SMALL INTESTINE

S1534 Lawlor Resident Award

Longitudinal Analysis of Gluten-Free Dietary Adherence and Psychosocial Health in Gluten-Associated Disorders: The UCLA Celiac Collective

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Introduction: Gluten-free diet (GFD), the mainstay of gluten-associated disorder (GAD) treatment, relies on patient adherence to optimize its therapeutic value. Psychological distress and decreased quality of life are associated with GFD non-adherence, but longitudinal assessments of psychosocial health's impact on dietary adherence are lacking. Our study is the first to explore mental health status and motivational factors (eg, motivation type, perceived competence), and their connection with longitudinal changes in GFD adherence.
Methods: In our prospective study, we invited patients with self-reported GAD from the UCLA Celiac Collective to complete baseline (2016-2020) and follow-up (2022) surveys. Surveys captured demographics, medical history, and responses to the Celiac Dietary Adherence Test; Celiac Symptom Index; PRO Measurement Information System's depression, anxiety, sleep disturbance, fatigue, pain, and social satisfaction domains; Celiac Disease Quality of Life; Treatment Self-Regulation Questionnaire for motivation; Perceived Competence Scale; and Health Care Climate Questionnaire for relationship with healthcare provider. Cross-sectional analyses of GFD adherence, the primary outcome, used McNemar's and Wilcoxon rank-sum tests; the longitudinal analysis assessed changes in GFD adherence from baseline to follow-up with *T*-tests.
Results: We identified 449 patients with GAD at baseline (87.9% female, mean age 48). Of those patients, 123 completed follow-up. Factors consistently associated with higher GFD adherence at baseline and follow-up were lower scores for disease activity, anxiety, depression, fatigue, pain; and higher competence, autonomous motivation, and social satisfaction. Forty-4 patients whose GFD adherence declined over time also saw an increase in disease activity, depression, fatigue; and a decrease in social satisfaction.
Conclusion: This is the first longitudinal study to show that intrinsic (or autonomously) motivated GAD patients who perceived themselves to be competent in complying with a GFD achieved better adherence, which in turn was linked to less distress and disease activity. This pattern emerged at both assessment periods and suggests that specific motivational behaviors may drive GFD adherence and its therapeutic value. Providers, whose influence is demonstrated by the positive association seen between patient-provider relationships and adherence, should treat GAD by considering these intrinsic behavioral qualities while addressing mental health disorders.

Table 1. Measures associated with GFD adherence

	Non-Adherence (CDAT ≥ 13)	Adherence (CDAT < 13)	*P*-value	Worsened Adherence	Improved Adherence	*P*-value
Celiac Disease Activity (CSI)						
Baseline	45.0 (37.0-51.0)	29.0 (23.0-36.0)	< 0.001	+4.2 (8.3)	-2.8 (8.2)	0.002
Follow-up	38.5 (36.0-45.5)	32.0 (25.5-36.5)	< 0.001			
Anxiety (PROMIS-29)						
Baseline	9.0 (6.0-12.0)	6.0 (4.0-8.0)	< 0.001	+1.2 (2.7)	0.0 (3.9)	0.154
Follow-up	9.0 (6.0-11.0)	7.0 (4.0-9.8)	0.018			
Depression (PROMIS-29)						
Baseline	7.0 (4.2-10.0)	4.0 (4.0-6.0)	< 0.001	+1.1 (2.1)	-0.6 (3.3)	0.014
Follow-up	7.0 (5.0-9.0)	4.0 (4.0-5.0)	< 0.001			
Sleep (PROMIS-29)						
Baseline	10.0 (8.0-12.0)	7.0 (5.0-9.0)	< 0.001	+0.4 (2.6)	+0.3 (3.3)	0.842
Follow-up	9.0 (7.0-11.0)	8.0 (6.0-10.0)	0.185			
Fatigue (PROMIS-29)						
Baseline	13.0 (9.5-16.0)	8.0 (6.0-10.0)	< 0.001	+1.5 (2.8)	-1.1 (3.2)	< 0.001
Follow-up	12.0 (10.0-15.0)	8.0 (6.5-10.5)	< 0.001			
Satisfaction with Social Interaction (PROMIS-29)						
Baseline	10.0 (7.0-12.0)	14.0 (12.0-16.0)	< 0.001	-1.0 (3.5)	+1.0 (3.2)	0.019
Follow-up	10.0 (8.0-12.0)	15.0 (12.0-16.0)	< 0.001			
Pain (PROMIS-29)						
Baseline	9.0 (5.5-14.0)	4.0 (4.0-8.0)	< 0.001	+0.6 (4.0)	-0.8 (4.0)	0.15
Follow-up	8.0 (6.8-12.0)	5.0 (4.0-7.2)	< 0.001			
Relationship with Healthcare Provider (HCCQ)						
Baseline	21.0 (12.0-33.5)	25.0 (15.0-36.0)	0.05	-0.4 (11.2)	+4.7 (8.6)	0.051
Follow-up	23.5 (14.2-29.8)	27.5 (19.5-38.2)	0.044			
Competence (PCQ)						
Baseline	26.0 (22.8-28.0)	28.0 (27.0-28.0)	< 0.001	+0.2 (2.2)	+1.7 (4.2)	0.068
Follow-up	28.0 (25.0-28.0)	28.0 (28.0-28.0)	0.002			
Autonomous Motivation (TSRQ)						
Baseline	40.0 (36.0-42.0)	42.0 (38.5-42.0)	< 0.001	-1.1 (6.4)	+1.1 (6.8)	0.172
Follow-up	40.0 (35.0-42.0)	42.0 (41.8-42.0)	0.003			
Controlled Motivation (TSRQ)						
Baseline	16.5 (8.2-23.0)	13.0 (7.2-23.0)	0.444	-3.2 (7.9)	-0.8 (6.4)	0.187
Follow-up	12.5 (6.0-22.0)	12.0 (6.8-20.0)	0.912			
Amotivation (TSRQ)						
Baseline	4.0 (3.0-8.0)	4.5 (3.0-9.0)	0.988	-0.4 (3.9)	-0.1 (4.4)	0.759
Follow-up	5.0 (3.0-8.0)	4.0 (3.0-7.8)	0.628			
Quality of Life (CDQoL)						
Baseline	62.0 (49.0-77.0)	50.0 (37.0-59.5)	< 0.001	-1.1 (12.6)	-2.5 (12.4)	0.786
Follow-up	50.0 (43.2-63.5)	45.5 (40.0-52.5)	0.252			

Data on the right ("Worsened Adherence" & "Improved Adherence") represent change of score means (SD) from baseline to follow-up. CDAT: Celiac Dietary Adherence Test; CSI: Celiac Symptom Index; PROMIS: PRO Measurement Information System; CD-QOL: Celiac Disease Quality of Life; TSRQ: Treatment Self-Regulation Questionnaire; PCS: Perceived Competence Scale; HCCQ: Health Care Climate Questionnaire
Data from the left half of the **Table** (under "Non-Adherence" & "Adherence") represent score medians (Q1-Q3) at baseline and follow-up.

S1535 Outstanding Research Award in the Small Intestine Category (Trainee)

Common Variable Immunodeficiency-Like Enteropathy Associated With Rituximab B-Cell Depletion Therapy

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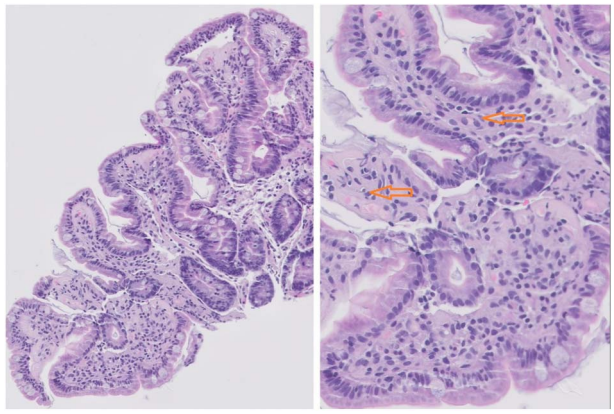
Introduction: Rituximab (RTX), is a monoclonal antibody therapy directed at CD20 positive B lymphocytes, increasingly used in both malignant and autoimmune conditions. RTX therapy may result in hypogammaglobulinemia due to B cell depletion. The gastrointestinal side effects of the therapy are not well described. Here we report the clinical, endoscopic and histologic features of rituximab-associated enteropathy in a single center case series.
Methods: Retrospective analysis of medical charts of patients with enteropathy presenting to a tertiary care university hospital between 2008 and 2021 were reviewed. Patients with enteropathy identified using diagnostic codes for: CVID, enteropathy, malabsorption, weight loss and diarrhea. Patients with rituximab use were further characterized. Clinical data, laboratory values, endoscopy and pathology reports were analyzed.
Results: Ten patients with enteropathy associated with RTX therapy were identified without other competing etiology. Mean age was 64 years ± 17.4 and 5 (50%) patients were female. Lymphoma 8 (80%) was the most common indication for RTX therapy. Diarrhea and weight loss were the most common clinical presentation. The mean BMI on presentation was 18 kg/m^2 with a mean total weight loss of 13.5 kg ± 8.9. Seven (70%) patients were defined as severely malnourished based on clinical criteria. Mean albumin was 3.3 g/dL (3.5-5) and pre-Albumin 16 g/dL (normal >21). Serum levels of IgA, IgM and IgG were below lower limit of normal. Endoscopic appearance of the duodenal mucosa was abnormal in 3/8 cases (37.5%) and 3/5 (60%) cases had decreased plasma cells in the lamina propria with villous atrophy (Figure 1). Seven patients (70%) had a pathologic diagnosis consistent with CVID enteropathy. All of the patients received IVIG therapy (100%) although only 2 had symptomatic improvement. Prednisone (n = 6), budesonide (n = 2), infliximab (n = 2) or vedolizumab (n = 1) did not lead to clinical improvement. Six (60%) required long term parenteral nutrition (PN).
Conclusion: RTX is associated with a secondary CVID-like enteropathy with absent/decreased plasma cells in small bowel biopsies. Diarrhea with weight loss and severe malnutrition are common clinical features and patients often require parenteral nutrition. Treatment with IVIG and other immunosuppressive therapies rarely improves GI symptoms. RTX-associated enteropathy should be considered in patients presenting with diarrhea and/or weight loss and history of RTX use.



[1535] **Figure 1.** Small bowel mucosa showing a lack of lamina propria plasma cells (arrows).

S1536 Outstanding Research Award in the Small Intestine Category
Presidential Poster Award

Iron Deficiency and the Development of Celiac Disease

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Introduction: The prevalence of celiac disease is increasing; however, the cause for this increase is unknown. Iron deficiency is one suggested environmental trigger for celiac disease development. We aimed to evaluate this possible association through the use of Mendelian randomization (MR). MR capitalizes on the random allocation of single nucleotide polymorphisms (SNPs) at conception, and under certain assumptions can suggest causality.
Methods: We conducted a 2-sample MR study examining the relationship between SNPs associated with iron status and the presence of celiac disease. The SNPs were drawn from a meta-analysis of 3 genome-wide association studies (GWAS). The GWAS summary statistics from the UK Biobank, consisting of 336,638 White British individuals of whom 1,855 participants had celiac disease, were used to assess the association between these SNPs and celiac disease. The assumptions of MR were tested to evaluate for possible causality. We also performed an MR Egger test for pleiotropy.
Results: There are 4 SNPs strongly associated with systemic iron status. Using PhenoScanner, we found that these SNPs were not significantly associated with known risk factors for celiac disease. All 4 were available in the UK Biobank summary statistics, and utilizing these SNPs, we harmonized exposure and outcome associations. We found that higher iron status was negatively associated with risk of celiac disease (odds ratio per 1 standard deviation increase in serum iron: 0.65, 95% CI 0.47-0.91). We performed leave-one-out analyses and had consistent results and did not find one single variant to be driving this association. All 3 assumptions of MR appeared plausible.
Conclusion: We found that genetically lower iron levels were associated with an increased risk of having celiac disease. If the assumptions of MR hold, this association suggests a causal role of iron deficiency in celiac disease development. While unlikely to be the only environmental trigger, iron deficiency is prevalent in both the general and celiac population. One possible mechanism for this pathogenic role is that the transferrin receptor 1, which is upregulated in the setting of iron deficiency, directly transports gliadin peptides across enterocytes – a key step in celiac disease development. Our findings highlight a potential opportunity for celiac disease prevention.

S1537 Presidential Poster Award

Celiac Disease Is Associated With Cardiac Pathology: Results From a National Database

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Introduction: Celiac disease (CD) is a common and underdiagnosed immune-mediated systemic disorder triggered by dietary gluten with a strong genetic component and various manifestations. Few studies in the literature examine the relationship of CD to cardiac pathology such as dilated cardiomyopathy (DCM), myocarditis (MC), and pericarditis. The potential links between CD and these cardiac pathologies are numerous and include systemic inflammation, imbalanced nutrition, auto-immune contribution, and potential shared genetic predisposition. We aimed to describe these associations using a large healthcare database.
Methods: We queried IBM Explorys (Cleveland, OH), a database aggregating approximately 81 million patients drawn since 1999 from across the United States. Employing SNOMED Clinical Terms definitions, we described patients with DCM, idiopathic myocarditis, and pericarditis, excluding those with clear secondary diagnoses such as infection and lupus. Multivariate regression analysis was performed using IBM SPSS Statistics v26 (Chicago, IL). The analysis accounted for celiac disease as well as demographic variables and potential confounders, including age, gender, hypertension, obesity, diabetes, alcohol abuse and tobacco use.

Results: Of 80,920,060 patients included in the database (including 143,330 CD patients, or 0.18%), 848,960 were diagnosed with DCM (3,940/0.46% CD), 27,390 with idiopathic myocarditis (180/0.65% CD) and 63,990 with idiopathic pericarditis (460/0.71% CD). After running the multivariate regression analysis, CD was significantly associated with DCM (OR 1.56, 95% CI 1.50-1.60, $P < 0.001$), idiopathic myocarditis (OR 3.49, 95% CI 3.02-4.04, $P < 0.001$), and idiopathic pericarditis (OR 3.65, 95% CI 3.32-3.40, $P < 0.001$).
Conclusion: After adjusting for potential confounders, CD was significantly associated with occurrence of DCM, idiopathic myocarditis, and pericarditis as represented in this large United States healthcare database. This finding may have clinical implications for CD patients who present with chest discomfort or those controlled on a gluten-free diet with new concerns of upper abdominal or lower chest discomfort.

Table 1. Results of the Multivariate Logistic Regression Analyses Showing

Risk Factor	OR for DCM	*P*-value	OR for Myocarditis	*P*-value	OR for Pericarditis	*P*-value
Celiac Disease	1.55 [1.50-1.60]	< 0.001	3.49 [3.02-4.04]	< 0.001	3.65 [3.32-4.00]	< 0.001
Age ≥ 65	2.28 [2.27-2.29]	< 0.001	2.05 [2.00-2.10]	< 0.001	2.45 [2.41-2.49]	< 0.001
Female gender	0.64 [0.64-0.65]	< 0.001	0.63 [0.62-0.65]	< 0.001	0.65 [0.64-0.66]	< 0.001
White ethnicity	1.15 [1.14-1.15]	< 0.001	1.96 [1.91-2.01]	< 0.001	2.16 [2.12-2.20]	< 0.001
Tobacco use	1.63 [1.62-1.65]	< 0.001	NA		NA	
Alcohol use	2.31 [2.29-2.33]	< 0.001	NA		NA	
Diabetes	1.92 [1.92-1.93]	< 0.001	NA		NA	
Obesity	1.86 [1.85-1.87]	< 0.001	NA		NA	
Hypertension	7.82 [7.77-7.87]	< 0.001	NA		NA	

Odds Ratio (OR), Confidence Intervals, and *P*-values. DCM = Dilated Cardiomyopathy.

S1538 Presidential Poster Award

Clinicopathologic Characterization and Genotype Correlation of Duodenal Polyposis Among Hispanics With Familial Adenomatous Polyposis

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Introduction: Prognosis of individuals with familial adenomatous polyposis (FAP) has improved due to the implementation of specialized surveillance guidelines and prophylactic colectomy. Nonetheless, patients remain at increased risk for other neoplasia such as duodenal cancer. The Spigelman Classification for duodenal polyposis was designed to estimate a patient's risk for duodenal cancer based on endoscopic and histopathologic findings. Prior studies have reported variation in colonic polyposis burden following a genotype-phenotype pattern according to APC gene mutation. The genotype-phenotype relationship in duodenal polyposis remains less clear.
Methods: A total of 71 patients with FAP who had undergone at least one complete esophagogastroduodenoscopy (EGD). Age, sex, tobacco use, alcohol use, and BMI were among the sociodemographic factors evaluated. Study population was divided into mutations in the APC gene between codons 1000-1500 (Group 1) versus mutations before codon 1000 and after codon 1500 (Group 2). All participants were recruited as part of the UPR Medical Sciences Campus IRB approved prospective familiar cancer registry (PURIFICAR - http://purificar.rcm.upr.edu/).
Results: Seventy-one patients with genetic diagnosis of FAP were evaluated. Duodenal adenomas were present in 42 (59%) patuents. 120 EGDs were evaluated for the study, with a mean of 1.7 EGDs per patient (Range = 1–4). Patients with duodenal adenomas on EGD were more likely to have APC mutations between codons 1000-1500 (65.7% vs 20%; $P = 0.001$). Differences in Spigelman stage between the 2 genotype groups were not statistically significant (*P*-value = 0.86). Furthermore, bivariate analysis of gender, age at last contact, BMI, tobacco use, and alcohol use were not independently associated with Spigelman stage. No cases of duodenal or ampullary cancer were observed after an overall follow-up of 304.6 patient-years.
Conclusion: Our study provides the first phenotypic and genotypic characterization of duodenal polyposis among Hispanic patients with FAP. Duodenal polyposis was significantly associated with APC mutations within the 1000-1500 codon cluster. No other clinicodemographic or environmental associations were independently associated with presence of duodenal polyposis; no cases of duodenal cancer were observed in 304.6 patient-years of follow up. Our observations provide insightful information about the impact of APC genetic mutation location and endoscopic surveillance.

S1539 Presidential Poster Award

Second Primary Malignancy in Gastrointestinal Stromal Tumors: Insights From a Population-Based Analysis

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Introduction: Gastrointestinal stromal tumor (GIST) are the most frequent mesenchymal neoplasms in the digestive tract. Second primary malignancies (SPM) have been reported frequently, either synchronously or during follow-up, in patients diagnosed with GIST. We analyzed the incidence and location of SPM in patients with GIST.
Methods: We conducted a retrospective cohort study using the Surveillance, Epidemiology, and End Results database (SEER) to filter out patients diagnosed with GISTs. Patients with GIST diagnosed between 1975 and 2019 and confirmed on histopathology were included, while those diagnosed at autopsy or lost to follow-up were excluded. SPM was defined as a second tumor diagnosed more than 60 days after the initial GIST diagnosis. Standardized incidence ratio (SIR) and absolute excess risk (AER) were calculated using SEER*Stat software (version 8.4.0.1). P-values and 95% confidence intervals (95% CI) were generated assuming Poisson distribution of observed incidence of SPM.
Results: Overall, 3,202 GIST patients were included (mean age 62.36 years). SPM was reported in 328 (10.2%) patients. Patients with GIST had a significantly greater risk of developing SPM in any location as compared to the general population with SIR = 1.25 (95% CI = 1.11-1.39) and AER of 32.86 per 10,000 population. The most common site for SPM was the digestive tract, specifically the colon (SIR = 1.53, 95% CI = 1.01-2.22) and stomach (SIR = 2.50, 95% CI = 1.29-4.37). Other locations where site-specific risk was significantly increased were the lungs, bronchus and trachea (SIR 1.69), soft tissues (SIR 5.27), skin (SIR 1.71), kidney (SIR 2.34), thyroid (SIR 4.13), and chronic myeloid leukemia (SIR 4.15) (Table 1). Increased risk of SPM was reported for patients aged 50-75 years (SIR = 1.28) but not for patients younger than 50 or older than 75. There was a significantly increased risk of developing SPM in all races; the SIR for Caucasians, African-Americans, and other races (American Indian/AK Native, Asian/Pacific Islander) were 1.19, 1.45, and 1.34, respectively. Increased risk of SPM was reported for latency 2-11 months (SIR 2.31) but not for latencies 12-59 months (SIR 1.06), 60-119 months (SIR 1.05), or 120+ months (SIR 1.29).
Conclusion: Patients with GIST are at a high risk of developing SPM, especially tumors of the digestive and respiratory tract, along with chronic myeloid leukemia. Data suggests a higher incidence of SPM in patients aged 50-75 years and with 2-11 months latency.

Table 1. Secondary Primary Malignancy Sites

Location	Observed	Expected	SIR	95% CI	AER
All Sites	328	263.3	1.25#	1.11-1.39	32.86
All Solid Tumors	287	227.57	1.26#	1.12-1.42	30.18
Stomach	12	4.8	2.50#	1.29-4.37	3.66
Colon	27	17.69	1.53#	1.01-2.22	4.46
Sigmoid Colon	10	4.34	2.30#	1.1-4.24	2.87

Table 1. (*continued*)

Location	Observed	Expected	SIR	95% CI	AER
Lung, and bronchus	60	35.46	1.69#	1.29-2.18	12.49
Skin excluding Basal and Squamous	22	12.86	1.71#	1.07-2.59	4.64
Soft Tissue including Heart	8	1.52	5.27#	2.27-10.38	3.29
Kidney	19	8.1	2.34#	1.41-3.66	5.53
Chronic Myeloid Leukemia	4	0.96	4.15#	1.13-10.62	1.54
Thyroid	16	3.87	4.13#	2.36-6.71	6.16

#represents $P< 0.05$.
SIR : Standardized incidence ratio, AER : Absolute excess risk, 95%CI : 95% Confidence Interval.

S1540 Presidential Poster Award

Effect of Probiotics on Mental Health and Their Association With Serum Neurometabolites in Adults With Depression or Anxiety: A Systematic Review and Meta-Analysis

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Introduction: Tryptophan metabolism is implicated in the development of mental illnesses such as depression and anxiety. Breakdown of the essential amino acid tryptophan produces neurometabolites (namely kynurenine and serotonin) through the classic kynurenine pathway, the neurochemical pathway, and the microbiome-derived metabolic pathway in the gut. Probiotics targeting the gut can regulate the production of these neurometabolites and offer affordable supplements for managing mental illnesses. The objective is to conduct a systematic review and assess the effectiveness of probiotic intake in altering the kynurenine:tryptophan ratio (K:T ratio), serum neurometabolites, and symptoms of depression and anxiety in adults.
Methods: We conducted literature searches via PubMed, SCOPUS, Cochrane (Reviews and Trials), CINAHL, ClinicalTrials.gov and PsycINFO from inception to 2022. We included randomized controlled trials that assessed the impact of probiotics on serum neurometabolites and depression or anxiety scores versus placebo in adults with at least 4 weeks of follow-up. The Cochrane Risk of Bias Tool 2.0 was used to assess the methodological quality of studies. Forest plots were generated for outcomes that had sufficient trials.
Results: We identified a total of 851 studies and 4 studies were included in the meta-analysis. Three studies assessed the impact of probiotics on depression and showed significant improvement in symptom reporting scores (standardized mean difference [SMD] = −0.49; I^2 = 0%; CI = −0.80 to −0.19). Three studies assessed change in anxiety scores with probiotic intervention (SMD = −0.47; I^2 = 0%, 95% CI = −0.80 to −0.14). Three studies assessed the effect of probiotics on kynurenine and showed a significant reduction (SMD = −0.41; I^2 = 0%, 95% CI = −0.72 to −0.10). Three studies assessed the effect of probiotics on tryptophan (SMD = −0.19; I^2 = 74%, 95% CI = −0.81 to 0.42). Results for K:T ratio and changes in serum serotonin levels are forthcoming.
Conclusion: Overall, probiotics significantly improved depression and anxiety symptoms and reduced kynurenine levels in adults. However, there was no significant change in serum tryptophan levels. Few studies reported data that could be included in this meta-analysis. More research is needed to assess the impact of probiotic intake on gut neurometabolic metabolism. This systematic review has important implications for further studying the relationship between the gut and mental health.

S1541 Presidential Poster Award

Algorithmic Approach to Differentiate Between Non-Specific and Specific Etiologies of Chronic Terminal Ileitis

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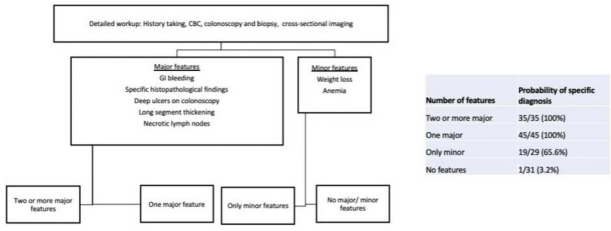
All India Institute of Medical Sciences, Delhi, Delhi, India.

Introduction: Chronic isolated terminal ileal (TI) involvement (terminal ileitis) may be seen in numerous etiologies including Crohn's disease (CD) and intestinal tuberculosis (ITB) in addition to other etiologies that may improve with symptomatic management alone. We aimed to improve our previously suggested algorithm to distinguish patients who would merit specific treatment from those who do not.
Methods: Patients (n=153) with isolated TI involvement (ulcers or nodularity) following-up at All India Institute of Medical Sciences (2007-2022) were retrospectively reviewed. A specific (ITB/CD) diagnosis was made based on standardized criteria and other relevant data was collected. The cohort was utilized for validation of previously suggested algorithm while multivariate analysis with bootstrap validation was used to develop a revised algorithm (Figure).
Results: We included 153 patients (mean age 36.9 years, 70% males, median duration of symptoms 1.5 years) with isolated terminal ileal involvement of whom 109 (71.2%) received a specific diagnosis (69 CD, 40 ITB). Multivariate regression and validation statistics suggested that based on a combination of clinical (blood in stools, weight loss, hemoglobin), radiological (necrotic lymph nodes, long segment ileal involvement) and colonoscopic findings (presence or absence of deep ulcers), an optimism corrected c-statistic of 0.975 and 0.958 could be reached with and without histopathological findings respectively. Based on these, a revised algorithm was developed which showed a sensitivity of 99.08% (95%CI:94.99 - 99.98) and a specificity of 75.0%(95%CI:59.66 - 86.81). The PPV was 90.76%(95%CI:85.47 - 94.25) and NPV was 97.06%(95%CI:82.32 – 99.57) with an overall accuracy of 92.16% (95%CI:86.70 - 95.88). This was a more sensitive and specific than the previous algorithm (accuracy 83.6%, sensitivity 95.4%, and specificity 54.4%).
Conclusion: We suggested a revised algorithm to stratify patients of terminal ileitis into those who have a specific etiology and those who need only symptomatic treatment. Our algorithm has the potential to avoid missed diagnosis as well as unnecessary side effects of treatment.



[1541] **Figure 1.** Algorithm for terminal ileitis (TI) evaluation

S1542 WITHDRAWN

S1543

Disordered Eating in Adults With Gluten-Associated Disorders: The UCLA Celiac Collective

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Introduction: Previous studies have identified disordered eating (DE) in up to 50% of adolescents and adults with gluten-associated disorders (GAD). Since a gluten-free diet (GFD) is the mainstay of therapy, non-adherence is associated with worse outcomes and decreased quality of life. Case studies and systematic studies have described reduced adherence to GFD in individuals with comorbid celiac disease and DE. Our aim was to assess this correlation in a cohort of patients with GAD.
Methods: Participants in the UCLA Celiac Disease Collective registry completed multiple validated patient-reported outcomes (PRO) surveys including the Celiac Dietary Adherence Test (CDAT), Celiac Symptom Index (CSI), and PRO Measurement Information System (PROMIS) for depression, anxiety, fatigue, and social interaction satisfaction. In addition, they completed the 28-item self-report questionnaire Eating Disorder Examination Questionnaire (EDE-Q 6.0), a validated tool for evaluating eating disorder pathology using 4 subscales (restraint, eating concern, shape concern, weight concern) and a global score.
Results: Preliminary results show that more than 50% of participants with GAD have significant DE, comparable to previous data in CD. Individuals with GAD have higher global EDE-Q scores compared to the general population. Worse GFD adherence correlates with higher EDE-Q score, which was statistically significant in the eating concern domain with a trend for restraint, shape concern, and weight concern domains, and global score. Participants with comorbid DE have worse symptom severity and higher prevalence of depression and anxiety. Severity of DE is also associated with comorbid fatigue and decreased social interaction satisfaction (Table).
Conclusion: This study is the first to show that adults with comorbid GAD and DE have worse disease activity and psychological distress. It supports previous studies associating DE with GFD non-adherence. Providers are advised to screen for DE in their GAD population.

Table 1. Measures associated with DE in Individuals with GAD

	Eating Disorder Presence		*P* value
	No (N=13)	Yes (N=14)	
GAD Diagnosis[1]			
Celiac Disease	12 (92.3%)	12 (85.7%)	–
Dermatitis Herpetiformis	0 (0.0%)	1 (7.1%)	–
Other Gluten Associated Disorder	1 (7.7%)	1 (7.1%)	–
GFD Adherence (CDAT)			
	9.6 (2.4)	13.8 (3.8)	0.02
Celiac Disease Activity (CSI)			
	26.9 (7.3)	39.4 (8.1)	0.004
CSI Subcategories[1]			0.035
Active Disease	0 (0.0%)	3 (25.0%)	–
Moderate Disease Control	2 (25.0%)	7 (58.3%)	–
Disease Remission	6 (75.0%)	2 (16.7%)	–
PROMIS-29 Categories			
Anxiety	5.6 (1.9)	8.3 (2.8)	0.026
Depression	4.8 (1.7)	7.5 (3.5)	0.049
Sleep	8.6 (3.0)	8.2 (3.0)	0.761
Fatigue	7.4 (2.1)	12.2 (2.9)	0.002
Social Interaction	14.8 (1.3)	10.3 (3.1)	< 0.001
Pain	5.9 (2.2)	8.0 (4.5)	0.342

Data represent score means (standard deviation) unless otherwise noted.
[1]Reported by frequency.

S1544

Celiac Disease Is Associated With Idiopathic Inflammatory Myopathies

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Introduction: Celiac disease (CD) is a chronic systemic immune-mediated disorder that occurs in genetically predisposed people upon ingestion of gluten. The complications and associations of CD are wide-ranging, with notable associations with other autoimmune diseases. Few studies have described the association of CD with inflammatory myopathies, such as polymyositis (PM), dermatomyositis (DM), and inclusion body myositis (IBM). However, there have been no studies interrogating the association in large cohorts. We investigated these relationships using a large nation-wide patient dataset.
Methods: Utilizing IBM Explorys (Cleveland, OH), a database encompassing over 80 million patients in the United States and spanning the last 2 decades, we defined groups of patients identified with 3 idiopathic inflammatory myopathies using SNOMED Clinical Terms. We collected age, gender, and race-based demographics, in addition to comorbidities including hypothyroidism, alcohol use disorder, tobacco use, and CD, and subsequently performed binary logistic regression using IBM SPSS Statistics v26 (Chicago, IL) to determine significant associations.
Results: In the database, which contained 80,920,060 patients at the time of querying, there were 143,330 CD patients (0.18%). 18,270 (0.02%) of the total patients met the definition of dermatomyositis, with 160 (0.88%) of these carrying the diagnosis of CD, while there were 8,970 (0.01%) polymyositis patients (of which there were 70 / 0.78% with CD) and 1,520 (0.002%) IBM patients (30 / 2% with CD). Following logistic regression that incorporated demographics and several comorbidities, CD was significantly associated with all inflammatory myopathies: the OR for CD in dermatomyositis was 3.55 (95%CI 3.11 — 4.04, $P < 0.001$), 3.23 in polymyositis (95%CI 2.89 — 3.60, $P < 0.001$) and 11.60 in IBM (95%CI 9.02 — 14.92, $P < 0.001$) (Table).
Conclusion: In this very large aggregate of patients across the United States, CD was significantly associated with the idiopathic inflammatory myopathies of polymyositis, dermatomyositis, and IBM after accounting for a number of possible confounders. Clinicians should be aware of this relationship when assessing myalgia in celiac disease patients.

Table 1. Results of the multivariate regression analysis with odds ratios (OR), confidence intervals, and *P*-values (significant if <0.05) for each inflammatory myopathy

Risk Factor	OR for PM	*P*-value	OR for DM	*P*-value	OR for IBM	*P*-value
Celiac Disease	3.23 [2.89-3.60]	< 0.001	3.55 [3.11-4.04]	< 0.001	11.60 [9.02-14.92]	< 0.001
Female gender	1.34 [1.31-1.38]	< 0.001	1.36 [1.32-1.40]	< 0.001	0.47 [0.43-0.52]	< 0.001
Age ≥65	2.51 [2.45-2.57]	< 0.001	2.26 [2.20-2.33]	< 0.001	9.16 [8.20-10.25]	< 0.001
White race	1.49 [1.45-1.53]	< 0.001	1.71 [1.65-1.76]	< 0.001	1.88 [1.69-2.09]	< 0.001
Hypothyroidism	3.59 [3.49-3.69]	< 0.001	3.33 [3.21-3.45]	< 0.001	3.08 [2.76-3.43]	< 0.001
Alcohol use	1.64 [1.53-1.76]	< 0.001	1.76 [1.61-1.93]	< 0.001	3.01 [2.47-3.67]	< 0.001
Tobacco use	1.93 [1.86-2.00]	< 0.001	1.93 [1.84-2.02]	< 0.001	1.45 [1.25-1.68]	< 0.001

S1545

Gender Disparities in Symptoms and Autoimmune Disease in Celiac Disease Patients: A Population-Based Study

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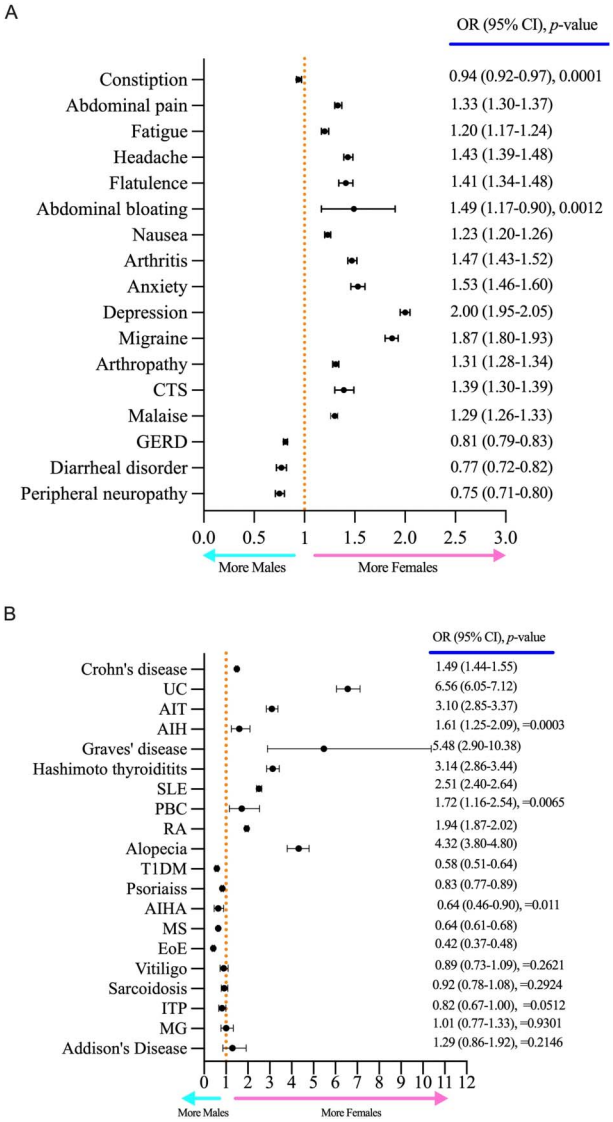
Introduction: Celiac disease (CD) is an immune-meditated inflammatory condition characterized by enteropathy after exposure to gluten. Previous studies suggest gender-based differences in symptom presentation and associated disorders, but this has yet to be explored on a large scale. As such we sought to investigate this area by querying a nationwide database.
Methods: We used a commercial database (Explorys Inc, Cleveland, OH) which includes electronic health record data from 26 major integrated US healthcare systems. Based on Systematized Nomenclature of Medicine – Clinical Terms (SNOMED-CT), we identified all patients (age >18 years) with a diagnosis of CD from 1999 to 2022. Based on gender, the study population was divided into 2 groups: Female and Male. Data on symptoms and autoimmune conditions were gathered.
Results: Of the 70,383,890 individuals in the database, we identified 136,690 (0.19%) patients with CD. In the CD cohort, the majority of patients were White 113,110 (83%), in age between 18-65 years old 97,220 (71%) and predominantly females 102,910 (75%). Abdominal pain, abdominal bloating, nausea, flatulence, headache and arthropathy were seen more in females compared with males. Whereas diarrhea and peripheral neuropathy were seen more in males (Figure A). Some concomitant autoimmune diseases were more frequent in women including autoimmune thyroiditis, Crohn's disease, ulcerative colitis, rheumatoid arthritis, autoimmune hepatitis and alopecia, while others predominated in men such as eosinophilic esophagitis, type 1 diabetes and psoriasis (Figure B).
Conclusion: Significant gender-based disparities were found in this large scale nationwide study of celiac disease patients in the United States. Females exhibited a wide variety of presenting symptoms. Autoimmune conditions were seen in both genders with high rates of concomitant Crohn's disease, lupus, and rheumatoid arthritis in particular. These conditions were found at higher frequencies than type 1 diabetes which has a clear shared genetic susceptibility with celiac disease. Further investigation into these gender differences and their impact on clinical management of celiac disease is warranted.



[1545] **Figure 1.** Forest plot of symptoms (A) and autoimmune conditions (B) in gender based-celiac disease patients. All *P*-values are < 0.0001 unless stated otherwise. Univariate analysis used to calculate OR. OR; odd ratio, CI; confidence interval. Abbreviations: CTS: Carpal tunnel syndrome, UC; ulcerative colitis, AIT; autoimmune thyroiditis, AIH; autoimmune hepatitis, SLE; systemic lupus erythematosus, PBC; primary biliary cholangitis, RA; Rheumatoid arthritis, T1DM; type1 diabetes, AIHA; autoimmune hemolytic anemia, ITP; idiopathic thrombocytopenic purpura, MS; multiple sclerosis, MG;myasthenia gravis EoE; Eosinophilic esophagitis

S1546

Gluten-Associated Disorders and Overlap Irritable Bowel Syndrome: The UCLA Celiac Collective

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Introduction: Treating gluten-associated disorders (GAD) requires knowledge of both pathophysiology and comorbid psychosocial factors. While a gluten-free diet (GFD) results in disease improvement or remission, adherence to treatment often proves to be challenging. Irritable bowel syndrome (IBS)-like symptoms are commonly reported in GAD, and account for up to 20% of non-responsive celiac disease (NRCD). Our study aims to characterize patients with GAD and comorbid IBS and explore the role of non-adherence in this group.
Methods: We conducted a cross-sectional study involving patients with GAD from the UCLA Celiac Collective registry, who were invited to complete online IBS surveys in 2022. Surveys captured demographics, medical history, and responses to the Rome IV Criteria, IBS Symptom Severity Scale (IBS-SSS), Celiac Dietary Adherence Test (CDAT), Celiac Symptom Index (CSI), Patient Reported Outcomes Measurement Information System (PROMIS) domains of depression, anxiety, sleep disturbance, fatigue, pain, and social satisfaction. Chi-square, Fisher's exact and Wilcoxon rank-sum tests were used to identify parameters associated with presence of IBS, our primary outcome.
Results: Our preliminary results show that a third of GAD patients had overlap IBS (Table). These patients had worse anxiety, depression, fatigue, pain, and satisfaction with social interactions compared to the general population. Patient-provider relationships were better for those with overlap IBS compared to patients with only GAD. Subjects with GAD and IBS had only a trend of having more anxiety, depression, fatigue, and pain than those without IBS. Non-adherence to GFD was not more common in patients with overlap IBS.
Conclusion: Patients with GAD and overlap IBS have more psychological distress than the general population, and likely more than GAD patients without IBS. More interestingly, GAD patients share the same prevalence of non-adherence, regardless of the presence of IBS, which suggests that overlap IBS is not the result of advertent nor inadvertent exposure to gluten. Treatment of GAD with IBS overlap should prioritize and assess for the presence of psychosocial comorbidities. Providers may appropriately be monitoring these patients closely, as evidenced by stronger patient-physician relationships reported in the overlap IBS group.

Table 1. Measures associated with presence of IBS

	GAD	GAD + Overlap IBS	*P*-value
Demographics			
Female gender[1]	90.5%	90.0%	1
Age	57.0 (51.0-63.0)	44.0 (36.0-51.2)	0.128
GAD Diagnosis[1]			
Celiac Disease Dermatitis Herpetiformis +/- Celiac Disease Non-Celiac Gluten Sensitivity/Wheat Sensitivity	81.0% 19.0% 0.0%	60.0% 30.0% 10.0%	–[2]
GAD Disease Duration			
	8.0 (2.0-12.0)	4.5 (1.0-10.0)	0.317
IBS Symptom Severity (IBS-SSS)			
	–	344.5 (323.8-377.2)	–
IBS-SSS Categories[1]			
Mild Moderate Severe	– – –	0.0% 16.7% 83.3%	–
GFD Adherence (CDAT)			
	12.5 (10.8-15.2)	13.0 (9.0-14.0)	0.905
Celiac Disease Activity (CSI)			
	35.5 (31.8-37.2)	39.0 (35.0-43.0)	0.106
Relationship with Healthcare Provider (HCCQ)			
	19.0 (10.5-36.5)	39.0 (28.0-42.0)	0.013

Data represent score medians (Q1-Q3) unless noted otherwise. 1(superscript): Reported by frequency. 2(superscript): *P*-value not measured as categories not mutually exclusive. GAD: gluten-associated disorder; IBS: irri**Table** bowel syndrome; IBS-SSS: IBS Symptom Severity Scale; CDAT: Celiac Dietary Adherence Test; CSI: Celiac Symptom Index; HCCQ: Health Care Climate Questionnaire.

S1547

Feasibility of Video Capsule Endoscopy in Patients With Surgically Altered Gastric Anatomy: Propensity-Matched Study

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[1]Cleveland Clinic, Cleveland, OH; [2]Cleveland Clinic Foundation, Cleveland, OH; [3]Central Michigan University College of Medicine, Saginaw, MI.

Introduction: There is very scarce information about feasibility and safety of video capsule endoscopy (VCE) in patients with and without surgically altered gastrointestinal anatomy (SAGIA). We compared the safety and feasibility of VCE in patients with SAGIA and to those with normal anatomy (NA).
Methods: Patients undergoing VCE at our Institution were included (2010-2022) (Table A). Patients with SAGIA undergoing VCE were 2:1 matched based on gender to patients with normal anatomy (NA). SAGIA group comprised of gastrectomy, gastric bypass, enterectomy, colectomy, esophagectomy or Whipple's procedure. Primary outcomes were completion rate, gastric and small bowel transit time, and adverse event rate.
Results: Amongst 9,584 patients undergoing VCE, 77 patients with SAGIA were matched to 154 patients with NA. Complete data was available for 139 patients with NA which comprised the final control group. Gastric bypass was the most common surgery (n=31 [40.3%]) followed by gastrectomy (complete or partial, n=29 [37.6%]). Video capsule was more likely to be placed by swallowing in SAGIA group in comparison with NA group (76.6% vs 38.8%, $P< 0.001$). Patency capsule prior to CE was performed more often in SAGIA group compared to NA group (22.1% vs 5.0%, $P< 0.001$). VCE outcomes are presented in Table B. Study completion rates were similar between SAGIA and NA (97.4% and 95.5%, $P=0.56$). Rates of gastric (1.5% vs 0.7%, $P=0.5$) and small bowel (1.5% vs 37%, $P=0.6$) capsule retention were similar between the SAGIA and NA groups. No patients in either group required intervention for capsule retrieval. Follow up of radiographic imaging was performed more frequently in patients with SAGIA as compared to those with NA (27.3 vs 3.6%, $P< 0.001$).
Conclusion: VCE is safe in patients with SAGIA with comparable completion rates to patients with NA. Patients with SAGIA undergoing VCE are more likely to undergo patency capsule prior to VCE and radiographic imaging post VCE despite the lower frequency of actual capsule retention in patients with SAGIA. This study did not support routine patency capsule or radiographic follow-up in patients with SAGIA.

Table A. Baseline characteristics, type of surgery and indications for video capsule endoscopy in patients with normal anatomy and surgically altered gastrointestinal anatomy (SAGIA)

A. Baseline characteristics, type of surgery and indications for video capsule endoscopy in patients with normal anatomy and surgically altered gastrointestinal anatomy (SAGIA)

Variable	Group	SAGA	Normal anatomy	P value
N		77	139	
Age at the time of VCE		55.58 (15.82)	59.50 (15.87)	0.083
Gender (%)	Female	41 (53.2)	74 (53.2)	1
Race (%)	African/Black	17 (22.1)	40 (29.2)	0.696
	Asian	1 (1.3)	2 (1.5)	
	Caucasian	58 (75.3)	94 (68.6)	
	Hispanic	1 (1.3)	1 (0.7)	
BMI		29.60 (10.11)	28.03 (7.38)	0.191
Capsule placement	Endoscopically placed	18 (23.4)	85 (61.2)	< 0.001
	Swallowed	59 (76.6)	54 (38.8)	
Comorbidities				
HTN (%)		37 (48.1)	113 (81.3)	< 0.001
Autoimmune diseases (%)		4 (5.2)	4 (2.9)	0.46

Table A. (*continued*)

A. Baseline characteristics, type of surgery and indications for video capsule endoscopy in patients with normal anatomy and surgically altered gastrointestinal anatomy (SAGIA)				
Variable	**Group**	**SAGA**	**Normal anatomy**	**P value**
Hypothyroidism. (%)		18 (23.4)	23 16.5)	0.277
Scleroderma. (%)		3 (3.9)	1 (0.7)	0.131
Diabetes (%)		26 (33.8)	60 (43.2)	0.194
Parkinsonism. (%)		0 (0.0)	4 (2.9)	0.299
Indications for VCE				
Abdominal pain (%)		9 (11.7)	3 (2.2)	0.009
Concern for IBD (%)		2 (2.6)	1 (0.7)	0.29
Iron deficiency anemia (%)		23 (29.9)	33 (23.7)	0.335
Occult GI bleeding. (%)		10 (13.0)	2 (1.4)	0.001
Overt GI bleeding. (%)		34 (44.2)	108 (77.7)	< 0.001
Small bowel polyps. (%)		5 (6.5)	0 (0)	0.005
Other. (%)		26 (33.8)	0 (0.0)	< 0.001
Prior Surgeries				
Gastrectomy (%)		29 (37.6)		
Gastric bypass (%)		31 (40.3)		
Enterectomy (%)		7 (9.1)		
Colectomy/ICR (%)		3 (3.9)		
Esophagectomy (%)		1 (1.3)		
Whipple's procedure (%)		6 (7.8)		
B. Outcomes of video capsule endoscopy in patients with normal anatomy vs surgically altered gastrointestinal anatomy (SAGIA)				
Outcomes		**SAGIA**	**Normal anatomy**	**P value**
Small bowel transit time (min)		316.56 (142.63)	310.89 (159.59)	0.82
Total transit time (min)		731.00 (197.08)	773.97 (168.97)	0.141
Result				
Completion		75 (97.4)	128 (95.5)	0.598
Gastric retention at Day 15		1 (1.3)	1 (0.7)	
Small bowel retention at Day 15		1 (1.3)	5 (3.7)	
Follow up imaging (%)		21 (27.3)	5 (3.6)	< 0.001
B: Footnote: Continuous variables are presented as mean (SD) and categorical variables are presented as n (%).				

S1548

GI Dysmotility Symptoms Are Not Associated With Increased L-Dopa Requirements in Parkinson's Disease Patients

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Introduction: Gastrointestinal (GI) motility disorders like gastroparesis or small bowel bacterial overgrowth are thought to impair efficacy of L-dopa, which is absorbed in the proximal small intestine, resulting in motor fluctuations among Parkinson's Disease (PD) patients that prompt dosage increases. While symptoms of dysmotility are frequently reported in PD, results are mixed concerning their effect, if any, on L-dopa pharmacokinetics. In this study, we compare L-dopa equivalent daily dose (LEDD) and motor function among patient with 5 upper GI symptoms to study the possible relationship between dysmotility symptoms and increased L-dopa requirement.
Methods: Two hundred PD patients evaluated by outpatient neurology at Mass General Brigham Healthcare between 2018-2019 were included. History of 5 upper GI symptoms (dysphagia, nausea, vomiting, epigastric pain, and bloating), as well as LEDD were obtained from the medical record. Unified PD Rating Scale (UDPRS) part III motor exam scores off medication were extracted from clinical notes. Differences between LEDD and UDPRS motor scores among patients with and without a history of each of the 5 GI symptoms were calculated via Student t-tests.
Results: Dysphagia was significantly associated with increased LEDD (230mg, $P=0.0067$) as well as increased motor severity (4.6 UDPRS, $P=0.036$). In contrast, nausea was associated with decreased LEDD (-187 mg, $P=0.030$) despite similar motor severity (1.1 UDPRS, $P=0.63$) to those without nausea (Table). There were no differences in LEDD and UDPRS scores among patients with and without vomiting, epigastric pain, and bloating.
Conclusion: Except dysphagia, we found that GI dysmotility symptoms were not associated with increased LEDD nor increased motor severity. This suggests that symptomatic GI dysmotility may not be directly related to poor L-dopa absorption, a common clinical question for PD patients referred to GI. While dysphagia was linked with higher LEDD, this may have stemmed from higher UDPRS scores in this population. Interestingly, those with nausea required lower LEDD despite comparable motor severity. Nausea, a frequent symptom of delayed intestinal transit, may extend absorption time leading to better motor outcomes on less L-dopa. However, further studies are needed to discern any underlying mechanism. Our results nonetheless indicate that clinical management of dysmotility may not reduce motor fluctuations and the need for increased L-dopa as previously presumed.

Table 1. **Changes in L-dopa Requirements and Motor Severity in the Presence of Dysmotility Symptoms**

GI Symptom	ΔLEDD, mg (SD)	*P* value	ΔUDPRS III (SD)	*P* value
Nausea	**-187 (86)**	**0.03**	1 (2)	0.633
Vomiting	11 (95)	0.91	-2 (2)	0.343
Dysphagia	**230 (84)**	**0.007**	**5 (2)**	**0.036**

Table 1. (*continued*)

GI Symptom	ΔLEDD, mg (SD)	*P* value	ΔUDPRS III (SD)	*P* value
Epigastric pain	101 (104)	0.33	0 (3)	0.881
Bloating	62 (99)	0.533	1 (2)	0.764

LEDD—L-dopa equivalent daily dose UDPRS—Unified Parkinson's Disease Rating Scale.

S1549

Somatic Mutations Within the Epitope-Binding Groove DNA Sequence of At-Risk HLA DQA1 and DQB1 Genes Are the Cause of Celiac Disease

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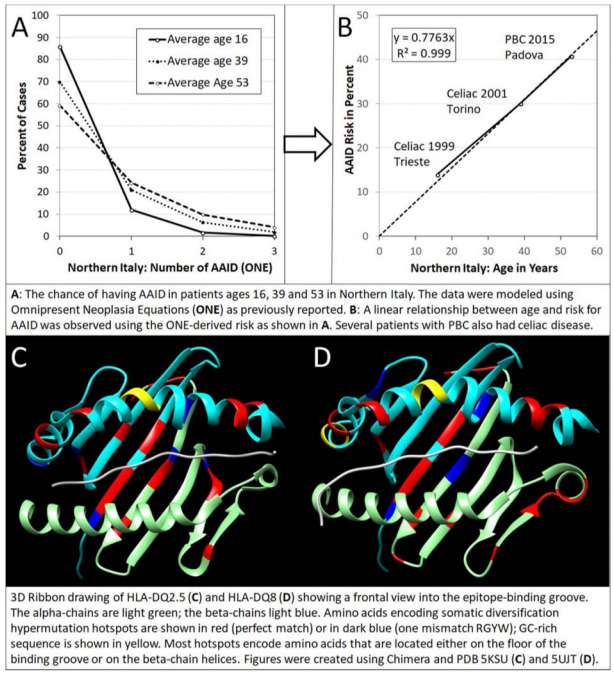
Introduction: Celiac disease (CD) is an inherited autoimmune disease (AID) that occurs after exposure to gluten, is associated with other AIDs and in many persons is associated with the presence of anti-gliadin and -transglutaminase antibodies (TGA). However, despite decades of research the disease mechanism is not understood. We proposed that the key mechanism of AID consists of a constant rate of somatic mutations within the epitope-binding groove of at-risk HLA genes, that are amplified by mutations in other genes identified by GWAS (DDW 2022). We aimed to investigate whether existing epidemiological and genetic data are compatible with somatic DNA mutations causing CD. We assume that one gene causes CD and any additional autoimmune diseases (AAID) in at-risk persons.
Methods: Published data were reviewed for the presence of disease patterns compatible with a constant rate of DNA mutations. HLA haplotypes DQA1*05:01/DQB1*02:01 (DQ2.5) and DQA1*03:01/DQB1*03:02 (DQ8) associated with increased risk for CD were evaluated for DNA sites prone to mutation. GWAS studies were analyzed for (1) HLA; (2) likely autoimmune target; (3) signal amplification factors.
Results: Three studies from northern Italy showed that the number of AID per person adhered to an exponential distribution supporting a constant rate of mutations (Figure A). The chance for an AAID was 0.8% per year per person (Figure B). Analysis of the DQ2.5 and DQ8 haplotypes shows presence of somatic diversification hypermutation hotspots (HH, Figure C, D), i.e., mutation-prone DNA loci, many involving amino acids at the base of the epitope-binding groove. GWAS studies show a strong signal for HLA and lesser signal for amplification factors, but do not show a likely autoimmune target as seen in type 1 diabetes (insulin), Graves' disease (TSHR), and vitiligo (MC1R, OCA2). The Table shows a comparison of GWAS findings.
Conclusion: Existing epidemiological and genetic data are compatible with a somatic DNA mutation mechanism as the cause of CD. The configuration and composition of the peptides causing CD within the mutated epitope-binding groove require a specific baseline configuration – hence only a few DQ alleles are associated with CD risk – and one or more mutations – hence inheritance with incomplete penetrance that depends on the number of HH and the number of mutations required to initiate disease. Transglutaminase is not an autoantigen causing CD but TGA are a result of the gluten-induced chronic inflammatory process of the small bowel mucosa.



A: The chance of having AAID in patients ages 16, 39 and 53 in Northern Italy. The data were modeled using Omnipresent Neoplasia Equations (**ONE**) as previously reported. **B**: A linear relationship between age and risk for AAID was observed using the ONE-derived risk as shown in **A**. Several patients with PBC also had celiac disease.

3D Ribbon drawing of HLA-DQ2.5 (**C**) and HLA-DQ8 (**D**) showing a frontal view into the epitope-binding groove. The alpha-chains are light green; the beta-chains light blue. Amino acids encoding somatic diversification hypermutation hotspots are shown in red (perfect match) or in dark blue (one mismatch RGYW); GC-rich sequence is shown in yellow. Most hotspots encode amino acids that are located either on the floor of the binding groove or on the beta-chain helices. Figures were created using Chimera and PDB 5KSU (**C**) and 5UJT (**D**).

[1549] **Figure 1.** (A, B) Chance of having AAID in patients ages 16, 39, and 53 in Northern Italy (C, D) Ribbon drawing of HLA-DQ2.5 and (D) HLA-DQ8

Table 1. **Our interpretation of a GWAS (FinnGen Release 7) for HLA-mediated AID is as follows**

	Type 1 Diabetes	Graves' Disease	Celiac Disease
Main GWAS signal	HLA	HLA	HLA
Autoantigen GWAS signal	Insulin	TSHR	None?
Amplifier GWAS signal in FinnGen Release 7 (top 3)	Insulin PTPN22 HIST1H2BA	TSHR PTPN22 CTLA4	LPP SH2B3 CCR3
Cases / Controls	7,337 / 255,551	1,421 / 231,654	2,953 / 296,917
Antibodies against GWAS signal	Yes	Yes	No
Germline mutations in autoantigen cause disease like AID	Yes	Yes	No
Symptom prevention	No	No	Gluten-free diet
Tissue affected by AID	Local cell destruction	Local cell activation	Inflammation small bowel mucosa

Table 1. (*continued*)

	Type 1 Diabetes	Graves' Disease	Celiac Disease
Contact with external antigen	No	No	Yes - small bowel
External antigen as cause of AID	No	No	Yes

The key event is the interaction between HLA protein, content of the epitope-binding groove and the T-cell receptor; this event can be changed through mutations in one or more of each component. In HLA-mediated AID, the HLA signal will be dominant. If the key antigen causing AID is a self-antigen, any mutability of the self-antigen may result in a second signal in GWAS (Autoantigen GWAS signal: Type 1 diabetes and Graves' disease); germline mutations in these proteins may cause symptoms like those seen with AID. All other signals represent proteins that can amplify the main HLA signal. If the key antigen is a non-self-antigen as in CD, an obvious autoantigen will not be present on GWAS, and mutations in any of the proteins identified on GWAS do not cause symptoms like those seen in AID.

S1550

Fatty Liver and Metabolic Syndrome in Patients with Celiac Disease: A Systematic Review and Meta-Analysis

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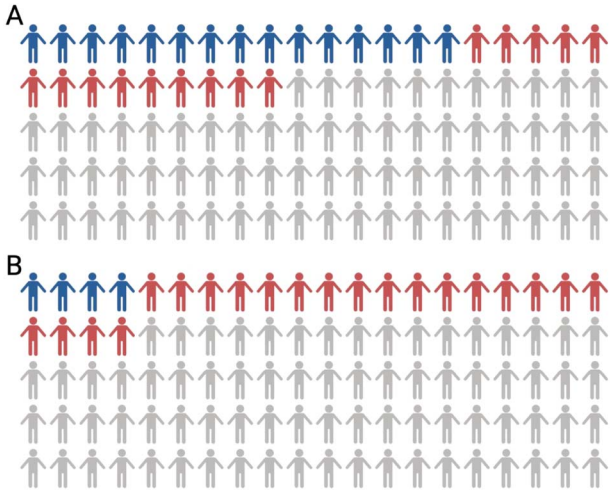
Introduction: Studies have suggested a high prevalence of fatty liver and metabolic syndrome in patients with celiac disease (CeD). We conducted a systematic review and meta-analysis to assess the prevalence of fatty liver and metabolic syndrome in treatment-naïve patients with CeD and in patients on a gluten-free diet (GFD).
Methods: The PubMed, Embase, and the Cochrane Library databases were searched for original studies. We included full-text articles published in the English language after 1990 that used well-defined criteria for CeD, fatty liver and metabolic syndrome. Of 185 studies identified, 7 were included for the analysis. Random effects model was used to calculate pooled prevalence.
Results: The pooled prevalence of fatty liver in treatment-naïve patients with CeD was 15.3% (0.153 [95%CI 0.056-0.285], n=867). After initiation of GFD, the prevalence increased to 29.1% (0.291 [95%CI 0.172-0.427],n=869). The pooled prevalence of metabolic syndrome in treatment-naïve patients with CeD was 4.3% (0.043 [95%CI 0.024-0.067], n=1239), which increased to 24.2% (0.242 [95%CI 0.195-0.293], n=1239) with the initiation of GFD (Figure). We did not observe any significant publication bias. Subgroup analysis was performed to further explain heterogeneity.
Conclusion: The present review has suggested a high prevalence of fatty liver and metabolic syndrome in patients with CeD. With the initiation of GFD, the prevalence of fatty liver and metabolic syndrome increases further. Patients with CeD should thus be screened and monitored for the development of fatty liver and metabolic syndrome. They should be counseled appropriately regarding their diet and inclusion of physical activity in their lifestyle.



[1550] **Figure 1.** Schematic representation of the results: (A) Of 100 treatment-na¨ve patients with celiac disease (CeD), 15 have fatty liver (FL) at baseline (blue). After initiation of a gluten-free diet (GFD), the prevalence of FL increases to 29. (B) Of every 100 treatment-na¨ve patients with celiac disease (CeD), 4 have metabolic syndrome (MS) at baseline (blue). After initiation of GFD, the prevalence of MS increases to 24

S1551

Prevalence and Predictors of Follow-Up Endoscopic Biopsy in Patients With Celiac Disease in the United States

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Introduction: Though endoscopy with small intestinal biopsy is usually required to confirm the diagnosis of celiac disease, the role of follow-up biopsy is less certain. It can be considered if symptoms persist, or for surveillance since persistent villous atrophy may predict disease outcomes. Guidelines do not specifically recommend for or against follow-up biopsy in asymptomatic patients, and the practice patterns regarding follow-up biopsy in the U.S. are poorly understood. We aimed to characterize the frequency and predictors of a second intestinal biopsy in patients with biopsy-diagnosed celiac disease.
Methods: Using the IBM MarketScan Commercial Claims and Encounters database and Medicare Supplemental and Coordination of Benefits database, we identified patients with a diagnosis code for celiac disease and at least one claim for an endoscopic biopsy from 2009-2019. Patients were followed for at least 6 months after the 1st biopsy until they received a 2nd endoscopic biopsy or continuous insurance coverage ended. Predictors of 2nd endoscopic biopsy were assessed using a Cox proportional hazards model.
Results: A total of 30,737 patients were identified. Among them, with a median follow-up time of 22.8 months, 5,976 (19.4%) had a follow-up biopsy. The median time between the initial and follow-up biopsies was 16.8 months. Compared with patients younger than age 20 years, older patients were more likely to have a follow-up biopsy (Table). Frequency of follow-up biopsy was lower in more recent years. Patients with a greater comorbidity burden were more likely to have a follow-up biopsy. Gender and residence in a metropolitan region did not impact the likelihood of follow-up biopsy. Among patients who had a follow-up biopsy, 57.1% had diagnostic codes for celiac disease-related symptoms in the 30 days prior to the procedure.
Conclusion: Among patients with celiac disease, a follow-up biopsy was performed in 19% of cases. The practice is more common in adults than children. There were differences in frequency of follow-up biopsy based on age, comorbidity burden, and geography, but not by gender or urban versus rural residence. Many follow-up biopsies appear to be performed to investigate symptoms rather than as routine monitoring. Further research is needed to explore the impact of follow-up biopsy on outcomes.

Table 1. Multivariate Cox proportional hazards model of variables associated with the performance of a follow-up biopsy (* = $P < 0.05$)

	Adjusted hazard ratio
Age (years)	
≤19	Referent
20-29	1.45 (1.30-1.62)*
30-39	1.49 (1.35-1.64)*
40-49	1.62 (1.49-1.77)*
50-59	1.69 (1.55-1.83)*
60-69	1.76 (1.59-1.94)*
≥70	1.54 (1.36-1.75)*
Year, 1st endoscopic biopsy	
2009	Referent
2010	0.90 (0.81-0.99)*
2011	0.87 (0.79-0.96)*
2012	0.85 (0.77-0.94)*
2013	0.85 (0.77-0.94)*
2014	0.85 (0.76-0.94)*
2015	0.83 (0.74-0.94)*
2016	0.77 (0.68-0.87)*
2017	0.82 (0.72-0.94)*
2018	0.73 (0.62-0.86)*
2019	0.77 (0.55-1.08)
Sex	
Male	Referent
Female	0.98 (0.93-1.04)
Region	
Northeast	Referent
North Central	0.92 (0.86-0.99)*
South	0.98 (0.92-1.04)
West	1.12 (0.81-1.54)
Unknown	0.80 (0.74-0.87)*
Metropolitan statistical area (MSA)	
MSA	Referent
Non-MSA	0.96 (0.88-1.04)
Unknown	0.82 (0.64-1.05)
Elixhauser Comorbidity Index	
0	Referent
1	1.09 (1.01-1.17)*
2 or greater	1.28 (1.20-1.37)*

S1552

Alzheimer's Disease Occurs More Frequently in Patients With Celiac Disease: A Nationwide Population-Based Cohort Study

Andrew Ford, MD[1], Manik Aggarwal, MD[1], Almaza A. Albakri, MD[2], Motasem Alkhayyat, MD[1], Arjun Chatterjee, MD[1], Claire Jansson-Knodell, MD[1], Alberto Rubio Tapia, MD[3].
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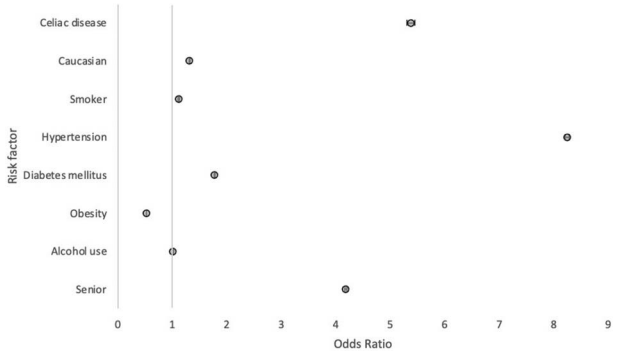
Introduction: Alzheimer disease (AD) affects greater than 5 million Americans and is increasingly prevalent among an aging population. Cognitive decline in general has been linked to chronic inflammation and altered intestinal microbiome. Celiac disease (CD) is an autoimmune enteropathy characterized by immune-mediated damage caused by exposure to gluten. This study was designed to describe the epidemiology and risk association of Alzheimer disease in patients with CD.
Methods: We surveyed a multi-institutional database (Explorys Inc., Cleveland, OH); an aggregate of electronic health record data from 26 major US health systems. Cohorts of patients with AD and CD were generated using Systemized Nomenclature of Medicine – Clinical Terms (SNOMED-CT). AD was defined as SNOMED terms for "Alzheimer's disease" or "Alzheimer's dementia" with prescribed Alzheimer's treatment (donepezil, memantine, galantamine, or rivastigmine). Patients with vascular dementia or Parkinson's disease were excluded. Univariate and multivariate analyses were performed on the data, and associations were reported as adjusted odds ratios (aORs) with 95% confidence intervals (CIs) using IBM SPSS Statistics version 25.
Results: Of 25,298,080 individuals in the database (2017-2022), 88,770 were diagnosed with celiac disease (0.35%). Among CD patients, 970 (1.1%) carried an AD diagnosis compared to 194,030 (0.8%) of the non-CD patients ($P < 0.0001$). After multivariate analysis, CD was associated with increased odds of Alzheimer's disease (aOR = 5.38, 95% CI = 5.30-5.45, $P < 0.0001$) (Figure).
Conclusion: This large population-based cohort study demonstrated a significantly higher odds of Alzheimer disease among celiac disease patients compared to those without celiac disease (Table). Larger prospective work would be beneficial to identify possible mechanisms. Further work may examine possible interventions to minimize the odds of developing AD in this population and to assess the effect of gluten-free diet adherence on its development.



[1552] **Figure 1.** Risk factors for development of Alzheimer's disease

Table 1. Baseline characteristics of patients with celiac disease

Celiac Disease	With Alzheimer's Disease (n [%])	Without Alzheimer's Disease (n [%])	Multivariate Analysis (aOR [95% CI])
Total	970	87800	5.38 (5.30-5.45)
Risk factor			
Alcohol use	40 (4.1)	2900 (3.3)	1.01 (1.00-1.02)
Obesity	180 (18.6)	30510 (34.7)	0.53 (0.53-0.53)
Diabetes mellitus	390 (40.2)	29430 (33.5)	1.77 (1.77-1.78)
Hypertension	810 (83.5)	38880 (44.3)	8.25 (8.21-8.28)
Smoker	290 (29.9)	10460 (11.9)	1.12 (1.11-1.13)
Caucasian	890 (91.7)	71120 (81.0)	1.31 (1.31-1.32)
Senior (Age >65)	890 (91.7)	33050 (37.6)	4.18 (4.17-4.20)

S1553

Association of EoE and Celiac Disease in a National VA Database

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Introduction: Conflicting evidence exists regarding the association between Eosinophilic Esophagitis (EoE) and Celiac Disease, leaving clinicians questioning whether it is necessary to perform small bowel biopsies during endoscopy for patients with EoE. We sought to assess the relationship between EoE and Celiac Disease in a large population-based study to better assess the utility of this procedure in this setting.
Methods: We performed a nationwide, retrospective case control study using patient data from the Department of Veterans Affairs electronic health records (EHR). Using ICD9 and ICD10 codes, patients with EoE (530.13, K20.0) and Celiac Disease (579.0, K90.0) were identified. The prevalence of EoE in Celiac Disease and the prevalence of Celiac Disease in EoE, as well as odds ratios of the respective conditions, were determined.
Results: Using a random sample of 1,110,189 VA patients, we identified 1,022 cases of EoE (0.092%) and 1,285 cases of Celiac Disease (0.116%), where the prevalence of Celiac Disease within the EoE cohort was found to be 1.08% (n=11), reflecting an elevated odds of Celiac in EoE (OR= 9.46, 95% CI: 4.89,16.32) (Table). Additionally, the prevalence of EoE within the Celiac cohort was 0.86%, which also indicated an elevated odds of EoE in Celiac Disease (OR= 9.46, 95% CI: 4.89,16.32) (Table). Adjusting for race, ethnicity, and gender resulted in a similar OR (OR=8.66, CI: 4.47, 14.95). When stratified by race and ethnicity, we found that all patients with both EoE and Celiac Disease were White and non-Hispanic. When stratified by gender, 10 of the 11 patients with both EoE and Celiac Disease were found to be male.
Conclusion: The results of our study show that there is an increased odds of Celiac Disease within EoE and vice versa in VA patients when compared to the general population. These results suggest that in EoE and Celiac patients, Celiac Disease and EoE should be actively looked for and that there should be a low threshold to biopsy, especially if there are any symptoms or mucosal changes that are suggestive of disease. However, further research into the association of Celiac Disease and EoE with prospective studies are needed.

Table 1. Prevalence of EoE and Celiac Disease among patients in the VA EHR

	Patients with EoE	Patients without EoE
Patients with Celiac Disease	11	1,274
Patients without Celiac Disease	1,011	1,107,893

S1554

Rectal Evacuation Disorder Associated With a Higher Rate of Small Intestinal Bacterial Overgrowth Diagnosis Compared to Slow Transit Constipation

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Introduction: Small intestinal bacterial overgrowth (SIBO) is being more and more recognized as a potential comorbidity in patients with constipation, likely causing additional symptoms, and posing as a challenge to the management of symptoms in this patient population. We aimed to compare the rate of SIBO in patients with Rectal Evacuation Disorder (RED) to that in patients with slow transit constipation (STC).
Methods: The electronic medical records of patients aged 18 or more were screened for the diagnoses of RED and STC between year 2015 and present at a tertiary care center in Northern California. We then screened the results for a diagnosis of SIBO based on a hydrogen breath test and an established SIBO diagnosis within one year. Chi-squared statistic was used to compare the rate of SIBO in patients with RED to that in patients with STC.
Results: 319 patients were identified with RED, of whom 256 (80.3%) were female. 954 patients were identified with STC, of whom 597 (62.6%) were female. The mean age (sd) was 57.7 (15.9) in RED and 58.7 (19.9) in STC. The rate of a SIBO diagnosis was significantly higher in patients with RED (7.84%) compared to patients with STC (1.78%) (P=0.000081).

Conclusion: RED is associated with an increased rate of a SIBO diagnosis compared to slow transit constipation. Untreated RED may offer one explanation for the recurrent nature of SIBO in patients with constipation, and RED should thus be suspected and screened for in patients with constipation who are diagnosed with SIBO. Prospective research is needed to better understand.

S1555

Perspectives, Experiences, and Concerns Among Patients With Short Bowel Syndrome (SBS) and Their Caregivers: Insights From Social Media Platforms

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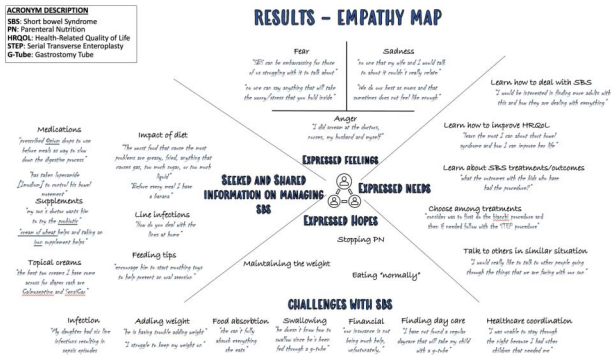
Introduction: SBS is a rare, chronic and severe disease resulting from physical loss and functional deficiency of portions of intestine, primarily due to surgical resection. Patients experience a collection of clinical features such as diarrhea, bloating, and weight loss, and frequently need parenteral nutrition (PN), which significantly impacts their health-related quality of life (HRQOL). In this study, we analyzed social media posts to examine the perspectives, experiences, and concerns among patients with SBS and their caregivers.
Methods: We extracted 20,221 publicly available posts from SBS-specific sites and other forums (e.g. Careplace, Reddit) between 1/1/10-1/11/21. After applying SBS keyword filters (e.g. short bowel, short gut, SBS) and manual review, we identified 316 relevant posts. We used an open coding technique to qualitatively analyze the posts. Posts were read multiple times and codes were generated inductively, sorted, and grouped into themes and subthemes. Afterwards, we developed an empathy map to visualize the different themes and subthemes.
Results: Overall, 70.6% and 14.9% of posts were made by caregivers and patients, respectively; the source for 14.6% of posts was unclear. See Figure for empathy map. Forums were used to seek and share information on how to manage SBS including medication and supplement use, diet, infections, rashes, and feeding tips. Challenges associated with SBS were discussed, such as financial struggles, finding daycare for kids with SBS, coordinating with healthcare providers, managing infections related to PN, gaining weight, absorbing food, and swallowing issues. The need for support was expressed, such as feeling lonely and not understood. There were reports about needing to learn about SBS treatments, how to choose among treatment options, how to handle SBS associated conditions, and how to improve their HRQOL. Common feelings expressed were fear, sadness and anger, and common hopes expressed were stopping PN, maintaining weight, and eating normally.
Conclusion: Our analysis supports previous evidence on the marked burden SBS has on physical, social, and mental health for both patients and caregivers. Additionally, this analysis highlights how social media platforms can be used to derive insights on the perspectives, experiences and concerns in a rare disease such as SBS.



[1555] **Figure 1.** Empathy map

S1556

Enteropathy in Primary Immunodeficiency Diseases: A Systematic Review of Cases

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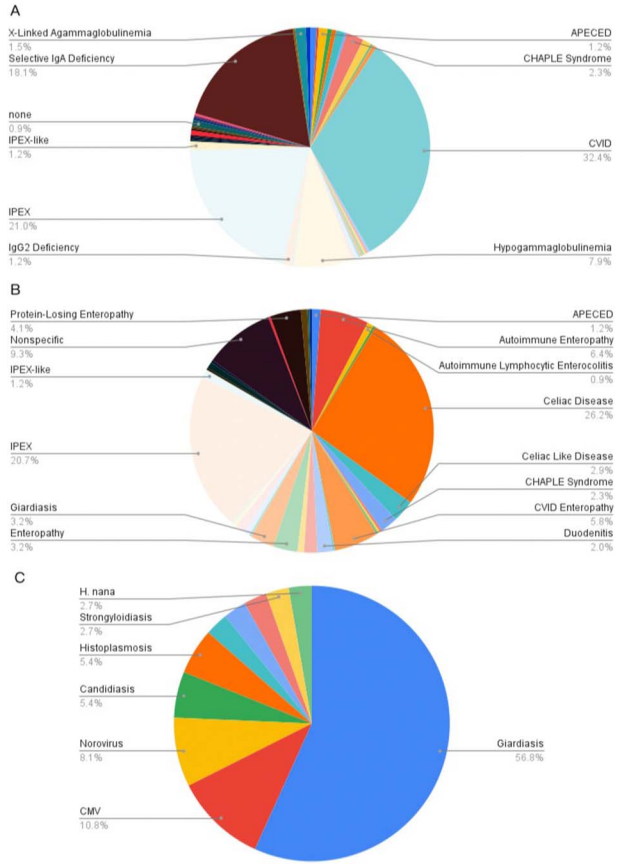
Introduction: Inborn errors of immunity are a group of primary immunodeficiency disorders caused by over 400 genetic defects. Enteropathy has been common in PID patients, which presents with chronic diarrhea, malabsorption, growth delay, iron deficiency, and failure to thrive. This article systemically reviewed the clinical presentations, treatments, and genetic defects of enteropathy observed in PIDD.
Methods: We have reviewed published cases with the clinical diagnosis of both enteropathy and PIDD using 3 databases (Pubmed, Scopus, EMBASE). A total of 346 cases met our inclusion criteria.
Results: The most common enteropathy-associated PIDD is common variable immunodeficiency (32.4%), IPEX (21%), selective IgA deficiency (18.1%), and hypogammaglobulinemia (7.9%; Figure). Celiac disease (26.2%) is the most common enteropathy presentation in PIDD, followed by IPEX (20.7%), autoimmune enteropathy (6.4%), and CVID enteropathy (5.8%). Selective IgA deficiency and CD/Celiac like disease were also frequently reported. More than half of documented PIDD-related CD showed positive serology test results and histopathological findings. Eighty-eight percent of PIDD-related CD cases are responsive to a gluten-free diet. FOXP3 mutation (70) was the most common gene mutation in PIDD, followed by *CTLA-4* (17), *CD55* (8), *NFKB1* (8), *GoF-STAT1* (5), *GoF-STAT3* (5), and *C1-INH* (4; Table)). *CTLA-4* mutation was found related to CVID, hypogammaglobulinemia, and autoimmune enteropathy. NFKB1was found mainly linked to CVID. We observed frequent giardiasis (21), norovirus (3), CMV (4), Candidiasis (2), and histoplasmosis (2) infections causing enteropathy in PIDD. No significant difference in treatments of the enteropathy between PIDD and non-PIDD was noticed.
Conclusion: Enteropathy can be common clinical presentations in IEIs. With early recognition of clinical manifestations and enteropathy-associated gene mutation, PIDD can be diagnosed and treated timely, preventing complications and mortalities.



