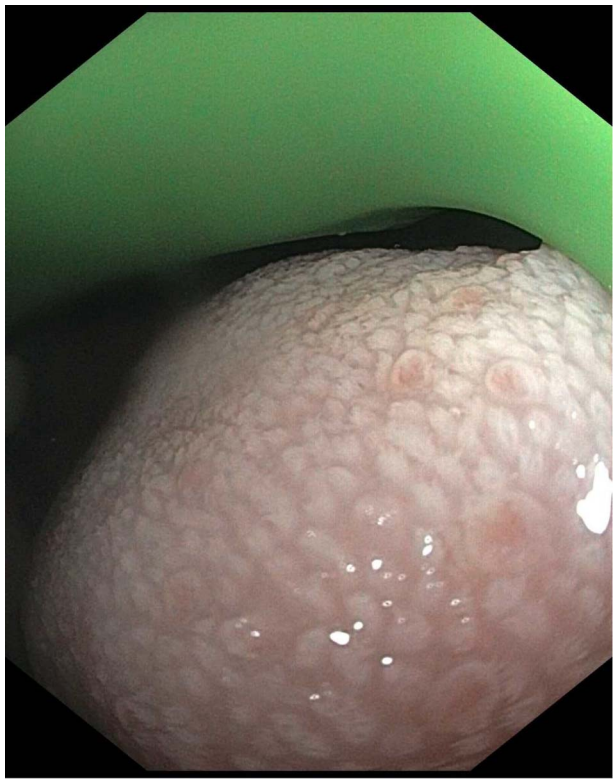
**Introduction:** Oral thrush is one of the most common infections that affect the oral cavity. The oral candidiasis lesions classically present as whitish confluent plaques on oral cavity examination of tongue, palate, and buccal mucosa. Oral thrush has long been associated with overgrowth of Candida albicans in the mouth in dialysis dependent patients, diabetics, immunocompromised, and who are on corticosteroid therapy. In this abstract we present a case of oral actinomycosis mimicking oral thrush upon presentation.
**Case Description/Methods:** 51-year-old female a history of squamous cell esophageal cancer status post distal esophagectomy and gastric pull through operation 5 years ago. She with a presented with a chief complaint of odynophagia and recurrent episodes of dysphagia for solid foods that has been worsening since past few weeks. About 6 months ago the patient had an upper endoscopy, which was significant for acute gastritis and pyloric stenosis, which was dilated. No obvious thrush was identified during that visit. Patient was not on any immunosuppressive medication. A repeat upper endoscopy was performed at this visit which was revealing of thrush in the oropharyngeal area and chronic gastritis. Tongue brushings were obtained for cytopathological analysis, which were suggestive of actinomyces infection. The patient was started on amoxicillin therapy for 2 weeks, which resulted in resolution of thrush as well as her clinical symptoms. Other treatment alternatives for actinomycosis include doxycycline, clindamycin, ceftriaxone, and imipenem (Figure).
**Discussion:** Actinomyces is a rare bacterium often found in the head and neck region. They are partially acid-fast, filamentous gram-positive microorganisms. They were originally classified with fungi organisms as they possess hyphae. Actinomyces is considered to cause chronic suppurative infection. However, actinomycosis infection of the tongue is rare, it usually presents as an abscess formation, draining sinus tracts, fistulae, and tissue fibrosis. The gold standard for the diagnosis of Actinomyces is histological examination. Identification of the underlying pathogen is crucial for management of these patients. Candida is treated with antifungals, however, for actinomyces the treatment is penicillin group of antibiotics. Therefore, it is important to have awareness for actinomyces in the setting of oral thrush for appropriate treatment.



[2513] **Figure 1.** Endoscopic Findings.

**S2514**

**More Than Skin Deep: Kaposi Sarcoma of the Gastrointestinal Tract**

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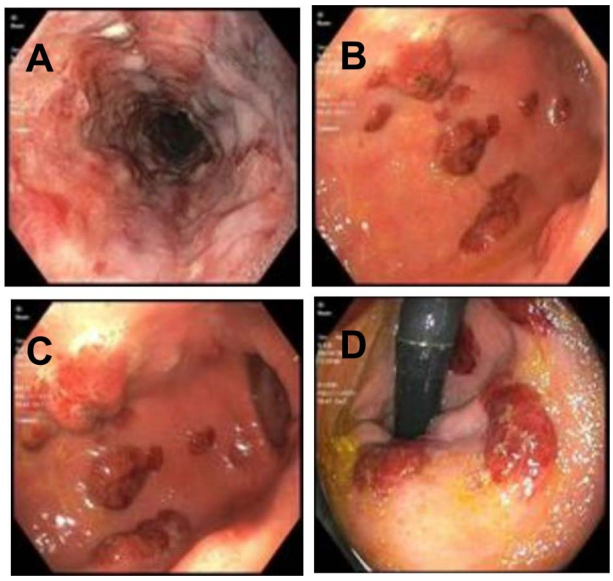
**Introduction:** Although gastrointestinal Kaposi sarcoma is usually asymptomatic, some patients present with abdominal pain and warrant endoscopic evaluation. Here, we report a case presentation of 40-year-old male with no past medical history who presented with a 1-year history of dysphagia, abdominal pain, and decreased oral intake.
**Case Description/Methods:** Social history was significant for unprotected sexual intercourse. Physical exam was significant right upper quadrant abdominal pain and diffuse painless, purple, maculopapular skin lesions. Initial labs included: WBC 5.2 K/cumm, Hg 12.9 g/dL, MCV 82.5 fL, platelet 220 K/cumm, BUN 16 mg/dL, Cr 0.8 mg/dL, AST 18 U/L, ALT 19 U/L, alkaline phosphatase 87 U/L, total bilirubin 0.3 mg/dL, albumin 3.6 g/dL. Other labs included: positive HIV-1 antibody, HIV-1 RNA 343K copies/mL, CD4 count of 38/cumm. Positive syphilis with 1:1 titer. Chlamydia, gonorrhea, and acute viral hepatitis panel were negative. CT abdomen and pelvis showed thickening of the ascending colon and prominent omental, mesenteric, inguinal lymphadenopathy. On EGD, grade D esophagitis and numerous violaceous lesions seen throughout the gastric antrum, fundus, and duodenal bulb. On colonoscopy, several vascular lesions ranging in size from 2-10 cm from rectum to terminal ileum. Cecal and ascending colon biopsies confirmed Kaposi sarcoma with immunohistochemical stains positive for human herpes virus 8. He was started on highly active antiretroviral therapy (HAART) with emtricitabine, tenofovir, and dolutegravir for HIV as well as atovaquone and azithromycin for prophylaxis after consultation with infectious disease. He was also started on pantoprazole for grade D esophagitis and treated with penicillin G for late latent syphilis. He was discharged and referred to medical oncology. On follow-up, patient has completed multiple cycles of paclitaxel. Dysphagia and abdominal pain have improved (Figure).
**Discussion:** It is important to recognize the utility of endoscopy when patients present with symptomatic gastrointestinal manifestations of Kaposi sarcoma. Visceral involvement of Kaposi sarcoma is associated with poor prognosis. Treatment is usually palliative and aimed at improving symptoms and preventing progression. Depending upon severity and disease burden, HAART is the first-line therapy. Antiretrovirals may decrease proportion of new lesions, promote regression of existing lesions, and improve survival with or without chemotherapy.




[2514] **Figure 1.** (A) Severe grade D esophagitis. (B-C) Numerous purple lesions throughout gastric antrum. (D) Retroflexion with additional violaceous lesions at the gastric fundus.

**S2515**

**Chicken Bone Causing Gastric Perforation With Liver Penetration: Successfully Managed With Endoscopic Removal and Clipping**

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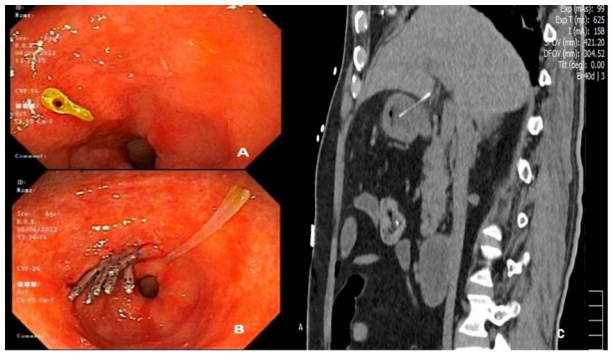
**Introduction:** Perforation of gastric wall by a chicken bone has rarely been reported. Most foreign bodies (FB) pass through the gastrointestinal tract uneventfully within one week. However, we report a case of chicken bone-induced gastric perforation managed by endoscopic retrieval and closure with clips.
**Case Description/Methods:** A 70-year-old-man presented with sharp epigastric abdominal pain with nausea and vomiting 4 hours after eating chicken wings. He was hemodynamically stable. The abdomen was soft and non-tender, with normoactive bowel sounds on examination. CT scan revealed a small pneumoperitoneum in the upper abdomen and a 3.4 cm linear radiopaque density within the lumen of the gastric antrum and partially outside directed posteriorly, abutting the left hepatic lobe. An EGD revealed a 3.5 cm long, narrow bone with a hollow center protruding through the antral wall along the lesser curvature. One endoclip was placed adjacent to the FB for easier identification of the location of the perforation. The FB was then extracted from the wall with rat tooth forceps. The gastric wall defect was then closed with 6 endoclips in a zipper-like fashion. Finally, the FB was retrieved with rat tooth forceps by allowing the sharp end to trail with the withdrawal of the gastroscope. Post-procedure, the patient was started on a clear liquid diet 48 hours later, which he tolerated well, and was discharged on post-procedure day 4 (Figure).
**Discussion:** Accidental foreign body (FB) ingestion is common. Most ingested FB pass spontaneously, with only 10%-20% requiring non-operative intervention. Serious complications include impaction or perforation. A foreign body that perforates the bowel wall may have a wide spectrum of clinical manifestations, from acute generalized peritonitis to chronic abscess formation. Patients with foreign bodies in the stomach, duodenum, and large intestine rarely have perforation due to thick walls and may have delayed presentation. We report the second case of endoscopic retrieval of a chicken bone causing gastric perforation managed endoscopically. Endoclips are often used as a hemostatic tool or to close endoscopic mucosal resection sites. Endoscopic intervention is more likely to be successful in cases of FB ingestion in the absence of peritonitis. In this case, the endoscopic removal of FB resulted in successful non-surgical management of gastric perforation. Placement of initial endoclip prior to bone removal allowed successful closure despite loss of gas after FB removal.




[2515] **Figure 1.** A-Upper Endoscopy shows a sharp hollow chicken bone in gastric antrum. B-Upper Endoscopy showing removal of chicken bone followed by endoscopic clipping. C- CT Abdomen/Pelvis without contrast shows a foreign body perforating the gastric wall and penetrating the liver.

**S2516**

**Peppered in Appearance: A Rare Case of Pseudomelanosis of the Upper Gastrointestinal Tract**

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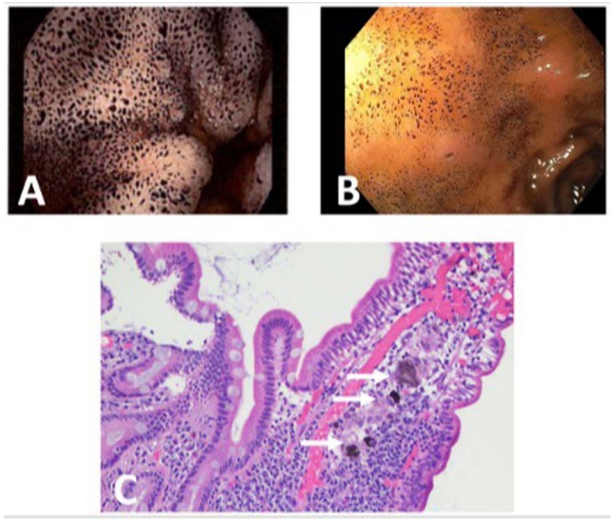
**Introduction:** Pseudomelanosis of the upper gastrointestinal tract (GI) is a rare condition characterized by a diffuse black-brown speckled pigmentation within the intestinal mucosa. Usually identified incidentally on endoscopy, upper intestinal pseudomelanosis is more frequently seen in the duodenum, but can rarely also be seen in the gastric body and jejunum. While this condition has been reported in literature, pathogenesis and clinical course are still largely unknown. Here we describe a case of pseudomelanosis in a patient referred for iron deficiency anemia.

**Case Description/Methods:** An 80-year-old woman with a history notable for hypertension, type 2 diabetes, chronic kidney disease, coronary artery disease, gastroesophageal reflux disease, hypothyroidism, and celiac disease was referred for evaluation of iron deficiency anemia. On review, she had undergone screening colonoscopy 2 years ago, notable for scattered diverticulosis, and upper endoscopy 5 years ago notable only for mild gastritis. Medications included: aspirin, hydralazine, metoprolol, losartan, atorvastatin, pantoprazole, ferrous sulfate, and insulin glargine. She initially underwent video capsule endoscopy, revealing mild non-erosive gastropathy and scattered black pigmentation in the duodenum (Figure 1A). She subsequently underwent upper endoscopy demonstrating scattered pigmentation in the gastric antrum, duodenum, and proximal jejunum (Figure 1B). Duodenal biopsies revealed pigment laden macrophages within the mucosa consistent with pseudomelanosis duodeni (Figure 1C).
**Discussion:** Pseudomelanosis of the upper intestinal tract is a rare and poorly understood condition. While considered benign, it has been associated with various conditions including hypertension, diabetes mellites, chronic kidney disease, gastrointestinal bleeding, and with medications including oral iron supplements and diuretics—many of which were seen in this case. Unlike colonic pseudomelanosis, which is histologically characterized by accumulation of lipofuscin within the colonic mucosa and is associated with laxative use, pseudomelanosis of the upper intestinal tract is histologically distinct, characterized by accumulation of ferrous sulfate containing compounds. To date, pathogenesis of the condition remains unclear. Given the rarity of upper intestinal pseudomelanosis, prognosis and treatment have also yet to be determined. This reports aims to increase awareness of this rare and incompletely understood condition.