[1556] **Figure 1.** A. The common enteropathy presentations in PIDD B. The common PIDD associated with enteropathy C. The common infectious enteropathy in PIDD

Table 1. The most common gene mutations associated with enteropathy in PIDD

Gene Mutation	Case Number
FOXP3	70
CTLA-4	17
CD55	8
NFKB1	8
GoF-STAT1	5
GoF-STAT3	5
C1-INH	4
AIRE	3
LRBA	3
BTK	2
TTC37	2
WASP	2
ATM	1
BACH2	1
CD25	1
CD3 gamma	1
CYBB	1
DOCK8	1
F12-NG	1
ITBG2	1
MEFV	1
NCF2	1
NLRP12	1
PIK3CD	1
RAG1	1

Table 1. (*continued*)

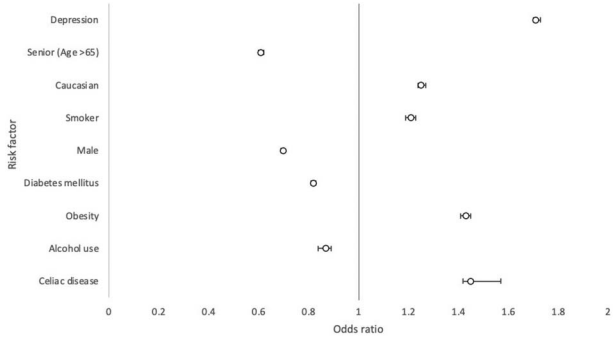
Gene Mutation	Case Number
RMRP	1
SAMD9	1
KMT2D	1
TACI	1
TTC7A	1

S1557

Migraine Disorder Occurs More Often in Celiac Disease Patients: A Nationwide Population-Based Cohort Study

Andrew Ford, MD[1], Manik Aggarwal, MD[1], Motasem Alkhayyat, MD[1], Almaza A. Albakri, MD[2], Abel Joseph, MD[1], Claire Jansson-Knodell, MD[1], Alberto Rubio Tapia, MD[3].
[1]Cleveland Clinic Foundation, Cleveland, OH; [2]Royal Jordanian Medical Services, Amman, Al Karak, Jordan; [3]Cleveland Clinic, Cleveland, OH.

Introduction: Migraine disorder is a relatively common condition affecting nearly 15 percent of the general population. This disorder has been linked to multiple gastrointestinal diseases including celiac disease (CD), an autoimmune enteropathy characterized by immune-mediated damage resulting from gluten exposure. We aimed to further characterize the risk and epidemiologic association between CD and migraine disorder with this study.
Methods: We queried a multi-institutional database (Explorys Inc., Cleveland, OH); an aggregate of electronic health record data from 26 major US health systems. We formed cohorts of patients with CD and migraine disorder using Systemized Nomenclature of Medicine – Clinical Terms (SNOMED – CT). Migraine disorder was defined as SNOMED terms for "Migraine disorder" with prescribed migraine treatment (drug class: "migraine treatment"). Patients with diagnoses of cluster or tension headaches were excluded. Univariate and multivariate analyses were performed on the data, and associations were reported as adjusted odds ratios (aORs) with 95% confidence intervals (CIs) using IBM SPSS Statistics version 25.
Results: Of 30,142,740 adult patients in the database between 2017-2022, 91,390 (0.30%) carried a celiac disease diagnosis. Among celiac disease patients, 6,360 (6.9%) carried a migraine disorder diagnosis compared to 474,360 (1.6%) of the non-CD patients ($P < 0.0001$). After multivariate analysis, celiac disease was associated with an increased odds of migraine disorder (aOR = 1.49; 95% CI 1.42-1.57; $P = < 0.0001$) (Figure, Table).
Conclusion: This large population-based cohort study demonstrated significantly higher odds of migraine disorder among patients with celiac disease compared to patients without the disease. Our findings are in line with those from previous observational studies showing the same. Larger prospective studies would be beneficial in identifying the role of gluten exposure in this association.



[1557] **Figure 1.** Risk factors for development of migraine disorder

Table 1. Prevalence of risk factors among celiac disease patients with respective adjusted odds ratios (aORs) by multivariate analysis.

Celiac Disease	With Migraine Disorder (n [%])	Without Migraine Disorder (n [%])	Multivariate Analysis (aOR [95% CI])*
Total	6360	85030	1.45 (1.42-1.57)
Risk factor			
Alcohol use	240 (3.77)	1590 (1.87)	0.87 (0.84-0.89)
Obesity	2230 (35.1)	18230 (21.4)	1.43 (1.41-1.45)
Diabetes mellitus	390 (22.3)	17680 (20.8)	0.82 (0.81-0.83)
Male	560 (8.81)	18780 (22.1)	0.70 (0.69-0.70)
Smoker	720 (11.3)	5040 (5.93)	1.21 (1.19-1.23)
Caucasian	5720 (89.9)	56830 (66.8)	1.25 (1.24-1.27)
Senior (Age >65)	920 (14.5)	19550 (23.0)	0.61 (0.60-0.62)
Depression	3850 (60.5)	24840 (29.2)	1.71 (1.70-1.73)

*$P < 0.0001$ for all aORs.

S1558

Risk of *Clostridioides difficile* Infection in Patients With Celiac Disease: Insight From a U.S.-Based Population Study

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Introduction: Little is known about the association between Clostridioides difficile infection (CDI) and celiac disease. The aim of the study is to describe the risk of CDI in patients with celiac disease and to describe the clinical outcomes in patients with celiac and CDI.
Methods: We queried a commercial database (Explorys IncTM, Cleveland, OH, United States), and obtained an aggregate of electronic health record data from 26 major integrated United States healthcare systems comprising 360 hospitals in the United States from 2017 to 2022. Diagnoses were organized into the Systematized Nomenclature of Medicine Clinical Terms (SNOMED–CT) hierarchy. We compared

the incidence of new CDI among patients with celiac disease versus those without celiac disease (controls). Univariate and multivariate analyses were performed on the data, and associations were reported as adjusted odds ratios (aOR) with 95% confidence intervals (CI).

Results: We identified 90,060 patients with celiac disease and 25,807,720 controls. The incidence of new CDI was 1.31% (1,180) in celiac disease patients, and 0.35% (92,330) in controls, yielding an odds ratio (OR) of 3.69 (95% CI 3.49-3.91; $P < 0.0001$). Treatment and clinical outcomes were not significantly different. After controlling for common CDI risk factors, the multivariate analysis model uncovered that celiac patients were more likely to develop CDI OR: 1.34 (95% CI: 1.2-1.4, $P < 0.0001$) compared to controls (Table).

Conclusion: In a large US population-based study, patients with celiac disease had a significantly higher incidence of CDI than controls. Clinical outcomes were similar between the groups. Clinicians should be vigilant and consider CDI in celiac patients with new or recurrent diarrhea.

Table 1.

Demographics	Celiac Patients (n=90,060)	Control Patients (n=25,807,720)
Age 18-65 years (%)	65,220 (72.4%)	18,331,970 (71%)
Age >65 years (%)	24,840 (27.6%)	7,475,750 (29%)
Female (%)	66,800 (74.2%)	13,219,060 (51.2%)
Race: White (%)	73540 (81.7%)	14,072,590 (54.5%)
Smoker (%)	15,120 (16.8%)	2,327,950 (9%)
Alcohol abuse (%)	2,530 (2.8%)	552,010 (2.1%)
Hypertension (%)	42,250 (46.9%)	8,063,140 (31.2%)
Diabetes (%)	25,560 (28.4%)	3,446,990 (13.4%)
Obesity (%)	26,170 (29.1%)	3,535,170 (13.7%)
Clostridioides difficile infection management and clinical outcomes		
Developed Clostridioides difficile infection*	1,180 (1.31%)	92,330 (0.35%)
Treated with metronidazole	820 (69%)	56,490 (61.1%)
Treated with vancomycin	770 (65.2%)	55,270 (59%)
Treated with fidaxomicin	70 (5.9%)	4,370 (4.7%)
Colectomy**	10 (0.84%)	970 (1.05%)
Multivariable model with Clostridioides difficile infection being the outcome		
Risk Factors	Odds Ratio	95% CI; *P-value*
Age ≥65 yr vs < 65yr	2.988	2.962-3.014; < 0.0001
Gender (female vs male)	1.194	1.184-1.204; < 0.0001
Race (White vs rest)	1.483	1.467-1.498; < 0.0001
Antibiotics	3.657	3.6-3.7; < 0.0001
Acid suppressive therapy***	4.268	4.224-4.313; < 0.0001
Inflammatory bowel disease	6.031	5.93-6.13; < 0.0001
Celiac disease	1.342	1.284-1.403; < 0.0001

*1st occurrence of CDI after celiac diagnosis.
**Colectomy performed within 60 days of CDI diagnosis (excluded patients with a history of IBD, ischemic colitis, and neoplasia of the colon).
***Acid suppressive therapy includes prior use of proton pump inhibitors or H2 blockers

S1559

Reduction of Video Capsule Endoscopy Reading Times Using Artificial Intelligence

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Introduction: Video capsule endoscopy (VCE) is an innovation that has revolutionized care within the field of gastroenterology, but the time needed to read the studies generated has often been cited as an area for improvement. The purpose of this study is to create a machine learning model capable of significantly reducing capsule endoscopy reading times.

Methods: In this study, we have trained a convolutional neural network, ResNet50, using videos from the KID dataset to confidently exclude normal images on VCE, while retaining abnormal ones. The CNN was exposed to 3 full length capsule endoscopy videos A threefold cross-validation scheme was employed whereby the model was trained using 2 of the aforementioned videos, tested on the third and this was repeated for all possible video combinations. We trained our model for 9 epochs and further improved the predictions of our model by adding rotated versions of abnormal segments to our training data.

Results: Our study identified abnormal frames in 3 KID videos as outlined in the Table. We were able to reduce the video length by 47%, on average, and captured frames from 118 of the 119 abnormal segments labeled by the expert physician. We were able to develop an algorithm that successfully detected 99% of abnormal segments while reducing the reading time for a physician by over 43%.

Conclusion: Our model demonstrated high levels of accuracy with significant reduction in physician reading time. Our results are reassuring and demonstrate the benefit of CNN in processing VCE images. We believe our study lays an excellent foundation for further validation in large multicenter trials.

Table 1. Description of the various characteristics of the of the 3 KID videos in addition to the reduced number of frames produced and abnormal segments detected by the ResNet50 model

Video Name	Total Number of Frames	Number of Abnormal Segments	Reduced Number of Frames by ResNet50	Number of Abnormal Segments Detected by ResNet50
KID Video 1	28480 (96 min)	22	14828 (49 min)	22
KID Video 2	117565 (391 min)	86	84672 (282 min)	86
KID Video 3	74762 (249 min)	11	27099 (90 min)	11

S1560

Small Intestinal Bacterial Overgrowth Among Persons Living With HIV

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Introduction: Chronic gastrointestinal symptom management remains a priority among persons living with HIV (PLWH) despite advances in HIV antiretroviral therapy (ART). Diarrhea, abdominal discomfort, and weight loss are commonly reported by PLWH and negatively impact quality-of-life. In longitudinal studies, these symptoms remain prevalent over follow-up despite ART initiation, suggesting a small bowel pathology not addressed by HIV suppressive therapy. Small intestinal bacterial overgrowth (SIBO) manifests similar symptomatology and is associated with immunodeficiency, yet data on SIBO/HIV co-prevalence are lacking in the literature. We hypothesized that HIV infection increases the risk for SIBO and that markers of HIV disease progression further enhance SIBO risk.
Methods: In a pilot, retrospective cohort study, we abstracted electronic medical record data from symptomatic patients who underwent hydrogen-dextrose breath testing (HBT) for evaluation of SIBO at the Atrium Health Wake Forest Baptist Digestive Health Clinic. We included adults with an established HIV diagnosis (N=29) prior to HBT. Controls were matched 2:1 (N=58) for relevant demographic and historical factors.
Results: In the HIV group, viral RNA was < 20 copies/mL in 89% (N=25/28) and peripheral CD4+ counts >300/μL in 96% (N=22/23) within 3-6 months of HBT. A SIBO diagnosis was made in 41.4% of the HIV group compared to 31.0% of controls (RR: 1.33, P=.340). Bloating, excessive gas, and abdominal cramping were the 3 most common symptoms in each group. Symptoms failed to cluster by group in a principle-components redundancy analysis (P=.286). Breath methane was reduced in the HIV vs control group (P=.019) while hydrogen was similar (P=.577). In the HIV group, mean viral RNA and CD4+ counts were 7 and 28 copies/mL (P=.418) and 861 and 665 cells/μL (P=.280) among SIBO(-) and SIBO(+) subjects, respectively. Breath methane was inversely associated with CD4+ counts (P=.004) and hydrogen was not (P=.497).
Conclusion: In this pilot, HIV infection was controlled and trended towards an enhanced SIBO risk without reaching statistical significance. Decreased breath methane was observed among PLWH with clinically stable CD4+ counts, suggesting that small bowel dysbiosis accompanies controlled infection. In this limited sample, SIBO was as prevalent in symptomatic PLWH as in controls. An awareness of SIBO among PLWH may broaden therapeutic options for chronic symptom control.

S1561

Food Is Medicine: A Novel Educational Intervention to Improve Nutritional Competency in Graduate Medical Education

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Introduction: Nutrition plays a critical role in the pathogenesis of many chronic conditions, and thus it is essential that physicians and trainees possess a strong knowledge base of key nutritional concepts. This is especially relevant to gastroenterologists, who care for patients at high risk for developing nutritional abnormalities due to complications of their gastrointestinal diseases. However, physicians receive minimal nutritional instruction throughout their medical training due to curricular and time constraints and often feel unprepared to effectively counsel patients on key aspects of nutrition. This study aimed to evaluate resident physicians' attitudes toward nutrition and to improve competency in nutritional counseling using a novel educational intervention.
Methods: We implemented a novel nutrition training intervention, aimed at senior internal medicine and internal medicine-pediatrics residents. A scripted presentation was developed on fundamental nutritional concepts and evidence-based nutritional considerations on topics deemed highest priority by the American Society for Clinical Nutrition Committee on Medical/Dental School and Residency Nutrition Education. Resident physicians were surveyed using an abbreviated version of a validated nutrition survey and questionnaire evaluating attitudes on nutrition and objective nutritional competence before and after receiving the presentation. Comparison of pre- and post-intervention scores was made by 2-sample t-test.
Results: Among the 39 resident physicians who completed the survey, 85% believed physicians are not adequately trained to counsel patients on nutrition, and 95% expressed the desire to improve their competency in nutrition. Mean scores of objective nutritional competence were significantly improved after administration of the nutritional intervention (63.6% ±11.8 vs 72.2% ±11.5; P=0.017, CI -3.29 to -0.34), as was subjective confidence in counseling patients on all surveyed nutritional concepts (Table).
Conclusion: Our study suggests that resident physicians value nutritional education and desire to improve their knowledge of nutrition, but the majority are not satisfied with the current quality of nutrition training. Our novel educational intervention is a simple, efficient method of improving objective nutritional competency among resident physicians that can be easily implemented while respecting time constraints associated with graduate medical training.

Table 1. Basic participant demographics as well as observed measures of subjective confidence and objective competence in nutritional counseling

	Baseline Participant Demographics	
	Pre-Intervention Survey Completion	**Post-intervention Survey Completion**
PGY2	22	12
PGY3	14	4
PGY4	2	1
Male	29	12
Female	10	6
Objective Competence Scores		
Pre-Intervention (n=39)	Post-intervention (n=18)	*P*-Value
63.60%	72.20%	0.0169
Measurement of Subjective Confidence in Nutritional Counseling		
Nutritional Topic	Pre-Intervention (n=39)	Post-Intervention (n=18)	*P*-Value
Food constituents	2.85	4.12	< 0.0001
Body mass index	3.08	4.06	0.0055
Basic metabolism	3.46	4.67	0.0007
Vitamins	3.00	4.17	0.0003
Omega-3/6 fats	3.11	3.72	0.0409
Dietary cholesterol	3.55	4.28	0.0103
Type 2 diabetes	3.90	4.50	0.0033
Osteoporosis	3.26	4.00	0.0032
HIV	2.08	3.89	< 0.0001

Objective competence was determined utilizing 22 questions from a validated nutrition assessment survey, and scores are reflected as percentage correct. Subjective confidence was determined using a validated questionnaire assessing comfort level and knowledge of nutritional interventions in commonly-encountered diseases in primary care, and was scored on a scale of 1-5 from least to most comforTable, respectively. HIV - human immunodeficiency virus. PGY – post-graduate year.

S1562

Subclinical Celiac Disease in the U.S. Hispanic Population: Results From the Los Angeles County Department of Health Services

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Introduction: Anemia is a common extraintestinal manifestation of celiac disease (CeD). Although perceived as a predominantly White disease with a 1% prevalence, CeD also affects other underreported populations, such as the Hispanic population. We aimed to estimate the prevalence of CeD in Hispanic patients presenting with unexplained anemia.
Methods: A cross-sectional study was completed through a clinical management database and electronic medical record after receiving Institution Review Board approval. Adult Hispanic patients with unexplained anemia who underwent upper endoscopy with duodenal biopsies between 2013 and 2020, were included. This study was conducted at the Los Angeles County Department of Health Services, a safety-net public health care system with a predominantly Hispanic (65%) population. Electronic medical records were queried for the diagnosis of biopsy-proven CeD.
Results: Two hundred six subjects underwent upper endoscopy for unexplained anemia, of which 61 were excluded (18 underwent an upper endoscopy without biopsy, 7 were non-Hispanic, and 36 patients had normal hemoglobin levels; Table). Among all Hispanic patients referred for endoscopic evaluation of unexplained anemia (N = 145), the mean age was 53.9 years, and 66% were female. The overall prevalence of biopsy-proven CeD was 4.8% (N = 7/145). In patients with iron-deficiency anemia (IDA) specifically, the prevalence of CeD was 4.5% (N = 3/67). Of the 7 patients with confirmed CeD, only one in retrospect had associated diarrhea. None of the patients had a family history of CeD. Interestingly, only one of 5 tested patients had positive IgA tissue transglutaminase antibody, and the remainder were seronegative. Other CeD-related laboratory testing including vitamin B12 and liver function tests were unremarkable; thyroid stimulating hormone (TSH) levels were within normal range except for one patient with elevated levels due to poorly controlled hypothyroidism.
Conclusion: The prevalence of CeD in adult Hispanic patients with unexplained anemia (unspecified or IDA) is similar to the prevalence in White patients, a finding not previously reported in the literature. Screening for CeD in patients with unexplained anemia in general, and IDA specifically, is of value in Hispanic patients as well as White ones. Seronegative CeD requires endoscopic evaluation with duodenal biopsies for diagnosis.

Table 1. Clinical and Lab Characteristics of Patients with Biopsy-Confirmed Celiac Disease tTG: IgA tissue transglutaminase within <4 months from index endoscopy; TSH: thyroid stimulating hormone; M: male; F: female; AST: aspartate aminotransferase; ALT: alanine transaminase

ID	Hemoglobin (Ferritin)	AST/ALT	Total Bilirubin	IgA tTG	Vitamin B12	TSH	Other Comorbidities
49 F	8.5	33/17	0.5	None	652	12.8	Hypothyroidism, Rheumatoid arthritis on sulfasalazine and methotrexate
42 F	11.6 (70.7)	None	None	< 1	596	None	Refractory GERD s/*P* fundoplication
57 F	8.4 (7.6)	42/51	0.6	>100	NA	None	Hypothyroidism, Chronic hepatitis C
34 F	11.7	16/12	0.6	< 1	325	None	None
40 M	10.7	29/40	0.6	None	481	0.83	Substance abuse, Coronary artery disease with NSTEMI, Osteoporosis
50 F	9.9 (5.4)	22/23	0.5	< 1	291	2.1	Stage II breast cancer
44 F	9.5 (3.1)	29/33	0.6	< 1	NA	1	Type II diabetes

S1563

ACE Inhibitor Use Is Associated With Angioectasias on Video Capsule Endoscopy (VCE) in Patients With Iron Deficiency Anemia

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Introduction: Small bowel bleeding poses a clinical challenge in patients with vascular lesions. Bleeding recurrence after endoscopic therapy is high and medical therapies have limited efficacy. In those with left ventricular assist devices (LVADs), the use of angiotensin converting enzyme inhibitors (ACEi) has been associated with a reduced rate of gastrointestinal bleeding. Conversely, the antiplatelet effects of ACEi have been described thought due to their effects in decreasing platelet aggregation. Our aim was to determine whether ACEi or angiotensin receptor blocker (ARB) therapy had an impact on the rate of positive VCE small bowel findings in those with iron deficiency anemia (IDA).
Methods: Data was collected from consecutive inpatient and outpatient VCE examinations performed between January 1, 2009 to March 1, 2018 at a single U.S. tertiary medical center performed for IDA. VCE studies were excluded in those with incomplete small bowel examinations (9) or technical failure (1). Patient demographics, laboratory values, medication use and endoscopic findings were recorded. VCE findings were based on the P0-P2 grading system. The primary outcome of interest was a positive (P2) VCE. Data were analyzed using Wilcoxon rank-sum test for continuous variables and Fischer's exact test for dichotomous variables. Bivariate logistic regression was performed to identify independent factors predictive of positive VCE.
Results: Eighty-two VCE were included in the final analysis. Median age was 67.6 (IQR: 57.1-75.5) years and 38 (46.3%) patients were male. Thirty-seven (45.1%) VCE examinations were positive with the most common finding of angiectasia in 21 (55.3%). Risk factors for positive VCE are listed in Table 1. In univariate analysis, ACEi therapy associated with both positive VCE (P=0.014) and the presence of angiectasia (P=0.021), whereas ARB therapy did not associate with positive VCE. ACEi findings remained significant in bivariate analysis after controlling for cardiac and pulmonary comorbidities.
Conclusion: In this single center study the presence of ACEi use was associated with small bowel and/or stomach angiectasias on VCE in univariate analysis and when controlled for cardiac and pulmonary risk factors. Further studies are required to determine the mechanistic impact of ACEi on gastrointestinal bleeding risk such as the reduction in von Willebrand factor or factors that affect platelet aggregation.

Table 1. Univariate predictors of positive video capsule endoscopy in iron deficiency anemia

	Negative VCE (n = 45)	Positive VCE (n =37)	*P*-value
Age, years, mean (SD)	63.6 (15.8)	68.6 (9.3)	0.303
Male sex, n (%)	21 (46.7)	17 (46)	1
White race, n (%)	22 (48.9)	20 (54.1)	0.223
Active smoking, n (%)	6 (14)	11 (29.7)	0.105
Outpatient location, n (%)	31 (68.9)	21 (56.8)	0.357
Chronic kidney disease, n (%)	10 (22.2)	13 (35.1)	0.224
Chronic liver disease, n (%)	3 (6.7)	4 (10.8)	0.696
Congestive heart failure, n (%)	10 (22.2)	13 (35.1)	0.224
Diabetes mellitus, n (%)	15 (33.3)	15 (40.5)	0.645
Cardiac valvular disease, n (%)	7 (15.6)	6 (16.2)	1
Coronary artery disease, n (%)	14 (31.1)	14 (37.8)	0.641
Cardiac arrhythmia, n (%)	**10 (22.2)**	**18 (48.7)**	**0.019**
Chronic lung disease, n (%)	10 (22.2)	13 (35.1)	0.224
Left ventricular assist device, n (%)	2 (4.4)	2 (5.4)	1

Table 1. (*continued*)

	Negative VCE (n = 45)	Positive VCE (n =37)	*P*-value
Blood transfusion in the preceding 4 weeks, n (%)	17 (37.8)	16 (43.2)	0.656
Aspirin, n (%)	20 (44.4)	14 (37.8)	0.654
Thienopyridine, n (%)	3 (6.7)	2 (5.4)	1
Coumadin, n (%)	**1 (2.2)**	**5 (13.5)**	**0.086**
SSRI/SNRI, n (%)	7 (15.6)	9 (24.3)	0.404
ACEi	**7 (15.6)**	**15 (40.5)**	**0.014**
ARB	9 (20)	7 (18.9)	1
Hemoglobin, g/dL[1]	9.9 (1.7)	9.2 (2.3)	0.216
Platelets, 10^9/L[1]	225.4 (67.5)	208.6 (84.9)	0.202
BUN, mg/dL[1]	21.9 (13.1)	26.7 (22)	0.869
Creatinine, mg/dL[1]	1.3 (0.5)	1.9 (1.5)	0.197
Ferritin, ng/mL,[1]	128 (259)	39.6 (28.7)	0.966
Percent saturation, %[1]	14.8 (15.1)	13.7 (15.4)	0.76

S1564

Association of SIBO Symptoms with Breath-Test Results and Response to Treatment

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Introduction: Small intestinal bowel overgrowth (SIBO) is a common gastrointestinal disorder caused by excessive microbial growth in the small intestine, leading to a wide range of symptoms such as abdominal distention, bloating, nausea, vomiting, diarrhea, constipation, steatorrhea, and weight loss. We aimed to explore the association between patients' presenting SIBO symptoms to their breath-test results and response to treatment to determine whether the symptoms could potentially be used as a predictive tool for aiding in the diagnosis and treatment of SIBO.
Methods: A retrospective analysis was performed on a single-center study population from 2019 to 2021. The study population consisted of patients who had tested positive for SIBO for the first time. Chi-squared test and Fisher's exact test were the statistical analyses used to assess for any associations between symptoms and breath test results and associations between symptoms and response to treatment. This study received IRB approval.
Results: 174 patients tested positive for SIBO. The most common presenting symptom was bloating/distention (79.9%), followed by pain/discomfort (66.7%), diarrhea (48.9%), constipation (36.8%), nausea/vomiting (28.2%), weight loss (16.1%), flatulence (13.8%), and steatorrhea (6.9%). Breath tests were positive for hydrogen, methane, and both gases in 76.5%, 18.4%, and 5.2% of patients respectively. Most patients were treated with rifaximin (69%), followed by rifaximin + neomycin combination (6.9%), metronidazole (4.6%), ciprofloxacin (4.6%), amoxicillin-clavulanate (4.6%), and other (2.3%). The *P*-values for the associations between symptoms and breath-test positivity (hydrogen or methane) and the associations between symptoms and response to therapy are outlined in our Table. None of the associations had a *P*-value < 0.05. Response to treatment using rifaximin + neomycin combination vs rifaximin alone had a *P*-value = 0.5.
Conclusion: There was no statistically significant relationship between symptoms and breath-test positivity. There was also no statistically significant relationship between symptoms and response to therapy. In addition, there does not appear to be an increased efficacy of using rifaximin + neomycin combination compared to rifaximin alone.

Table 1. ***P*-values for the association between symptom and breath test positivity (hydrogen or methane) and the association between symptom and response to therapy**

Symptoms	Association with breath-test results (hydrogen/methane)	Association with response to therapy
Bloating/distention	$P = 0.08$	$P = 0.20$
Pain/discomfort	$P = 0.37$	$P = 0.52$
Diarrhea	$P = 0.60$	$P = 0.099$
Constipation	$P = 0.35$	$P = 0.37$
Nausea/vomiting	$P = 0.20$	$P = 0.57$
Weight loss	$P = 0.22$	$P = 0.60$
Flatulence	$P = 0.19$	$P = 1.0$
Steatorrhea	$P = 0.95$	$P = 0.68$

S1565

Association of Small Bowel Angioectasia With Cardiac, Pulmonary, Liver, Renal and Systemic Diseases

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Introduction: Small bowel angioectasias (SBA) are characterized by regions of local accumulation of dilated blood vessels in the intestinal wall. While angioectasias can occur anywhere in the gastrointestinal tract, a significant source for obscure GI bleed is within the small bowel. The mechanism behind SBA development is not fully understood. Studies have shown that SBAs may be associated with conditions such as aortic valve stenosis, liver cirrhosis, and renal failure. The purpose of this study is to identify comorbid medical conditions that may be associated with SBA.
Methods: Patients admitted to Thomas Jefferson University Hospital and Methodist Hospital with small bowel angioectasia between 2018-2022 were identified using *ICD-10* coding. Each patient's diagnosis was confirmed via endoscopy or capsule reports. Patients with only gastric and/or colonic angioectasias were excluded. Patient demographics and comorbidities were evaluated.
Results: Overall, 447 patients with SBA were identified; 237 were female and 210 were male. Thirteen patients were below 50 years old and 7 were >90 years old; 44 patients were between 51-60 years old, 140 were between 61-70, 148 were between 71-80, and 88 were between 81-90. 261 patients were White and 151 patients were Black. Other racial groups were Hispanic (12), Asian (9), Native American (1), and Unknown (13). Hypertension and hyperlipidemia were seen in 331 and 213 patients, respectively; 137 patients had diabetes. COPD affected 127 patients. CAD occurred in 123 patients. Anemia was present in 122 patients. Atrial fibrillation was seen in 115 patients. Heart failure and CKD were both seen in 111 patients. Cancer was seen in 103 patients. Other comorbidities include: GERD (99), sleep apnea (71), aortic stenosis (53), liver cirrhosis (50 patients), hypothyroidism (47), stroke (45), ESRD (45), asthma (26), mitral valve regurgitation (23), and pulmonary hypertension (23) (Table).
Conclusion: This study sought to identify common comorbidities in SBA. Key characteristics were chronicity and cardiopulmonary effects. Kidney disease and malignancy were also common. Heyde syndrome (aortic stenosis with angioectasias) is a known cause of GI bleed. Yet aortic stenosis was only seen in 12% of patients in this study. Other comorbidities were more prevalent. The results demonstrate that a variety of chronic diseases may be associated with angioectasia. Further research is needed to understand the risk for GI bleeding in the presence of chronic disease.

Table 1. Twenty most common comorbidities found in patients with small bowel angioectasia

Comorbidity	# Of Patients	% Of total
Hypertension	331	74
Hyperlipidemia	213	48
Diabetes Mellitus	137	31
COPD	127	28
CAD	123	28
Anemia	122	27
Atrial fibrillation	115	26
Heart Failure	111	25
Chronic Kidney Disease	111	25
Cancer	103	23
GERD	99	22
OSA	71	16
Aortic Stenosis	53	12
Cirrhosis	50	11
Hypothyroidism	47	11
Stroke	45	10
ESRD	45	10
Asthma	26	6
Mitral Regurgitation	23	5
Pulmonary Hypertension	23	5

The number of patients with each disease is listed, along with the percentage of total patients who have the condition. The total number of patients observed was 447.

S1566

Evaluation of the Pharmacokinetics of PGN-OB1 Following Oral Administration of an Oral Biotherapeutics Delivery System (OBDS) in Yucatan Swine

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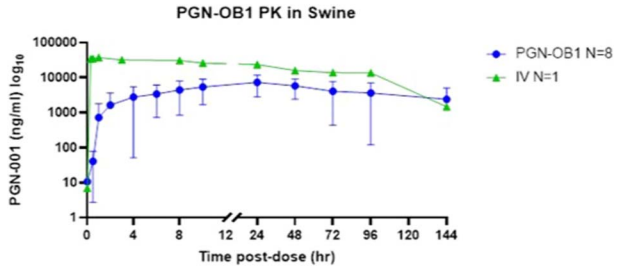
Introduction: Biologics/peptides/nucleic acids are highly effective drugs; however, oral delivery of these therapeutics has proved to be difficult due to the harsh conditions of the upper gastrointestinal tract (GIT) and the poor absorption rate in the small intestinal mucosa. We aimed to develop an oral biotherapeutic delivery system (OBDS) that protects these biomolecules from degradation in the upper GIT and increases oral bioavailability via needless injection in the submucosal space of the small intestine. We describe the development of models to assess the functional capability of the OBDS capsule. A Yucatan minipig model was chosen to better represent the pharmacokinetic properties of submucosal injection in humans and evaluate the injection efficiency of OBDS *in vivo*.
Methods: Due to prolonged and variable gastric residence times in the swine model, the semi-autonomous OBDS capsules containing a variant of adalimumab (PGN-001), PGN-OB1, were administrated by intraduodenal endoscopic placement (ID) and released into the proximal small intestines of the female Yucatan swine. Blood samples post-ID dosing were collected to evaluate the injection efficiency of the OBDS compared with the IV control group.
Results: All OBDS capsules were successfully advanced through the pyloric sphincter via endoscopic placement, without early deployment, and released in the proximal duodenum to naturally transit and deploy *in vivo*. Out of 13 animals dosed, 8 animals showed detectable drug levels (Figure), and an oral bioavailability average of 22% or 25% (range from 7-55%) excluding an animal showing a late deployment at 72hr post-dose. The variability in the swine model is believed to be due to physiologic variability that may not directly translate to humans including variability in small intestinal transit time, and more frequent and larger, gas and fluid pockets in the swine.
Conclusion: Here we have provided proof-of-concept of a semi-autonomous OBDS device that can achieve as high as 55% bioavailability of a variant of adalimumab that is a magnitude higher than current oral protein or peptide delivery technology in the market and at levels much closer to the subcutaneous route of administration estimated in human trials. The OBDS capsules could potentially provide a better alternative for the non-invasive route of administration with better patient compliance.



[1566] **Figure 1.** Plasma concentration of PGN-001 treated with ID and IV over time

S1567

Celiac Disease Patients Have Higher Risk for *Clostridium difficile* Infections: A Population-Based Study

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Introduction: Clostridium difficile infection (CDI) is a common gastrointestinal pathology with the incidence in the United States to 20-30 per 100 000 population. Previous studies showed association of Celiac disease and CDI, however the prevalence of CDI in patients with celiac disease has not been described in literature. Our aim is to estimate the prevalence of CDI among celiac patients, and to assess whether celiac disease is an independent risk factor for CDI.

Methods: A large multi-center database (Explorys Inc., Cleveland, OH, USA) of aggregated electronic health records of 26 different healthcare systems with a total of 360 hospitals and more than 70 million patients across the United States was utilized for this study. A cohort of patients with Celiac disease was identified. Later, a second sub-cohort of those who also had CDI was identified. We excluded all the patients age < 18, patients with inflammatory bowel diseases, immunosuppressed, HIV, Roux-en-Y bypass. Statistical Package for Social Sciences (SPSS version 25, IBM Corp) was used for statistical analysis, and for all analyses, a 2-sided *P*-value of < 0.05 was considered statistically significant. Multivariate analysis was performed to adjust for multiple factors including age, sex, race, smoking, use of proton pump inhibitors, diagnosis of celiac disease.
Results: Among the 80,884,400 individuals screened in the database there were total of 143,180 with Celiac disease (0.17%). The baseline characteristics of patients with Celiac disease are represented in Table. Celiac disease was independently associated with a higher risk of developing CDI (OR= 2.592). Other factors associated with risk of CDI included age 18-65 (OR= 2.764), White race (OR =1.880), smoking (OR= 1.540), use of proton pump inhibitors (OR= 8.468) as in the Figure.
Conclusion: Celiac disease seems to be an independent risk factor for CDI. Further studies are required to investigate specifics of pathophysiology, diagnosis and treatment of CDI in patient with celiac disease.

	OR	95% CI	p value
Age>65	2.764	2.740- 2.788	<0.0001
Male	0.782	.775-.789	<0.0001
Caucasian	1.880	1.861-1.900	<0.0001
Celiac Disease	2.592	2.479-2.711	<0.0001
PPI*	8.468	8.392-8.545	<0.0001
Smoking	1.540	1.522-1.559	<0.0001

*proton pump inhibitors

[1567] **Figure 1.** Multivariable analysis for risk factors in individuals with Clostridium Difficile Infection

Table 1. Baseline Catachrestic of Study Population.

		Celiac disease (%) N=143,180	No celiac disease (%) N=80,741,220
Age	18-65	9,7340 (67.9)	47,893,510 (59.3)
	>65	39,400 (27.5)	21,266,990 (26.2)
Sex	Male	1,640 (1.15)	189,850 (0.24)
Race	Caucasian	117,430 (82.0)	42,011,260 (52.0)
Comorbidities	Clostridium difficile	2,050 (1.43)	231,830 (0.29)
	Hypertension	45,710 (31.9)	14,183,650 (17.5)
	Type 2 Diabetes	20,050 (13.9)	5,651,930 (6.9)
	Hyperlipidemia	51,170 (35.7)	11,782,380 (14.5)
	Smoking	19,480 (13.6)	3,874,810 (52.0)
Medications	PPI	54,790 (38.27)	86,37,610 (10.70)

S1568

Spectrum of Body Mass Index in Patients With Celiac Disease

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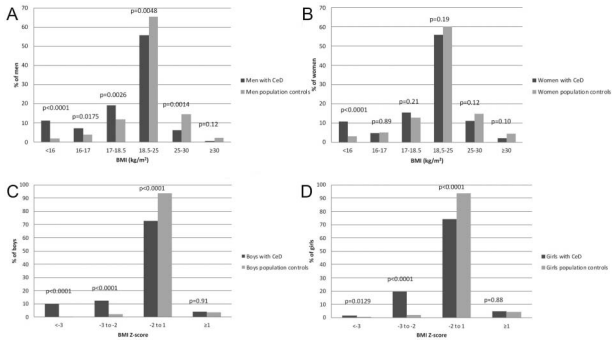
Introduction: Although generally perceived to be mostly underweight, patients with celiac disease (CeD) can be of normal weight or even overweight/obese. We conducted a comparative cross-sectional study to assess the body mass index (BMI) of treatment-naïve patients with CeD in comparison to the population controls.
Methods: In a retrospective analysis of a prospectively maintained database of treatment-naive patients with CeD, we included 583 patients (419 adults [43.7% males] and 164 adolescents [43.9% males]). Details extracted from our database included demographic details, height, weight, laboratory and histopathological parameters. The data of 375,460 adults (12.7% males) and 71,315 adolescents (12.1% males) of Indian National Family Health Survey-4 was considered as the population controls.
Results: Although majority of adult and adolescent patients with CeD patients had BMI in normal or underweight range, a significant proportion were overweight or obese (adults 10.3%, adolescents 3.7%) (Figure). The mean BMI in patients with CeD was significantly lower than the population controls for adults (men 19.8 vs 21.8 kg/m^2, $P < 0.001$; women 20.5 vs 21.8 kg/m^2, $P < 0.001$) and adolescents (BMI Z-scores: boys -1.4 vs -0.5, $P < 0.001$; girls -1 vs -0.4, $P < 0.001$). Higher proportion of adults (men 11.2% vs 1.9%, $P < 0.001$; women 10.7% vs 3.2%, $P < 0.001$) and adolescents (boys 10.4% vs 0.2%, $P < 0.001$; girls 1.6% vs 0.2%, $P = 0.01$) with CeD were severely underweight compared to population controls.
Conclusion: While majority of adult and adolescent patients with CeD have a lower BMI compared to population controls, a significant proportion can be overweight and even obese.



[1568] **Figure 1.** Graphs depicting comparison of adult and adolescent patients with celiac disease (CeD) against population controls from India. (A) Spectrum of body mass index (BMI) in men with CeD as compared to population controls. (B) Spectrum of BMI in women with CeD as compared to population controls. (C) Spectrum of BMI in boys with CeD as compared to population controls. (D) Spectrum of BMI in girls with CeD as compared to population controls

S1569

Gender-Based Association of Celiac Disease With Systemic Lupus Erythematosus and Other Rheumatologic Conditions: Analysis From National Inpatient Database

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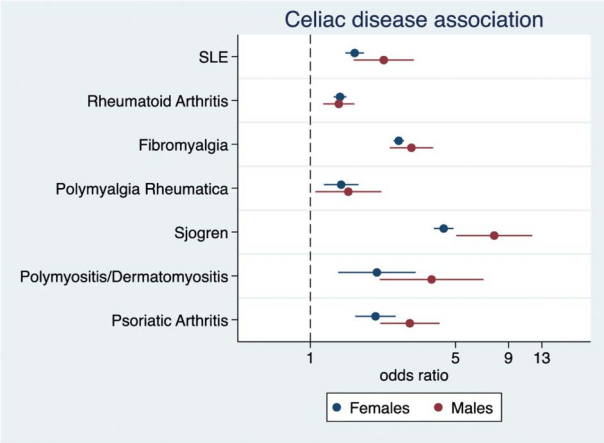
Introduction: Celiac disease is an immune mediated disorder of the digestive system that often presents with extraintestinal manifestations. The association of celiac disease with dermatitis herpetiformis, Type I Diabetes, IgA deficiency and chromosomal defects like Turner's and Down's syndrome is well known. However, the association of rheumatological diseases with Celiac disease remains controversial, especially based on gender. The aim of our study was to examine the association of rheumatological diseases with Celiac disease, stratified by gender.
Methods: All adult hospitalized patients from January 2016 to December 2019 in nationwide inpatient sample (NIS) were captured. The study population included all patients with a diagnosis of Celiac disease using *ICD-10* codes (International Classification of Diseases, tenth edition). Those without Celiac disease were included as the Control group. We identified patients with a diagnosis of SLE (Systemic Lupus Erythematosus), RA (Rheumatoid Arthritis), Fibromyalgia, Polymyalgia Rheumatica (PMR), Polymyositis/Dermatomyositis (PM/DM), Sjogren syndrome and Psoriatic Arthritis (PsA) using *ICD-10* codes. We used linear regression for comparing continuous variables and Chi-square test for categorical variables. The association between Celiac disease and rheumatological conditions was analyzed using multivariate logistic regression model.
Results: Our study included a sample size of 122,238,462 patients, of which 158,910 (0.13%) patients had Celiac disease and 122,079,552 (99.87%) patients did not. In the study group, highest risk was seen with Sjogren in both males (aOR=7.41) and females (aOR=3.93), with $P< 0.001$, followed by PM/DM (aOR=4.44 in males and aOR=2.11 in females, P-value < 0.001) compared to the control group. We also noted a higher risk of Celiac with SLE (aOR=1.72), RA (aOR=1.34), Fibromyalgia (aOR= 2.09), PMR (aOR=1.65) and PsA (aOR=2.71), all with $P< 0.001$ (Figure, Table). Similarly in males, greater risk was seen with SLE (aOR=2.51), RA (aOR=1.34), fibromyalgia (aOR=2.59), PMR (aOR=1.52), and PsA (aOR, 2.63).
Conclusion: There is a significantly greater association of Rheumatological conditions with Celiac disease, more so in males than in females. Although, more studies are warranted to determine the causal relationship, knowledge about the association between them can help with early diagnosis and better management of these patients.



[1569] **Figure 1.** Graph 1

Table 1. Association of Celiac disease with Rheumatologic diseases

Rheumatologic diseases	Females		Males	
	Unadjusted odds ratio	Adjusted odds ratio[a]	Unadjusted odds ratio	Adjusted odds ratio[a]
SLE*	2.49[b]	1.72[b]	2.89[b]	2.51[b]
Rheumatoid Arthritis	1.83[b]	1.34[b]	1.55[b]	1.34[b]
Fibromyalgia	3.06[b]	2.09[b]	3.44[b]	2.59[b]
Polymyalgia Rheumatica	1.65[b]	1.35[b]	1.65[b]	1.52[b]
Polymyositis/dermatomyositis	2.86[b]	2.11[b]	4.51[b]	4.44[b]
Psoriatic arthritis	2.71[b]	1.57[b]	3.32[b]	2.63[b]
Sjogren	6.68[b]	3.91[b]	10.25[b]	7.37[b]

*SLE- Systemic Lupus Erythematosus.
[a]Adjusted for age, race, Elixhauser score and other rheumatologic conditions.
[b]significant and/or $P<0.001$.

S1570

Diagnosis and Treatment of Celiac Disease Among Adults: A Qualitative Study Among Gastroenterologists

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Introduction: Gastroenterologists (GI) play a key role in the diagnosis and treatment of persons with Celiac disease (CeD). Data is lacking regarding the management of CeD among GI. We conducted a qualitative study to assess the management of CeD among US GI.
Methods: In-depth 1-on-1 phone interviews with board certified GI (minimum 70% of time in direct patient care; minimum of 50 CeD patients/year). To reduce bias, the study was conducted by Cadence, an independent market research firm. Prior to the interviews, a list was prepared that covered the topics of interest. Data was coded and categorized, and a general description of each theme was formulated.
Results: Participants were 18 GI, with mean practice experience 14.1 $\pm$ 7.7 years, and mean annual CeD patients treated 119 $\pm$ 97. Theme 1 - Typical Patient - Patients are diverse in their demographic characteristics, and are predominantly White, female, with ages ranging from 18 to 55 years. They typically present with abdominal symptoms that are also consistent with irritable bowel syndrome or ulcerative colitis Some present with nutrient deficiencies. Theme 2 – Referral Patterns - Most patients are referred from primary care physicians, and about 25% of referrals received a prior diagnosis of CeD. Theme 3 – Diagnostic Workup - The typical diagnostic process for GIs begins with serological tests, followed by endoscopy. Theme 4 – Patient Categorization - Although duodenal biopsies are graded by pathologists (Marsh scale), this is not useful for patient management. Categorization of patients by disease severity is not typically used, but patients may be informally classified based on the number and nature of their symptoms. Theme 5 – Therapeutic Approach - The primary therapeutic approach for patients with CeD is a Gluten Free Diet (GFD). Patients might also be treated to address acute symptoms (e.g., anti-

diarrheals, anti-nausea) and 50% of GI use steroids for severe or refractory cases. Theme 6 - Biggest Challenge in Treating CeD Patients - The biggest challenge is compliance with a GFD, which is of concern in about 40% of patients. GIs believe that there is a major unmet need for CeD treatments beyond a GFD.
Conclusion: More patients are now being evaluated for CeD driven by increasing awareness of the disease. GI respondents consider CeD to be a straightforward disease to diagnose and treat, and the primary therapeutic approach is a GFD. However, compliance with a GFD is challenging for many patients, and new treatment options for CeD are needed.

S1571

Device-Assisted Enteroscopy (DAE) in the Surveillance of Intestinal Polyposis in Peutz-Jeghers Syndrome

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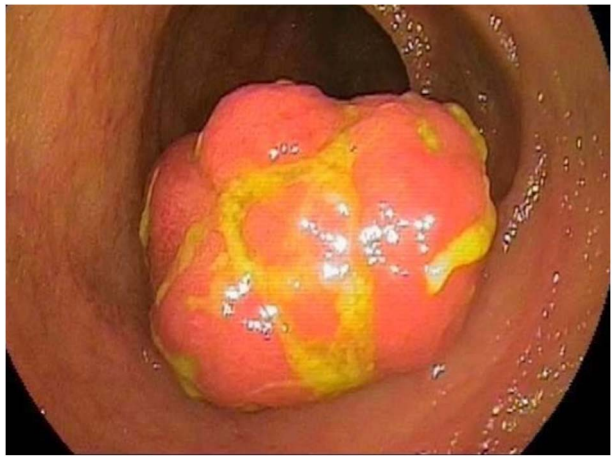
Introduction: Peutz-Jeghers Syndrome (PJS) is an autosomal dominant, genetic disorder characterized by the formation of hamartomatous polyps in the gastrointestinal tract. These polyps are a source of significant morbidity and mortality in this population due to associated complications of intestinal obstruction, bleeding, and malignant transformation. Surveillance of intestinal polyposis in patients with PJS has not been extensively studied, although centers which perform a significant volume of device-assisted enteroscopies (DAEs) encounter multiple cases each year. With the progressive adoption of DAE, examination of the deep small bowel for monitoring and prophylactic polypectomy has the potential to prevent complications of PJS and minimize the need for repeated laparotomy.
Methods: After obtaining IRB approval, electronic health records were used to identify all DAEs performed on patients with PJS at 3 US referral centers (LSU, Duke, and UMass) between January 1, 2007 and January 1, 2020. Electronic medical records were reviewed to collect and analyze multiple data points. Primary endpoints included the complications associated with DAE performed for removal of intestinal hamartomas and the rate of laparotomy in PJS patients prior to and after index DAE. Secondary data points included patient characteristics, procedural details, and size/location/distribution of small bowel hamartomas.
Results: Twenty-four patients met our inclusion criteria. Of these, 18 (75%) had previously undergone small bowel surgery for complications related to small bowel hamartomas. Between 2007 - 2020, a total of 46 DAEs were performed in these patients with an average of one surveillance exam every 2.5 years. A total of 131 polypectomies were performed during our study period with an observed complication rate of 1.5%, including one bowel perforation requiring surgery. Only one patient underwent surgery related to small bowel hamartomas following initial DAE surveillance exam over a total of 366 years of aggregated follow-up (Figure).
Conclusion: Endoscopic management of small bowel polyps in patients with PJS using DAE is an effective strategy for prophylactic removal of hamartomas. DAE surveillance and endoscopic polypectomy is safe and may decrease the need for repeated laparotomy due to complications from intestinal hamartomas in patients with PJS.



[1571] **Figure 1.** Hamartomatous polyp in the ileum removed with saline injection-lift technique and hot snare polypectomy

S1572 WITHDRAWN

S1573

Incidence and Risk of Diseases of the Musculoskeletal Systems and Connective Tissues in Celiac Disease Patients

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Introduction: Celiac disease (CD) is an autoimmune-based reaction to dietary gluten found predominantly in wheat, barley, or rye. The main pathogenesis occurs in the intestinal mucosa by lymphocytic infiltration with subsequent destruction of the intestinal villi architecture. We aimed to identify the prevalence level of diseases of the musculoskeletal system and connective tissues in hospitalized CD patients.
Methods: NIS database was queried from September 2015 to December 2019 to retrieve records of patients admitted with a principal or secondary diagnosis of CD. We compared the incidence of diseases of the musculoskeletal system and connective tissues in CD (cases) to patients who did not have CD (controls). Controls were 1:1 fixed ratio nearest neighbor (greedy) propensity score-matched using the patient's age, sex, and race. We used clinical classification software refined (CCSR) for ICD-10-CM diagnosis v2021 and identified diseases of the musculoskeletal system and connective tissues. We used the Rao-Scott chi-square test on the weighted sample. We used alpha = 0.01 and P value < 0.001 considered statistically significant. Statistical analysis is performed in R (Studio 1.4).
Results: A total of 178,590 records (no outpatient) identified with CD in the weighted sample. Compared to non-CD matched patients, CD patients have an increased prevalence of juvenile arthritis (OR, 5.15 99% CI, 2.42 – 10.99; $P < 0.001$), systemic lupus erythematosus (OR, 3.27; 99% CI, 2.84 – 3.78; $P < 0.001$), osteoporosis (OR, 2.97; 99% CI, 2.68 – 3.29; $P < 0.001$), scoliosis (OR, 1.79; 99% CI, 1.48 – 2.17; $P < 0.001$), rheumatoid arthritis (OR, 1.78; 99% CI, 1.58 – 2.03; $P < 0.001$), stress fracture (OR, 1.68; 99% CI, 1.19 – 2.36; $P < 0.001$), spondylopathies/spondyloarthropathy (OR, 1.46; 99% CI, 1.33 – 1.61; $P < 0.001$, gout (OR, 1.44; 99% CI, 1.26 – 1.64; $P < 0.001$), and low back pain (OR, 1.21; 99% CI, 1.08 – 1.35; $P < 0.001$). CD patients are at low risk of osteomyelitis (OR, 0.74; 95% CI, 0.60 – 0.94; $P < 0.001$) and muscle disorders (OR, 0.83; 95% CI, 0.70 – 0.97; $P < 0.01$) (Table).
Conclusion: CD is a multisystem autoimmune condition with clinical presentations including musculoskeletal and rheumatological complaints. CD appears to have an increased risk of having another autoimmune disorder, little is known about the risk factors, pathogenesis, and relationship between CD and rheumatological conditions like SLE, Spondyloarthropathies, and Gout. We propose screening for CD in some patients and a need for rheumatologic follow-up.

Table 1. **Prevalence of Musculoskeletal System and Connective tissues in CD vs age-, sex-, race- matched non-CD patients, Weighted NIS 2015Q4 to 2019.**

Variables	CD = No (n= 178584) 50%	CD = Yes (n= 178590) 50%	OR (99%CI)	*P* value
Infective arthritis	469 (0.26%)	424 (0.24%)	NA	0.5029
Osteomyelitis	1665 (0.93%)	1250 (0.69%)	0.74 (0.60 – 0.94)	0.0007
Rheumatoid arthritis and related disease	3600 (2.02%)	6335 (3.55%)	1.78 (1.58 – 2.03)	< .0001
Juvenile arthritis	70 (0.03%)	360 (0.20%)	5.15 (2.42 – 10.99)	< .0001
Osteoarthritis	21839 (12.2%)	22750 (12.7%)	NA	0.0892
Tendon and synovial disorders	910 (0.51%)	1135 (0.64%)	NA	0.0331
Musculoskeletal pain, not low back pain	7885 (4.41%)	7390 (4.13%)	NA	0.0792
Spondylopathies/spondyloarthropathy	8640 (4.84%)	12355(6.92%)	1.46 (1.33 – 1.61)	< .0001
Osteoporosis	5030 (2.82%)	14160 (7.93%)	2.97 (2.68 – 3.29)	< .0001
Pathological fracture	840 (0.47%)	1175 (0.66%)	1.40 (1.06 – 1.85)	0.0018
Stress fracture	770 (0.43%)	460 (0.25%)	1.68 (1.19 – 2.36)	< .0001
Acquired foot deformities	585 (0.30%)	530 (0.33%)	NA	0.4643
Scoliosis	1515 (0.84%)	2700 (1.52%)	1.79 (1.48 – 2.17)	< .0001
Acquired deformities (excluding foot)	835 (0.47%)	605 (0.33%)	0.72 (0.53 – 0.99)	0.0092
Systemic lupus erythematosus	2405 (1.35%)	7640 (4.27%)	3.27 (2.84 – 3.78)	< .0001
Muscle disorders	2875 (1.61%)	2375 (1.33%)	0.83 (0.70 – 0.97)	0.0025
Disorders of jaw	230 (0.13%)	385 (0.22%)	1.675 (1.03 – 2.73)	0.0059
Aseptic necrosis and osteonecrosis	500 (0.28%)	570 (0.32%)	NA	0.3447
Gout	3565 (1.99%)	5080 (2.84%)	1.44 (1.26 – 1.64)	< .0001
Low back pain	6060 (3.39%)	7265 (4.07%)	1.21 (1.08 – 1.35)	< .0001

S1574

Management of Home Parenteral Nutrition in Gastroenterology Office Infusion Centers

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Introduction: Home parenteral nutrition (PN) is commonly provided to patients (pts) with gastrointestinal dysfunction. There remains a lack of data on the provision of PN through gastroenterology (GI) office infusion centers (OICs). We evaluated the management of pts in this setting.
Methods: A retrospective study was conducted in pts receiving PN through GI OICs over a 6-year period. Pts were selected for PN therapy by their gastroenterologist. The physician and PN-trained pharmacist managed the PN formula. PN-trained nurses performed pt teaching, drew weekly labs, and provide catheter care in the OIC. All pts self-administered PN with solution and supplies dispensed weekly from the pharmacy. Data included demographics, PN indications, comorbidities, nutritional status, PN regimen, and home vs hospital PN initiation. Pt weight (wt) changes were assessed, and catheter-related blood stream infections (CRBSI) occurring during PN therapy were measured. Descriptive statistics were utilized.
Results: A total of 17 PN pts (71% female) were identified with a mean (SD) age of 44 (17) years. PN was initiated in the OIC in 8 pts (47%). Indications for PN included severe malnutrition/malabsorption (n=6), small bowel obstruction (n=4), intestinal motility disorder (n=4), GI fistula (n=2), and short bowel syndrome (n=1). Underlying disease included inflammatory bowel disease (IBD) in 8 pts (47%) and malignant disease in 3 pts (18%). Most (76%) were on a limited oral diet as tolerated. The median [IQR] duration of PN therapy was 10.3 [1-21] months, and 10 pts (59%) received >3 months of therapy. All pts received cyclic PN with protein, carbohydrate, and fat. Mean (SD) caloric intake of PN was 28 (6) kcal/kg/day and was administered over 9-18 hours. Stable or improved wt changes were reported in 14 pts (82%) at a median [IQR] increase of 4.5 [1-7] kg. Three pts experienced declining wt (2 malignant disease, 1 uncontrolled IBD). The CRBSI rate was 0.81% per 1000 days, which was lower than the previously reported rates in adults of 3.1% and 4.58% per 1000 days.
Conclusion: The GI OICs provide collaborative care settings for the management of PN using trained nurses, pharmacists, and readily accessible gastroenterologists. PN was successfully initiated in the home setting in almost half of study pts. Positive weight changes were recorded in 82% of pts, and there was a lower rate of catheter infections compared to reported rates with traditional home care. GI OICs offer safe and effective provision of home PN.

S1575

The Use of Post-Prandial Breath Hydrogen to Monitor Antibiotic-Induced Changes in the Activity of the Gut Microbiome

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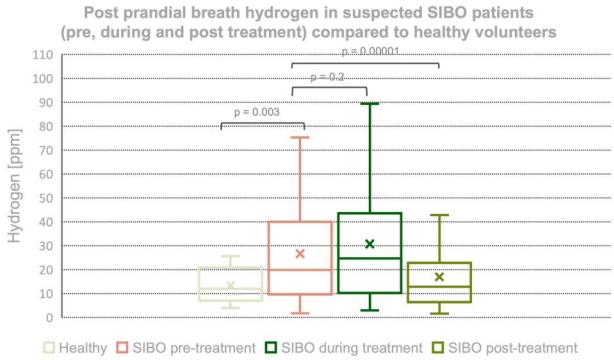
Introduction: Small intestinal bacterial overgrowth (SIBO) is a common condition that is associated with a range of non-specific GI symptoms. Patients are often prescribed an empirical trial of antibiotics, however, response to treatment can vary. Due to the heterogeneous nature of symptoms experienced, post-prandial breath hydrogen (PPH_2) may assist in identifying beneficial changes in the activity of the gut microbiome in those with a dysbiotic phenotype.
Methods: Sixteen suspected SIBO patients measured their PPH_2 using an at-home, app-connected breath analyzer (AIRE, FoodMarble). Readings were taken at 0, 30, 60, and 90 min after their morning and evening meals for 7 days pre and post-treatment. There were 14/16 patients who followed this protocol during antibiotic treatment. Six controls measured their PPH_2 levels for 7 days. Gas normalization was defined as +2 SD of the mean PPH_2 in controls.
Results: Patients produced significantly more PPH_2 (P=0.003), 27 ppm ± 22 ppm (mean ± SD) than controls (13 ppm ± 7 ppm). 5/14 patients saw a transient increase (P=0.2) of 23 ppm ± 23 ppm (mean delta ± SD) of PPH_2 during treatment. Post-treatment, patients produced significantly less PPH_2 (P=0.00001), 17 ppm ± 13 ppm, more closely matching that of healthy controls (Figure).
Conclusion: For the first time, PPH_2 was tracked before, during, and after antibiotic treatment. PPH_2 was significantly greater in patients. Interestingly, for some patients, PPH_2 increased during the treatment period, likely due to a dynamic rearrangement of certain microbial populations. Post-treatment, the level of PPH_2 in patients was more similar to that of healthy volunteers. The collection of PPH_2 data may be useful to identify those who produce elevated gas levels due to dysbiosis and to determine the normalization of their microbiome in response to antibiotics.



[1575] **Figure 1.** Post prandial breath hydrogen of suspected SIBO patients (n=16) before, during and after antibiotic treatment compared to healthy controls (n = 6)

S1576

Cardiovascular Diseases in Celiac Disease Patients

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Introduction: Celiac disease (CD) is a common immune-mediated disease of the small bowel caused by exposure to gluten in genetically sensitive individuals. CD is associated with a modestly increased risk of cardiovascular diseases, but the evidence base is limited. In this study, we aimed to analyze the prevalence of cardiovascular diseases in CD patients.
Methods: NIS database was queried from September 2015 to 2019 to retrieve records of patients admitted with a principal or secondary diagnosis of CD. We compared the incidence of cardiovascular diseases in CD (cases) to patients who did not have CD(controls). Controls were 1:1 fixed ratio nearest neighbor (greedy) propensity score-matched using the patient's age, sex, and race. We used clinical classification software refined (CCSR) for ICD-10-CM diagnosis v2021 and identified a spectrum of cardiovascular diseases. We used the Rao-Scott chi-square test on the weighted sample. We used alpha = 0.01 and *P* value < 0.001 considered statistically significant. Statistical analysis is performed in R (Studio 1.4).
Results: A total of 178,590 records identified with CD in the weighted sample. Compared to non-CD matched patients, CD patients have an increased prevalence some cardiovascular diseases including cardiac and circulatory congenital anomalies (OR, 2.34; 99% CI, 1.83 – 3.01; $P < 0.001$), vasculitis (OR, 2.39; 99% CI, 1.66 – 3.43; $P < 0.001$), acute hemorrhagic cerebrovascular disease (OR, 1.98; 99% CI, 1.42 – 2.75; $P < 0.001$), chronic phlebitis (OR, 1.87; 99% CI, 1.26 – 2.76; $P < 0.001$), peripheral and visceral vascular disease (OR, 1.71; 99% CI, 1.55 – 1.89; $P < 0.001$), pericarditis and pericardial disease (OR, 1.70; 99% CI, 1.29 – 2.24; $P < 0.001$), chronic rheumatic heart disease (OR, 1.48; 99% CI, 1.25 – 1.76; $P < 0.001$, hypotension (OR, 1.50; 99% CI, 1.37 – 1.65; $P < 0.001$), acute phlebitis (OR, 1.48; 99% CI, 1.27 – 1.73; $P < 0.001$), conduction disorders (OR, 1.32; 99% CI, 1.20 – 1.45; $P < 0.001$), and cardiac dysrhythmias (OR, 1.13; 99% CI, 1.06 – 1.20; $P < 0.001$). CD patients are at low risk of essential hypertension (OR, 0.89; 95% CI, 0.85 – 0.94; $P < 0.001$) and heart failure (OR, 0.78; 95% CI, 0.73 – 0.84; $P < 0.001$) (Table).
Conclusion: Our study showed that CD patients are an increased likelihood of some cardiovascular diseases, however little is known about the risk factors and pathogenesis. Therefore, more studies are needed to determine the risk factor and mechanisms for developing cardiovascular diseases in CD patients.

Table 1. Prevalence of Cardiovascular Diseases in CD vs age-, sex-, race- matched non-CD patients, Weighted NIS 2015Q4 to 2019

Variables	CD = No (n= 178584) 50%	CD = Yes (n= 178590) 50%	OR (99%CI)	*P* value
Cardiac and circulatory congenital anomalies	819 (0.46%)	1915 (1.07%)	2.34 (1.83 – 3.01)	< .0001
Chronic rheumatic heart disease	2300 (1.29%)	3385 (1.89%)	1.48 (1.25 – 1.76)	< .0001
Endocarditis and endocardial disease	460 (0.26%)	515 (0.29%)	NA	0.4468
Myocarditis and cardiomyopathy	5525 (3.10%)	6410 (3.59%)	1.17 (1.04 – 1.31)	0.0008
Pericarditis and pericardial disease	745 (0.42%)	1265 (0.71%)	1.70 (1.29 – 2.24)	< .0001
Essential hypertension	57753 (32.3%)	53330 (29.9%)	0.89 (0.85 – 0.94)	< .0001
Acute myocardial infarction	4465 (2.50%)	4135 (2.32%)	NA	0.1410
Coronary atherosclerosis and other heart disease	28964 (16.2%)	29915 (16.8%)	NA	0.1196
Acute pulmonary embolism	1905 (1.07%)	1909 (1.08%)	NA	0.8876
Pulmonary heart disease	4800 (2.69%)	5605 (3.14%)	1.17 (1.04 – 1.33)	0.0008
Conduction disorders	7445 (4.17%)	9710 (5.43%)	1.32 (1.20 – 1.45)	< .0001
Cardiac dysrhythmias	24389 (13.7%)	26990 (15.1%)	1.13 (1.06 – 1.20)	< .0001
Cardiac arrest and ventricular fibrillation	864 (0.48%)	685 (0.38%)	NA	0.0383
Heart failure	24919 (14.0%)	20100 (11.3%)	0.78 (0.73 – 0.84)	< .0001
Cerebral infarction	2825 (1.58%)	2725 (1.53%)	NA	0.5653
Acute hemorrhagic cerebrovascular disease	450 (0.25%)	890 (0.49%)	1.98 (1.42 – 2.75)	< .0001
Occlusion or stenosis of precerebral or cerebral arteries without infarction	1490 (0.83%)	2025 (1.14%)	1.36 (1.10 – 1.68)	0.0001
Peripheral and visceral vascular disease	6610 (3.70%)	11045 (6.19%)	1.71 (1.55 – 1.89)	< .0001
Hypotension	7470 (4.19%)	10975 (6.14%)	1.50 (1.37 – 1.65)	< .0001
Acute phlebitis; thrombophlebitis and thromboembolism	2435 (1.36%)	3580 (2.00%)	1.48 (1.27 – 1.73)	< .0001
Chronic phlebitis; thrombophlebitis and thromboembolism	625 (0.19 %)	335 (0.35%)	1.87 (1.26 – 2.76)	< .0001
Vasculitis	365 (0.20 %)	870 (0.49%)	2.39 (1.66 – 3.43)	< .0001

S1577

Outcomes of Acute Mesenteric Ischemia in Patients With End-Stage Renal Disease: A Nationwide Database Study

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Introduction: Acute mesenteric ischemia is an uncommon disorder occurring secondary to an abrupt obstruction in blood flow to the intestines resulting in bowel infarction. It is associated with an extremely high mortality rate. Only limited studies have evaluated this lethal disease among patients with end-stage renal disease (ESRD). We aim to determine outcomes in patients diagnosed with acute mesenteric ischemia with and without ESRD.
Methods: A retrospective analysis was performed by utilizing the National Inpatient Sample database (2016, 2017, and 2018) and the International Classification of Diseases, Tenth Revision codes to identify the patients > 18 years old with the principal diagnosis of acute mesenteric ischemia. We compared the all-cause in-hospital mortality, length of hospital stay (LOS), and total costs between the ESRD cohort vs non-ESRD cohort. Categorical variables were compared using the chi-square test, and continuous variables were compared using the t-test.
Results: We Identified 169,245 patients with acute mesenteric ischemia, of whom 10,493 (6.2%) patients had ESRD, and 158,752 (93.8%) patients did not have ESRD. In-hospital mortality was significantly higher in patients with ESRD (OR 1.96, 95% CI: 1.66–2.32; P = 0.00). Hyperlipidemia (P < 0.01), cardiovascular comorbidity (P = 0.01), and older age (P = 0.02) were identified as predictors of higher mortality. Patients with acute mesenteric ischemia and ESRD have high odds of peritonitis (OR 2.97, 95% CI: 2.26–3.91; P = 0.00), sepsis (OR 15.7, 95% CI: 13.5–18.26; P = 0.00), and ileus (OR 1.46 95% CI: 1.10–1.95; P = 0.008) compared to patients without ESRD. Additionally, patients with ESRD had a longer LOS (7.4 days vs 5.3 days; P < 0.00), and higher total hospital cost ($91,520 vs $58,175; P < 0.00) compared to patients without ESRD (Table).
Conclusion: In patients admitted for acute mesenteric ischemia, ESRD is an independent risk factor for increased inpatient mortality. Compared to patients without ESRD, patients with ESRD have a higher risk of peritonitis, sepsis, and ileus and contribute to longer LOS and increased healthcare costs.

Table 1. Primary and secondary outcomes of acute mesenteric ischemia in ESRD vs Non- ESRD cohort

Frequency in %	ESRD (N=10,493)	Non-ESRD (158,752)	OR [95% CI]	*P*-value
In-hospital mortality	8.5	4.5	1.96 [1.66-2.32]	0.00
Peritonitis	3.1	1.9	2.97 [2.26-3.91]	0.00
Sepsis	7.1	3.8	15.7 [13.5-18.26]	0.00
Ileus	3.1	1.7	1.46 [1.10-1.95]	0.01
Patient's characteristics				
Mean age	68	70		
Female	52	66		
Race				
Caucasian	45.8	79.9		0.00
African American	32.1	9.2		
Hispanic	14.1	6.6		
Asian	4.0	2.0		
Native American	1.0	0.3		
Others	2.7	1.8		
Insurance				
Medicare	84.6	72.9		0.00
Medicaid	7.4	6.8		
Private	7.4	1.8		
Others/Uninsured	0.5	1.8		
Bed size				
Small	14.8	19.3		0.00
Medium	28.5	30.1		
Large	56.5	50.4		
Hospital Region				
Northeast	20.9	21.0		0.1
Midwest	24.6	22.2		
South	30.0	39.2		
West	17.5	17.2		
Teaching hospital	74.3	66.6		0.00
Chronic comorbidity				
Diabetes mellitus	62.0	28.8		0.00
Chronic heart failure	45.1	18.9		0.00
Obesity	14.3	12.3		0.01
Dyslipidemia	46.2	46.8		0.6
Coronary artery disease	51.2	31.8		0.00

S1578

Sleep Disturbances and Sexual Dysfunction in Patients With Gluten-Associated Disorders

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Introduction: Sleep disturbances and sexual dysfunction are well documented in patients with gluten-associated disorders (GAD). However, the effect of strict adherence to a gluten-free diet (GFD) and its effect on sleep and sexual health is less understood, largely due to a lack of a large-scale study population. This study assesses factors associated with sleep and sexual health, including GFD adherence.
Methods: Validated surveys were sent in 2022 to participants enrolled in the Celiac Collective registry. A cross-sectional study was conducted incorporating demographics and medical history, along with multiple patient-reported outcomes (PRO) tools such as the Celiac Dietary Adherence Test (CDAT), Celiac Symptom Index (CSI), Treatment Self-Regulation Questionnaire (TSRQ) for motivation, and PRO Measurement Information System (PROMIS) domains of sleep disturbance and sexual function. Our primary outcomes were prevalence of sleep disturbance or sexual dysfunction in patients with GAD compared to the general population and their association with GFD adherence. Statistical analyses were conducted using chi-square, Fisher's exact and Wilcoxon rank-sum tests.

Results: Preliminary results demonstrate that 64.9% and 84.6% of subjects with gluten-associated disorders have sleep disturbance or sexual dysfunction, respectively (Table). Both are higher rates than the general population. Sleep disturbances are associated with shorter disease duration and higher amotivation scores, while sexual dysfunction is associated with longer disease duration and female sex.
Conclusion: This study demonstrated that patients with GAD have higher sleep and sexual dysfunction compared to the general population, and they tend to have worse psychological distress than people with GAD without these disturbances. Female sex, disease duration, and autonomous (intrinsic) versus controlled (extrinsic) motivation also affect rates of sexual dysfunction and sleep disturbances in patients with GAD. Providers can target sleep quality and sexual function to improve quality of life of people with gluten-associated disorders.

Table 1. Measures associated with presence of sleep disturbance or sexual dysfunction. Data represent score Median (Q1-Q3) unless noted otherwise

	GAD	GAD + Sleep Disturbance	GAD + Sexual Dysfunction	*P*-value
Self-Reported Patients				
Sleep Disturbance = n (%)	12 (35.1%)	24 (64.9%)		
Sexual Dysfunction = n (%)	4 (15.4%)		22 (84.6%)	
GFD Adherence (CDAT)				
	14.0 (10.0-14.0)	14.0 (12.0-16.0)		0.325
	13.0 (11.8-14.0)		14.0 (12.0-16.0)	0.526
Celiac Disease Activity (CSI)				
	36.5 (30.8-47.2)	37.0 (32.0-42.0)		0.942
	34.0 (31.2-37.5)		40.5 (36.2-45.5)	0.162
CSI Categories	*Sleep / Sex*			*Sleep / Sex*
Active Disease	37.5% / 0%	9.5%	25%	
Moderate Disease Control	37.5% / 75%	71.4%	60%	0.112 / 0.586
Disease Remission	25% / 25%	19%	15%	
Anxiety (PROMIS-29)				
	7.0 (6.0-10.0)	7.5 (6.0-12.0)		0.634
	7.0 (6.2-8.0)		9.5 (7.0-12.0)	0.184
Depression (PROMIS-29)				
	6.0 (4.0-6.0)	5.5 (4.0-11.0)		0.610
	6.5 (4.8-8.8)		6.0 (4.0-10.0)	0.970
Sleep (PROMIS-29)				
	6.0 (5.0-8.0)	10.0 (9.0-11.0)		< 0.001
	11.0 (9.5-12.2)		10.0 (8.0-11.0)	0.297
Fatigue (PROMIS-29)				
	8.0 (6.0-13.0)	12.0 (9.5-14.5)		0.174
	11.0 (8.5-13.0)		12.5 (9.8-14.2)	0.612
Satisfaction with Social Interaction (PROMIS-29)				
	13.0 (9.0-16.0)	10.0 (8.0-14.0)		0.234
	11.5 (7.8-15.2)		10.0 (8.0-13.0)	0.694
Pain (PROMIS-29)				
	10.0 (6.5-14.0)	8.0 (7.0-12.0)		0.727
	8.0 (8.0-9.5)		9.0 (6.8-12.0)	0.963
Relationship with Healthcare Provider (HCCQ)				
	25.5 (16.0-31.2)	21.0 (14.0-26.0)		0.304
	19.0 (12.0-25.2)		18.0 (9.0-27.0)	0.710

S1579

Prevalence and Characteristics of Gastrointestinal Symptoms in Hospitalized COVID-19 Patients in Douala—Cameroon, West Africa

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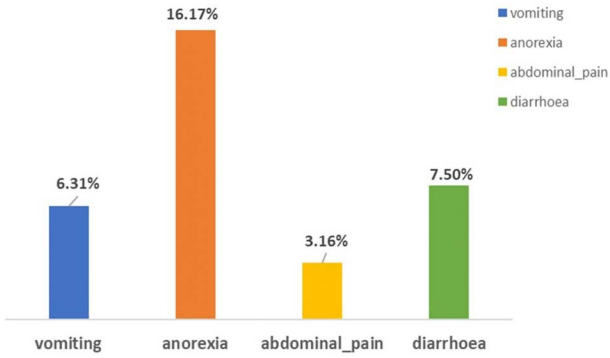
Introduction: Coronavirus 2019 (COVID-19) disease caused by SARS-CoV-2 primarily affects the respiratory system, but a multi-systemic involvement has been reported, including the gastrointestinal tract (GIT) causing GIT symptoms. This study aims to characterize GIT symptoms in hospitalised COVID-19 patients in 2 tertiary hospitals in Cameroon.
Methods: We conducted a retrospective analysis of hospital records of patients admitted at the COVID-19 units of 2 tertiary medical centers in Cameroon, between March 2020 to December 2020. Data on demographics, clinical characteristics, and laboratory and radiologic data of COVID-19 confirmed patients were included in the final analysis. Demographics included age and gender, clinical characteristics included gastrointestinal symptoms on admission (vomiting, anorexia, abdominal pain, and vomiting), and radiologic data were defined as findings consistent with COVID-19. R version 4.2 was used to analyze descriptive data, the chi-square test was used to test for associations, and P was considered significant at $P< 0.05$.
Results: Overall, A total of 650 files were reviewed, and 507 participants met the inclusion criteria and were included in the final analysis. 305 (65%) were males, and the mean was age 54 ± 14 years (Table). Patients were generally overweight to obese (BMI 30.7±6.8kg/m2), with the majority having a cardiovascular risk factor or comorbid condition 301(59.4%). Baseline demographics were not different among those presenting with gastrointestinal symptoms compared to those without. A total of 142 (28.%) patients had at least one gastrointestinal symptom. The most common symptoms were anorexia 82 (16.%), diarrhea 38 (7.5%), and vomiting 32 (6%) (Figure). The average day of appearance of GI symptoms before admission was 8.8 days. Laboratory results on admission revealed no significant differences in leukocyte count, hemoglobin, platelets, and liver enzymes. The median duration of hospital stay was 7 days, and it did not differ between those with or without GI symptoms (7 vs 8 days $P= 0.3$). Overall mortality in-hospital mortality was 161(31.7%); there was no statical significant difference in the mortality rate of patients who presented with GI symptoms (30 vs 23.6 $P= 0.16$).
Conclusion: One in 4 persons hospitalized for COVID-19 in Douala had at least a gastrointestinal symptom. Therefore, COVID 19 testing should be considered for patients with GI symptoms.



[1579] **Figure 1.** Prevalence of Gastrointestinal symptoms in hospitalized COVID-19 patients

Table 1. Sociodemographic characteristics of hospitalized COVID-19 patients

Variable	n (%)
Age (mean ± SD)	54.24 ±14.25
Gender	
Male	305 (65)
Female	202(39.6)
Level of education	
Primary	90 (17.6)
Secondary	194 (38.3)
University	221 (43.6)
BMI (Mean ± SD)	30.75 ±6.83

S1580

History of Tonsillectomy Is Seen to Have a Minimal Impact on the Small Intestinal Microbiome

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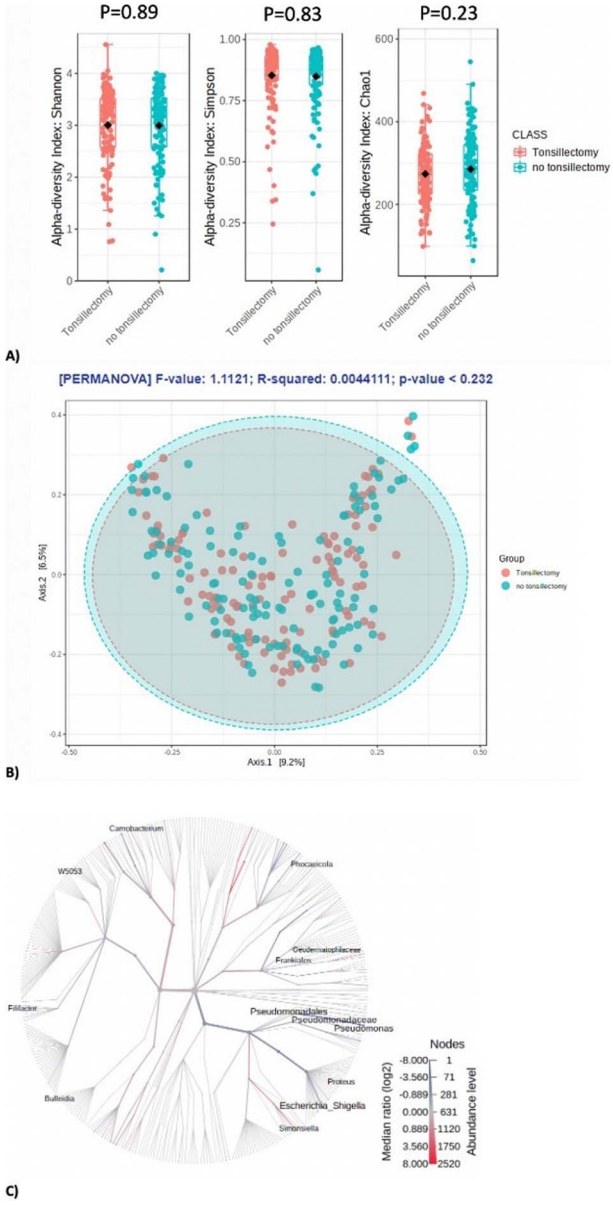
Introduction: Tonsillar tissue is an important immune reservoir and serves as an early activator of the immune system. Despite the link with infection and immunity, to date there is no study examining the effect of tonsillectomy on the small bowel microbiome. Here, we compare small bowel microbial profiles in subjects with or without tonsillectomy.
Methods: The REIMAGINE study is a novel large-scale study using validated methods for duodenal aspirate collection and microbiome sequencing in subjects undergoing upper endoscopy without colon prep. Subjects were divided into 2 groups: Self-reported undergoing a tonsillectomy (T+), and those who did not undergo a tonsillectomy (T-). Groups were matched for sex, age ±5 years, and BMI ±3 kg/m^2. Duodenal aspirates were collected, and microbial DNA isolated using the MagAttract PowerSoilDNA Kit. V3 and V4 libraries were sequenced on MiSeq. Reference based Operational Taxonomic Unit clustering was performed using SILVA v132 database. Taxonomic analyses were performed with CLC Microbial Genomics Module v.2.5 and MicrobiomeAnalyst. Blood was collected for inflammatory biomarkers and biochemical analysis, including lipid and glucose profiles.
Results: 253 subjects were included (T+, N=126, mean age = 62±15, mean BMI = 26.12±5.55) (T-, N=127, mean age = 63±13, mean BMI = 25.62±5.75). There was no significant difference in blood level of any analyte between groups. There was no statistical difference in duodenal microbial alpha-diversity (P >0.05) or beta-diversity (P >0.05) between the T+ group and T- group (Figure A, B). The most prevalent phylum in the core duodenal microbiome of the T+ group was Firmicutes, followed by Actinobacteria and Proteobacteria. While Firmicutes was also the most prevalent phylum in the T- group, Proteobacteria moved up in rank to the second most prevalent phylum, and Actinobacteria dropped to third most prevalent. There were no major higher order taxonomic differences between T+ and T- groups (Figure C). At genus level, *Streptococcus* (FC= 0.1, FDR P-value=1) (an organism known to infect the oropharynx) was not significantly different between groups. The only genus change observed was an increase in the relative abundance of *Granulicatella* in the T+ group (FC= 2.02, FDR P-value=0.01).
Conclusion: Our findings demonstrate that history of tonsillectomy was associated with very few changes in the small bowel microbiome. Of note, organisms such as *Streptococcus* were not altered in subjects with prior tonsillectomy.



[1580] **Figure 1.** A) Alpha-diversity box plots comparing the duodenal microbial diversity in the Tonsillectomy group (T+), and the no Tonsillectomy group (T-), as determined using 3 different indices, Shannon's, Simpson's and Chao1. B) Principal Component Analysis (PCA) plot of the duodenal microbiome beta-diversity of Tonsillectomy group (T+) compared to no Tonsillectomy group (T-). C) Heat tree illustrating minimal changes to the microbiome in subjects who reported undergoing a tonsillectomy (T+) relative to those who did not (T-). Nodes represent taxonomic levels, and greater line thickness denotes greater relative abundance. Taxa with higher relative abundance in the T+ group when compared to the T- group are shown in red. Taxa with higher relative abundance in the T- group when compared to the T+ group are shown in blue. Taxa with similar relative abundance in both groups are shown in gray

S1581

Development of Ex Vivo and In Vivo Models to Assess the Performance of an Oral Biotherapeutic Delivery System Capsule

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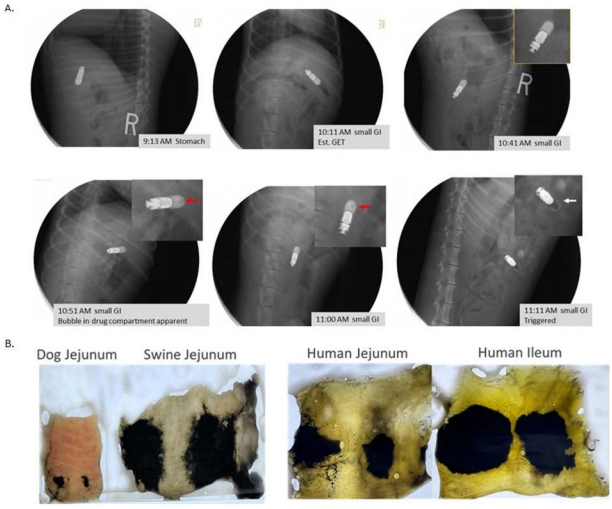
Introduction: Biologics/peptides/nucleic acids are highly effective drugs; however, oral delivery of these therapeutics has proved to be difficult due to the harsh conditions of the upper gastrointestinal tract (GIT) and the poor absorption rate in the small intestinal mucosa. We aim to develop an oral biotherapeutic delivery system (OBDS) that prevents drug degradation in the upper GI and increases bioavailability via submucosal injection. The OBDS capsule operates autonomously and triggers a needleless injection to deposit the liquid drug payload into the submucosal space of the proximal small intestine for absorption. The objective of this study was to assess the autonomous trigger function and performance in the beagle dog model.
Methods: Two versions of autonomous OBDS capsules (Type 1 and 2) were filled with contrast reagent, iohexol (OMNIPAQUE 350), and orally dosed to beagle dogs. Fluoroscopy images were taken every 5-15 mins post-dose to visualize capsule deployment of iohexol to assess the deployment time and approximate location post gastric emptying in the intestine. In parallel, OBDS capsules with Black India ink were inserted into intact sections of the small intestine in a wet chamber and manually triggered to compare ink deposition on *ex-vivo* small intestine tissues of dog vs swine vs human.
Results: We observed 10 out of 12 capsules dosed orally deployed approximately at the small intestine (Figure A), one deployed in the colon, and one did not deploy due to manufacturing defects. The overall triggering time was consistent in each group, with trigger type 1 capsules having an average deployment of 1h and 8 min ± 5 min post gastric emptying (N=7), approximately 14 minutes faster than trigger type 2 capsules (N=4). Lower and less consistent ink deposition on the beagle dog GI tissue was observed compared with swine or human tissue (Figure B). This suggests a lower capacity of canine tissue to accept a liquid bolus as expected due to differences in canine intestinal anatomy compared to humans and swine.

Conclusion: We have successfully shown ≥ 83% deployment accuracy of autonomous OBDS capsules in the small intestine without early deployment in the stomach. These results suggest the beagle dog model can be utilized to assess the consistency and reproducibility of the OBDS capsule. However, an assessment of submucosal injection will need to be performed in the swine model for human translation of the oral bioavailability.



[1581] **Figure 1.** In-Vivo and Ex-vivo performance of OBDS device. A. Representative image of capsule deployment in the small intestine. The drug module is noted with the red arrow. The layer of tungsten on the piston is noted by the White arrow. B. Ex-vivo tissue deposition of India ink in intact bowel tissue in dog vs swine vs human tissue

S1582

Abnormal Glucose Breath Test Does Not Correlate With Patient's Self-Reported Symptoms and Their Severity

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Introduction: Glucose Hydrogen Breath Testing (GBT) is commonly used for diagnosing Small Intestinal Bacterial Overgrowth (SIBO). However, patients present with clinical symptoms (sx) and their predictive value for a positive (pos) GBT remains unclear. AIM: To assess the prevalence of self-reported GI sx profiles in adults with unexplained upper GI symptoms and correlate this with GBT.
Methods: We retrospectively analyzed all adult patients presenting to a single medical center from 6/20 –12/21 for GBT. A pos GBT for SIBO was defined by the North American Consensus (≥20 ppm H2 increase over basal within 90 minutes (mins) and/or ≥10 ppm CH4 at any time). Pts completed a GI questionnaire assessing 17 key sxs: regurgitation (RG), chest pain (CP), heartburn (HB), belch (BH), abdominal pain (AP), bloating (BT), gas (GS), nausea (NA), vomit (VT), abdominal cramps (AC), indigestion (ID), distension (DN), fullness (FL), early satiety (ES), diarrhea (DR), constipation (CN), and fatigue (FT). Sxs measured by frequency (None, < 1/week (wk), 1/wk, ≥ 1/wk), intensity (None, Mild, Moderate, Severe), and duration (None, < 10 mins, 10 – 30 mins, ≥ 30 mins). Sub-groups, GERD, Dyspeptic, and IBS were constructed assessing the mean summation scores for Frequency, Intensity, and Duration between negative (neg) and pos GBTs. Univariate analysis and logistic regression analysis was performed. A *P*-value of < 0.05 was considered statistically significant.
Results: We evaluated 184 pts, mean age of 51.7 (18 – 85), 78.3% female, 82.8% Caucasian, and mean BMI of 27.2. No significant differences were seen between GI sx frequency and intensity categories for pts with GBT neg and pos groups. A trend for a higher percentage of pos GBT pts reporting FL (P=0.09) and DR (P=0.07) in sx duration categories of 10 -30 mins (FL: 30.8% vs 27.5%; DR: 31.8% vs 23.4%) and ≥ 30 mins (FL: 51.3% vs 41.3%; DR: 37.2% vs 22.1%) was noted. Regression analysis showed a trend toward a pos GBT for VT frequency of Less than 1/wk: (P = 0.07; OR = 2.99; 95% CI: 0.91, 9.83) and NA intensity of Severe: (P = 0.09; OR = 2.24; 95% CI: 0.86, 5.84). No differences were seen between the GERD, Dyspeptic, and IBS sub-groups (Figure).
Conclusion: The pre-test predictability of GI symptoms as it relates to a pos or neg GBT is poor. The yield for a pos GBT varies between 31%-55% and depends on age. In routine practice, when the index of clinical suspicion is high for SIBO, clinicians should consider objective testing with GBT rather than empiric antibiotic treatment.

Mean Summation Score	Negative Glucose Hydrogen Breath Test (n = 93)	Positive Glucose Hydrogen Breath Test (n = 91)	P-Value
Frequency			
Mean GERD Category	2.3 (2.0)	2.5 (2.1)	0.62
Mean Dyspeptic Category	10.7 (4.7)	11.5 (5.4)	0.33
Mean IBS Category	4.6 (2.2)	4.7 (2.2)	0.83
Intensity			
Mean GERD Category	2.3 (2.0)	2.5 (2.1)	0.62
Mean Dyspeptic Category	10.7 (4.7)	11.5 (5.4)	0.33
Mean IBS Category	4.6 (2.2)	4.7 (2.2)	0.83
Duration			
Mean GERD Category	2.6 (2.3)	2.6 (2.2)	0.96
Mean Dyspeptic Category	10.4 (4.3)	11.2 (5.0)	0.25
Mean IBS Category	4.8 (2.3)	5.1 (2.3)	0.46

	Age Group: 18-59 N = 2682	Age Group: 60-80 N = 1514	Age Group: ≥ 81 N = 213	P value
Positive GBT	30.2%	39.8%	54.9%	<0.001
Positive GBT: H2 Only	70.6%	68.4%	47.8%	<0.001
Positive GBT: CH4 Only	21.0%	19.4%	36.9%	<0.001
Positive GBT H2 and CH4	8.4%	12.2%	15.3%	<0.001
Mean Peak H2 (ppm)	21.7	30.2	29.6	<0.001
Mean Peak CH4 (ppm)	9.6	13.6	27.4	<0.001
Peak H2 (minutes)	63.6	73.7	76.2	<0.001
Peak CH4 (minutes)	62.2	68.8	61.1	0.02

[1582] **Figure 1.** (Left) Mean Summation Scores for Self-Reported Frequency, Intensity, and Duration Responses to GI Symptoms Relative to the Gastroesophageal Reflux Disease (GERD), Dyspeptic, and irritable bowel syndrome (IBS) Groups. (Right) Prevalence of Positive Glucose Breath Test (GBT) Among Age Cohorts and Glucose Breath Test Measurements

S1583

Surveillance With Double Balloon Enteroscopy in Adult Patients With Peutz-Jeghers Syndrome: A Single Center Experience

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Introduction: Peutz-Jeghers syndrome (PJS) is an inherited polyposis syndrome caused by mutation in the STK11 gene, and characterized by hamartomatous polyps throughout the gastrointestinal tract, most often in the small bowel (60-90%) and colon (50-64%). Presenting symptoms in PJS are often related to symptomatic polyps, including intestinal obstruction, abdominal pain, and bloody stools. For PJS patients, current recommendations support screening upper endoscopy, colonoscopy, and video capsule endoscopy at age 8-10, and if no polyps identified then again at age 18. Small bowel screening is recommended every 2-3 years. Deep enteroscopy, via antegrade or retrograde balloon enteroscopy, is often required for clearance of larger polyps visualized on video capsule endoscopy (VCE). The optimal follow-up strategy and modality of screening and surveillance of small bowel polyps is controversial. We aimed to evaluate if proactively performing deep enteroscopy vs serial VCE in adult PJS patients reduces risk of requiring subsequent surgical intervention.
Methods: We retrospectively collected data from adult patients over 18 years of age with Peutz-Jeghers syndrome who underwent deep balloon enteroscopy for small bowel surveillance. Patients within this subset underwent proactive screening with antegrade double balloon enteroscopy performed approximately every one to 2 years. Data collected included number of prior small bowel surgeries before onset of and during deep enteroscopy surveillance, and complications.
Results: Thirteen patients underwent deep enteroscopy from 2010 through 2021. Average number of prior small bowel surgeries was 1.9 (SD 1.4), average number of balloon enteroscopies during surveillance period 2.8 (SD 1.4), and average number of surgeries during balloon enteroscopy surveillance 0.46 (SD 0.66). There was a significant difference between number of surgeries before and during surveillance period (P=0.002).

Conclusion: The initial standard screening modality for small bowel polyps and malignancy, VCE, can be complicated by increased risk of capsule retention given likelihood of prior surgery and possibility of obstructing polyps. Additionally, within the PJS cohort, there is a high yield for pathology which may ultimately require therapeutic deep enteroscopy anyhow. Our study, though small, does indicate that on a proactive balloon enteroscopy surveillance protocol there is a significantly lower risk of requiring small bowel surgery.

S1584

Prevalence of Non-*Helicobacter pylori*Peptic Ulcers in Patients With Chronic Pancreatitis: A Population-Based National Study

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Introduction: Acute and chronic pancreatitis are recognized as a continuum of the same disease, with a significant overlap in clinical manifestations. The association between peptic ulcer disease (PUD) and acute pancreatitis (AP) has been described in the literature. However, there is no available data on the prevalence of PUD in patients with chronic pancreatitis (CP).
Methods: The aim of this study was to investigate if CP is associated with increased overall risk of PUD, gastric ulcers, and duodenal ulcers. Data was collected from a commercial database (Explorys Inc, Cleveland, OH), an aggregate of EHR data from 27 integrated healthcare systems in the US between 12/2016-12/2021. We identified patients with CP based on Systemized Nomenclature of Medicine – Clinical Terms and excluded patients with *Helicobacter pylori* infection of the gastrointestinal tract. We compared the prevalence of peptic ulcer, gastric ulcer, and duodenal ulcer after at least 30 days following a diagnosis of CP to a cohort without CP.
Results: Of the 33,660,510 patients in the database without *H. pylori*, we identified 66,550 cases of CP. We identified 2,480 cases of peptic ulcer, 2,680 cases of gastric ulcer and 1,420 cases of duodenal ulcer in patients with CP. In comparison, we identified 160,270 cases of peptic ulcer, 201,600 cases of gastric ulcer and 81,920 cases of duodenal ulcer in patients without CP. Overall prevalence for peptic ulcer, gastric ulcer, and duodenal ulcer in patients with CP was 3727/100000, 4027/100000 and 2134/100000 people, respectively. In comparison, overall prevalence for peptic ulcer, gastric ulcer, and duodenal ulcer in patients without CP was 477/100000, 600/100000 and 244/100000 people, respectively. The prevalence ratios (PR) for developing each diagnosis after at least 30 days of a CP diagnosis were 7.81 (95% CI 7.51-8.12, $P<$ 0.001), 6.71 (95% CI 6.47-6.97, $P<$ 0.001), and 8.75 (95% CI 8.31-9.22, $P<$ 0.001), respectively. Table 1 describes the prevalence of PUD among various demographics.
Conclusion: In this large population-based study, peptic ulcer, gastric ulcer, and duodenal ulcer were all significantly more prevalent among patients with a diagnosis of CP. Future investigation will be useful in understanding the pathophysiology of this association.

Table 1. Prevalence of peptic ulcer, gastric ulcer and duodenal ulcer in patients with and without chronic pancreatitis

	Chronic Pancreatitis (CP)	No CP	Prevalence in CP /100,000	Prevalence in No CP /100,000	Prevalence Ratio	95% CI	*P*-value
Peptic Ulcer	2480	160270	3726.52	477.08	7.81	7.51-8.12	< 0.001
Adults (18-65)	1640	74130	3964.23	374.11	10.60	10.1-11.1	< 0.001
65)"}" > Elderly (>65)	840	85440	3362.69	1088.74	3.09	2.89-3.30	< 0.001
Caucasian	1700	119480	3739.55	657.53	5.69	5.43-5.96	< 0.001
African American	670	24730	5153.85	648.38	7.95	7.38-8.57	< 0.001
Asian	20	2860	2985.07	536.31	5.57	3.61-8.58	< 0.001
Male	1210	64750	3587.31	434.60	8.25	7.81-8.73	< 0.001
Female	1270	95450	3861.36	516.79	7.47	7.08-7.89	< 0.001
Gastric Ulcer	2680	201600	4027.05	600.11	6.71	6.47-6.97	< 0.001
Adults (18-65)	1730	88420	4181.77	446.23	9.37	8.95-9.82	< 0.001
65)"}" > Elderly (>65)	940	112280	3763.01	1430.75	2.63	2.47-2.80	< 0.001
Caucasian	1830	149090	4025.52	820.48	4.91	4.69-5.13	< 0.001
African American	740	28950	5692.31	759.02	7.50	6.99-8.05	< 0.001
Asian	30	3690	4477.61	691.96	6.47	4.56-9.19	< 0.001
Male	1260	77990	3735.55	523.46	7.14	6.76-7.54	< 0.001
Female	1420	123300	4317.42	667.58	6.47	6.15-6.81	< 0.001
Duodenal Ulcer	1420	81920	2133.73	243.85	8.75	8.31-9.22	< 0.001
Adults (18-65)	860	31370	2078.80	158.31	13.10	12.3-14.0	< 0.001
65)"}" > Elderly (>65)	560	49880	2241.79	635.61	3.53	3.25-3.83	< 0.001
Caucasian	960	60480	2111.75	332.84	6.35	5.96-6.76	< 0.001
African American	400	11250	3076.92	294.96	10.40	9.46-11.5	< 0.001
Asian	20	1720	2985.07	322.54	9.26	6.00-14.3	< 0.001
Male	850	45120	2520.01	302.84	8.32	7.78-8.90	< 0.001
Female	570	36660	1733.05	198.49	8.73	8.04-9.48	< 0.001

SMALL INTESTINE

S3358 Presidential Poster Award

Exploring Probe-Based Confocal Laser Endomicroscopy as a Novel Tool in Graft Monitoring After Small Bowel Transplantation

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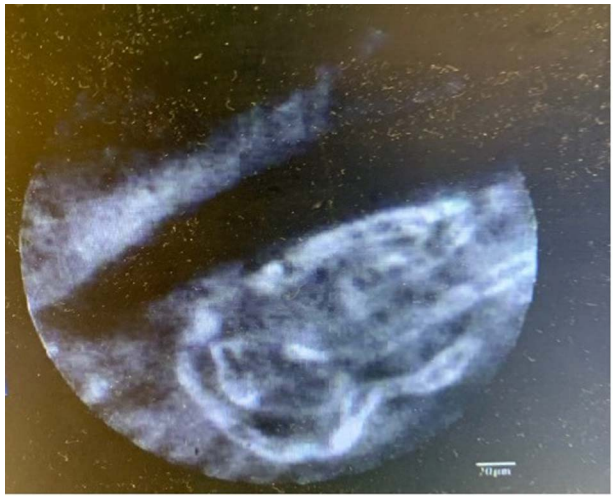
Introduction: The most common and severe complication early in Small Bowel Transplant (SBT) is graft rejection making timely detection key to graft preservation. Endoscopic surveillance with protocol biopsy is the gold standard for graft monitoring. Confocal Laser Endomicroscopy (CLE) has emerged as a modality for real-time diagnosis at a histological scale. However, its role in SBT is not known. Herein, we report 3 cases of SBT patients and compare CLE results with standard protocols.
Case Description/Methods: Case 1: A 73-year-old man underwent SBT for sclerosing mesenteritis and total enterectomy. CLE and ileoscopy was performed 15 times post-transplant without findings of acute cellular rejection (ACR). Patient expired of sepsis on day 30 post-transplant. Case 2: A 17-year-old male with familial adenomatous polyposis complicated by total colectomy, hepatoblastoma s/p resection and chemotherapy underwent SBT for large desmoid tumor requiring total enterectomy. CLE and ileoscopy was performed 14 times post-transplant without findings of ACR. Case 3: A 23-year-old female underwent SBT for intestinal neuronal dysplasia after chronic rejection from 1st transplant 10 years ago. Ileoscopy done at 20 days post-transplant showed congested and ulcerated mucosa in the proximal ileum. CLE images and histopathology showed findings consistent with ACR. Immunosuppression was increased and reassessment with CLE and ileoscopy done.
Discussion: Cases 1 and 2 show normal CLE assessment. Normal villus architecture, non-edematous, and adequate microcirculation suggesting low suspicion for ACR. This correlated with normal pathology characterized by less than 6 apoptotic bodies per 10 crypts, no intravascular neutrophils, and negative viral cytopathology. However, in case 3, CLE images showed significant villus congestion and decreased microcirculation (Figure) indicative of ACR. Biopsies confirmed ACR. Rejection protocols were rapidly initiated.Repeat CLE and ileoscopy showed recovery of mucosal congestion and return of microcirculation. This correlated with complete resolution of ACR on pathology. In the absence of endoscopic criteria for small bowel graft rejection, biopsies are the gold standard. Enhanced technology for in vivo visualization of grafted bowel is needed and would allow for examination of a greater surface area of the bowel than limited biopsies. CLE is a promising tool that could significantly improve early transplant graft monitoring. Further data and prospective studies are needed.



[3358] **Figure 1.** CLE images showing significant villus congestion, and decreased microcirculation.

S3359 Presidential Poster Award

Diaphragm Disease: A Crohn's Mimicker?

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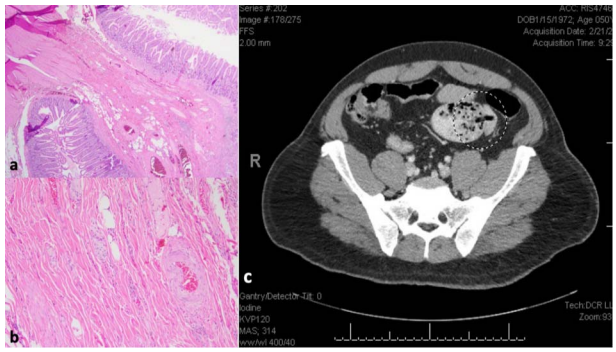
Introduction: Diaphragm disease of the small bowel is a rare disease typically associated with NSAID use. The disease is characterized by multiple strictures appearing as 'pinhole' sized lumens caused by mucosal thickening. Often the condition goes misdiagnosed until the lumen is visualized, with clinical presentations initially consisting of recurrent bowel obstructions, anemia and/or protein loosing enteropathy.
Case Description/Methods: A 50-year-old male with a history of hypertension, incarceration, and prior recurrent small bowel obstructions and prior exploratory laparotomy with lysis of adhesions, presented with recurrent abdominal pain, nausea, vomiting and constipation. A year prior to presentation, the patient was hospitalized for similar symptoms, with colonoscopy demonstrating a fibrotic stricture at the ileocecal valve, which was only able to be traversed after balloon dilation. The mucosa appeared endoscopically normal, however, biopsies of the terminal ileum demonstrated chronic active focal ileitis, suggestive of Crohn's disease. The patient then experienced two more hospitalizations for small bowel obstruction, which resolved with supportive care and steroid administration. During this hospitalization, CT scan demonstrated a small bowel obstruction with several transition points in the mid small bowel. Intravenous corticosteroids were started, and nasogastric tube was placed with improvement. He adamantly denied any current or past prolonged NSAID use. It was decided that patient would benefit from a surgical small bowel exploration. Several strictures were seen in the proximal small bowel, and therefore proximal and distal resection of jejumum with a primary end-to-end enteroenterostomy anastomosis was performed. Histopathological evaluation of the resected specimen showed small bowel with circumferential membranes with submucosal neuromuscular and vascular hyperplasia, displaced mucosal glands, and associated mucosal active and chronic ileitis with ulceration, compatible with a diagnosis of diaphragm disease. (Figure)
Discussion: NSAIDs can have deleterious effects on the small bowel including, ulceration, structuring, erosion formation, perforation and/or the formation of fibrous diaphragms that can lead to obstruction. Diaphragm disease is uncommon, especially in the absence of prior NSAID use. Clinically, it is important to recognize this group of patients as obstructive symptoms due to structuring/diaphragm disease are unlikely to resolve without some form of surgical intervention.



[3359] **Figure 1.** a + b) Submucosal fibrosis and neuromuscular and vascular hyperplasia. c) Focal narrowing and proximal dilation of a loop of small bowel with transition point within the mid small bowel.

S3360 Presidential Poster Award

Laparoscopic-Assisted Enteroscopy with "Shar Pei" Technique for Resection of Deep Small Bowel Polyps

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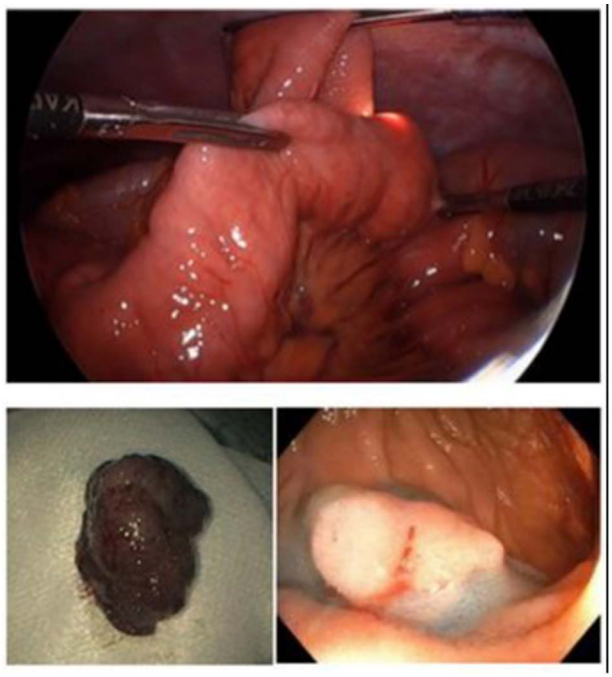
Introduction: Balloon enteroscopy (BE) has transformed the management of small bowel disease. However, BE still has limits in terms of reaching deeper parts of the small bowel. Laparoscopic-assisted enteroscopy (LAE) has emerged as an effective procedure for small bowel polyps. We present 2 cases of LAE using a novel Shar Pei technique.
Case Description/Methods: Case 1: A 71-year-old female who underwent bidirectional endoscopy and video capsule endoscopy (VCE) for work-up of iron deficiency anemia. VCE revealed a non-obstructing polypoid lesion with minimal oozing in the proximal small bowel at 15% small bowel transit time (SBTT). Anterograde BE revealed a flat, 10-mm polyp in the proximal jejunum. Due to unstable positioning, the polyp was removed incompletely via piecemeal cold snare polypectomy (CSP). She subsequently underwent LAE with Shar Pei technique to pleat the small bowel over the enteroscope until the polyp was reached. The polyp was removed en bloc via endoscopic mucosal resection (EMR). Case 2: A 23-year-old female with Peutz-Jeghers syndrome was found to have 2 polypoid lesions in the small bowel on surveillance imaging. VCE revealed 2 polyps at 32% and 82% SBTT, respectively. The polyps were not reachable via anterograde and retrograde BE. A LAE was performed and the small bowel was pleated using the Shar Pei technique until a 50 mm semi-pedunculated polyp was visualized by the endoscope 320 cm from the Ligament of Treitz. The polyp was removed en bloc via EMR. Next, a colonoscope was advanced to the terminal ileum which was pleated laparoscopically until three polyps were seen 25 cm past the ileocecal valve that were removed with en bloc EMR. (Figure)
Discussion: LAE is an effective, minimally invasive technique for the management of deep small bowel pathology. LAE preserves the mucosal integrity eliminating the need for anastomoses. LAE has a high detection rate for small intestinal disease and is generally safe. No immediate complications were noted in our cases. Our novel Shar Pei technique was named after the dog breed whose characteristic wrinkled skin resembles the folds of the small bowel. The technique entailed laparoscopically advancing the small bowel over the endoscope while it remained stationary. The proximal end of the small bowel was stabilized during polyp resection to secure the endoscope and prevent telescoping backwards. LAE with the Shar Pei technique is a novel, promising tool for the diagnosis and treatment of small bowel polyps.



[3360] **Figure 1.** Panel 1. (Top) Laparoscopic-assisted enteroscopy with Shar Pei technique was utilized to telescope the small bowel over the endoscope until the 50-mm polyp was successfully reached at 320 cm from the Ligament of Treitz and resected with EMR (bottom left). This technique was also used to resect a 10-mm jejunal polyp with en bloc EMR (bottom right).

S3361 Presidential Poster Award

Liposarcoma Masquerading as IgG4-Related Disease

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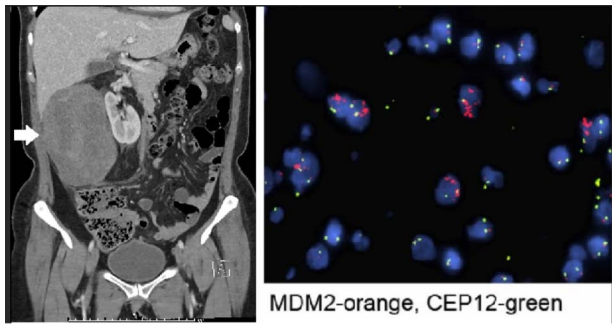
Introduction: IgG4-related disease (IGRD) is characterized by lymphoplasmacytic tissue infiltration by IgG4-positive plasma cells and small lymphocytes and is known to involve multiple organs, sometimes simultaneously.[1] Retroperitoneal involvement is common with IGRD, often presenting as mass-like lesions and fibrosing disease such as retroperitoneal fibrosis[2] and sclerosing mesenteritis[3]. While there is no established association, very few cases have been reported in literature about strong IgG4 positivity and sarcomas.[3,4] Investigation usually warrants biopsy, as imaging studies and serologies cannot differentiate between IGRDs and sarcomas. We present a case of an aggressive liposarcoma mimicking IGRD.
Case Description/Methods: A 40-year-old female presented with right upper quadrant (RUQ) pain for 3 months. Patient was hemodynamically stable with physical exam demonstrating fullness and tenderness in RUQ. CT abdomen revealed a 12.9x11.8x8.7 cm heterogenous macro-lobulated mass located below the inferior hepatic border in the retroperitoneal space and hepatorenal recess (Figure A). CT guided biopsy of the mass revealed increased IgG4-positive cells identified by immunohistochemical studies, however, FISH MDM2 amplification studies were inconclusive. Patient's serum IgG4 levels were elevated, and she was started on prednisone therapy. At 2 month follow-up, the patient's serum IgG4 levels had decreased but upon physical examination, the mass had increased in size and was firmer. Decision was made to repeat CT-guided biopsy, wherein MDM2 amplification studies revealed that the mass was consistent with well-differentiated liposarcoma (Figure B). She underwent liposarcoma resection, lymph node dissection, and right nephrectomy (adherent to the mass). Histopathologic analysis revealed a final diagnosis of stage I well-differentiated liposarcoma. Patient now follows for surveillance of her liposarcoma.
Discussion: IGRD and liposarcoma can have common initial presentations and at times overlapping serological markers such as IgG4 levels, which necessitates histopathologic analysis for confirmation of the disease. In addition, raised IgG4 levels are not specific and can be elevated in infections and autoimmune conditions[5]. Our case emphasizes the importance of considering liposarcomas in retroperitoneal masses with elevated IgG4 levels, as they can mimic IGRD. A repeat biopsy should be considered if the initial diagnosis is doubtful, or patient responds poorly to corticosteroids.



[3361] **Figure 1.** A) Coronal view of the CT scan showing a macrolobulated heterogenous mass measuring 10.6x10.5x14.1cm, B) MDM2 expression of mass using FISH.

S3362 Presidential Poster Award

MEN2B-Associated Chronic Intestinal Pseudo-Obstruction

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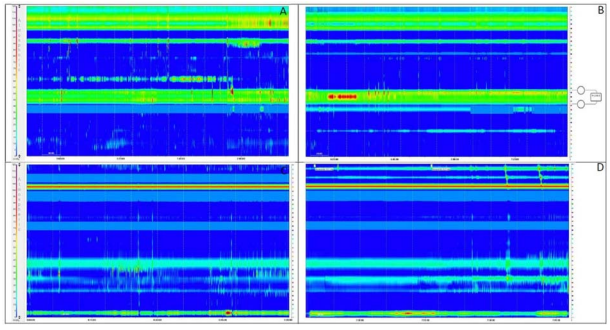
Introduction: Multiple endocrine neoplasia type 2B (*MEN2B*) is a rare genetic disorder characterized by medullary thyroid cancer and pheochromocytoma. Gastrointestinal (GI) involvement, if present, is often observed with onset at birth or shortly thereafter. GI symptoms may manifest as constipation that can progress to megacolon while involvement of other portions of the GI tract are less commonly reported. We report a case of *MEN2B* who presented with progressive gastrointestinal symptoms and was found to have chronic intestinal pseudo-obstruction (CIPO).
Case Description/Methods: A 23-year-old female with past medical history of *MEN2B* presented to our clinic for evaluation of abdominal pain, bloating, and loose stools. Since childhood, the patient had chronic abdominal distension and intermittent episodes of abdominal pain and distension. 8 months prior to presentation, her symptoms became increasingly severe and required several hospitalizations for IV hydration. Her symptoms continued to progress and became associated with significant weight loss, nausea, and anorexia, at which time she was seen in our clinic. On physical examination, her vital signs were stable, and she was in no acute distress. Examination notable for a thin female (BMI 16) with a distended, tympanic abdomen with hyperactive bowel sounds and diffuse tenderness. Laboratory findings and infectious workup were largely unremarkable. CT imaging showed marked distension of the entire colon as well as the small bowel without definite transition point. She underwent a bidirectional endoscopy which was notable for a normal upper endoscopy, gross dilation of the colon, and no stricture in the terminal ileum. Antroduodenal manometry and colonic manometry (**Figure**) performed at the time of endoscopy were consistent with CIPO of neuropathic origin. She was started on prucalopride 2 mg daily with significant improvement of her symptoms.
Discussion: We present an unusual case of *MEN2B*-associated gastrointestinal involvement manifesting as CIPO which suggests that small intestinal dysmotility may need to be considered in the workup and management of gastrointestinal symptoms in *MEN2B* patients. While gastrointestinal involvement in *MEN2B* classically presents with a pseudo-Hirschprung's type megacolon, this case also demonstrates that identification of chronic intestinal pseudo-obstruction, particularly in young patients, should also prompt workup for *MEN2B*.



[3362] **Figure 1.** Antroduodenal manometry with notable absence of MMCIII in the fasted state (panel A), and lack of stimulation in response to octreotide (panel B) suggestive of neuropathic CIPO and consistent with the presumed pathology of diffuse intestinal ganglioneuromatosis. Colon manometry revealed no high-amplitude phasic contractions (panel C) with a non-propagating response isolated to the proximal 10-15cm of the right colon to bisacodyl stimulation (panel D).

S3363 Presidential Poster Award

A Case of Progressive Refractory Diarrhea due to Zollinger-Ellison Syndrome

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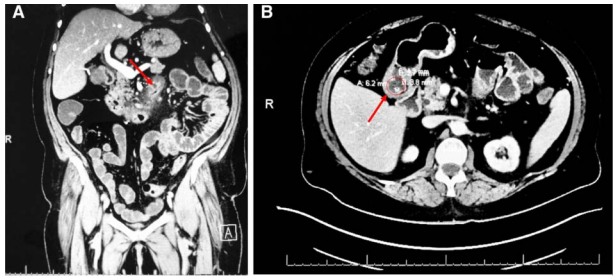
Introduction: A 64-year-old female with a history of diabetes mellitus presents with acute on chronic diarrhea and refractory acid reflux is found to have Zollinger-Ellison syndrome.

Case Description/Methods: The patient had a two-year history of non-bloody diarrhea which worsened (7 bowel movements/day) in the two months prior to presentation. She described associated nausea, reflux, and abdominal pain. Notably, her mother had similar symptoms, requiring small bowel resection (records not available). Previous workup included a colonoscopy and an esophagoduodenoscopy (EGD) showing duodenal ulcerations with negative biopsies for celiac disease. She was started on a proton-pump inhibitor (PPI), colestipol, and loperamide without improvement. Medication review, stool infectious studies, and cross-sectional imaging had been unrevealing. On presentation, she had stable vitals and physical exam significant for diffuse abdominal tenderness. Labs were notable for a hemoglobin of 10.2 gm/dL (grams per deciliter) and an acute kidney injury (AKI). Abdominal Computed Tomography (CT) suggested multiple, small duodenal ulcers with a polypoid lesion in the gastric antrum (Figure). EGD revealed esophagitis, multiple small duodenal ulcerations (D1, D2), and gastric mucosal atrophy. Repeat colonoscopy was unremarkable. The fasting gastrin level was 211 pg/mL (picogram per milliliter). The secretin stimulation test showed serum gastrin blood levels of 121, 258, and 483 pg/mL at 2, 5, and 10 minutes respectively consistent with Zollinger-Ellison Syndrome. The Dotatate PET/CT scan showed metastatic somatostatin receptor expressing portocaval adenopathy without a tracer-avid primary neuroendocrine tumor. She was started on lanreotide and is now referred to oncology.
Discussion: This case represents an instructive example of Zollinger Ellison Syndrome (ZES), often discussed, but rarely diagnosed. In this case, the patient presented with progressive diarrhea, a subtle family history, and with several prior evaluations. ZES is a rare gastrin-secreting neuroendocrine tumor (NET) associated with multiple endocrine neoplasia (MEN-1), which leads to refractory acid reflux and chronic diarrhea, as seen in our patient. Non-specific gastrointestinal symptoms and empiric PPI use can delay the diagnosis. The gastric pH, gastric level, secretin stimulation, and Dotatate PET/CT are used to evaluate and localize the tumor.



[3363] **Figure 1.** A, B: Computed Tomography (CT) abdomen showed multiple, small duodenal ulcers and 4 millimeter (mm) enhancing polyp at the gastric antrum (red arrows).

S3364 Presidential Poster Award

A Case of Systemic Mastocytosis Diagnosed Endoscopically

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Introduction: Mastocytosis, or mast cell proliferation has an annual incidence of 2/100,000. Only 10% of all Mastocytosis is considered systemic (SM); the most common site of involvement is the bone marrow, however of those ~60% will also have gastrointestinal symptoms. The colon is often affected most and endoscopically, there is mucosal nodularity, loss of normal architecture and friability with pathologically positive dense mast cell infiltration of the lamina propria. Indolent SM is characterized by both gastrointestinal and cutaneous symptoms, in the absence of bone marrow suppression and end-organ damage. It is extremely rare and often missed due to the complexity of diagnosis. We present a patient with abdominal pain, flushing and nausea who was diagnosed endoscopically with Systemic Mastocytosis, likely indolent type.
Case Description/Methods: A 39 YO F with no significant PMH presented to the GI clinic with dull and non-radiating RLQ pain associated with nausea and flushing of her face, lips, and ears. The patient appeared well-nourished and her examination, including perianal and digital rectal exams were unremarkable. She had a normocytic anemia (Hbg 11.7 g/dL, MCV 90.6 fL), but otherwise complements, CRP and ESR were normal. A CT abdomen/pelvis demonstrated a soft tissue nodule near the ileocecal region measuring 1.5x1.2 cm, concerning for a carcinoid tumor. Colonoscopy revealed numerous 5-15 mm yellow-white mucosal nodules with central hyperpigmentation visualized in the entire colon. There were discontinuous areas of non-bleeding and ulcerated mucosa in the transverse colon, ascending colon and cecum. Cold forceps biopsies from the ileal and colonic mucosa revealed sheets of eosinophils mixed with clusters of mast cells, showing atypical morphology including oval to short-spindled nuclei and focal clustering. IHC stains were positive for CD117, CD25, and Tryptase. OnkoSight KIT Sequencing detected Tier 1 genomic alterations in KIT pAsp816Val, strongly supporting the diagnosis of Systemic Mastocytosis. She was referred to Rheumatology for further workup.
Discussion: Mastocytosis is a rare disease characterized by heterogeneous clinicopathological features. In the absence of classical cutaneous lesions, bone marrow suppression and/or serum tryptase elevations, the diagnosis of Indolent Systemic Mastocytosis can be easily missed. As many patients lack cutaneous symptoms at time of diagnosis, the clinical suspicion should remain high if other more common diseases can be excluded.

S3365

Autoimmune Enteropathy: A Rare Cause of Small Intestinal Villous Atrophy in Adults

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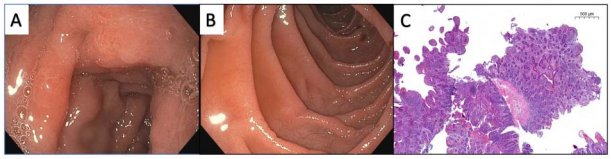
Introduction: Autoimmune enteropathy (AIE) is a rare cause of small intestinal villous atrophy. This condition is more frequently diagnosed in children, with only a handful of case series describing its presentation in adults. We present a case of adult-onset AIE in which diagnosis was made based on its unique endoscopic and histologic findings.
Case Description/Methods: A 54 year-old previously healthy woman was hospitalized with profuse watery diarrhea, abdominal pain, and weight loss of six months duration. Stool testing was negative for infection. Inflammatory markers were mildly elevated. Computer tomography imaging showed hyperenhancement of the small bowel consistent with enteritis, without evidence of stricture or abscess. She underwent esophagogastroduodenoscopy which revealed diffuse edema of the duodenal mucosa with villous blunting and cracked-earth appearance (**Figure A, B**). She had normal appearing mucosa of the distal small bowel and colon on video capsule endoscopy and colonoscopy, respectively. Pathology from duodenal biopsies showed chronic active duodenitis characterized by marked villous blunting, reduced goblet and Paneth cells, neutrophilic and lymphocytic infiltration of deep crypts, and basal layer apoptosis (**Figure C**). Similar findings were seen in random ileal biopsies, while random colon biopsies were normal. Anti-enterocyte antibodies and anti-transglutaminase antibodies were negative. Patient was treated for AIE with intravenous methylprednisolone and she required supplemental parental nutrition. Within days of initiating steroids, her diarrhea had improved and she was transitioned to oral prednisone. Several days later she was weaned off parenteral nutrition and stable for discharge with plans to introduce a thiopurine at follow-up.
Discussion: AIE should be suspected in a patient with chronic diarrhea, signs of severe malabsorption, and characteristic histologic features in absence of other causes of villous atrophy such as Celiac disease. Specific histologic features of AIE include absence of goblet cells and Paneth cells and presence of crypt apoptotic bodies in intestinal biopsies. Development of autoantibodies against enterocytes are associated with this condition, but not required for diagnosis. Steroids are the mainstay of therapy and have variable effect. We aim to raise awareness of AIE, as the rarity of this condition can make its diagnosis elusive.



[3365] **Figure 1.** 1A. EGD shows villous blunting and edema of duodenal mucosa. Figure 1B. EGD shows cracked-earth appearance of duodenal mucosa. Figure 1C. Duodenal biopsy reveals chronic active duodenitis characterized by marked villous blunting, reduced goblet and Paneth cells, neutrophilic and lymphocytic infiltration of deep crypts, and basal layer apoptosis.

S3366

Duodenitis as the Initial Manifestation of Adult Onset Henoch-Schonlein Purpura

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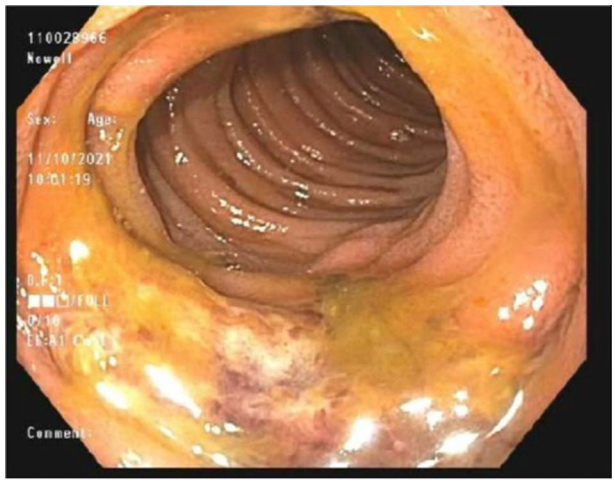
Introduction: Henoch-Schönlein Purpura (HSP) is the most common pediatric vasculitis. It classically presents with petechiae in the absence of thrombocytopenia and/or palpable purpura in addition to at least one of the four clinical symptoms of: abdominal pain, arthralgia, hematuria, and proliferative glomerulonephritis or leukocytoclastic vasculitis with predominantly IgA on histological evaluation. HSP presents primarily in pediatric populations, often in children prior to the age of 10. Adult-onset HSP has an incidence of 4/100,000. Common gastrointestinal symptoms include nausea, vomiting, and abdominal pain. Gastrointestinal manifestations occur in about 20% of patients prior to skin involvement. We present the case of a 30 year old female who presented with Adult Onset HSP Duodenitis.
Case Description/Methods: A 30 year old female presented with three episodes of hematemesis and two days of generalized abdominal pain. In addition she reported arthralgias and a lower extremity rash. Her exam revealed a palpable petechial rash on her bilateral buttocks, thighs, and legs as well as generalized abdominal tenderness. Initial labs revealed a white blood cell count of 19.29 K/μL. Contrast-enhanced computed tomography (CT) scan revealed wall thickening, submucosal edema, and surrounding fat stranding centered at the duodenum and proximal jejunum consistent with duodenitis. An esophagogastroduodenoscopy (EGD) was performed which revealed friable mucosa and numerous nonbleeding cratered and linear ulcers throughout the duodenum with the most prominent ulcers located in the second portion. Duodenal biopsies were consistent with mucosal ulceration. Left buttock biopsy revealed a leukocytolastic vasculitis with IgA deposition in vessel walls confirming the diagnosis of HSP. The patient was treated with intravenous corticosteroids for four days and had significant clinical improvement with resolution of her abdominal pain and vomiting. She was discharged with an oral steroid taper. (Figure)
Discussion: Although HSP remains primarily a disease of childhood, adult-onset HSP does occur and often with non-classical presentations. The small intestinal epithelial villi are most commonly affected due to the presence of end capillaries, making it susceptible to ischemic injury. It is therefore important to consider HSP in patients with duodenitis and extra-intestinal manifestations of arthralgias and rashes. The disease course is often self-limited however corticosteroids may lead to more rapid improvement in symptoms.



[3366] **Figure 1.** Duodenal Ulceration.

S3367

Olmesartan-associated Collagenous Gastrointestinal Disorders: Case Report and Review of the Literature

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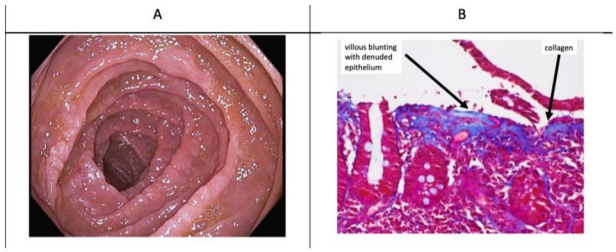
Introduction: Collagenous inflammation of the gut traditionally affects the colon, as part of microscopic colitis, and rarely the small bowel, as collagenous sprue. Angiotensin II receptor blocker (ARB) use has been implicated in sprue-like enteropathy. We present a rare case of olmesertan-associated pan-collagenous gastroenterocolitis.
Case Description/Methods: A 73-year-old man, with a history of tribenzor use (olmesartan-amlodipine-hydrochlorothiazide, 40 mg once a day for 5-10 years) for complicated hypertension and gastroesophageal reflux disease, developed severe diarrhea and weight loss. His work up included normal stool testing for parasitic and bacterial pathogens, normal pancreatic elastase, normal thyroid function test, normal celiac serology (tissue transglutaminase IgA, deaminated gliadin peptide IgA and IgG, and total IgA), and absent celiac genes (HLA-DQ2 and/or DQ8). He underwent pan-endoscopic evaluation showing a nodular gastric antrum and edematous duodenum with fissures and loss of folds, along with patchy nodularities of the right colon on colonoscopy. Histologic findings were consistent with subepithelial collagen thickening of his gastric antrum, duodenum, ileum, and right colon. He was diagnosed with malabsorption due to olmesartan-associated collagenous gastrointestinal (GI) disorders (CGID), and his symptoms resolved entirely following olmesartan discontinuation. (Figure)
Discussion: Our case highlights the effects of olmesartan on the entire GI tract and is the first time patchy collagenous pan-gastroenterocolitis, or CGID, as we prefer to term it, has been associated with ARB use. Healthcare providers should suspect this entity even in patients who have been using ARBs for years.



[3367] **Figure 1.** Endoscopic images of the edematous duodenum with scalloping and loss of folds (A) and corresponding histologic findings of subepithelial collagen and villous blunting of the epithelium on trichrome stain x 100 (B).

S3368

Bleeding From a Rare Omental Gastrointestinal Stromal Tumor Found on Capsule Endoscopy

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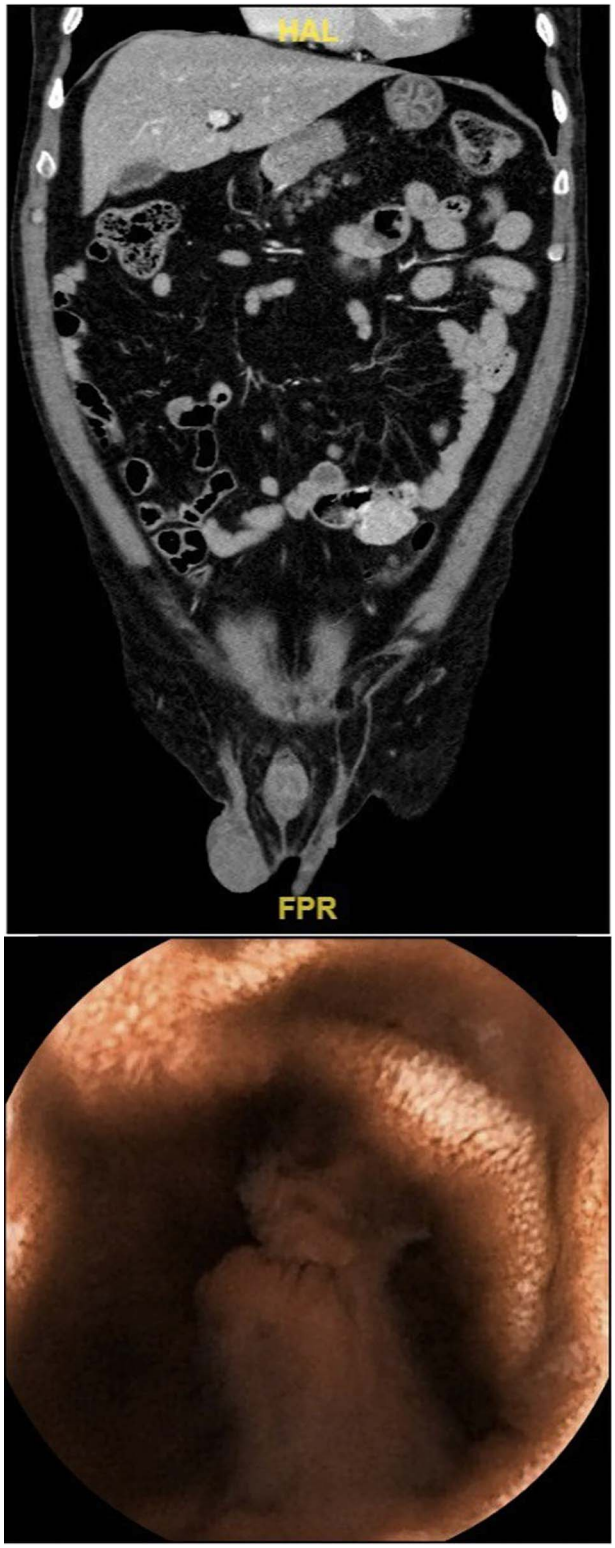
Introduction: Gastrointestinal stromal tumors (GISTs) are the most common mesenchymal tumors involving the gastrointestinal tract compromising approximately 1 -2 % of gastrointestinal cancers. They can arise from the bowel wall as a subepithelial neoplasm in the stomach and small intestine, and less than 5% of them arise as extragastrointestinal tumors in the retroperitoneum, mesentery and omentum. They occur in older adults aged 65 to 69 years old, and vary in their presentation depending on their location. We present a unusual case of small bowel gastrointestinal bleeding from a GIST adherent to the omentum.
Case Description/Methods: 58 year old male with a history of diabetes mellitus type II, hypertension, recent prolonged hospitalization for disseminated nocardia infection on long term antibiotics and a Decadron taper presented with several days of epigastric pain, nausea and non-bloody diarrhea. He denied any signs of gastrointestinal bleeding such as melena or hematochezia. His labs were notable for a hemoglobin of 4.2 g/dL with a mean corpuscular volume of 78.5 fL that was previously noted to be within normal limits. Computed tomography of the abdomen and pelvis with contrast showed a 4 cm circumscribed, hyperdense enhancing exophytic solid mass arising from the small bowel in the left lower quadrant with adjacent hyperenhancement. The patient subsequently underwent capsule endoscopy which showed fresh blood in the mid jejunum with more active bleeding surrounding a possible small bowel tumor. Due to ongoing bleeding and anemia, the patient underwent laparoscopic small bowel resection and lymphadenectomy of a small bowel tumor adherent to the omentum with about 10 cm of proximal and distal margins. Pathology was consistent with a low-grade gastrointestinal stromal tumor approximately 3.0 x 3.0 x 2.3 cms with tumor free margins and no evidence of lymphovascular spread. Thereafter, the patient remained hemodynamically stable with improvement of his blood counts and was discharged home. (Figure)
Discussion: While GISTS are common findings in the gastrointestinal tract, only a small percentage of them can cause gastrointestinal bleeding needing surgical intervention. Surgical resection remains the mainstay of treatment for localized, non-metastatic GISTs that present with bleeding as it can both stop the bleeding and resect the lesion. However, other modalities of treatment include endoscopic intervention depending on the location or transcatheter arterial embolization.



[3368] **Figure 1.** Top: CT Abdomen and Pelvis with contrast demonstrating a 4 cm circumscribed hyperdense enhancing exophytic solid mass arising from the small bowel in the left lower quadrant. Bottom: Capsule endoscopy showing a small bowel tumor.

S3369

Small Bowel Diverticulitis: A Unique Urinary Presentation

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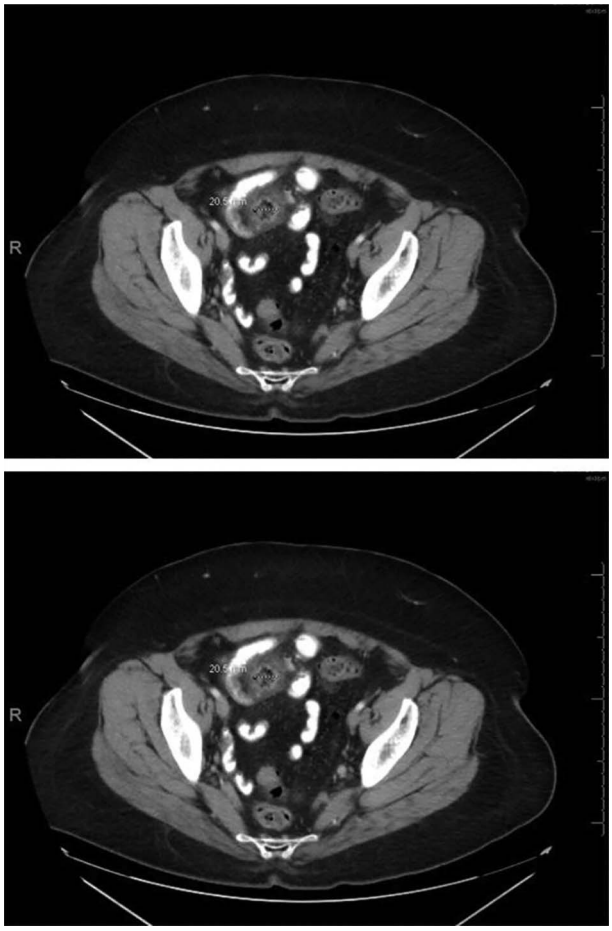
Introduction: Small bowel diverticula (SBD) are rare and often discovered incidentally. Although usually asymptomatic, complications such as diverticulitis, obstruction, abscess, and perforation can occur. Given the rarity of SBD, no established treatment guideline exists. We present a case of a healthy patient with urinary symptoms who was found to have SBD.
Case Description/Methods: A healthy 54-year-old female presented to her PCP with sharp suprapubic abdominal pain with dysuria and gross hematuria. Physical examination demonstrated suprapubic tenderness upon palpation. A urinalysis and urine culture were unremarkable. However, there was concern for urinary tract infection (UTI) and patient was started on Cephalexin 500mg BID for 14 days. Two days later, patient presented to the ER with worsening symptoms. She now had associated nausea and vomiting with fever. She exhibited guarding and rebound tenderness on physical examination. A CMP was unremarkable with normal LFTs. She had a WBC count of 11.8 K/mm^3. Given worsening suprapubic pain, a CT abdomen and pelvis with contrast was obtained which showed severe acute small bowel diverticulitis involving a 6 cm long distal ileal loop adjacent to the bladder. Surgery services were consulted for intervention and recommended inpatient observation and initiation of IV antibiotics. The patient refused and wished to go home. She was discharged with Ciprofloxacin 500mg BID and Metronidazole 500mg TID for 14 days. The patient was scheduled to follow up with GI clinic. A repeat CT abdomen and pelvis with contrast showed near complete resolution of the inflammatory features of small bowel diverticulitis. Patient reported retuning to her baseline health. (Figure)
Discussion: Most patients with SBD are asymptomatic. However, symptoms may reflect local inflammation as seen in our patient who presented with suprapubic pain, dysuria, and gross hematuria. No established treatment guidelines exist for SBD and thus colonic diverticulitis management is used as a guide. Asymptomatic patients do not require treatment. For patients with uncomplicated SBD, management includes diet adjustment and antibiotics. For complicated SBD, as evidenced by perforation on imaging or hemodynamic instability, surgical management is often required.



[3369] **Figure 1.** (top): CT abd and pelvis w/contrast showing large small bowel diverticulitis measuring 2.1 cm. (bottom): CT abd and pelvis w/contrast showing marked improvement of the small bowel diverticulitis with near complete resolution of inflammatory changes and wall thickening.

S3370

Chronic Intestinal Obstruction in Surgically Treated Hirschsprung's Disease: A Rare Presentation

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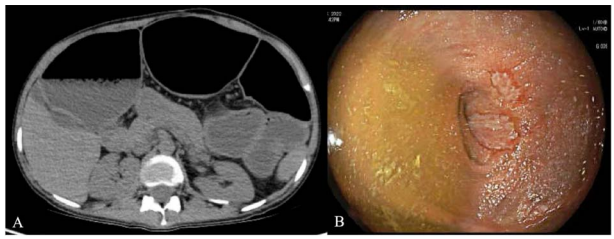
Introduction: Hirschsprung's Disease (HD) is exceedingly rare and represents a failure of ganglion cell migration to the gut. Longitudinal studies examining long-term outcomes of surgically treated HD are extremely limited. We discuss a patient who underwent surgical treatment for HD as an infant and presented in adulthood with severe abdominal pain and weight loss.
Case Description/Methods: A 34-year-old female presented with severe abdominal pain and 15-pound weight loss over 1 month. She had a history of HD that involved the entire colon diagnosed at 6 months which was treated with the Duhamel procedure at age 1. Her postoperative course was complicated by recurrent small bowel obstruction (SBO) requiring multiple surgeries for adhesiolysis. She continued to pass gas; bowel movements were at baseline. Physical exam revealed mild distention and tenderness in the periumbilical region. CT showed marked small bowel dilation proximal to the anastomosis, multiple areas of focal narrowing, and significant stool burden (Figure A). Endoscopy revealed nonobstructive ileal stenosis proximal to the anastomosis and ileal ulcerations with biopsies showing acute and chronic inflammation with cryptitis (Figure B). Empiric treatment for small intestinal bacterial overgrowth and Hirschsprung-associated enterocolitis were initiated without improvement. She was also trialed on mesalamine with no benefit. Manometry revealed impaired recto-anal inhibitory reflex possibly due to residual HD and Type 1 dyssynergic defecation. She was started on prucalopride with some improvement of her abdominal pain. Providing non-invasive venting options remains an ongoing challenge.
Discussion: The curative treatment for HD is surgical resection and the limited studies on long-term outcomes of these patients have demonstrated that constipation or diarrhea are common, and obstructive symptoms or dyssynergia are rare. Potential reasons for the complications presented in this case include mechanical obstruction from strictures and possible residual aganglionosis from insufficient resection. Additionally, prolonged bowel dilation may lead to impaired motility, evacuation difficulties, chronic dilation, and ischemic changes. There is a need for longitudinal studies at dedicated colorectal centers to provide effective transition of care from childhood to adulthood as well as a formalized approach to the management of postoperative complications of HD in adulthood, especially as these issues closely relate to functional status and quality of life.



[3370] **Figure 1.** A: CT demonstrating areas of narrowing and marked dilation proximal to the anastomosis B: Endoscopic view of the ileum demonstrating ulceration and stenosis at the anastomosis site.

S3371

A Rare Case of Iron Deficiency Anemia Due to Lung Cancer Metastasis to Small Bowel

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Introduction: Lung cancer remains the leading cause of cancer related death in the United States, likely due to its high malignant potential. While the brain, bone, liver, and adrenal glands are common sites of metastasis for lung cancer, metastasis to the GI tract remains rare. Here we present a rare case of primary lung cancer with symptomatic metastasis to the small bowel.
Case Description/Methods: A 48 year old M was referred to the Gastroenterology clinic for iron deficiency anemia and abdominal pain. A year prior, he was diagnosed with CK7 positive poorly differentiated Adenocarcinoma of the lung after a biopsy from a right upper lobe pleural mass. He underwent local radiation and chemotherapy with Carboplatin and Pemetrexed, followed by immunotherapy with Durvalumab. Follow up imaging in the interim showed excellent response to therapy. A month prior to presenting to the GI clinic, he developed recurrent mid abdominal pain associated with food intake. Labs revealed iron deficiency anemia with Hemoglobin 5.2 g/dL, MCV 64.5 um ^{3}, Iron 9 mcg/dL. He denied overt GI bleeding. EGD and colonoscopy were negative for malignancy, however video capsule endoscopy showed areas of mucosal ulceration throughout the middle of the exam. Small bowel enteroscopy redemonstrated mucosal changes in the jejunum. Pathology from jejunal biopsies identified CK7 positive carcinoma, consistent with lung metastases to the small bowel. Follow up PET scan disclosed diffusely metastatic disease. Unfortunately, no treatment options remained, and patient died shortly thereafter.
Discussion: Symptomatic disease spread to the small bowel in lung cancer has been estimated to be 1.8%,[1] however autopsy studies have indicated rates of up to 11.9%[2]. This discrepancy is likely due to minimal symptom burden associated with small bowel metastases. In addition, small bowel metastasis is often an extremely late presentation of disease, as nearly all patients with small bowel metastasis are found to have additional sites of metastasis[3], thus lending to an extremely poor prognosis. This case was particularly interesting in that this patient's only indications for metastatic disease were iron deficiency anemia abdominal pain due to small bowel metastasis. A high index of suspicion is needed for patients with a history of lung cancer who develop abdominal pain or iron deficiency anemia for the detection of metastasis.

S3372

Biopsy-Confirmed Celiac Disease After COVID-19 Infection

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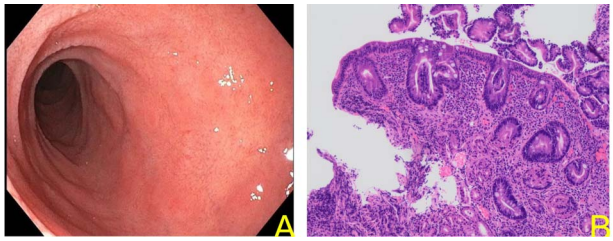
Introduction: Post-COVID gastrointestinal (GI) symptoms are increasingly common in clinical practice and can sometimes overlap with other common, treatable GI disorders, such as Celiac Disease (CeD). We present the case of post-COVID GI symptoms subsequently diagnosed with CeD.
Case Description/Methods: A 28-year-old man with no prior medical history presented with nausea, vomiting, poor appetite, and weight loss after recovery from PCR-confirmed COVID-19 infection. Family history is remarkable for a sibling with type 1 diabetes mellitus. He endorsed an unintentional 40-pound weight loss with fatigue over the past 4 months, but he denied any constipation or loose stools. Family history is remarkable for a sibling with type 1 diabetes mellitus. He sought emergent care twice prior to being referred to gastroenterology clinic at which time abdominal imaging was unremarkable. Symptoms did not improve with a trial of proton pump inhibitors. Physical exam at time of consultation was remarkable for obesity. Laboratory work revealed elevated tissue transglutaminase IgA 57 U/mL (normal $<$ 19 U/mL). He was also found to be deficient in vitamin D, vitamin B12, and folate. Upper endoscopy revealed duodenal scalloping and atrophy (Figure A) where biopsies confirmed CeD, Marsh 3B (Figure B). He was started on a gluten free diet and his symptoms resolved. Tissue transglutaminase IgA normalized (9 U/mL) within 6 months of making dietary changes.
Discussion: Post-COVID symptoms are overall varied and non-specific with potential to overlap with other GI conditions. There has been a reported increased prevalence of CeD in all age groups during the SARS-CoV-2 pandemic[1]. SARS-CoV2 enters mucosal membranes via angiotensin-converting enzyme 2, which is present in the small bowel mucosa[2]. Our case highlights that weight loss, and other "red flag" symptoms should prompt further evaluation despite being a common post-COVID symptom. We contemplate what role, SARS-CoV-2 could have had in disrupting small bowel mucosa integrity, potentially leading to CeD in genetically susceptible individuals.1. Trovato CM et al. COVID-19 and celiac disease: A pathogenetic hypothesis for a celiac outbreak. International Journal of Clinical Practice. 2021;75(9). 2. Hoffman M et al. SARS-CoV-2 cell entry depends on ACE2 and TMPRSS2 and is blocked by a clinically proven protease inhibitor. Cell. 2020;181(2):271-280.



[3372] **Figure 1.** A: Duodenal scalloping and atrophy on upper endoscopy B: Subtotal villous atrophy, Marsh 3B.

S3373

Atypical GBS as the Presenting Symptom of Celiac Disease: Recognizing Extra-intestinal Presentations of Celiac Disease

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Introduction: Celiac disease (CD) is an immune-mediated enteropathy in response to gluten that occurs in 1% of the population. Classic presenting symptoms include bloating, weight loss, and diarrhea. But, extraintestinal symptoms such as dermatitis herpetiformis, iron deficiency anemia, asymptomatic liver abnormalities, and more rarely, neurologic symptoms can be seen. We present a case of a patient who presented with worsening polyneuropathy and hepatitis leading to a final diagnosis of CD.
Case Description/Methods: A 44-year-old male with history of recent left-chest shingles complicated by paralysis of right face and left leg weakness was initially admitted for suspected atypical Guillain Barre Syndrome (GBS) and treated with prednisone and a 5-day course of intravenous immunoglobulin (IVIG). The patient was readmitted a few weeks later with worsening neurologic status. A thorough infectious workup was unremarkable. The patient was also noted to have abnormal liver enzymes on prior admission that initially improved, but were noted to be worse on readmission up to ALP 1103, AST 634 and ALT

689. Given no prior history of liver disease, patient underwent a thorough workup including negative viral hepatitis, normal liver US, abdominopelvic CT showed scattered hypoattenuating lesions in the liver, one measuring up to 2.2 cm. He underwent a liver biopsy which demonstrated acute hepatitis with findings concerning for autoimmune versus drug-induced hepatitis. Autoimmune hepatitis panel was negative, however, he was found to have a tissue transglutaminase IgA >250 U/mL. Finally, he underwent upper endoscopy with biopsies that demonstrated findings consistent with celiac disease. Liver enzymes improved with gluten avoidance, but neurologic impairments persisted.
Discussion: Atypical extra-intestinal symptoms at presentation of CD can result in diagnostic delay and permanent neurological disability if not treated. CD has been found in as many as 9% of patients with elevated liver enzymes. It is imperative not only to recognize neurologic presentations of CD but also the spectrum of possible liver impairments. Adherence to a strict gluten-free diet has been shown to improve symptoms of both neuropathy and liver enzyme abnormalities, but must be instituted quickly.

S3374

Asymptomatic Multiple Lymphomatous Polyposis: An Atypical Presentation of Mantle Cell Lymphoma

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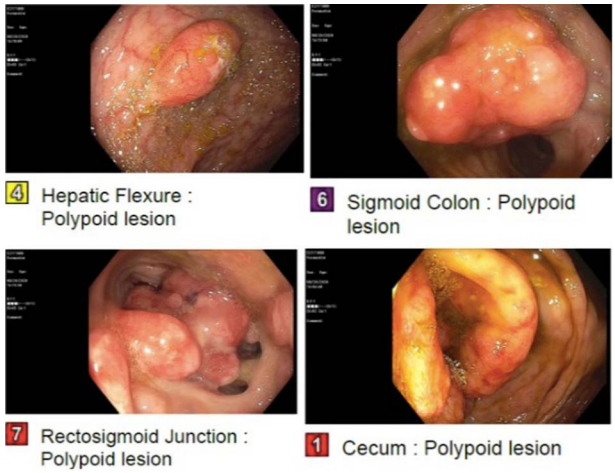
Introduction: Multiple lymphomatous polyposis (MLP) is a rare type of mantle cell lymphoma that affects the GI tract. Patients with MLP generally present with generalized GI symptoms including abdominal pain and imaging, including endoscopy, can be unrevealing. We present an unusual case of a patient without GI symptoms who was found to have MLP affecting areas of the duodenum, colon, and rectum.
Case Description/Methods: We present a 71-year-old female with a past medical history of IBS, previous diverticulitis s/p sigmoidectomy, history of transverse myelitis and DM who presented to the hospital with progressively worsening shortness of breath for the past 6 weeks. She denied any B-symptoms (fevers, night sweats, weight loss). Subsequent labs did not identify any abnormalities. CTA chest revealed a large right pleural effusion and significant lymphadenopathy in the axilla and supraclavicular regions bilaterally. A thoracentesis was performed which revealed cytology consistent with MCL. A CT abdomen/pelvis demonstrated a mass in the pylorus and duodenal bulb and a questionable mass in the sigmoid colon. A bidirectional endoscopy was performed which showed polypoid lesions in the duodenum, hepatic flexure,sigmoid colon, recto-sigmoid colon. Biopsy of the polyps revealed mantle cell lymphoma . PET scan showed lymphadenopathy in the axillary, mediastinal, hilar, retroperitoneal regions, splenomegaly, and FDG avidity in the gastrocolic ligament. The patient was initially treated with r-AraC chemotherapy regimen and was later transitioned to Bendamustine/Rituximab regimen. She also had a bone marrow biopsy which showed normocellular marrow and was scheduled to receive bone marrow transplant. (Figure)
Discussion: MLP is an extremely rare phenomenon and is characteristically seen in patients with MCL. It is characterized by numerous GI polypoid lesions involving different areas in the gastrointestinal tract. Common clinical symptoms reported for patients with MPL include weight loss, nausea, vomiting, diarrhea and intestinal malabsorption. Some cases have even reported chylous ascites and intestinal obstruction secondary to intussusception. This case highlights a unique clinical presentation of shortness of breath and a rare endoscopic finding of MLP to diagnose MCL. As MPL has a poor prognosis, early detection and treatment is imperative. In summary, we report an unusual patient with high-risk MCL who had asymptomatic MLP identified only at staging bidirectional endoscopy.



[3374] **Figure 1.** A bidirectional endoscopy was performed which showed polypoid lesions in the duodenum, hepatic flexure,sigmoid colon, recto-sigmoid colon.

S3375

Capsule Endoscopy as a Diagnostic Tool for Metastatic Melanoma of the Small Bowel

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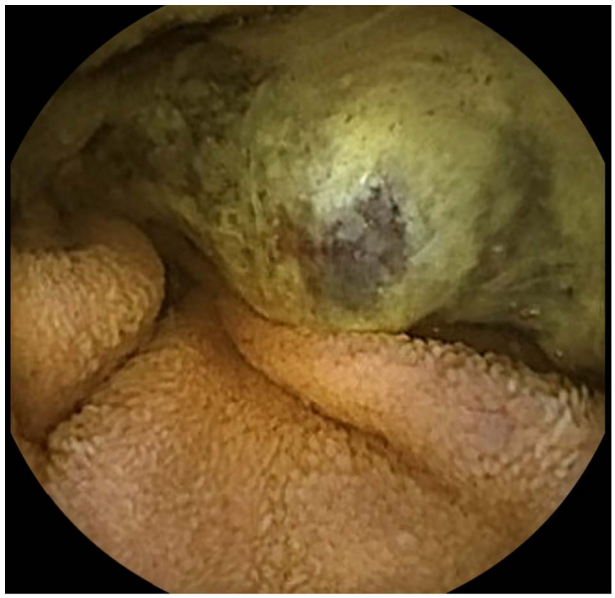
Introduction: The detection of gastrointestinal malignancies in the small bowel remains to be a challenge for both clinicians and radiologists due to nonspecific clinical findings and inconclusive diagnostic testing. Endoluminal studies are beneficial but have their limitations on particular areas of bowel. Capsule endoscopy is a noninvasive imaging study with a high yield in diagnosing these cases.
Case Description/Methods: A 53 year-old male with a history of melanoma of the back was treated with surgical excision and immunotherapy presented with a 2 month history of progressive exertional dyspnea and fatigue. Denied any complaints of overt bleeding, abdominal pain, or weight loss. No family history of gastrointestinal malignancies. Noted to have iron deficiency anemia with a hemoglobin of 6.7 g/dL. He was admitted and received blood transfusion with improvement of his symptoms and wanted to pursue outpatient endoscopies. However continued to have ongoing symptoms following discharge. He underwent an upper endoscopy which was unremarkable and a colonoscopy that showed a small transverse colon polyp and small internal hemorrhoids which did not explain his anemia. Therefore he underwent a small bowel capsule endoscopy that revealed a partially obstructing ulcerated mass with active oozing in the proximal small bowel, seen in fig. 1. He underwent an urgent upper endoscopy with push enteroscopy that demonstrated a large fungating multilobulated jejunal mass that was biopsied. Pathology confirmed the mass to be a MART-1, S100, and HMB45 positive melanoma, suspected to be a metastatic lesion given his history. A CT scan of his abdomen and pelvis was done for staging purposes that showed new metastatic tumoral implants in the mesentery adjacent to the small bowel loops. (Figure)
Discussion: Malignant melanomas in the gastrointestinal tract are rare and could be either a true primary or a metastatic lesion. They are difficult to diagnose as only 1-5% are detected in early stages of imaging. Routine endoscopic modalities have minimal benefit in diagnosing small bowel lesions. These malignancies are aggressive and are diagnosed in late stages with conventional studies and have a poor prognosis. Therefore capsule endoscopy is a simple and noninvasive test that can help provide crucial information in establishing diagnosis.