[2516] **Figure 1.** (A) Capsule endoscopy demonstrating scattered black-brown pigmentation within the duodenal mucosa. (B) Endoscopic view of the duodenal bulb with scattered foci of pigmentation. (C) Duodenal biopsy demonstrating pigment laden macrophages (white arrows) consistent with pseudomelanosis.

**S2517**

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**Pill Prep Problems? Erosive Gastritis and Peptic Ulcers due to Sodium Sulfate-Based Tablet Bowel Prep**

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**Introduction:** SUTAB is the second ever tablet formulation for bowel preparation. It became FDA approved in 2020 based on 2 randomized studies in which SUTAB provided noninferior bowel cleansing when compared to other commonly used FDA approved preparations. These randomized studies focused only on colonoscopies, possibly ignoring SUTAB's effects on the upper GI tract. Endoscopists have recently been noticing significant adverse side effects from SUTAB, notably erosive gastritis and peptic ulcers in patients scheduled for same day bidirectional endoscopy. These findings were not noted by the studies used for FDA approval. SUTAB is a sodium sulfate based tablet composed of 3 main active ingredients: sodium sulfate, magnesium sulfate, and potassium chloride. SUTAB is taken as a 2-day split dose regimen of 24 tablets with large volumes of water. The most common adverse side effects published for SUTAB include nausea, abdominal distension, vomiting, and abdominal discomfort. Any mention of mucosal ulcerations focused on colonic ulcerations in patients with suspected inflammatory bowel disease or in concurrence with the use of stimulant laxatives.
**Case Description/Methods:** 5 cases of same day bidirectional endoscopy were examined from 9/2/2021 to 11/15/2021. Indications for upper endoscopy included a history of Barrett's esophagus and/or gastroesophageal acid reflux disease and all patients used SUTAB as the sole bowel preparation. In all cases there was no previous history of erosive gastritis or peptic ulcer disease, however, all upper endoscopies noted signs of moderate to severe erosive gastritis. Two of the cases also noted similarly appearing linear gastric ulcers with black eschars. All patients were subsequently placed on antacid therapy and follow-up endoscopies were performed to assess ulcer healing, which was noted at the time.
**Discussion:** SUTAB is a relatively new bowel preparation on the market with very little published data on its known side effects. The convenience of a tablet formulation for a bowel preparation that achieves successful bowel cleansing attracts patients and doctors, but it comes with the cost of possible adverse side effects in the upper GI tract. The ingredients in SUTAB are corrosive agents, specifically, potassium chloride has been linked to erosions that are found in the mucosa of the GI tract with prolonged exposure. To better understand this recently noted phenomena, more research is needed to help prevent these unwanted and potentially dangerous side effects.

**S2518**

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**Rare Gastrointestinal Manifestations of Metastatic Breast Cancer**

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**Introduction:** Breast cancer is the most common cancer and a leading cause of mortality among women. Breast cancer commonly metastasizes to the lung, liver, bones, and adrenal glands. However, there are rare instances where breast cancer can metastasize to the GI tract, most commonly the stomach. We present a case of a 65-year-old woman diagnosed with breast cancer in 1997 and found to have metastases to the stomach and cecum 19 and 21 years later, respectively.
**Case Description/Methods:** A 65-year-old female with a past medical history of infiltrating lobular breast carcinoma (ER-positive) status post resection and chemotherapy and PUD presented 19 years later with refractory nausea. EGD showed localized moderate inflammation characterized by congestion, erythema, and friability in the stomach. Pathology (IHC staining) revealed tumor cells that were ER- and CAM5.2-positive and PR-negative. These findings were consistent with metastatic carcinoma with a breast primary. The patient had a subsequent PET scan that was positive for metastasis to the bone, spine, and pelvis and was restarted on hormonal-based chemotherapy. Two years later the patient presented with nausea, vomiting, and loss of appetite. CT of the abdomen with contrast showed a new finding of a 1.2 cm metastasis to the cecum. Colonoscopy showed altered vascular, atrophic, ulcerated mucosa in the cecum and thickening of mucosal folds in the proximal ascending colon. Pathology (IHC staining) revealed neoplastic cells positive for GATA-3 and negative for CDX-2, which support the diagnosis of infiltrating carcinoma from breast primary. The patient was continued on several different lines of chemotherapy.
**Discussion:** Metastatic disease of the breast to the GI tract is a relatively rare presentation. It can be difficult to detect due to recognition of GI symptoms that are attributed to chemotherapy or a primary GI disease/malignancy. This can lead to delays in diagnosis and treatment. Due to this patient's initial nausea, an EGD was conducted which revealed gastritis. Biopsies were positive for metastatic carcinoma in the stomach consistent with a primary breast cancer. Subsequent colonoscopy revealed metastatic disease to the cecum and ascending colon. Previously reported cases have shown that metastatic breast cancer to the GI tract can be diagnosed up to 30 years later. This highlights the importance of including primary breast cancer metastasis in the differential of ambiguous gastrointestinal symptoms.

S2519

**Post-Transplant Lymphoproliferative Disorder Presenting as Iron Deficiency Anemia: A Case Report**

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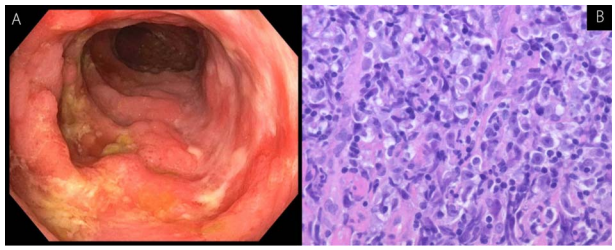
**Introduction:** Post- Transplant Lymphoproliferative Disease (PTLD) is a rare but well-known complication of transplant recipients on immunosuppression. Even less common is monomorphic B cell type PTLD in a patient negative for the Epstein Barr Virus. Its presentation is highly variable and associated with poor outcomes with later detection.
**Case Description/Methods:** A 29-year-old male presented with nausea, vomiting, fatigue, and 10-pound unintentional weight loss over several weeks. He had a past medical history of congenital solitary kidney, Focal Segmental Glomerular Sclerosis and underwent deceased donor renal transplant 13 years ago. He had remained on chronic immunosuppression (Mycophenolate, tacrolimus, prednisone). His exam was notable only for conjunctival pallor. Labs revealed WBC 10.8 (15% lymphocytes), anemia (Hgb 5.7), iron saturation 6% and thrombocytosis (platelets 1154). Contrast enhanced CT Abdomen and Pelvis revealed necrotic mesenteric lymphadenopathy and thickened small bowel loops in the left hemiabdomen. Upper endoscopy was normal. Colonoscopy exposed an ulcerated lesion with nodular mucosa in the terminal ileum (Figure 1A). Biopsy revealed monomorphic PTLD Diffuse Large B Cell Lymphoma (DLBCL) with CD20 and CD30 expression (Figure 1B). EBV and CMV staining were negative. Further workup with bone marrow biopsy showed no evidence of leukemia or lymphoma. FISH showed trisomy of chromosome 8, 14, and 18, but no bcl2-IgH fusion translocation or C-MYC translocation. He was treated with weekly Rituximab/Ruxience biosimilar followed by the CHOP chemotherapy regimen. However, surveillance imaging showed increase in size of lymph nodes. He continued to deteriorate over the next 3 months and died thereafter.
**Discussion:** Transplant recipients are vulnerable to PTLD, with greater than 85% of cases of PTLD occurring in the first-year post-transplant. In our case, we described an aggressive course of B cell type monomorphic PTLD 13 years post-transplant in an EBV seronegative patient on chronic immunosuppressants. Most patients are treated with Rituximab and/or CHOP chemotherapy, however, patients with solid organ transplants often do not tolerate such aggressive treatment. New treatments with low toxicity are needed to further improve outcomes for patients with PTLD. Our case also highlights the importance of adequate small bowel evaluation with terminal ileum intubation during colonoscopy and further advanced imaging if necessary for workup of iron deficiency anemia.




[2519] **Figure 1.** A. ulcerated lesion with nodular mucosa in the terminal ileum B. histology slide showing monomorphic PTLD Diffuse Large B Cell Lymphoma (DLBCL).

S2520

**Severe Nausea and Hyperemesis Are Not Always Gastrointestinal: Non-GI Immune-Related Adverse Events Presenting as Upper GI Symptoms**

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**Introduction:** Cancer therapy using immune checkpoint inhibitors (ICI) are increasingly utilized in the treatment of various types of advanced cancers, such as renal cell carcinoma (RCC), lung cancer and melanoma. While the mechanism of action in using the body's innate immune system to fight cancer have been positive, there are documented cases of autoimmune side effects called immune-related adverse events (irAE). We present a patient on ICI that was thought initially to be having GI irAE but was, in fact, something else.
**Case Description/Methods:** A 61-year-old male with metastatic RCC status post right nephrectomy on Nivolumab and Ipilimumab was admitted due to 2-months of progressive generalized weakness and persistent nausea and vomiting. On presentation, vital signs demonstrated he was hypotensive at 88/66 mmHg and tachycardic at 102 bpm. Physical exam, laboratory parameters (CBC, CMP) and abdominal CT were unremarkable. The GI service was consulted to place a percutaneous endoscopic gastrostomy (PEG) for palliative feeding, but given the patient was on ICI therapy further evaluation was needed. A TSH (21.3mcIU/mL) was elevated and AM cortisol (< 0.5mcg/mL) was low. Hydrocortisone and levothyroxine therapy were immediately instituted. Thyroid and adrenal antibodies were negative and there was no serologic evidence of pituitary dysfunction consistent with an endocrine irAE. An esophagogastroduodenoscopy (EGD) was performed to evaluate for GI irAE, which was unremarkable. His symptoms resolved and he was able to eat with no issues. The patient ultimately did not need a PEG.
**Discussion:** Programmed cell death-1 (PD-1) and cytotoxic T-lymphocyte antigen 4 (CTLA-4) are proteins primarily involved in suppressing the immune reaction to self-antigens, resulting in immunologic tolerance. Unfortunately, neoplastic cells use the same protein activity to evade the body's immune response. Nivolumab and Ipilimumab selectively inhibit (PD-1) and (CTLA-4), respectively, and lead to an increase in baseline T-cell specific immune response against tumor cells. This process can also result in autoimmune responses to various systems in the body, including gastrointestinal, endocrine, hepatic, pulmonary, dermatologic, and renal systems. With the increased utility of ICI in treating various malignancies, it is of the upmost importance to recognize different irAE to avoid delay in starting lifesaving treatments and to avoid unnecessary procedures like this particular case.

S2521

**Sevelamer: An Underreported Cause of Enteritis and Colitis**

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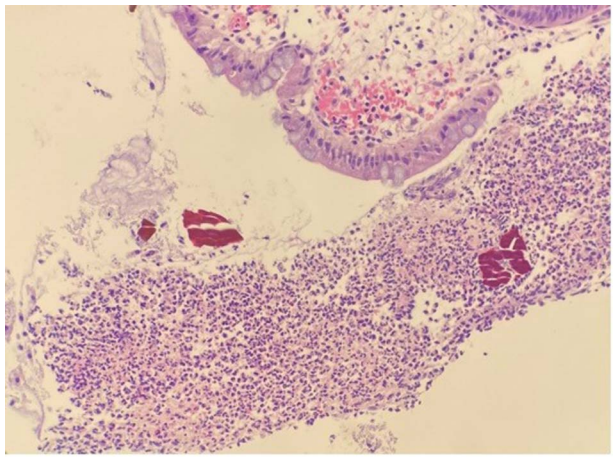
**Introduction:** Hyperphosphatemia is a common metabolic derangement in patients with end-stage renal disease (ESRD). In addition to dietary constraints, phosphate binders are needed to treat hyperphosphatemia. Sevelamer is the most used resin-based phosphate binder and can crystallize to form concretions. Crystals formed by other resin-based binders such as polystyrene sulfonate (kayexalate) are a well-known cause of gastrointestinal (GI) mucosal injury. However, sevelamer is an underreported culprit. Here, we describe a case of ileitis and colitis caused by sevelamer crystals.
**Case Description/Methods:** A 65-year-old male veteran with ESRD, secondary hyperparathyroidism treated with sevelamer, and cocaine abuse was admitted for abdominal pain and bloody diarrhea for 3 days. CT scan of the abdomen and pelvis revealed inflammation of the ileum and rectosigmoid. He was started on ciprofloxacin and metronidazole. Stool studies were negative. A colonoscopy revealed an ulcerated and erythematous terminal ileum (TI) between 15cm and 7cm proximal to the ileocecal valve. There was mild ulceration and erythema in the ascending colon. The worst ulceration was between 40cm to 30cm from the anal verge with additional ulceration seen in the rectosigmoid. The colonic mucosa between the ulcerations were normal appearing. Biopsies taken from the TI, ascending colon, and descending colon revealed chronic ileitis and colitis with ulceration, granulation tissue associated crystals, crypt abscess, and fibrinopurulent debris and negative for infection or malignancy. A review the medication reconciliation confirmed no exposure to kayexalate and, thus, concluded that sevelamer was the cause. The patient was advised to stop taking sevelamer. A follow up colonoscopy 6 months later revealed complete mucosal healing (Figure).
**Discussion:** GI mucosal injury is a rare and underrecognized adverse effect of sevelamer. A limited number of reported cases describe a range of symptoms such as nausea, vomiting, constipation, dysentery, and acute abdomen requiring surgery. The enteritis and colitis caused by sevelamer can be overlooked and presumed to be infectious or ischemic etiology. The pathology can also be missed because of misidentification or failure to identify crystals on histology. Increased awareness of this underreported complication of sevelamer is important for these reasons and for directing appropriate therapy with cessation of this medication in the setting of enteritis or colitis.



[2521] **Figure 1.** H&E stain showing fibrinopurulent exudate (ischemic /acute erosive pattern colitis) and the presence of Sevelamer crystals.

**S2522**

**Spur of the Moment Removal: A Rare Complication of Esophageal Manometry**

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**Introduction:** Esophageal manometry is a safe procedure that analyzes the contraction pressure of the esophagus. The well-known risks are patient discomfort, nasal trauma, and retching. To our knowledge and search of present literature, there has not been a reported incident of esophageal manometry probe becoming lodged in the nasopharynx. Here, we present a case of manometry probe meeting resistance upon attempted extubation, ultimately requiring endoscopic removal.
**Case Description/Methods:** A 53-year-old female with a history of GERD and hiatal hernia presented for routine esophageal manometry as part of anti-reflux surgery workup. The manometry probe was inserted into the right naris and advanced without difficulty. The procedure was performed without complications. Upon extubation, the probe met resistance. Multiple providers attempted extubation, all unsuccessful. The patient experienced increasing discomfort, and efforts to extubate were immediately stopped. A small amount of blood was noted in the right nares, and intranasal lidocaine 2% gel was administered. Planned EGD was then performed with manometry probe in place. Blood was seen in the posterior oropharynx, and manometry probe was visualized passing through the nares and seen in the oropharynx. The gastroscope was then advanced into the stomach where the end of the probe was visible. Removal of the probe was unable to be performed even under sedation, and ultimately the probe had to be sacrificed. The end of the manometry probe was grasped using a snare and the distal end was removed through the mouth. Due to the large connectors, the proximal end of the manometry probe exiting through the naris was cut, and the distal end of the probe was pulled through the mouth without resistance. EGD procedure was then completed. The patient was subsequently evaluated by ENT for nasal obstruction and was found to have a septal deviation with a very large right posterior septal spur that was contacting the lateral nasal wall on nasal endoscopy. We suspect that the spur acted similarly to a one-way valve; allowing easy insertion of the probe and difficulty with removal given the positional anatomy of the spur.
**Discussion:** In conclusion, we report a novel adverse event from routine manometry procedure leading to traumatic removal and ultimately sacrificing the manometry probe due to a large posterior nasal spur.

**S2523**

**Stuck Scope: Failed Endoscopic Withdrawal After Hemospray**

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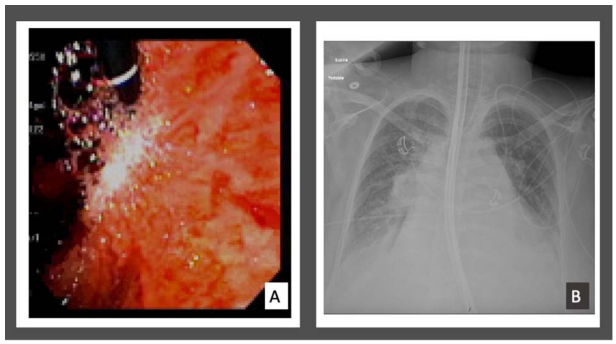
**Introduction:** Hemostatic spray is a non-contact endoscopic tool that utilizes inert bentonite powder to achieve hemostasis in an acute gastrointestinal hemorrhage. To date, reports of adverse effects following this treatment have been rare. We report an unusual complication of a failed endoscope withdrawal after use of hemostatic spray.
**Case Description/Methods:** A 43-year-old female, with past medical history of significant alcohol use, presented to an outside ED with large volume hematemesis. She was started on a proton pump inhibitor and octreotide infusion and admitted to the ICU for a suspected upper GI bleed. She underwent an EGD, which showed large amount of bright red blood in the cardia and gastric fundus, alongside a large clot. Active bleeding followed clot removal attempt. The hemospray was applied in a retroflex direction, aimed at the blushing beneath the clot in the fundus. After multiple sprays, hemostasis was achieved. However, the endoscope could not be withdrawn after and adhered at the level of the GE junction by the spray coating. Patient was subsequently intubated for airway protection. Attempts to retroflex and spray water, to dilute the hemospray were limited by scope mobility. Attempts to torque, withdraw or advance the scope resulted in increased bleeding. An attempt to insert another scope alongside the previous one was made, however there was limited space. Due to risk of esophageal perforation with repeat attempt of endoscope removal, patient was transferred to a tertiary care hospital. The GI team was able to remove the scope with scope traction and only encountered mild resistance– 6 hours after the sentinel EGD. Endoscopy was repeated in 48 hours to find source of initial bleeding, however only portal hypertensive gastropathy was encountered with no active bleeding (Figure).
**Discussion:** Only one such case of a retained endoscope after hemospray use has been reported, where the scope was safely removed under direct endoscopic visualization after 48 hours –the time given to allow for complete elimination of powder from upper GI tract. In our case, the strong adherence between the mucosal surface and surface of endoscope by the bentonite powder likely inhibited endoscope movement, leading to failure of withdrawal of the scope. The slow elimination of hemospray after a few hours likely made scope removal easier. Our case not only reports this rare potential complication of hemospray, but also highlights that safe removal may be possible after 6 hours per-oral without endoscopic visualization.




[2523] **Figure 1.** A - Endoscope position at gastric fundus B - Chest X-ray post-intubation, showing retained scope.

**S2524**

**Successful Retrograde Deep Distal Small Bowel Enteroscopy Using a Novel Device**

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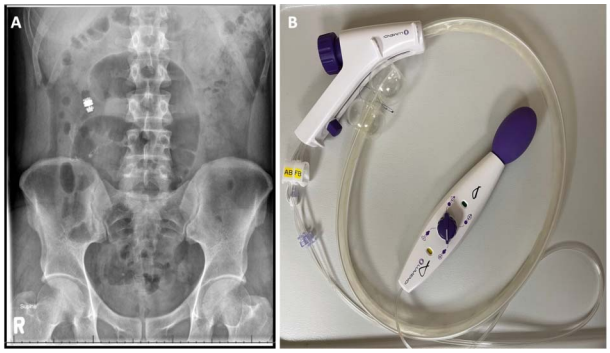
**Introduction:** Small bowel evaluation is challenging due to its length and contractility. Several techniques have been developed including video capsule endoscopy, push enteroscopy, device-assisted enteroscopy, and intraoperative enteroscopy, all with limitations. We report the first case of retrograde enteroscopy utilizing the DiLumen device (Lumendi Ltd, Westport, CT) attached to a standard colonoscope.
**Case Description/Methods:** A 39-year-old male with history of Meckel's diverticulectomy, presented with chronic intermittent abdominal pain accompanied by nausea and vomiting. Physical exam and laboratory data were unremarkable. CT abdomen/pelvis showed post-surgical changes from Meckel's diverticulectomy, mild thickening of ileum proximal to the anastomosis with inflammatory mesenteric changes suggestive for inflammatory bowel disease (IBD) but no obvious strictures. EGD and Ileocolonoscopy were performed without endoscopic or pathologic evidence of IBD. A video capsule endoscopy (VCE) was performed with capsule retention noted. Patient's symptoms remained at baseline. Endoscopic capsule retrieval was attempted with retrograde balloon enteroscopy using a standard colonoscope, with assistance of the DiLumen device. The anastomotic site was found at about 80 – 100cm proximal to the ileocecal valve with a tight stricture present. A through-the-scope balloon dilation was performed up to 10 mm with adequate dilation effect. Unfortunately, the colonoscope was unable to pass the stricture. Biopsies of the stricture showed no evidence of IBD. Patient was referred to colorectal surgery for further management (Figure).
**Discussion:** Balloon assisted enteroscopy (BAE) requires expertise given the prolonged procedure time and technical device management aspects. Retrograde BAE is more challenging compared to the anterograde approach due to colonic navigation using an enteroscope. DiLumen is an endoscopic accessory sheath consisting of 2 balloons that can be manually inflated and deflated as needed. The balloons can facilitate endoscope navigation by shortening and straightening the colon similar to double BAE. A larger colonoscope, rather than an enteroscope, can then be used to perform the procedure. In our case, with a colonoscope, an ileal anastomotic stricture was reached and dilation performed successfully. This opens the potential for retrograde deep ileal intubation, especially in those with a tortuous or redundant colon, where an enteroscope may have difficulty reaching that location.




[2524] **Figure 1.** A: Abdominal plain film showed retained video capsule device in the small bowel; B: Dilumen double balloon device.

**S2525**

**Successful Surgery-Sparing Endoscopic Removal of an ICV Polyp With the Use of Dilumen Device**

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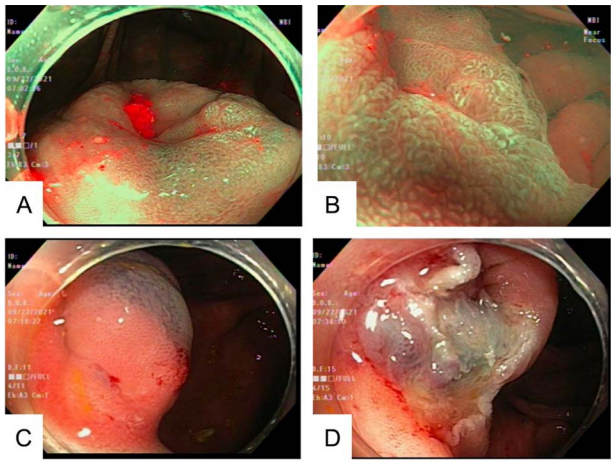
**Introduction:** Ileocecal valve (ICV) polyps are technically challenging to remove endoscopically and reoccur with a rate of 18.6%. We report on a case of an ileocecal valve polyp that underwent multiple failed attempts of endoscopic removal. It was successfully removed using the Dilumen device.
**Case Description/Methods:** A 72-year-old White male with known past medical history of CAD, paroxysmal atrial fibrillation not on anticoagulation, referred for completion polypectomy of a 10mm flat ileocecal valve (ICV) polyp after 2 failed colonoscopy removal attempts seen by referring gastroenterologist due to colon tortuosity and polyp location. Pathology of the partially removed polyp was adenoma with high grade dysplasia (HGD). Vitals, physical exam and laboratory data were unremarkable. Patient refused surgical management and prefers endoscopic treatment, if possible. He agreed to a third colonoscopy and, given history of failed attempts, we utilized the Dilumen device to assist with the endoscopic procedure. The device provided stability in positioning the colonoscope that allowed complete removal of the residual ICV polyp via en bloc endoscopic mucosal resection (EMR). The edges were treated with snare tip coagulation. Pathology came back as adenoma with HGD with clear margins. Patient is doing well post-procedure with no issues (Figure).
**Discussion:** ICV polyps usually pose a great challenge for EMR due to its location and tendency to extend into the terminal ileum and around the valve orifice. This makes it technically difficult to completely resect the lesion and poses a high risk for recurrence. In fact, a study showed that only 76.3% of the large polyp (size >20mm), can be resected endoscopically in the ICV versus 91.3% resection rate in non-ICV area. Frequently, surgical referral would be the alternative option but more complications can occur after invasive surgery. In order to avoid surgery, a few novel techniques have been developed. Dilumen is an endoscopic accessory sheath consisting of 2 balloons that can be manually inflated and deflated as needed. The balloons can facilitate endoscope navigation by shortening and straightening the colon similar to double balloon assisted enteroscopy. This improves access for the endoscope to maneuver in between and remove the polyp. In our patient, it helped greatly in removing the difficult ICV polyp and the patient was able to avoid undergoing surgery.




[2525] **Figure 1.** A - Narrow Band Imaging (NBI) of the ICV polyp B - Near focus evaluation of the ICV polyp C - Submucosal lifting of the ICV polyp D - Post EMR of the ICV polyp.

**S2526**

**Should Endoscopists Look for an Inguinal Hernia Before Beginning a Colonoscopy?**

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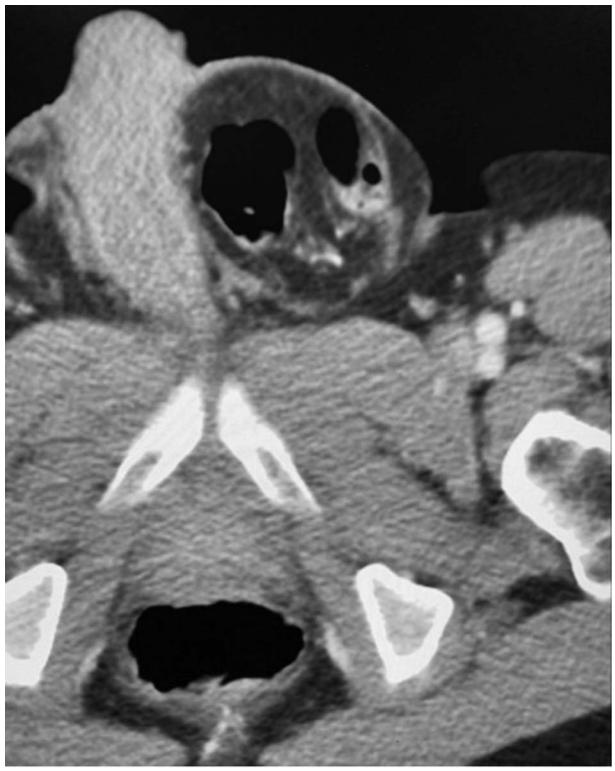
**Introduction:** Colonoscopes ca potentially become incarcerated in inguinal hernia sacs during routine colonoscopies. The entrapment of colonoscopes may occur when patients present with unknown inguinal hernias. Entrapment can occur during the insertion or withdrawal of the scope. We report a case of a screening colonoscopy resulting in entrapment of a colonoscope in a left inguinal hernia, which was managed by applying pressure to the hernia site.
**Case Description/Methods:** AH is a 66-year-old, white male who presented to the ambulatory surgical center for a screening colonoscopy. During the procedure, the scope repeatedly ended up in the blind end. It was evident the patient had a left inguinal hernia, and the scope was entering the hernia sac (Figure). Subsequently, the endoscopist withdrew the scope to prevent scope entrapment in the hernia sac, while the assistant put pressure on the hernia site under the patient's gown to reduce the hernia sac. The colonoscopy was reattempted, while the assistant continued to push on the hernia site, and the procedure was completed without further difficulties.
**Discussion:** There have been 2 previous case reports of scope entrapment in a patient's hernia sac resulting in the scope being removed using either surgery, fluoroscopy, or the pulley method.[1, 2] It is best to avoid performing a colonoscopy for patients with an irreducible hernia. For a reducible hernia, applying pressure on the hernia site may ensure that the scope does not enter the hernia sac. If a patient has an unknown hernia in an open-access colonoscopy, then the endoscopist may not be prepared for a hernia until they start the procedure. Gastroenterologists should be aware of the consequences of patients presenting with unknown inguinal hernias, especially if they are irreducible, to avoid potential complications and emergent operations. Physicians performing colonoscopies should be cognizant of the potential risk to patients with large, irreducible inguinal hernias. Larger studies are needed to definitively mandate this recommendation.