[3375] **Figure 1.** Video Capsule Endoscopy demonstrating an ulcerated mass visualized in the jejunum with partial obstruction.

S3376

Brunner Gland Hamartomas: Uncommon Presentations and Endoscopic Management

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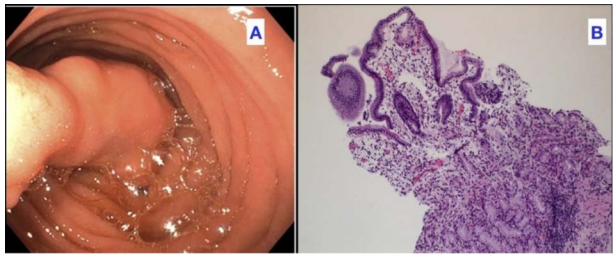
Introduction: Brunner gland hamartoma (BGH) is rare benign tumor of the duodenum that requires a high clinical suspicion to diagnose. Although it may be discovered incidentally, it can be the etiology of the underlying gastrointestinal bleeding (GIB) or intestinal obstruction. Here, we present a case of anemia and vomiting due to BGH followed by a literature review of the current practice of the diagnosis and management of large BGH.
Case Description/Methods: A 66-year-old man was referred to the gastroenterology clinic for iron deficiency anemia (IDA), vomiting, and abdominal discomfort. He reported no early satiety, unintentional weight loss, or change in bowel habits. Physical exam was significant for pallor and abdominal exam was soft and non-tender. Laboratory work up was significant for normocytic anemia with hemoglobin of 9.6 g/dL and iron deficiency with iron level of 7mcg/dL, ferritin of 3.5 ng/mL, transferrin saturation of 7%, and total iron binding capacity 411 mcg/dL. Intrinsic factor antibody, folate, TSH, and comprehensive metabolic profile were normal. Patient underwent an EGD that revealed a 2cm hiatal hernia, grade B esophagitis, and a large 50mm partially obstructing pedunculated duodenal polyp extending from the duodenal sweep to the 2nd portion of the duodenum (Figure A). Multiple attempts at endoscopic hot snare removal with endoloop were made without success. Biopsies of the polyp showed benign Brunner's gland hyperplasia (Figure B). Patient was then referred to an advanced endoscopist for resection of the large pedunculated duodenal polyp. The patient's IDA and abdominal symptoms have significantly improved following resection of the duodenal polyp with no further vomiting or intermittent abdominal discomfort. The IDA was deemed to be second to the duodenal BGH.
Discussion: Brunner gland hamartomas are extremely rare benign hyperplastic lesions of the GI tract. Small BGHs have an average size of 1.6cm and are commonly asymptomatic. Large hamartomas (>2cm) are likely to present with GI bleeding, anemia, or obstructive symptoms. Management of BGH is either endoscopic or surgical and is usually based on the tumor size (typically 0.5-5 cm) and malignancy potential. On the other hand, surgery is reserved for lesions that are large, endoscopically unresectable, or have a suspicion for malignancy. In summary, we suggest that clinicians should be cognizant of considering BGH in their differential, especially in case of obstruction, occult GI bleeding, and IDA.



[3376] **Figure 1.** A. Esophagogastroduodenoscopy demonstrating the pedunculated duodenal polyp. B. Histological evaluation of the pedunculated duodenal polyp. The biopsy demonstrates the polypoid duodenal mucosa with the Brunner's gland hyperplasia, chronic inflammation, and focal villous blunting. No evidence of increased intraepithelial lymphocytes, adenomatous changes, or dysplasia.

S3377

Atypical Disseminated Mycobacterium Avium Complex: A Rare Case of Chronic Severe Diarrhea in a Patient with HIV

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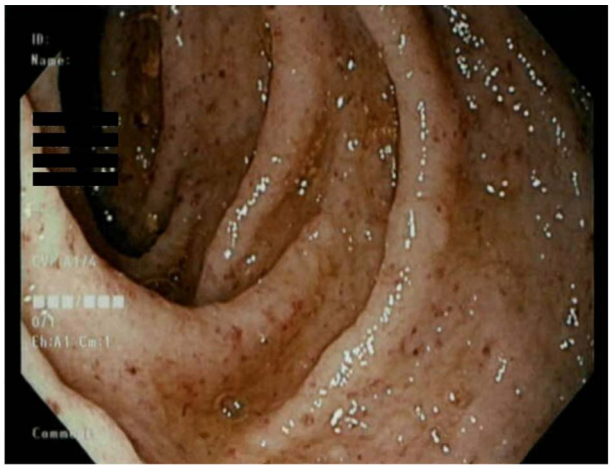
Introduction: The global use of highly active antiretroviral therapy (HAART) has led to a dramatic decline in the incidence of disseminated Mycobacterium Avium Complex (MAC) to ≤ 2 cases per 1000 person-years. We present a unique case of chronic diarrhea secondary to disseminated MAC which was initially thought to be cytomegalovirus(CMV) viremia.
Case Description/Methods: A 41-year-old man with Human Immunodeficiency Virus(HIV)presents with progressively worsening loose, non-greasy, non-bloody, non-mucoid diarrhea for 6 weeks. Associated with recent 10-pound unintentional weight loss, and dyspnea. Outside records report splenectomy for a splenic rupture with tissue culture positive for acid-fast bacilli(AFB) non-Mycobacterium tuberculosis, non-MAC by PCR 3 months prior, started on therapy but reports nonadherence. Cachectic, hypotensive, tachycardic, tachypneic on exam. Labs showed hemoglobin 6.9 gm/dl, MCV 79.5 FL, platelets of 138,000/microL, pre-renal AKI with creatinine 1.36 mg/dl, lactic acid of 3.0mMol/L, and albumin of 2.1 gm/dl . WBC, bilirubin, ALT, AST, and ALP were normal. Extensive lab workup showed low CD4 count of 7 cells/

uL, and high CMV PCR at 4,068 IU/mL. Qualitative fecal fat positive. Negative blood cultures, fungal culture, mycobacterial blood, and sputum cultures. Negative stool testing for Clostridium difficile, fecal leukocyte antigen, cryptosporidium, Giardia antigen, Salmonella, Shigella, Campylobacter species, ova and parasites. CT abdomen/pelvis showed a loculated fluid collection in the splenic bed that was drained with negative cultures. On EGD and colonoscopy, the duodenum and terminal ileum (TI) had petechial lesions which on pathologic examination revealed diffuse AFB and villous blunting in TI. Normal colonic biopsies. Biopsies were negative for CMV, celiac disease, and Whipple's disease. Diagnosed with disseminated atypical MAC(most likely M. kansasii) in the setting of HIV-AIDS. CMV viremia was thought to be an incidental finding without GI manifestations. Treatment for disseminated MAC with ethambutol, isoniazid, rifampin, azithromycin, pyridoxine, and for HIV with Dolutegravir & Truvada started. However, he deteriorated despite treatment and died from multi-organ failure 16 days later. (Figure)

Discussion: Diarrhea, a common gastrointestinal symptom in AIDS, has many etiologies. DMAC is associated with higher morbidity and mortality. So physicians must be familiar with the causes and utilize endoscopic interventions as needed for effective diagnosis and treatment in AIDS patients.



[3377] **Figure 1.** Petechial lesions in the terminal ileum noted on endoscopy.

S3378

Bleeding GIST Treated Successfully With Hemospray in a Patient With Neurofibromatosis Type 1

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Introduction: Gastrointestinal stromal tumors (GISTs) are rare subepithelial mesenchymal neoplasms of the gastrointestinal tract that are typically KIT (CD117) positive. GISTs can present with a wide range of symptoms including gastrointestinal hemorrhage. Small bowel bleeding from a GIST can be difficult to treat endoscopically and there is a paucity of data on the use of TC-325 (hemospray) for these tumors.
Case Description/Methods: A 47-year-old man with a past medical history significant for neurofibromatosis type 1 and multifocal small bowel GISTs presented to our center with symptomatic anemia. The patient described previous episodes of melena. The patient's initial hemoglobin was 2.2 g/dl. He was initially treated with a transfusion of 4 units packed red blood cells. CT abdomen and pelvis with IV contrast was obtained which revealed a centrally located 6 x 5cm small bowel mass with heterogeneous enhancement and intraluminal signal concerning for active gastrointestinal hemorrhage. A push enteroscopy was performed using a pediatric colonoscope via the mouth and advanced deeply into the small bowel. A large ulcerated submucosal mass with active bleeding was found at the junction of the fourth portion of the duodenum and proximal jejunum. Submucosal injection of 1:10,000 diluted epinephrine around the ulcer base failed to achieve hemostasis. Next, the hemospray catheter was advanced to several centimeters proximal to the bleeding ulcer. Hemospray was deployed and immediate hemostasis was achieved after coating the area.
Discussion: The majority of GISTs are sporadic and found in the stomach. Patients with neurofibromatosis type 1 however, have a higher incidence of GISTs, are often multifocal, and most commonly are found in the small intestine as demonstrated here. Endoscopic treatment of small bowel tumor bleeding is challenging because of the location and nature of the bleeding. Conventional treatments such a hemoclips or bipolar electrocautery usually fail to stop tumor bleeding. TC-325 or hemospray is a novel inert powder which accelerates the coagulation cascade leading to rapid hemostasis of actively bleeding lesions. Hemospray should be considered first line treatment for malignant bleeding because of its ease of use, wide field of application and demonstrated success.

S3379

Capecitabine-Induced Rapid Onset Terminal Ileitis in Patient With Stage III Sigmoid Adenocarcinoma

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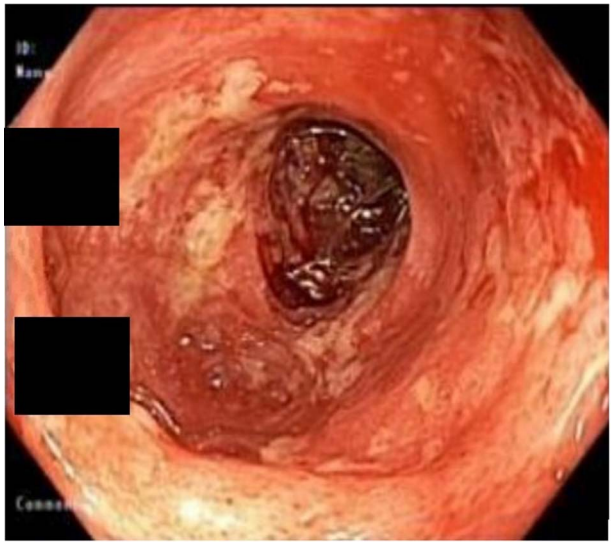
Introduction: Capecitabine is a chemotherapy drug used in the treatment of colorectal cancer, particularly when a single agent is desired. It is known to cause diarrhea, usually mild, in the second or third week of treatment. We present of case of rapid onset diarrhea secondary to terminal ileitis beginning two days after starting capecitabine therapy.
Case Description/Methods: A 42-year-old female with a history of T3N2 colon adenocarcinoma s/p left hemicolectomy two months prior presented with one day history of abdominal pain and watery blood-tinged diarrhea. Physical examination was unremarkable and patient was hemodynamically stable. Labs, including stool studies, were within normal limits. She had completed her first cycle of capecitabine three days prior to presentation. CT of abdomen showed diffuse small bowel wall thickening most prominent in ileum with reactive edema. Colonoscopy revealed erythematous and friable mucosa with ulceration and exudate leading to the diagnosis of terminal ileitis (Figure). Biopsy showed severe active ileitis with focal surface erosion and increased number of eosinophils in the ilium. Patients was discharged after seven days of IV antibiotics and opted for different chemotherapy agent for further treatment.
Discussion: Capecitabine-induced terminal ileitis is a rare but serious adverse effect that clinicians must watch for in patients being treated with this medication. It has been seen within one week of initiation of therapy in patients with dihydropyridine dehydrogenase deficiency (DPD). This is the only case we have found to demonstrate terminal ileitis development within one week of starting capecitabine in a patient without DPD deficiency. We feel that this is an important case to discuss because we do not want clinicians to attribute the signs and symptoms of terminal ileitis to just a benign medication associated diarrhea, particularly when occurring so quickly after treatment initiation. Clinicians need high index of suspicion, and these patients need urgent workup to prevent poor outcomes.



[3379] **Figure 1.** Colonoscopy revealed erythematous and friable mucosa with ulceration and exudate in the terminal ilium.

S3380

Atherosclerotic Small Bowel Ischemia Causing Intestinal Obstruction and Volvulus

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Introduction: Obstructions are the most common events requiring surgical intervention in the small intestine. Several factors can cause an obstruction, with adhesions from previous surgeries being most common. It is rare for the obstruction to be caused by volvulus of the small bowel, and increasingly more rare for that volvulus to be caused by ischemia due to atherosclerosis. Presented herein is a unique case of small bowel obstruction due to volvulus of the small bowel that resulted from severe bowel ischemia.
Case Description/Methods: The case discusses the medical and surgical management of a 70-year-old man who presented to the emergency department with abdominal pain. After a thorough history, physical exam, and diagnostic testing, the patient was diagnosed with small bowel obstruction due to volvulus, secondary to atherosclerotic ischemia of the small intestine, and rushed to surgery. After the surgical intervention and hospital stay, the patient made a complete recovery and was asymptomatic at follow-up appointments.
Discussion: Small bowel obstructions occur mostly due to adhesions from prior surgeries. It is exceedingly rare that they may occur due to volvulus. This case is unique in that the inciting event was significant small bowel ischemia, likely from uncontrolled hyperlipidemia and atherosclerosis as evidenced by the CT findings of a severely atherosclerotic abdominal aorta. The ischemia then made way for the necrotic segment of the small bowel to twist upon its mesentery and cause a closed loop obstruction and further compromise of the blood supply to the small intestine. The presentation of a volvulus in the small intestine is not as easily recognizable on radiographs as a sigmoid and cecal volvulus. The classic "birds beak" phenomenon characteristic of any volvulus on contrast enhanced plain film was not seen in this case. Imaging was consistent with a small bowel obstruction, but the etiology was unclear until an exploratory laparotomy was performed. It is likely that this patient had a chronic underlying process of mesenteric ischemia before presenting with this volvulus that was never diagnosed. Ischemia must be considered as a differential or possibly as an underlying factor for future management when working up a patient for bowel obstruction. Swift intervention in this scenario likely prevented outcomes such as perforation and severe hemodynamic instability. This patient would benefit from cholesterol lowering medications and possibly endovascular repair to prevent future recurrence.

S3381

Bruner's Gland Hyperplasia Mimicking Duodenal Carcinoma

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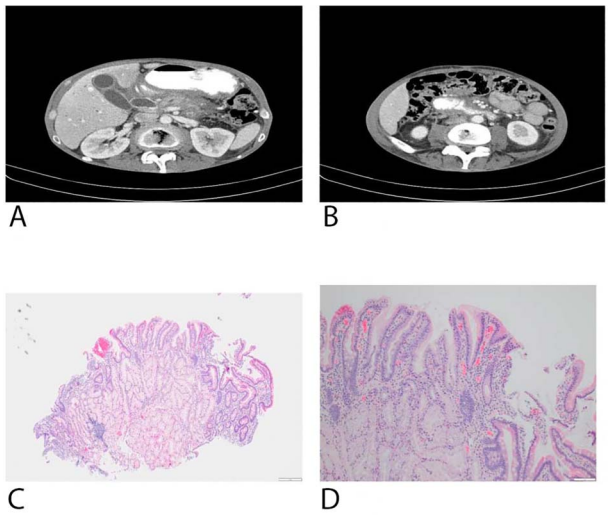
Introduction: Brunner's gland hyperplasia are rare benign tumors, arise from Brunner's glands in the duodenum. Brunner's gland hyperplasia is asymptomatic in many patients but it can present with GI bleed or small bowel obstruction. We are presenting an interesting case of a patient with Brunner's Gland hyperplasia resembling a duodenal carcinoma.
Case Description/Methods: A 47 years old male presented with epigastric pain. He had a history of alcohol use and abuse, chronic pancreatitis, pancreatic pseudocyst and hypertension. On physical exam he had epigastric tenderness, he was admitted to the hospital multiple times for acute and chronic pancreatitis. Labs showed an alpha fetoprotein at 1.3, comprehensive metabolic panel was only remarkable for sodium of 132.WBC was 8.8, Hemoglobin of 13. A CT scan was ordered to assess his pancreatic pseudocyst demonstrated large duodenal mass involving the first and the second portions of the duodenum concerning for primary duodenal neoplasm such as duodenal carcinoma (Figure A, B) the mass measures 4.9x3.2 cm in size, appears to impinge on the head and neck of pancreas, although it alternatively, it is possible that it represents a pancreatic mass.so we proceeded with upper GI endoscopy which showed erythema and congestion of the duodenal bulb, which was biopsied. Pathology report of the duodenum showed reactive epithelial changes and Brunner's gland hyperplasia (Figure C, D).
Discussion: Risk factors of Brunner's gland hyperplasia include high gastric acid secretion, H. pylori infection, chronic pancreatitis, inflammatory stimulation, mucosal injury, etc. The lesions are mostly located at the proximal duodenum, and their occurrence gradually decreases with increasing the distance from the pyloric ring: 57% in the duodenal bulb.Most patients are asymptomatic, if symptomatic, symptoms are usually nonspecific, like dyspepsia, abdominal distension, abdominal pain, nausea, vomiting, gastrointestinal bleeding and obstruction.It can be diagnosed by CT, on contrast-enhanced CT usually shows specific imaging features, including a mass in the bulb and descending parts of the duodenum (Figure A, B). It is important to think about Brunner gland hyperplasia in a differential diagnosis when evaluating a duodenal mass. The differential should also include mucosal polypoid masses such as adenomatous polyps and adenocarcinomas, lymphoma, carcinoid tumors, mesenchymal neoplasms such as gastrointestinal stromal tumors, leiomyomas, leiomyosarcomas, neurogenic tumors; and metastatic disease.



[3381] **Figure 1.** A, B are CT Abdomen with contrast showing Brunner's gland hyperplasia C, D nodular peptic dudodenitis is characterized by hyperplastic Brunner glands that fill the lamina propria which creates the nodular mucosal appearance on endoscopy. The reactive epithelial changes and foveolar hyperplasia at the surface epithelium are characteristic of chronic duodenopathy.

S3382

Budesonide in Treating a Patient With Microscopic Enteritis

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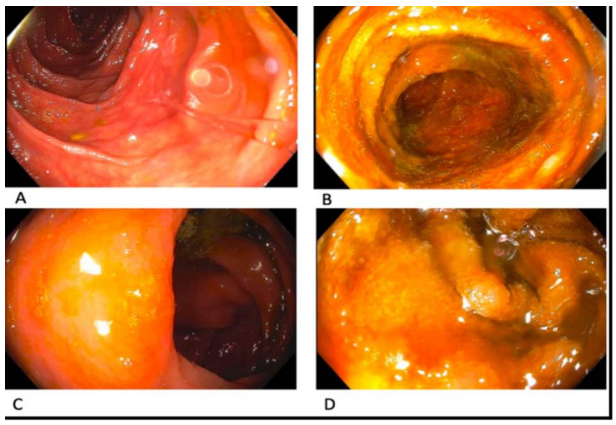
Introduction: Microscopic enteritis is an inflammatory condition in the small bowel that causes chronic diarrhea and bloating. It is associated with gluten intolerance, allergies, drug therapy, inflammatory bowel disease, and autoimmune conditions. Despite the prevalence of risk factors for microscopic enteritis, the understanding of the disease remains limited.
Case Description/Methods: A 33-year-old man with past medical history of major depressive disorder presented with chronic diarrhea. Seven months ago, he started to have 2-5 loose bowel movements daily, with Bristol stool consistency ranging from 4-7. The diarrhea did not improve with fasting and was not nocturnal, but heavy meals worsen his symptoms. The patient denied having abdominal pain, bright red blood per rectum, incontinence, weight loss, fever, or chills. The patient attempted to take pancrelipase, loperamide, probiotics, increased fiber intake, with no improvement in his symptoms. Workup was negative for tissue transglutaminase and stool culture for gastrointestinal pathogens, but significant for fecal calprotectin >200 mcg/g. Colonoscopy showed non-bleeding internal hemorrhoids, normal entire colon and terminal ileum (Figure). Pathology of the biopsy taken from the terminal ileum showed small intestinal mucosa with mild intraepithelial lymphocytes suggesting a diagnosis of inflammatory bowel disease vs microscopic enteritis. The patient was started on budesonide 9 mg by mouth daily for 2 months, which led to diarrhea resolving and remaining asymptomatic off the medication.
Discussion: Microscopic enteritis is an inflammatory condition that affects the small bowel. It can be caused by infection, gluten intolerance, medications, or autoimmune diseases. Workup for microscopic enteritis involves testing for endomysial antibodies, anti-tissue transglutaminase, HLA typing and immunoglobulin titers. To avoid missing this diagnosis in a patient with chronic diarrhea, in addition to the common practice of random colonic biopsies to rule out microscopic colitis, intubation of the terminal ileum along with biopsies should be performed. Histologically, the epithelium in microscopic enteritis is infiltrated with lymphocytes, and lamina propria has increased eosinophils and plasma cells. Given the known response of microscopic colitis to budesonide, a trial of budesonide was started and was sufficient to resolve the patient's symptoms and lead to complete clinical remission.



[3382] **Figure 1.** A. descending colon; B. ascending colon; C. ileo-cecal valve; D. terminal ileum.

S3383

Clicking Time Bomb: Unusual Management Approach of Gardner's Syndrome

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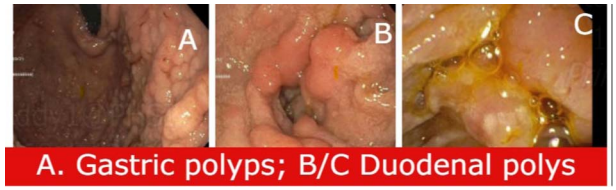
Introduction: Gardners syndrome is a variant phenotype of Familial adenomatous polyposis (FAP) due to mutation in chromosome 5q APC tumor suppressor gene with inevitable colon cancer by the age of 35 in up to 95 %. Other Intestinal manifestations include gastric and duodenal polyps which can progress to malignancy. A prophylactic proctocolectomy is practiced against colon cancer in FAP patients, leaving them with a reduced length of intestines. Patients experience significant morbidity and the clinical picture becomes further complicated if further polyps develop in the stomach or small intestine. Here we report a case of FAP with previous proctocolectomy presenting with gastric and duodenal adenomas.

Case Description/Methods: A 36-year-old female with a history of Gardners syndrome was diagnosed at the age of sixteen and underwent proctocolectomy soon thereafter. She undergoes bi-annual colonoscopies for surveillance. She also has a history of bilateral adrenal adenomas, desmoid tumor in the proximal lower extremities bilaterally, and Thyroid cancer – for which she underwent thyroidectomy and radiation. She presented to the emergency department complaining of generalized weakness associated with headache and exertional dyspnea. Conjunctival pallor was noticed. The patients' symptomatic anemia was improved after two units of PRBC transfusion. Endoscopy a week later showed innumerable medium sessile polyps located diffusely in the proximal stomach with antral sparing as well as large sessile polyps in the duodenal bulb and first portion of the duodenum. (Figure)
Discussion: The incidence of gastric and duodenal polyps in a patient with FAP is 90%. The risk of progression to periampullary carcinoma is 3-12 % in the patients. However, they can also present with malabsorption and iron deficiency anemia. Duodenal polyps can be monitored and classified as proposed by Spigelman which is based on polyps' size and number, histology, and grade of dysplasia. It can be divided into 4 stages based on scoring from 0-12. Patients with duodenal polyps can be managed by endoscopic surveillance every 1-5 years depending on the Spigelman staging. Celecoxib is suggested for stage 3. Endoscopy with EUS and duodenectomy (pancreas sparing or pylorus sparing) is recommended for stage 4. Knowledge about Spigelman classification is required in managing duodenal polyps in patients with FAP. Given the prophylactic proctocolectomy, every effort has to be made to preserve the rest of the intestine in these patients.



[3383] **Figure 1.** Gastric and Duodenal polpys.

S3384

Choledocystoduodenal Fistula Effectively Treated With a High-Dose Proton Pump Inhibitor: A Case Report

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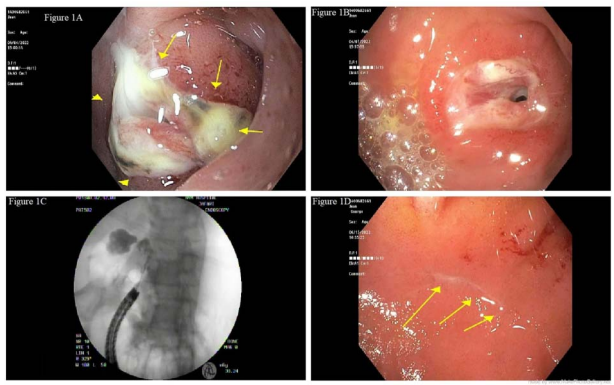
Introduction: Choledocystoduodenal fistulas are a rare complication of biliary or peptic ulcer disease. Approximately 91%-94% of spontaneous internal biliary fistulas are caused by stones in the biliary tract. The second most common cause is peptic ulcer. We present a case of a patient found to have a choledocystoduodenal fistula caused by peptic ulcer disease successfully treated with high-dose PPI.
Case Description/Methods: A 73-year-old female with a history of duodenitis and gastritis presented for chronic intermittent abdominal pain. Physical exam was positive for epigastric tenderness. Labs were normal. Imaging revealed a large stool burden, mildly distended gallbladder without gallstones and a normal biliary tree. She was started on a once-daily PPI and a bowel regimen. EGD was performed and demonstrated a large necrotic duodenal lesion with copious purulent drainage (Figure A). Gastric biopsy was negative for H. pylori. The patient was readmitted for persistent abdominal pain, weight loss and loss of appetite. On physical exam, she had epigastric tenderness. She was afebrile and lab work was normal. CT abdomen and pelvis demonstrated small bowel thickening involving the duodenal bulb and first portion of the duodenum and air within the gallbladder with mild pericholecystic inflammatory changes. An EGD performed thereafter revealed the same duodenal bulb ulcer with a likely fistula (Figure B); injection of contrast through the tract under fluoroscopy confirmed a fistula to the gallbladder (Figure C). The patient was discharged on high-dose twice daily PPI, a short course of amoxicillin and clavulanic acid. Her symptoms resolved within 8 weeks. A surveillance EGD performed to assess healing demonstrated a healed scar at the site of the prior ulcer and fistula (Figure D).
Discussion: Cholecystoenteric fistulas are a rare complication of gallstone disease and peptic ulcer disease, with an autopsy reported incidence rate of 0.1%-0.5%. Historically, surgical treatment is the most common treatment method for cholecystoenteric fistulas. Our case suggests that a high proton pump inhibitor dose for 8-12 weeks may be sufficient for the fistula to heal. By implementing medical management for this condition, it may be possible to avoid the morbidity and mortality associated with laparoscopic or open fistula repair.



[3384] **Figure 1.** A. Initial EGD showing large necrotic duodenal lesion with copious purulent drainage B. Second EGD demonstrating duodenal bulb ulcer C. Fluoroscopy with contrast injection demonstrating fistula tract communication with gallbladder D. Follow-up EGD after treatment with high dose PPI demonstrating healed choledocystoduodenal fistula with a small residual duodenal ulcer.

S3385

Collagenous Sprue and POEMS Syndrome: Association or Coincidence?

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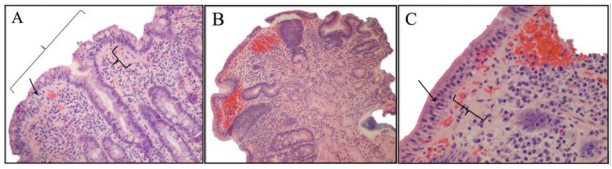
Introduction: We present a case of collagenous sprue (CS), a rare enteropathy of unknown etiology with subsequent diagnosis of POEMS syndrome, another rare entity and speculate on an association. CS typically presents similarly to celiac disease (CD) with chronic diarrhea, weight loss, and malnutrition. CS is most frequently associated with CD, and if untreated can lead to substantial mortality. The presence of a subepithelial collagen band (SCB) on histology differentiates CS from CD, as both have villous atrophy. Polyneuropathy, Organomegaly, Endocrinopathy, Monoclonal protein, and Skin changes syndrome (POEMS) presented shortly after this patient's sprue symptoms, suggesting a possible association through an immune-mediated mechanism.
Case Description/Methods: A 75-year-old male with coronary artery disease and hypertension reported intermittent diarrhea, bloating, and 20lbs weight loss over six months. He had no relevant family history. His medications included losartan and aspirin. Physical exam was notable for a thin male (BMI 23.3) with a benign abdomen. His hemoglobin was 13.8 g/dL. CD serologies (anti-tissue transglutaminase IgA 158U, gliadin IgA 201U, and endomysial antibody 1:40) were positive; CD diagnosis was confirmed by an EGD biopsy of the proximal duodenum which revealed villous atrophy and intraepithelial lymphocytosis (Figure A). He was started on a gluten-free diet but did not follow up. One year following his CD diagnosis, the patient was diagnosed with POEMS after being referred for peripheral neuropathy and an elevated IgM kappa monoclonal protein. Remission was achieved with melphalan and dexamethasone. Four years after initial CD diagnosis, he had continued weight loss (12lbs) and was referred for GI evaluation. A repeat duodenal biopsy showed a SCB of 32.2μm along with previous villous atrophy, consistent with CS (Figures B and C). On review, a SCB was present from the initial duodenal biopsy but to a lesser degree (28.6μm).

Discussion: POEMS rarely results in GI manifestations with no previous cases of small bowel involvement. Thus, it is unclear if the diagnosis of both CS and POEMS within a short period of time was a coincidence or if there is a connection given their proposed similar immune-mediated pathogenesis. Due to the proinflammatory environment, it is plausible that CS and/or CD may be triggering the production of POEMS' monoclonal protein. Research investigating CS's pathogenesis and additional cases of CS with POEMS is needed to further support this hypothesis.



[3385] **Figure 1.** A. Initial proximal duodenal biopsy under high-power view demonstrating villous atrophy (large outer bracket), intraepithelial lymphocytosis (arrow) , and 28.6um subepithelial collagen band (small inner bracket). B. Repeat proximal duodenal biopsy under low-power view demonstrating villous atrophy. C. Repeat proximal duodenal biopsy under high-power view demonstrating intraepithelial lymphocytosis (arrow) and 32.2um subepithelial collagen band (bracket).

S3386

Celiac Disease and Adrenal Insufficiency: A Case of Autoimmune Polyglandular Disease

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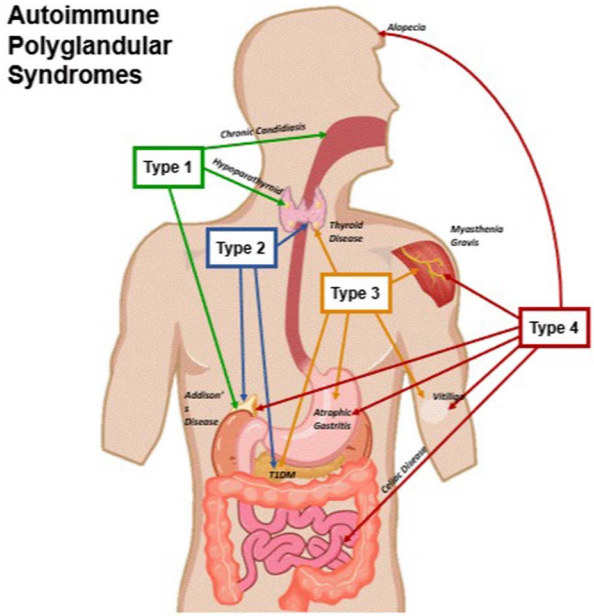
Introduction: Our cases illustrates that celiac disease is an important clue to diagnose adrenal insufficiency due to its association with Addison's disease. We present a case of a man with celiac disease and Grave's disease who presented with 6 days of vomiting and loose stools with severe hyponatremia found to have adrenal insufficiency. He received a new diagnosis of autoimmune polyglandular disease type 4, a rare disease characterized by multiple autoimmune disorders affecting both endocrine and non-endocrine organs.
Case Description/Methods: A 39 year old man with Graves status post iodine ablation on levothyroxine and celiac disease presented with 6 days of vomiting, loose stools, and poor oral intake. His family members complained of similar symptoms. He presented at an urgent care center whose labs revealed hyponatremia with sodium of 110 mmol/L. In the ED he was hypotensive with physical exam revealing a non-focal neurological exam, non-tender abdomen, and bronze skin according to his wife. Labs came back with worsening hyponatremia at 106 mmol/L. He was admitted to the ICU where he received aggressive hydration with normal saline for the initial diagnosis of hypovolemic hyponatremia in the setting of a positive Norovirus test and history of severe diarrhea. His sodium level did not improve so a cortisol level was ordered which came back low at 2.4 mcg/dL. An ACTH stimulation test demonstrated inadequate stimulation from the adrenal glands which prompted the administration of hydrocortisone. His sodium began to improve with the addition of fludrocortisone and hydrocortisone to 127 mmol/L. His 21 hydroxylase antibodies came back positive confirming primary adrenal insufficiency. Due to his celiac disease, Grave's disease, and Addison's disease he received the diagnosis of autoimmune polyglandular type 4 disease. (Figure)
Discussion: Our case demonstrates that adrenal insufficiency should be considered as the cause of hyponatremia in patients with celiac disease. A positive norovirus became a distractor from his pertinent medical history of celiac disease. Autoimmune polyglandular disease can have an insidious onset as circulating autoantibodies and lymphocytic infiltration may not cause acute symptoms until an inciting event triggers a potentially fatal adrenal crisis. Furthermore, these symptoms have been reported in all ages ranging from infants to older adults. Therefore, a cortisol level can be obtained to avoid precipitation of an adrenal crisis in patients with celiac disease and hyponatremia.



[3386] **Figure 1.** Illustration of the types of autoimmune polyglandular disorders.

S3387

Celiac Disease With Concurrent Stiff Person Syndrome

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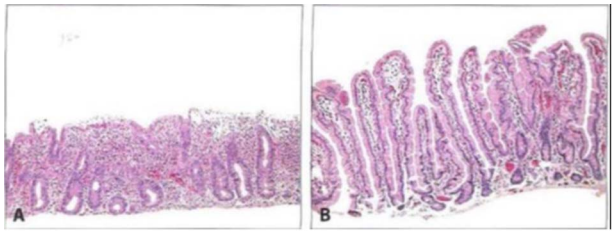
Introduction: Celiac disease (CD) is an immune-mediated gastrointestinal disease with a prevalence of 1% in the general population. It is instigated by the ingestion of gluten in genetically predisposed individuals and has strong associations with HLA-DQ2 and DQ8. It classically manifests with gastrointestinal symptoms, however, extra-intestinal symptoms including neurologic and dermatologic findings such as ataxia and dermatitis herpetiformis can occur. Stiff person syndrome (SPS) is a rare, relapsing-remitting, neurological disorder characterized by truncal and proximal limb muscle rigidity and spasms. We report a rare case of celiac disease in concurrence with stiff person syndrome and review additional autoimmune associations.

Case Description/Methods: A 35-year-old white female with a past medical history of celiac disease, hypothyroidism, and polycystic ovarian syndrome presented with an acute flare of stiff person syndrome. Her symptoms included painful, dystonic posturing in all extremities as well as abdominal spasms and sustained contractions for one week. She was admitted to the neurology service and was treated with plasma exchange and baclofen. Gastroenterology was consulted due to one episode of rectal bleeding with associated abdominal pain and bloating following gluten ingestion. She was placed on a strict gluten-free diet, which resulted in resolution of rectal bleeding and as well as continued improvement of neurologic symptoms. (Figure)
Discussion: Neurological manifestations of celiac disease have been described in the literature. These include gluten ataxia, restless leg syndrome, as well as other movement disorders such as SPS. Celiac disease and stiff person syndrome are strongly associated with other autoimmune diseases. The central genetic factors lie on the major histocompatibility complex. HLA alleles linked with celiac disease are also associated with stiff person syndrome and type I diabetes. Autoantibodies, specifically against glutamic acid decarboxylase, gliadin, and tissue transglutaminase, are theorized to have a significant role in these disease mechanisms. This case highlights the importance of a thorough gastrointestinal diagnostic workup in patients with stiff person syndrome and other movement disorders because of the concomitant nature of autoimmune diseases and disorders. Immune responses in celiac disease may be implicated in the pathogenesis of concurrent SPS and controlling CD may be helpful in resolving and preventing exacerbations of neurological diseases.



[3387] **Figure 1.** A: Celiac disease, flattened villi. B: Normal villi.

S3388

Chronic Norovirus Infection in a Renal Transplant Recipient Resembling Allograft Rejection

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Introduction: We describe an immunocompromised individual with chronic diarrhea who developed an acute kidney injury in the setting of chronic Norovirus infection.
Case Description/Methods: A 75 year-old female with cadaveric renal transplant 10 years prior (on immunosuppression) presented with acute right flank pain, worsening fatigue and unintentional weight loss of 80 lbs for over a year. She was tachycardic and hypotensive with a non-tender right lower quadrant renal transplant. Laboratories showed a bicarbonate of 12 mEq/L, blood urea nitrogen of 35 mg/dL, creatinine of 2.4 mg/dL (baseline creatinine 0.9) and white blood cell count of 4.4 K/mcL. Urine studies showed 3+ protein and protein/creatinine ratio of 7,900 mg/g. Tacrolimus level was appropriate. A renal transplant ultrasound was nonspecific, revealing mildly elevated resistive indices measuring 0.8-0.9 suggestive of transplant dysfunction. Serology for Epstein-Barr virus and Cytomegalovirus was negative. Renal biopsy was obtained and was not consistent with either rejection or polyomavirus nephropathy. Patient reported episodes of watery diarrhea. Over the past year, she had painless intermittent diarrhea often requiring hospitalization. A positive stool viral PCR for Norovirus from 6 months ago was discovered on hospital records. Stool studies of C. difficile, Legionella, Shigella, Campylobacter, Salmonella, Cryptosporidium/Cyclospora and Giardia were negative and positive for Norovirus. She was treated with oral immunoglobulin (**IG**) with a decrease in diarrhea. Her creatinine improved to 1.3.
Discussion: Norovirus infection may progress to chronic infection after a solid organ transplant. Generally self-limiting, norovirus infection in immunocompromised individuals can lead to prolonged symptoms and viral shedding for many years. Minimal information is available in terms of prevalence of chronic Norovirus infection among solid organ transplant recipients. One study showed a prevalence of 4.6% among solid organ recipients. Of these, 22.8% developed chronic infection, with a median shedding period of 218 days. Currently, there are no approved human antiviral agents that are effective against Norovirus. Treatment using oral IG was extrapolated from its role in management of primary immunodeficiency conditions. In our patient, viral shedding continued but oral IG provided symptomatic relief, decreasing the progression of kidney injury. Physicians should be made aware of this particular presentation and treatment options in renal transplant patients.

S3389

Chronic Diarrhea Caused by Duodenal Diverticulum

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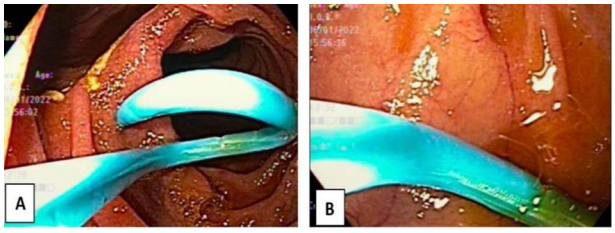
Introduction: Diverticula are bulging sacs that can occur in any part of the gastrointestinal tract. While mostly seen in the large intestine, 9–23% of small bowel diverticula are found incidentally via endoscopy. Small intestinal bacterial overgrowth (SIBO) is characterized by an abnormally increased bacterial burden in the small intestine with symptoms including abdominal distension and pain, bloating, and diarrhea. Small intestinal diverticulosis is a known risk factor for SIBO. Small bowel aspirates and culture is considered gold standard for diagnosing SIBO, while aspirates from diverticula is not routine practice for diagnosis. We present a case of SIBO diagnosed by aspirates from a large duodenal diverticulum (DD).
Case Description/Methods: A 62-year-old female with chronic diarrhea, hepatic steatosis without cirrhosis, GERD, and diverticulosis presented to the gastroenterology clinic for continued complaints of diarrhea and bloating. The patient noted 4-5 bowel movements per day. Vital signs, physical exam, and previous workup including stool culture, *Clostridium difficile* study, colonoscopy with biopsies, fecal calprotectin and elastase, abdominal imaging, and celiac serologies were unremarkable. Patient tried pancrelipase with no noticeable difference in diarrhea. An esophagoduodenoscopy (EGD) performed showed multiple fundic gland polyps in the stomach and a large 40 mm diverticula in the 3rd portion of the duodenum. Duodenal and gastric biopsies obtained ruled out celiac disease, pathogens, and *Helicobacter pylori*. Aspiration of duodenal diverticular fluid was significant for >100,000 colony forming units per mL (CFU/mL) *Escherichia coli*, *Enterococcus faecium*, and *Streptococcus mitis*. Disaccharidases (lactase, sucrase, maltase, palatinase, glucoamylase) were within normal limits. Patient was initiated on rifaximin 550mg TID for 14 days with improvement of her chronic symptoms. (Figure)
Discussion: While most DD are asymptomatic, small, and require no treatment, intervention is necessary if the patient develops SIBO, bowel obstruction, diverticulitis, bleeding, or perforation. SIBO can be diagnosed via small bowel aspirates showing >1000 CFU/mL. Large DD can lead to SIBO by creating a protected environment for bacterial species. Treatment of diarrhea due to SIBO and large DDs are treated with antibiotic therapy, like our patient who was prescribed rifaximin and noted improvement of symptoms. It is important to understand that while DD are uncommon, large diverticulum can lead to chronic diarrhea and SIBO.



[3389] **Figure 1.** [A] EGD findings with a large duodenal diverticulum in the upper left corner; [B] Process of aspirating duodenal diverticulum fluid.

S3390

Celiac Disease and Systemic Eosinophilia: A Case Report

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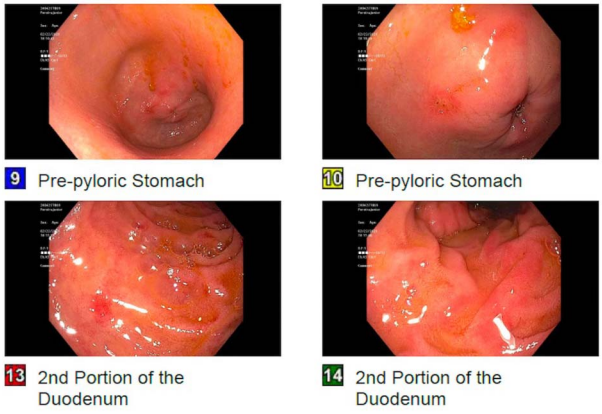
Introduction: Celiac disease is an immune mediated reaction to the gluten protein. this disease primarily affects the small intestine.it occurs in population with genetic predisposition, usually resolves with gluten free diet regimen. it was reported the association between eosinophilic gastrointestinal diseases and celiac disease but in the following case will discuss patient with significant peripheral eosinophilia and celiac disease without Eosinophilic gastrointestinal disorder.
Case Description/Methods: 45 years old male, with history of hypertension, who presented with progressively worsening intermittent colicky lower abdominal pain for the past 2 months. Associated with recurrent episodes of nausea and vomiting, and non-bloody watery diarrhea.Patient Labs were significant for Hemoglobin 12 gm/dL, WBC's 26,000 with 62% Eosinophils (16,500 /uL), Platelet of 332 Thou/uL. Peripheral blood smear was negative for parasites, stool was negative for parasites or bacterial infection. Patient went upper GI endoscopy that showed grade A reflux esophagitis, congestive gastropathy, Duodenitis, and non-bleeding gastric ulcer, Biopsies were obtained during the EGD. Pathology showed Duodenal mucosa with mild villous atrophy and focal increased intraepithelial lymphocytes suggestive of celiac disease,chronic gastritis, esophageal biopsy was negative for increased eosinophils. Patient was started on gluten free diet that was resulted in gradual resolution of his symptoms and also normal eosinophilic count after 1 month. (Figure)
Discussion: Patient with celiac disease can present with typical gastrointestinal symptoms such as diarrhea, weight loss, bloating, abdominal pain, and also non-gastrointestinal abnormalities such as abnormal liver function test, iron deficiency anemia, and it may be asymptomatic. In this case patient presented with typical symptoms but was associated with significant peripheral eosinophilia, without evidence eosinophilic gastrointestinal disorder.



[3390] **Figure 1.** EGD showing stomach and duodenum.

S3391

Chronic Sclerosing Mesenteritis: An Uncommon Cause of Chronic Diarrhea

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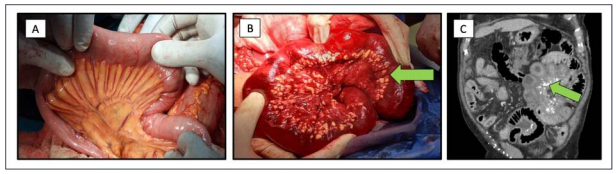
[1]Cooper Medical School of Rowan University, Camden, NJ; [2]Cooper University Hospital, Camden, NJ.

Introduction: Sclerosing mesenteritis is an uncommon condition where chronic inflammation and necrosis of mesenteric adipose tissue leads to progressive fibrosis. It typically presents in the fifth decade of life and its etiology is unclear, although it has been linked to abdominal trauma, autoimmune processes, ischemia, and malignancy. Depending on the degree of fibrosis it can present with symptoms such as abdominal pain, palpable mass, or bowel obstruction. Reexamination of imaging has shown the true prevalence of this disease to be higher than the diagnostic prevalence (2.5% vs 0.6%), indicating most cases are asymptomatic. Chronic diarrhea is a particularly atypical presentation of this condition, being reported in only one-quarter of diagnosed patients.
Case Description/Methods: Here we present a case of chronic intermittent diarrhea in a 86 year old man with a history of biopsy-confirmed sclerosing mesenteritis. Over three years, the patient had recurrent watery diarrhea without abdominal pain treated with loperamide, culminating in a refractory episode requiring admission. Imaging showed scattered calcifications and soft tissue densities in the small bowel mesentery, consistent with chronic sclerosing mesenteritis. Computed tomography angiography ruled out mesenteric ischemia, a known complication of sclerosing mesenteritis. Workup also revealed normal leukocyte and lactic acid levels, as well as negative stool cultures and Clostridioides difficile test. After three days of supportive care, the patient's diarrhea resolved and he was discharged. (Figure)
Discussion: This case highlights the association between sclerosing mesenteritis and chronic diarrhea. The lack of symptoms commonly associated with sclerosing mesenteritis, such as palpable mass or abdominal pain, shows that nonspecific gastrointestinal symptoms like diarrhea can serve as the disease's primary presentation. Therefore, it is prudent to consider this disease in the workup of chronic diarrhea and obtain imaging when indicated, as these patients may qualify for advanced therapies. Since immunosuppressive agents are the standard of care for symptomatic sclerosing mesenteritis, the escalation of this patient's diarrhea to a point requiring hospitalization suggests that diarrhea may be sufficient justification for initiation of immunosuppressive therapy in diagnosed patients, although further research is needed to establish this utility.



[3391] **Figure 1.** (A) Normal Mesentery. (B) Mesentery with calcifications, as seen in sclerosing mesenteritis. (C) Coronal slice of patient's abdominal CT, showing calcifications in mesentery (green arrow) consistent with sclerosing mesenteritis.

S3392

Disseminated Histoplasmosis Causing Peritoneal Implants in Patient Receiving Tumor Necrosis Factor-Alpha Inhibitor

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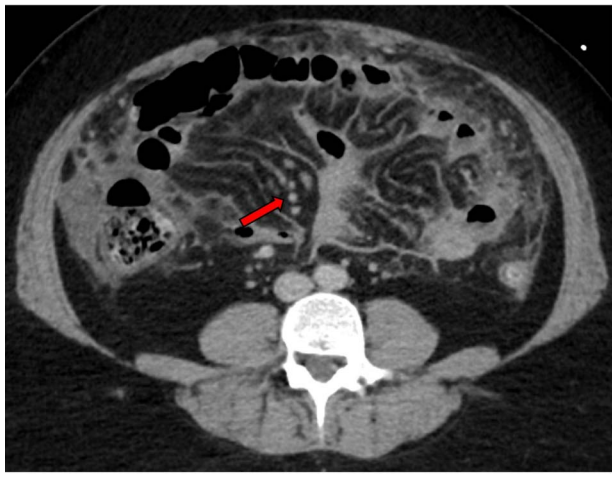
Introduction: Histoplasmosis is an endemic mycosis commonly found in the Ohio River valley and presents with various clinical presentations ranging from asymptomatic to disseminated disease. Most patients present with pulmonary symptoms, while the immunocompromised subset can have multiple organ involvement with varying symptomology. An increasing number of patients are being treated with tumor necrosis factor (TNF)-alpha inhibitors, which dampens immune response and puts these patients at risk of opportunistic infections. We report a rare presentation of disseminated peritoneal histoplasmosis initially mimicking peritoneal carcinomatosis in a patient with rheumatoid arthritis on a TNF-alpha inhibitor.
Case Description/Methods: A 43-year-old female with known history of rheumatoid arthritis (RA) on leflunomide, infliximab, and recent steroid course presented to the ED for abdominal pain and shortness of breath. On admission, the patient was febrile, tachycardic, and required supplemental oxygen. CT chest/abdomen/pelvis demonstrated right-sided pleural effusion, diffuse mesenteric nodules along the anterior abdominal wall concerning for malignancy, and moderate ascites (Figure A). Tumor marker CA 125 was elevated at 341 U/mL and surgical oncology was consulted for diagnostic laparoscopy with drainage of ascites, which was initially concerning for peritoneal carcinomatosis. Surprisingly, the biopsy revealed chronic inflammation, granulomas, and fungal yeast cells with morphological features consistent with histoplasmosis. She underwent thoracentesis, and pleural studies were consistent with exudative effusion with lymphocyte predominance, and cultures confirmed histoplasmosis. Initial broad antibiotic therapy for sepsis of unknown origin was transitioned to itraconazole and she was eventually discharged. More than a year after hospitalization, she remains on itraconazole due to persistent histoplasmosis. Her RA treatment was switched to upadacitinib, with some control of her symptoms.
Discussion: Diagnosis of histoplasmosis can be challenging because of its heterogeneous presentation. Disseminated histoplasmosis with peritoneal implants is exceeding rare, and most cases have been reported in ESRD patients on continuous peritoneal dialysis. Other cases of peritoneal histoplasmosis have been reported in immunosuppressed patients, which mimicked pathologies such as malignancy and IBD. It is essential to be vigilant of uncommon presentations of common disease, especially in immunocompromised patients.



[3392] **Figure 1.** Diffuse nodularity to the mesentery most prevalent along anterior abdominal wall right paracolic gutter regions concerning for metastatic disease to the mesentery (Arrow). Mild bowel wall thickening seen involving descending colon.

S3393

Diffuse Large B Cell Lymphoma Presenting as Bleeding and Hypoalbuminemia: A Case Report

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Introduction: Diffuse large B cell lymphoma (DLBCL) accounts for 38-57% of primary gastrointestinal (GI) lymphomas. Initial presentation can include nonspecific symptoms, such as weight loss, fatigue, and nausea. Imaging studies are typically nonspecific as well. Therefore, patients are commonly misdiagnosed or diagnosis is delayed. 20-30% of GI lymphomas are in the small intestine but are rarely in the jejunum. We present a rare case of jejunal DLBCL and illustrate the diagnostic difficulties.
Case Description/Methods: A 49-year-old male presented with weeks of dizziness, dyspnea, hematochezia, and 30-pound unintentional weight loss over 4 months. Labs were notable for anemia and severe hypoalbuminemia (1.9 g/dL). Esophagogastroduodenoscopy and colonoscopy revealed diverticulosis and hemorrhoids, which were the presumed etiology of his symptoms. One month later, the patient presented similarly. Imaging demonstrated bilateral pulmonary emboli and intramural thickening of the jejunum. For worsening hypoalbuminemia (1.3 g/dL), the patient was scheduled for outpatient work-up. However, he presented again for acute GI bleed and severe anemia. Push enteroscopy revealed patchy mucosal changes in the jejunum and biopsies were consistent with DLBCL. The patient was started on chemotherapy and reported improvement in symptoms and resolution of his GI bleed.
Discussion: Primary GI DLBCL is rarely found in the jejunum due to the lack of lymphoid tissue, making this case noteworthy. GI DLBCL is difficult to diagnose, since patients may be asymptomatic or present with nonspecific symptoms. Small intestinal DLBCL can present with GI bleeding, which can easily lead to a misdiagnosis of a diverticular or hemorrhoidal bleed. Imaging studies can be nonspecific and obtaining confirmation through biopsy can prove difficult due to its location, further complicating the diagnosis. One unique aspect of this case that led to the diagnosis was the severity of hypoalbuminemia. The patient was not malnourished, had no clinically apparent protein-losing enteropathy, and no proteinuria or liver failure, making underlying malignancy likely. Differential diagnoses of small bowel wall thickening on imaging is wide and includes infections, primary inflammatory processes such as IBD, and neoplastic lesions. DLBCL may have unusual clinical presentations, subtle laboratory findings and may not be easily diagnosed with initial imaging; thus, clinicians should remain vigilant of this possible diagnosis.

S3394

CT of a Greyhound? A Case of Superior Mesenteric Artery Syndrome

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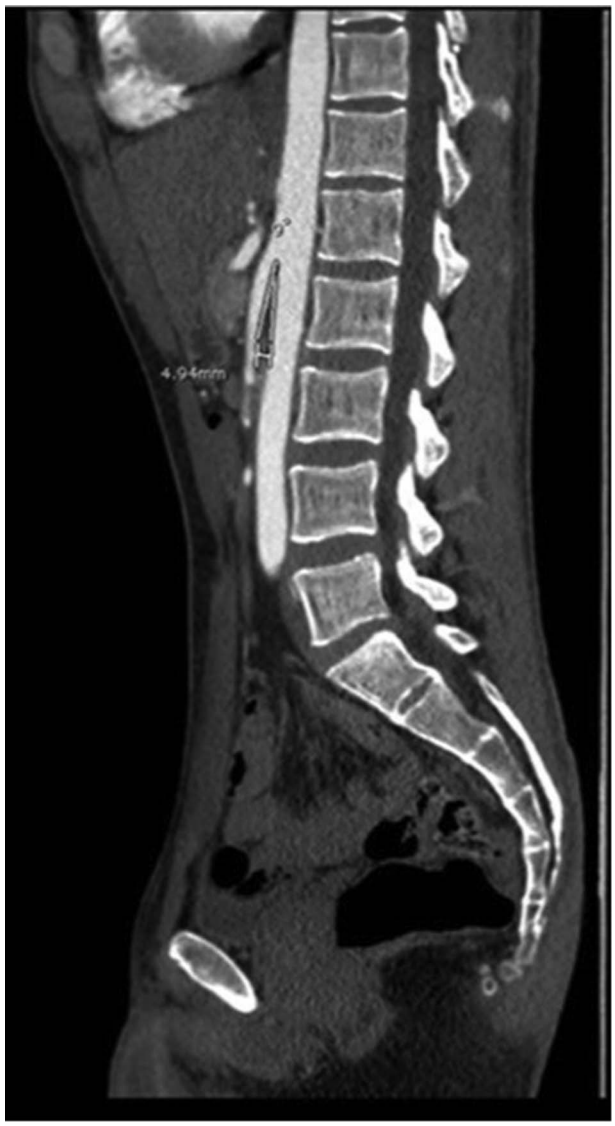
Introduction: Superior Mesenteric Artery Syndrome (SMA) is a rare disease defined by compression of the duodenum due to loss of the intervening fat pad between the superior mesenteric artery and aorta leading to nausea, vomiting, and abdominal pain. Diagnosis requires a high index of suspicion as late diagnosis can lead to significant morbidity and mortality.
Case Description/Methods: A 31 year old active duty male presented with unintentional weight loss of 40 pounds and progressive oral intolerance for 6 months. Prior to his more precipitous weight loss, he had intentionally been losing weight in preparation for his military physical fitness assessment. After achieving his target weight, he noted continued weight loss. For 5 months his symptoms persisted and expanded to include post-prandial epigastric pain, nausea, and vomiting resulting in a hospital stay for intractable symptoms. Upper endoscopy performed revealed significant compression of the third portion of the duodenum and retained residue in the stomach despite 24 hours of fasting. Subsequent cross sectional imaging showed an aortomesenteric angle of 9 degrees with a distance of less than 5mm. He was referred to nutrition for conservative management with aggressive caloric repletion of 3-4000 calories/day, slowly regaining weight over the next 6 months. (Figure)
Discussion: We present a case of SMA syndrome which developed after intentional weight loss for military service requirements, with endoscopic findings concerning for duodenal outlet obstruction. SMA is an rare diagnosis with incidence reports at 0.1-0.3%. Patients most at risk of developing SMA syndrome typically have chronic debilitating illnesses that lead to severe weight loss. The normal angle between the superior mesenteric artery and aorta ranges from 38 to 65 degrees with a distance of 10 to 28mm. Symptoms typically begin with an aortomesenteric angle of 22-28 degrees and distance of $<$ 8mm. Most patients can recover with aggressive nutritional support however 25-30% fail to gain weight in the first 6 weeks, requiring surgical intervention. This case illustrates that even otherwise healthy patients who make efforts at rapid weight loss can be at risk of this rare condition and highlights the importance of maintaining a high index of suspicion in patients with unexplained weight loss as the symptoms are non-specific. For this patient, not only was the presenting weight loss abnormal but the severity of the angle to less than 9 was significant and notable.



[3394] **Figure 1.** Abdominal CT providing diagnosis of SMA.

S3395

Enterocolitis as the Initial Presentation of Systemic Lupus Erythematosus

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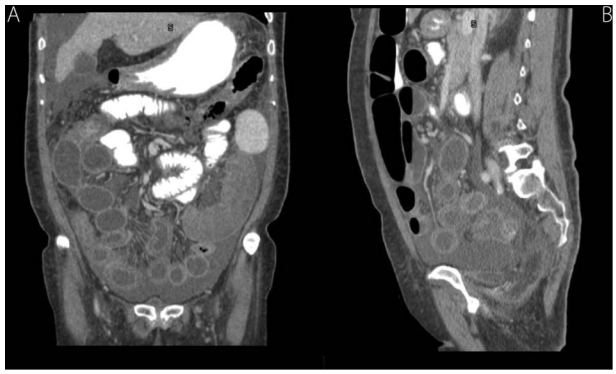
Crozer Chester Medical Center, Upland, PA.

Introduction: Systemic Lupus Erythematosus (SLE) is a chronic inflammatory autoimmune disease that can have gastrointestinal manifestations related to immune complex deposition and vasculitis. Symptoms are non-specific and include abdominal pain and diarrhea.
Case Description/Methods: A 66-year-old Asian woman with no prior medical history presented to the hospital with two months of generalized abdominal pain, nausea and watery diarrhea for 2 months. She reported a 10 lb unintentional weight loss. Initial labs revealed hemolytic anemia that was coombs positive with hemoglobin of 6.5 g/dL, as well as proteinuria. Contrast-enhanced CT of the abdomen & pelvis revealed moderate ascites, with normal appearing liver. Additionally evident was diffuse mural thickening of the descending colon, sigmoid colon, with the distal jejunum and ileum demonstrating "target signs" (Figure A & B). Stool studies were negative for infectious etiology. Upper endoscopy and colonoscopy revealed esophagitis, gastritis but normal appearing duodenum and colon. Single balloon enteroscopy revealed mild jejunitis and ulceration with biopsies suggesting chronic inflammation. Biopsies were negative for amyloidosis. Paracentesis revealed a serum ascites albumin gradient (SAAG) of < 1.1. She met the SLICC criteria for a diagnosis of SLE with coombs positive hemolytic anemia, positive ANA titer of 1:1280, anti ds-DNA titer 1:320, low completement levels and ascites. Her radiologic and endoscopic findings were attributed to lupus related enterocolitis. She was begun on intravenous methylprednisolone. Her symptoms progressively improved, after which steroids were tapered and she was transitioned to hydroxychloroquine therapy.
Discussion: We report here a case of enterocolitis as the initial presentation of SLE. Mesenteric vasculitis is the underlying mechanism, and patient can develop ascites as well as noted in our patient. Common sites of bowel involvement are the jejunum (80%) and the ileum (85%). Symptoms are generally non-specific. CT imaging has become the gold standard in diagnosis with findings of bowel wall edema ("target sign") and engorgement of mesenteric vessels ("combs sign"). Lupus enterocolitis occurs in the setting of active SLE, and is visualized endoscopically as ischemic enteritis or chronic colonic ulcerations. This disease demonstrates favorable response to steroids. Early suspicion and prompt management is essential to prevent complications such as bowel ischemia or perforation.



[3395] **Figure 1.** Computerized tomography of the abdomen & pelvis in coronal [A] and sagittal views [B] demonstrated diffuse mural thickening of the jejunum and ileum in a "target sign" pattern.

S3396

Duodenal Polyposis: An Incidental Finding of Duodenal-Type Follicular Lymphoma

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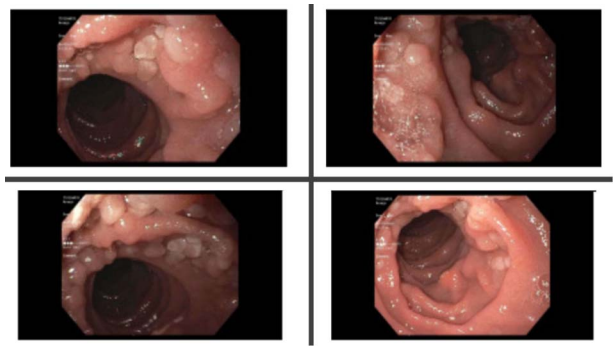
Introduction: Duodenal polyps commonly present as incidental findings on upper endoscopies (EGD), and often represent regenerative/hyperplastic nodules. The majority of neoplastic lesions are either adenomatous lesions of intestinal or gastric phenotype. A newly recognized entity of duodenal polyposis is duodenal-type follicular lymphoma (DFL). We present a rare case of duodenal polyposis from DFL as an incidental finding on a screening endoscopy
Case Description/Methods: A 58-year-old non-smoking man with no significant medical history underwent EGD for longstanding GERD symptoms refractory to PPI therapy in absence of other symptoms and unrevealing physical examination. Family history was notable for gastric carcinoma in father at age 58. Due to his risk factors, EGD was performed and notable for gastric inflammation and duodenal polyposis with multiple semi-sessile polyps in the second portion of the duodenum. Gastric biopsies revealed moderate acute and chronic gastritis, with a positive immunohistochemical stain for H-Pylori. Duodenal mucosa had abundant lymphoid aggregates, comprising of mainly CD20+ B-cells. B-cell follicles were positive for BCL2, BCL6, and CD10. Alongside the markedly nodular duodenum, these findings were consistent with B-cell lymphoma. Laboratory studies were largely unremarkable with normal CBC with borderline high uric acid 8.0 mg/dl (3.4-7.8 mg/dl), and LDH 226 u/L (125-220). Secondary involvement by a systemic B-cell lymphoma was excluded by a normal bone marrow and PET scan. Whole body PET scan was negative for any evidence of FDG avid malignancy. Patient was diagnosed with a lowgrade, stage-1 FL. Treatment options included localized radiotherapy with a curative intent or expectant monitoring off therapy. The patient's chronic GERD symptoms were attributed to gastritis caused by H. pylori, which isn't considered to play a role in the development of DFL. (Figure)
Discussion: DFL is a newly recognized variant of follicular lymphoma, with a low grade on presentation and benign evolution. The most common endoscopic finding is the presence of white nodular lesions confined within the duodenum, as described in this case, with rare appearances in other areas of the small intestine. Due to excellent prognosis and rare progression even in absence of treatment, watchful waiting is frequently favored. Endoscopists should be aware of the well-defined appearance of DFL, and the importance of ruling out extra duodenal involvement in order to present the patient with appropriate treatment options.



[3396] **Figure 1.** Polyposis noted in the second portion of the Duodenum.

S3397

Eosinophilic Enteritis: An Uncommon Cause of Ascites

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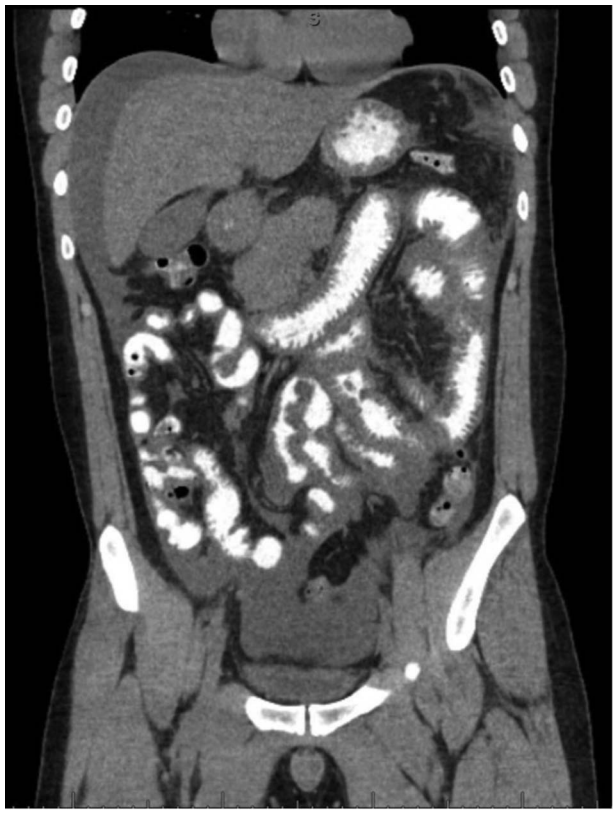
Introduction: Eosinophilic enteritis (EE) is a rare subtype of eosinophilic gastrointestinal disease characterized by eosinophilic infiltration of the small intestinal wall in the absence of secondary cause. The disease is further classified by depth of involvement: mucosal, muscular and subserosal, each with unique phenotype. Of these, subserosal is the rarest and eosinophilic ascites is regarded as the idiosyncratic feature of this pattern. We report a patient presenting with gastrointestinal symptoms and eosinophil rich ascitic fluid. Prior similar flare and robust steroid response supported a diagnosis of EE.
Case Description/Methods: A 23-year-old Asian male presented with 1-week history of nausea, vomiting and abdominal distension. 5 years ago, he had similar symptoms. Imaging was notable for ascites and thickening of esophagus and jejunum. Abdominal fluid analysis revealed eosinophilic ascites. After exclusion of other causes of eosinophilia, he underwent bi-directional endoscopy. Mucosa was normal in appearance and only esophageal biopsies were notable for eosinophilic infiltration. A presumptive diagnosis of EE was made and symptoms resolved rapidly with prednisone. On current admission serology was notable for 32% eosinophils; other lab parameters were within normal limits. Imaging again noted ascites and small bowel thickening (Figure). Ascitic fluid was noted to comprise of 76% eosinophils after paracentesis. Alternate causes of eosinophilia were again excluded and symptoms resolved within 2 days after initiation of prednisone.
Discussion: EE is classified by layer of gut wall involvement. Mucosal causes diarrhea, pain and malabsorption; muscular predisposes to obstruction and subserosal tends towards ascites. The subserosal variant is rarest and endoscopic biopsy is commonly non-diagnostic due to remoteness of the serosa from the lumen. In most cases, laparoscopic full-thickness biopsy is necessary for histological confirmation. Evaluation must also exclude alternate causes of eosinophilia such as parasitic infections, adrenal dysfunction etc. Despite negative biopsy, the presence of peripheral eosinophilia, eosinophilic ascitic fluid and quick resolution of symptoms with prednisone, strongly supported a diagnosis of EE in this patient. Support for the use of glucocorticoids, the mainstay of therapy, is gleaned from small sample case studies, underscoring the necessity of supplemental research in this area. A combination of peripheral and ascitic eosinophilia should prompt consideration of EE.



[3397] **Figure 1.** Coronal section abdominal computerized tomography showing abdominal and pelvic ascites in addition to small intestinal wall thickening.

S3398

Duodenal Follicular Lymphoma: Two Distinct Presentations of a Rare Disease Entity

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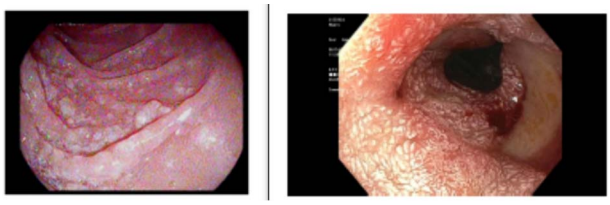
Introduction: Follicular Lymphoma (FL), a B cell neoplasm, is the second most common type of nodal NHL with frequent duodenal involvement mostly presenting with non-specific abdominal pain. Primary Gastrointestinal Non-Hodgkin Lymphoma (PGINHL) is a rare entity accounting for 2% of all small intestinal malignancies. Duodenal Follicular Lymphoma (DFL) only accounts for 1 to 6% of PGINHL. We present two cases of DFL with variable symptoms.
Case Description/Methods: Case 1: A 58-year-old female presented with nausea, vomiting, acid reflux, and 60-pound weight loss over one year. She denied any fever, night sweats, family history of malignancy and had a normal upper and lower endoscopy 5 years ago. EGD showed nodular mucosa in the 2nd portion of the duodenum confirmed to be low-grade FL on biopsy. Metastatic workup showed stage 4 lymphoma. There was no evidence of bulky lymphadenopathy and diagnosis of DFL was made. Patient finished 6 cycles of R-CHOP therapy due to concern for recurrent/residual DFL and currently remains indolent. **Case 2:** A 41-year-old male presented with coffee-ground emesis, melena, and 10 lbs weight loss over a month in absence of NSAID use. He also had fever, palpitations, and diaphoresis. Patient's grandfather had colon cancer and the mother had non-Hodgkin lymphoma and breast cancer. EGD and colonoscopy 9 months prior to presentation showed erosive gastritis and tubular adenoma, respectively. Repeat EGD showed abnormal duodenal mucosa with an area of oozing at the site of the biopsy. Pathology report supported the diagnosis of low-grade DFL. Metastatic workup showed a mediastinal mass, a large 7.7 × 4.9 × 7 cm mesenteric nodal mass, and a 5.8 cm × 5.9 cm duodenal mass involving the head of the pancreas. The patient moved to Iowa for further workup (Figure).
Discussion: DFL is an extremely rare PGINHL with an indolent course and good prognosis. DFL has unique features in that immunohistochemically it is similar to nodal FL, however, gene expression analysis groups it closer to MALT lymphoma, and thus, treatment options are heterogeneous with no established guidelines. 'Wait and Watch' strategy is often implemented in patients with low-grade disease, however, chemotherapy, radiation therapy, and immunotherapy have also been explored. Treatment of DFL with antibiotics owing to its similarity to and association with MALT lymphoma and H. pylori respectively has had mixed results. The two cases described above highlight heterogeneous presentation, dyspepsia, and upper GI bleed, of a rare malignancy - DFL.



[3398] **Figure 1.** The image on the left shows whitish nodular mucosa in the second portion of the duodenum in Case 1. The image on the right shows abnormal duodenal mucosa in Case 2.

S3399

Duodenal Metastasis From Primary Adenocarcinoma of the Lung

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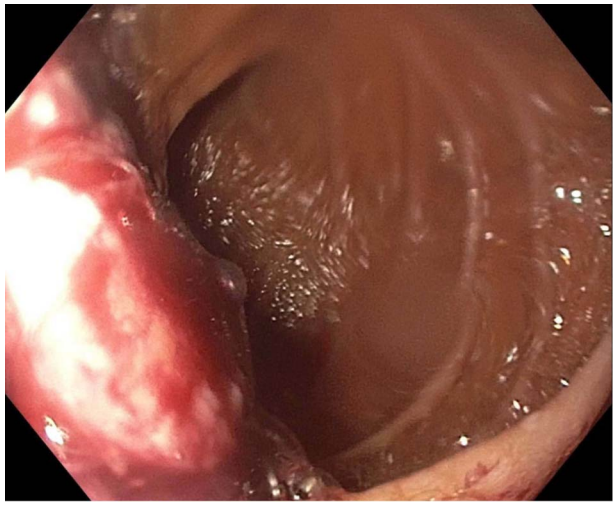
Introduction: Adenocarcinoma (NSCLC) is the most common primary lung cancer in the United States, making up 30% of all lung cancers and accounting for nearly 25% of all cancer mortality. Lung cancer metastasis occurs in many organs, including the adrenal glands, bone, lymph nodes, brain and liver. More commonly associated with small cell lung carcinoma, metastasis of non-small cell lung carcinoma to the stomach and small bowel have been rarely reported, many of which were found incidentally or during autopsy. We present a case of a patient with NSCLC who developed metastasis to the duodenum almost four months after diagnosis.
Case Description/Methods: The patient is a 43 year old female with a medical history of NSCLC and recent pulmonary embolism treated with eliquis presented with intermittent episodes of hematemesis and hemoptysis for three weeks. She described medium sized blood clots during episodic coughing and vomiting. On exam, she was afebrile, BP was 187/78, pulse 140, RR 26, and SpO2 94% on room air. She was ill appearing exhibiting epigastric tenderness without guarding. Lab studies revealed a Hg of 9.3, ALK 296, AST 67, ALT 56 and an elevated lactic acid at 4.4. All other lab tests were normal. Two large bore IVs were placed, initiated PPI, type and screened. Eliquis was stopped and the patient was transferred to the ICU for further monitoring. An EGD revealed a 2.5 cm fungating mass in the first portion of the duodenum, which did not require cauterization or clipping. Pathology reported a malignant mass and cells consistent with metastatic carcinoma from the lung primary. After discussing the pathology results with the patient, a decision was made to discontinue anticoagulation as the risk of bleeding outweigh the risk of pulmonary embolism (Figure).
Discussion: Common primary tumors that metastasize to the duodenum are renal cell carcinoma, melanoma, breast cancer and small cell carcinoma of the lung. Metastasis to the small intestine may be indolent and a challenge to diagnosis given vague symptoms like nausea, vomiting, abdominal pain and lack of overt bleeding, which can lead to a delay in endoscopy and diagnosis. In this case, anticoagulation likely exacerbated bleeding resulting in a need for an EGD. Duodenal metastasis is a grim prognosis with a survival rate less than 12 months. The case displays that distant metastasis from a primary malignancy can present with vague gastrointestinal symptoms and should not be discarded in certain clinical settings.



[3399] **Figure 1.** 2.5 cm fungating mass in the first portion of the duodenum.

S3400

Elevated Ascitic Fluid Amylase? Think Twice It Could Be a Foreshadowing of Intestinal Perforation

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Introduction: Elevated ascitic fluid amylase concentration is characteristic of pancreatic ascites. Nevertheless, ascitic amylase values greater than 2000 u/L can also be seen in intestinal perforation. We present a case of a patient without evident risk factors for fungal peritonitis whose ascitic fluid was notable for yeast and elevated amylase with a final diagnosis of intestinal perforation.
Case Description/Methods: A 54-year-old female with a medical history of metastasized transitional cell urethral cancer arrived at our institution due to persistent non-bloody vomiting, diffuse abdominal pain, and worsening abdominal distension for a month. Initial evaluation was a remarkable food distended, tense abdomen with normoactive bowel sounds and positive fluid wave. Images were remarkable for large amounts of ascites, with a faint enhancement of the peritoneal surface consistent with acute peritonitis and findings concerning partial small bowel obstruction with free retroperitoneal air of unknown origin. The patient was started on intermittent suction via a nasogastric tube. Diagnostic and therapeutic paracentesis was performed. CT urogram was performed due to concern of bladder perforation, which was remarkable for diverticular microperforation. Ascitic fluid analysis was consistent with secondary peritonitis, with yeast species and markedly elevated ascitic amylase at >7,500 u/L in the setting of normal pancreatic enzymes. Empiric coverage for fungal peritonitis was started with caspofungin. Hospitalization was complicated by shock and acute abdomen. Emergent imaging revealed duodenal jejunal mesenteric ischemia with perforation of the proximal jejunum and peritonitis. The patient underwent exploratory laparotomy with palliative gastrostomy, lysis of adhesions, and peritoneal lavage. After the intervention, blood cultures came back positive for candida glabrata. The patient completed therapy for fungemia and was discharged to hospice care.
Discussion: Candida peritonitis is most seen as secondary peritonitis. It generally occurs as a result of translocation of micro-organisms across the bowel wall, perforation of a hollow viscus, or instrumentation of the gastrointestinal tract. It is associated with high morbidity and mortality. This case raises awareness of careful evaluation of elevated ascitic amylase with concomitant yeast species in ascitic fluid gram stain, as they can both be indicators of intestinal perforation where a surgical approach can be lifesaving.