[2526] **Figure 1.** CT of the abdomen revealing left inguinal hernia.

**REFERENCES**
1. Tan VP, Lee YT, Poon JT. Incarceration of a colonoscope in an inguinal hernia: Case report and literature review. World J Gastrointest Endosc 201316;5(6):304-7.
2. Torrealba M, Matlock R, Petrun B, Zera R, Malli A. Incarcerated Colonoscope in a Left Inguinal Hernia During Diagnostic Colonoscopy. ACG Case Rep J 2021;8(4):e00564.

**S2527**

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**The Xiphisternum as a Gastric Subepithelial Lesion**

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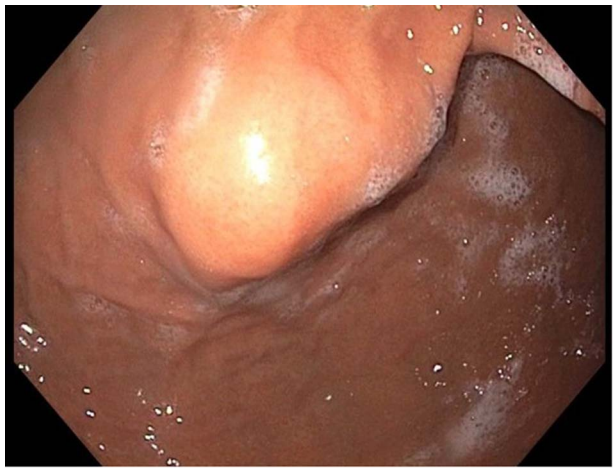
**Introduction:** The xiphisternum, comprised of cartilage surrounding a core of bone, is located inferior to the sternal body and enlarges with age. The process is usually directed anteriorly relative to the sternal body and the abdominal cavity. However, in approximately 10% of individuals the bony structure is angulated more than 10 degrees posterior to the orientation of the sternal body. Although this anatomical variant is typically asymptomatic, it may be encountered during endoscopy. As such, endoscopists should be familiar with its appearance to ensure correct identification.
**Case Description/Methods:** A 66-year-old man presented to an outpatient gastroenterology clinic for assessment of diarrhea and bloating. He had a background medical history significant for colon adenocarcinoma with prior right hemicolectomy, prostate cancer, and B Cell lymphoma. During diagnostic EGD, an incidental medium-sized gastric subepithelial lesion was identified (Figure 1). Biopsy of the lesion demonstrated gastric mucosa with foveolar hyperplasia and minimal chronic inflammation. Endoscopic ultrasound was obtained for further characterization. The lesion was visualized causing indentation on the anterior wall of the gastric body. No mucosal abnormalities were present. With respiration, the stomach "rolled over" this lesion, suggesting that the origin was extramural. Endosonography demonstrated a hyperechoic, multilayered, shadowing lesion external to the stomach. Palpation of the epigastrium resulted in indentation of the stomach just below the lesion. A review of sagittal sections of an abdominal CT, performed for unrelated reasons, clarified the underlying etiology for the lesion identified during endoscopy.
**Discussion:** The CT image demonstrates a posteriorly directed xiphisternum adjacent to the gastric body, causing protrusion of the gastric wall into the stomach which appeared as a subepithelial lesion during endoscopy. Upon visualizing this structure for this patient specifically, it was important to consider lymphoma, or compressive lymphadenopathy, as a differential diagnosis. However, with an intraprocedural examination and information available at the time of the EGD, it is likely that the endoscopic ultrasound could have been avoided. This was likely an incidental finding and not related to his presenting diarrhea and so no intervention was pursued.



[2527] **Figure 1.** Subepithelial gastric lesion identified during endoscopy.

**S2528**

**Think "Inside the Loop:" A Novel Method for Repositioning a Migrated G-J Tube**

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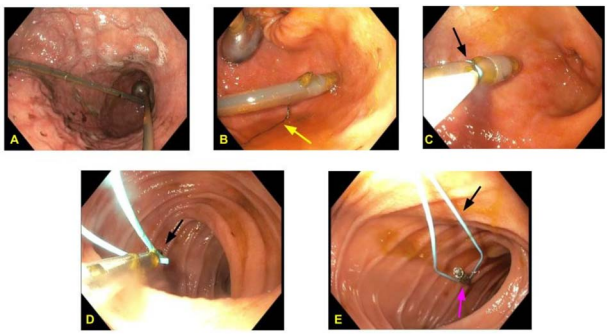
**Introduction:** Gastrojejunostomy tube (G-J tube) migration is common in patients requiring long term percutaneous enteral feeding. These patients often present with forceful vomiting and decreased PO intake. This case discusses a method for repositioning a migrated G-J tube using an endoloop (detachable snare device) and Resolution Clips.
**Case Description/Methods:** A 26-year-old female with a history of cystic fibrosis (CF), chronic malnutrition requiring enteral feeds, CF-related liver disease and variceal bleeding presented with 5 days of abdominal pain and coffee ground emesis. Initially, the patient was tachycardic and vomiting intermittently. Bright red blood was visible in the emesis bag. Physical exam was notable for epigastric tenderness without peritoneal signs. The skin around the G-J tube insertion site was non-tender with no signs of infection. CT of the abdomen revealed gastrosplenic varices and appropriate positioning of the enteral tube. There was no evidence of GI perforation or obstruction. An EGD demonstrated a patent gastrostomy tube in the gastric body. The jejunal tail was coiled up against the inflamed lumen of the stomach. The suture at the tip of the tail had degraded, dislodging the tube from its original position along the jejunal wall. There were blood clots in the body of the stomach but no evidence of active bleeding. Given the suspicion that the migrated feeding tube was the cause of the patient's symptoms, a decision was made to reposition it. An endoloop was tied around the tip of the G-J tube to serve as a substitute for the suture. A cold snare was then used to secure the endoloop and drag the tail into the jejunum. The endoloop was secured to the intestinal wall using 2 Resolution Clips. The patient's symptoms resolved after the EGD and she was discharged in stable condition (Figure).
**Discussion:** Migration of a G-J tube is commonly associated with severe motility disorders, such as cystic fibrosis. Oftentimes, the tube will loop inside the stomach or continue toward the esophagus. The tube itself can cause gastric outlet obstruction, leading to gastric distention, nausea and vomiting. These patients are an aspiration risk and should be initially managed with IV hydration, antiemetics, and stopping of tube feeds. In addition, it is important to assess for complications such as sepsis, hemorrhage, buried bumper, or perforation of the GI tract. If the feeding tube is patent, it is reasonable to have it repositioned based on clinical judgment and provider expertise.




[2528] **Figure 1.** Upper Endoscopy: A. Intact gastrostomy with a patent G-J tube present in the gastric body. The jejunal tail is coiled up in the lumen of the stomach. B. The tip of the G-J tube with surrounding trauma to the body of the stomach, characterized by edema, erythema, and inflammation. The thread at the tip of the G-J tube had degraded (yellow arrow). C. An endoloop was tied at the neck of the tip of the G-J tube (black arrow) to serve as a substitute for the suture. D. A cold snare was used to secure the endoloop (black arrow) and drag the tip of the G-J tube into the jejunum. E. The endoloop (black arrow) attached to the tip of the G-J tube was secured to the wall of the jejunum using a Resolution Clip (pink arrow).

**S2529**

**Threading the Needle: Safely Removing Double-Edged Sharp Foreign Bodies**

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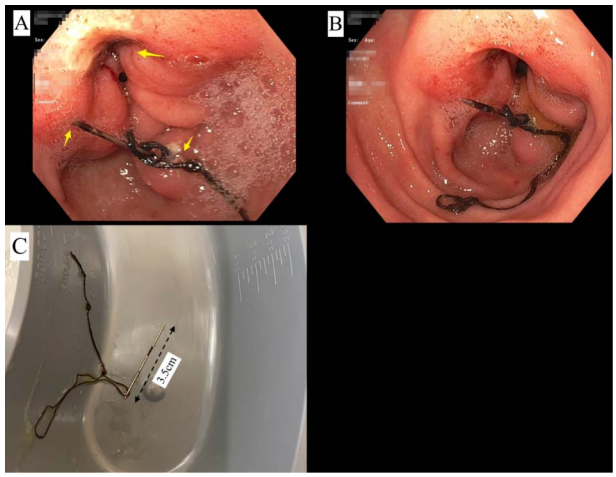
**Introduction:** Foreign body ingestion is a common phenomenon among individuals with psychiatric disorders, alcohol intoxication, developmental delay, or in incarcerated individuals. Most ingested foreign bodies pass spontaneously without complication but 10-20% require endoscopic intervention. Impaction, perforation, or obstruction often occurs at angulations or narrowing. Early recognition and urgent upper endoscopy (EGD) for foreign body removal may improve clinical outcomes. Here we present a case of melena and abdominal pain secondary to foreign body ingestion.
**Case Description/Methods:** An 83-year-old male with a past medical history of pulmonary embolism on warfarin, alcohol use disorder, and anxiety presented after 2 black, tarry stools and light-headedness. On admission his vitals were stable and labs were notable for an International normalized ratio (INR) of 3.3 and a Hemoglobin (Hgb) of 12.3g/dL. He was treated with Vitamin K and intravenous pantoprazole twice daily. The next day his Hgb was found to be 8.2g/dL and an EGD was performed revealing antral ulcerations and a 3.5cm sewing needle and thread with both ends penetrating opposite walls of the antrum (Figure A, B). Multiple attempts at removing the needle with rat tooth and jumbo forceps were unsuccessful and the procedure was aborted. Follow up computed tomography imaging did not reveal perigastric

fluid or pneumoperitoneum post-procedurally. On hospital day 3 a repeat EGD was attempted and showed the needle was only perforating mucosa along one side of the gastric body; it was able to be removed via overtube and raptor device (Figure C).

**Discussion:** Endoscopic removal of sharp ingested foreign bodies can prove to be challenging. Several risk factors including age and duration of impaction are associated with adverse events, such as laceration, perforation, and ulcers. Double pointed/sharp objects are particularly challenging given they are harder to grasp and maneuver. That, in addition to luminal contractions, likely contributed to the difficulty retrieving the needle in this case despite using recommended tools. Had the second endoscopic retrieval attempt failed, the patient would have required surgical evaluation. Overall, the benefit of further endoscopic retrieval attempts versus the risk of perforation from the foreign body or complications from the procedure itself must be weighed case by case.




[2529] **Figure 1.** Endoscopic Evaluation of Patient with Foreign Body A – Foreign body wedged between opposite ends of pylorus (Highlighted by top arrow) B – Full view of sewing needle with thread in gastric antrum C – Sewing needle after removal.

**S2530**

**Utility of Hemostatic Powder Spray for Management of Recently Placed Percutaneous Gastrostomy Tube Tract Bleeding**

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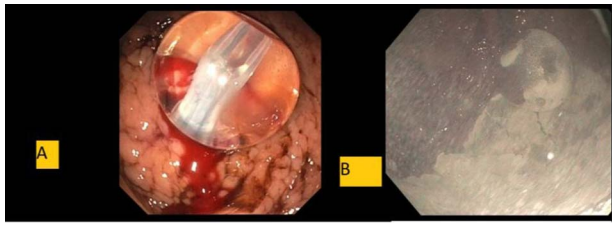
**Introduction:** Hemodynamically significant bleeding is an uncommon adverse event following percutaneous gastrostomy (PG) tube placement. The usual approach to managing bleeding involves conservative measures, interventional radiology guided interventions and surgery in rare cases. Endoscopic management of bleeding related to recently placed PG tube traditionally has been limited. Here we describe the use of hemostatic powder for management of overt PG tube tract bleeding not responsive to conservative management.

**Case Description/Methods:** 76-year-old male with a history of squamous cell carcinoma of the right posterolateral tongue presented to the hospital for curative surgical management. A percutaneous gastrostomy (PG) tube was placed by interventional radiology for nutritional support following surgery. Following PG tube placement, the patient developed frank hematemesis, melena, and bloody output from the PG tube. Laboratory data showed worsening anemia with a rapid decrease in hemoglobin from 7.4 g/dL to 4.4 g/dL.. PG tube traction was performed at bedside and patient was transferred to the intensive care unit due to hemodynamic instability and packed red blood cells transfusion was initiated. An emergent upper endoscopy was performed showing active bleeding from the recently placed PG tube tract despite applying PG tube balloon traction (Figure 1A). PG tube balloon traction was discontinued and hemostatic powder spray was applied to the opening of PG tube tract (Figure 1B). After application of hemostatic powder spray, no further bleeding was seen, and balloon traction was reapplied. Patient did well after the procedure and the PG tube was able to be used after 48 hours.

**Discussion:** Most bleeding related to PG tube placement is limited and can be controlled by simple pressure over the abdominal wound or tightening the bumper against the abdominal wall to compress the gastrostomy tract. If these measures fail patient may require interventional radiology guided intervention or surgery, since endoscopic intervention is limited due to our inability to access the bleeding source within the PG tube tract without removing the tube. Hemostatic powder spray allows us to deliver bentonite to the bleeding within the tract itself by absorbing water and creating a barrier that leads to mechanical tamponade and concentration of clotting factors, resulting in enhanced coagulation. In addition, using hemostatic powder spray allows us to salvage the PG tube.




[2530] **Figure 1.** (A)- Active PG tube tract bleeding despite balloon traction **Figure** 1(B)- Stopped PG tract bleeding after application of hemostatic powder spray.

**S2531**

**When the Potassium Is Low, Should You Look for a Colon Mass?**

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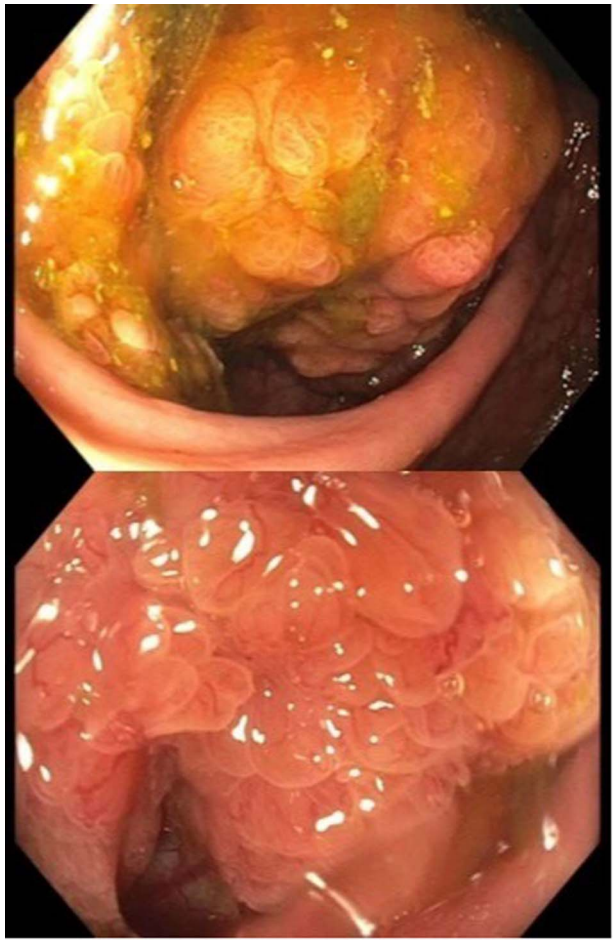
**Introduction:** Mckittrick-Wheelock syndrome is a rare condition that presents with severe electrolyte disturbances including severe hyponatremia, hypokalemia and renal failure in the setting of distal colorectal tumors. The most common tumors are typically benign villous adenomas, and the most commonly reported site of involvement is the rectum. The tumors are typically large and low in the rectum making them difficult to remove endoscopically. Diagnosis is clinical including laboratory tests, Computed tomography (CT) abdomen and pelvis with contrast and colonoscopy.

**Case Description/Methods:** 64-year-old female presented with acute onset of altered mental status and diarrhea. She was hemodynamically stable. Physical exam was notable for tenderness to palpation diffusely over her abdomen and she appeared ill. Her white blood cell count was 25.7 K/MM$^3$, hemoglobin 16.7 g/dL with an MCV of 75 fL, and platelets 406 K/ MM$^3$. Her chemistry panel showed a sodium 121 mmol/L, potassium 2.0 mmol/L, Chloride 108 mmol/L, bicarbonate 7 mmol/L, serum creatinine 12.12 mg/dL, BUN 173 mg/dL, calcium 8.3 mg/dl, albumin 4.0 g/dL, protein 7.4 g/dL, aspartate aminotransferase 41 U/L, alanine aminotransferase 25, alkaline phosphatase 83 U/L, bilirubin total 0.9 mg/dL. CT abdomen and pelvis without contrast showed an 8.5 x 6.1 cm fungating rectal mass approximately 7 cm from the anal verge. Flexible sigmoidoscopy showed a large polypoid rectal mass approximately 2-thirds of the circumference of the proximal rectum (Figure). Pathology was consistent with a tubular adenoma without

high grade dysplasia or malignancy. The rectal mass could not be resected endoscopically. She required hemodialysis for acute on chronic renal failure. She then underwent laparoscopic hand-assisted low anterior resection with colorectal anastomosis. Final pathology was consistent with a large, villous adenoma with focal high-grade dysplasia, no invasion, thirteen benign lymph nodes, and margins negative for dysplasia.

**Discussion:** Mckittrick-Wheelock syndrome is a rare presentation of large villous adenomas or adenocarcinomas most commonly seen in the rectum. It presents as secretory diarrhea, acute renal failure, hyponatremia, hypokalemia and hypoproteinemia. Prognosis is good if renal function can be recovered quicily and if the tumor is resected. However there is typically a delay in diagnosis due to its rarity.



[2531] **Figure 1.** Flexible Sigmoidoscopy demonstrates a large polypoid rectal mass encompassing approximately 2-thirds of the circumference of the proximal rectum.

**S2532**

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**Unusual Presentation of a Large Duodenal Lipoma**

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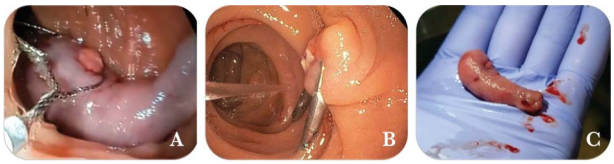
**Introduction:** Duodenal lipomas are rare benign tumors that are usually asymptomatic. This is the case of a patient who presented with symptomatic anemia secondary to upper GI bleeding due to a large duodenal lipoma.

**Case Description/Methods:** A 72 y/o male presented with weakness, dyspnea and melena for 2 days. His last colonoscopy showed 2 lipomas in the ascending and transverse colon. Rectal exam showed dark stool. Hemoglobin was 8.2 g/dL. BUN was 12.8 mg/dL, creatinine 0.7 mg/dL and an occult blood test was negative. Upper endoscopy showed a large, ulcerated and pedunculated duodenal polyp in the second portion, measuring approximately 4 cm. Two hemoclips were placed at the base and hot snare polypectomy was used to remove the polyp. After removal, 2 hemoclips were placed for adequate hemostasis. Histopathology showed submucosal lipomatosis, prominent vessels and an overlying duodenal erosion. Immunostain for MDM2 was negative, favoring a benign lipoma. His admission was uneventful with resolution of melena. On 6-month follow-up, the patient denied further bleeding episodes, with stable hemoglobin at 13.5 g/dL (Figure).

**Discussion:** Lipomas are benign tumors found in the subcutaneous tissue. They rarely occur in the gastrointestinal (GI) tract, with an estimated prevalence of 4%. When present, they are generally found in the colon. Duodenal lipomas are rare, with few reported cases found in literature. Most arise from the submucosa and may be identified as a low-density lesion with the same radiodensity as fat on CT scan or as a hyperechoic mass arising from the submucosa in EUS. They are frequently asymptomatic, and in most cases discovered incidentally. If asymptomatic, observation is typically recommended. Symptoms may include early satiety, gastric outlet obstruction, pain, and intussusception. Hemorrhagic duodenal lipomas are an even rarer occurrence, with severe bleeding usually being caused by an overlying mucosal erosion or ulceration. Some reports suggest that mucosal pressure atrophy may lead to ulcer formation due to necrosis of the underlying mucosa. When symptomatic, management includes endoscopic or surgical resection. Management is dictated by operator experience, tumor size and location. Endoscopic options include wound closure with clips and use of snare polypectomy. Complications may include perforation and delayed bleeding. Duodenal lipomas may present with nonspecific and vague symptoms. For this reason, it is important to consider this in the differential diagnosis of obscure GI bleeding.




[2532] **Figure 1.** A. Snare polypectomy of duodenal lipoma B. Post-polypectomy site C. Resected large pedunculated duodenal polyp measuring 4 cm with distal ulceration.

**S2533**

**An Unusual Finding Lurking in the Colon**

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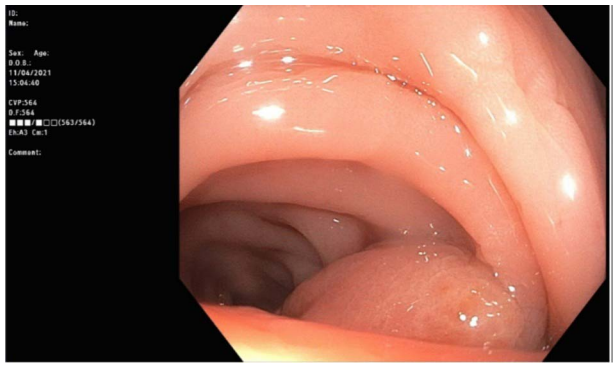
*University of Tennessee Health Science Center, Memphis, TN.*

**Introduction:** Deep Invasive gastrointestinal endometriosis (DIGIE) is rare. Out of all endometriosis cases, 7-12% of cases can have gastrointestinal involvement, ranging from single lesions, to multiple lesions, to full colonic stenosis. Symptoms can include dyspareunia, dysmenorrhea, infertility, dysphasia, diarrhea, constipation, bloating, and rectal bleeding. Here we present a patient who was incidentally found to have endometrioma.
**Case Description/Methods:** A 24-year-old female with a past medical history significant for polycystic ovarian syndrome, HIV and endometriosis, presented to the hospital with chief complaints of fever, dysuria, hematochezia, and lower abdominal pain which had been ongoing for 3-4 days. On admission, the patient was noted to be hemodynamically stable. Abnormal laboratory results included hematocrit 32.6% (reference range 36-46%), hemoglobin 10.2 g/dL (reference range 12-16 g/dL), mean corpuscular volume 72.4 fL (reference range 80-100 fL), ferritin 15.8 ng/mL (reference range 6.3-137 ng/mL), total iron binding capacity 306 mcg/dL (reference range 265-497 mcg/dL), and iron 19 mcg/dL (reference range 37-170 mcg/dL). The following day, she underwent a colonoscopy which showed isolated diverticulosis involving the sigmoid colon. There was an active oozing diverticulum that was successfully treated with endoscopic clip placement. At the rectosigmoid junction, there was a 3 cm submucosal lesion bulging into the lumen. This appeared to have a smooth surface and was without ulceration. Biopsies were taken and the pathology showed submucosal granulation tissue with chronic inflammatory reaction and iron deposition. CD10 specific immunostains were positive, suggesting the diagnosis of an endometrioma (Figure).
**Discussion:** This case highlights an incidental finding of DIGIE. It is important to think about colonic involvement in patients with endometriosis who have GI complaints. Rarely, these lesions can cause obstruction and may require surgery. There are currently no formal guidelines or recommendations, with most of these patients being managed on a personalized basis. Management for these patients includes symptomatic therapies, progresterones, colonic shaving, disc excision, and laparoscopic resection. As for our patient, she pursued conservative symptomatic management with close outpatient Gynecology follow up.




[2533] **Figure 1.** A 3 cm submucosal lesion bulging into the lumen at the rectosigmoid junction.

**S2534**

**Late-Onset Juvenile Polyposis and Hereditary Hemorrhagic Telangiectasia Overlap Syndrome**

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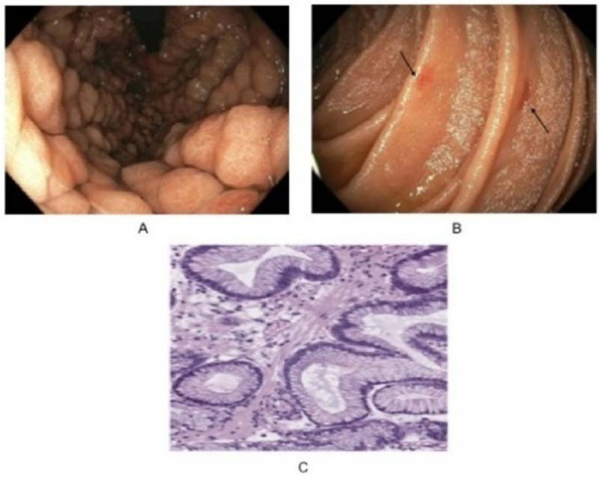
**Introduction:** Juvenile Polyposis Syndrome (JPS) and Hereditary Hemorrhagic Telangiectasia (HHT) are rare disorders inherited in an autosomal dominant manner. The SMAD4 gene mutation causes a combined syndrome of JPS and HHT. We report a case of late-onset JPS-HHT overlap syndrome.
**Case Description/Methods:** A 72-year-old man presented with orthostasis and melena for 3 days. He had a lifetime history of recurrent epistaxis. Family history revealed 2 daughters with a history of gastrointestinal (GI) bleeding due to arteriovenous malformations (AVMs) and had required surgical gastrectomies for polyposis due to SMAD4 gene mutation. Physical exam revealed mild tachycardia. Rectal exam showed melena. There were no mucocutaneous telangiectasias. Hemoglobin (Hb) level was 4.4 g/dl and he had an elevated BUN to creatinine ratio. He was resuscitated and transfused. Upper endoscopy revealed multiple non-bleeding semi-sessile polyps in the stomach (Figure 1A). Colonoscopy was unremarkable and CT angiography showed no active bleeding. Capsule endoscopy a week later displayed numerous small bowel angiectasias that were subsequently treated with argon plasma coagulation (Figure 1B). Gastric biopsies showed foveolar hyperplasia and edema of the lamina propria consistent with inflammatory polyp of JPS (Figure 1C). He met the diagnostic criteria for the JPS-HHT overlap syndrome. He declined genetic testing for SMAD4 mutation. Follow-up included oral iron supplementation, and Hb level monitoring. Screening for GI malignancy as well as pulmonary and central nervous system AVMs was recommended.
**Discussion:** JPS often comes to clinical attention by age 20 years with most presenting with bleeding or anemia due to GI polyps. Individuals with JPS due to SMAD4 mutations often exhibit features of HHT [1] with GI bleeding related to HHT often presenting in the 4th decade of life. Our patient had delayed onset penetrance of the GI manifestations of both HHT and JPS with a first GI bleed at age 72 years. It is important for gastroenterologists to be aware of the varied age-related GI manifestations of the JPS-HHT overlap syndrome.




[2534] **Figure 1.** A. Upper GI endoscopy showing multiple non-bleeding semi-sessile polyps in the stomach. B. Small Bowel Enteroscopy showing angiodysplastic lesions in the small intestine. C. Gastric biopsy showing foveolar hyperplasia and edema of lamina propria, consistent with inflammatory polyp of JPS.