S3401

Duodenal Diverticulitis Mimicking Acute Pancreatitis

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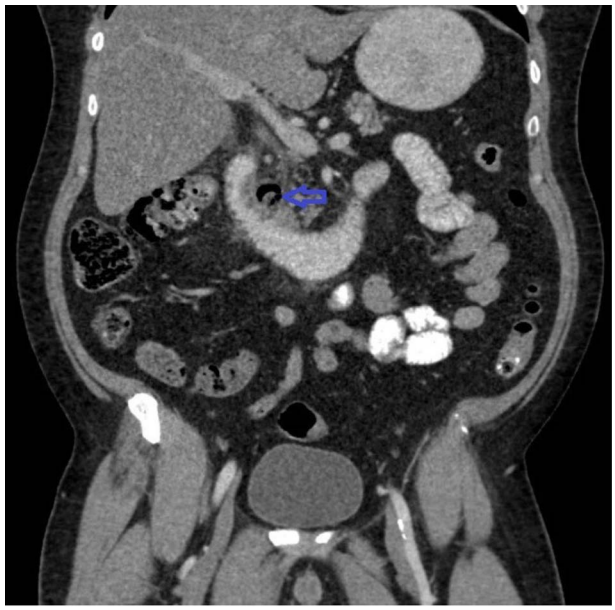
Introduction: Duodenal diverticulum is a frequent and common asymptomatic incidental finding and is commonly located at the second part of the duodenum. However, complications have been reported such as inflammation of the diverticulum, hemorrhage and perforation. Its presentation can mimic arrays of abdominal pathologies. In our case, patient presented with epigastric pain similar to the presentation of acute pancreatitis.
Case Description/Methods: Patient is a 52 yo male with medical history of type 2 DM and hypertension who presented to the ED for epigastric pain for two days duration. It was a sudden onset moderate progressive stabbing epigastric abdominal pain radiating to the back, associated with nausea, however patient denies vomiting, changes in bowel habits, fever, or chills. Upon arrival to the ED, his vital signs were within normal limits, his blood work demonstrated leukocytosis of 11.8 K/CMM, Hb 13.3 g/dL with MCV of 80 fL and CMP values were within normal limits. Blood cultures showed no evidence of microorganisms, he had an US of the abdomen which showed hepatic steatosis and hepatomegaly with no evidence of cholecystitis, chest X-Ray was also done which had unremarkable findings. Further radiological studies were ordered including CT abdomen and pelvis with contrast (Figure) which had significant findings of inflammatory changes in the second portion of the duodenum in the setting of duodenal diverticulum, so the patient was admitted for management of duodenal diverticulitis and was started on Flagyl IV 500 mg every 8 hour interval and Ciprofloxacin IV 400 mg daily for one day and then antibiotics were changed to Unasyn 3 g every 6 hour interval. Patient's abdominal pain resolved after 4 days of antibiotics. He had an EGD which showed small erosion in the antrum as well as mild duodenitis with diverticular deformity, no ulcers or masses were noted in the duodenum. Prior to discharge, MRI of the abdomen was ordered which showed duodenal diverticulitis that was regressing compared to prior CT study. Patient was discharged home with a follow up with a gastroenterologist two weeks after discharge.
Discussion: Duodenal diverticulitis is a possible differential diagnosis in acute abdominal pain. It is diagnosed with computed tomography showing inflammatory changes in duodenal diverticulum and managed medically with antibiotics covering gram negative bacteria as with our patient, however if complications like perforation occur, more aggressive interventions should be taken.



[3401] **Figure 1.** CT abdomen and pelvis with contrast showing significant inflammatory changes in the second portion of the duodenum in the setting of duodenal diverticulum.

S3402

Enterocolitis Caused by Mycobacterium Avium Complex in an HIV-Infected Patient

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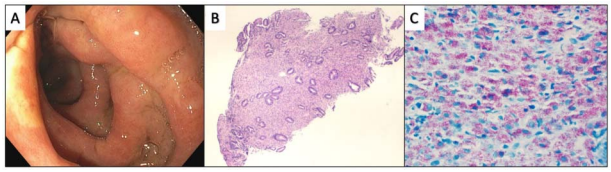
Introduction: Diarrhea in patients with human immunodeficiency virus (HIV) can be due to a variety of etiologies including infection, malignancy, and antiretroviral therapy (ART). With increased use of ART, diarrhea due to opportunistic infections has become less common. Here we report a rare case of chronic diarrhea due to *Mycobacterium avium* complex (MAC) infection involving the duodenum, colon, and rectum of a patient with advanced HIV.
Case Description/Methods: A 41-year-old man with HIV infection diagnosed two years earlier presented with progressively worsening diarrhea. Two months prior, his CD4 count was stable at 50 cells/uL and his HIV level was undetectable while on dolutegravir/lamuvidine. He had history of MAC mycobacteremia under treatment with ethambutol, azithromycin, and rifabutin. He also had cytomegalovirus (CMV) viremia treated with valganciclovir. He reported compliance with the above medications. He had eight-month history of frequent, watery diarrhea associated with malaise, nausea, bloating, abdominal pain, and weight loss. He was hospitalized for severe malnutrition and electrolyte abnormalities. Evaluation of his diarrhea included negative *C. difficile* toxin, enteric pathogen panel, and ova and parasite exam. CMV was detected in the blood at 17,000 IU/mL and HIV viral load was 6,166 IU/mL. Esophagogastroduodenoscopy was performed and revealed diffuse erythema and edema affecting the mucosa of the entire duodenum with associated villous blunting and fissuring (**Figure A**). Flexible sigmoidoscopy revealed normal appearing mucosa to the level of the ascending colon. Pathology from duodenal biopsies showed extensive histiocytic inflammation with presence of acid-fast bacillus organisms (**Figures B and C**). Random biopsies from the colon and rectum showed similar findings and no evidence of CMV. Acid-fast bacillus blood cultures grew *Mycobacterium avium-intracellulare*. His MAC treatment was changed to amikacin, linezolid, rifampin, and ethambutol with subsequent improvement in his diarrhea.
Discussion: Gastrointestinal tract involvement by MAC is usually part of a disseminated MAC infection in patients with advanced HIV, especially those with CD4 lymphocyte counts $<$ 50 cells/uL. The duodenum is most often affected, followed by the rectum. We report this case to raise awareness that MAC enterocolitis remains an important etiology of chronic diarrhea in patients with advanced HIV.



[3402] **Figure 1.** A, EGD showing duodenal involvement by MAC causing diffuse erythema and edema with villous blunting and fissuring. B, H&E stain of duodenal mucosa with extensive histiocytic inflammation. C, Ziehl-Neelsen stain of duodenal mucosa with acid-fast bacillus positive organisms consistent with *Mycobacterium avium* complex.

S3403

Duodenal Obstruction Secondary to Metastatic Urothelial Carcinoma: A Novel Presentation of a Common Malignancy

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Introduction: Urothelial carcinoma, also known as transitional cell carcinoma (TCC), is the most common urological malignancy. It commonly spreads to the pelvic lymph nodes, lung, bone and liver and rarely metastasizes to the gastrointestinal tract. Only a few cases of duodenal obstruction secondary to metastatic TCC have been reported to date. Herein we report a case of a patient with a recently diagnosed right renal mass presenting with intractable nausea and vomiting secondary to duodenal obstruction from local metastasis of TCC.
Case Description/Methods: An 80-year female diagnosed with right renal mass on CT scan 2 months prior presented with nausea, vomiting, diarrhea and 15-pound weight loss. CT of the abdomen without contrast revealed an ill-defined renal mass with extension into the perinephric soft tissues and multiple low-attenuation lesions in the liver concerning for metastasis. The visualized portion of the small bowel on the CT was unremarkable. A liver biopsy of the mass revealed metastatic TCC with immunohistochemical stains positive for CK 7, CK 20 and GATA-3. Gastroenterology team was consulted for intractable nausea and vomiting. An upper endoscopy revealed duodenopathy with friable tissue and mottling in the second portion of the duodenum. A severe luminal stenosis prevented further endoscope advancement. Duodenal mucosa biopsies revealed findings consistent with TCC. Plans for palliative duodenal stent insertion to relieve the symptoms of the obstruction were deferred due acute clinical decline; the patient was transitioned to hospice care.
Discussion: Duodenal and rectal obstructions from urological malignancies are relatively uncommon and only a few cases of symptomatic metastatic TCC to the duodenum have been reported in literature. Malignant obstructions are not always identified on imaging and can be difficult to diagnose unless patients are symptomatic. In our case, persistent nausea and vomiting was attributed to generalized malaise from her known malignancy, and duodenal obstruction was only identified following endoscopy. Duodenal obstructions due to malignancies are generally treated with either surgical bypassing, colostomy, or

endoluminal stent placement. However, these interventions are only palliative, and the diagnosis confers a poor prognosis. We aim to provide further knowledge and clinical experience regarding duodenal obstruction secondary to TCC for early identification and management.

S3404

Duodenal Penetration: A Late Complication of Inferior Vena Cava Filter Placement

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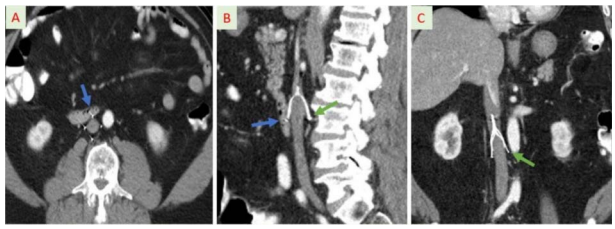
Introduction: Venous thromboembolism (VTE), including deep venous thrombosis (DVT) and pulmonary embolism (PE), with an annual incidence of 1 out 1000. The treatment of DVT includes anticoagulation or inferior vena cava (IVC) interruption via filter placement. However, IVC filter placement is associated with several perioperative and delayed complications including thrombosis of the access site (vena cava thrombosis), migration of the filters from their original position, vessel, and/or organ penetration.
Case Description/Methods: A 79-year-old male, with a past medical history of IVC filter placement for management of recurrent DVT and PE, presented to the ED with abdominal pain. He denied nausea, vomiting, or hematochezia. His vitals were stable and physical exam was significant for mild epigastric tenderness. Initial workup was unremarkable including lipase and abdominal ultrasound. A computed tomography (CT) of the abdomen and pelvis with contrast showed IVC filter perforation and extension into the duodenum (Figure A, B and C). Upper endoscopy was recommended but the patient did not want to proceed with invasive procedures. He was explained that his abdominal pain might be due to duodenal penetration/perforation by the IVC filter. The risks and benefits of surgical removal of IVC filter were explained to the patient. He opted for non-operative measurements. His pain resolved and he was discharged home. 2 years after the identification of strut perforation of the duodenum, the patient remains asymptomatic.
Discussion: Penetration, perforation, or malposition of IVC filters may be a clinically under-recognized complication of IVC filter placement. Symptomatic patients accounted for nearly 1/10th of all penetration. IVC filter penetration should be one of the differentials in patients with a history of IVC filter placement presenting with vague abdominal symptoms. CT scan and EGD should be considered for further evaluation. Although excellent outcomes with low complication rates have been reported in cases with surgical removal of IVC filters in symptomatic patients. Most cases only need a follow-up CT scan and EGD to assess for further complications. In our patient with IVC filter penetration through the duodenal wall, conservative management was successful.



[3404] **Figure 1.** Duodenal penetration of IVC filter (blue arrows), IVC struts out of vessel (green arrow).

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S3405

Duodenal Mass With Lymphadenopathy: A Case of Gastrointestinal Coccidioidomycosis

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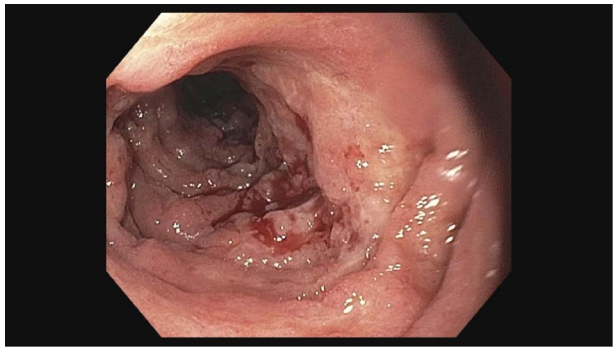
Introduction: Coccidioides is endemic to the SW region of the US. Clinical presentation of infection varies greatly with the majority of cases being asymptomatic. When symptomatic, Coccidioides infection usually results in a self-limited pulmonary illness. Disseminated disease accounts for 1% of cases and usually occurs in patients who are immunocompromised. GI infection with Coccidioides is exceedingly rare with less than 10 cases present in the literature. We present the case of disseminated coccidiomycosis that included the GI tract.
Case Description/Methods: 51 yo Caucasian male presented to the emergency room with a 1d history of lethargy and confusion. His past medical history included HTN, HLP, RnY gastric bypass, and previously treated Aspergillus pulmonary infection. VS on presentation were significant for fever of 104 F. His physical examination findings were largely unremarkable except for his neurologic assessment which was significant for decreased awareness and lethargy. There were no focal deficits. Lab work including CBC and CMP were unremarkable. CT scan of the brain demonstrated a subacute appearing right lentiform infarction. MRI was then undertaken and showed leptomeningeal findings most consistent with meningitis. He was started on empiric antibiotics for meningitis with the addition of acyclovir and amphotericin. LP was significant for a total protein of 154, glucose of 30, polys 12 (elevated) and elevated eosinophils (2). CSF gram stain and VDRL were negative. Coccidiomycosis IgG and IgM were both positive. CT A/P with contrast showed thickened appearance of distal duodenum with associated mesenteric and retroperitoneal lymphadenopathy. To further characterize the duodenal abnormality and evaluate cause of anemia EGD and Colonoscopy were undertaken. The EGD was unremarkable except for the presence of an ulcerated mass with a small amount of oozing blood in the second and third portion of the duodenum. Biopsies of the affected area demonstrated mixed inflammation with numerous intramucosal fungal spherules consistent with Coccidioides species. Colonoscopy was limited by poor preparation however the cecum was reached and no obstructing mass was identified. The patient was continued on amphotericin B with gradual overall improvement in his condition. He was transitioned to oral lifelong fluconazole. Repeat EGD will be undertaken in three months (Figure).
Discussion: Gastrointestinal infection with Coccidioides is exceedingly rare but should be considered in patients living in endemic regions.



[3405] **Figure 1.** Duodenum.

S3406

Enteropathy Associated T-cell Lymphoma Disguised as Crohn's Disease

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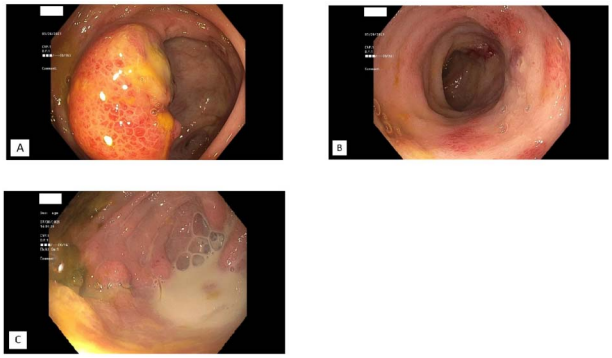
Introduction: Enteropathy associated T cell lymphoma (EATL) is a rare condition accounting for less than 5% of gastrointestinal lymphomas. This disease is difficult to diagnose in the early stages with most patients being diagnosed at stage IV. Presenting symptoms include abdominal pain, weight loss, fatigue, fevers, and night sweats. Given the overlap of symptomatology with Crohn's disease (CD), diagnosis of EATL can be delayed.
Case Description/Methods: A 56 year-old man arrived with fevers, chills, and diarrhea for 3 months. Exam was notable for mild abdominal tenderness. Labs showed a WBC of 43K, positive C diff toxin, elevated fecal calprotectin, elevated CRP to 23, and negative tissue transglutaminase. CT abdomen showed terminal ileitis and colitis. The patient was treated with oral vancomycin with improvement of his diarrhea. Six months later the patient was seen in clinic. He was again having significant diarrhea, abdominal pain, fevers, chills, night sweats, and a 50-pound weight loss. Colonoscopy was significant for deep ulcerations of the terminal ileum. Biopsies showed chronic ileitis with ulceration and focal collections of epithelioid histiocytes suggestive of granulomas. There was no evidence of dysplasia or metaplasia seen on biopsy. Following the biopsy results the patient was diagnosed with CD. One week later, he was admitted with weakness, fatigue, and worsening abdominal pain. CT enterography revealed an enteroenteric fistula. The patient underwent exploratory laparotomy with resection of the small intestine and end ileostomy. Pathology showed CD-30 positive T cell lymphoma indicating EATL. The background intestinal mucosa also revealed features suggestive of gluten-sensitive enteropathy with villous atrophy and increased intraepithelial lymphocytes. Ultimately this patient was started on chemotherapy but died of complications related to chemotherapy, intra-abdominal abscesses, and renal failure less than 2 months following diagnosis of EATL (Figure).
Discussion: This case illustrates the difficulty in diagnosing EATL. Patient initially was diagnosed with CD given the clinical presentation and endoscopic findings. It was not until the patient underwent small bowel resection due to enteroenteric fistula that the diagnosis of EATL caused by gluten-sensitive enteropathy was made by pathology. Negative celiac serologies made this case a more challenging one. Although rare, EATL must be considered in the differential for patients presenting with similar symptomatology as patients suspected to have CD.



[3406] **Figure 1.** A, Ulcerations at the ileocecal valve (B) Ulcerations at the terminal ileum (C) Frank pus in the small bowel.

S3407

Ethacrynic Acid-Associated Seronegative Enteropathy

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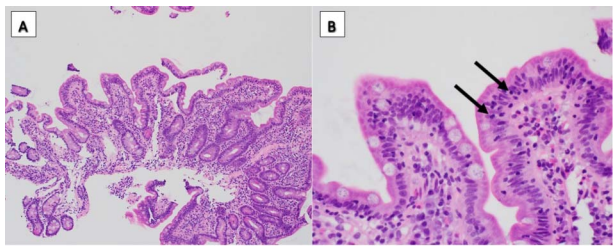
Introduction: Seronegative enteropathy is defined as negative serological celiac disease (CeD) testing, ie, tissue transglutaminase (tTG), deaminated gliadin peptide (DGP), anti-endomysial antibody (EMA), in patients with villous atrophy on small bowel biopsies. Many common medications have been implicated in causing a reversible enteropathy, mainly olmesartan. We describe the first case of a patient who developed severe diarrhea and subsequent diagnosis of seronegative enteropathy following initiation of ethacrynic acid.
Case Description/Methods: A 78-year-old male with a history of hypertension, hyperlipidemia, and chronic kidney disease presented for outpatient gastroenterology consultation with a complaint of 16 days of moderate volume, watery stools occurring 3-5 times per day. He was started on ethacrynic acid by his nephrologist for volume overload approximately 6 weeks prior to presentation. He was also taking magnesium, vitamin E and vitamin C supplements, which he stopped, without improvement in diarrhea. He continued to take losartan for high blood pressure, which he had been on for many years. Infectious and chronic diarrhea workup returned negative except for an elevated calprotectin level (753 ug/g; normal $<$ 49 ug/g). Colonoscopy and ileoscopy were normal, with negative random biopsies for microscopic colitis. Upper endoscopy was normal, however duodenal biopsies showed mild to moderate villous blunting (Figure A) and increased intraepithelial lymphocytes (Figure B), consistent with a Marsh 3a-3b lesion. CeD serological testing were normal with subsequently absent HLA DQ2 and DQ8. After stopping ethacrynic acid, the patient's diarrhea resolved within 2 days and repeat duodenal biopsies 5 months later showed resolution of his enteropathy.
Discussion: Seronegative enteropathy is a clinical challenge as many conditions and medications may cause severe small intestinal damage. Seronegative celiac disease was excluded in this patient as his celiac genes were absent and his enteropathy resolved without a gluten-free diet. This is the first case to our knowledge of seronegative enteropathy associated with ethacrynic acid, with clinical and histological remission upon drug discontinuation. It is important for clinicians to recognize each potential drug associated with seronegative enteropathy, in order to properly diagnose and manage an easily reversible condition. Incorrect interpretation of the duodenal biopsies could have led to a misdiagnosis of CeD and an unnecessary gluten free diet.



[3407] **Figure 1.** A, Duodenal biopsies showing villous blunting (H&E stain, 100× magnification); (B) Duodenal biopsies with arrows pointing to increased intraepithelial lymphocytes (H&E stain, 400× magnification).

S3408

Excellent Treatment Response of an Aggressive Primary Duodenal Neuroendocrine Carcinoma to Oxaliplatin-Based Chemotherapy

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Introduction: Primary duodenal neuroendocrine carcinoma (NEC) is a rare and highly aggressive malignancy with very poor prognosis. There is no established treatment due to its rarity. Treatment regimens used for small cell lung cancer (SCLC) are used to treat neuroendocrine carcinoma, due to its clinical and histopathological similarities. Therapeutic strategies are poorly understood and not well defined. There is no standardization of therapy even for limited disease and usually multimodal treatment approach is used. Etoposide based treatment regimens have been used mostly in advanced stages. We herein report a case of an aggressive primary duodenal neuroendocrine tumor with excellent response to oxaliplatin-based chemotherapy regimen.
Case Description/Methods: We describe a case of a 72-year-old caucasian male who presented to emergency room with abdominal bloating and constipation. He was found to have peritoneal carcinomatosis and marked hydronephrosis in computed tomography (CT) of the chest/abdomen/pelvis. He was further evaluated by urology, oncology, and gastroenterology team. He had paracentesis with removal of 4 L of ascitic fluid. He underwent esophagogastroduodenoscopy, endoscopic ultrasound, and a colonoscopy for further assessment. He was found to have a large mass in the duodenum which upon biopsy was consistent with grade 4 poorly differentiated neuroendocrine carcinoma. Positron emission tomography dotadate scan, peritoneal biopsy and peritoneal fluid cytology further confirmed metastatic neuroendocrine carcinoma. His tumor markers showed elevated Ca 19-9 and chromogranin levels at presentation. Due to his poor performance status and concerns for intolerability to etoposide, he was started on FOLFOX chemotherapy. He completed 12 cycles of chemotherapy with near complete resolution of his disease as evidence by his positron emission tomography dotadate scan and improvements in his tumor markers. Due to excellent response and disease remission, he is currently on xeloda with omission of oxaliplatin.
Discussion: Primary duodenal neuroendocrine carcinoma is a relatively rare malignancy with reported incidence of 0.4-2% among all duodenal malignancies. The prognosis is dismal due to presentation as advanced stage at diagnosis. Oxaliplatin based regiments have been shown to have promising anti-tumor activity in gastrointestinal neuroendocrine cancers, however the available data in duodenal gastrointestinal neuroendocrine cancers are very limited.

S3409

Features of Recurrent Mechanical Obstruction With Nonobstructive Duodenal Neuroendocrine Tumor

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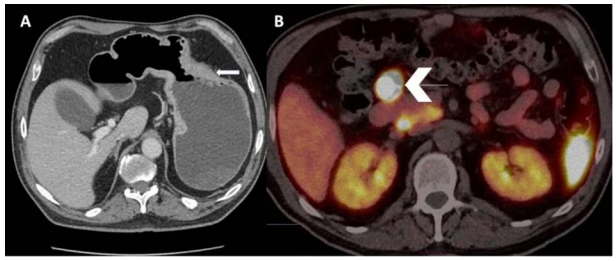
Introduction: Neuroendocrine tumors (NET) are a rare cause of bowel obstruction which is usually mechanical in nature. We present an unusual case of bowel obstruction in a patient with NET where no mechanical obstruction was found despite extensive work up.
Case Description/Methods: A 56-year-old male was admitted after 9-10 admissions to another facility with bowel obstruction and 50-pound weight loss over 10 months. His symptoms included abdominal pain, distention, and vomiting. He had no prior abdominal surgeries. Previous CT abdomen revealed small bowel obstruction with transition point in the terminal ileum. Current CT showed gastric outlet obstruction (Figure A). Formerly he was managed nonoperatively with nasogastric tube (NGT) decompression. Prior esophagogastroduodenoscopy and colonoscopy did not reveal an obstruction. NGT output was 2.5 L. Push enteroscopy, video capsule endoscopy did not reveal any mechanical obstruction. MR enterography showed two right mesenteric nodules, larger of which was 2.1 x 2.3 cm in size. These were moderately FDG avid on PET-CT. Endoscopic ultrasound biopsy revealed a well differentiated NET. The lesions were intensely somatostatin receptor positive on Gallium-68 DOTATOC PETCT (Figure 1B). Pancreatic polypeptide was 2145 pg/mL (0-435), chromogranin A 1458 ng/ml (0-103), serotonin 278 ng/mL (50-200), and gastrin 208 ng/mL (0-100). He underwent partial duodenal resection with primary repair and peripancreatic lymph node resection. No obstructive pathology was found intraoperatively. Histopathology confirmed well differentiated NET with metastatic peripancreatic nodes. He has had no admissions over the past 3 months and has gained 33 pounds.
Discussion: NET are rare tumors and use of appropriate diagnostic modalities are key to timely diagnosis. Despite improvement and availability of diagnostic modalities, most patients have metastatic disease at diagnosis. NET presenting with bowel obstruction usually have an obstructing tumor. We wish to bring to light the unusual presentation of NET with mechanical obstruction on imaging but extensive negative work up for an actual obstructing lesion. It is possible that mesenteric inflammation and edema secondary to NET may have caused recurrent obstruction in this case. NET can present with mechanical obstruction and are often missed on conventional imaging. Gallium-68 DOTATOC PETCT may be a reasonable investigation in a patient with unexplained recurrent gastrointestinal obstruction.



[3409] **Figure 1.** A, CT abdomen with dilated stomach and duodenum and decompressed distal small bowel suggestive of gastric outlet obstruction (arrow) (B) Ga-68 DOTATOC PET/CT scan showing somatostatin receptor positive peripancreatic nodules consistent with metastatic neuroendocrine tumor (chevron).

S3410

Foreign Body Reaction Following Use of Submucosal Lifting Agent for Endoscopic Resections

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Introduction: Submucosal injection of lifting solutions is essential for both endoscopic mucosal resection (EMR) and endoscopic submucosal dissection (ESD). In mucosal resections, a lifting solution is injected into the submucosal layer to separate the submucosa from muscularis propria to facilitate resection of the targeted lesion. ORISE^{TM} gel (Boston Scientific) is one of the lifting solutions that was recently approved by the Food and Drug Administration in 2018. While ORISE^{TM} gel has become the popular solution, there is a concern that it could affect histologic findings. Here we report a foreign-body reaction associated with ORISE^{TM} gel injection.
Case Description/Methods: A 72-year-old male with a history of Barrett's esophagus was first found to have duodenal adenoma in the 3rd portion in 2017. 4 years later the patient was found to have polypoid tissue in the 2nd portion of the duodenum. Endoscopic mucosal resection (EMR) was attempted and the tissue was lifted by submucosal injection of 4 mL ORISE^{TM} gel, however, the procedure was aborted because the center of the polyp was fixed and could not be lifted. Biopsies were taken with cold forceps which showed duodenal adenoma without evidence of dysplasia. 65 days later, the patient underwent repeat esophagogastroduodenoscopy (EGD) to remove the lesion. ORISE^{TM} gel injection was attempted, however, the lesion could not be lifted due to severe fibrosis thus hybrid EMR was used to excise the lesion and 25 mm of mucosa was resected. Pathologic examination revealed fragments of adenoma. In addition, submucosa showed foreign material with associated foreign giant cell reaction, consistent with the site prior to the procedure.
Discussion: In this case, we report a foreign giant cell reaction followed by ORISE^{TM} Gel use. The foreign body cell reaction occurs when particles are large (sizes $>$ 10 μm) enough so that macrophages induce the foreign body giant cell formation to degrade the material. These changes can be seen as early as 3 weeks later and also seen as late as 5 months later. One of the biggest advantages of ORISE^{TM} gel is that it is a long-lasting mucosal lift but this may have backfired. Given the severe foreign body reaction into the submucosal layer, the lesion needs to be completely removed after injection of ORISE. Also, longer-term observation is needed to ascertain whether histologic changes affect subsequent mucosal resection.

S3411

Extensive Hepatic Lesions From a Primary Duodenal Neuroendocrine Carcinoma in a Young Male

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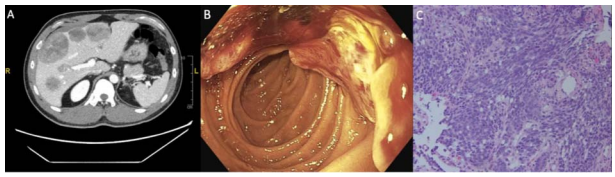
Introduction: Neuroendocrine neoplasms (NEMs) are relatively rare, accounting for 0.5% of newly diagnosed malignancies. NEMs can be found throughout the body with two-thirds arising in the GI tract and incidence increasing among duodenal NEMs. These malignancies have a female predominance and generally occur in the 6th decade of life. This is a case of advanced metastatic disease from an unexpected duodenal neuroendocrine carcinoma in a healthy young male.
Case Description/Methods: A 29-year-old male, with no PMH, acutely developed sharp RUQ abdominal pain. Prior to this, he was in his usual state of health. In the ED, the patient was hemodynamically stable with tenderness to palpation in the RUQ. Initial labs showed WBC 12.10 × 10^3/uL, Hgb 11.7 g/L, AST 55 U/L, ALT 173 U/L, ALP 127 U/L, and TBIL 0.8 mg/dL. CT A/P showed multiple well-circumscribed liver lesions, largest measuring 8.6 × 7.8 cm, with portal venous invasion (Figure A). CA 19-9 and CEA were negative. There was elevation of AFP 1,577 ng/mL and LDH 817 U/L, but negative hCG. A1AT, ANA, AMA, ASMA, ALKM, and ceruloplasmin were negative. Liver biopsy showed a poorly-differentiated malignant neoplasm of unknown primary. EGD was performed revealing a fungating mass in the second portion of the duodenum (Figure B). Pathology favored a high-grade poorly-differentiated neuroendocrine carcinoma (Figure C). He was discharged with palliative cisplatin/irinotecan initiation.
Discussion: Gastrointestinal NEMs have been increasing in prevalence due to improved awareness. When carcinoid symptoms are absent, detection is dependent on imaging and endoscopy after the patient presents with complications from metastasis or mass effect. Following biopsy, GI NEMs are classified by WHO 2010 criteria into 3 groups based on mitotic count and Ki-67 index. Group 3 tumors are classified as poorly-differentiated neuroendocrine carcinomas with Ki-67 >20% with higher rates of angioinvasion and metastatic potential. Lymph node metastasis occurs in 60% of duodenal NETs, while liver mets occur in < 10%. In our case, the patient's Ki-67% index was >90%, characterizing it as a Group 3 NEM. Characterization is important given the impact on prognosis and treatment. Group 3 NEMs have the overall worst prognosis. Since >90% of duodenal NEMs do not cause a clinical syndrome and are not associated with metastatic disease, surgical or endoscopic resection is preferred. However, if there is metastatic disease or high-grade histologic features, chemotherapy is the mainstay of treatment.



[3411] **Figure 1.** A, CT A/P with multiple large heterogeneously enhancing liver lesions concerning for metastatic malignancy of unknown primary (B) Large fungating mass located in the second portion of the duodenum (C) Duodenal mass, H&E stain, 20×, high-grade poorly differentiated epithelial malignancy composed of nested cells with indistinct nucleoli, moderate eosinophilic cytoplasm, and brisk mitosis.

S3412

Functional Medicine Approach to Care Improves Quality of Life and Blood Alcohol Levels in a Patient With Autobrewery Syndrome

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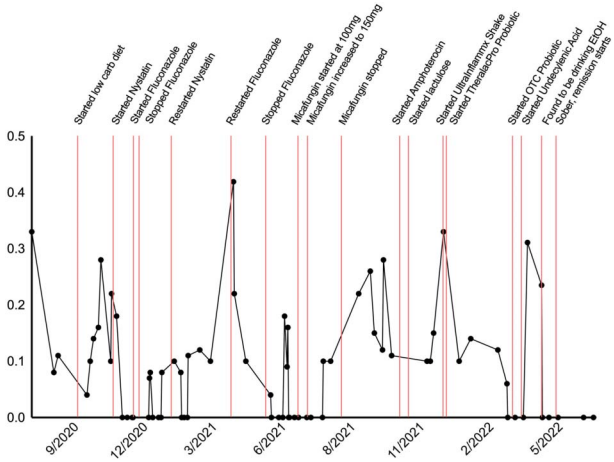
Introduction: Autobrewery syndrome, or gut fermentation syndrome (GFS), is a rare condition characterized by elevated blood alcohol concentration (BAC) in the absence of alcohol consumption. Treatment involves antifungals and a low carb diet, which are often ineffective. In this case, we describe a patient with GFS who failed traditional therapy, was unable to complete activities of daily living due to high BAC, and responded well to a functional medicine approach to care with sustained improvement in BAC.
Case Description/Methods: In 7/2020, a 58-year-old man with T2D, obesity, HTN and gout developed recurrent episodes of dysarthria, behavioral changes, and somnolence. On multiple occasions in the ER, his BAC level was found to be elevated. The patient and family denied alcohol use. He was ultimately diagnosed with GFS. He self-initiated a strict low-carb diet with initial success in controlling his BAC from 11/2020 to 12/2020. From 10/2020 to 10/2021, he underwent multiple courses of antifungal medications (nystatin, fluconazole, micafungin, and oral amphotericin). The longest period of remission (BAC 0) was 41 days while on micafungin. After 41 days on micafungin and a restricted diet, his BAC increased. Over this time, he lost 80 lbs. In 11/2021, he established with a functional medicine GI and dietitian. Stool metabolomics showed no obvious abnormalities. The 5 R protocol (remove, replace, repair, reinoculate, rebalance) was introduced. He started with an aggressive bowel regimen with prucalopride and lactulose. He completed 15 days of high-potency Bacillus subtilis probiotic, followed by a transition to a more diverse probiotic. He added daily intake of a medical food to his restricted diet. New foods were then introduced, one at a time, in the evening, with subsequent monitoring of BAC. In 2/2022, he started Undecylenic acid (10-Undecenoic Acid) as fatty acid support. Lifestyle modifications were added to emphasize more exercise, daily outdoor walks, optimal sleep, and decreasing stress. He did well until BAC levels spiked in 3/2022 due to consumption of alcohol, triggered by cravings that developed when the gut production of alcohol decreased. After alcohol cessation, BAC levels have remained undetectable for 60+ days with continued diet modifications and supplements (Figure).
Discussion: GFS is difficult to treat with minimal traditional treatment options. This case is a patient with GFS that failed all medical options and is now thriving after a functional medicine approach to care.



[3412] **Figure 1.** BAC levels continued to spike with medical therapies and have sustained improvement with functional medicine care.

S3413

Falsely Positive Anti-Gliadin Antibodies in a Non-Celiac Patient With Multiple Sclerosis

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Introduction: Celiac disease (CD) is a small bowel autoimmune enteropathy mediated by antibodies against dietary gluten. We present a case of a patient with multiple sclerosis with persistently positive IgA antibodies against deamidated gliadin peptide (DGP) who was ultimately demonstrated not to have CD after decades of gluten avoidance.
Case Description/Methods: A 64-year-old male with a history of multiple sclerosis (MS) and a family history of celiac disease in his mother presented to clinic to clarify his CD diagnosis. On prior testing he had positive DGP IgA (39 U) and weakly positive tissue transglutaminase (tTG) IgG serologies (8 U/mL, upper limit of normal 5 U/mL). However, prior esophagogastroduodenoscopy (EGD) was reportedly unremarkable. Based on serologies, he had been told he had CD and followed a gluten-free diet for years. On presentation, he denied bloating, diarrhea, abdominal pain, or weight changes. Labs were notable for an elevated DGP IgA level of 38 U with normal DGP IgG, tTG IgA, and tTG IgG serologies. On EGD while on a gluten free diet, the duodenum appeared normal, and pathology showed normal duodenal mucosa without villous atrophy (VA). Studies were repeated after several months of dietary gluten reintroduction. DGP IgA remained persistently elevated at 37 U, DGP IgG and tTG IgA remained within normal limits, and tTG IgG was borderline at 6 U/mL, of questionable significance. Genotyping was positive for HLA-DQ8 but negative for HLA-DQ2. Repeat EGD after gluten reintroduction demonstrated a normal-appearing duodenum without evidence of VA on pathology. The patient was informed that he did not have celiac disease.
Discussion: Serologies in CD can be impacted by autoimmune disease. This patient had MS-related immunogenicity to DGP in the absence of CD. Several studies have noted that patients with MS without CD often have higher titers of DGP IgA antibodies, which may be due to antibody cross reactivity or even increased gut permeability to DGP. When serology is discordant, as in this case, duodenal biopsy should be performed, ideally after a gluten challenge. If repeat biopsy and tTG IgA serologies remain negative after gluten challenge, CD is highly unlikely. However, these patients should be closely followed for development of symptoms of CD, as latent CD is also possible.

S3414

Goblet Cell Adenocarcinoma Presenting as Appendicitis

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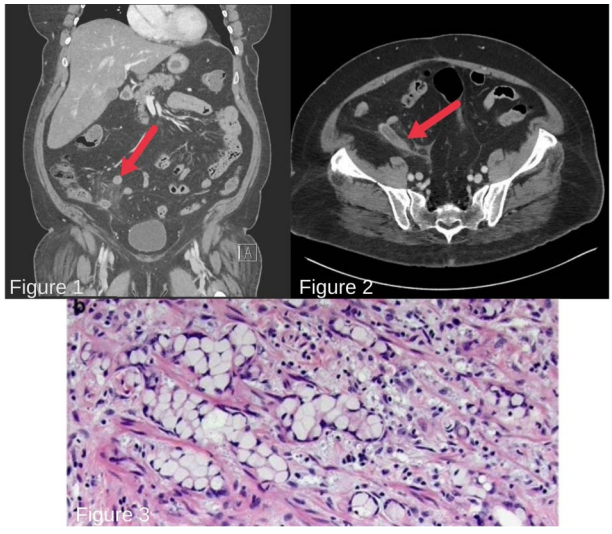
[1]FMH College of Medicine & Dentistry, Lahore, Punjab, Pakistan; [2]AdventHealth Orlando, Orlando, FL; [3]Methodist Dallas Medical Center, Dallas, TX; [4]Allama Iqbal Medical College, Sarnia, ON, Canada; [5]Allama Iqbal Medical College, Mississauga, ON, Canada.

Introduction: Goblet Cell Adenocarcinomas (GCAs) are a very rare subtype of appendiceal tumors, with an estimated incidence of 1 per 2 million people. They are not only rare but also unique in terms of their origin. Whereas most cancers arise from one cell line, goblet cell adenocarcinomas are a hybrid of neuroendocrine cells and epithelial cells. The aim of this abstract is to provide an overview of this unique clinical entity.
Case Description/Methods: We present a case of a 62-year-old female with past medical history of hypertension, diabetes mellitus and hyperlipidemia who presented to the ED with a one day history of right lower quadrant abdominal pain that had migrated from the periumbilical area, associated with non-bloody vomiting. On arrival, she was stable and her physical examination was significant for tenderness in the right lower quadrant and a positive Rovsing and obturator sign. Labs were remarkable for leukocytosis with left shift. CT scan of the abdomen was done which revealed an inflamed appendix and subsequently patient was taken for a laparoscopic appendectomy. Pathology of the surgical specimen revealed a well differentiated T1NxMx GCA with negative margins and no lymphovascular or perineural invasion. A decision was made to proceed with a right hemicolectomy for the purpose of staging and treatment. Pathology sent after right hemicolectomy revealed no residual GCA with 21 lymph nodes negative for metastasis and negative surgical margins (Figure).
Discussion: First described in 1969, GCAs mostly occur in the 5th and 6th decade of life with equal prevalence in both genders. GCAs most commonly present as acute appendicitis but can also present with abdominal pain associated with abdominal mass and weight loss. They can metastasize to the peritoneum, omentum, abdominal wall, and ovaries. Majority of GCAs are diagnosed incidentally on pathological examination of inflamed appendix after appendectomy. The mainstay of treatment is surgical resection with the addition of adjuvant chemotherapy for node-positive (stage III) disease or higher. Due to the rarity of this condition, there is lack of consensus regarding the extent of surgical resection. For stage I disease appendectomy alone maybe sufficient, but higher stages may require hemicolectomy. Prognosis depends on the grade and stage at diagnosis, with one retrospective analysis reporting 5-year survival rates of 100%, 76%, 22%, and 14% for stages I, II, III, and IV, respectively.



[3414] **Figure 1.** 1 and 2 - CT scan of the abdomen showing distended appendix with appendiceal wall thickening and hyperemia, as well as trace periappendiceal edema. 3 - Histologically conventional Goblet cell adenocarcinoma has a "crypt cell pattern" characterized by small round clusters of goblet cells in acinar configuration, resembling colonic crypts. [Pathology slide from Mod Pathol. 2016;29(10):1243-1253. DOI: 10.1038/modpathol.2016.105]).

S3415

Gastroenterological Manifestations in a Patient With Common Variable Immunodeficiency

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Introduction: Gastrointestinal manifestation of common variable immunodeficiency (CVID) can present with protein-losing enteropathy (PLE). A wide spectrum of liver injuries can occur with CVID, including nodular regenerative hyperplasia (NRH).
Case Description/Methods: 41 y.o. female with PMH of CVID on IgG, chronically elevated LFTs, and nutritional deficiencies. Patient presented with 8-year history of diarrhea, bloating, weight loss, abdominal pain, hepatic encephalopathy (HE), new ascites, and shock. Previously was diagnosed with celiac disease and has strictly followed a gluten-free diet with no improvement. EGD on the current presentation

showed visible changes in duodenal mucosa including villous flattening. Biopsies this time revealed villous flattening and absence of plasma cells consistent with CVID enteropathy. Liver biopsy 5 years prior to admission showed no liver disease. Serologic workups, including anti-gp210 and anti-sp100, were negative. Biopsy showed nodular regenerative hyperplasia and non-cirrhotic portal hypertension, which was confirmed by reticulin stain. Patient was started on budesonide 9 mg daily for treatment of PLE, and marked improvement of diarrhea was noted. Non-cirrhotic portal hypertension was managed with furosemide and spironolactone. HE was managed with oral lactulose. The patient was started on enteral and parenteral supplements.
Discussion: Duodenal villous atrophy which is a diagnostic criterion for celiac disease can be seen in multiple other conditions including immune disorders such as CVID. In CVID patients with gastrointestinal symptoms, up to 80% of biopsies from different sites in the GI tract will show histological abnormalities. Given their common presentation, celiac disease can be initially diagnosed. The absence of plasma cells and apoptosis presence in duodenal biopsy can suggest PLE diagnosis. Serological studies are not reliable for celiac disease diagnosis due to deficiency of immunoglobulin production in CVID. Steroids can be used for the treatment of PLE in CVID. Hepatic manifestations in CVID are extremely varied and often misdiagnosed given they can mimic other diseases. NRH is a potentially fatal complication of CVID. Recognition of this complication allowed for symptom resolution within two days of starting lactulose therapy. NRH in CVID represents an autoimmune-like liver disease that might be driven by a local vasculopathy, and thus severe cases can warrant a trial of immunosuppression.

S3416

Gastrointestinal Stromal Tumor Presenting as Small Bowel Obstruction in the Setting of Neurofibromatosis Type 1

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Introduction: Gastrointestinal stromal tumors (GISTs) are mesenchymal masses that occur throughout the GI tract, predominantly in the stomach. GISTs typically present with symptoms of early satiety, vomiting, and signs of GI bleeding. They are normally sporadic but can be associated with certain hereditary conditions including Neurofibromatosis type 1 (NF-1). Here we describe the case of a patient presenting with small bowel obstruction and perforation secondary to GIST related inflammation.
Case Description/Methods: A 46 year old man with a history of NF-1 and chronic constipation presented with worsening abdominal pain and distention for several days. He denied nausea or emesis but endorsed sharp, persistent abdominal pain. Physical examination showed diffuse abdominal distention and tenderness without guarding or rebound tenderness, as well as diffuse cutaneous neurofibromas. Serologic studies included normal WBC count and lactic acid level. CT abdomen/pelvis with IV contrast revealed small bowel obstruction with a transition point in the ileum and diffuse lymphoid tissue thickening along the mesenteric roots. The patient failed to improve with conservative management via NG tube. Exploratory laparotomy was performed, revealing a thickened hemorrhagic mesentery in the central portions of the small bowel and also a large adhesive band leading to an internal hernia. A focal perforation was noted in the mid-jejunum, and partial small bowel resection was performed. Pathology report confirmed the presence of two tumors measuring 0.8 cm and 0.2 cm within the jejunal wall at the junction of the mesentery, associated with serositis, a mural abscess and focal necrosis, and fibrinous adhesions. Tumor cells were positive for S100 and SOX10, as well as CD117 and DOG1, consistent with GIST. The patient recovered uneventfully, and subsequent video capsule enteroscopy revealed no findings of additional small bowel GISTs.
Discussion: The most common presentation of small bowel GISTs is hemorrhage and anemia. Rarely, GISTs may present with small bowel obstruction. With sporadic GISTs the obstruction is typically associated with a large tumor causing mechanical obstruction or intussusception. However, NF-1 patients may have GIST tumors which are small and multifocal, causing intense localized inflammatory changes which can result in bowel obstruction. Thus it is an important diagnosis to consider in patients with new onset small bowel obstruction, especially in patients with predisposing conditions to GIST such as NF-1.

S3417

Hyperlipasemia Due to Duodenal Obstruction Secondary to Gastrostomy Tube Migration

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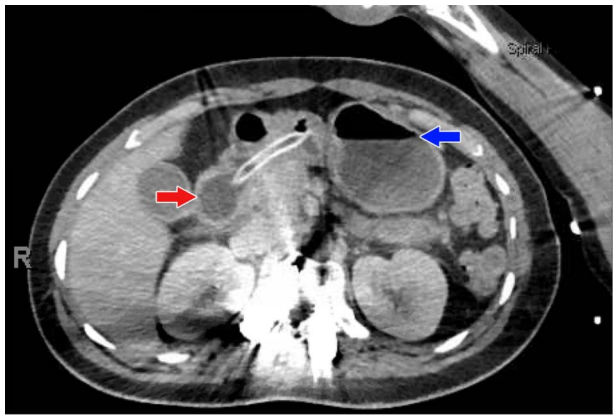
Introduction: Elevated lipase levels of three or more times the upper limit of normal along with abdominal pain and correlated findings on CT imaging can often diagnose a case of acute pancreatitis. Although uncommon, there are alternative causes of hyperlipasemia not related to acute pancreatitis that clinicians should be aware of. Here we present a case of significantly elevated lipase secondary to duodenal obstruction caused by gastrostomy tube migration.
Case Description/Methods: A 30-year-old nonverbal woman with a past medical history of cerebral palsy, refractory grand mal seizures, and chronic (fluoroscopically placed) gastrostomy tube (last changed about one month prior) presented to the hospital for breakthrough seizures, non-bilious vomiting of tube feeds, and abdominal pain. She arrived afebrile with a blood pressure of 165/85 mmHg and otherwise normal vitals. On examination, the patient was nonverbal and appeared uncomfortable with a moderately tender and distended abdomen. The external portion of her gastrostomy tube did not have signs of surrounding infection. Admission laboratory studies were remarkable for a white blood cell count of 13.3 k/ul and lipase of 8216 IU/L. Abdominal CT (Figure) showed a normal pancreas, but noted air fluid levels within the stomach as well as the balloon of the percutaneous feeding tube inflated within the second portion of the duodenum, concerning for obstruction. Patient was treated with a nasogastric tube to allow for decompression and the Interventional Radiology service repositioned the gastrostomy tube back into the stomach. After adjustment, the gastrostomy tube placement was confirmed and feeds were re-started. Patient was asymptomatic and tolerating her tube feeds without issue in less than 48 hours from initial presentation.
Discussion: While this case initially appeared to be a straightforward diagnosis of acute pancreatitis, the imaging proved otherwise. Moreover, the patient's rapid clinical improvement after alleviating the obstruction helped support the alternative diagnosis. It is likely that the obstruction inhibited forward flow of the pancreatic enzymes and instead increased diffusion into the bloodstream causing elevated lipase levels. Clinicians should be aware of the possibility of small bowel obstruction as a cause of hyperlipasemia.



[3417] **Figure 1.** CT abdomen showing percutaneous feeding tube with balloon inflated in the second portion of the duodenum (red arrow) and mild fluid level within the stomach (blue arrow).

S3418

IgM Multiple Myeloma Presenting With Diarrhea

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Introduction: IgM Multiple Myeloma (MM) is a rare subtype of MM consisting of < 1% cases of MM. It is distinguished from Waldenstrom Macroglobinemia, which also produces IgM, by the absence of somatic mutation MYD88. We present a patient with a chief complaint of diarrhea which unknowingly led to his hematological diagnosis.

Case Description/Methods: A 64 year old male with RA-SLE overlap syndrome on steroids, and recent COVID19 pneumonia, had presented with 5 episodes of watery diarrhea every day and 40 lb weight loss within 2 months. CT revealed small bowel enteritis and stool studies, including C. diff, cultures, ova and parasites were negative. Diarrhea persisted despite antibiotics, therefore an EGD and Colonoscopy were performed which showed duodenal lymphangiectasia and a normal colon. Duodenal biopsy revealed eosinophilic deposits in the villous lamina propria which stained for IgM and stained negative under congo red ruling out amyloidosis. SPEP and a bone marrow biopsy revealed monoclonal IgM spikes and plasma cells in the bone marrow suggesting MM along with a co-existing population of CLL. Next-generation sequencing was negative for MYD88, supporting IgM MM instead of Waldenstrom. He developed a protein-losing enteropathy with dramatic hypoalbuminemia (albumin 0.9) and lower extremity edema and DVTs. He was started on chemotherapy and frequent albumin infusions. His diarrhea completely resolved, however not in time, as his other medical comorbidities lagged behind and he developed anasarca and continued to deteriorate.
Discussion: Plasma cell dyscrasias such as IgM MM or more commonly Waldenstrom have rarely been reported to cause GI symptoms. GI involvement can include direct GI infiltration of plasma cells, IgM deposition, or the finding of a plasmacytoma. It has been speculated that IgM deposits can lead to interstitial viscosity and obstructive lymphangiectasia leading to diarrhea and a protein-losing enteropathy as in our patient. Protein loss has led him to have hypoalbuminemia and possibly loss of antithrombotic proteins that have caused DVTs. Few case reports have suggested that treating the underlying cause with chemotherapy stops diarrhea entirely. Although our patient's diarrhea ceased, we believe that it was not in time for him to entirely recover from the later complications of the disease. We hope that this case can help clinicians to attempt prompt treatment of patients when they find GI specimens showing IgM deposits and they suspect a plasma cell dyscrasia.

S3419

Ileocolitis Associated With the Checkpoint Inhibitor Palbociclib (PAL)

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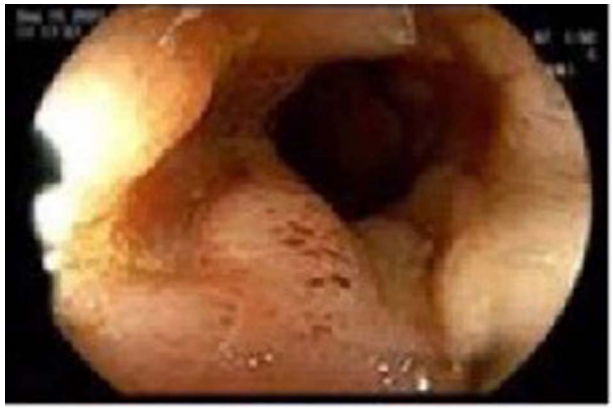
Introduction: Cancer treatments are continuously being updated to optimize survival. Newer checkpoint inhibitors (CDK4/6 inhibitors) have demonstrated promising responses in ER-positive breast cancers. We report a rare side effect of ileocolitis with Palbociclib (PAL) treatment.
Case Description/Methods: A 30-year-old female with biopsy confirmed stage IV ER+, PR+, HER2− ductal breast carcinoma with metastasis to liver and bone presented with abdominal pain, diarrhea, and hematochezia 2 months into therapy with letrozole and PAL. Subsequent colonoscopy 4 months after onset of symptoms showed diffuse colitis with congestion, erythema, and shallow ulceration scattered through the whole colon, and several superficial aphthous ulcerations in the ileum. Stool for C. difficile by PCR was negative and biopsies revealed nonspecific ileitis and colitis. There was concern for IBD vs drug induced enteritis/colitis. PAL was held and the patient was started on prednisone taper with resolution of symptoms after 1 month. Given concern for disease progression, PAL was restarted 1 month later with return of symptoms and hospital admission. Laboratory studies showed WBC 1.84 TH/uL, Hgb 7.6 g/dL, plt 24 TH/uL, LDH 4211 U/L, ALT 450 U/L, AST 221 U/L, alk phos 223 U/L, total bilirubin 1.3 mg/dL, ESR 49 mm/hr and CRP 45 mg/L. Infection was excluded via stool studies including C. difficile PCR and blood culture. Colonoscopy was performed and showed erythema, friability, congestion, erosions, and ulceration in the TI compatible with ileitis (Figure) as well as patchy erythema, edema, erosions, and ulcers throughout the colon with skip lesions and sparing of the rectum. Biopsies were reported as normal TI and colon. PAL was discontinued indefinitely and the patient had complete resolution of symptoms within weeks.
Discussion: CDK4/6 inhibitors are new first-line treatment for metastatic HR-positive breast cancer. There are no prior reports of PAL induced enterocolitis without concomitant radiation. In our patient, symptoms could not be easily distinguished from infectious colitis or IBD clinically. Ileal and colonic biopsies showed nonspecific ileitis and colitis or were reported as normal. As CDK4/6 inhibitors become used more as the first-line treatment of metastatic HR-positive breast cancer, additional evidence of GI related toxicities may emerge. Checkpoint inhibitor induced ileitis/colitis should be considered in a patient with new onset hematochezia and diarrhea after initiation of these agents.



[3419] **Figure 1.** Erythema, friability, congestion, erosions, and ulceration in the terminal ileum.

S3420

Hypocupremia Presenting 14 Years After Roux-en-Y Gastric Bypass

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Introduction: Copper deficiency is a well-established, but under recognized sequela of bariatric surgery. We present a case of acquired copper deficiency in a patient, 14 years after Roux-en-Y gastric bypass.
Case Description/Methods: A 39-year-old female with history of Roux-en-Y gastric bypass (RYGB) 14 years ago and cholecystectomy 3 years ago, was referred to the gastroenterology clinic for epigastric pain with nausea, vomiting and diarrhea ongoing for a few months. Her review of systems was also positive for fatigue, lack of energy and bilateral hand numbness. Initial work up showed a white blood cell count of 2.5 k/m^3 with low neutrophil count of 28%, macrocytosis with MCV of 103.8 fL, cobalamin of 239 pg/ml, folate of 12.7 ng/ml, low copper of 8 μg/dL, and elevated zinc of 126 μg/dL. She was started on copper supplementation. A bidirectional endoscopy to evaluate for abdominal pain and diarrhea was also performed that showed a small anastomotic ulcer but was otherwise normal. Within 4 weeks of copper supplementation, her copper level improved to 92 μg/dL. The patient also reported resolution of weakness and hand numbness. Her leukopenia, neutropenia and macrocytosis also normalized as shown in Table. Her diarrhea resolved with cholestyramine.
Discussion: Micronutrient deficiencies are a well-known complication of bariatric surgery. These occur due to a combination of decreased intake as well as poor absorption. Acquired copper deficiency can be seen in 10-20% of patients after RYGB and can lead to hematological and neurological sequelae if not diagnosed and managed in a timely manner. American Society for Metabolic & Bariatric Surgery recommend copper supplementation for all post RYGB patients with 2 mg/day along with annual screening for copper deficiency even in the absence of symptoms. Our patient also had zinc overload, which is an additional risk factor for copper deficiency. Zinc competes with copper for absorption, and therefore can lead to decreased intestinal uptake. Despite being a well-recognized mineral deficiency post bariatric surgery, copper deficiency remains underdiagnosed as it has a variable clinical presentation and is often confounded by other micronutrient deficiencies, especially cobalamin. While the hematological abnormalities resolve within 4-8 weeks of repletion, the neurological recovery is variable and can sometimes be irreversible, making prompt recognition and management of copper deficiency essential.

Table 1. Comparison of Lab Values Pre and Post Copper Supplementation. These labs show the initial severe copper deficiency (March 2021), which improved dramatically with copper supplementation. With adequate copper levels, the leukopenia, neutropenia, macrocytosis, and zinc overload all improved as well. Abbreviations: White blood cell count (WBC), Hemoglobin (Hgb), Hematocrit (Hct), Mean Corpuscular Volume (MCV)

Laboratory	March-2021	May-2021	January-2022
WBC (K/mm^3)	2.5	4.8	3.6

Table 1. (*continued*)

Laboratory	March-2021	May-2021	January-2022
Hgb (Gm/dL)	12.3	12.7	12.9
Hct (%)	38	40.4	40.1
MCV (fL)	103.8	100	95.7
Platelet count (k/mm3)	191	160	158
Neutrophil (%)	28	56.8	39.9
Cobalamin (pg/mL)	239	630	453
Folic Acid (ng/mL)	12.7	not measured	23.8
Copper (μg/dL)	8	92	80
Zinc (μg/dL)	126	not measured	114

S3421

HIV/AIDS, and MAC vs COVID Diarrhea

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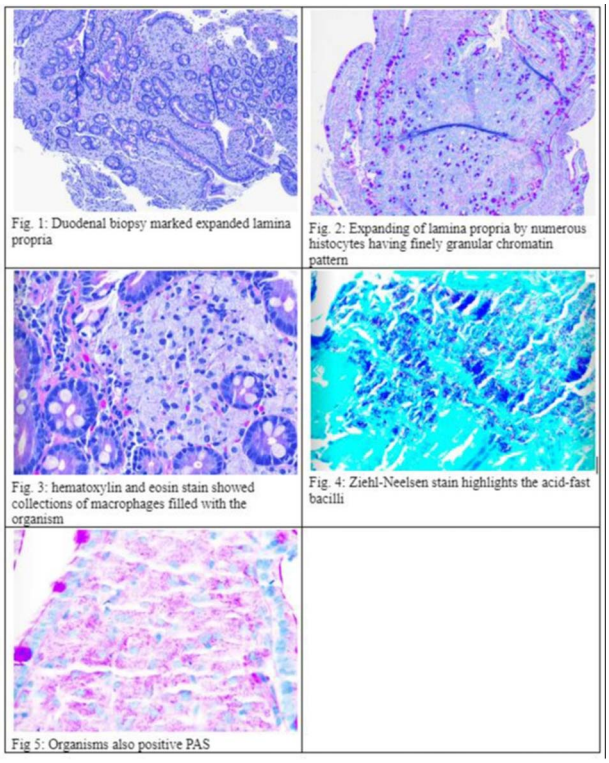
Introduction: During the COVID pandemic, it was difficult to differentiate COVID diarrhea vs other causes, especially in the HIV/AIDS population. HIV infected patients are at high risk for GI infections. Biopsy of the small intestine identifies etiologies which help to differentiate infection, inflammatory, and neoplastic GI disease.
Case Description/Methods: A 39-year-old male presented with two months of epigastric abdominal pain, severe diarrhea, and dehydration during the COVID pandemic in 2020. Past medical history of AIDS wasting syndrome. The patient was severely hypotensive. Exam was notable for mild epigastric tenderness,and hepatomegaly. Complete blood count (CBC) revealed a Hemoglobin (Hg) of 4.3 g. COVID 19 was positive. Gastrointestinal panel PCR was negative. CD4 count 1, HIV RNA viral load 48,700. MRI of the abdomen and pelvis showed diffuse bowel wall thickening in the small intestines. On hospital day 2, Esophagogastroduodenoscopy (EGD) and Colonoscopy were performed for anemia and diarrhea, which revealed abnormal variegated mucosa in the duodenum. Colonoscopy showed a normal mucosal appearance. The pathology report of the duodenum revealed macrophages in the lamina propria, which stained positive for Acid Fast Bacilli. Duodenal biopsy showed markedly expanded lamina propria with numerous histiocytes having finely granular chromatin pattern (Panel 1 and 2). The patient was diagnosed with Mycobacterium avium-intracellulare enteritis.
Discussion: This 39-year-old male presented with chronic diarrhea without fever or respiratory symptoms. The most pertinent question for the health care team was whether COVID 19 caused the diarrhea.. Endoscopy with biopsy of the small intestine was essential for an accurate diagnosis. MAC infection in HIV has been dramatically declining since the widespread use of antiretroviral therapy and prophylaxis against MAC infection compared to the 1990s (from 65.3/1000 in 1992 to 2/1000 in 2015) (1). It is important to differentiate MAC from other diseases with a macrophage infiltration or inclusions: including Whipple's, histoplasmosis, and macroglobulinemia. Patients with MAC enteritis present with fever, and watery diarrhea with diffuse small bowel malabsorption which mimics Whipple's disease. However, the key to differentiate these two entities is doing a duodenal biopsy showing foamy macrophages with PAS-positive acid-fast bacilli (2).



[3421] **Figure 1.** Histology finding.

S3422

Hepatic Parenchyma and Cholecystectomy Clip in Duodenum? An Interesting Case of Pancreatic Adenocarcinoma

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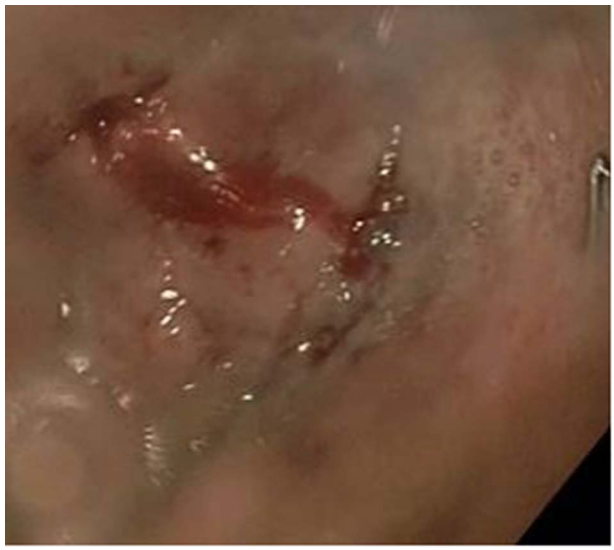
[1]Louisiana State University Health Sciences Center, Shreveport, LA; [2]Louisiana State University, Shreveport, LA.

Introduction: Pancreatic ductal adenocarcinoma (PDAC) is a leading cause of cancer-related death in many industrialized countries. Patients with PDAC typically report non-specific symptoms such as abdominal pain, weight loss, and jaundice. Given the lack of effective screening, many patients are diagnosed at the time of metastasis. Here we present a case of a patient who was diagnosed with metastatic PDAC with contained perforation in the duodenum.
Case Description/Methods: A 74-year-old man with a history of prostate cancer requiring prostatectomy presented with a 2-month history of abdominal pain, nausea, vomiting, unintentional weight loss, and melena. He had normocytic and normochromic anemia with normal liver enzymes and lipase. Tumor markers ordered around the same time as EGD, including CEA/PSA, were unremarkable, but CA 19-9 was 6959 U/mL. EGD revealed a large necrotic, ulcerated duodenal mass in the first portion with suspicion of contained perforation. A metallic object was also identified which was likely a cholecystectomy clip as confirmed on subsequent CT. Pathology showed necrotic tissue and hepatic parenchymal cells, but there were no diagnostic neoplastic elements. CT abdomen showed a proximal duodenal mass with ulcer-like outpouching extending into the gallbladder fossa, abnormal perfusion of liver segments 4 and 5 with soft tissue mass adjacent to the pancreatic head suspicious of locally invasive primary duodenal malignancy. Whipple's procedure was performed, given the lesion's appearance and extent, and for diagnosis. Pathology revealed a poorly differentiated ductal adenocarcinoma of the head of the pancreas, with a signet ring pattern invading the duodenum wall, anterior surface of the pancreas. There was lymph node metastasis. The patient elected for hospice, given his poor prognosis (Figure).
Discussion: Patients with PDAC often report epigastric pain, jaundice, and weight loss. Our patient presented with abdominal pain and weight loss. However, the melena and anemia raised concern for GI bleed; hence an EGD was performed, which led to the diagnosis of pancreatic adenocarcinoma with contained perforation in the duodenum. There are no practical screening tools for pancreatic cancer. Liver enzymes, CA 19-9, in addition to ultrasound abdomen or abdominal CT, depending upon the patient's symptoms and age, can be considered as part of work up. Curative treatment for PDAC is surgical resection with negative surgical margins and adjuvant therapies; however, the five-year survival rate remains low.



[3422] **Figure 1.** Necrotic duodenal lesion with cholecystectomy clip.

S3423

Incidental Finding of a Duodenal-Type Follicular Lymphoma in NASH Cirrhosis: A Rare Malignancy With Favorable Outcomes

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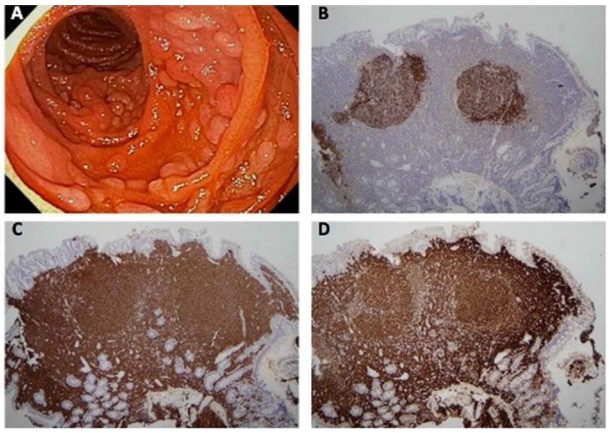
Introduction: Duodenal follicular lymphoma (DFL) are uncommon, only being recognized as a new entity by the World Health Organization (WHO) in 2016. Over a 39-year period, the Surveillance, Epidemiology, and End Results (SEER) registry only found 1109 cases. Detection of lesions occurs in both symptomatic and asymptomatic patients. Treatment options include surgery, radiation, chemotherapy (including rituximab) and varying combinations. Curiously, many patients do very well with little to no treatment. At five years, progression-free survival is greater than 70% and overall survival ranges from 80-94%. A better understanding of the characteristics of FL of the GI tract relating to epidemiology, pathophysiology, treatment options, and long-term outcomes is needed.
Case Description/Methods: A 57-year-old female with nonalcoholic steatohepatitis (NASH) cirrhosis underwent upper endoscopy surveillance for gastroesophageal varices. For the year prior, the patient reported a poor appetite and 30-pound weight loss. She denied any personal or family history of malignancy. She otherwise reported no gastrointestinal symptoms. Endoscopic evaluation revealed multiple small round polyps in a diffuse distribution in the second portion of the duodenum (Figure A). Tissue in-situ hybridization was positive for BCL2/IGH fusion loci establishing a diagnosis of follicular B-cell lymphoma. Immunohistochemical staining patterns and morphology indicated a low-grade lesion (Figures B–D). Further work up included PET/CT, which was negative for systemic disease. Based on the indolent natural history of DFL, plans are for observation.
Discussion: Stage I/II gastrointestinal FL (GI-FL) are associated with >80% 5-year survival. Many of these patients do not require treatment. Given its rarity, little is known about its pathogenesis. DFL shows a female predilection in as high as a 2:1 ratio. The most common location is in the duodenum, though it can be seen in the large bowel and stomach. While little is known about its pathogenesis, gut microbiome disruption was associated with DFL in a case-control study. Disruption of the mucosal microbiota is also seen in nonalcoholic fatty liver disease (NAFLD).



[3423] **Figure 1.** (A) Endoscopic view of second portion of duodenum; (B) CD21 stain identifying architecture of lymphoid follicles with positively staining dendritic reticulum cells; (C) CD20 stain revealing lymphocytes within lymphoid follicles are overwhelmingly CD20-positive B cells; (D) BCL-2 stain defines follicles as neoplastic and confirms diagnosis of follicular B-cell lymphoma.

S3424

Imaging-Negative Double Jejunal Intussusception Diagnosed on Laparoscopy With Intraoperative Enteroscopy in a Patient With Peutz-Jeghers Syndrome

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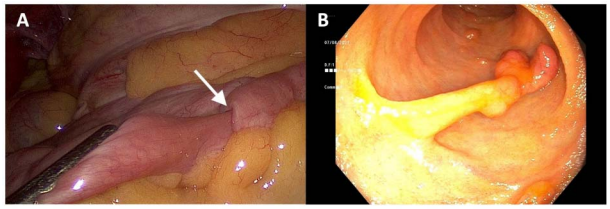
Introduction: Peutz-Jeghers syndrome (PJS) is a rare, autosomal dominant syndrome characterized by gastrointestinal polyposis, mucocutaneous pigmentation, and an increased risk of malignancy. Although generally benign, PJ-type polyps can be large and can act as lead points for intussusception. We describe the case of a 25-year-old female with PJS who presented with recurrent abdominal pain in the setting of negative cross-sectional imaging who was found to have a double intussusception on diagnostic laparoscopy with intraoperative enteroscopy.
Case Description/Methods: The patient's surgical history was notable for five reductions of small bowel intussusception related to PJ-type polyps since age 9. She initially presented with intermittent abdominal pain six months prior to this admission, at which time seventeen small bowel PJ-type polyps were resected via anterograde small bowel enteroscopy. Her pain persisted despite intervention, and magnetic resonance cholangiopancreatography (MRCP) and magnetic resonance enterography (MRE) four months prior to admission demonstrated two additional polyps in the terminal ileum which were resected via colonoscopy. The patient then presented with acutely worsening abdominal pain that she described as similar to her past episodes of intussusception. Although a computed tomography (CT) scan with IV contrast was unremarkable, there was high clinical suspicion for intussusception, so a multidisciplinary decision was made to proceed with diagnostic laparoscopy. Two jejunal intussusceptions related to large polyps were identified and reduced, and one large (25 mm) polyp was surgically resected (Figure A and B). On intraoperative enteroscopy via enterotomy, one smaller (15 mm) duodenal polyp and three large (20-25 mm) jejunal polyps were removed. The patient was discharged three days postoperatively with resolution of her pain.
Discussion: Patients with PJS require multidisciplinary care given their significantly increased risk of both gastrointestinal and extraintestinal malignancy and complications such as intussusception. While CT is generally the preferred imaging study when evaluating for intussusception in adults, there is limited data on its sensitivity, with estimates ranging from 58-100%. Our case highlights two important points: first, although PJ-type polyps are generally benign, they may grow rapidly; and second, even if imaging is unremarkable, clinicians should still consider intussusception in patients with predisposing risk factors and a compatible clinical picture.



[3424] **Figure 1.** (A) Laparoscopic view of a small bowel intussusception (arrow) related to a Peutz-Jeghers-type polyp. (B) Endoscopic view of a large hamartomatous Peutz-Jeghers-type polyp.

S3425

Is Pembrolizumab Associated With Celiac Disease?

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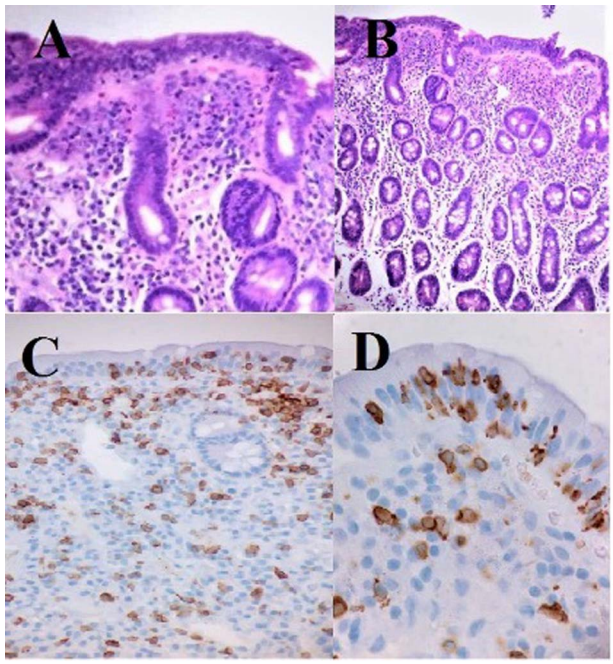
[1]SBH Health System, Bronx, NY; [2]The Brooklyn Hospital Center, Brooklyn, NY; [3]St. Barnabas Health System, Bronx, NY; [4]Blanchard Valley Health System, Findlay, OH.

Introduction: Immune checkpoint inhibitors (ICIs) have improved the survival rate and prognosis of patients with several advanced malignancies by enhancing the immunologic system and augmenting the antitumor cytotoxic T-lymphocyte response. However, their side effects are being observed more often because of the increase in their use. They are called immune-related adverse events (irAEs), such as hepatitis, colitis, and other immune reactions. We present the case of Pembrolizumab-induced Celiac disease after treatment of metastatic melanoma.
Case Description/Methods: A 49 y/o Female presented to the GI clinic for IDA, had a history of malignant melanoma diagnosed eight years ago. She presented with diffuse lymphadenopathy of the left arm and was diagnosed with malignant melanoma. Radiation therapy was initiated, and at her three months follow-up with PET-scan there was involvement of the left femur and hip and multiple nodules in abdominal and esophageal areas. This was deemed advanced metastatic malignant melanoma and was started on Pembrolizumab. After receiving this treatment for a year, she was in remission and has continued to have treatment every twelve weeks. Upper endoscopy and Colonoscopy were done to evaluate the etiology of IDA. Duodenal biopsy showed areas of inflammation of the first and second portions of the duodenum. The pathology report showed flattened villi and crypt hyperplasia; immunohistochemical showed increased CD3+ T cell lymphocytes in the surface epithelium consistent with Celiac Disease Image1. Blood tests was Positive TTG-IgA and HLA-DQ2. She was then placed on Gluten-free diet and proton pump inhibitors, showing improvement in the anemia and continues to be in remission (Figure).
Discussion: Celiac disease is an immune-mediated inflammatory disorder that affects the small bowel, triggered by the consumption of gluten. Gluten hypersensitivity leads to activation of T-cells in the lamina propia, producing mucosal inflammation and villous atrophy. Commonly resolves after withdrawal of wheat and cereals from the diet. Pembrolizumab is a monoclonal antibody that works by blocking programmed death receptor-1 (PD -1), it is used in the treatment of advanced cancer, one of them being Metastatic melanoma. However, it can cause irAEs during its use or even after. Only a few cases have been reported in the literature of pembrolizumab-induced celiac disease; We would like to emphasize the importance of a proper evaluation and follow-up of celiac disease in patients on Pembrolizumab treatment.



[3425] **Figure 1.** A and B, H&E stain 40X showing flattened villi and crypt hyperplasia, (C and D) low and high power view CD3+ T cell near the surface epithelium.

S3426

Incidental Terminal Ileum Polyp During a Screening Colonoscopy

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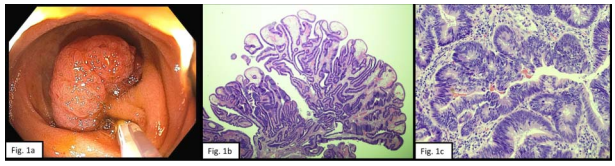
Introduction: Small intestinal adenomas can occur sporadically. While the duodenum is the most frequently involved site, an ileal adenoma is extremely rare, with no clear surveillance guidelines. It is controversial whether or not an ileoscopy should be performed during routine screening colonoscopy. We present a rare case of an incidentally found adenoma in the terminal ileum (TI) during a surveillance colonoscopy.
Case Description/Methods: A 69-year-old male with a past medical history of hypertension and colon polyps presented to the outpatient endoscopy unit for his third colonoscopy. His first colonoscopy was done at age 58. He had seven sub-centimeter adenomatous polyps at that time. An ileoscopy was not attempted, and he was instructed to repeat the exam in 3 years. A surveillance colonoscopy without ileoscopy was done at age 62. He only had a few left-sided hyperplastic polyps, but a repeat exam in 5 years was recommended, given his history. At this time, he did not have any complaints and denied any gastrointestinal symptoms. His vital signs and physical exam were unremarkable. Colonoscopy was performed, and the TI was intubated. A 3 cm pedunculated polyp (Figure A, B) was found and removed piecemeal with a hot snare, and retrieved with a Roth net. Two endoclips were placed to control post-polypectomy bleeding, with no bleeding at the end of the procedure. One mL of tattoo ink was injected submucosally 2-3 cm proximal to the polyp. Pathology revealed tubular adenoma (TA) with high-grade dysplasia (HGD) (Figure C), with margin negative for HGD. He also had seven sub-centimeter adenomatous polyps in his colon. He was instructed to repeat colonoscopy with ileoscopy in 6 months for surveillance and to get an upper endoscopy at that same time to rule out duodenal adenomas with a plan for video capsule endoscopy to clear the rest of his small bowel.
Discussion: Adenomas are commonly found in the colon; however, they can develop in the small bowel. While not well quantified, their frequency is low, with the duodenum and jejunum more commonly affected than the ileum. There are no available surveillance guidelines when adenomatous polyps are found in the TI. In our case, we completely resected an incidentally found large adenomatous TI polyp with HGD, which could have progressed to cancer and/or obstructive symptoms. This suggests a role for routine ileoscopy during screening colonoscopy.



[3426] **Figure 1.** (a) A large 3 centimeter pedunculated terminal ileum polyp. (b) A low-power photomicrograph of hematoxylin and eosin stain showing adenomatous polyp. (c) A medium power photomicrograph of hematoxylin and eosin stain showing an area demonstrating increased architectural complexity and loss of nuclear polarization, indicative of high-grade dysplasia.

S3427

Implicit Bias Contributing to Delayed Diagnosis in a Rare Case of Endoscopic Biopsy Proven Gastrointestinal Amyloidosis

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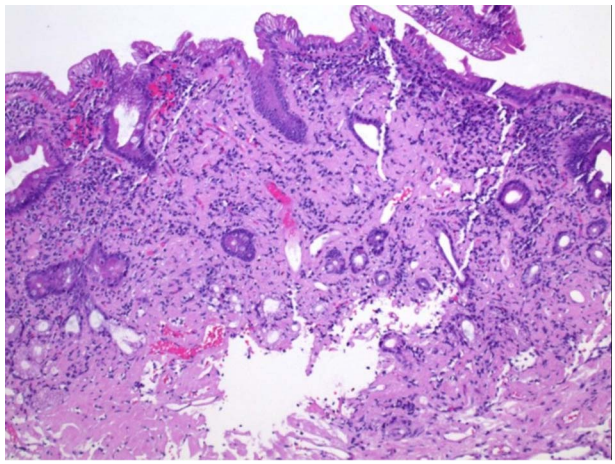
Introduction: Gastrointestinal amyloidosis often requires extensive workup to diagnose, but early diagnosis and initiation of treatment improves mortality. Implicit bias leads to narrow workups which can misdiagnose rare diseases. We present a case of gastrointestinal amyloidosis ultimately diagnosed by endoscopic biopsy. The rarity of this disease as well as bias that led to anchoring on more common diagnoses resulted in months of misdiagnosis.
Case Description/Methods: A 67 year old woman with a history of abdominal aortic aneurysm (AAA), substance use disorder in remission, and chronic malnutrition presented with worsening of chronic abdominal pain. Workup over the course of eight prior ED visits for abdominal pain was unremarkable. Her pain was attributed to functional pain or opioid use and withdrawal despite a lack of documented opioid relapse. On this presentation, expanding AAA size prompted surgical consult. The surgical team expressed concern that she would not tolerate an open repair with a BMI of 13.9; TPN was started. GI recommended endoscopy to assess for a malabsorptive process. Unfortunately the procedure was delayed 22 days as the patient felt her uncontrolled pain was not validated by the care team. After many care

conferences, she agreed to upper endoscopy which showed flattening of the villi of the duodenum. Microscopic testing from biopsies showed extensive amyloidosis (Figure). Amyloidosis resulting in intestinal ischemia was thought to be the cause of her pain and malnutrition.

Discussion: Implicit bias associated with the patient's history of addiction led to anchoring bias and initial narrow workup and misdiagnosis. Implicit bias amongst physicians regarding substance use disorders has been associated with suboptimal care. The severity of the patient's abdominal pain and malnutrition should have prompted earlier referral to GI and may have led to expedited diagnosis of intestinal amyloidosis. Additionally, the frequent misdiagnosis and dismissal of symptoms resulted in mistrust leading the patient to decline further workup. Recognizing implicit bias can allow clinicians to provide more equitable workup and care.



[3427] **Figure 1.** DUODENUM, BIOPSY Amyloidosis, extensive, involving mucosa and submucosa, perivascular and diffuse.

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S3428

Incidental Intramucosal Adenocarcinoma of the Appendix Presenting as Acute Appendicitis

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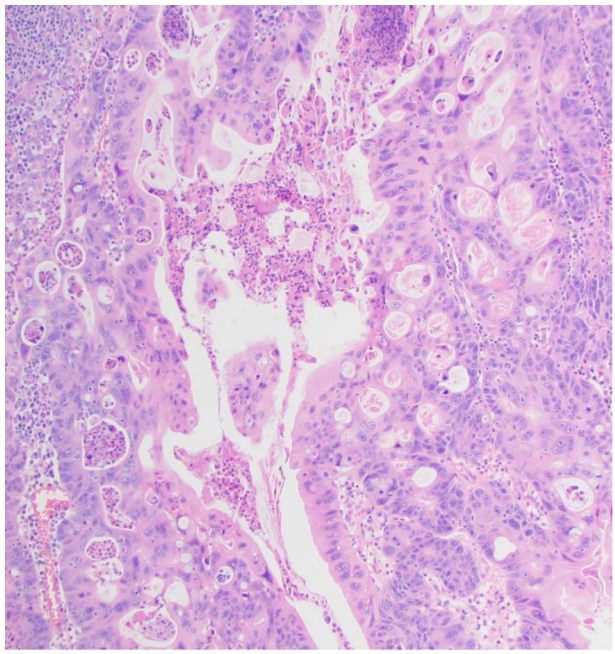
Introduction: Intramucosal adenocarcinoma arising in an adenomatous lesion of the appendix is a rare occurrence and has never been reported in a young patient per our literature search. This report is of a 29-year-old woman with this lesion and her course.

Case Description/Methods: The patient is a 29-year-old female who presented to an urgent care with complaints of nausea and right flank pain. She was subsequently referred to our emergency department, where a CT scan showed probable appendicitis characterized by enlargement, hyperenhancement, and wall thickening. Her medical history revealed that she was currently 3 months post-partum and taking Flagyl for bacterial vaginosis, and that she had a history of protein S deficiency, asthma, and remote liver lesions that were favored to represent hemangiomas and hepatic adenomas per imaging. Her vitals and labs were normal and physical exam revealed only abdominal and right flank tenderness. She was taken for a laparoscopic appendectomy, where the appendix was described as enlarged and inflamed without perforation intraoperatively. The resected appendix was sent to pathology where upon opening they discovered a 1.7 cm yellow-tan polypoid mass in the appendiceal tip. Histologic sections showed intramucosal adenocarcinoma arising in a traditional serrated adenoma with conventional-type adenomatous dysplasia and extensive high-grade dysplasia. No invasion was identified. The patient was discharged two days later and is now scheduled for a right hemicolectomy with node dissection (Figure).

Discussion: This report has detailed the course of a young patient with intramucosal adenocarcinoma of the appendix whose presentation was consistent with acute appendicitis by symptoms, radiology, and physical exam. Although this is a rare occurrence, this case serves as an important reminder that cancer can be found in patients of any age and may surface with an atypical presentation.



[3428] **Figure 1.** An area of intramucosal adenocarcinoma within an adenomatous lesion of the appendix.

S3429

Iron Deficiency Anemia and Small Bowel Strictures: Thinking Beyond Crohn's Disease

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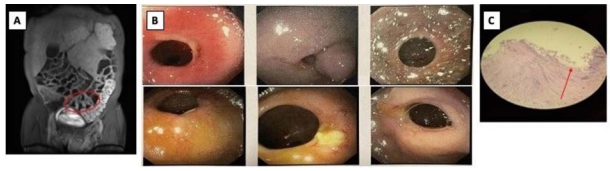
Introduction: NSAIDs are well known to cause gastrointestinal (GI) mucosal damage. We present a case of a 72-year-old male who was found to have Diaphragm Disease (DD).
Case Description/Methods: 72-year-old male with history of IDA, back pain on long-term NSAIDs, and testicular cancer s/p radiation presented to the ED for abdominal pain. History is notable for Crohn's disease in his sister and niece. EGD showed patchy gastritis and colonoscopy was negative for ileitis or colitis. Video capsule endoscopy showed linear erosions with segmental strictures and ulcers in the jejunum and ileum. Capsule passage was confirmed with imaging. MRE showed segmental circumferential bowel wall thickening of mid small bowel loop concerning for fibro-stenotic disease. On push enteroscopy, no strictures or ulcers could be reached. Next, he had a lap-assisted enteroscopy with 150 cm SB resection. Multifocal segmental strictures of the SB were noted mostly involving the jejunal segment. The resected SB segment had strictures with mucosal necrosis, acute and chronic inflammation with erosions and focal fibrinous membranes. No diagnostic morphologic changes of IBD or obliterative endarteritis and stromal cellular atypia found in radiation associated changes were noted. The histologic findings and clinical history led to a diagnosis of DD. After surgical intervention and cessation of NSAIDs his symptoms resolved (Figure).
Discussion: NSAIDs can cause many GI side effects including but not limited to small bowel erythema, erosions, and strictures, known as NSAID induced enteropathy. DD, characterized by concentric septa-like mucosal projections cause the SB lumen to narrow, can present with variable, non-specific symptoms. There is no established relationship between length of NSAID use and development of DD. Most cases are caused by chronic NSAIDs but there are reports of DD with use for weeks to months. Our case of a 72-year-old male with a non-specific GI complaint in the setting of a complex personal and family history highlights the importance of taking a complete history. When strictures were found in the SB this was not enough to finalize etiology as they can be seen in Crohn's, radiation induced enteritis and NSAID use. While cases of DD are well documented, the course of our patient's workup provides critical insight into diagnosing DD.



[3429] **Figure 1.** A, Magnetic Resonance Enterography: coronal fat saturated T1-weighted images of the abdomen acquired 5 minutes after contrast administration demonstrates thickened loops of bowel in the lower abdomen with delayed enhancement (red circle) (B) Multifocal segmental strictures of the distal small bowel visualized during laparoscopic assisted enteroscopy (C) Segment of resected small bowel showing mucosal necrosis and chronic inflammation/ulcers (red arrow).

S3430

Lactulose Works in the Large Bowel: But How Does It Improve Hepatic Encephalopathy in a Patient Without a Large Bowel?

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Introduction: Lactulose is a non-absorbable disaccharide which acts in the large bowel, and is commonly used in the treatment of hepatic encephalopathy. We present an interesting case of altered mental status due to hepatic encephalopathy successfully managed with lactulose in a patient with history of total colectomy.
Case Description/Methods: A 67-year-old male with non-alcoholic cirrhosis and inflammatory bowel disease (IBD) post total proctocolectomy with a continent ileostomy known as a "Kock-pouch" (K-pouch) presented to the hospital with flu like symptoms and altered mental status. He was subsequently found to be positive for COVID-19. At the time of initial evaluation, the patient was obtunded with an elevated ammonia level of 91 umol/L. Colorectal surgery was consulted as the patient was not able to empty his K-pouch. Recently, he complained of inability to catheterize and with bleeding from the stoma. Initial catheterization with a Water's tube yielded 400 cc of effluent. Nasogastric tube was placed through which he was receiving lactulose 30 mg q8 hours. The patient's mental status improved within 24 hours. The patient ultimately underwent flexible pouchoscopy with endoscopic dilation and placement of a 22 French mushroom catheter for decompression of the K-pouch.
Discussion: Lactulose is a non-absorbable disaccharide composed of galactose and fructose. The small intestine does not have the enzymes required to breakdown lactulose so it reaches the large bowel in its original form. In the large bowel, it is metabolized by colonic bacteria into monosaccharides and then to volatile fatty acids, hydrogen and methane. Lactulose decreases both the production and absorption of

ammonia mainly through the presence of gut bacteria. The question arises as to how lactulose decreased ammonia levels in this patient without a large bowel. One proposed mechanism is the translocation of bacteria normally found in the large bowel to the small intestine. Small Intestinal Bacterial Overgrowth (SIBO), is a condition causing an increased number of bacteria in the small intestine. Patients with IBD and structural abnormalities are at increased risk of developing SIBO. Lactulose is commonly used in the diagnosis through the administration of lactulose and subsequent measurements of hydrogen and methane gas in expired air. This condition, in our patient with history of ulcerative colitis and colectomy, is a proposed mechanism of the efficacy of lactulose in the treatment of hepatic encephalopathy.

S3431

Intestinal Strongyloidiasis and Hyperinfection Syndrome in an Immunocompetent Patient

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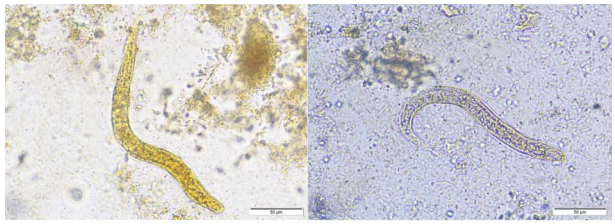
Introduction: Strongylodiasis is a term attributed to the many pathologies caused by the nematode helminth *Strongyloides stercoralis*. The condition is prevalent in around 70 countries with endemicity in tropical and subtropical climates, including the Southern United States. In chronically infected and immunocompetent individuals, the disease is generally asymptomatic with eosinophilia and stool larvae being the only indication of infection. A more disseminated form of the disease can lead to a debilitating condition known as *Strongyloides* hyperinfection syndrome (SHS), caused by a high intestinal parasitic load leading to multi-organ damage. SHS may reach 2% of patients with strongyloidiasis, typically occurring in the immunocompromised with underlying conditions such as hematologic neoplasias, advanced HIV, and organ transplantation. The risk of SHS in immunocompetent patients is not zero, however, as other conditions such as malnutrition, diabetes, chronic obstructive pulmonary disease, alcoholism, and chronic kidney disease are identifiable predisposing conditions for SHS.
Case Description/Methods: A 73-year-old South Asian male with well-controlled asthma and diabetes presented with worsening shortness of breath, cramping abdominal pain, and distension for the past month accompanied by a productive cough and constipation with no bowel movement for the last 4 days. Twenty years earlier, he had emigrated from Bangladesh to his current residence in the northeast United States. Initial exam was notable for wheezing and diffuse, mild abdominal tenderness. Workup showed a leukocytosis with eosinophilic predominance and computed tomography showed a short segment of mural thickening in the sigmoid colon/rectum. Treatment with nebulizers and bowel regimens failed to relieve his symptoms initially. Stool analysis was notable for the presence of rare *Strongyloides stercoralis* larvae and the patient was started on ivermectin with complete relief of pulmonary and gastrointestinal symptoms (Figure).
Discussion: SHS varies widely in symptomatology with gastrointestinal complaints ranging from abdominal pain to vomiting and watery diarrhea. *Strongyloides* is unique among intestinal nematodes in its ability to persist in humans for many years. Most cases of infection in diabetics are in those individuals with poor sugar control. Our case appears to be a waning of immunity in a likely chronic, asymptomatic strongyloidiasis patient housing the parasite since emigration from South Asia leading to SHS requiring urgent medical care.



[3431] **Figure 1.** (a) Iodine contrast staining of stool showing rhabditiform larva of Strongyloides stercoralis (b) Fecal agar plate culture showing rhabditiform larva of Strongyloides stercoralis.

S3432

Intermittent Superior Mesentric Artery Syndrome Secondary to Aggressive Low-Fat Dieting

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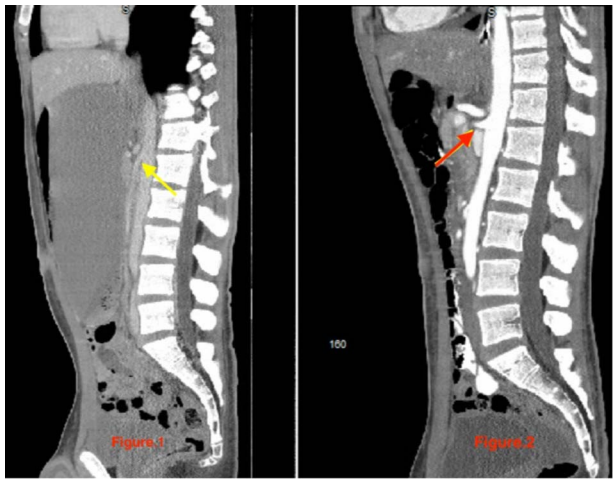
Introduction: Superior mesenteric artery (SMA) syndrome is a rare and potentially life-threatening disorder that is caused by the compression of the third part of the duodenum between the SMA and the origin. We present a rare case of intermittent SMA syndrome in a young patient who presented with symptoms of gastric outlet obstruction and was diagnosed with SMA syndrome based on the radiology on admission. During the hospital course, the symptoms improved and repeat imaging showed the resolution of the compression of the duodenum.
Case Description/Methods: A 19-year-old male patient with no past medical history presented with epigastric abdominal pain associated with non-bilious, non-bloody vomiting for two days. He endorsed 30 kilograms of intentional weight loss in the last six months, using a low-fat diet. On physical examination, the patient was severely malnourished with a body mass index (BMI) of 18. Abdominal examination showed moderate distention with mild tenderness in the epigastric area. CT scan of the abdomen revealed gastro-duodenal distension with a transition point at the third part of the duodenum due to compression between the SMA and aorta with an acute aortomesenteric angle close to 8 degrees. Gastric decompression was done using a nasogastric tube, draining 1500 mL of bilious fluid. IV hydration and electrolyte supplementation were given. Gastroduodenoscopy on day two of admission ruled out obstruction. By the third day of hospitalization, the patient's abdominal pain and vomiting resolved. CT angiography of the abdomen confirmed normalization of the aortomesenteric angle. Oral feeding was started and he was discharged on day six of hospitalization (Figure).
Discussion: SMA syndrome may have an acute, chronic, or intermittent presentation. The SMA is surrounded by a mesenteric fat pad which lies between the SMA and the aorta. In our case, the loss of the aortomesenteric fat pad secondary to acute weight loss placed the patient at risk for intermittent positional compression of his duodenum. This case highlights the importance of considering SMA syndrome in the context of aggressive dieting and eating disorders.



[3432] **Figure 1.** (1) is a CT contrast of the abdomen and pelvis on admission and the yellow arrow shows an acute aortomesentric angle with a compressed intervening duodenum. (2) is a CT angiography of the abdomen and pelvis performed on day 3 of admission and the red arrow shows normalization of the aortomesenteric angle with a patent duodenum.

S3433

Intussusception in an Elderly Patient: A Carcinoid Case Report

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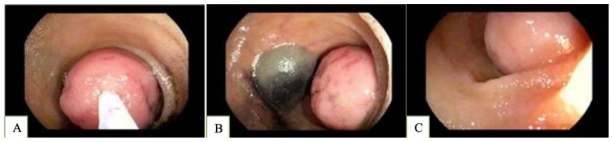
Introduction: Intussusception occurs when a proximal bowel segment slides into a distal bowel segment, which may cause bowel obstruction and intestinal ischemia. Although rarely seen in adults, intussusception is important to consider when evaluating a patient with abdominal pain as it can lead to life-threatening complications if undetected. Here, we describe a case of intussusception in an elderly gentleman secondary to a carcinoid tumor in his terminal ileum.
Case Description/Methods: A 65-year-old male, with a history of a large rectal tubulovillous adenoma with focal high-grade dysplasia that required surgical resection 10-years ago, hypertension, and diverticulosis presented with a several month history of relapsing lower abdominal pain associated with nausea. His abdominal exam post-attack was normal with the exception of mild tenderness in the right lower quadrant. An abdominal CT showed a 2.3 cm soft tissue density in the small bowel with transient, non-obstructive intussusception. Colonoscopy revealed a firm, submucosal mass 20 cm into the terminal ileum. A biopsy of the mass revealed that it was a carcinoid tumor and the patient has been scheduled for surgical resection (Figure).
Discussion: The majority of intussusception cases in adults involve the small bowel and are secondary to a pathological lead point such as a neoplasm, post-surgical adhesions, anatomical changes, endometriosis, or iatrogenic causes such as placement of a gastrostomy or jejunostomy tube. Symptoms can be relatively non-specific, and most patients will describe intermittent crampy abdominal pain, bloating, nausea, and vomiting. Given its rarity in the adult population and its non-specific presentation, intussusception can be a challenging diagnosis which is often delayed or missed altogether. Prompt diagnosis may prevent complications such as necrosis, perforation, and sepsis.



[3433] **Figure 1.** (A), (B), and (C) display colonoscopy images of a firm, submucosal mass 20 cm into the terminal ileum.

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S3434

Internal Duodeno-Pancreatic Fistula Likely Secondary to Concealed Perforation

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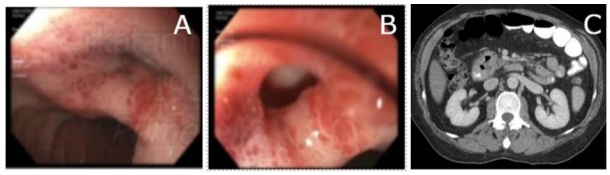
Introduction: Pancreatic fistulas can be classified as internal and external fistulas depending on whether they are opening onto the skin or internal structures. An internal fistula can be secondary to acute or chronic pancreatitis, malignancy, trauma, post-operative, or chronic inflammatory disease like ulcerative colitis or Crohn's disease. Except for post-operative pancreaticoduodenal fistulas, other types are uncommon and rarely reported. We report a case of an asymptomatic duodeno-pancreatic fistula likely secondary to concealed perforation.
Case Description/Methods: A 55-year-old male with a medical history of asthma and alcohol use presented to the hospital with multiple episodes of hematemesis associated with dizziness and palpitations for one day. He reports no use of NSAIDs or blood thinners but consumes one pint of vodka daily (Table). The abdomen was distended with tenderness on palpation in the epigastrium. No spider nevi or asterixis. Esophagogastroduodenoscopy (EGD) revealed recently bleeding grade II esophageal varices that were, completely eradicated and banded, erythematous mucosa of antrum and duodenum, and a duodenal fistula with a crater-like opening in the duodenum. Biopsy of the duodenum showed non-specific chronic inactive inflammation with no evidence of *Helicobacter pylori*. CT of the abdomen was done and it showed an air-containing fistulous tract is noted extending from the second portion of the duodenum medially to the pancreatic head. There is no gross evidence of mass. There was also some thickening of the gastric antrum and duodenum, fatty infiltration of the liver, and perisplenic varices. The patient was treated with IV fluids, Somatostatin analogs, and Beta-Blockers and was referred to an advanced gastroenterologist for evaluation with endoscopic ultrasound (Figure).
Discussion: Pancreaticoduodenal fistulas are commonly mostly post-operative in addition to other causes including pancreatitis, malignancy, duodenal ulcer, trauma, and inflammatory bowel disease. Pancreatico-duodenal fistula can be asymptomatic. Symptomatic patients can be managed conservatively using TPN, and somatostatin analogs. Patients that failed to respond may be managed with a minimally

invasive or surgical approach using diversion or resection techniques. For high output fistulas, a diversion can be achieved by percutaneous duodenostomy, or transhepatic biliary or trans biliary approach. Alternatively, a more invasive open surgical diversion or resection and anastomosis can be performed.

Table 1. Initial labs on admission

Laboratory Parameters	Values	Reference Range
Sodium	141	136 - 145 mmol/L
Potassium	2.6	3.5 - 5.3 mmol/L
Chloride	92	98 - 110 mmol/L
Blood Urea Nitrogen (BUN)	26	6 - 24 mg/dL
Creatinine	1.7	0.6 - 1.2 mg/dL
Aspartate Transaminase (AST)	65	10 - 36 U/L
Alanine Transaminase (ALT)	42	9 - 46 U/L
Total bilirubin	1.5	0.2 -1.2mg/dl
Albumin	4.0	3.6 -5.1 g/dl
Hemoglobin	11.8	13.5 - 17.5 g/dL
Platelets	271	150 - 450 × 10*3/uL
INR	1.36	0.9 -1.1
HS Troponin I	61	0-76 ng/L
Protime	16.0	9.9-13 sec
PTT	36.6	36.6
Total protein	10.4	6.4 -8.4g/dl



[3434] **Figure 1.** A and B, Duodenal fistula with crater-like opening; (C) Duodeno-Pancreatic fistula in Axial film with no evidence acute or chronic pancreatitis.

S3435

Jejunal Diverticula: A Lesser Known Cause for Massive Lower GI Bleeding

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Introduction: Small bowel diverticula are rare. We describe a case of Jejunal diverticula that caused massive lower GI bleeding requiring surgical intervention.
Case Description/Methods: A 77 year old male presented to our ER with sudden onset bright red blood per rectum. The patient first noticed 2 days ago that his stool was dark red followed by red streaking on toilet paper. A few hours later he developed rectal urgency followed by another large bloody bowel movement along with associated diaphoresis, LLQ cramping and presyncope. Past medical history included GERD, BPH, Hypertension and Polycythemia Vera. He had never undergone an EGD and his last screening colonoscopy was normal 5 years ago. Labwork revealed acute anemia with Hb of 7.7 g/dL (baseline 13) and one unit of RBCs was therefore transfused. Emergent EGD and colonoscopy were performed and these were unremarkable apart from sigmoid diverticulosis. The following day massive rectal bleeding recurred and this time a radionuclide bleeding scan was obtained which was positive for active bleeding in small bowel lumen at the LUQ. Interventional Radiology did not feel the area was approachable for embolization. 6 additional units of RBCs were given due to persistent bleeding. General Surgery was called and the patient was taken to OR for exploratory laparotomy with small bowel resection. Diffuse proximal Jejunal diverticulosis was found to be actively bleeding with blood traveling all the way to distal colon. 60 cm of Jejunum was transected 30 cm from the ligament of Treitz. The patient had an unremarkable postoperative recovery with no further episodes of bleeding and stable Hemoglobin on serial CBCs.
Discussion: Diverticulosis involving the small bowel, especially the Jejunum, is rare. These lesions have a higher incidence in the elderly and are acquired as a result of increased intraluminal pressure. They are thin and friable due to lack of muscularis layer. Concurrent colonic diverticulosis is common. Traditionally considered to be asymptomatic and incidental findings on imaging or surgical exploration, Jejunal diverticula can uncommonly cause lower GI bleeding. Traditional endoscopy is unhelpful given the lack of accessibility of the small bowel. CT Angiography and Nuclear Medicine bleeding scans may be beneficial to localize bleeding. Hemorrhage is usually brisk, requiring massive blood transfusion to maintain hemodynamics. Unstable patients should undergo emergent laparotomy so that resection of the involved segment can be performed.

S3436

Jejunal Gastrointestinal Stromal Tumor (GIST) as a Rare Cause of Lower GI Bleed

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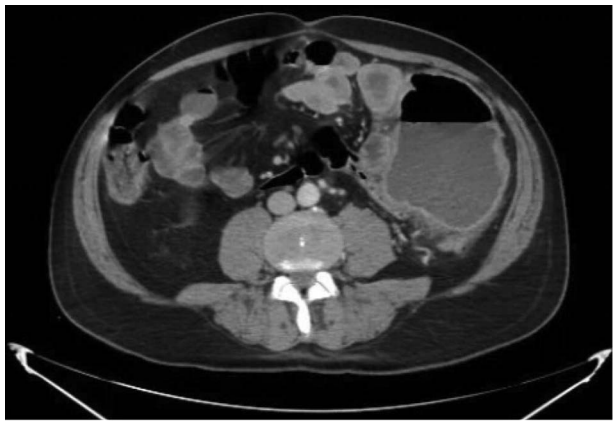
Introduction: Gastrointestinal stromal tumors (GIST) are mesenchymal neoplasms that range from asymptomatic to nonspecific presenting symptoms of nausea, vomiting, and abdominal fullness. The prevalence of GIST is approximately 1% of all gastrointestinal (GI) malignancies. We present a rare case of occult lower GI bleeding leading to a diagnosis of GIST in a 53-year-old patient.
Case Description/Methods: A 53-year-old male without prior medical history was evaluated after a syncopal episode. He was found to have hemodynamically significant anemia requiring multiple units of packed red blood. Endoscopy revealed gastric erosions without active bleeding. The patient became febrile after transfusion, prompting infectious workup including abdominal CT evaluation. A 11.4 x 8.6 x 13.8 cm a collection with wall thickening and adjacent mesenteric fat stranding was seen, contiguous within the jejunum. MRI evaluation confirmed these findings with an additional borderline enlarged para-aortic lymph node. Empiric antibiotic therapy with cefepime and metronidazole was initiated until infectious etiology was ruled out including negative blood cultures. Subsequently, the patient underwent exploratory laparotomy with small bowel resection. Intraoperative course was uncomplicated but significant for colonic and omental adhesions. Metastatic workup while awaiting biopsy results was negative. Histopathology reports confirmed spindle cell GIST of the jejunum with diffusely positive CD117 and patchy CD34 expression and negative margins. He was initiated on oral chemotherapy and cleared for discharge. (Figure)
Discussion: Overt GI bleeding as the initial symptoms of jejunal GIST is uncommon with literature review demonstrating 2 previous case reports of similar presentations. Arriving at a definitive diagnosis can be difficult given that bleeding can occur for years before being apparent to the patient. Also, endoscopic detection can be masked until the tumor size is rather large. Given these challenges, often GIST diagnosis

requires exploratory laparotomy and subsequent biopsy. Tumor excision with negative margins is performed with curative intent. This case illustrates the need to maintain a wide differential to GI bleeding due to malignancies in order to allow for early detection, intervention, and treatment initiation.



[3436] **Figure 1.** Abdominal CT demonstrating a large air fluid collection with wall thickening and adjacent mesenteric fat stranding.

S3437

Initial Presentation of Primary Duodenal Adenocarcinoma

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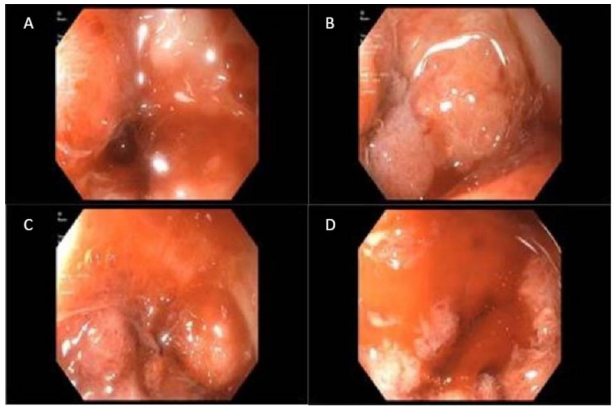
Introduction: Primary tumors arising from the small bowel are a rarity among GI cancers. Here we explore the path to a diagnosis of duodenal adenocarcinoma.
Case Description/Methods: A 65-year-old male with a history of diverticulosis and DVTs presented with a 3-day history of painless melena. Previously, he had separately presented with stable hematochezia, and with extensive lower extremity DVTs and saddle PE needing anticoagulation. In both cases, outpatient follow up for colonoscopy was advised. In the present case, the patient presented with a hemoglobin of 7.8 (previous 13.7), Alkaline Phosphatase of 188, AST 38 and ALT 61. A CT angiogram did not reveal a source. GI performed an EGD which demonstrated an infiltrative and fungating circumferential 40 mm mass in the second part of the duodenum that eroded through the wall (Figure). Biopsies showed grade G2 moderately differentiated adenocarcinoma. MRI abdomen confirmed a duodenal mass with extension to the pancreatic head and encasement of the common bile duct. Surgery took the patient for a Whipple's procedure. Intraoperative biopsies demonstrated moderately differentiated duodenal adenocarcinoma with metastasis in 6 out of 20 nodes. After discharge the patient was started on adjuvant FOLFOX chemotherapy for 3 months and continues to do well and follow up with oncology.
Discussion: Of primary small bowel tumors adenocarcinoma is the second most common, representing around 30-40% of small bowel cancers. Within the duodenum, tumors are most likely to arise in the second section, near the ampulla. Due to nonspecific symptoms most of these tumors are diagnosed at later stages. Tumors near the ampulla present earlier with biliary obstruction. Patients can present with abdominal pain, weight loss, jaundice, vomiting and less commonly GI bleed. Studies show that patients have a median life expectancy of 5.7 months following diagnosis. Treatment for these cancers is contingent on TMN staging. If caught early the cancerous tissue can be removed by endoscopic resection. With more extensive disease in the first or second portion of the duodenum treatment requires full pancreaticoduodenectomy. For those with a tumor in the third or fourth portion a segmental duodenectomy is preferred. The use of chemotherapy after surgery remains controversial with poor evidence of mortality benefit. It is vital in patients with nonspecific symptoms and no clear etiology to rule out small bowel cancers in order to improve their overall chance of survival.



[3437] **Figure 1.** Photos captured during EGD. A & B showing mass in the second portion of the duodenum. C & D showing bleeding after biopsy of mass.

S3438

Malignant Gastrointestinal Neuroectodermal Tumor of the Small Intestine: A Rare Cause of Iron Deficiency Anemia

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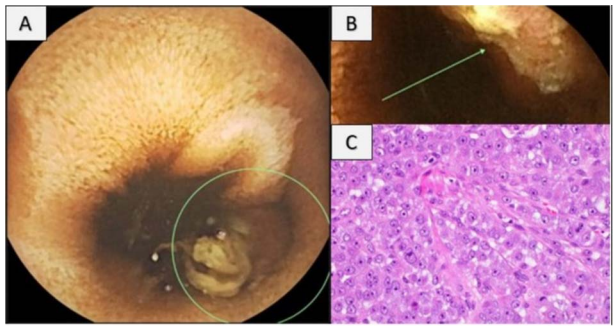
Introduction: Gastrointestinal neuroectodermal tumors (GNET) are a type of high-grade sarcoma of the gastrointestinal (GI) tract that share features with clear cell carcinoma of tendon sheaths and aponeuroses. Patients typically present with clinical signs and symptoms of small bowel obstruction, intussusception or abdominal mass. These tumors can arise in the stomach, colon, as well as metastasis to liver or regional lymph nodes. We present a case of ileal GNET in an otherwise asymptomatic patient with Iron-deficiency Anemia (IDA) diagnosed using video capsule endoscopy (VCE).
Case Description/Methods: A 58-year-old Hispanic male with a past medical history of GERD and obesity presented for evaluation of chronic microcytic anemia. Workup revealed: hemoglobin 8.6 g/dl, MCV 62.2, iron saturation 2.6%, transferrin 307 mg/dl, ferritin < 5 ng/ml, reticulocyte index of 1.34; serum folate, vitamin B12 and TSH levels were within normal limits. He was taking oral ferrous sulfate and vitamin C for IDA. He reported heartburn but denied melena, hematochezia, hematemesis, weight loss and had an unremarkable colonoscopy four years prior. Upper endoscopy revealed mild erosive esophagitis and mild chronic active gastritis with negative biopsies. Colonoscopy was unremarkable in the setting of optimal bowel preparation. VCE revealed an ulcerated mass occupying 35% of the ileal lumen. CT of the

abdomen noted asymmetric wall thickening with enhancement of the small bowel. The patient underwent laparoscopy-assisted enteroscopy with rendezvous technique allowing direct visualization of a 3 cm proximal ileum mass requiring open segmental resection with primary anastomosis. Pathology suggested malignant GNET or clear cell sarcoma-like tumor of the GI tract. Immunohistochemistry was positive for neural crest markers (S100, SOX10) and Vimentin; negative for CD117, DOG1, HMB45, MART1, MAA, chromogranin, and synaptophysin. Fluorescence in-situ hybridization was positive for EWSR1 (22q12) rearrangement. Given the clear surgical margins, absent regional lymph node spread and lack of standard guidelines for this rare pT1N0M0 tumor, surveillance imaging every three months without adjuvant radiation or systemic chemotherapy was recommended. (Figure)
Discussion: This case highlights the importance of VCE for small bowel assessment after negative bidirectional endoscopy to identify the etiology of IDA per American Gastroenterology Association guidelines. In this case, an ulcerated GNET was the etiology of IDA persisting despite iron supplementation.



[3438] **Figure 1.** A-Luminal view of small bowel tumor on Video Capsule Endoscopy (VCE) B–Close up view of ulceration of small bowel tumor on VCE C–Histopathology slide showing epithelioid cells with eosinophilic cytoplasm, pleomorphic nuclei with prominent nucleoli and vesicular nucleoli. Scattered mitotic figures identified.

S3439

Lemmel Syndrome Due to Periampullary Diverticular Food Impaction

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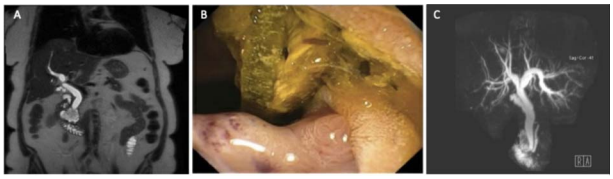
Introduction: Lemmel Syndrome is a rare cause of mechanical biliary obstruction attributed to periampullary duodenal diverticula (PAD). It was first described in 1934 as obstructive jaundice in the absence of biliary stones or malignancy. The majority of PAD are asymptomatic, however, about 5% have symptomatic disease. We present a symptomatic case.
Case Description/Methods: An 80-year-old woman with history of cholecystectomy, coronary artery disease, hypertension, and diabetes was transferred from a free-standing emergency room for acute pancreatitis. She had a one day history of postprandial epigastric pain with radiation to the back. Labs on presentation included lipase above 4000 U/L, BUN 27 mg/dL, AST 316 U/L, ALT 422 U/L, ALP 292 IU/L, total bilirubin 0.6 mg/dL. Abdominal ultrasound revealed 12mm common bile duct (CBD) with intrahepatic ductal dilatation. MRCP showed intra and extrahepatic biliary ductal dilatation with dilated pancreatic duct and a 3.6 cm fluid filled periampullary diverticulum. ERCP showed a large periampullary duodenal diverticulum with food impaction causing obstruction of the ampullary orifice which was removed with forceps. Cholangiogram revealed dilation of pancreatic, common bile, and intrahepatic ducts without any filling defects. Patient improved and was discharged home. (Figure)
Discussion: The incidence of duodenal diverticula is approximately 17% with PAD being the most common type. PADs are pseudo-diverticula most commonly found in the second part of the duodenum and they pose a cannulation challenge during ERCP with an increased risk of complications. PAD in the setting of biliary obstruction is known as Lemmel Syndrome. It can cause biliary obstruction by sphincter of Oddi dysfunction, direct obstruction, or via external compression by fluid or material-filled PAD. In our case, there was biliary compression due to a food-filled PAD. Cross sectional imaging is critical for diagnosis, however, ERCP is the gold standard. Several therapeutic modalities including endoscopic extraction of entrapped food or stones, extracorporeal shock wave lithotripsy, and laparoscopic diverticulectomy have been used. Our case required endoscopic extraction of diverticular food debris and adds to the limited published cases to date. Lemmel Syndrome requires a high index of suspicion when a PAD is found in the setting of biliary obstruction.



[3439] **Figure 1.** A. MRCP image showing a large periampullary diverticulum packed with debris. B. ERCP image of food impacted inside periampullary diverticulum. C. MRCP image demonstrating a dilated common bile duct in absence of choledocholithiasis.

S3440

Malignant Melanoma of Unknown Primary Presenting With Small Intestinal Masses

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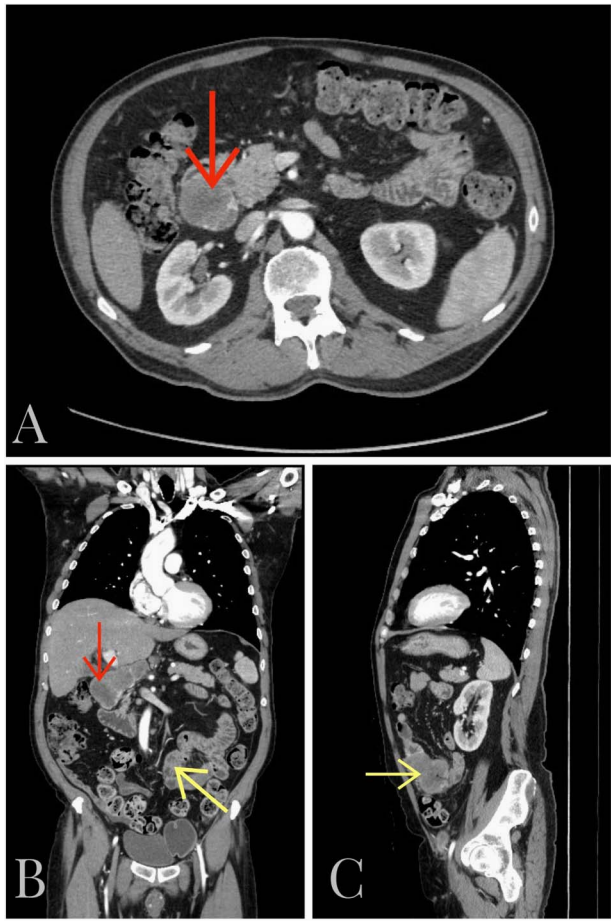
Introduction: GI melanomas are extremely rare and require thorough investigation. Most GI melanomas are metastatic from an oculocutaneous lesion, however, and if not found, are termed melanoma of unknown primary (MUP.) MUP represents only 2% of all melanomas yet it is a major cause of mortality. Common areas of metastasis involve the lymph nodes, small intestines, and subcutaneous sites. We present a case of MUP that developed large abdominal masses within 7 months.
Case Description/Methods: A 68 year old male presented with 1 week of LLQ abdominal pain and constipation. He had undergone a normal colonoscopy 1 year prior. He had normal vitals and labs excluding hemoglobin of 9.5 g/dL, compared to a baseline of 15.2 g/dL. CT scan of the abdomen and pelvis showed: a multi-lobulated heterogeneously enhancing mass centered in the LLQ involving the small bowel measuring 6.8 x 5.6 x 6.5 cm, as well as a 3.5 x 9.0 x 4.1 cm metastatic hilar lymph node encompassing the duodenal bulb. Of note, these findings were not visualized on imaging 7 months prior. Tumor markers were normal. EGD showed an extrinsic mass compressing the duodenal bulb with a superficial ulcer. Colonoscopy was normal. Percutaneous biopsy of the porta hepatis lymph node was done showing evidence of malignant melanoma. The patient had an extensive skin evaluation yielding no skin/ocular findings consistent with melanoma, making a diagnosis of MUP. (Figure)
Discussion: GI MUP has rarely been reported in the literature. Diagnosis is typically definitive in the absence of primary cutaneous, ocular, or mucosal melanoma. Melanoma first found in the GI tract requires a thorough investigation to determine the primary lesion. After a GI melanoma is diagnosed, a detailed physical exam of the lymph nodes. nasopharynx, eyes, anus, and skin must be performed. If no primary lesion is discovered, a PET scan should be done to determine if the GI melanoma is primary, metastatic, or of unknown origin.



[3440] **Figure 1.** (A) Axial view of contrast-enhanced CT showing a partially cystic heterogeneous mass in the hilar region encompassing the duodenal bulb measuring 3.5x 9.0x 4.1 cm (Red Arrow). (B) Coronal view showing RUQ mass causing duodenal bulb compression(Red Arrow), as well as partially visualizing LLQ mass (Yellow Arrow.) (C) Sagittal view of multi-lobulated heterogeneously enhancing mass centered in the LLQ involving the small bowel measuring 6.8 x 5.6 x 6.5 cm (Yellow Arrow.)

S3441

Minor Duodenal Papilla Somatostatinoma: A Rare Case Report

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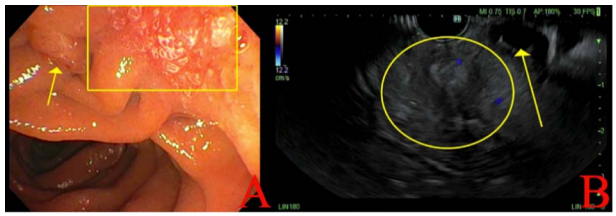
Introduction: Neuroendocrine tumor (NET) of the major duodenal papilla is a rare entity. However, NET of the minor duodenal papilla is even rarer and poorly studied. To our knowledge, only individual case reports and no comprehensive analyses are available in literature. We report a case of a somatostatin secreting NET located on the minor papilla with subsequent management outlined.
Case Description/Methods: A 67-year-old female presented with watery diarrhea accompanied by abdominal pain, dizziness, and lightheadedness. Initial work-up was negative for Clostridium difficile or enteric parasitic infection. Esophagogastroduodenoscopy (EGD) showed a 15mm single duodenal polyp just proximal to the ampulla and potentially involving the minor papilla [Figure 1]. Biopsy of the polyp revealed benign duodenal foveolar metaplasia. CT abdomen and pelvis with IV contrast was obtained due to persistent symptoms and showed an enhancing polypoid lesion at the junction of the second and third portions of the duodenum and near the level of the minor papilla with mild distension of both the common bile duct and pancreatic duct. Endoscopic ultrasound (EUS) with fine-needle biopsy (FNB) of the duodenal polyp at the minor papilla was performed and pathology showed grade I well-differentiated NET. Lab work demonstrated normal 5-HIAA and gastrin levels. Chromogranin A was elevated to 531.5 and measurement of VIP was 44.3. After multidisciplinary discussion, patient underwent Whipple procedure. Post-surgical pathology revealed a well-differentiated 2.8cm NET with Ki-67 less than 3% with negative margins, staining consistent with somatostatinoma. Since surgery, patient's symptoms have resolved.
Discussion: Duodenal NETs are usually located in the first or second portion of the duodenum, with 20% occurring in the periampullary region. Initial biopsies obtained by EGD did not show any evidence of NET, but FNB obtained during EUS did reveal well-differentiated NET. Management of NETs is based on size, location, histological grade, stage, and tumor type. Because of their rarity, there is no consensus on the definitive treatment. There are few case reports of successfully treated NETs endoscopically. Our patient necessitated surgical resection due to its location and potential for lymph node metastasis. EUS with FNB should be pursued in suspected cases of duodenal NET even with negative biopsies from EGD. Further studies are required to establish the optimal method of management of NETs, especially those of the major and minor papilla.



[3441] **Figure 1.** (A) A single 15mm sessile polyp was found at the minor papilla on endoscopy (boxed); the lesion was just proximal to the major papilla (arrow). (B) EUS: Circle showing intramural lesion (NET); arrow showing dorsal pancreatic duct.

S3442

No Endoscopy, No Diagnosis: A Rare Presentation of Multiple Myeloma

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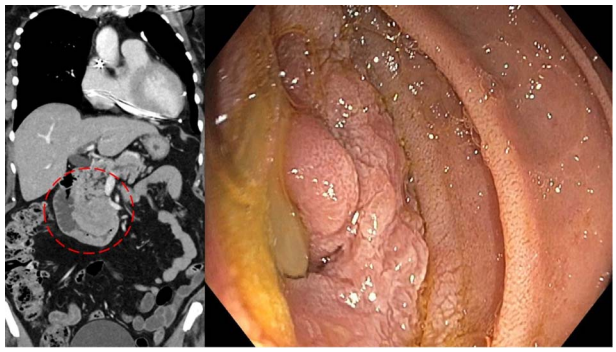
Introduction: Multiple myeloma (MM) is a malignant plasma cell dyscrasia typically found within the bone marrow, resulting in excess clonal serum immunoglobulins and ultimately end-organ dysfunction. Plasmacytomas are extramedullary collections of clonal plasma cell populations, which are only seen in 3.3% of patients at the time of MM diagnosis. Plasmacytomas are most commonly found in soft tissues of the head and neck and extremely rarely in the gastrointestinal system. Herein, we describe a case of MM diagnosed based solely on biopsy proven evidence of a singular vertebral plasmacytoma and a singular duodenal plasmacytoma.
Case Description/Methods: A 73-year-old woman presented with generalized weakness, chronic back pain, increasing, and new onset incontinence. Spinal imaging revealed multiple thoracic pathologic vertebral insufficiency fractures and a homogenous paraspinal tumor resulting in severe spinal canal stenosis. Subsequent abdominal imaging demonstrated a large (6cm) circumferential mass involving the third portion of the duodenum with adjacent retroperitoneal adenopathy. An esophagogastroduodenoscopy was performed, revealing a non-traversable friable mass in the duodenum as the suspect location. Histologic evaluation revealed sheets of plasma cells extending to the duodenal mucosa with notably absent carcinoma. Routine laboratory assessment revealed normal calcium, creatinine, hemoglobin, and protein electrophoresis. However, serum kappa free light chains were elevated and immunofixation revealed IgA monoclonality. Biopsy of the synchronous spinal lesion revealed morphologically and immunophenotypically similar tissue. Interestingly, bone marrow biopsy revealed a paucity of plasma cells. The patient underwent surgical spinal cord decompression and promptly started chemotherapy. (Figure)
Discussion: Gastrointestinal involvement is a rare finding in MM and is almost universally seen after the patient has been diagnosed via conventional criteria. Typically, this diagnosis is made when plasma cells comprise >10% of bone marrow specimen along with typical clinical features. This patient solely demonstrated lytic axial bone lesions with a bone marrow biopsy revealing too few plasma cells to complete immunohistochemical analysis. However, in the presence of >1 plasmacytomas, bone marrow involvement is not essential to make the diagnosis. This case demonstrates a unique instance when endoscopic evaluation was instrumental in the diagnosis of a classically marrow-based hematologic malignancy.



[3442] **Figure 1.** Large ill-defined mass adjacent to the third portion of the duodenum; seen on esophagogastroduodenoscopy as a circumferential, non-traversable and friable lesion, consistent with plasma cell neoplasm on tissue examination.

S3443

Multifocal Duodenal-type Follicular Lymphoma Found Incidentally on Diagnostic Endoscopy

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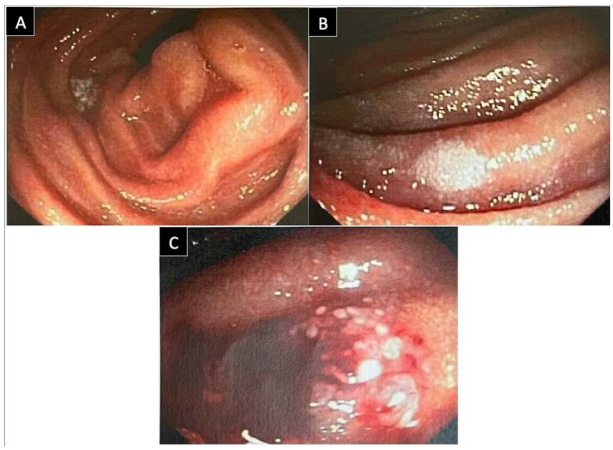
Introduction: Follicular lymphoma (FL) accounts for approximately 4% of gastrointestinal (GI) lymphomas (1). Of the follicular lymphomas found in the GI tract, duodenal-type follicular lymphoma (D-FL) make up 38-81% of cases (2-4). D-FL is often incidentally discovered, lack invasion into deeper layers of the GI tract, and are indolent in nature (5). The clinical stage at time of diagnosis often predicts probability of disease progression (6). Given the excellent prognosis and rare occurrence of progression to high stage FL or transformation to diffuse large B cell lymphoma, a "wait and see" therapeutic approach is often taken (4,7). We report a case of D-FL here.
Case Description/Methods: A 39-year-old female with no pertinent past medical history presented with refractory acid reflux. Her endoscopy was notable for gastritis, duodenal atrophy, and three 5-mm pale nodules in the second part of the duodenum (Figure 1A-1C). Initial biopsy of these polyps showed atypical lymphoid follicular aggregates. Outside pathologists' subsequent review noted atypical lymphoid cells positive for CD10, BCL-2, BCL-5, and CD20 markers, consistent with duodenal follicular lymphoma. Further workup with small bowel enteroscopy to the mid-jejunum and colonoscopy were normal. LDH, SPEP with immunofixation, HIV, hepatitis serologies, and full body CT were unremarkable. The patient was diagnosed with stage I D-FL per the Lugano staging system for gastrointestinal lymphomas, and a "wait and wait" approach was recommended by oncology.
Discussion: D-FL typically presents with multiple low-stage, white nodules in the second portion of the duodenum, as in our case. Because lesions are rarely solitary in 78-85% D-FL cases, thorough small bowel assessment with enteroscopy or capsule endoscopy is warranted (3,4). Radiation has been shown to be effective in limited disease and is the most common form of treatment (8-10). Chemotherapy, Rituximab, and surgery can also used as monotherapy or in combination therapy (4). However, the "watch and wait" approach has been found to be equally effective and is the most common approach amongst oncologists in the United States (11).



[3443] **Figure 1.** Endoscopic images of the duodenal second portion. Three distinct 5-mm pale nodules were seen and biopsies. First, second, and third lesions are seen in figure 1A, 1B, and 1C, respectively.

S3444

Not All That Fistulizes Is Crohn's Disease – Ischemic Jejunocolic Fistula

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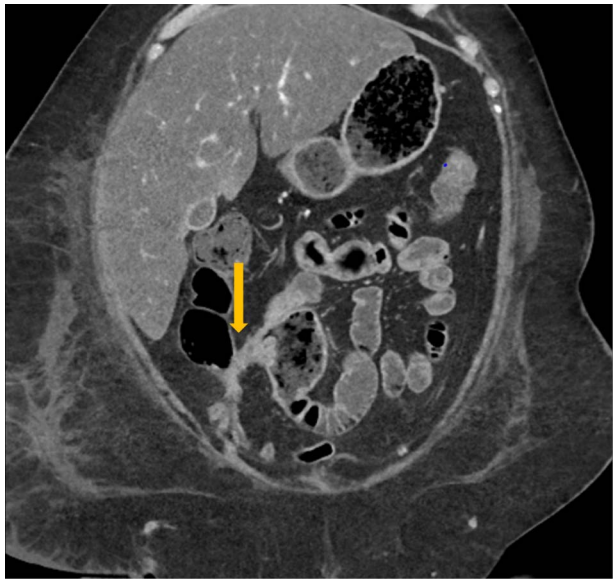
Introduction: Enterocolic fistulae lead to abnormal diversion of gastrointestinal contents from the small intestine to the colon, causing diarrhea and protein energy malnutrition. We describe a rare case of a jejunocolic fistula due to chronic mesenteric ischemia leading to severe chronic diarrhea.
Case Description/Methods: A 40-year-old female with morbid obesity, nicotine dependence, type 2 diabetes mellitus was evaluated for epigastric pain and chronic diarrhea. She also had sitophobia and post-prandial urgency, with weight loss of 70 pounds over the last 6 months. She had been diagnosed with extensive thrombosis involving the abdominal aorta with extensions into the celiac artery, common hepatic artery, and left gastric artery, with evaluation revealing a JAK2 V617F mutant disorder. She was diagnosed with chronic mesenteric ischemia. For the evaluation of her diarrhea, she underwent an EGD with gastric and small bowel biopsies that was normal. CT enterography demonstrated an entero-colic fistula (jejunum to cecum). She underwent a colonoscopy which confirmed a 4 mm fistula in the cecum. Random colonic biopsies were normal, and biopsies from the fistula showed active chronic inflammation. Patient also has elevated calprotectin to 2600. Overall evaluation favored this isolated fistula in the setting of complex atherothrombotic disease to be from an ischemic origin rather than related to Crohn's disease. The patient is currently being nutritionally optimized with plans for a surgical fistula takedown in the future. (Figure)
Discussion: Enterocolic fistulae are complex conditions associated with high morbidity and mortality rates. Most fistulae occur following abdominal surgery, in the setting of infection or breakdown of an intestinal anastomosis due to ischemia, tension, or distal obstruction. Spontaneous fistula formation is uncommon (15-20% of cases), and causes include inflammatory bowel disease (specifically Crohn's disease), diverticular disease, cancer, radiation enteritis and pancreatitis. Chronic ischemia is a very rare cause of spontaneous intestinal fistulization, with scant case reports described in literature. Enterocolic fistula can present with pain, diarrhea, and weight loss, and can be complicated by protein-energy malnutrition, abscesses, and sepsis. Management of the fistula depends on the underlying etiology, with importance given to nutritional optimization. Surgical takedown or endoscopic closure of fistulae remain a viable option in patients with localized disease.



[3444] **Figure 1.** CT enterography demonstrating a jejunocolic fistula (yellow arrow).

S3445

Microscopic Enteritis: A Rare Manifestation of T-cell Promyelocytic Leukemia

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Introduction: An abnormal infiltration of intraepithelial lymphocytes in the intestinal mucosa is described as microscopic enteritis (ME). ME is a heterogeneous condition that can be found in, but not limited to, celiac disease, autoimmune disorders, food protein intolerance, parasitic infections, and NSAID use. Thus, an obvious cause of ME is difficult to elucidate and requires thorough work up. We report a case of a patient who was found to have ME thought to be secondary to infiltrative T-cell promyelocytic leukemia (T-PLL).
Case Description/Methods: A 68-year-old male with a history of type II diabetes and hyperlipidemia presented with intermittent fever, abdominal pain, nausea, and emesis. He was found to have small bowel obstruction with severe enteritis of 3/4 of the small bowel (distal jejunum to the ileocecal valve). He had a small bowel resection with pathology revealing necrotizing lymphocytic enteritis. Bidirectional endoscopy revealed mild colonic, ileal and duodenal edema with biopsies revealing increased intraepithelial lymphocytes. He was empirically treated with intravenous methylprednisolone with resolution of fevers. He was discharged home with a follow up with gastroenterology for ME and with hematology for persistent leukocytosis. He continued to have recurrent fevers with persistent lymphocytic leukocytosis (14,200 WBC/microliter). Infectious (acid fast bacteria, fungi, cytomegalovirus, adenovirus, syphilis) and rheumatologic diagnostics were unrevealing. He underwent bone marrow biopsy and peripheral blood examination with immunostains, flow cytometry, and cytogenetic studies and was found to have clonal T-cell receptor gamma gene rearrangement concerning for T-PLL. He was treated with alemtuzumab; however developed worsening disease and disseminating opportunistic infections (cytomegalovirus and aspergillosis). He passed away shortly after his diagnosis.
Discussion: ME is a challenging diagnosis as its etiology can be difficult to elucidate. Prompt, timely evaluation should be prioritized to improve outcomes. T-PLL is a rare leukemia due to abnormal growth of T-lymphocytes and typically presents with leukocytosis, weight loss, and infection; however, gastrointestinal symptoms may be a presenting symptom.

S3446

Monomorphic Epitheliotropic Intestinal T-Cell Lymphoma: A Tough Battle to Win!!

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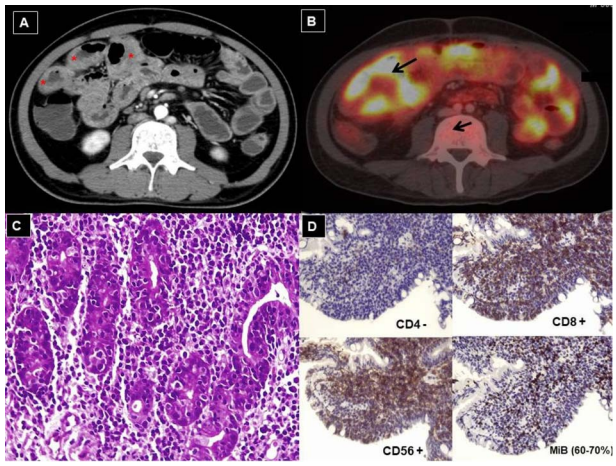
[1]VPS Lakeshore Hospital and Research Centre, Kochi, Kerala, India; [2]VPS Lakeshore Hospital, Kochi, Kerala, India; [3]Amrita Hospital, Kochi, Kerala, India.

Introduction: Monomorphic epitheliotropic intestinal T-cell lymphoma (MEITL), previously known as type 2 enteropathy-associated T-cell lymphoma (EATL), is an extremely rare and rapidly progressive type of non-Hodgkin's lymphoma without an established targeted therapy at present. Our purpose is to report the diagnostic and therapeutic challenges we faced in treating this condition associated with high mortality.
Case Description/Methods: A 36-year-old gentleman presented to us with a history of chronic small bowel type of diarrhoea for 3 months associated with significant weight loss and high-grade fever. Baseline investigations including the stool examination were normal except for elevated C-reactive protein. Since oesophagogastroduodenoscopy (OGD) and colonoscopy showed normal mucosal patterns, CT enterography was done to evaluate the small bowel which revealed jejunal thickening and mesenteric lymphadenopathy. Single balloon enteroscopy showed multiple ulcerations in the proximal and mid jejunum involving more than 50% of the circumference with necrotic base. PET CT demonstrated significant diffuse uptakes in proximal jejunum, spleen, paraaortic lymph nodes, and axial skeleton. Biopsy from jejunal ulcers revealed surface erosions and distorted crypt-villous architecture with dense infiltration by the monotonous population of medium-sized lymphoid cells with scanty cytoplasm and round hyperchromatic nuclei. Multiple atypical lymphoid cells and scattered mitotic figures were seen. These atypical lymphoid cells were positive for CD3, bcl2, CD8, and CD56 with a high MIB index (60 - 70%) and were negative for CD4 and CD30. No CD20 positive B cells were seen among the infiltrating cells. Immunohistochemistry (IHC) was consistent with MEITL. Tissue transglutaminase IgA antibody test was done to screen for celiac disease which was negative. The patient is on chemotherapy (CHOP Regimen) but with an unsatisfactory response at present (Figure).
Discussion: MEITL is a rare type of primary T cell lymphoma with an aggressive clinical course often presenting at an advanced stage of the disease. MEITL was reclassified from EATL in the 2016 WHO classification because of the lack of association with celiac disease. Small bowel evaluation and biopsy with IHC are the cornerstones of the diagnosis. Though there is no established therapy yet, the literature on stem cell transplantation has shown promising results.



[3446] **Figure 1.** Radiology and Histopathology images of a 36-year-old male diagnosed with MEITL. A) CT enterography showing diffuse jejunal wall thickening (Red asterisk). B) PET CT showing diffuse uptake in the jejunum and axial skeleton. (Black arrow). C) Biopsy from the jejunal ulcers demonstrating the epitheliotropism, i.e. Atypical lymphoid cells infiltrating the crypt epithelium. Immunohistochemistry reveals these cells are negative for CD4 but positive for CD8 and CD56 markers with Mib index of 60 to 70% as shown in D.

S3447

An Unusual Case of Diarrhea in a Patient With Lupus

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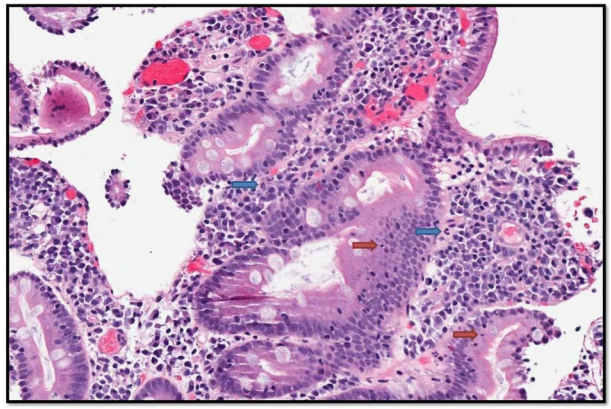
Introduction: The differential diagnosis of diarrhea in the setting of systemic lupus erythematosus (SLE) is broad and includes lupus-related as well as non-lupus-related etiologies. Lupus-related etiologies include but are not limited to mesenteric vasculitis, pseudo-obstruction, protein-losing enteropathy, and pancreatitis. Autoimmune enteropathy (AIE) is rare in adults, with fewer than fifty cases described in the medical literature. We report a case that responded effectively to Belimumab.
Case Description/Methods: A 31-year-old woman presented with intermittent ongoing diarrhea of six months duration. Workup for infectious causes included stool for ova and parasites, stool cultures, and C. difficile toxin, all of which were negative. Celiac disease antibody testing was negative. CT angiography of the abdomen revealed patent vasculature, devoid of stenosis or occlusion of the celiac artery, superior mesenteric artery, or inferior mesenteric artery. No evidence of inflammatory bowel disease (IBD) or microscopic colitis was seen on biopsies from the colon. Upper gastrointestinal endoscopy was performed. Duodenal biopsy illustrated cryptitis, glandular apoptosis and increased plasma cells in the lamina propria consistent with severe chronic duodenitis (Figure). A diagnosis of autoimmune enteropathy was presumed and she was initiated on treatment with oral steroids. As an outpatient, she was transitioned to Belimumab, a monoclonal antibody that inhibits B cell-activating factor. She remained in sustained remission on follow-up. The response to immunosuppression further supported our diagnosis of AIE.
Discussion: The dearth of adult case reports, and the non-specificity and heterogeneity of clinicopathologic findings make the diagnosis of AIE challenging. Diagnosis requires persistent diarrhea for more than six weeks, malabsorption, distinctive small intestinal histology, and exclusion of alternative causes of villous blunting. The damage is typically confined to the duodenum. Histological patterns can be classified as active chronic duodenitis, celiac disease-like, graft vs host disease-like, and mixed/no predominant pattern. Nutritional optimization and immunosuppression are the cornerstones of treatment. Treatment is

often initiated with steroids. Alternative drugs include 6-mercaptopurine, azathioprine, cyclosporine, and tacrolimus. Adalimumab, Vedolizumab, and Ustekinumab have also been successfully used. To our knowledge, this is the first case of AIE successfully treated with Belimumab.



[3447] **Figure 1.** A higher magnification image of the duodenal biopsy reveals neutrophils (blue arrows) within the lamina propria and apoptotic bodies in the glands, indicating active inflammation and glandular injury. There is also an increase in the plasma cells within the lamina propria, indicating chronicity. This pattern of inflammation is consistent with chronic enteritis. Though this is nonspecific, autoimmune enteropathy is within the differential diagnosis in the appropriate clinical context. (20x).

S3448

Amyloidosis Diagnosed by the GI Doctor

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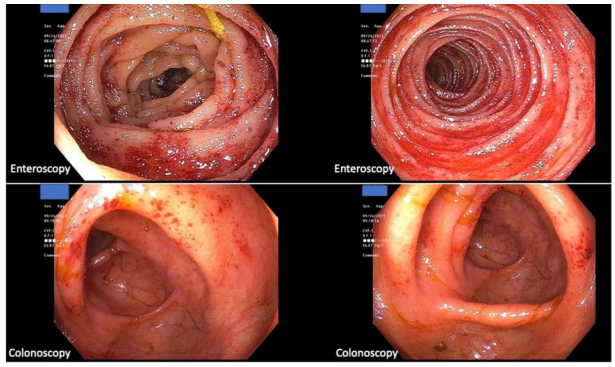
Introduction: Amyloid light-chain (AL) amyloidosis is the most common form of amyloidosis and is the main type to involve the gastrointestinal tract. Monoclonal Gammopathy of Undetermined Significance (MGUS) is a Plasma Cell Dyscrasia (PCD) with potential to precipitate amyloidosis. Our patient presented with a constellation of signs and symptoms that lead to his diagnosis.
Case Description/Methods: A 59-year-old man with a history of Hemoglobin S-C presented with recurrent admissions to our hospital for new-onset orthostasis, weight loss, and dyspnea progressing over a 9 month period. Physical exam was unimpressive at the first presentation. Labs were significant for a NT-Pro BNP of 17,100 pg/ml. Echocardiography and cardiac MRI showed a restrictive filling pattern with preserved ejection fraction, however a specific etiology was not identified. Serum immunofixation revealed monoclonal IgA light chain proliferation. Bone marrow biopsy confirmed MGUS. The patient later developed epigastric pain with associated chronic watery diarrhea, peripheral edema, and hypoalbuminemia. A CT demonstrated colonic wall thickening with trace abdominal ascites. Enteroscopy revealed severe inflammation of the duodenum and jejunum characterized by edema, friability, scalloping, and diffuse petechiae. Colonoscopy showed similar but patchy involvement throughout. Histology was initially inconclusive. However, our suspicion remained high so slides were sent to a GI specialty lab. Pathology ultimately demonstrated immunoglobulin light chain lambda amyloidosis. He developed nephrosis as well. A work-up for autoimmune, infectious, and malignant causes was negative. The patient was ultimately transferred to a nearby hospital with specialty care services. (Figure)
Discussion: AL amyloidosis develops as a result of monoclonal light chain deposition in various tissues. The cardiac and renal systems are most commonly involved. The main causes include PCDs and autoimmune illnesses. The diagnosis is often delayed. It should be suspected in a patient who presents with restrictive cardiomyopathy or otherwise unexplained heart failure. Our patient's illness manifested as restrictive cardiomyopathy and likely protein losing enteropathy. MGUS can precede amyloidosis by about 12 years so this was an important finding. Initial GI biopsies were inconclusive but ultimately cinched the diagnosis, highlighting the importance of remaining skeptical. Treatment is aimed at the underlying cause.



[3448] **Figure 1.** Endoscopy Images.

S3449

Primary Enterolith: A Rare Cause of Chronic Intestinal Obstruction

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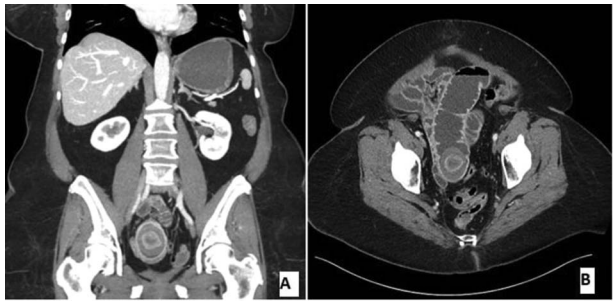
Introduction: Primary enterolithiasis or the formation of gastrointestinal concretions is an uncommon condition occurring in 0.3-10% of people. It is promoted by factors that cause intestinal stasis such as bowel kinking with intra-abdominal adhesion, blind intestinal loop/pouch, intestinal stenosis/strictures due to Crohn's disease, radiation enteritis and intestinal tumor. Complications of enterolithiasis include bowel obstruction, intestinal gangrene, intussusceptions, intestinal hemorrhage and perforation. Enterolithiasis causes mortality in about 3% of cases. While acute intestinal obstruction is described by a few cases, chronic obstruction has rarely been reported. Here, we describe a case of chronic partial bowel obstruction secondary to a large primary enterolith.
Case Description/Methods: A 71-year-old woman presented to gastroenterology clinic for chronic and recurrent abdominal pain. Her past medical history was significant for endometrial cancer for which she underwent total abdominal hysterectomy and chemo-radiation 10 years prior. She developed post-prandial nausea, vomiting and abdominal pain 5 years ago. The pain was initially thought to be from biliary colic, but cholecystectomy did not provide relief. During the last 6 months, she experienced increasing frequency of stereotypical abdominal pain episodes with nausea and vomiting, Physical exam demonstrated a benign abdomen without tenderness or distention. CT enterography was performed, and the coronal (Figure A) and axial (Figure B) images demonstrated a large lamellated intraluminal mass in the mid small

bowel and dilated loops of small bowel upstream of the mass. The patient was diagnosed with primary enterolithiasis proximal to a radiation induced stricture, causing chronic recurrent partial small bowel obstruction. Two weeks later, she underwent elective robotic-assisted small bowel resection with the retrieval of a brown, green enterolith measuring 5.5x3.8x3.5 cm. The patient was discharged on post-operative day 3 without complications.

Discussion: Primary enterolithiasis should be considered as a rare etiology of chronic abdominal pain and bowel obstruction in patients with pre-disposing factors that promote intestinal stasis. Treatment depends on the timely recognition of this entity and endoscopic or surgical management. With the technological advances, better patient outcomes as well mortality rates are expected.



[3449] **Figure 1.** CT enterography showing a large lamellated intraluminal mass in the mid small bowel A) Coronal view, B) Axial view.

S3450

Atypical Endoscopic and Clinical Presentation of Strongyloidiasis

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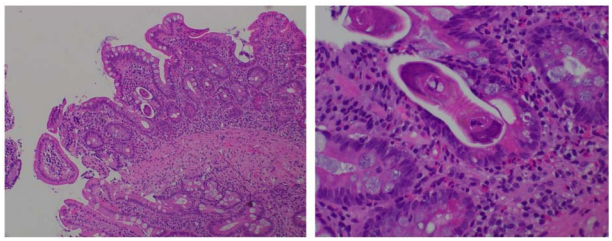
Introduction: Strongyloidiasis is a helminthic parasitic infection caused by the nematode Strongyloides stercoralis. Due to its replication cycle within the human host, acute infection classically presents sequentially with skin irritation, dry cough, and then non-specific gastrointestinal (GI) symptoms. Mild GI and dermatologic symptoms appear to be more prevalent in chronic infections. Here we present a case of strongyloidiasis manifesting in atypical symptoms with normal endoscopic findings.

Case Description/Methods: An 81-year-old female with a medical history of diverticulosis, hypertension, type 2 diabetes mellitus, hypercholesterolemia, and chronic kidney disease was admitted to the hospital for anorexia, abdominal pain, and unintentional 26-pound weight loss over 6 months. Computed tomography of chest, abdomen, and pelvis revealed a moderate left-sided pleural effusion and uncomplicated sigmoid diverticulitis. Pleural fluid studies showed exudative features, with negative cultures and cytology. She was started on ciprofloxacin and metronidazole for diverticulitis. The patient also had a chronic iron deficiency anemia with an average hemoglobin of 7.4 g/dL. Absolute eosinophil count, total IgE, sedimentation rate, and c-reactive protein were elevated. Enteroscopy up to the mid jejunum appeared normal except for a 4 cm hiatal hernia. Random duodenal biopsies showed chronic active duodenitis with several eosinophils and parasitic organisms (Figure). Colonoscopy was deferred due to acute diverticulitis. Strongyloides serum antibodies were positive. Stool studies were not analyzed due to lab errors. The patient was treated with oral ivermectin 200 mcg/kg/day for 14 days due to concern for disseminated infection and scheduled for an outpatient endoscopy and colonoscopy.

Discussion: This case highlights the need to keep parasitic infections on the differential when patients present with symptoms concerning for malignancy, such as weight loss, anemia, abnormal laboratory markers, and new pleural effusions. While *S. stercoralis* has been found to mimic inflammatory bowel disease, it has not been associated with diverticulitis thus far. In addition, typical endoscopic findings include edematous and erythematous mucosa with white villi, however, this patient had normal endoscopic findings despite the extent of her infection. The duodenal biopsies aided in making the diagnosis for this case and should be considered in patients with similar presentations.



[3450] **Figure 1.** Duodenal biopsies showing chronic active duodenitis with several eosinophils and parasitic organisms.

S3451

Work-Up of Hemoptysis and Interstitial Lung Disease Leading to the Diagnosis of Celiac Disease

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Introduction: Hemoptysis is the coughing up of blood that originates in the bronchial tree. Hemoptysis has a variety of etiologies including infections, vasculitides, malignancy, trauma and many others. The workup can be broad and should be guided by the clinical history and physical exam. We present a case of a patient in whom workup for hemoptysis and interstitial lung disease led to the diagnosis of celiac disease.

Case Description/Methods: Patient is a 47-year-old male with history of interstitial lung disease, emphysema, and chronic tobacco use who presented due to hemoptysis. Inflammatory markers and autoimmune workup were ordered and revealed positive celiac serologies. Patient had no GI complaints aside from occasional reflux and denied diarrhea. He underwent EGD which showed erythematous mucosa in the antrum, erythematous duodenopathy, and mucosal changes concerning for Barrett's esophagus. Histologic examination of the biopsies of the esophagus, stomach and duodenum showed Barrett's esophagus with no dysplasia, positive H. Pylori staining, and duodenal mucosa with mild villous blunting and increased intraepithelial lymphocytes. Due to hemoptysis and celiac disease, there was concern for Lane-Hamilton Syndrome. The patient underwent bronchoscopy with bronchoalveolar lavage (BAL) which was negative for hemosiderin. CT imaging of the chest showed worsening of his known interstitial lung disease with no acute abnormalities. He was placed on a gluten free diet which can result in improvement in both gastrointestinal and pulmonary symptoms.

Discussion: Celiac disease should be suspected in patients with symptoms of diarrhea and bloating which are worse when eating gluten-containing foods. Lane-Hamilton syndrome is a rare disorder in which pulmonary hemosiderosis coexists with celiac disease. Our patient has a history of interstitial lung disease and was diagnosed with celiac disease during the workup of his hemoptysis, raising suspicion for Lane-Hamilton syndrome. Although our patient's bronchoscopic evaluation was negative for pulmonary hemosiderosis, there have been reports of a possible association between interstitial lung disease and celiac disease in the absence of pulmonary hemosiderosis. Our patient will be followed closely to ensure resolution or improvement in pulmonary symptoms following treatment of his celiac disease.