**REFERENCE**

1. Lin HC, Fiorino KN, Blick C, Anupindi SA. A rare presentation and diagnosis of juvenile polyposis syndrome and hereditary hemorrhagic telangiectasia overlap syndrome. Clin Imaging. 2015 Mar-Apr;39(2): 321-4.

**S2535**

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***Aeromonas hydrophila* Gastroenteritis Presenting With Profound Watery Diarrhea Following Esophagogastroduodenoscopy**

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**Introduction:** An inadequate reprocessing process, biofilm formation, or defects in the endoscope could lead to endoscopy-associated infections. Leading pathogens of such infections are *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Escherichia coli*, and *Salmonella enteritidis*. Here we report a rare case of *Aeromonas hydrophila* infection following an esophagogastroduodenoscopy (EGD) which manifested with cholera-like watery diarrhea.
**Case Description/Methods:** A 55-year-old female with a history of Roux-en-Y gastric bypass (RYGB) complicated by a marginal ulcer, gastroesophageal reflux disease, and type 2 diabetes mellitus presented with 10 days of abdominal cramping, nausea, and profound watery diarrhea. The patient had diarrhea 10-15 times a day, including incontinence overnight, was unable to tolerate an oral diet, and lost 5 kg during this time. One day before the onset of symptoms, the patient received an EGD for assessment of a known marginal ulcer at an outside institution which showed resolution of ulcer. She denied any recent travel, exposure to freshwater, known sick contacts, or consumption of seafood or raw food. Physical examination was significant for epigastric and left upper quadrant abdominal tenderness with a palpable mass in the left upper quadrant without peritoneal signs. Labs were significant for elevated aminotransferases (AST, 106 IU/L; ALT, 318 IU/L) and alkaline phosphatase (157 IU/L) which later decreased during the hospital course. CT abdomen revealed possible intussusception at the jejunojejunal anastomosis, while the upper GI series was negative for the intussusception on the second day of admission. Stool culture was positive for *Aeromonas hydrophila*. Symptoms significantly improved after symptomatic treatment with intravenous volume replacement and a 7-day course of ciprofloxacin administration for prolonged diarrhea.
**Discussion:** Given symptoms consistent with acute gastroenteritis, positive stool culture, negative history of environmental exposure to a pathogen, and recent EGD, despite an unconfirmed source of infection, we hypothesize that the patient had an *Aeromonas hydrophila* infection most likely from a contaminated endoscope. The patient is suspected to have had several potential risk factors for *Aeromonas hydrophila* infection, including proton pump inhibitor use and possibly atypical microbiome following RYGB. *Aeromonas hydrophila* infection could be prevented by properly washing and drying the endoscope channel to remove biofilm.

**S2536**

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**A Case of Gastric Volvulus: Examining a Complication of Paraesophageal Hernia**

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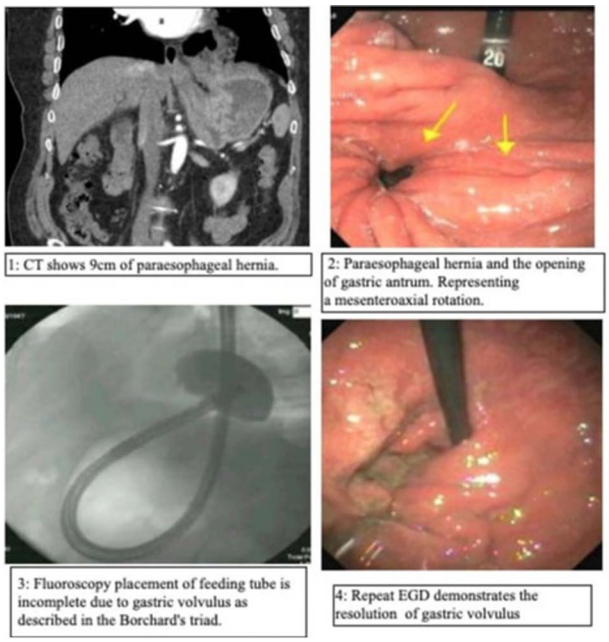
**Introduction:** Gastric volvulus (GV) is defined as rotation of the stomach beyond 180 degrees. This rotation can further leads to gastric outlet obstruction or strangulation. Without prompt intervention GV can progress to necrosis or stomach perforation. Overall case mortality of GV ranges from 30%-50%. Risk factors associated with GV are age above 50, diaphragmatic abnormalities, phrenic nerve paralysis, kyphoscoliosis, and other abdominal anatomic abnormalities. Symptoms of acute GV as described in Borchardt's triad often include severe epigastric pain, retching without vomiting and inability to pass a nasogastric tube. Borchardt's triad is seen in 70% of reported acute GV. Chronic GV can present with Borchard's triad but often more subtle and nonspecific symptoms, which makes diagnosis difficult. GV further classifies into organoaxial rotation and mesenteroaxial rotation. Mesenteroaxial rotation describes rotation of the antrum of the stomach above the gastroesophageal (GE) junction and is a less commonly seen form of GV. Esophagogastroduodenoscopy (EGD) offers an opportunity for nonsurgical intervention or temporary relief while confirming the anatomic rotations that are often not visualized on computer tomography (CT).
**Case Description/Methods:** A 73-year-old man with a history of gastroesophageal reflux disease (GERD) presented with 2 weeks of worsening abdominal pain, unintentional weight loss, early satiety, and dysphagia. Patient reported low intensity intermittent sharp abdominal pain in the epigastric region. He associated the pain with food intake and dysphagia with solids. He endorsed nausea but denied vomiting or hematemesis. He has no other significant medical history. His arrival vital signs and labs were all within normal range. CT angiogram of the abdomen revealed a left sided paraesophageal hernia. A subsequent EGD with attempted decompression was performed. Results showed opening of the antrum within the large paraesophageal hernia confirming mesenteroaxial GV. Subsequent day EGD confirmed the resolution of GV after detorsion attempt. Patient underwent surgical paraesophageal hernia repair prior to discharge. He returned to his usual state of health on a recent follow up visit (Figure).
**Discussion:** GV should be considered as a differential diagnosis for nonspecific abdominal pain. Complete resolution of GV may be achieved after prompt and early EGD. This case highlights that early EGD may prevent progression of acute complications and potentially decrease mortality.




[2536] **Figure 1.** Gastric volvulus on CT, EGD, and resolution.

**S2537**

**A Long and Dangerous Infection: Whipple's Disease Presenting With Concurrent Candida Esophagitis and *H. pylori* Gastritis**

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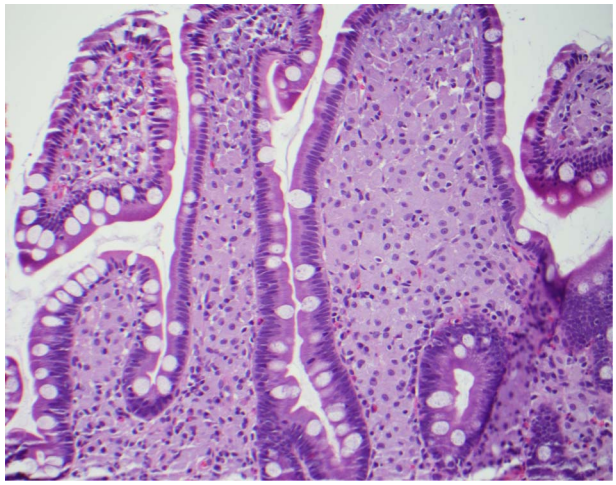
**Introduction:** Whipple's Disease (WD) is a rare condition which occurs from infection by *Tropheryma whipplei*. It is a debilitating illness and can lead to immunocompromise of the host. We present a case of a patient with WD who also had concomitant esophageal candidiasis and *H. pylori* gastritis.
**Case Description/Methods:** A 36-year-old male presented with a 3-month history of non-bloody diarrhea, nausea, migratory polyarthralgia, and weight-loss of 30 lbs. EGD and colonoscopy were performed. There were multiple, diminutive white plaques in the esophagus. Biopsies confirmed esophageal candidiasis. Gastric biopsies revealed *H. pylori* infection. The second portion of the duodenum displayed diffuse dilated lacteals with villous blunting. Similar findings were noted in the terminal ileum (TI). Biopsies of the duodenum, TI, and colon revealed foamy macrophage infiltration of the lamina propria that were intensely positive on PAS and PAS-D stains (Figure). Analysis for *Tropheryma whipplei* by DNA PCR was positive. He was started on ceftriaxone for WD with concurrent fluconazole for his esophageal candidiasis, which was presumed to be a complication of his immunocompromised state. Due to potential CNS and cardiac involvement, a lumbar puncture and a transthoracic echocardiogram were obtained, respectively, which were normal. After 2 weeks of receiving ceftriaxone, he was started on doxycycline and hydroxychloroquine with plans to continue treatment for at least one year with repeat endoscopy. His *H. pylori* infection was successfully treated with quadruple therapy. Three months after starting treatment, our patient had regained 20 lbs. and his other symptoms had completely resolved.
**Discussion:** A similar case of a patient with WD, concomitant esophageal candidiasis, and *H. pylori* gastritis has been reported in the literature. Both patients did not have specific risk factors for esophageal candidiasis, which supports a potential association between WD and esophageal candidiasis. The exact mechanism is unclear but may be related to the immunocompromised state of the host incurred from WD. The frequency of interval endoscopy is not defined in these patients and collaborating with infectious disease specialists to optimize follow-up is essential. Overall, WD is a rare condition that presents a diagnostic challenge, often resulting in an immunocompromised state with multisystem involvement. Long-term multidisciplinary follow-up is advisable after treatment with consideration for repeat endoscopic and histologic evaluation.



[2537] **Figure 1.** Foamy macrophages in the lamina propria, duodenum (20x).

S2538

**A Grate Lesson: Abdominal Pain Secondary to Ingested Wire Grill Brush**

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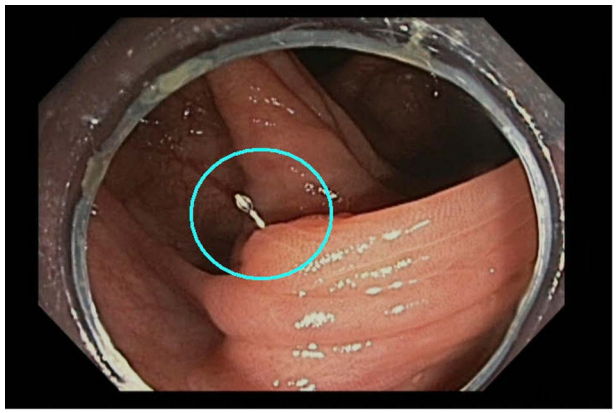
**Introduction:** An 18-year-old male presented with 3 days of right lower quadrant abdominal pain and 3 weeks of intermittent bright red blood per rectum. Pain was described as sharp 2/10 non-radiating pain at rest but 9/10 with movement. Review of systems was negative for diarrhea, constipation, nausea, emesis or any extra-intestinal symptoms. Patient denied any NSAID, alcohol or illicit drug use though did endorse a family history of Crohn's, Celiac disease and IBS in primary and secondary family members.
**Case Description/Methods:** Initial abdominal CT noted several nonspecific enlarged fluid-filled loops of small bowel within the right lower quadrant particularly at the terminal ileum. Upon evaluation with colonoscopy, a sharp metallic object was seen protruding about 1mm out of the cecal mucosa near the ileocecal valve (Figure). After several unsuccessful attempts at removing the object with biopsy forceps, a cold snare was looped around the end, closed, and the object was extracted through the working channel.
**Discussion:** Inspection revealed a sharp thin 1.5cm long metallic foreign body suspected to be a fragment of a wire brush used to clean barbeque grills. Retrospective analysis of CT imaging revealed the metallic object in question. Patient was symptom-free 4 days later with no recurrence of pain.



[2538] **Figure 1.** A sharp metallic object was seen protruding about 1mm out of the cecal mucosa near the ileocecal valve.

S2539

**A Large Inflammatory Pseudotumor of the Sigmoid Colon Causing Perforation**

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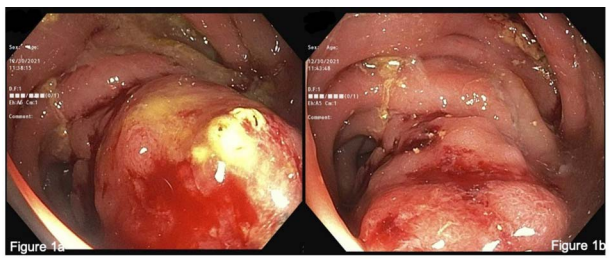
**Introduction:** Inflammatory pseudotumors (IPT) are rare non-malignant lesions. Lungs are the most common locations of origin. They remain extremely rare in the colon.
**Case Description/Methods:** An 80-year-old woman underwent colonoscopy for recent diverticulitis. Physical examination and laboratory workup were unremarkable. A 2-month prior Computer tomography (CT) scan revealed sigmoid diverticulosis with segmental wall thickening, suggestive of acute diverticulitis. Colonoscopy demonstrated a large, ulcerated, non-circumferential, nonobstructing, nonbleeding mass in the sigmoid colon (Figure). She developed bowel perforation and underwent exploratory laparotomy with sigmoid and left colectomy, and colostomy. Histopathology revealed numerous abscess sinuses within the colonic wall, extending from the diverticula. Extensive acute and chronic inflammation was also present. There was no evidence of dysplasia or malignancy.
**Discussion:** Inflammatory pseudotumors may have clinical and radiological features similar to malignancies, and may even recur after resection, however, malignant transformation is rare.




[2539] **Figure 1.** Colonoscopy demonstrating a large, ulcerated, non-circumferential, nonobstructing, nonbleeding mass in the sigmoid colon.