S3452

Missed Perforated Appendicitis Presenting as Flank Necrotizing Fasciitis - A Rare Phenomenon

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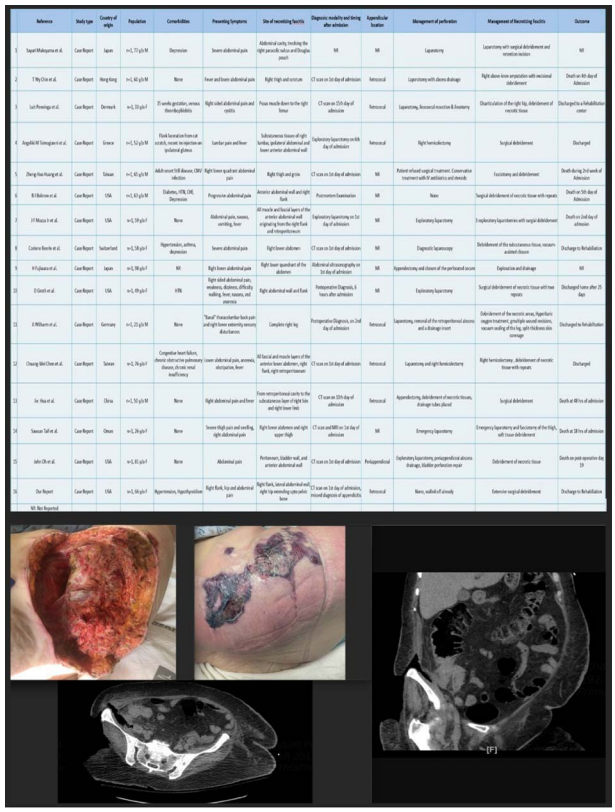
Introduction: Acute appendicitis is one of the most common surgical condition. Necrotizing fasciitis is a rare infection of the deeper layers of skin and subcutaneous tissues, rapidly spreading across fascial planes within the subcutaneous tissue with an average mortality rate of 20.6% and is a surgical emergency. Necrotizing fasciitis due to perforated appendicitis is even rarer.
Case Description/Methods: We present a case of necrotizing fasciitis of abdominal wall and right flank secondary to a perforated appendix and reviewed the available literature. Confusion with cellulitis, can delay aggressive therapy. A 66-year-old female presented with severe right flank pain following 2-weeks history of right abdominal pain. She had tachycardia and hypotension. On physical examination, she had a large necrotic wound in right flank with pus and blistering, and abdomen had no peritoneal signs. Her laboratory investigations revealed white cell count of 35,500/mm3. Non-contrast CT (patient was allergic to contrasts) showed features of necrotizing fasciitis in the flank with inflamed and walled-off cecum and appendix. Antibiotics were started and emergent surgery revealed grossly necrotic tissue with multiple pockets of pus in subcutaneous tissue, fascia, and muscles of flank and hip extending to retroperitoneum along with multiple pelvic abscesses. Due to walling-off, disseminated retroperitoneal and pelvic infection had no further intraperitoneal connection. Her wound cultures grew Bacteroides, E.Coli and Aerococcus. Patient was discharged once stable, on wound vac and extended antibiotic regimen. (Figure)
Discussion: Necrotizing fasciitis due to perforated appendix is rarely reported. Our literature review showed that only 16 cases (including this) have been reported with calculated mortality rate of 46.15% (3 cases did not report outcome). Retrocecal appendiceal location is mostly seen to be present. In our patient, the perforated appendix ruptured through peritoneum into retroperitoneal space and drained out into lateral abdominal wall through superior and inferior lumbar triangles (areas of relative weakness) causing extensive necrotizing fasciitis of this region with multiple abscess formation. This case illustrates the importance of early diagnosis of disease, progression, and prompt surgical intervention and why should we be vigilant for clues of a missed appendicitis. We should consider intra-abdominal pathologies in determining cause of necrotizing fasciitis presenting over abdomen or flank.



[3452] **Figure 1.** Review of literature of all the available worldwide cases of missed appendicitis presenting as necrotizing fasciitis, along with postoperative images of necrotizing fasciitis of flank and CT imaging of the same during patient's admission.

S3453

Small Bowel Diverticulosis and Malrotation: A New Spin on SIBO

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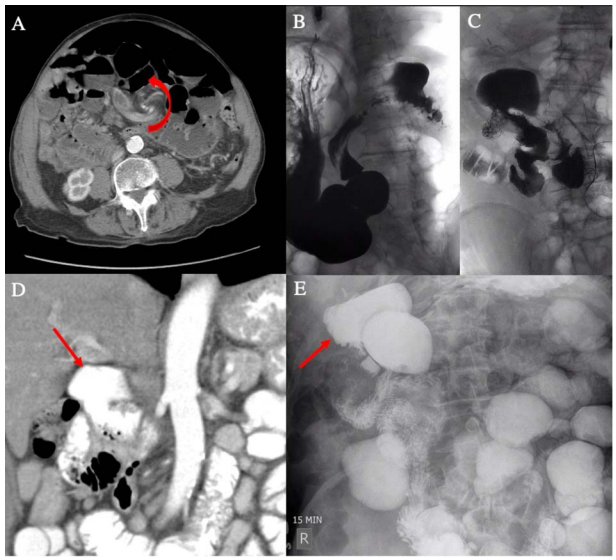
Walter Reed National Military Medical Center, Bethesda, MD.

Introduction: Small bowel diverticula are far less common than colonic diverticulosis. Formative mechanisms, including abnormal peristaltic activity and high intraluminal pressures, are shared between both varieties. Small intestinal bacterial overgrowth (SIBO) is defined by an abnormal abundance of bacteria in the small bowel, typically associated with abdominal pain, bloating, and diarrhea. Small bowel diverticula can predispose for SIBO. The constellation of symptoms is commonly attributed to or misdiagnosed as functional bowel syndromes. When appropriately recognized and treated, symptoms associated with bacterial overgrowth can be exquisitely responsive to antibiotic therapy.
Case Description/Methods: Our case involves a 93-year-old man with over ten years of chronic abdominal bloating, distension, watery stools, and intermittent, focal postprandial epigastric pain. Computed tomography imaging was significant for evidence of malrotation and focal caliber change within the small intestine (Figure A, D). Real-time fluoroscopic exam of barium traversing the duodenum revealed preferential filling of the large duodenal diverticulum rather than the true duodenal lumen (B, C). An upper gastrointestinal series revealed a corkscrew appearance of the duodenum, a 6.3-centimeter proximal post-bulbar duodenal diverticulum, and multiple jejunal and ileal large multi-centimeter diverticula (E). The collection of small bowel diverticular disease was postulated to cause a secondary bacterial overgrowth and focal epigastric discomfort given the degree of contrast stasis. He started a 10-day trial of rifaximin with prompt and dramatic clinical improvement. He experienced recrudescence of symptoms every few months and was managed with courses of rifaximin with good symptomatic control. The patient was also responsive to a full liquid diet aimed to reduce luminal distension.
Discussion: Our patient's anatomy and symptomatology suggest a linear causality that started with anatomic malrotation leading to intermittent increases in luminal pressures. This predisposed him for small bowel diverticula formation, which ultimately lead to stasis and a nidus for SIBO. There are isolated case reports of an association between malrotation and extensive small bowel diverticulosis. We present this case to alert physicians that "incidentally" found malrotation may be associated with symptomatic physiologic sequelae including SIBO.



[3453] **Figure 1.** A - Axial computed tomography (CT) of the abdomen with intravenous contrast demonstrating splanchnic swirling. B - Real-time barium swallow with view of the esophagus, stomach, pylorus, and proximal duodenum with large diverticulum. C - Oblique view of large proximal duodenal diverticulum with preferential filling and contrast stasis. D - Coronal CT of the abdomen with oral contrast with large proximal duodenal diverticulum. E - Abdominal x-ray with delayed oral contrast medium showing innumerable small bowel diverticula.

S3454

Endoscopic Management of Bouveret's Syndrome in a Comorbid Patient

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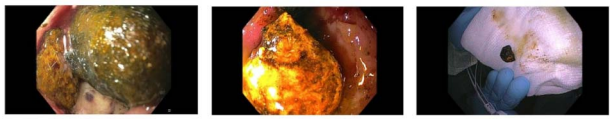
Introduction: Bouveret's syndrome is a gastric outlet obstruction (GOO) caused by an impacted gallstone in the duodenum or stomach via a bilioenteric fistula. The treatment is primarily surgical, but endoscopic therapy may be the only option for patients that are non-surgical candidates.
Case Description/Methods: A 78-year-old man presented with hematemesis and CT scan suggesting cholecystoenteric fistula leading to GOO. Due to multiple comorbidities, he was deemed a non-surgical candidate. Esophagogastroduodenoscopy (EGD) showed a large Forrest Class IIb ulcer in the duodenal bulb that was not amenable to endoscopic therapy. The patient then underwent endovascular embolization of the gastroduodenal and supraduodenal arteries. On day five, a second EGD showed a 3 cm gallstone impacted in the duodenal bulb. Under the gallstone was a partially obstructing Forrest Class IIc ulcer. Unsuccessful removal of the gallstone was attempted using a mechanical lithotripter basket and Roth net. On day ten, the gallstone was removed endoscopically using electrohydraulic lithotripsy (EHL) at a pulse rate of 10 and medium power setting. One stone fragment (~1 cm) remained, obscuring the lumen distal to the duodenal bulb. During the fourth EGD on day 14, the remaining gallstone was fragmented with rat-tooth forceps and lithotripsy basket. After balloon sweep of the presumed cholecystoduodenal fistula tract, contrast injected into the tract was seen draining into the duodenal bulb, precluding full fluoroscopic assessment of the fistula. The patient was discharged to a skilled nursing facility on day eighteen. (Figure)
Discussion: Bouveret's syndrome is a rare complication of cholelithiasis. Most common symptoms include epigastric pain, nausea, and vomiting. Patients can also present with non-specific signs, such as gastrointestinal bleeding, as did our patient. When surgical candidacy is limited by comorbid conditions, endoscopy is the preferred and possibly only therapeutic option. Gallstone removal may be done endoscopically, with mechanical, electrohydraulic, or laser lithotripsy. For impacted stones, mechanical fragmentation can be accomplished by using a basket, snare, forceps, mechanical lithotripsy, or EHL prior to extraction and removal. All stone fragments should be removed after mechanical fragmentation to avoid complications like gallstone ileus. In our case EHL was followed by mechanical fragmentation to accomplish complete fragmentation and removal.



[3454] **Figure 1.** Caption 1 is showing duodenal bulb gallstone and ulcer. Caption 2 is showing gallstone fragment at the duodenal bulb after using EHL. Caption 3 is showing the biggest gallstone fragment in the endoscopist hand.

S3455

Unusual Presentation of a Young Male Diagnosed With a Perforated Gastrointestinal Stromal Tumor (GIST) in the Jejunum With a Previous History of Multiple Myeloma (MM)

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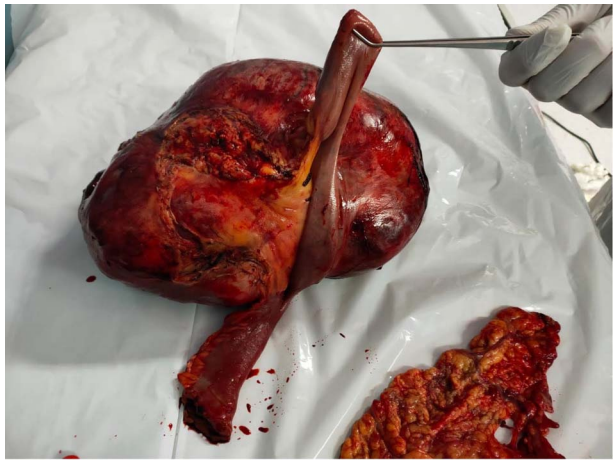
Introduction: Studies suggest an increase in the frequency of additional malignancies in patients with GIST. This relationship remains unclear and needs further exploration. We present a case of GIST in a patient with Multiple Myeloma.
Case Description/Methods: A previously healthy 38 years old male presented in 2016 complaining of weakness, fatigue, and back pain; he was diagnosed with Multiple Myeloma. Remission was achieved using chemotherapy and autologous bone marrow transplantation. He had a relapse of Multiple Myeloma on a bone marrow biopsy in 2020. Since then, he has been receiving chemotherapy. In March 2022, he presented to the ER complaining of abdominal distension, severe pain, and vomiting, based on a physical exam and CT. Scan, he was diagnosed with small bowel obstruction and peritonitis. Surgical exploration revealed a large abdominal perforated mass originated from the proximal jejunum, with adhesions to the root of the mesentery, urinary bladder, small bowel, transverse, descending, and sigmoid colons. Excision of the mass with resection of about 30 cm and re-anastomosis of the jejunum was performed. Postoperatively, the patient was admitted to the ICU for three days to control cardiac arrhythmia. He was discharged from the hospital ten days later. Pathology confirmed the diagnosis of GIST. Currently, the patient is receiving chemotherapy and doing well (Figure).
Discussion: Despite being the most common mesenchymal neoplasms of the gastrointestinal tract, GIST are still rare tumors that account for less than 1% of all GI tumors; they occur mostly in the stomach or small intestines. The average age of diagnosis is 50-70 years and is considered non-hereditary or sporadic. There are reports of co-existing GIST with adenocarcinomas within the GI tract or distant organs. In a single case series of 43 patients with GIST, 6 (14%) had an additional primary tumor, five within the GI tract. In a larger population-based study with 6,112 GIST patients, 1,047 (17.1) had additional cancers.

Despite that, so far, only one case of (MM) was reported in a patient with GIST in 2007 in Greece, which ended with the patient's death six months after the diagnosis of (MM). This case highlights the relationship between GIST and other primary neoplasms at a young age. It is unclear whether the GIST was due to genetic association, environmental exposure, or chemotherapy. Clinicians should look for tumors within or outside the GI tract, which could significantly affect the prognosis.



[3455] **Figure 1.** Resected GIST from with 30 Cm of the jejunum.

S3456

Olmesartan-Induced Enteritis: When Symptoms Do Not Improve With Drug Discontinuation

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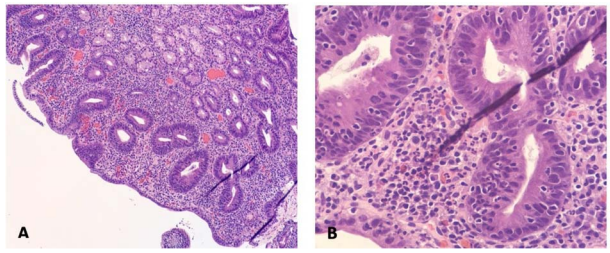
Introduction: Severe diarrhea is a well-known, but rare, complication of Olmesartan. The enteropathy caused by Olmesartan is histologically similar to Celiac disease, however celiac serologies are negative and it lacks a response to a gluten free diet. In most cases, symptoms resolve with the discontinuation of the medication. We present a case of Olmesartan induced enteropathy that only improved after the initiation of budesonide.
Case Description/Methods: A 73-year-old female with a history of hypertension presented with complaints of nausea, vomiting and watery diarrhea. Initial workup with bloodwork, stool studies and imaging were all unremarkable. Recent outpatient colonoscopy was unremarkable. Review of her medications did not reveal any typical culprits of microscopic colitis. Given persistent nausea and vomiting, an upper endoscopy was pursued. Celiac serologic testing, including issue transglutaminase (tTg) IgA and IgG, anti-gliadin IgA and anti-endomysial IgA antibodies were all normal. Qualitative IgA levels were within normal limits. Upper endoscopy was performed with duodenal biopsies revealing moderate active chronic duodenitis with blunting of villous architecture and increased intraepithelial lymphocytic infiltrate, suspicious for celiac sprue. At this point, a closer review of her medications revealed a prescription for Olmesartan. Olmesartan was discontinued, however she continued to have significant diarrhea, up to 2.5L daily, for over a week after discontinuation. Given persistent diarrhea, nausea and vomiting, resulting in significant electrolyte abnormalities, the decision was made to initiate Budesonide. She experienced rapid improvement in symptoms after the initiation of Budesonide and only reported two loose stools per day on discharge. (Figure)
Discussion: The initial workup for chronic diarrhea involves a thorough medication review. Physicians who prescribe Olmesartan should be aware of the enteropathy it can cause, as it is likely more common than expected. Other drugs such as azathioprine, colchicine, mycophenolate, methotrexate and neomycin have also been implicated in a similar enteropathy. Timely diagnosis can help decrease healthcare utilization and patient discomfort. In cases such as ours, the enteropathy can be severe enough to result in hospital admission. Immunosuppressant's such as steroids or azathioprine have shown great efficacy in treatment of these refractory cases.



[3456] **Figure 1.** Histology of duodenal biopsy showing (A) Moderate active chronic duodenitis with blunting of villous architecture and (B) Increased intraepithelial lymphocytic infiltrate.

S3457

Perforated Jejunal Diverticulitis With a Coloenteric Fistula Misdiagnosed as Gastroenteritis

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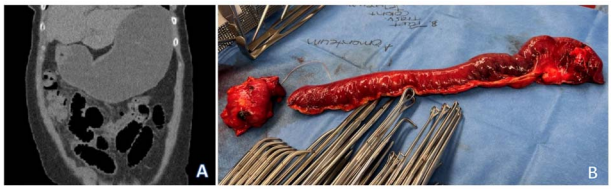
Introduction: Diverticular disease is one of the most common issues encountered by gastroenterologists. It is estimated that around one half of those above the age of 60 will have colonic diverticula and up to 25% of these individuals will experience a complication such as diverticulitis. In the small intestine, a majority of diverticula are found in the duodenum (79%). Diverticula of the jejunum and ileum are rather rare, affecting only 0.5%-2.3% of patients. In those with jejunoileal diverticula, their clinical course is largely asymptomatic but 10% of the time patients can develop complications. Here we present an interesting case of perforated jejunal diverticulitis with a coloenteric fistula that was misdiagnosed as gastroenteritis.
Case Description/Methods: A 74-year-old female presented to the hospital for 3 weeks of abdominal pain, diarrhea and fevers. Prior to this, she presented to an outside hospital on two separate occasions for her symptoms. During these admissions, she was told she had gastroenteritis and was subsequently discharged home on antibiotics. Eventually her pain became constant and she developed intractable vomiting therefore she presented to another hospital. Vitals were normal and the physical exam showed moderate tenderness of the epigastric area. White blood cell count was 21,000 uL and lactic acid was 2.1 mmol/L. CT scan without contrast showed nonspecific enteritis. Broad spectrum antibiotics were given. 24 hours later the abdominal pain worsened despite frequent narcotic administration. CT enterography was obtained, showing a 3.7cm x 2.8cm outpouching of the jejunum with significant thickening and stranding, that was inseparable from the transverse colon (Figure A). Exploratory laparotomy revealed ruptured jejunal diverticulitis that was fistulized to the mid transverse colon (Figure B). She then underwent a transverse colectomy and small bowel resection with anastomosis. Her postoperative course was uncomplicated and she was discharged 2 weeks later.

Discussion: Although jejunal diverticulitis may only occur in less than 2% of known diverticulosis, the occurrence can be fatal. Physical examination and the close evaluation of cross-sectional imaging are integral in its diagnosis. It is important to recognize that this disease may have an insidious presentation and may become a diagnostic challenge.



[3457] **Figure 1.** A. Enterography coronal view showing a focal outpouching in the jejunum measuring 3.7 x 2.8 cm, inseparable from adjacent transverse colon. B. Surgical specimen with resected transverse colon (left) and resected segment of jejunum fistulized into the colon (right).

S3458

Often Reported, Rarely Considered: A Case of ACEI-Induced Mesenteric Angioedema

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Introduction: Angiotensin converting enzyme inhibitors (ACEi) are commonly used in the treatment of cardiovascular and renal diseases. Angioedema is a well-known side effect of ACE inhibitors, but isolated visceral angioedema is a rare and under-recognized phenomenon. We present a case of a patient who underwent years of unnecessary and invasive testing to ultimately be diagnosed with ACEi induced mesenteric angioedema.
Case Description/Methods: A 49 year old male with history of type 1 diabetes mellitus and hypertension presented with 5 years of intermittent abdominal pain, nausea, vomiting and diarrhea. Labs were unremarkable including fecal calprotectin, fecal fat, Helicobacter pylori, and tissue transglutaminase IgA (normal total IgA). Multiple endoscopies were normal with negative biopsies. Trials of low FODMAP and lactose free diets as well as fiber, cholestyramine, dicyclomine, doxepin, steroids, mesalamine and antibiotics were all ineffective. Capsule endoscopy showed subtle and superficial erosion in the jejunum. Several computed tomography (CT) scans including CT enterography showed multifocal jejunal wall thickening without evidence of stricture and diffuse mesenteric edema with intermittent small volume of free fluid. Gastric emptying study was normal. HIDA scan revealed reduced gallbladder ejection fraction. During this time patient underwent two surgeries: an exploratory laparotomy showed mild changes of creeping fat in the jejunum, but no definitive evidence of Crohn's disease and cholecystectomy which failed to relieve symptoms. Extensive chart review led to a trial of holding the patient's lisinopril. At this time the patient has had no further abdominal symptoms.
Discussion: Visceral angioedema due to ACEi therapy is often overlooked and can pose a diagnostic challenge. Symptoms are non-specific and include abdominal pain, nausea, vomiting, diarrhea and ascites. Onset can occur days to years after starting an ACEi. Imaging may show segmental small bowel thickening, elongation of bowel loops, or mesenteric edema with no vascular compromise or adenopathy as well as abdominal free fluid without gross ascites. ACEi discontinuation typically leads to resolution of symptoms within 48 hours. This phenomenon occurs more often in females and African Americans. This was a case of a Caucasian male who had an extensive workup, including exploratory surgery, before the ACEi was held. Although this diagnosis is well reported, it is rare, and must be considered to avoid unnecessary tests and procedures.

S3459

Pembrolizumab-Induced Ulcerative Duodenitis Treatment Successfully Treated With Prednisone

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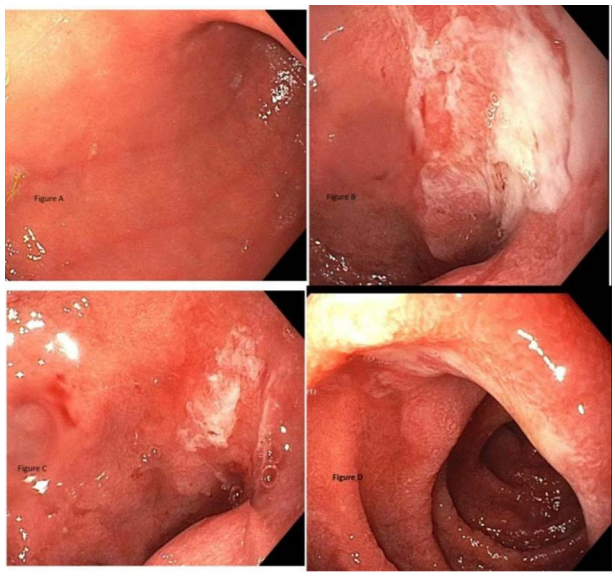
Introduction: Programmed death ligand-1 (PD-L1) inhibitors belong to a category of immune checkpoint inhibitors (ICIs) and have been used to treat melanoma, lung cancer, renal cell carcinoma Hodgkin's lymphoma, and gastric cancer. Among gastrointestinal adverse events, colitis occurs most frequently, while gastritis is of rare occurrence as only handful of cases have been reported. Here, we present a unique case of isolated ulcerative duodenitis associated with PD-L1 uses successfully treated with steroid.
Case Description/Methods: 57-year-old female with PMH of malignant melanoma with osseous metastasis treated with Pembrolizumab (Keytruda) for 8 months, last dose received 1 month prior to presentation. She presented with 10-day history of acute onset nausea, non-bilious vomiting, and LUQ pain. There was no history of NSAID use. Workup including CBC, LFTs, contrast enhanced CT abdomen was unremarkable. EGD revealed multiple superficial duodenal ulcers (see figure) in the bulb and 2nd part with background of mucosal inflammation. Biopsies showed marked active duodenitis with villous atrophy, cellular atrophy, cryptitis, crypt abscess and crypt dropout consistent with medication induced injury. She was started on 1 mg/kg (60 mg), tapered by 10 mg/week. Her symptoms resolved by end of 1 week of treatment.
Discussion: There have been less than 20 cases of PD-L1 associated upper GI adverse events reported in literature. EGD findings have been variable including normal, mild gastritis, gastric erosions, hemorrhagic gastritis, atrophy, and ulceration. Our case is unique as the patient had isolated duodenal findings. The exact mechanism of injury is unknown, although several mechanisms of tissue injury due to self-reactive CD8-positive T cells and plasma antibody-mediated autoantibody production from CD4-positive T cells have been proposed. Our patient responded well with tapering dose of prednisone, however few refractory cases required treatment with anti-tumor necrosis factor (TNF)-α antibody, infliximab. It is imperative to carefully examine the duodenum including 2nd/3rd part when excluding ICI related upper GI adverse effects.



[3459] **Figure 1.** Esophagogastroduodenoscopy showing stomach (fig A), ulcerated mucosa in duodenal bulb (fig B and C) and in 2nd part of duodenum (fig D).

S3460

Olmesartan-Induced Enteropathy With Associated Anti-Enterocyte Antibody

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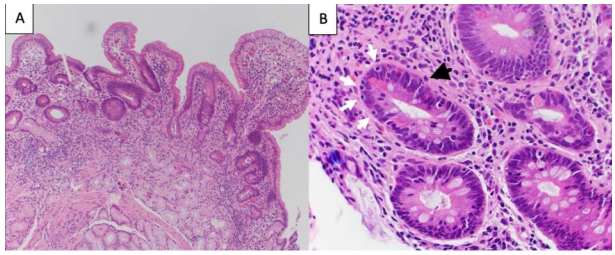
Introduction: Olmesartan-induced enteropathy (OIE) is a well-described drug injury that presents with severe diarrhea and weight loss. The mechanism of injury is poorly understood though believed to involve immune-mediated enteropathy in addition to direct damage of intestinal epithelial cells. We describe a severe presentation of OIE with associated positive anti-enterocyte antibody.
Case Description/Methods: A 74-year-old female presented with a seven month history of weight loss, diarrhea, nausea, and vomiting. Initial evaluation demonstrated a stool osmotic gap of 42 consistent with secretory diarrhea and negative celiac serologies. Cross-sectional imaging revealed extensive inflammation throughout the stomach and duodenum. A subsequent esophagogastroduodenoscopy (EGD) with biopsies revealed esophageal necrosis, active gastritis, and severe mucosal changes of atrophy and scalloping in the duodenum. Olmesartan for hypertension was held and she was started on twice daily IV PPI for the esophageal necrosis. Due to persistent and worsening symptoms over the following four weeks, she underwent a repeat EGD and was given IV steroids. Duodenal biopsy showed chronic duodenitis with villous blunting and scattered apoptotic epithelial cells compatible with Olmesartan-induced injury (Figure). Indirect immunofluorescence demonstrated IgG linear staining of apical membranes indicative of circulating anti-enterocyte antibodies. Diarrhea, weight loss, and difficulty with PO intake persisted the following two weeks despite steroid therapy and nutrition support with enteral access for tube feeds. She declined immunosuppressive agents or total parental nutrition and elected to discharge home on a steroid taper. Four months after discharge, she reported complete resolution of her diarrhea while on Budesonide and significant weight gain with both enteral feeds and PO intake.
Discussion: When OIE was first described, 3 of 19 patients were positive for anti-enterocyte antibody indicating potential association. These antibodies may circulate in a hyper-immune state such as the setting of Olmesartan use in certain individuals. The clinical presentation and histology of OIE and autoimmune enteropathy (AIE) are similar, which further supports an immune-related mechanism of injury in OIE. It is unclear if anti-enterocyte antibody presence indicates Olmesartan may cause AIE or is associated with OIE itself. We present this case to draw awareness to this relation and need for further investigation of the mechanism of injury in OIE.



[3460] **Figure 1.** Panel A - The duodenal mucosa showed mild to moderate villus blunting and widening by an increased chronic inflammatory infiltrate in the lamina propria. (Hematoxylin and eosin stain, 100x original magnification). Panel B - There were scattered epithelial apoptotic bodies in the crypts (black arrow) and focal increase in intraepithelial lymphocytes (white arrows). (Hematoxylin & eosin stain, 400x original magnification).

S3461

Patient With Short Bowel Syndrome Discharged Home With Full Oral Intake by Utilizing Medium-Chain Fatty Acids: A Case Report

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Introduction: Short bowel syndrome (SBS) requires lifelong use of nutritional therapy for malabsorption associated with extensive small bowel resection. Medium-chain triglyceride (MCT) is a favorable energy source because of its more rapid digestion and absorption than of long-chain fatty acid and absorption through the large intestine. We describe the case of an SBS patient who was discharged on a full oral intake of MCTs and additional foods.
Case Description/Methods: A 78-year-old female had a history of colorectal cancer surgery. She underwent extensive small bowel resection for intestinal necrosis caused by a thrombus of the superior mesenteric artery. The remaining small intestine was the jejunum up to 110 cm from the Treitz ligament, with no residual ileum. The patient was 150 cm tall, weighed 59.0 kg, and had nutritional requirements of 1755 kcal energy and 70 g protein. Oral intake was initiated on postoperative day (POD) 5 in combination with intravenous nutrition. Diarrhea continued at a frequency of 2-5 times/day. Since nutrient uptake

was affected due to lack of appetite, we attempted (1) seeking healthy sip feed and other nutritional supplements, (2) adding mainly familiar foods such as natto and yogurt, and (3) ensuring efficient energy intake by adding MCT oil. As nutritional dosage increased, diarrhea events increased to 5-7 times/day, and the patient became anxious about voiding. On POD28, MCT oil was added to ensure sufficient energy intake. To avoid fatty diarrhea, the lipid energy ratio was set at approximately 30%, of which 30%-40% was derived from MCT oil. The frequency of excretion increased from 6 to 13 times/day; bowel habit gradually improved, and the amount of feces per excretion decreased. Food intake also showed gradual improvement, and at the time of discharge, energy intake was 1300 kcal and protein intake was approximately 50 g, which did not meet the required amount, but skeletal muscle mass improved from 15.6 to 16.8 kg and Alb from 2.4 to 2.7 g/dL, and the patient was discharged on POD54.
Discussion: In nutritional management of SBS, the length of the remaining small intestine, location of resected small intestine, and presence/absence of an ileocecal area are important. In this case, the upper small intestine, a high-absorption site, remained, and the patient could consume relatively high-quality protein. Therefore, the use of MCTs, which have high utilization efficiency, increased protein utilization efficiency.

S3462

Oh, My Blister Pack! A Case of Ingestion and Perforation

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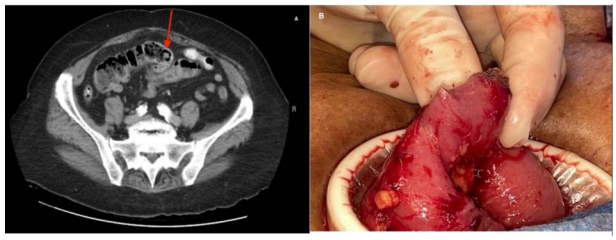
Introduction: Perforation of the small bowel resulting in resection due to foreign body ingestion occurs in less than 1% of all foreign body ingestions (FBI). Commonly FBI occurs in children and the elderly, due to shared risk factors including age and poor cognitive function. The main purpose of blister pill packs is to prevent tampering and most importantly seal individual tablets for protection and safety against ultraviolet light. Here we describe accidental ingestion of a blister pill pack presenting as abdominal pain, resulting in a small bowel resection.
Case Description/Methods: An 86-year-old female presented with a chief complaint of abdominal pain. She rated the pain at an 8 out of 10. She noted that movement made the pain worse, and no activity made it better. She denied any hematochezia, melena, diarrhea, or constipation. Initial diagnostic workup for abdominal pain included computed tomography (CT) of the Abdomen and Pelvis. CT scan revealed a partial small bowel obstruction with dilated, fecalized small bowel proximal to the blister pack and non-distended small bowel distally (Figure A). Subsequently, the patient was taken to the operating room, where the blister pack was turned 180 degrees and found to be adherent to the right lower quadrant of the small bowel (Figure B). Small bowel resection was completed successfully with anastomosis of the bowel. Patient had resumed diet with resumption of bowel movements within 10 days post-operation.
Discussion: Foreign body ingestion is a common occurrence in children and elderly people. Most locations of impaction include the stomach and esophagus. Rare effects of blister pack ingestion may include small bowel obstruction leading to perforation as seen in our case. Clinicians should be aware of a broad differential when considering ingestion of an unknown substance, including small bowel obstruction, perforation, or aspiration.



[3462] **Figure 1.** 1a: Left Image 1b: Right.

S3463

Primary Intestinal Follicular Lymphoma

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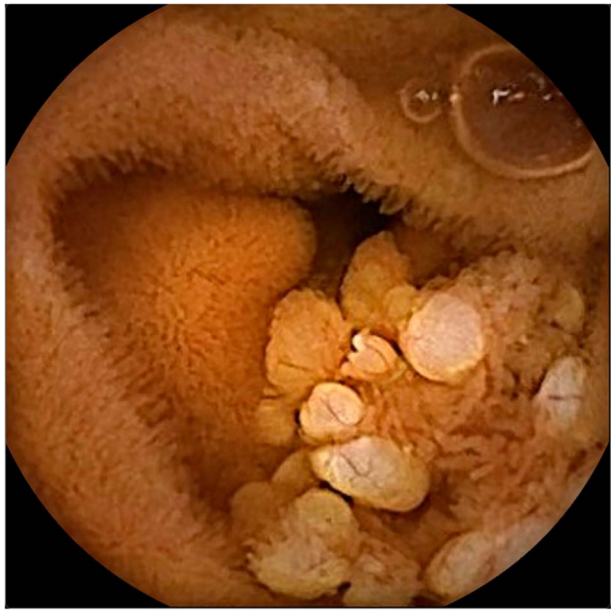
Introduction: Follicular Lymphoma (FL) mostly presents as a nodal lymphoma, however primary extranodal involvement is rare. The gastrointestinal tract is the most common site of extranodal FL, and with rates of FL increasing it remains important to recognize the characteristic endoscopic features of the disease. It typically carries an indolent course that may not even require treatment. We present a case of primary intestinal FL that involved the entire small intestine.
Case Description/Methods: A 53-year-old Hispanic male with a history of gastrointestinal reflux disease (GERD), presented with refractory GERD symptoms despite maximal PPI therapy. An upper endoscopy was performed and revealed an irregular Z line, gastritis and numerous, patchy, small (< 1cm), white nodules in the second and third portion of the duodenum. Multiple biopsies were taken from the nodules. Pathology revealed Grade I-II FL. Capsule endoscopy was performed and revealed numerous similar lesions throughout the entire small intestine. He was referred to Oncology and underwent PET-CT scan which revealed multiple FDG-avid abdominopelvic lymph nodes. After discussion with Oncology, the patient elected for a watch and wait approach. Repeat PET-CT scan 6 months later revealed multiple abdominopelvic lymph nodes that are no longer FDG-avid. He remains asymptomatic and will be monitored closely off therapy. (Figure)
Discussion: The most common primary intestinal lymphomas are diffuse large B-cell lymphoma (DLBCL) and mucosa associated lymphoid tissue (MALT) Lymphoma. FL only comprises approximately 1-2% of primary intestinal lymphomas. The small intestine is the most common location, with the second portion of the duodenum as the most likely site of involvement. It is rarely seen in the colon and stomach and there are no reported cases of esophageal involvement. Rates of diagnosis are on the rise, thus highlighting the importance of endoscopic recognition as it may often be seen in the second portion of the duodenum on routine upper endoscopy. Given the indolent course of FL, a watch and wait approach may be reasonable. Other options for therapy include chemotherapy, Rituximab, or radiation.



[3463] **Figure 1.** White nodular mass in duodenum visualized on capsule endoscopy consistent with patient's known diagnosis of Follicular Lymphoma.

S3464

Primary Aortoduodenal Fistula as a Complication of Mycotic Aortitis

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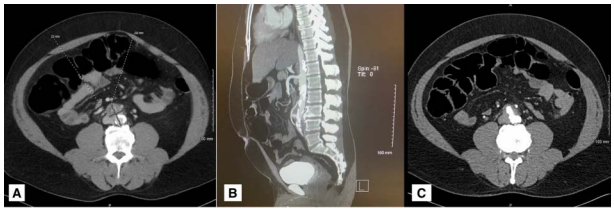
Introduction: Mycotic aortitis is an uncommon diagnosis that is almost universally fatal if not diagnosed and treated quickly. Primary aortoenteral fistula is a rare, equally life-threatening complication of mycotic aortitis due to localized inflammation of the aorta and the anatomical proximity of the bowel. The duodenum is the most common segment of bowel involved as it is located directly anterior to the aorta. Just over 250 cases have been documented between 1951 and 2010. With mortality approaching 100%, a high clinical suspicion is required for timely diagnosis and treatment.
Case Description/Methods: Here we present the case of a 54-year-old male with a 3 month history of vague abdominal pain. He was recently diagnosed with an unspecified abdominal mass, suspected to be malignancy. A PET scan revealed a hypermetabolic retroperitoneal mass with a biopsy planned at a later date. Before biopsy, the patient had acutely worsening abdominal pain and presented to the emergency department. An abdominal angiogram revealed an irregular saccular infrarenal abdominal aortic aneurysm with a mural thrombus and periaortic fat stranding with mural enhancement consistent with aortitis (Figure A, B, C). These findings were in the same location as the previously noted hypermetabolic retroperitoneal mass. Evaluation also revealed Methicillin sensitive *Staphylococcus aureus* bacteremia. The patient was diagnosed with mycotic aortitis and initiated on Ertapenem. He subsequently underwent an open procedure for aortic aneurysm repair with extensive debridement, where he was found to have a primary aortoduodenal fistula (PADF) with extensive spillage of duodenal contents. The area was debrided and the fistula repaired with plans to repair the aneurysm at a later time following resolution of severe localized inflammation.
Discussion: PDAF is a difficult diagnosis to make due to its low incidence and often vague symptoms. The classic triad of symptoms, in order of most to least common, includes gastrointestinal bleed, abdominal pain, and a pulsatile abdominal mass. However, this triad is only present in 10-11% of cases. Although gastrointestinal bleeding is the most common symptom, it is not present in over 60% of patients. Even with proper treatment, aortitis and primary aortoenteral fistulas both have an exceedingly high mortality rate. Quickly recognizing mycotic aortitis and planning an open surgical intervention led to the diagnosis of PADF in our patient which may have prevented his mortality.



[3464] **Figure 1.** A) Transverse slice from CT abdomen and pelvis revealing 23mm x 38mm abdominal aortic aneurysm. B) Sagittal slice from CT Abdomen and pelvis with IV contrast revealing abdominal aortic aneurysm with thrombus. C) Transverse slice from CT abdomen and pelvis with IV contrast revealing extravasation of blood from the abdominal aorta into the duodenum and surrounding tissues.

S3465

Protein-Losing Enteropathy Associated With SHOC2 Gene Mutation in Noonan-Like Syndrome

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Introduction: Protein-losing enteropathy (PLE) is the abnormal loss of protein through the gastrointestinal tract and has been associated with many diseases. The group of disorders including Noonan syndrome and Noonan-like syndromes (NS) consist of various dysmorphic features and congenital abnormalities. Multiple gene mutations paired with diverse phenotypic presentations have made identifying relationships between genotype and phenotype challenging, particularly with PLE in NS. In rare cases, PLE has been reported with NS, but few genetic associations have been made. Our case describes a possible connection between PLE and the SHOC2 gene mutation.
Case Description/Methods: A 21-year-old female with a history of NS presented to our outpatient clinic with lower extremity edema and diarrhea with four watery bowel movements per day for 3 months. Labs revealed albumin of 2.4 g/dL, creatinine of 0.35 mg/dL, and potassium of 3.1 mEq/L. Acute hepatitis panel and anti-tissue transglutaminase were negative. Stool studies were positive for *Clostridioides difficile* and she was treated with oral vancomycin. She had an unremarkable esophagogastroduodenoscopy and colonoscopy. Computed tomography enterography suggested altered lymphatic drainage, and a

lymphangiogram showed abdominal collateral lymphatics and iliac lymphangiectasia. Overall, these findings were consistent with PLE. A high protein diet and medium-chain triglyceride supplementation were initiated, and she had normalization of serum albumin levels. Genetic analysis showed a SHOC2 gene mutation.
Discussion: PLE consists of loss of serum proteins through the digestive tract, which may initially present as hypoalbuminemia in the absence of liver disease. This disorder is important to recognize due to the association between low albumin and increased mortality risk. Lymphatic disease presenting with lymphedema, chylothorax, and lymphangiectasia is well known in NS and may play a role in PLE development. PLE has been tied to the mutation of protein tyrosine phosphatase non-receptor type 11, but to our knowledge, has not been reported with SHOC2 mutation. Because little is understood about PLE in NS, there is no standard treatment. However, supplementation with medium-chain triglycerides and periodic albumin infusions has proven effective. The association between PLE and NS may be more common than acknowledged. This case identifies SHOC2 mutation with PLE and encourages consideration of PLE in NS in hopes of decreasing mortality and increasing quality of life.

S3466

Primary T-Cell Lymphoma of the Small Intestine Presenting as Perforation Peritonitis

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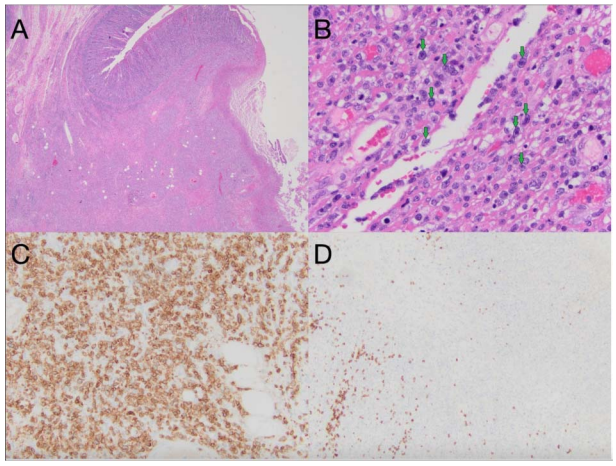
Introduction: Intestinal T-cell lymphomas are rare primary T-cell lymphomas. The two most common types are enteropathy-associated T-cell lymphoma (EATL) and monomorphic epitheliotropic intestinal T-cell lymphoma (MEITL). MEITL was newly defined by the 2016 revision of the World Health Organization, it was previously known as EATL type II. EATL is associated with celiac disease whereas MEITL is not. Both types of lymphoma usually present with non-specific symptoms.
Case Description/Methods: We present the case of a 65 year old female patient with no known relevant medical history presented with an acute onset of abdominal pain. X-ray showed pneumoperitoneum and the patient was taken to surgery. During surgery, a perforation in the small bowel was identified and a segment of the small bowel was resected. Histologic examination of the specimen showed full-thickness involvement by atypical medium-sized lymphoid cells with areas of necrosis. The overlying epithelium showed increased intraepithelial lymphocytes. Immunostains showed that the neoplastic cells are positive for CD2, CD3, and CD8 with aberrant loss of CD5 and CD7. The findings were most consistent with monomorphic epitheliotropic intestinal T-cell lymphoma. Histologic images with select immunostains are seen in the attached figure. (Figure)
Discussion: We present a rare case of intestinal T-cell lymphoma that was interestingly first diagnosed after it perforated the small bowel. MEITL carries an incidence of 0.25% of all malignant lymphomas and <5% of all digestive tract malignant lymphomas. It is unfortunately a very aggressive malignancy that carries a poor prognosis and a high mortality rate. Oftentimes, MEITL also represents a diagnostic challenge. Careful histologic examination is key in diagnosing this entity of disease.



[3466] **Figure 1.** A- A transmural lymphoid infiltrate is seen with mucosal ulceration (20x). B- The tumor cells are medium-sized to large (400x). C- The tumor cells are positive for CD3. D- The tumor cells show aberrant loss of CD5.

S3467

Primary Duodenal Adenocarcinoma: A Rare Malignancy

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Introduction: Primary duodenal adenocarcinoma (PDA) is extremely rare, representing 0.3 – 1% of all gastrointestinal malignancies. It poses a significant challenge to the physicians due to late non-specific symptoms that delays the diagnosis. Surgical resection is the only treatment option although PDA with distant or nodal metastasis have a very poor prognosis.
Case Description/Methods: A 33-year-old male patient presented with epigastric abdominal pain, nausea, vomiting, lipase > 2000, elevated LFTs and hyperbilirubinemia, consistent with acute pancreatitis. CT abdomen and pelvis revealed an irregular mass in the second and third portion of the duodenum near ampulla, dilation of pancreatic and common bile duct along with multiple hepatic lesions, consistent with metastatic malignancy. EGD showed non bleeding duodenal ulcer along with enlarged lymph nodes in the peripancreatic region. Biopsy of the ulcer was suggestive of moderately differentiated invasive duodenal adenocarcinoma with background of high-grade dysplasia. Due to metastatic malignancy, patient was started on folinic acid, fluorouracil and oxaliplatin without undergoing surgery. Percutaneous biliary drain was placed with resolution of pancreatitis and hyperbilirubinemia. Patient also completed a course of antibiotics due to pseudomonas bacteremia and sepsis secondary to pancreatic phlegmon that was drained. Pain was management with fentanyl patch and oral opioids. Patient was later discharged home with outpatient follow up.
Discussion: This case highlights the importance of primary duodenal adenocarcinoma (PDA) which is a very rare malignancy. Its etiology is unknown, however patients with familial adenomatosis polyposis, Peutz-Jeghers syndrome, Crohn's and celiac disease are at higher risk. Due to non-specific symptoms including anorexia, postprandial fullness, nausea, vomiting, jaundice and abdominal pain, early diagnosis is a challenge for physicians. PDA is most commonly seen in second part of the duodenum with an incidence of 50 -70%. It can be diagnosed with the help of contrast imaging studies along with EGD and biopsy. Management is usually surgical resection with tumors of the second part of the duodenum, proximal and distal infiltrating tumors requiring pancreaticoduodenectomy to ensure radical resection. Lympho-vascular invasion, lymph node metastasis and perineural invasion are features associated with poor prognosis and high mortality. For unresectable tumors, palliative surgery or endoscopic treatment is usually indicated.

S3468

Radiation-Induced Ileovesical Fistula: A Rare Presentation of Chronic Diarrhea

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Introduction: Chronic diarrhea (CD) is a common gastrointestinal manifestation. Etiologies include infectious diarrhea, IBD, IBS, malabsorption, medication-induced and post-surgical. It is a leading cause of healthcare utilization mostly due to the broad workup needed to achieve a diagnosis. Here we present a rare presentation of CD caused by longstanding effects of radiation.

Case Description/Methods: This is a case of a 50-year-old woman with a history of cervical cancer treated with radiation therapy 20 years ago and recurrent cystitis who was admitted due to 2 month history of CD, decreased urine output, and dysuria. The patient reported more than 5 episodes of watery diarrhea daily associated with abdominal and suprapubic discomfort. Vital signs were remarkable for tachycardia. On physical exam, she was found underweight and malnourished with a non-distended abdomen with high pitch bowel sounds. Labs showed preserved renal function with metabolic acidosis(MA). A urinalysis revealed pyuria and bacteriuria. Fecal leukocytes, stool culture and ova and parasite resulted negative. Empiric therapy for UTI and C. difficile were provided due to recent antibiotic use for recurrent cystitis. After ruling out C. difficile and no improvement of symptoms, loperamide was started without improvement. A CTE was ordered to rule out any structural cause of diarrhea but came unremarkable. Additional workup for less common causes of diarrhea like viral, parasitic and metabolic tests were ordered but all were negative. A colonoscopy was done but no endo-histologic abnormality was found. After 2 weeks, the patient reported an increased frequency of diarrhea and anuria, but renal function remained stable reason for which an enterovesical fistula (EVF) was suspected. A CT cystography was then performed confirming our suspicion. Due to poor nutritional status, the patient was not a candidate for surgery. To divert the urine and decrease the fistula's flow to the ileum, nephrostomies were placed. After 3 days the CD and MA resolved hence, the patient was discharged.
Discussion: Here we portray a case of an Ileovesical fistula (IVF) causing CD, recurrent UTI, and severe MA caused by the effect of radiation. It is important to report this case to raise awareness of the importance of the history of radiation in patients with CD to decrease invasive and expensive tests in the diagnostic process. Also, to highlight the benefits of nephrostomies as a tool in the management of IVF and its impact on the quality of life of the patients.

S3469

Recurrent Small Bowel Perforation due to IgG4 Mediated Enteropathy

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Introduction: Small bowel perforation is a rare but serious complication from causes such as trauma, neoplasm, obstruction, or complications from IBD. We present a case of recurrent small bowel perforation due to IgG4 mediated enteropathy (IME).
Case Description/Methods: A 44F with PMHx of endometriosis presents in distress with 3 months of intermittent LUQ, non-radiating abdominal pain with normal bowel movements. Temperature was 97 F, pulse 86, and BP 107/75. Her abdomen was diffusely tender with rebound and guarding. Labs include a leukocyte count of 14 with left shift, a Hg of 12, an anion gap acidosis, and lipase of 206. CT abdomen revealed free intraperitoneal air and fluid consistent with bowel perforation. Emergent ex lap revealed bilious ascites and a 5 mm mid small bowel perforation. After primary resection, she was discharged on post op day 3. Pathology revealed ischemic necrosis, gross perforation, and absence of granulomas. Over the next 3 months, she had many ED visits for abdominal pain where workup was negative for perforation but consistent with ileus. Colonoscopy and EGD were normal. MRE revealed dilated thick loops of small bowel and calprotectin has high normal. 4 months after initial presentation, she presented with abdominal pain, hypotension and tachycardia. CT revealed pneumoperitoneum and ex lap showed pinpoint perforation in the ileum. Small and large bowels were otherwise normal. Rheumatologic workup yielded + ANA, an IgG4 level of 255. Re- evaluation of pathology revealed elevated levels of IgG4. Special stains revealed increased IgG4 cells, however representing less than 40% of total IgG cells, and no definite storiform fibrosis. She was treated with prednisone 40 mg daily for one month with prolonged taper and has remained asymptomatic for one year.
Discussion: We describe recurrent small bowel perforation due to IME. Diagnosis requires histopathological storiform fibrosis, lymphoplasmacytic infiltrate, obliterative phlebitis and eosinophilic infiltration. Immunohistochemical features include IgG4+ cells per HPF $>$ 10 or IgG4+/IgG+ cell ratio $>$ 40%. Diagnosis requires 2 out of 3 histologic criteria to be met with IgG4 to IgG plasma cell ratio of 40% to support the diagnosis. Treatment is steroids with or without a biologic agent to achieve remission, which may take months to respond. It is unclear whether maintenance therapy with a steroid sparing agent is beneficial to patients. High index of suspicion is crucial to prevent dire consequences by straightforward therapy.

S3470

Severe Immune Checkpoint Inhibitor Enteritis Treated With Infliximab: Insurance Steered the Wheel

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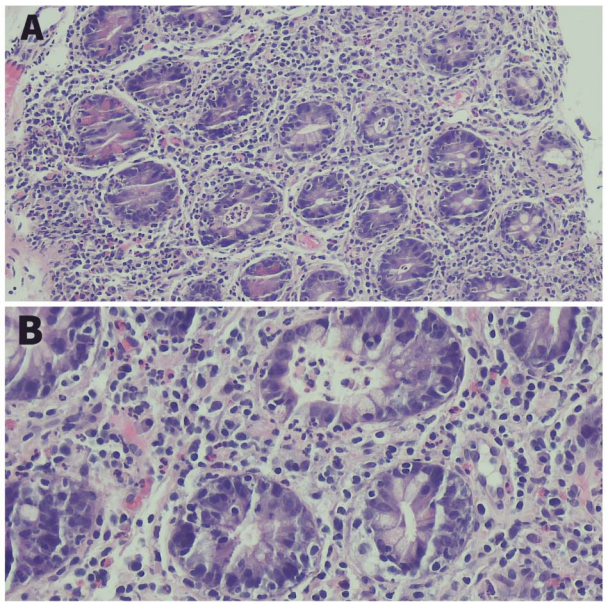
Introduction: There is limited published data regarding severe steroid-refractory isolated ICI enteritis. Vedolizumab seems to be effective in treating ICI colitis with a better safety profile when compared to Infliximab. However, some insurance companies are not approving it for therapy. We present a case of severe isolated steroid-refractory ICI enteritis successfully treated with Infliximab following an insurance denial for Vedolizumab coverage.
Case Description/Methods: A 59-year-old female with a history of metastatic renal cell carcinoma (RCC) status post left nephrectomy who has recently been treated with Axitinib and Pembrolizumabt therapy, presented with acute abdominal pain and diarrhea. Immunotherapy was discontinued and she received high-dose steroid therapy for 7 days for presumed ICI-induced diarrhea with no improvement. Abdominal exam was concerning for abdominal distention. Abdominal imaging showed extensive pneumatosis in mid-distal small bowel, moderate pneumoperitoneum, and diffuse small bowel perforation. She underwent an emergent exploratory laparotomy with distal ileal resection (22.5cm) and end-ileostomy. Her hospital course was complicated by intraabdominal abscesses and persistent high volume ostomy output (9-10L/day) despite a trial Imodium, high dose steroids, tincture of opium, octreotide, and fiber. Infectious workup, fecal elastase, celiac screen, VIP, TSH, gastrin levels were normal. Ileoscopy was performed showing normal mucosa, however, biopsies showed an increase in lamina propria inflammatory cells, cryptitis, crypt abscesses, and apoptotic bodies, consistent with chronic active enteritis/immunotherapy-related changes (Figure). Vedolizumab was denied by insurance despite multiple appeals and therefore, Infliximab 5 mg/kg was started. A significant improvement was noticed following one dose of Infliximab. The patient had complete resolution of symptoms and eventual ileostomy reversal.
Discussion: Infliximab is effective in treating severe isolated ICI enteritis. Although current literature favors Vedolizumab given its higher safety profile, especially given infection and malignancy in our case, the insurance company denied our request and appeal. Ongoing studies showing a higher safety profile of Vedolizumab in treating ICI enteritis will likely change insurance approval processes in the coming years.



[3470] **Figure 1.** Inflammatory cells in lamina propria, cryptitis, crypt abscesses, and apoptotic bodies A: Small bowel biopsy Hematoxylin & Eosin (H&E) stain, 100X B: Small bowel biopsy Hematoxylin & Eosin (H&E) stain, 200X.

S3471

Retrieval of Retained Video Endoscopy Capsule Reveals Small Bowel Adenocarcinoma

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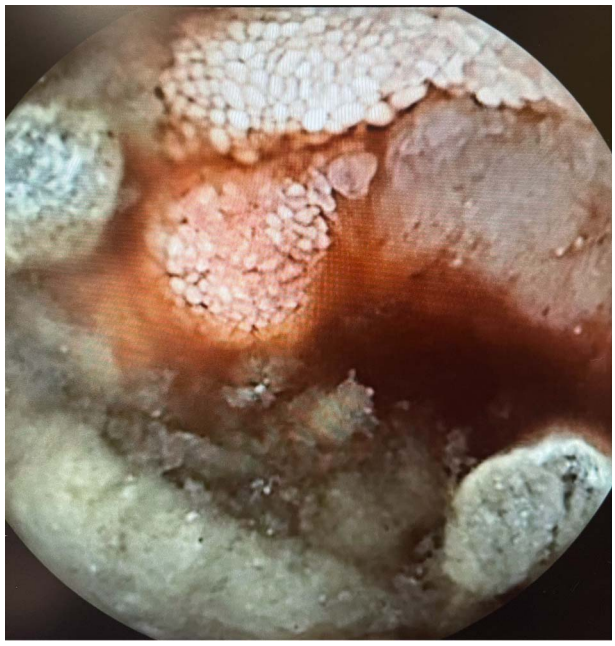
Introduction: Capsule endoscopy (CE) has been useful for evaluating diseases of the small bowel, such as bleeding. However, the most common and serious complication is capsule retention in the gastrointestinal (GI) tract. Rarely, the investigation of a GI bleed with CE reveals small bowel adenocarcinoma (SBA). Notably, limitations of small bowel exploration make it difficult to diagnose this devastating disease. Here we present a case of endoscopic retrieval of a retained capsule leading to a diagnosis of small bowel adenocarcinoma.
Case Description/Methods: A 78-year-old female with medical history including atrial fibrillation on apixaban was seen in the GI clinic for recurrent acute on chronic anemia and heme positive stools. Occasionally, she had minimal rectal bleeding and intermittent dark stool attributed to hemorrhoids and supplemental iron tablets. Esophagogastroduodenoscopy and colonoscopy were negative for a GI source of bleeding. CE was performed and showed fresh blood in the proximal small bowel at around the 3.5-hour mark. At the 6-hour mark, fresh blood was noted again along with failure of capsule to reach the cecum. Patient was contacted urgently and admitted to the hospital. She was asymptomatic upon admission. Later, she developed vomiting and abdominal pain. Subsequent imaging showed retained capsule in left side of abdomen. A push enteroscopy was performed which revealed the retained capsule at 150 cm in the proximal jejunum. An ulcerated mass was found to be obstructing the capsules path (Figure). Surgery performed an exploratory laparotomy with segmental resection of jejunum and partial gastrectomy. Pathology of the lesion returned with moderately differentiated adenocarcinoma. Patient was sent home with follow up in oncology clinic.
Discussion: Capsule retention occurs in approximately 1–3% of video capsule endoscopies. Retention often requires endoscopic or surgical removal. In this case, the exploration to retrieve a retained capsule revealed the uncommon diagnosis of small bowel cancer. Of the GI tumors, less than 6% are found to be SBA. SBAs typically occur without identifying risk factors. The diagnosis is typically made incidentally while investigating an intestinal obstruction or GI bleed. Treatment of SBA is surgical resection of the primary tumor and loco-regional lymph nodes. The main prognostic factors are margin of resection and nodal invasion, which are highly in favor of the patient in this case.



[3471] **Figure 1.** Ulcerated lesion in proximal jejunum found to be small bowel adenocarcinoma on biopsy.

S3472

Rumble in the Tummy: Atypical Location of Abdominal Thrombosis Post-COVID

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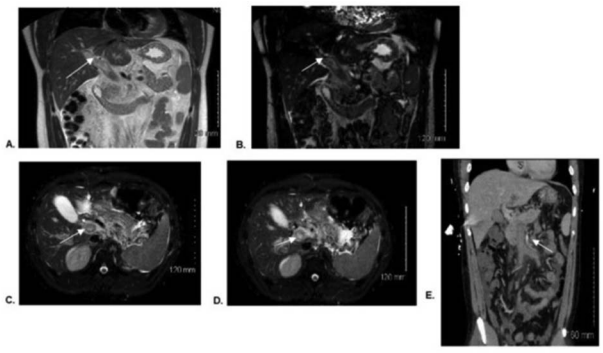
Introduction: More than 532 million cases of COVID-19 infection have been documented, and hundreds of thousands are being added every day. COVID-19 is linked to a number of co-morbidities, one of which, hypercoagulability, has piqued the interest of many researchers. Here, we present a case of a portal vein and superior mesenteric vein thrombosis in a patient with subclinical COVID-19 infection.
Case Description/Methods: A healthy 30-year-old male presented 3 weeks after having mild, asymptomatic COVID with epigastric abdominal pain, nausea, and vomiting. His only complaints when he had COVID was a decreased appetite, but no other gastrointestinal (GI) or respiratory symptoms. Upon admission, initial CT with contrast showed thrombophlebitis of superior mesenteric vein (SMV) and portal vein (PV), decreased contrast within the intrahepatic PVs; follow up MR Angiography revealed PV intraluminal filling defect beginning just distal to the superior mesenteric vein and splenic vein confluence and involving the intrahepatic PV with periportal edema. He denied any prior clots nor family history of clotting disorder. GI and hematology were consulted. He received catheter-directed tPA for 2 days due to abdominal pain and follow up CT angiogram with no evidence of occlusion in arteries. The cause for his extensive abdominal vasculature thrombosis is thought to be secondary to COVID hypercoagulability, and the location has not been well delineated. Abdominal pain improved, he continued anticoagulation (AC), did not require repeat sessions with plans for close follow up imaging. (Figure)
Discussion: Klok et al. reported a crude cumulative thrombosis incidence in COVID19 ICU patients is of 57% (95% CI 47–67%). In terms of intestinal damage, prothrombotic effects of the virus rises the incidence of mesenteric ischemia from 0.09–0.2% in the normal population to 1.9–3.8% in COVID-19 patients. Thrombosis is a known outcome, but in our case, this location is atypical, and he required catheter directed tPA by IR due to abdominal pain, that is a large measure and not typically done for thrombosis so it is also an aggressive management for thrombosis (done due to unrelenting abdominal pain). Hereby, we conclude that any vascular complication without an underlying reason should raise suspicion of an underlying COVID-19 infection, and more research should be done to determine the risk of such complications.



[3472] **Figure 1.** A,B: MRI Coronal view. Portal vein intraluminal filling defect beginning just distal to the superior mesenteric vein and splenic vein confluence and involving the intrahepatic portal vein with periportal edema Figure C,D: MRI Axial View of portal vein intraluminal filling defect with periportal edema Figure E: CT images post tPA show The portal vein, and SMV appear prominent in size and demonstrate perivascular stranding which can be seen with thrombosis.

S3473

Sclerosing Mesenteritis in the Setting of Follicular Lymphoma in a Young Male

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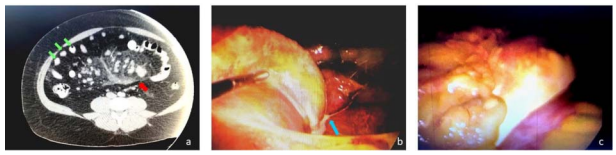
Introduction: Sclerosing mesenteritis, also called mesenteric panniculitis, describes a rare fibroinflammatory disorder. Acute or insidious in onset, it predominates in males over 60. Small intestine involvement is most common and can present as idiopathic, autoimmune or malignant. We report an unusual case of a previously healthy young male with mesenteritis related to follicular lymphoma.
Case Description/Methods: A 30-year-old male with a past medical history of obesity (BMI 45) and hyperlipidemia presented with mild diffuse abdominal pain and slightly elevated liver enzymes (AST 58, ALT 118). Family history included a grandfather with lymphoma in his 50s. CT Abdomen showed enlarged mesenteric lymph nodes measuring between 2.2-2.7 cm and coalescent, rim-enhancing cystic lesions with low-attenuation in the root of the small bowel mesentery (a). An area of fat around the mesenteric vessels and a fibrous band separating inflamed fat from mesentery was also seen, called fat ring sign and pseudocapsule, respectively. Laparoscopy was performed and the band was taken down with bipolar energy. Distorted omentum, small bowel edema, prominent lymph channels and chylous ascites were noted (b,c). Lymph node biopsy showed 50% of monoclonal B-cells expressing CD19, CD20 bright, CD10, and dim kappa light chain on flow cytometry, indicating follicular B-cell lymphoma. (Figure)
Discussion: Mesenteritis presents with nonspecific symptoms and is usually diagnosed in older adults with histories of abdominal surgeries. Along with histopathology, cytometry, and CT, PET can further diagnose this condition, as increased contrast uptake suggests co-existing mesenteric deposits, particularly in patients with lymphoma. Although mild cases have shown to resolve spontaneously, glucocorticoids and tamoxifen are first line therapy, while colchicine, azathioprine or cyclophosphamide are used adjunctly. Tamoxifen can be continued indefinitely to downregulate fibrosis, as the rate of recurrence and complications, including obstruction and perforation, is clinically significant. Follow up with patients is imperative as co-existing malignancies develop in up to 70% of patients with mesenteritis and include lymphomas, urogenital, and gastrointestinal carcinomas. Although literature is limited, mesenteritis should be included in the differential for nonspecific GI symptoms, regardless of advanced age. As in our case, this can treat potential malignancies early and avoid morbid complications, especially in a setting of familial history of cancer.



[3473] **Figure 1.** a: Enlarged mesenteric lymph nodes (green arrows) noted on initial CT with fat ring sign (red arrow). b: Prominent lymph channels with chylous ascites leading up to nodes were noted on laparoscopy (blue arrow). c: Distorted omentum with associated small bowel edema.

S3474

Sevelamer-Induced Ischemic Ulcerative Colitis

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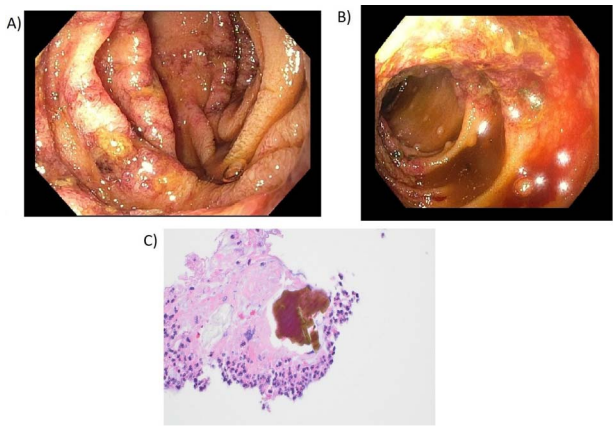
Introduction: Sevelamer is phosphate binder used to treat hyperphosphatemia in patients with end stage renal disease (ESRD) on hemodialysis. Rarely, it has been shown to be a cause of ischemic colitis. This case provides an example of a patient presentation and key findings when considering sevelamer induced small bowel ischemic colitis.
Case Description/Methods: A thirty-five-year-old male with ESRD on peritoneal dialysis presented with two days of hematochezia and severe abdominal pain. Diarrhea is described as maroon colored with four episodes in the span of 24 hours. Infectious work-up, including clostridium difficile and comprehensive GI panel, was negative for acute infection. CRP was elevated to 42.6 mg/L, WBC 12.9 10*3/uL, and hemoglobin 7.5 g/dL. CT angiography of the abdomen showed two short segments in the small bowel with inflammation concerning for ischemia. Colonoscopy and push enteroscopy were performed on day three of hospitalization showing ulcerative inflammation in D4 and in the terminal ileum 6cm proximal to the ileocecal valve. Biopsy results at the terminal ileum and D4 were showed mucosal necrosis and acute inflammation with crystalloid structures consistent with pill-associated mucosal necrosis. Further history was obtained from patient, he had been started on sevelamer a few weeks prior to hospitalization. Sevelamer was discontinued resulting in resolution of patient's symptoms (Figure).
Discussion: Although a rare complication, sevelamer has been documented to cause ischemic colitis (Yuste, Keri). These patients present similarly to the one discussed above with melena and abdominal pain. One of the hallmark findings reported on histopathological exam is "fish-scale" crystalloid structures in the mucosa. A review from 2017 examined published case reports in which sevelamer had caused ischemic colitis and the crystalloid structures were present in 13 out of 16 patients (Yuste). In ESRD patients on sevelamer, who present with symptoms consistent with gastrointestinal bleeding, it is important to recognize the sevelamer as a cause of ischemic colitis and to stop the sevelamer immediately. Furthermore, video capsule endoscopy or push enteroscopy should be considered if standard upper endoscopy and colonoscopy do not identify the source, as sevelamer induced enteritis can occur throughout the intestinal tract. If sevelamer is not stopped, complications such as perforation, acute anemia, stricture formation, and ulceration can continue to occur and worsen.



[3474] **Figure 1.** A) Diffuse ulcerated mucosa of the fourth portion of the duodenum. B) Patchy ulcerations of the terminal ileum. C) Terminal ileum biopsy showing crystalloid structures in an area of necrosis suspicious for pill-associated mucosal necrosis.

S3475

Renal Cell Carcinoma of the Small Bowel: An Unusual Site for Metastasis

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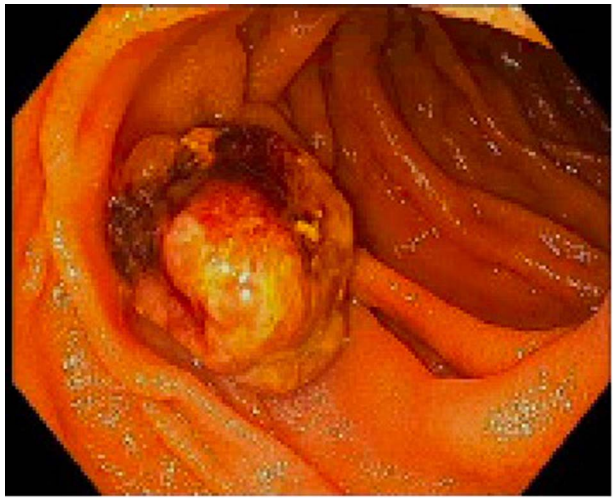
Introduction: Renal cell carcinoma (RCC) is a malignancy that accounts for 80,000 new cases and approximately 14,000 deaths yearly in the United States alone. Although it has several subtypes, the most prevalent is clear cell carcinoma. Most people are asymptomatic until the disease has advanced, in which they can present with various symptoms of hematuria, weight loss and abdominal pain. RCC often stays localized to the kidney, but metastatic disease does occur. The most common sites of metastases are the lungs, bone, lymph nodes, liver, and adrenal glands. While there are other less common sites, the small bowel remains extraordinarily infrequent.
Case Description/Methods: An 81-year-old male with a medical history of metastatic undifferentiated carcinoma of unknown primary presented with a one-month history of symptomatic anemia and melena. He reported a 40-lb weight loss over the last year and a newly noted lump on his chest. Laboratory work-up was significant for anemia with a hemoglobin of (8.6) from his baseline (15). He underwent a mammogram, which showed multiple masses within both breasts. An ultrasound guided right breast biopsy was performed, which revealed a poorly differentiated malignancy not of breast origin. Additional work-up with both upper and lower endoscopy revealed a large fungating mass, measuring 15 to 18 mm with bleeding involving the third portion of the duodenum. Biopsy was negative for malignancy at that time. Over the next few weeks, he continued to experience fatigue and melena, requiring hospital admission. Due to worsening anemia, he had a repeat upper endoscopy, again noting the presence of a large fungating mass. Repeat biopsies from the mass indicated poorly differentiated carcinoma, and the slides were sent to Mayo Clinic for additional review. Final pathology report suggested the possibility of a renal primary and molecular diagnosis was consistent with clear cell renal cell carcinoma (Figure).
Discussion: Metastasis to the small bowel is rare and when it does occur the most common primary is melanoma. In the few case reports of RCC metastasis to the small bowel GI bleeding is a common symptom. Due to the rarity of RCC metastasis to the duodenum there is no standard treatment protocol. In a patient with widespread metastasis systemic therapy or palliative treatment options are better options compared to surgical resection. Although the small bowel is not a common site for metastasis, especially RCC, it should be considered for undifferentiated carcinoma of the small bowel.



[3475] **Figure 1.** 3rd portion of the duodenum.

S3476

Recurrent Gastrointestinal Stromal Tumor With Extensive Peritoneal Metastasis and Adhesions to the Abdominal Aorta

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Introduction: Gastrointestinal stromal tumor (GIST) is a rare tumor of gastrointestinal (GI) mesenchymal (nonepithelial) origin. We report a case of GIST to address the importance of postoperative surveillance in reducing the risk of metastases and improving the mortality rate.
Case Description/Methods: A 44-year-old male presented with one week of diffuse abdominal pain. No nausea, vomiting, fever, diarrhea, constipation, or hematemesis; status post-laparotomy for a confined jejunal mass indicative of GIST. Examination revealed a non-tender palpable RIF mass. CTA revealed multiple hypodense, heterogeneously enhancing lobulated abdominopelvic masses in the mesentery, indicating peritoneal metastases. The patient had surgical debulking, multiple tumors were discovered intraoperatively, with the largest (15 cm) in the right iliac fossa extending into the retroperitoneum, adhering to the abdominal aorta, and affecting the gastrocolic ligament. Tumors were excised and the postoperative course was uneventful. Repeat CTA revealed enhanced residual tumors arising from the gastric fundus wall and nodular lesions in the right mesentery, jejunum, and pelvis.
Discussion: GIST develops from multipotential mesenchymal stem cells and is linked to mutations in the c-KIT and PDGFRA genes. They are frequently asymptomatic, but they can bleed and cause obstructive symptoms. GIST can be malignant in up to 20% of cases. In patients with suspected GIST, computed tomography (CT) is used to assess the primary tumor. PET CT scans are extremely sensitive. These tumors usually metastasize to the liver, then to the peritoneum, as seen in this patient. Estimating the risk of recurrence after GIST resection is critical when selecting patients who may benefit from adjuvant imatinib. Tumor size, mitotic rate, and tumor origin have been identified as the most reliable predictors of recurrence and/or metastasis in these tumors. A history and physical examination every 3 to 5 months for 5 years, then annually, is recommended for a completely resected GIST, along with CTA every 3 to 6 months for 3 to 5 years, then annually. Patients receiving imatinib with locally advanced or metastatic disease should have a history, physical examination, laboratory studies, and an abdominopelvic CT scan.
Conclusion: Although GIST rarely metastasizes, the undiagnosed distant metastatic tumor 5-year survival rate is 55%. Physicians should schedule frequent follow-up visits to identify and provide necessary interventions.

S3477

Running Into a Wall: A Case of Bouveret Syndrome

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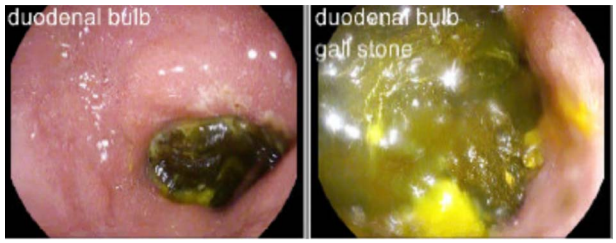
Introduction: Bouveret syndrome is characterized by gastric outlet or duodenal obstruction due to stone impaction in the pylorus or duodenum in the setting of cholecystogastric or most commonly cholecystoduodenal fistula. It is a very rare complication of cholethiasis, seen in 0.3-0.5% of cases.1 Due to its disproportionate presentation in the elderly, complexity, and non-specific presentation in combination with no set workup or management guidance, it has mortality rates ranging from 12-30%.[1]
Case Description/Methods: A 75 year-old female with a history of decompensated cryptogenic cirrhosis underwent routine MRI HCC screening, which identified signs of cholecystitis, pneumobilia, and fistula between the gallbladder and duodenum. The patient reported a postprandial "gallbladder attack" with epigastric/RUQ pain and nausea one week prior to MRI, with intermittent smaller attacks since. She was advised to present to the ED, where her exam was normal and labwork mildly cholestatic. CT abdomen/pelvis recapitulated the MRI findings. She underwent EGD, which demonstrated a large non-obstructive gallstone in the duodenal bulb. Due to resolving symptoms and lack of expertise in stone removal, no endoscopic intervention was performed. The patient was deemed too high-risk for surgery given her cirrhosis. She was discharged on antibiotics, and is currently planned for TIPS for risk optimization. (Figure)
Discussion: This rare case was discovered via routine HCC MRI screening. The postprandial symptoms described likely represent intermittent obstructions, relieved by gallstone transit. While there are no guidelines for Bouveret syndrome management, strategies have developed. Management starts with diagnosis, usually via CT or MRI.[2] EGD for diagnostic and sometimes therapeutic purposes is then performed. Endoscopic removal options include baskets or nets, mechanical lithotripsy, and specialized procedures including electrohydraulic, shockwave, or laser lithotripsy.2 Surgical options are considered for lack of endoscopic technical expertise or EGD failure. Cholecystectomy with fistula repair is controversial; often it cannot be performed due to comorbidity and risk, leaving patients at risk for recurrence and even

malignancy related to the unremoved fistula.[2] In our patient's case, her cirrhosis represented a hurdle in terms of surgical risk. In the end, the dearth of data regarding outcomes in this rare condition makes creating definitive guidelines difficult, and pushes management towards an individually-tailored approach.



[3477] **Figure 1.** EGD Demonstrating Gallstone in the Duodenal Bulb.

S3478

Successful Diagnosis and Treatment of Radiation Enteropathy With Single Balloon Enteroscopy and Hyperbaric Oxygen: A Case Report

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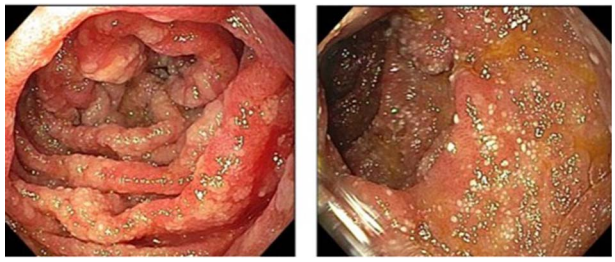
University of Maryland Medical Center, Baltimore, MD.

Introduction: Radiation enteropathy (RE) is a major complication of radiotherapy treatment for patients with abdominal and pelvic malignancies. Intestinal damage is due to disruption of angiogenesis, leading to capillary fibrosis and bowel ischemia. These pathologic changes can present clinically as small bowel obstruction from luminal stenosis or obscure GI bleeding (undiagnosed after upper endoscopy (EGD) and colonoscopy), which represents 5% of all GI bleeding and is typically found in the small intestine. However, endoscopy of the small bowel is challenging given its length. Deep enteroscopy via balloon assisted enteroscopy (BAE) is a valuable tool that permits exploration of the small bowel, especially given the risks associated with capsule endoscopy (CE) in patients with suspected RE. This case highlights the utility of BAE in diagnosing RE, and such awareness could improve the diagnostic timeline of patients who present with similar symptoms after undergoing abdominal radiation.
Case Description/Methods: A 78-year-old male with diffuse large B-cell lymphoma involving the small bowel, underwent consolidation radiation of the abdomen. He was later hospitalized for melena and symptomatic anemia requiring transfusions. Both initial and repeat endoscopic work-up showed no evidence of active bleeding. CE was also performed and did not identify a source of bleeding. Ultimately, Single Balloon Enteroscopy (Olympus, Center Valley, PA) was pursued and revealed segmental diffuse lymphangiectasia and erythema in the distal duodenum and proximal jejunum, changes consistent with moderate to severe RE. Additionally, multiple angioectasias with oozing were appreciated and successful Argon Plasma Coagulation (Erbe Med) in focal areas was performed. Given a diagnosis of RE, the patient was referred for hyperbaric oxygen therapy (HBOT) which led to resolution of his bleeding and symptomatic improvement. (Figure)
Discussion: This case was notable for its successful use of Balloon-Assisted Enteroscopy in the diagnosis and treatment of radiation enteropathy. While capsule endoscopy was utilized, it was unsuccessful in making the correct diagnosis and put the patient at risk for capsule retention. Furthermore, while many small studies have shown positive outcomes of HBOT in treating RE, it is still often underutilized. This case demonstrates the successful use of HBOT to treat RE and should be considered in patients with ongoing symptomatic anemia due to radiation injury given its favorable success rates in terminating bleeding.



[3478] **Figure 1.** Enteroscopic findings of segmental radiation injury in the small bowel using Single Balloon Enteroscopy.