S2540

**A Rare Case of Metastatic Esophageal Granular Cell Tumor**

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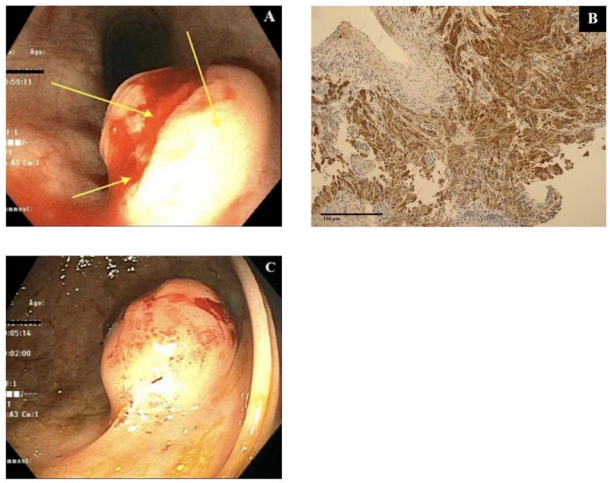
**Introduction:** Granular cell tumors (GCTs) are thought to originate from Schwann cells and rarely affect the gastrointestinal (GI) tract. Reported cases of GI involvement indicate the esophagus is most affected, while the colon is rarely involved. The majority of GCTs are benign, though approximately 1% can be malignant. We present a rare case of esophageal GCT with metastases to the lung and cecum.
**Case Description/Methods:** A 59-year-old female with history of colon polyps presented to our gastroenterology office for surveillance colonoscopy. She was incidentally found to have iron-deficiency anemia, and double endoscopy was arranged. Esophagogastroduodenoscopy found a 3 cm submucosal esophageal mass in the upper esophagus with multiple non-bleeding gastric angiodysplastic lesions, and one duodenal lesion, which were treated with argon plasma coagulation. A colonoscopy was aborted due to poor preparation. The esophageal mass biopsy found a granular cell tumor with S100 positivity. She was referred for endoscopic submucosal dissection (ESD) for her esophageal mass but was unfortunately lost to follow up. The patient underwent a series of imaging, including a PET-CT scan showing a 7 mm nodule in the left upper lung lobe with minimal metabolic activity. Three months later, a CT thorax without contrast showed enlargement of the left upper lobe nodule to 1 cm with multiple other small nodules noted. The patient underwent an endobronchial ultrasound with lung biopsy yielding granular cell tumor. Repeat colonoscopy ultimately found a 20 mm polyp in the cecum with pathology again identifying a

granular cell tumor. Repeat PET-CT scan re-demonstrated the mildly FDG avid left upper lobe nodule measuring 1.1 cm but also found a focal area of increased activity in the left anorectal wall. The patient began to experience significant dysphagia and was again referred for ESD which will be completed in the near future (Figure).

**Discussion:** Granular cell tumors may present localized or with metastases to various organs. The diagnosis of GCT is made via histopathology. In our case, an esophageal GCT was found with suspected spread to the lungs and cecum. Although most tumors are benign with a favorable prognosis, patients may develop symptoms such as dysphagia, cough, abdominal pain, nausea, or chest pain, especially when involving the GI tract. The definitive treatment is surgical or endoscopic removal, as chemotherapy and radiation are rarely effective. Long-term surveillance is essential as tumors may recur following resection.




[2540] **Figure 1.** Upper endoscopy imaging showing 3 cm submucosal mass in upper esophagus (A). Immunohistochemistry image showing positive S100 staining of esophageal mass (B). Colonoscopy imaging showing 20 mm polypoid lesion in the cecum (C).

**S2541**

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**A Rare Case of Erosive Gastritis and Melena Related to Gastric Mucosal Calcinosis**

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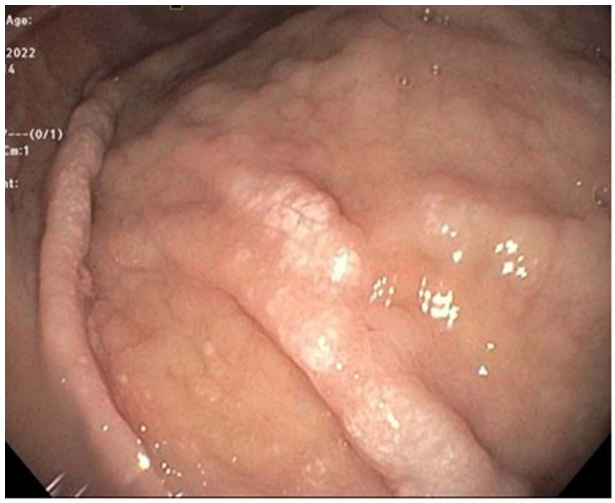
*University of Kentucky College of Medcine, Lexington, KY.*

**Introduction:** Gastric mucosal calcinosis (GMC) is a rare disorder typically found incidentally or at autopsy. GMC can rarely lead to upper gastrointestinal bleeding if it results in severe erosive or ulcerative gastritis. We present a case of a patient with GMC secondary to End-Stage Renal Disease (ESRD).

**Case Description/Methods:** A 74-year-old male with history of ESRD on peritoneal dialysis, atrial fibrillation, HFrEF, and hypothyroidism was admitted for generalized weakness, abdominal pain, and 4 days of melena. He had been hospitalized one week prior for peritonitis, which was treated with vancomycin and ceftazidime. Review of systems was also notable for odynophagia. He denied dyspnea, chest pain, hematochezia, or hematemesis. Vital signs were notable for tachycardia but were otherwise within normal limits. Physical exam was significant for an irregularly irregular cardiac rhythm, mild abdominal distension, and moderate diffuse abdominal tenderness. Labs were notable for white cell count of 16 per μl, hemoglobin 7.7 g/dl, platelets 236,000 per μl, K 2.5 mmol/l, Ca 6.4 mmol/l, and phosphorus 4.2 mmol/l. CT images showed mild wall thickening of the descending and sigmoid colon with mild mesenteric stranding which may represent colitis. Stool PCR was positive for Clostridium difficile. He subsequently underwent esophagogastroduodenoscopy (EGD) that demonstrated patchy areas of whitish mucosa (Figure) associated with moderate gastric erythema, edema, and erosions in addition to thickening of gastric folds. Gastric biopsies revealed gastric mucosal calcinosis. He was started on fidaxomicin for C. diff colitis. He was continued on proton pump inhibitors with resolution of his melena.

**Discussion:** GMC, as found in this patient, can be part of metastatic calcinosis, which is the most common type of GMC. It can occur in up to 13% of ESRD patients. Gastric tissue is thought to be more prone to calcification given its relatively intracellular alkalinity. Endoscopic evidence of gastric calcifications is generally seen as 1-5 mm white flat plaques in fundus, body, or antrum. Diagnosis is important in this patient population as GMC can rarely cause ulcerations/necrosis of gastric tissue leading to acute upper gastrointestinal bleeding. Therefore, GMC should be always considered in the differential diagnosis in patients with long history of ESRD presenting with melena.



[2541] **Figure 1.** Endoscopic appearance of gastric mucosal calcinosis.

S2542

**A Rare Case of Renal Cell Carcinoma Diagnosed on EGD**

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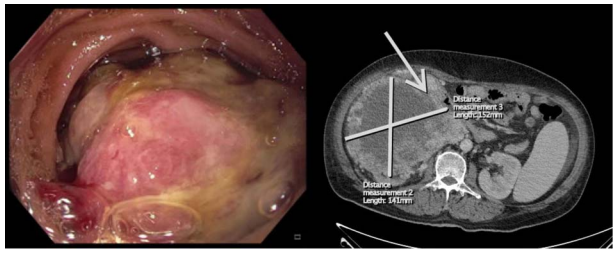
**Introduction:** Neoplasms of the duodenum account for a fraction of overall gastrointestinal tumors. Even when a lesion is found in the duodenum, it is more likely to be of secondary neoplastic origin than primary. Here we present a unique case of primary renal cell carcinoma (RCC) diagnosed on EGD.
**Case Description/Methods:** A 55-year-old male with a history of alcohol use disorder presented to the emergency department for lightheadedness, fatigue, and 100-pound unintentional weight loss over 3 months. On exam, the patient was found to be hypotensive, tachycardic, and jaundiced. Laboratory values were pertinent for a Hemoglobin of 4.4 g/dl. After adequate resuscitation, esophagogastroduodenoscopy (EGD) was performed that showed a large partially obstructive, infiltrative, and ulcerated mass with no bleeding in the second part of the duodenum. Histology from the duodenal mass biopsy revealed clear cell carcinoma of renal origin with mucosal ulceration. An abdominal computerized tomography (CT) scan showed a heterogeneous, centrally necrotic right renal mass causing mass effect on the second and third portions of the duodenum, right hemicolon, and hepatic flexure. The scan was also suggestive of duodenal invasion by the mass. Solid pulmonary nodule biopsy was unequivocal for metastasis. The patient was discussed in tumor board and deemed to be a candidate for cytoreductive surgery with partial duodenal resection and systemic immunotherapy if metastasis was confirmed on future biopsies (Figure).
**Discussion:** Duodenal metastasis from RCC is very rare and poses a diagnostic challenge. The most common presentation is with upper gastrointestinal hemorrhage or obstruction, though sometimes it can present with perforation, intussusception, or obstructive jaundice. Duodenal involvement is often from direct infiltration or metastatic spread. Delay in care and large tumor size are the likely reasons for the rare presentation seen in this patient. This case highlights the importance of considering all neoplasms of extraintestinal origin when duodenal lesions are identified endoscopically.




[2542] **Figure 1.** Left: 2nd portion of duodenum. Right: CT Abdomen/Pelvis.

S2543

**A Rare Complication of Percutaneous Endoscopic Gastrostomy Tube: A Case Report**

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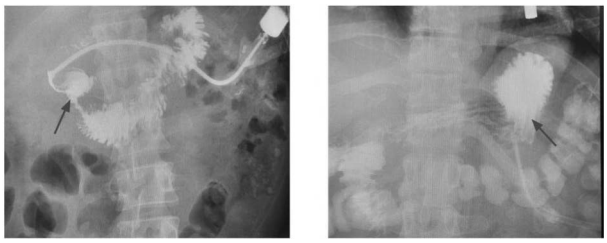
[1]The Wright Center for Graduate Medical Education, Clark Summit, PA; [2]Trinity Health of New England, Springfield, MA; [3]The Wright Center for Graduate Medical Education, Scranton, PA; [4]The VA Medical Center Wilkes Barre, Wilkes Barre, PA.

**Introduction:** Percutaneous endoscopic gastrostomy (PEG) tube procedure is performed globally for various indications for patients whose oral intake is not safe or unable to meet their nutritional needs. Commonly performed for long-term enteral nutrition and medication use. Though it is a safe and effective procedure, complications are not uncommon. We report a rare complication of the PEG tube as migration of internal balloon into the pylorus
**Case Description/Methods:** A 43-year-old man with diabetes, traumatic brain injury, stroke, and ulcerative colitis. Underwent PEG tube placement for long-term nutrition and medication use. PEG tube being used uneventfully for more than 2 years. He presented to hospital with complaints of resistance to tube feeds and abdominal pain for one day. On arrival the patient is in mild abdominal pain, otherwise vital signs were in normal range. On physical examination, he has a 20F PEG tube in the left upper quadrant area. The external bolster is almost close to the feeding port. Unable to pull the PEG tube outwards, it felt like the internal balloon stuck inside the stomach. Even under gentle pressure, the patient was complaining of pain. The imaging of the tube was performed using gastrografin instilled through the tube. Which showed the internal balloon /distal tip of the tube is inside the proximal aspect of the duodenum (Figure, left panel). It was confirmed that the PEG tube has migrated into the pylorus. At the bedside, deflated the internal balloon and the PEG tube was removed with some resistance. A new 20F PEG tube was inserted through the same track, the internal balloon inflated and the external bolster was secured properly. Post-placement of the new PEG tube, the repeat imaging using gastrografin , showed the internal balloon is within the stomach (Figure, right panel). Patient was discharged home with proper instructions to prevent and avoid complications of a similar type.
**Discussion:** The complications associated with PEG tubes range from simple tube clogging to serious buried bumper syndrome. The tube migration is a rare complication, which may occur due to increased peristalsis which could push the internal balloon into the pylorus. Patients with this complication are at risk of gastric outlet obstruction, even block the pancreatic, biliary ducts leading to serious complications like pancreatitis. Our case provides useful insight into the importance of recognizing the tube migration early and treating it appropriately to prevent any further complications and minimize patient suffering.



[2543] **Figure 1.** Left: Internal balloon is seen inside the proximal aspect of the duodenum, (arrow); Right: Internal balloon is seen within the stomach. (arrow).

S2544

**Acute Symptomatic Hyponatremia Following Single Balloon Enteroscopy With Water Immersion: Be on the Lookout**

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[1]Icahn School of Medicine at Mount Sinai - NYC Health + Hospitals/Elmhurst, Elmhurst, NY; [2]Elmhurst Hospital Center, Elmhurst, NY.

**Introduction:** The water immersion endoscopic method is a safe and often used endoscopic technique. We report a unique case of electrolyte abnormalities in a single balloon enteroscopy secondary to this procedure
**Case Description/Methods:** A 70-year-old female with HTN and hypothyroidism presented for single balloon enteroscopy for removal of a retained capsule. The capsule was initially performed for the of work up of melena and iron deficiency anemia after a negative upper endoscopy and colonoscopy. Physical exam and labs prior to the procedure were normal, including a sodium level of 139. The patient underwent the planned enteroscopy with water emersion with an estimated amount of 4 L of water being used. Shortly after completion of the procedure, the patient was noted to be delirious and have an altered mental

status. The patient became aphasic, began clenching her fists, and was shaking. A sodium level post-procedure was obtained and was 113. A head CT scan was performed and no central involvement was noted. The patient was managed with 3% hypertonic saline and her symptoms resolved. Over the next 2 days patient's sodium level normalized and the patient was discharged from the hospital.
**Discussion:** This is the first reported case of hyponatremia secondary to water immersion endoscopy. While there is an abundance of reports describing hyponatremia in urologic and gynecologic procedures those procedures generally use glycine and mannitol as their irrigate. With regards to GI procedures, hyponatremia secondary to polyethylene glycol-electrolyte preparation has infrequently been reported. Free water irrigation/immersion is generally regarded as safe during gastroenterological procedures. Our case, brings awareness to the possibility of symptomatic hyponatremia following prolonged enteroscopy with the use of large volume water irrigation/immersion. Absorption of ingested water and most solutes occur in the proximal small intestine. If a large amount of fluids are necessary then normal saline can be utilized instead of water. Limiting water to 1.5 liters and suctioning excess water can help minimize these complications. Clinicians should be aware of this serious complication when performing these procedures.

**S2545**

**AL Amyloidosis Presenting as Isolated Gastric Lesion Found on Endoscopy in Patient With Recurrent Emesis**

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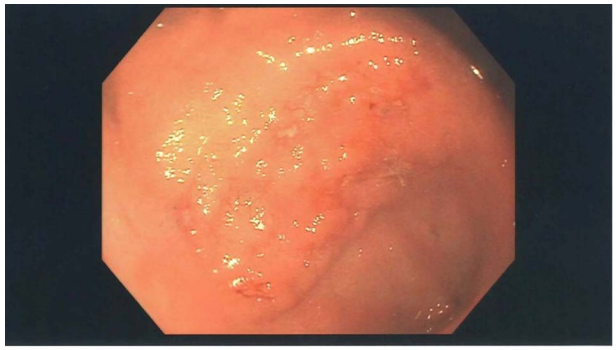
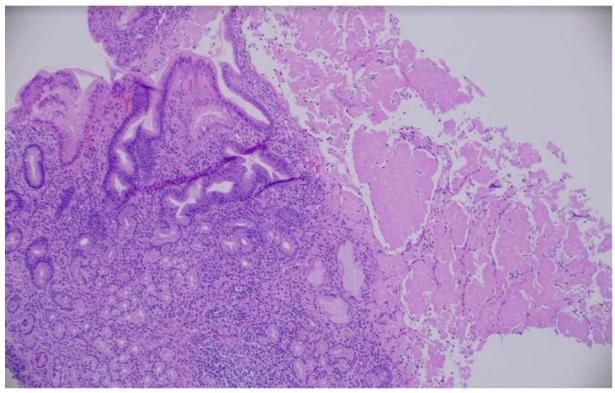
*Dell Medical School at the University of Texas at Austin, Austin, TX.*

**Introduction:** Isolated gastric amyloid is a rare condition with a variable presentation. The following is a case of gastric amyloid presenting as a single large lesion in a patient with few nonspecific symptoms.
**Case Description/Methods:** A 55-year-old woman with a history of Lennox-Gastaut Syndrome underwent endoscopy for emesis workup. The procedure showed a large, friable, ulcerated lesion in the gastric antrum on the lesser curvature (Figure A). The lesion measured approximately 3 x 8 cm and was concerning for malignancy. However, biopsies of the lesion were consistent with active chronic gastritis with submucosal light chain (AL) amyloid deposition, lambda type (Figure B).
**Discussion:** Gastric amyloidosis is a rare condition, occurring in approximately only 3% of patients with amyloidosis. In light chain amyloidosis, it most often manifests in patients with the lambda subtype, as in the case presented here. The symptoms are vague and protean, making a clinical diagnosis difficult. Patients may complain of epigastric discomfort and may demonstrate signs of dysmotility. Ultimately, the definitive diagnosis and classification of amyloid must be made with histologic confirmation. In this case, the patient was unable to communicate her symptoms because of her intellectual disability, but did have constipation and increased emesis in the absence of other reported symptoms or findings consistent with systemic amyloidosis. Extensive workup to exclude systemic amyloidosis was unrevealing. This particular case is notable for the presentation of the disease with a single, large, friable ulcer, adding to the literature concerning the variable presentation of gastric amyloidosis on endoscopy. Considering the difficulty in clinical diagnosis and the morbidity of this disease, the authors propose a low threshold to consider pathologic examination for gastric amyloidosis in patients with nonspecific symptoms and abnormal findings on endoscopy.





[2545] **Figure 1.** A: This image was taken from endoscopy and shows a large friable, ulcerated lesion that was found in the patient's antrum on the lesser curvature. It was later found to be consistent with AL amyloidosis. B: This is a histology slide from the biopsy that was taken from the gastric ulcer, demonstrating fluffy pink amyloid tissue, found to be consistent with AL amyloidosis, lambda subtype.

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**S2546**

**All Roads Lead to SMAD4: Menetrier's Disease in Association With a SMAD4 Mutation**

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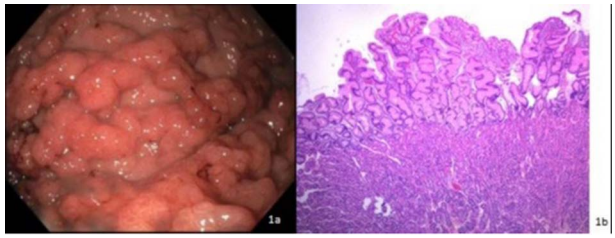
**Introduction:** Menetriere's Disease (MD) is a rare protein losing enteropathy. A common histological feature of MD is massive foveolar hyperplasia (expansion of mucus cells). Medical therapies include Cetuximab and Octreotide but total gastrectomy remains the mainstay as a curative option. We report a case of MD, unrelated to infection in a patient with a germline SMAD4 mutation.

**Case Description/Methods:** A 39-year-old male with past medical history of hypertension, and an extensive orthopedic history was referred for iron deficiency anemia. Initial workup was notable for WBC 9,700cells/uL, Hemoglobin 6.2g/dL, Hct 23%, MCV 80.1 fL, Platelets 423 k/uL, Ferritin 13.2 ng/mL, Iron 40 ug/dL, total iron binding capacity 304 ug/dL, Transferrin 243 mg/dL, and transferrin saturation 12%. Symptoms included fatigue and exertional dyspnea but no gastrointestinal symptoms such as melena, hematochezia, abdominal pain, or diarrhea. Family history was notable for Juvenile Polyposis Syndrome (JPS) and a benign gastric mass removed by a partial gastrectomy in his father. He underwent endoscopic workup for further assessment. Upper endoscopy demonstrated severe gastritis and diffusely thickened gastric folds with thick mucus secretion in the body and cardia. (Figure 1A) The antrum appeared normal. Body and cardia biopsies showed diffuse foveolar hyperplasia with cystically dilated foveolar glands and edematous, mildly inflamed lamina propria consistent with MD. (Figure 1B) Biopsies were negative for H. Pylori and lacked viral cytopathic changes. Due to his family history of JPS, he was referred for genetic testing which revealed a SMAD4 gene mutation. The patient was admitted several weeks later for worsening anemia and renal insufficiency and diagnosed with atypical Hemolytic Uremic Syndrome and unfortunately passed away from complications

**Discussion:** SMAD4 is an intracellular signaling mediator of the Transforming growth factor beta (TGF-β) pathway. Loss of TGF- β stimulation leads to unopposed TGF-a processes, causing features consistent with MD (decreased stomach acidity, hyperplasia of mucus cells, and gastric antralization). Only a handful of documented associations have been published between MD and SMAD4. In one study, with overlap of MD and JPS, SMAD4 was hypothesized to be the causative mutation for both. Despite lacking confirmatory genetic testing in our patient's father, it is safe to assume the autosomal dominant SMAD4 mutation is the culprit in both individuals despite the differential expression as disease.




[2546] **Figure 1.** Endoscopic and Pathologic appearance of Menetriers Disease associated with SMAD4.

**S2547**

**An Unusual Cause of Gastric Outlet Obstruction**

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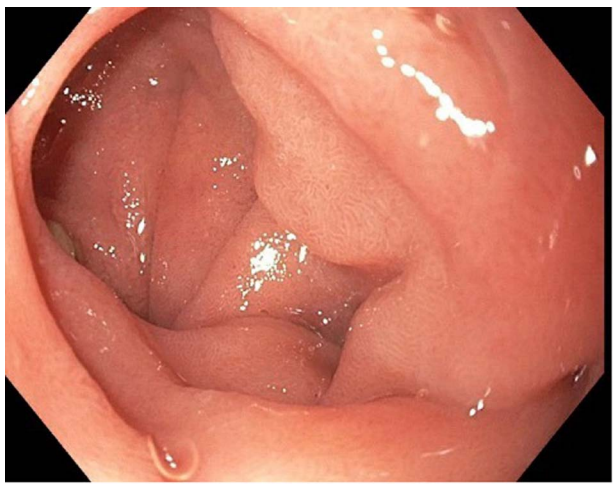
**Introduction:** Gastric outlet obstruction (GOO) is often an initial sign of upper gastrointestinal neoplasms causing mechanical obstruction that is characterized by abdominal pain and vomiting. Neoplasms that most often cause GOO include gastric, pancreatic, and biliary tract malignancies. We report an 82-year-old female who presented with nausea, vomiting, and right upper quadrant pain, without urinary symptoms, who was found to have a GOO due to high grade urothelial carcinoma.

**Case Description/Methods:** An 82-year-old female with no gastrointestinal or urinary history presented with nausea, vomiting and right upper quadrant abdominal pain with no urinary or systemic symptoms. A computer tomography (CT) showed severe right hydronephrosis related to a 3.5 x 2.2 cm ill-defined soft tissue density at the ureteropelvic junction, which extended to the lower pole calyx of the right kidney. The patient left against medical advice and returned 5 days later due to increasing symptoms. A repeat CT showed increased distention of the stomach secondary to encasement of the duodenum from the neoplasm. Urinalysis was negative for blood, casts, transitional epithelium, and squamous epithelium. A CT urogram showed severe right hydronephrosis secondary to an irregular mass that appeared to infiltrate into the surrounding fat and abutted the duodenum, inferior vena cava and right psoas muscle. An upper endoscopy (EGD) to further evaluate for gastric origin did not find mucosal disease, but it did show a severe extrinsic deformity in the third portion of the duodenum (Figure). The patient then underwent a cystoscopy with right retrograde pyelogram and right ureteral stent placement. Unfortunately, her cytology was positive for high grade urothelial carcinoma. Ultimately, the patient felt too weak to proceed with any procedures and chose to transition to hospice care.

**Discussion:** GOO most often can be caused by infiltrative disease, peptic ulcer disease, gastric polyps, and malignancy. Upper tract urothelial carcinoma represents 5% of urothelial cancers with few cases causing GOO reported. Although this patient did not present with hematuria of flank pain, this case highlights, patients presenting with intractable vomiting or abdominal pain should undergo imaging.



[2547] **Figure 1.** EGD showing a severe extrinsic deformity in the third portion of the duodenum.

**S2548**

**An Unusual Case of Abdominal Hematoma Presenting After Long Quiescence Period**

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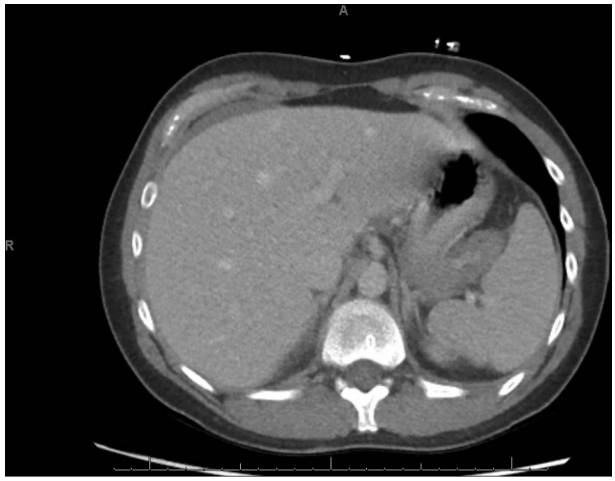
**Introduction:** Incidental abdominal masses are often detected during routine imaging for abdominal pain. Abdominal hematomas usually present a few days after recent trauma and are associated with peritonitis requiring surgical intervention. We report a case of symptomatic abdominal hematoma, presenting as an abdominal mass, 1 year after motor vehicle accident (MVA).

**Case Description/Methods:** 24-year-old female presented with sudden onset sharp epigastric and left upper quadrant (LUQ) abdominal pain, radiating to bilateral shoulders, along with decreased oral intake, nausea, and bilious vomiting. On physical exam, she was hemodynamically stable, abdomen was soft, non-distended, with epigastric and LUQ tenderness, bowel sounds normal, otherwise unremarkable exam.

Initial blood work was unremarkable. Computed tomography of abdomen and pelvis (CTAP) revealed a 5.6x 2.1x 5.4 cm mass located between stomach and spleen without associated adenopathy, suspicious for gastrointestinal stromal tumor (Figure). On EUS, the lesion appeared poorly defined, multicystic and likely originating outside the gastric wall. There was also a moderate amount of free fluid in the abdomen. A fine needle biopsy was obtained, but the sample was largely blood clot. The next day, the patient developed sharp abdominal pain with guarding, blood work revealed hemoglobin levels drop from 12.9 to 7.6 g/dl. CTAP angiography showed development of large amount of intraperitoneal free fluid, without active extravasation, and grade 1 splenic laceration, likely chronic. The non-enhancing hyperdense mass was again noted, unchanged in size and location. Laparoscopy revealed a ~6cm hematoma and evacuated 2L old blood. Postoperative diagnosis was spontaneous hemoperitoneum secondary to trauma of short gastric vessel. Pathology result showed blood, inflammation, and rare degenerated epithelial cells. Upon further interrogation, patient endorsed MVA 1 year ago but did not seek medical care due to no symptoms. Patient was monitored after surgery and upon significant improvement in her symptoms and blood work, she was discharged.

**Discussion:** It is rare for abdominal hematomas to be quiescent for one year and then suddenly present as peritonitis without any further inciting event. Our patient could have suffered from major morbidity, given the drastic drop in hemoglobin level, without urgent intervention. Physicians should have high clinical suspicion and investigate for possible trauma, in acute abdominal pain cases with an incidental abdominal mass on imaging.




[2548] **Figure 1.** Computed tomography of abdomen and pelvis revealing a hypo-echoic mass located medially adjacent to the spleen and laterally adjacent to the stomach. No surrounding fat stranding or lymphadenopathy noted.