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S3479

Small Bowel Diaphragm Disease Presenting With Iron Deficiency Anemia and Abdominal Pain

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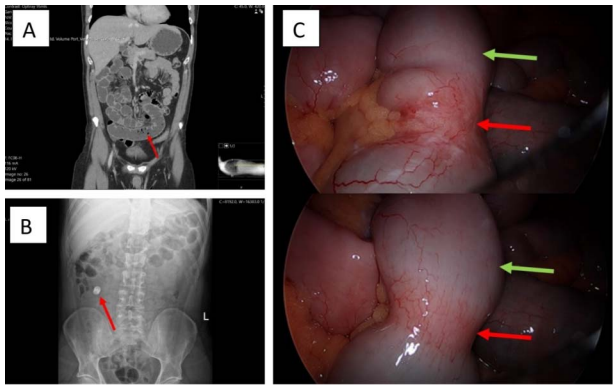
Introduction: Small bowel diaphragm disease (SBDD) is a rare condition characterized by circumferential strictures causing intermittent or complete small bowel obstruction, as well as unexplained abdominal pain or occult gastrointestinal bleeding commonly secondary to chronic NSAID use.
Case Description/Methods: A 50-year-old male with no past medical or surgical history presents with intermittent recurring hypogastric abdominal pain with occasional vomiting and chronic fatigue. His lab work was significant for microcytic anemia secondary to iron deficiency. Previous EGD and colonoscopy one month before to identify the cause of iron deficiency anemia were normal. CT scan of abdomen/pelvis showed moderately dilated small bowel loops involving the distal jejunum and proximal ileum suggestive of an incomplete closed-loop partial obstruction without an obvious cause (Figure A). MRI ruled out suspected small bowel adenocarcinoma or any other significant cause of bowel obstruction. A video capsule endoscopy could not be completed because the patency capsule did not pass through as seen on XR (Figure 1B). A diagnostic and therapeutic laparoscopy was performed, where a careful inspection of the small bowel from the ileocecal valve to the duodenojejunal flexure showed three distinct areas of congested appearing, thickened, narrowed small bowel within the ileum were observed (Figure C) with associated mesenteric lymphadenopathy. The diseased segment of the small bowel with the associated mesenteric vessels was removed followed by hand-sewn end-to-end anastomosis. Histology of the resected small bowel showed ulceration with elongated mucosal folds and submucosal fibrosis confirming the diagnosis of SBDD. Postoperatively, the patient improved clinically, and repeat anemia workup showed significant improvement (Table).
Discussion: The most common presentation of SBDD includes a history of extensive NSAID use, anemia, and obstructive symptoms. Imaging can potentially be used to diagnose SBDD pre-operatively. The best diagnostic and therapeutic intervention for diaphragm disease is laparoscopy which will show dilations and strictures in the affected bowel segment. Definitive treatment consists of resection and stricturepalsty of the affected small bowel. SBDD should be considered as a differential in patients with chronic abdominal pain, obstructive symptoms, and iron deficiency anemia regardless of history of NSAID use.



[3479] **Figure 1.** A. Coronal CT abdominal scan - Signs of dilated small bowel and fecalization (red arrow) suggesting small bowel obstruction. B. Abdominal XR - showing the Patency capsule that did not pass through the small bowel (red arrow). C. Laparoscopy images showing macroscopic changes in the small bowel. Strictures (red arrows) and dilations (green arrows) are visible in both images.

Table 1. Summary of lab values at presentation and 2- and 8-months post-resection of the disease small bowel. L= lower than normal values and H= Higher than normal values

Lab Results	At presentation	2-month Post-Operative	8-month Post-Operative
RBC count (million cells/mcL)	3.97 (L)	4.78	4.95
Hb (g/L)	91 (L)	121 (L)	135
Hematocrit (%)	0.308 (L)	0.392 (L)	0.424
MCV (fL)	78 (L)	82 (L)	86
MCH (pg)	22.9 (L)	25.3 (L)	27.3 (L)
MCHC (g/dL)	295 (L)	309	318
RDW (%)	15.4 (H)	17.3 (H)	15.8 (H)
Ferritin (μg/L)	6 (L)	13 (L)	21 (L)

S3480

Symptomatic Duodenal-Type Follicular Lymphoma Treated With Bendamustine-Rituximab: A Case Report

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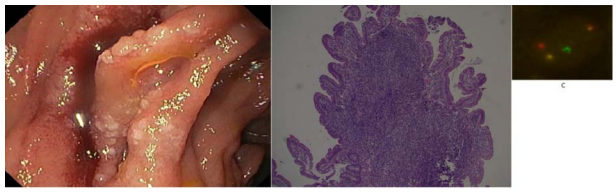
Introduction: Follicular lymphomas (FL) of the gastrointestinal tract are rare. They are usually discovered incidentally, and the terminal ileum is the most prevalent location. We present a patient with a rare variant of FL involving the distal duodenum and proximal jejunum and its treatment with bendamustine-rituximab.
Case Description/Methods: A 46-year-old female presented due to ongoing nausea, vomiting and diarrhea multiple times a day since many months. Her physical exam and laboratory data including stool studies were unremarkable. She underwent workup at an outside facility including an esophagogastroduodenoscopy (EGD) and colonoscopy, biopsies from which were unremarkable. Her computerized tomography (CT) scan of the abdomen showed mesenteric lymphadenopathy but was otherwise unremarkable. She reported that a capsule endoscopy was also done which showed ulcers in her small bowel. She was scheduled for an EGD with single balloon enteroscopy which showed nodular areas with white plaques in the distal duodenum and proximal jejunum. Her biopsies including immunohistochemistry and fluorescence in-situ hybridization (FISH) showed low-grade duodenal-type follicular lymphoma (DTFL) with translocation t(14;18) in the bcl-2 locus. Because of debilitating symptoms, she was treated with bendamustine-rituximab with resolution of her nausea and vomiting and significant improvement in her diarrhea. She was given loperamide and colestipol for mild persistent diarrhea. A repeat EGD with double balloon enteroscopy was performed after completing treatment which showed no gross abnormalities and biopsies did not demonstrate the presence of any lymphoproliferative disorder. (Figure)
Discussion: DTFL is a newly recognized entity in the WHO classification update. It is usually detected incidentally on endoscopy and diagnosed at a low grade and stage and stays localized to the duodenum in most cases. Due to the excellent prognosis, a wait and watch strategy is recommended. This case is unique because our patient presented with debilitating symptoms which are not classic of DTFL and hence treatment was initiated with chemotherapy resulting in significant improvement of her symptoms.



[3480] **Figure 1.** A- Nodular areas with white plaques seen on balloon enteroscopy B- Histopathology demonstrating atypical lymphoid infiltration into lamina propria. The neoplastic cells are positive for CD20, CD10 and bcl-2 C- t(14;18)(q32;q21) (IGH/BCL2) translocation is detected by FISH. One green, one orange and two yellow fusion signals (x1000).

S3481

Small Bowel Obstruction Caused by Neurofibromatosis

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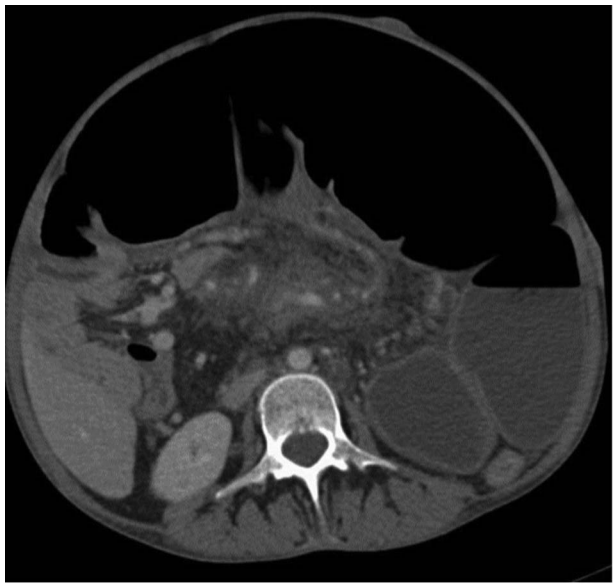
Introduction: We report an unusual case of acute small bowel obstruction in a woman with Neurofibromatosis type 1 (NF1) caused by numerous gastrointestinal neurofibromas. NF1 is an autosomal dominant genetic disorder that is classically characterized by the development of cutaneous neurofibromas, but less commonly, it is associated with a variety of gastrointestinal tumors including plexiform neurofibromas, mucosal ganglioneuromas, and gastrointestinal stromal tumors [1]. Up to 25% of patients with NF1 are reported to have a gastrointestinal tumor of some kind, but it is estimated that only 5% develop gastrointestinal symptoms including bleeding, abdominal pain, abdominal distention, constipation, and very rarely small bowel obstruction (SBO) [2].

Case Description/Methods: Patient is a 26-year-old female with a past medical history significant for poorly controlled Crohn's disease who presented due to abdominal pain and hematochezia. Physical exam was notable for pain in the right upper quadrant. CT abdomen and pelvis with contrast showed high-grade small bowel obstruction with tethering of small bowel centrally concerning for active Crohn's disease. Colorectal surgery was consulted and performed laparotomy with ileocecectomy and end ileostomy formation. The terminal ileum and cecum were sent to pathology for further analysis. The pathology report showed "wall thickening with extensive neural hyperplasia (ganglioneuromatosis) and plexiform neurofibromas. The features seen in the resection favor intestinal neurofibromatous proliferations with secondary chronic mucosal changes. There is no transmural inflammation, granulomas or other features typically seen in Crohn's disease." (Figure)
Discussion: This patient was initially thought to have SBO due to Crohn's disease flare, but pathology report showed that the underlying cause was secondary to plexiform neurofibromas. A systematic review of the literature shows that this is a rare finding as only 25 cases were identified from 1972 to 2013 of NF1 patients who underwent laparotomy for SBO [2]. SBO is a rare but possible manifestation of NF1.



[3481] **Figure 1.** CT Abdomen / Pelvis showing high grade small bowel obstruction.

S3482

Small Bowel Enteropathy Induced by Immune Checkpoint Inhibitors: A Case Series

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Introduction: While there is a growing understanding of the identification and management of colonic complications of immune checkpoint inhibitors (ICI), less is known about proximal intestinal complications. In this case series, we present 3 patients who developed small bowel enteropathies (SBE) after initiation of ICI.
Case Description/Methods: **Case 1:** A 68-year-old female with stage IV non-small cell lung adenocarcinoma on pembrolizumab developed diarrhea with 7 watery bowel movements daily. EGD was normal and colonoscopy showed minimal sigmoid erythema endoscopically with histologic duodenitis and colitis. Despite treatment with vedolizumab, she remained steroid-dependent. Repeat EGD and sigmoidoscopy showed variable villous abnormality in the duodenum with associated epithelial lymphocytosis, focal active duodenitis, and resolution of colitis. Serologic evaluation revealed hypogammagobulinemia. Nine months after last dose of pembrolizumab, and ultimately therapy with intravenous immunoglobulin therapy and open-capsule budesonide, her symptoms resolved. **Case 2:** A 42-year-old male with metastatic melanoma treated with ipilimumab and nivolumab developed grade 2 diarrhea with steatorrhea, nausea, vomiting, and unintentional weight loss. EGD was endoscopically normal with biopsies showing active duodenitis with normal colonoscopy. He was initially treated with steroids, but due to persistent symptoms transitioned to infliximab. After receiving 3 doses of infliximab, he had complete resolution of symptoms. **Case 3:** A 59-year-old female with celiac disease (CD) and metastatic melanoma was started on nivolumab. She developed abdominal pain, early satiety, softer stools, and unintentional weight loss. EGD revealed non-bleeding duodenal erosions and duodenal mucosal changes consistent with celiac disease. Biopsies showed subtotal villous atrophy with increased intraepithelial lymphocytes. CD typing did not suggest refractory sprue. Her presentation was suggestive of a nivolumab-induced exacerbation of CD. After a year of intermittent steroid therapy and adherence to a gluten-free diet, symptoms resolved. Repeat EGD with biopsies showed normal villi and reduced intraepithelial lymphocytes.
Discussion: In this case series, we present three patients who developed SBE, highlighting the challenge of its diagnosis and management. SBE should be suspected in individuals with altered stools and no evidence of classic colitis.

S3483

Superior Mesenteric Artery Thrombosis and Intestinal Ischemia as a Consequence of Sars-Cov-2 Infection

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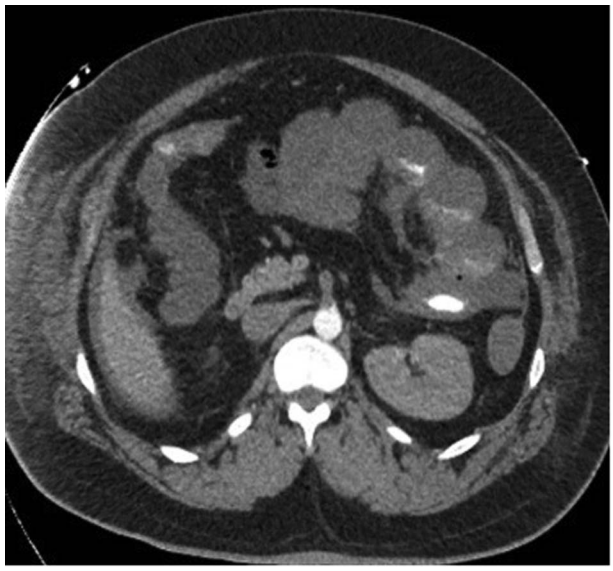
Introduction: We present a patient with devastating thrombosis of the superior mesentery artery associated with SARS-CoV-2.
Case Description/Methods: A 47 yo man with a PMH of obesity and hypothyroidism presented with malaise, fever, chills, diarrhea, productive cough, and dyspnea for 9 days. He was febrile and tachycardiac with a SpO2 of 87% on RA. His breath sounds were distant. Creatinine and D-dimer were mildly elevated, and he had a positive SARS-CoV-2 PCR. CXR showed hazy patchy bilateral opacities. He was started on dexamethasone, nebulizer treatment, oxygen, and prophylactic enoxaparin. Subsequently, he was on BIPAP and baricitinib in the ICU. On hospital day 8, he developed mild abdominal discomfort, and an abdominal X-ray showed an overall paucity of bowel gas with thickened folds in a partially distended loop of bowel. On day 9, he required intubation with mechanical ventilation and a vasopressor. Repeat D-dimer was markedly elevated (21,380), with lactate 2.2 mmol/L. Non-contrast CT demonstrated mildly prominent fluid-filled bowel from the duodenojejunal junction to the splenic flexure, with mesenteric edema and interloop ascites. CTA showed complete occlusion of the SMA at its origin, with no evidence of atherosclerotic disease in the aorta and its branches. In the OR, a gangrenous bowel from the ligament of Treitz to the mid transverse colon was noted. Revascularization of the SMA and/or excision of the vast majority of the patient's bowel was deemed futile. He expired the next morning. (Figure)
Discussion: Thrombosis in COVID-19 may be due to inflammation; endothelial injury by a viral affinity for ACE2 receptors in the respiratory tract, heart, GI tract, and distal vasculature; activation of tissue factor pathway; excessive thrombin generation; increased fibrin formation; and polymerization with fibrinolysis shutdown. Hypoxia in severe COVID-19 may stimulate thrombosis by increasing blood viscosity and a hypoxia-inducible transcription factor-dependent signaling pathway. The management approach is different from center to center with some institutes having anticoagulation protocol based on D- dimer levels and others doing pharmacological thromboprophylaxis, preferably with iv UFH or LMWH. Health care providers should have a high index of suspicion regarding this life-threatening complication of COVID-19 so that timely intervention can be done. Also, Future research is needed to better understand the role of coagulopathy and anticoagulation treatment in managing patients with COVID-19 infection.



[3483] **Figure 1.** Axial image of CTA of the abdomen shows thrombus with complete occlusion of the superior mesenteric artery at its origin.

S3484

Small Intestine Bacterial Overgrowth After COVID-19 Infection

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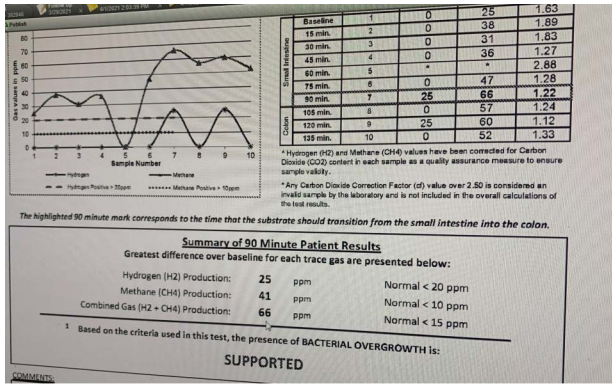
Introduction: Gastrointestinal manifestations of COVID-19 includes diarrhea, which usually resolve after the acute infection. Covid-19 has been observed to cause a disruption of the gut equilibrium. We present a case of post-COVID SIBO in a patient with no other potential cause.
Case Description/Methods: Patient with past medical history of hyperlipidemia, OSA and BPH presented to hospital with complains of dyspnea. Based on imaging studies and lab work, diagnosis of COVID infection was made. Patient did not require steroid or Remdesivir therapy. He received symptomatic treatment and discharged in two days. Patient was admitted to hospital for diarrhea and bloating months after initial presentation. CT Abdomen was negative for inflammation, workup for infectious causes was negative. Patient was symptomatically treated and discharged home, however he continued to have recurrent episodes of diarrhea and frequent complain of severe bloating. EGD was unremarkable and biopsy for H Pylori was negative. Colonoscopy was negative and biopsy results did not show microscopic colitis or functional bowel disorder. Stool studies was negative for malabsorption. Lactulose hydrogen breath test confirmed the diagnosis to be SIBO. Patient was prescribed Rifaximin and Neomycin and his symptoms completely resolved. (Figure)
Discussion: Low gastric acid secretion and intestinal dysmotility are two main factors causing SIBO. Viral infections damage the enteric epithelial cells, causing decreased Motilin production, resulting in reduced migrating major complexes causing dysmotility and favoring overgrowth of bacteria. It has long been established that there is a cross walk between lungs and gut microbiota. Studies have shown that disruption of gut microbiota resulted in increased risk of developing respiratory illnesses (Chunxi et al., 2020) and gut microbes also play role in immunity against respiratory infections (Willer & Viemann 2021). Likewise, it has been established that respiratory illnesses negatively affect gut microbiome as well. Enterocytes express Angiotensin-converting enzyme 2 (ACE2) protein, which is a major receptor for SARS-CoV-2. It is hypothesized that COVID causes alteration of gut microbiota resulting in "gut dysbiosis" causing increased bacterial growth however further research is on SARS-Cov2 and gut microbiota is needed to have a better understanding of GI manifestation of COVID infection.



[3484] **Figure 1.** Lactulose hydrogen breath test results.

S3485

Small Bowel Obstruction Secondary to a Duodenal Tubulovillous Adenoma: A Rare Case

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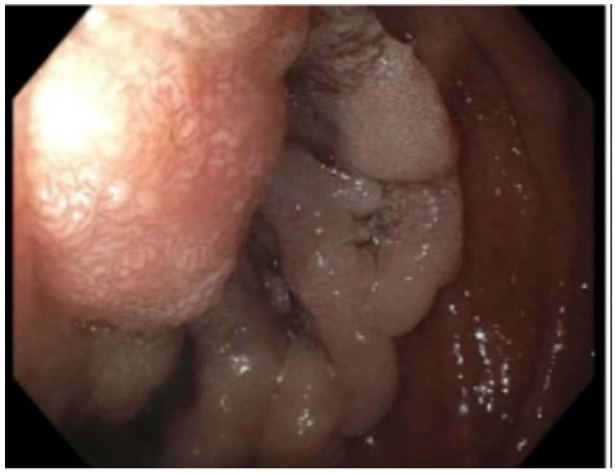
Introduction: Bowel obstruction is a common gastrointestinal emergency involving an interruption of normal intraluminal content flow. Small bowel obstructions alone account for approximately 15% hospital admissions in the United States. A rare, but lethal cause of mechanical obstructions are small bowel tumors. This paper reveals a rare case of mechanical obstruction secondary to a periampullary duodenal tubulovillous adenoma.

Case Description/Methods: Our patient is an 88-year-old Hispanic male with a past medical history of prostate cancer and long segment Barrett's esophagus who presented to the emergency department with coffee ground emesis 5 days prior to admission with associated epigastric abdominal pain. The patient was unable to tolerate any oral intake including liquids since the onset of symptoms. He denied NSAID or anticoagulant use. The esophagus and stomach were found to be dilated and filled with fluid. Given these findings, the patient was deemed to be a high risk for aspiration and underwent an emergent esophagogastroduodenoscopy (EGD). During the procedure, a large frondlike/villous mass was found within the third portion of the duodenum. This mass encompassed nearly 75% of the lumen of the duodenum. Biopsies were taken and the pathology revealed a tubulovillous adenoma with foci of high-grade dysplasia (Figure). The results were discussed with the patient with several treatment options, including surgical intervention for treatment or symptomatic management were discussed with the patient and his family. The patient was against surgical intervention and requested time to discuss the results with his family.
Discussion: Duodenal adenomas have an overall incidence of 0.4% of the lesions found during upper endoscopic studies. Furthermore, tubulovillous tumors of the duodenum are extremely rare, accounting for less than one percent of all duodenal neoplasms. The presence of duodenal adenomas is seen in up to 90% of patients with FAP, most commonly in the ampulla, periampullary regions, or distal duodenum. This adds to the uniqueness of our case, as our patient had no prior history of FAP. Although most of these patients are asymptomatic, they may occasionally display symptoms such as abdominal pain, melena, or weight loss. In addition, these tumors can also cause complications such as pancreatitis, duodenal obstructions or intussusception.



[3485] **Figure 1.** Upper endoscopy which revealed a large frondlike/villous mass in the third portion of the duodenum which encompassed nearly 75% of the lumen of the duodenum.

S3486

Small Bowel Intussusception Secondary to a Large Lipoma Presenting as Acute Gastrointestinal Bleeding

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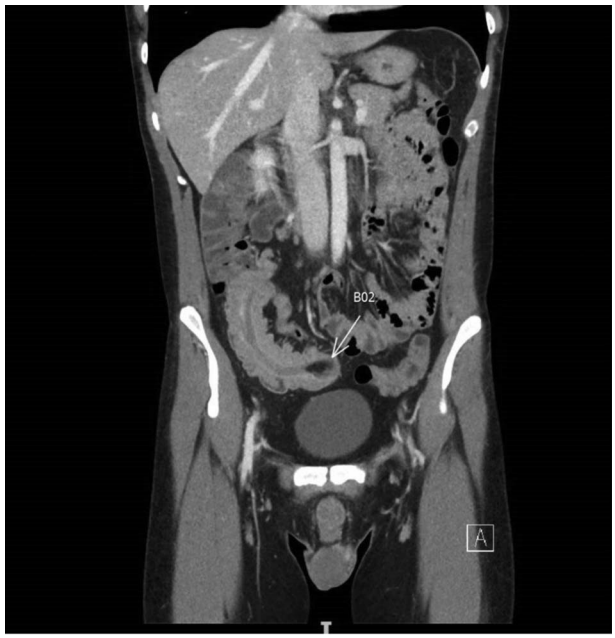
Introduction: Small bowel intussusception in adults is a rare disorder that represents less than 5% of all intussusception cases. This entity in adults is frequently associated with secondary causes such as benign and malignant tumors. We present a rare case of adult intussusception due to a small bowel lipoma, presenting with acute gastrointestinal bleeding without abdominal pain, treated successfully with surgical resection.
Case Description/Methods: A 28-year-old male with chronic headaches and daily ibuprofen use for 4 months presented with acute onset of hematochezia. Physical exam was remarkable for tachycardia to 130 bpm. Abdomen was soft and non-tender. Levels of hemoglobin were 6.5 g/dL and required blood transfusion. Esophagogastroduodenoscopy and colonoscopy were performed and were notable for fresh blood seen in the terminal ileum, thought to be originating from a more proximal small bowel source. CT angiogram of the abdomen was obtained and showed a fat density lesion within a loop of small bowel and a 15 cm segment of small bowel wall thickening at this level and proximal to it, representing a segment of intussusception. Diagnostic laparoscopic surgery was performed. Small bowel intussusception was found in the mid ileum around a soft intraluminal polypoid protrusion, and subsequently, 20 cm of small bowel were resected. Pathologic examination revealed a 10 × 3 × 3 cm polypoid protrusion consistent with a submucosal lipoma serving as an intussusception lead point. The polypoid protrusion was indurated and ulcerated at the tips. The postoperative course was uneventful, and the patient was discharged after two days (Figure)
Discussion: Intussusception in adults is often a challenging diagnosis with non-specific signs that require high clinical suspicion. History, physical exam, and blood tests can aid in the process, but imaging is usually needed to establish the diagnosis. Nearly 90% of cases of intussusception have a pathological lead point, most commonly a neoplasm. Herein, we report a rare case were a lipoma was the cause of intussusception. Interestingly, this patient presented with an acute bleed and drop in hemoglobin, possibly related to ischemia from the telescoping of the bowel and the development of ulcerations that were also demonstrated on final pathology. Lipomas of the small intestine are rare benign tumors with no malignant potential and are usually asymptomatic. Early recognition is critical since definitive treatment of intussusception is surgical intervention and should not be delayed.



[3486] **Figure 1.** A large fat density lesion with some septations measuring 3.7 cm. There is surrounding marked thickening of the loop of small bowel proximal to this extending over a length of 15.2 cm, representing a segment of intussusception.

S3487

Superior Mesentery Artery Syndrome Presenting in an Otherwise Healthy Female

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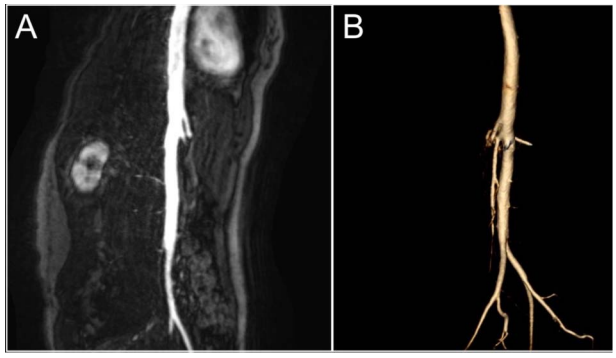
Introduction: Superior Mesentery Artery (SMA) Syndrome is a rare gastrovascular disorder due to narrowing of the space between the aorta and superior mesenteric artery, also known as aortomesenteric angle, which can compress the third part of the duodenum. Clinically, SMA syndrome in adults is associated with conditions, such a malignancy, trauma, extreme weight loss, or corrective spinal surgeries. We report a case of SMA syndrome in a patient without comorbidities to alert clinicians of the non-specific manifestations of this rare phenomenon.
Case Description/Methods: A 41-year-old female presented with a 30-year-history of post-prandial epigastric pain and acid reflux. She also reported early satiety, frequent belching, nausea, and constipation. She denied vomiting, diarrhea, hematochezia, and weight loss. Physical examination revealed epigastric tenderness to palpation. Laboratory studies were unremarkable. Upper endoscopy showed patchy erythema and few erosions in the antrum consistent with gastritis. Colonoscopy, gastric emptying study, and abdominal ultrasound were unremarkable. CT abdomen and pelvis showed nondilated bowels without thickening. Magnetic resonance angiography abdomen showed narrowing of the aortomesenteric distance at 5 mm, decreased aortomesenteric angle at 23 degrees, and mild narrowing of the left renal vein as it passes between the aorta and superior mesenteric artery (Figure). SMA syndrome was diagnosed. She was started on metoclopramide 5mg twice a day with subsequent improvement of her acid reflux by 90 percent and resolution of her epigastric pain.
Discussion: SMA Syndrome may present with insidious non-specific signs and symptoms, which can lead to delayed diagnosis. When diagnosis is delayed, it can result in significant morbidity and mortality. Diagnosis is challenging. Gastric emptying studies may be falsely negative when the duodenum obstruction is relieved by positional changes. CT or magnetic resonance angiography allows visualization of the vascular compression. In mild cases without small bowel obstruction, prokinetic pharmacotherapy may help decrease symptoms. Surgical correction may be needed. Clinicians should consider SMA syndrome in select patients who present with symptoms suggestive of small bowel obstruction when gastrointestinal studies are unrevealing.



[3487] **Figure 1.** Decreased aortomesenteric angle of 23 degrees. A. Sagittal view. B. 3-dimensional reconstruction view.

S3488

Symptomatic Meckel's Diverticulum in an Adult Presenting With Recurrent Gastrointestinal Bleeding

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Introduction: Meckel's diverticulum is a gastrointestinal congenital malformation that results due to persistence of the vitelline duct, and is commonly diagnosed in children before the age of two. Rarely, symptomatic Meckel's diverticulum can present in adults, as in this case with recurrent gastrointestinal bleeding.

Case Description/Methods: A 51-year-old male with a history of iron deficiency anemia presented to the hospital with hematochezia associated with mild epigastric tenderness. He had a similar presentation one year ago with unrevealing EGD, colonoscopy, and video capsule endoscopy. Three months ago, repeat colonoscopy was notable for a single nonbleeding diverticulum in the terminal ileum and outpatient double balloon endoscopy was recommended, however not yet done due to current hospitalization. He presented with tachycardia and hypotension as well as a hemoglobin 10.6g/dL (previously 11.4g/dL few weeks prior) and mildly elevated BUN. CT abdomen and pelvis was negative for any active bleeding. Repeat EGD and colonoscopy was unremarkable except for blood-tinged fluid interspersed in the colon but again the source of bleeding remained obscure. Given the history of a previous finding of a terminal ileum diverticulum and persistence of hematochezia, a Meckel's scan was performed revealing abnormal uptake in the right mid to lower abdomen suspicious for Meckel's diverticulum containing ectopic gastric mucosa. Patient was taken to the operation room; a large Meckel's diverticulum was noted at approximately two feet proximal to the ileo-cecal valve along with hemorrhagic inflammatory changes within the local mesentery. Patient underwent partial small bowel resection and appendectomy with pathology revealing a Meckel's diverticulum of the ileum with gastric heterotopia. Patient had an uncomplicated recovery postoperatively along with resolution of hematochezia.
Discussion: A finding of Meckel's diverticulum is common in the pediatric population; however, it is a rare diagnosis in adults. This case highlights the importance of considering Meckel's diverticulum for instances of recurrent gastrointestinal bleeding, especially in patients who are still symptomatic despite an extensive workup. It is also important to notice that Meckel's diverticulum can be missed on video capsule study.

S3489

The Uncommon Acute Abdomen: Enteropathy-Associated T-Cell Lymphoma Revealed by Jejunal Perforation

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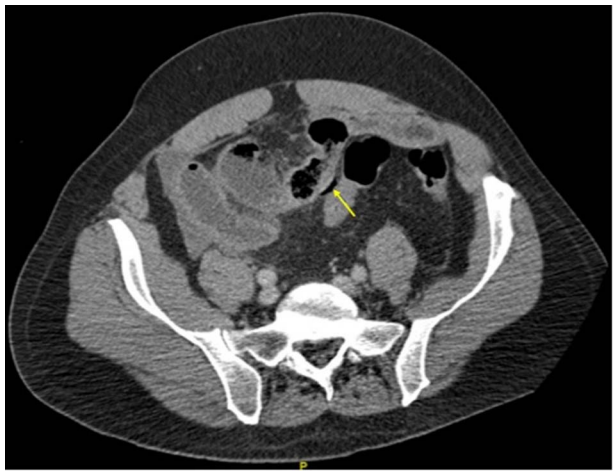
Introduction: Long-standing Celiac disease that is untreated or refractory to gluten-free diet can have dire consequences. Among these, Enteropathy-associated T-cell Lymphoma (EATL) is a rare form of non-Hodgkin's intestinal lymphoma that carries a poor prognosis with high mortality. This neoplasm can manifest as complicated small bowel disease such as obstruction or perforation. We present a patient with Celiac Disease presenting with an acute abdomen found to have jejunal perforation due to EATL.
Case Description/Methods: A 51-year-old male with history of biopsy-confirmed Celiac Disease (CD) presented for severe abdominal pain. Vital signs were stable and laboratory findings showed leukocytosis and lactic acidosis. Abdominal CT revealed closed loop obstruction and pneumatosis with hollow viscus perforation. He was taken to the OR for urgent exploratory laparotomy where purulent fluid and a perforation in the mid-jejunum was discovered. Subsequent jejunal resection with primary anastomosis was performed. Post-operative course was complicated by ileus and urinary retention but ultimately made full recovery. Biopsy of the resected jejunum revealed malignant lymphoid infiltrate in a background of mild villous blunting, increased intraepithelial lymphocytes, ulceration, perforation, and acute serositis consistent with EATL. Follow-up endoscopy was consistent with known CD, but otherwise unremarkable. T-cell receptor rearrangement study and bone marrow biopsy were negative. He was initiated on BV-CHP chemotherapy and achieved complete remission after 6 cycles. After induction, he opted for consolidation (BEAM) chemotherapy followed by autologous bone marrow transplant. Unfortunately, this treatment course was complicated by septic shock with multi-organ failure and ultimately decision was made for comfort care. (Figure)
Discussion: Strict gluten-free diet (GFD) is the primary prevention of EATL in CD. Non-compliance to GFD has been shown to significantly increase risk of developing EATL. Small bowel perforation is a rare, but well-documented complication of EATL that must be recognized in CD patients presenting with acute abdomen. Current treatment of EATL with chemotherapy and autologous bone marrow transplant has shown promising improvement in survival rates and reduced mortality rate. For CD patients, strict adherence to GFD with close clinical monitoring is of utmost importance to improve quality of life, minimize progression of EATL, and prevent necessity of risky advanced interventions as highlighted in our case.



[3489] **Figure 1.** Axial view of Abdominal CT showing perforation of small bowel.

S3490

The Great Masquerader: Whipple's Disease Presenting as Sarcoid-Like Granulomas

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Introduction: Whipple's disease (WD) can be mistaken for sarcoidosis given its overlapping symptoms and presence of granulomas. We present a patient with history of sarcoidosis with progression despite immunosuppression, later found to have WD.
Case Description/Methods: A middle-aged female presented with abdominal pain, vomiting, diarrhea, and weight loss for three years. She had AKI, hypercalcemia, and renal biopsy with non-caseating granulomas leading to sarcoidosis diagnosis. She improved with steroids and methotrexate. Workup for mediastinal lymphadenopathy and pulmonary disease was negative. Months later, GI symptoms recurred. EGD and colonoscopy with biopsies were unremarkable. MRE and gastric emptying study were normal. She received several courses of IV steroids for presumed sarcoidosis flares. On admission, a PET scan had intense FDG avidity in the small and large bowel and no extraintestinal uptake. IV solumedrol was initiated for presumed flare with plans to initiate outpatient TNF inhibitor therapy. However, she was readmitted for failure to thrive. Push enteroscopy revealed friable mucosa in the duodenum with biopsies showing numerous foamy histiocytes, distended villi with lipoid vacuoles, and positive periodic acid Schiff (PAS), consistent with WD. Importantly, no granulomas were identified. She was started on IV ceftriaxone with symptomatic improvement.
Discussion: WD is a rare, systemic bacterial infection caused by *T. whipplei* and presents with symptoms including diarrhea, weight loss, and vomiting. Diagnosis is challenging as it can mimic sarcoidosis with granulomas found in multiple organs and responds to steroids initially. In extraintestinal WD, PAS is often negative due to low density of *T. whipplei*; thus, it is vital to use PCR or electron microscopy to rule out WD in such cases. While it is possible that the patient had sarcoidosis and later developed WD in the setting of immunosuppression, the lack of typical hilar and mediastinal lymphadenopathy and only brief improvement in symptoms with steroids, argue against sarcoidosis. Antibiotics are the treatment for WD; however, patients on immunosuppressive therapy before antibiotics must be monitored for IRIS. Prompt diagnosis and treatment of WD is necessary to prevent endocarditis, CNS manifestations, and death. WD should be considered in the differential diagnosis of sarcoidosis, particularly in steroid-unresponsive disease.

S3491

The Unusual Culprit: A Rare Case of Severe Anemia Secondary to Hypocupremia

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Introduction: Acquired Copper deficiency is often overlooked, as daily requirements are significantly low[1]. Depletion of cellular lines, neuropathy, and cognitive decline have been associated[2,3]. In the absence of common risk factors such as enteropathies, or denture adhesives, copper deficiency is atypical[4].There are no available reports on such rare occurrences. We present a case of severe symptomatic anemia from copper deficiency in the absence of common risk factors.
Case Description/Methods: An 84-year-old female with history of atrial fibrillation presented with one-month complaint of worsening fatigue, weakness and dyspnea. She was malnourished, pale with a small non-bleeding external hemorrhoid on rectal examination. On admission had hemoglobin of 4g/dL, macrocytosis 111.5fL, platelet count of 144 10^3/mcL and white blood cell count of 2.86x10^3/mcL with neutropenia 1.49x10^3/mcL. Xarelto was discontinued and multiple blood transfusions given (goal hemoglobin more than 7g/dL). Ferritin, iron and total iron binding capacity remained low despite 2 weeks of high dose oral iron regimen. Endoscopic studies were unremarkable for bleeding. After thorough evaluation for other causes of anemia, giving her history of denture use, zinc and copper levels were tested. Copper and ceruloplasmin were deficient, 46mcg/dL and 14mg/dL respectively, and zinc was normal 60mcg/dL. She was started on IV copper gluconate and discharged on oral formulations. At six weeks follow-up, her neutropenia and thrombocytopenia resolved, and hemoglobin remained above 7g/dL.
Discussion: Copper deficiency is rare given high dietary source and low daily requirement, except in cases of enteropathies, bariatric surgery or zinc intoxication, yet the actual source of deficiency is unknown [3,5]. Copper deficiency evaluation is triggered by neurological symptoms (38%) despite its similar incidence to cooper anemia (46%)[6]. Given the patient's poor copper-rich diet and absorption, evident by her low serum copper and ceruloplasmin levels, and responsiveness to oral supplementation, confirms that the rare etiology of dietary deficiency is more common that initially thought. Copper replacement is ambiguous, however removing offending agent and supplementing might suffice[2,3,9]. Diet induced hypocupremia is rarely reported but may be more prevalent than thought, especially in elderly. Evaluation for less common causes should be considered prior to invasive and expensive testing and allow for timely treatment.

S3492

Unraveling an Unusual Case of Intussusception With Deep Enteroscopy

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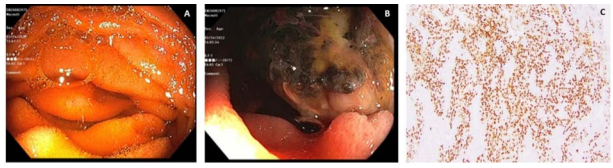
Introduction: Intestinal melanoma is a rare entity arising either from metastatic depositions or from primary mucosal involvement. Intestinal melanoma presenting as small bowel intussusception is exceedingly rare. Reported cases in the literature have been primarily managed surgically. We present a case of jejuno-jejunal intussusception due to intestinal melanoma successfully diagnosed and reduced with deep enteroscopy
Case Description/Methods: A 45-year-old man with history of chronic hepatitis B who presented with acute left upper quadrant abdominal pain, nausea, and vomiting. Review of systems was positive for one month of constipation, dark stool, and fatigue. Laboratory data were unremarkable apart from a mild iron deficiency anemia. CT scan of the chest, abdomen and pelvis revealed two large left upper lobe lung masses with ipsilateral mediastinal lymphadenopathy and pleural effusion, and a jejuno-jejunal intussusception with upstream dilation of the small intestine and stomach. Instead of surgery, it was opted to pursue deep enteroscopy for possible diagnosis and therapy. Utilizing an enteroscope, the jejuno-jejunal intussusception was reached, which initially demonstrated a severely narrowed lumen with normal tissue (Figure A). The area was eventually traversed and undone revealing a large, ulcerated, hyper-pigmented mass (Figure B), which was biopsied. Tissue immunohistochemical staining was positive for S100 protein and Sox10 establishing a diagnosis of melanoma (Figure C). Patient had significant symptomatic improvement after the procedure. A full skin and eye exam was normal. PET scan showed an extensive extra-nodal metastatic tumor burden. After a multidisciplinary discussion, the decision was made to proceed with medical therapy utilizing immunotherapy. Surgical management was going to be based on treatment response. Unfortunately, the disease progressed quickly prior to treatment and patient died due to complications of post-obstructive pneumonia
Discussion: This report of primary intestinal melanoma resulting in jejuno-jejunal intussusception describes a novel approach utilizing deep enteroscopy to diagnose and decompress intestinal intussusception. In the right patient population, this approach can have multiple added benefits including avoidance of emergency surgery, symptomatic relief, and reducing the extent of intestinal resection. More research is needed to determine if deep enteroscopy should be added to the treatment algorithm for adult intestinal intussusception.



[3492] **Figure 1.** A) Site of intussusception B) Hyper-pigmented, large, ulcerated jejunal mass C) Diffuse nuclear positivity for SOX10 staining in tumor cells, consistent with melanoma.

S3493

Two Cases of Perforated Jejunal Diverticulum With Severe COVID-19

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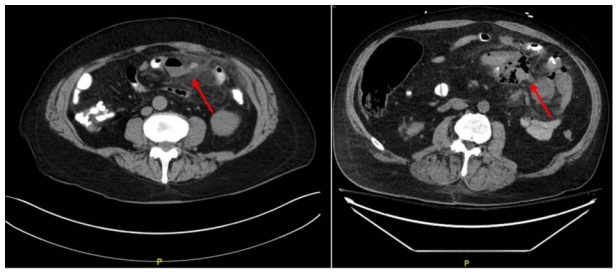
Introduction: Jejunoileal diverticulitis is very rare with a high mortality rate of 21-40%. The rates of small bowel diverticulitis with perforation associated with COVID are unknown. Here, we present cases of complicated jejunal diverticulitis with perforation in two patients with severe COVID infection.
Case Description/Methods: The first patient is a 69-year-old man who presented to our Emergency Department with shortness of breath, cough, and diarrhea. He was diagnosed with severe COVID-19 despite being vaccinated. He was started on methylprednisolone and remdesivir. 13 days into admission, the patient developed pneumomediastinum. Computed Tomography Abdomen/Pelvis (CT A/P) for further evaluation showed pneumoperitoneum and perforated jejunal diverticulitis. Given the severity of COVID pneumonia requiring high-flow oxygen, continued use of corticosteroids (methylprednisolone 40mg BID), and risk of surgical complications with COVID, conservative management with antibiotics and bowel rest was pursued. The patient developed an abscess in the mesentery and pelvis for which a drain was placed percutaneously by Interventional Radiology. He unfortunately developed progression of the perforation and passed away from septic shock. The second patient is a 57-year-old male with hypertension, deep vein thrombosis who presented with worsening shortness of breath, cough, fever. He was diagnosed with severe COVID (unvaccinated) and started on baracitinib, remdesivir, methylprednisolone. Ten days into admission he developed severe abdominal pain, nausea, vomiting, and worsening leukocytosis. CT A/P showed new abscess involving the left upper quadrant with a contained jejunal perforation. The patient was initially managed with bowel rest, antibiotics and pain control. However, he developed worsening leukocytosis and pain with peritonitis on exam. He was taken emergently to the operating room and had proximal perforated jejunum resection and lysis of adhesions. He ultimately had small bowel re-anastomosis. He continued to improve clinically and was discharged home. (Figure)
Discussion: It is unclear if the complication of perforated diverticulum in these cases is due to the underlying inflammatory response due to COVID, steroid-induced disruption of intestinal mucosal barrier or incidental diverticulitis with evolution to complicated perforation. However, each case reminds us how important it is to take abdominal complaints in COVID seriously as jejunoileal diverticulitis has a high mortality rate.



[3493] **Figure 1.** Left panel- Patient 1 CT Abdomen/Pelvis transverse view showing jejunal perforation with surrounding inflammation (arrow). Right panel- Patient 2 CT Abdomen/Pelvis transverse view showing thickened loops of jejunum with interloop abscess (arrow).

S3494

Whipple's Disease Without Arthropathy in an Immunocompromised Patient

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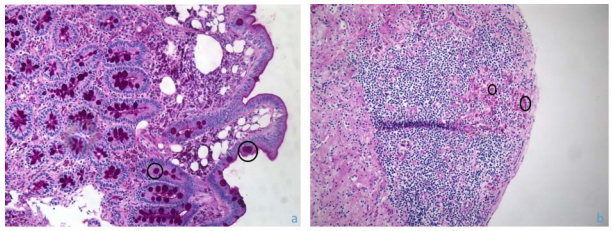
Introduction: Whipple's disease (WD) is a rare disorder caused by the pathogen *Tropheryma whipplei*. It presents with non-specific symptoms which can mimic the presentation of other gastrointestinal pathogens, especially in immunocompromised patients. We report a unique presentation of WD involving an immunocompromised patient who experienced non-specific gastrointestinal symptoms without arthralgia, which is a characteristic symptom of this disease.
Case Description/Methods: A 67-year-old man with a history of chronic hepatitis B infection and Human immunodeficiency virus (HIV) infection presented with weight loss, nausea, vomiting and myalgia. Of note, patient did not have arthralgia. Physical exam revealed cervical lymphadenopathy and diffuse abdominal tenderness. Patient was anemic (hemoglobin of 8.6 g/dL) with a normal white count. Endoscopy demonstrated erythema in the gastric body, lymphangiectasia of the duodenum, and increased granularity of the terminal ileum. Mucosal biopsies revealed macrophages in the lamina propria with focal histiocytic aggregates throughout the small bowel and cecum, with positive PAS staining, consistent with Whipple's disease. Confirmatory T. whipplei PCR testing was positive (Figure).
Discussion: WD is a rare diagnosis that must be considered in the differential diagnoses of patients presenting with unexplained nausea, vomiting, diarrhea and anemia. Furthermore, in patients with HIV, the possibilities would also include opportunistic gastrointestinal pathogens. Classic WD is characterized by diarrhea, weight loss, abdominal pain and extra-intestinal involvement manifesting as joint pain, endocarditis, dementia, supranuclear gaze palsy, and mediastinal lymphadenopathy. Diagnosis involves biopsy of affected tissue demonstrating foamy macrophages with PAS (+) substance, which can be confirmed by PCR or immunohistochemistry. Treatment involves an initial course of penicillin or ceftriaxone followed by a prolonged course of Bactrim, or a one-year course of doxycycline and hydroxychloroquine, followed by lifelong doxycycline therapy, which is more likely to prevent disease relapse. In the absence of suppressive therapy, relapse of WD is common and may lead to further complications, including neurological involvement.



[3494] **Figure 1.** Periodic Acid-Schiff-Staining Macrophages (circles) Within the Lamina Propria of the Duodenal Bulb (a) and Cecum (b) as Seen Via Light Microscopy with a 20x Objective Lens.

S3495

What Do These Eosinophils Mean? Treatment Naive Systemic Lupus Erythematosus Presenting With Eosinophilic Ascites, Eosinophilic Pleural Fluid, and Enteritis of Unclear Etiology

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Introduction: Eosinophilic ascites (EA), lupus enteritis (LE), and eosinophilic enteritis (EE) are rare systemic lupus erythematosus (SLE) complications. When they do occur, overlapping diagnostic criteria causes problems in distinguishing between a SLE-related etiology or EE. For example, intestinal eosinophil infiltration associated with EE is also consistently found in disseminated SLE autopsy case reports. Although both conditions are treated with steroids, therapies after treatment failure are different. We present a rare case of a SLE patient with this diagnostic dilemma with suboptimal response to SLE therapies, questioning whether an EE treatment algorithm may be more appropriate.
Case Description/Methods: An afebrile 55 year old female with untreated SLE presented with constipation and a mildly tender distended abdomen. Imaging described moderate ascites, continuous small bowel thickening, ileus, and bilateral pulmonary infiltrates with pleural effusions. Significant results included a white blood cell count of 3.8 without eosinophils, erythrocyte sedimentation rate 5, C-reactive protein $<$ 1, positive ANA (1:160), positive double stranded DNA, and low C3/C4 complement. Paracentesis yielded an exudative eosinophilic (71%) ascitic fluid with a low serum-ascites albumin gradient of 0.9 g/dL, high protein of 3.9 g/dL, and no evidence of malignancy or infection. She also had exudative eosinophilic pleural fluid. Endoscopy revealed mild cecal ischemia, mild gastritis, and a normal terminal ileum without eosinophils. Rheumatology diagnosed her with SLE serositis with peritoneal and pleural involvement complicated by LE, but they were unable to rule out EE. She responded to intravenous steroids but deteriorated when she was switched to oral prednisone. She then failed a regimen of hydroxychloroquine and oral prednisone. During her most recent hospitalization, the patient was transitioned to a regimen of hydroxychloroquine, mycophenolate mofetil, and oral prednisone.
Discussion: It is unclear whether our patient's sub-optimal therapy response is due to SLE or EE, with our case illustrating the difficulties in determining a diagnosis. Determining a diagnosis influences treatment and the patient's clinical course. In EE, treatment includes dietary therapy, antihistamines, leukotriene antagonists, and mast-cell stabilizers. However, our patient's symptoms are attributed to serositis, leading to a focus on escalating her SLE immunosuppressive therapy rather than re-examine the diagnosis.

S3496

You Shall Not Pass: Rare Duodenal Bulb Adenocarcinoma Causing Gastric Outlet Obstruction

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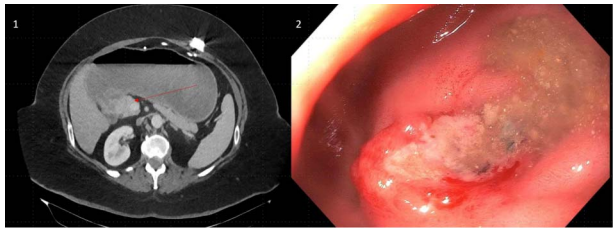
Introduction: Duodenal carcinoma accounts for only 1-2% of diagnosed gastrointestinal cancers in the United States. Due to its insidious onset, advanced disease is often established at the time of diagnosis resulting in poor outcomes for patients. Although duodenal adenocarcinomas have been found to comprise around half of all small bowel adenocarcinomas, disease arising from within the duodenal bulb in the D1 segment is a vanishingly rare phenomenon.
Case Description/Methods: A 52-year old female with a past medical history of morbid obesity with placement of laparoscopic adjustable gastric band 14 years prior presented with frequent emesis, inability to tolerate oral diet and a 22kg weight loss. Recent esophagogastroduodenoscopy during a previous admission revealed a cratered ulcer in the duodenal bulb. An upper GI series was obtained, showing a 2cm mass in the bulb apex which was confirmed by computed tomography. A repeat esophagogastroduodenoscopy was performed which identified a firm and friable ulcer with contact bleeding with complete obstruction of the duodenal bulb. Subsequent histopathology confirmed the presence of invasive, moderately differentiated adenocarcinoma. (Figure)
Discussion: Small bowel tumors comprise about 2% of all GI tumors. Adenocarcinoma accounts for about 30% of all small bowel tumors. The duodenum comprises 53% of all small bowel neoplasms. Of duodenal tumors, about 57% occur in the D2 segment of the duodenum. One 2014 meta-analysis of small bowel cancers between 1990-2012 from an institutional registry (excluding ampullary cancers arising from the bile duct mucosa) identified only 30 cases of primary duodenal adenocarcinoma with none arising from D1. A previous 1991 literature review cites 47 cases of primary duodenal adenocarcinoma arising from D1, however, anatomic distinction between the duodenal bulb and the distal D1 segments was not made. Although small bowel tumors are the third leading cause of gastric outlet obstruction, lesions are typically asymptomatic. In a study of 217 primary duodenal adenocarcinoma, the median survival was 20 months with 35% presenting with metastatic disease (39% present with stage III disease). Although it is difficult to distinguish the prevalence of duodenal bulb neoplasms, it has been clearly demonstrated that neoplasms arising from the D1 segment of the duodenum are extremely rare. By presenting this case, we hope to increase the index of suspicion for small bowel neoplasms as part of the differential for gastric outlet obstruction.



[3496] **Figure 1.** CT demonstration of duodenal bulb mass causing gastric outlet obstruction; Figure 2: Endoscopic image of duodenal bulb tumor.

S3497

Tubulo-Villous Adenoma of the Distal Ileum Presenting With Small Bowel Obstruction-Like Symptoms

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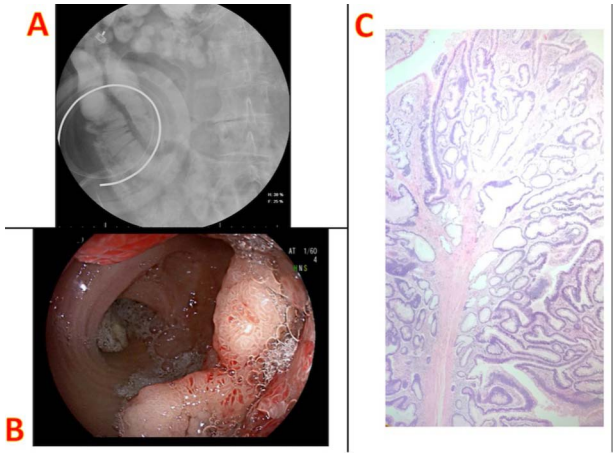
Introduction: Small bowel tumors (SBT) are rare and account for < 3% of all gastrointestinal tumors in the United States. Given their insidious presentation with non-specific symptoms, they can evade detection and cause delays in diagnosis.
Case Description/Methods: A 74-year-old lady came to the ER with abdominal pain, nausea, vomiting and abdominal distention since two days. Past medical history included esophageal spasm, GERD, achalasia, hypertension, CKD with anemia and fibromyalgia and had undergone total abdominal hysterectomy. At her last colonoscopy 10 years ago a 5 cm tubular adenoma was excised and a repeat was advised after 5 years, which she did not follow up with. EGD 8 years ago showed grade 1 reflux esophagitis, normal stomach and small bowel. Pulse was 100/minute and BP was 132/77. Abdomen was soft and distended, with tenderness in the right upper quadrant and epigastrium. Hb was 9.2 g/dL, consistent with her baseline. Though abdominal CT suggested SBO with a transition point just proximal to the ileocecal valve, a small bowel series ruled out an SBO. The patient was made NPO and NGT was placed. Colonoscopy revealed a partially obstructing, medium-sized, fungating, friable, infiltrative, circumferential, villous mass, 8-10 cm away from the ileocecal valve. Histopathology confirmed tubulovillous (TV) adenoma with no signs of dysplasia or malignancy. The patient underwent robotic-assisted ileocecectomy and recovered well. Follow up colonoscopy 4 months later showed healing ileocolonic anastomosis, with no stricture or ulceration. (Figure)
Discussion: Though the small bowel constitutes 90% of the surface area of the GI tract, it contributes to < 2% of GI malignancies. Reasons for this include the more liquid contents causing less mucosal irritation than solid colonic contents and rapid transit. Adenomatous polyps, the most common benign SBTs, have an epithelial origin. Histological types include tubular, villous and tubulovillous. Villous components, atypia or large size increase the risk for malignancy. Presenting symptoms are nonspecific and they are commonly found unexpectedly during surgery in patients with SBO. Evaluation in symptomatic patients includes endoscopy with biopsies. Push, double-balloon, or video capsule endoscopy may be needed. Diagnostic laparoscopy or surgical exploration can help establish a definitive diagnosis. Given their potential for malignant transformation, TV adenomas must be removed, and patients should be monitored for recurrence.



[3497] **Figure 1.** A: XR small bowel with oral contrast suggestive of mucosal irregularity with narrowing around distal ileum. B: Colonoscopy showing partially obstructing, circumferential villous mass, 8-10 cm from the ileocecal valve. C: Histo-pathology showing epithelial finger-like projections away from the muscularis mucosae, lined by dysplastic epithelium.

S3498

Multifocal Small Bowel Intussusceptions in an Adult with Strongyloidiasis

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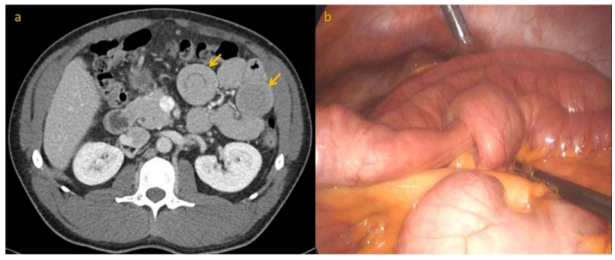
Introduction: Multifocal adult intussusceptions (AI) are rare with challenging diagnostic workup. Etiology of most adult intussusceptions are structural lesions, such as surgical adhesions or malignant neoplasm.

Case Description/Methods: A 54-year-old man presented with severe abdominal pain. His medical history included diverticulosis and pulmonary histoplasmosis, but no prior abdominal surgeries. He works in construction, and he denied international travel. Vital signs were normal and labs notable for leukocytosis (17 x10^9/L). Physical examination revealed abdominal distension and tenderness to palpation of the left abdomen. Contrast CT of the abdomen showed multiple proximal small bowel intussusceptions without identifiable lead points or obstruction (a, arrows). HIV, histoplasmosis, tuberculosis, and celiac serologies were negative. He underwent exploratory laparoscopy with reduction of 9 segments of intussuscepted bowel involving the jejunum and proximal ileum, all without identifiable lead point (b). CT enterography post-operatively was normal. Antegrade double balloon-assisted enteroscopy showed no mucosal abnormality and jejunal biopsies were unremarkable. Patient was discharged with symptom resolution. He re-presented 2 months later with recurrent left-sided abdominal pain. Laboratory investigation showed leukocytosis (10.7 x10 ^{9}/L) with mild eosinophilia (0.63 x10^9/L). CT enterography showed a possible short-segment intussusception at the proximal jejunum without lead point. Comprehensive autoinflammatory and infectious workup revealed positive Strongyloides Antibody, IgG. Patient was treated with ivermectin and has remained symptom free without further events. (Figure)

Discussion: AI is unusual and requires a high index of clinical suspicion combined with appropriate imaging to establish an early diagnosis.[1] More than 90% are caused by a lead point or structural abnormality (e.g., tumor) and CT is best modality for diagnosis.[1,2] Helminth infection is a well-recognized cause of pediatric intussusception, but few cases of helminth-related AI have been reported, and none secondary to Strongyloides have been documented. Our patient's peripheral eosinophilia, abdominal pain, recurrent multifocal intussusceptions, and resolution of these symptoms after treatment of Strongyloides infection suggest Strongyloides as the etiology of his multifocal AI. This case highlights the importance of a risk factor driven differential in the workup of AI.



[3498] **Figure 1.** CT abdomen showing multifocal small bowel intussusceptions (a) and exploratory laparoscopy demonstrating segment of intussuscepted bowel (b).

S3499

Gastrointestinal Involvement With Vasculitis: A Rare and Difficult Distinction Between Intestinal Tuberculosis and Inflammatory Bowel Disease

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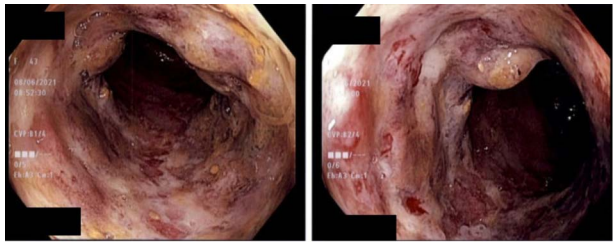
Introduction: GI involvement is common in forms of vasculitis and presentation can mimic Inflammatory Bowel Disease (IBD) and infectious enteritis. We describe a 43 year old female with a history of Tuberculosis (TB) who presented with symptoms of IBD. Endoscopy showed circumferential ileal inflammation with biopsies ruling out Crohn's Disease and microorganisms. Serology and kidney biopsy revealed P-ANCA positive Crescentic glomerulonephritis and Drug-Induced SLE and treatment included plasmapheresis, steroid taper and mycophenolate.

Case Description/Methods: A 43 year old female with a history of latent TB had recently discontinued Isoniazid due to adverse effects of abdominal pain and nausea. She presented with worsening abdominal pain and initial labs of anemia (hgb 6.9 g/dL), CRP 44.37 mg/L, ESR 80 mm/hr, Fecal Calprotectin 375 ug/mg and creatinine 2.49 mg/dL. CT showed retroperitoneal lymphadenopathy, multiple calcified lymph nodes, and thickened terminal ileum. Colonoscopy exposed 5-7 cm of continuous ileal ulcerative inflammation with spontaneous bleeding (Figure A) with biopsies positive for increased neutrophils and negative for Chron's and microorganisms. Worsening kidney injury required dialysis. Serology was positive for P-ANCA and renal biopsy showed Crescentic Glomerulonephritis with segmental sclerosis, interstitial fibrosis and tubular atrophy. She was treated with plasmapheresis and steroids and was evaluated at a tertiary center where they diagnosed drug-induced SLE. Treatment included mycophenolate, prophylactic Bactrim and steroid taper.

Discussion: Blood vessel inflammation occurs in vasculitis with various forms inducing GI symptoms and kidney injury. Abdominal pain is the most common symptom followed by GI bleeding. This clinically overlaps with IBD and Intestinal Tuberculosis (ITB) as they all primarily affect the ileocecal region in young females. They differ histologically with ITB having large caseating granulomas and AFB stain, vasculitis with leukocytoclastic infiltrate, and Crohn's with non-caseating granulomas. There are no standardized treatments for GI vasculitis. Therapies reported include steroids, 5-ASA, Anti-TNF, cyclophosphamide, IVIG and plasmapheresis. Surgical management is conservative, with perforation being managed by resection. The diagnosis of GI vasculitis has historically been difficult and can be life threatening if missed. This carries high importance in endemic areas of TB and vasculitis and providing awareness may eliminate late diagnosis and surgery.



[3499] **Figure 1.** The small bowel was intubated showing a normal distal ileum however the following 5-7 cm of small bowel showed continuous severe ulcerative inflammation with erythema and spontaneous bleeding. More proximally, the ileal mucosa was again normal appearing.

S3500

Cecal GIST Presenting as a Fecalith: A Case Report and Literature Review

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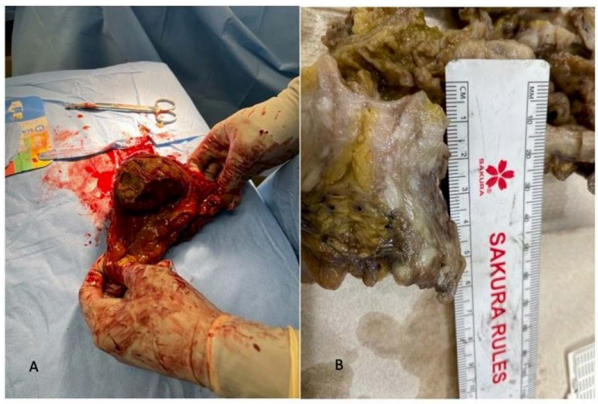
Introduction: A fecalith is a mass of accumulated hardened fecal matter that usually arises in the colon, most commonly the sigmoid and the rectum. Very rarely does a fecalith appear in the small intestine. GISTs are rare soft tissue sarcomas that can occur anywhere along the gastrointestinal tract. It is documented in the literature that fecaliths can mimic tumors. However, this report is the first documented case of a fecalith obscuring the presentation of a GIST, and even more unique given it was found in the cecum. Thus, the seemingly benign presentation of a fecalith may be a more concerning manifestation and consequently prompt further workup.

Case Description/Methods: A 58-year-old Hispanic male with a past medical history of HIV and melanoma, presented to the ED with a week of right lower quadrant abdominal pain. Physical exam was remarkable for severe pain on deep palpation of the right lower quadrant. Laboratory studies and vitals were normal and tumor markers, carcinoembryonic antigen, CA 19-9, and CA-125, were negative. A CT abdomen/pelvis scan was significant for colonic wall thickening, a "mass-like structure" in the cecum extending to the level of the ileocecal valve and enlarged peri-colonic lymph nodes. A colonoscopy was performed that showed a large fecalith in the cecum. After a failed attempt at removing the fecalith via colonoscopy, the patient underwent laparoscopic hand-assisted right colectomy the following Monday. The surgery was successful, and the patient's postoperative course was unremarkable. He was discharged on postoperative day seven. The pathology report indicated that the mass-like fecalith structure was a low-

grade multifocal GIST of spindle cell type involving 5 cm of the bowel wall, 2 mitoses per 5mm2, Ki 67 was less than 5%, pT2N0. A GIST neotype profile indicated the tumor was of the KIT D820G (Exon 17) missense mutation variant. It was decided the patient would benefit from oncology follow-up for adjuvant imatinib as well as follow up CT scans once every 3 months. (Figure)
Discussion: This case report depicts an unusual presentation of a fecalith and GIST in the cecum. While the patient underwent a thorough workup, the true etiology of his abdominal pain was not realized until surgery. This was likely due to the large fecalith's atypical location that obscured the presentation of the GIST. This case report is of clinical relevance because it depicts a diverse way that GISTs may present, and can hopefully expedite the recognition and appropriate treatment of similar cases.



[3500] **Figure 1.** A) Surgical resection of the fecalith in the cecum B) A 5 cm mass, later identified as a GIST, was present within the core of the fecalith.

S3501

Chronic Mesenteric Ischemia Causing Chronic Pneumatosis Intestinalis With Pneumoperitoneum

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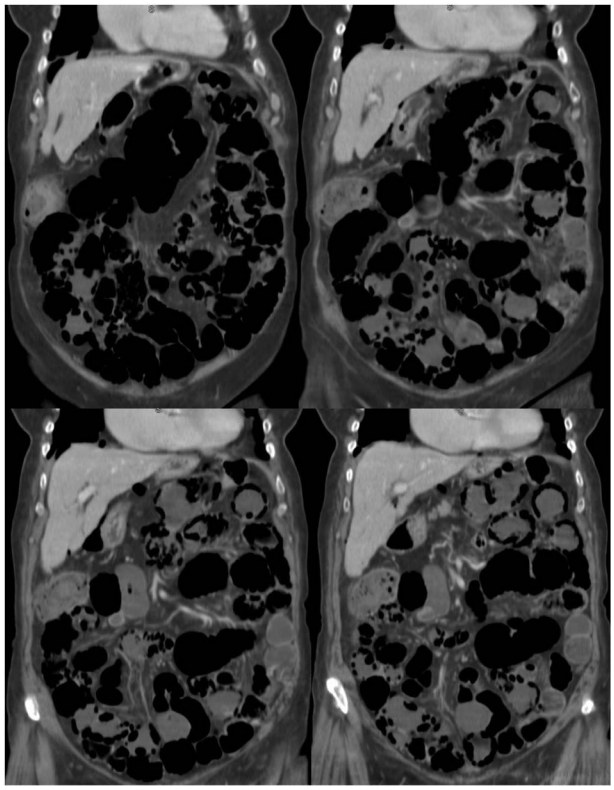
Introduction: Pneumatosis intestinalis (PI) is a condition in which gas is entrapped in the intestinal wall. In the context of abdominal pain and pneumoperitoneum, PI may reflect an intrabdominal catastrophe; however, there are other, more benign causes. We present a rare case of persistent PI with pneumoperitoneum secondary to chronic mesenteric ischemia.
Case Description/Methods: An 86-year-old female with past medical history of hyperlipidemia and coronary artery disease presented with an acute exacerbation of chronic abdominal pain. Pain was worsened by eating and physical exertion. On physical exam she was nontoxic appearing with abdominal distention and generalized tenderness without peritoneal signs. Abdominal x-ray was concerning for intraperitoneal free air. CT abdomen and pelvis with contrast revealed extensive PI throughout multiple loops of dilated small bowel with pneumoperitoneum. Additionally, atherosclerotic calcifications were seen throughout the abdominal vasculature, including at the origin of the celiac and superior mesenteric arteries. After a discussion with general surgery, the patient elected conservative management and was discharged with hospice care. Unexpectedly, her condition remained stable; she graduated from hospice and followed with her primary care provider for the next seven years. She was placed on cilostazol for chronic mesenteric ischemia. Interventional radiology determined endovascular revascularization would not be feasible given the extent of disease. Her abdominal pain was improved with the aversion of fibrous foods, fatty foods, and meats; when pain and bloating persisted, she would make herself nil per os until symptoms resolved. She underwent CT scans at three years and six years after her initial presentation that showed persistent dilated small bowel with PI and pneumoperitoneum. She continued to do well with conservative management. (Figure)
Discussion: PI with pneumoperitoneum is associated with gastrointestinal disease requiring emergent surgery. Initially, these alarming findings were concerning for acute mesenteric ischemia with bowel necrosis and perforation. However, the stability of her symptoms and radiographic findings indicated a severe case of chronic mesenteric ischemia. This case was unique given the profundity of these findings. This is an important reminder that PI with pneumoperitoneum is a nonspecific radiographic sign requiring careful clinical interpretation.



[3501] **Figure 1.** Initial CT showing extensive pneumatosis intestinalis with pneumoperitoneum.

S3502

A Deceptive Diagnosis: Gastric Outlet Obstruction in the Setting of Disseminated Tuberculosis

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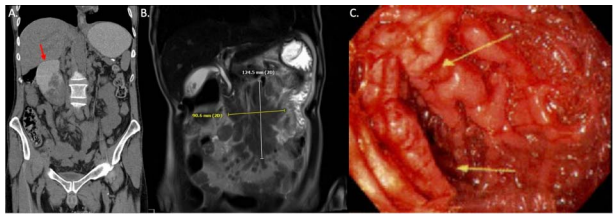
Introduction: Tuberculosis (TB) continues to have a significant disease burden and while the most common manifestation of TB is pulmonary, primary abdominal tuberculosis is exceedingly rare. Duodenal involvement occurs in only about 0.3 to 2.3% of patients with TB. Even in parts of the world where TB is endemic, gastroduodenal tuberculosis accounts for only about 2% of disease burden. We present a rare case of a 52 year-old male who presented with primarily abdominal findings of constipation, epigastric pain, and intractable vomiting. The patient was initially mistaken to have lymphoma but diagnosed with gastric outlet obstruction secondary to disseminated tuberculosis.
Case Description/Methods: A 52 year-old male with history of HIV presented to the ED with complaint of intractable non-bloody vomiting and severe epigastric abdominal pain. He reported a history of worsening constipation for which he had been seen in multiple EDs prior. On admission, the patient was found to have a lactic acidosis of 3.4 with severe abdominal distention and oral intolerance. A nasogastric tube was placed for gastric decompression. Computed tomography and magnetic resonance imaging revealed a conglomerate mesenteric nodal mass concerning for lymphoma. The mass was found to be compressing the horizontal segment of the duodenum resulting in a duodenal outlet obstruction. Gastroenterology was consulted and performed an endoscopic ultrasound which revealed diffuse mucosal changes and moderate stenosis of the second portion of the duodenum. Enlarged celiac, peripancreatic, and porta hepatis lymph nodes were observed with appearance suggestive of lymphoma. Fine needle aspiration was positive for acid fast bacilli. Due to concerns for active pulmonary TB, the patient underwent bronchoscopy and bronchoalveolar lavage with cultures positive for acid fast bacilli and positive nucleic acid amplification (NAA) testing for Mycobacterium tuberculosis. The patient was started on RIPE therapy. (Figure)
Discussion: Gastric outlet obstruction is an infrequently described presentation of TB. Our case highlights the importance of having a high index of suspicion for disseminated tuberculosis in an immunocompromised patient. Diagnosis requires either demonstration of caseating epithelioid granulomas or presence of acid-fast bacilli in tissue. GDTB is usually responsive to RIPE therapy. Surgical intervention is reserved for complications such as abscess, fistulas, or perforation.



[3502] **Figure 1.** (A) Computed tomography and (B) Magnetic resonance imaging revealed a conglomerate mesenteric nodal mass concerning for lymphoma. The mass was found to be compressing the horizontal segment of the duodenum resulting in a duodenal outlet obstruction. (C) EGD revealed diffuse mucosal changes and moderate stenosis of the second portion of the duodenum.

S3503

Neuroendocrine Tumor of Ampulla of Vater in Existing Tubulovillous Adenoma

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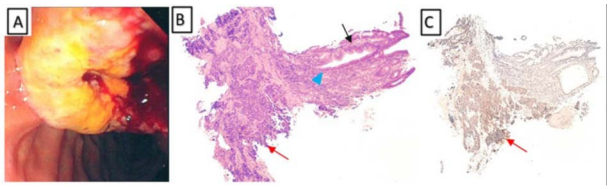
[1]Hurley Medical Center, Flint, MI; [2]Emory University School of Medicine, Atlanta, GA.

Introduction: Primary neuroendocrine carcinoma (NEC) of the ampulla of vater(AoV) is an extremely rare occurrence comprising 2% of periampullary neoplasms and < 1 % of gastrointestinal NEC. Likewise, ampullary adenoma is an uncommon occurrence with high predisposition for transformation to adenocarcinoma. Accordingly, their co-existence is sparsely reported. Herein we present a rare case of ampullary high grade neuroendocrine carcinoma (HGNEC) arising from a pre-existing tubulovillous adenoma.
Case Description/Methods: A 89 year old Jehovah witness male was evaluated for anemia 4 years ago and was found to have biopsy proven AoV tubulovillous adenoma. He was lost to follow-up after 6 months of surveillance esophagoduodenoscopy (EGD). He currently presents with severe bleeding per rectum with clots for one day. On presentation, he was hypotensive and with bright red blood mixed with stool on digital rectal exam. Laboratory work up revealed hemoglobin 8.1g/dL, alkaline phosphatase 748U/L, ALT 54U/L, AST 49U/L, Total bilirubin 1.8mg/dL and direct bilirubin 1.5mg/dL with elevated PT 17.6seconds. Given his ongoing bleeding, low hemoglobin and being a Jehovah witness, he was treated with IV iron sucrose, Aranesp, vitamin K, tranexamic acid. CT of the abdomen revealed extensive intra and extrahepatic bile duct dilation, common bile duct dilation (2.5 cm) and pancreatic duct dilation (1.2 cm). On MRCP, a small T2 hypointense lesion was identified near AoV. ERCP showed a large ulcerated mass measuring about 3 cm at the papilla, and the biopsy was significant for high grade small cell neuroendocrine carcinoma arising in a tubulovillous adenoma with immunohistochemical stains positive for CDX2, CD 56, SATB2 and synaptophysin. Ki-67 stain(cell proliferation marker) revealed positive in 100% of neuroendocrine cells. Biliary drain placement for hyperbilirubinemia (direct bilirubin 9.8mg/dL, 1 month from presentation) was done. Further course of action to be determined after correction of anemia and bilirubin. (Figure)
Discussion: One study reported 50% of ampullary HGNEC is associated with adenoma but pathogenesis is still unclear. Although a HGNEC was identified in pre-existing adenoma, we need to further evaluate the possibility of adenocarcinoma arising from adenoma as this could affect further management of chemotherapy. The ampullary HGNEC identified appears aggressive with rapid growth and high Ki-67 index. Pancreatoduodenectomy with local lymph node dissection is warranted for concerns of early nodal metastasis at this time.



[3503] **Figure 1.** (A) Endoscopic image of large ulcerated ampullary tumor with bleeding (B) H & E stain X40 showing tubulovillous adenoma (black arrow) segment of normal AoV epithelium (blue arrow head) and nest of neuroendocrine cell (red arrow) (C) Immunohistochemical stain with synaptophysin x40 showing uniform staining of the neuroendocrine cells (red arrow).

S3504

A 64-Year-Old Male With Recurrent Brunneroma

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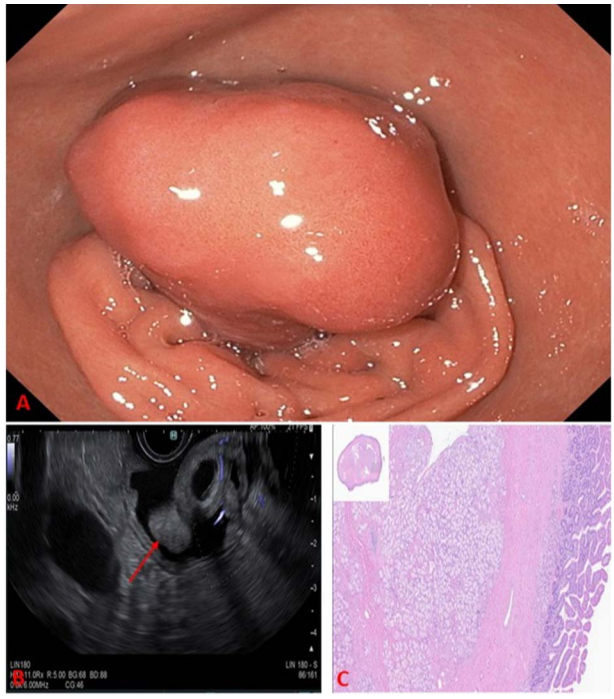
Introduction: Brunneroma (also termed Brunner's gland hyperplasia, adenoma, or hamartoma) is a rare benign tumor arising within the alkaline-producing exocrine glands located in the submucosal layer of the duodenum. Most patients with brunneroma are asymptomatic, but others may present with gastric obstruction-like symptoms or upper gastrointestinal bleed. Less than 200 cases have been reported. We present a case of a patient with recurrent Brunneroma.
Case Description/Methods: A 64-year-old male was evaluated for intermittent achy abdominal pain in the umbilical region associated with postprandial bloating and early satiety. He had a similar presentation twelve years ago and underwent esophagogastroduodenoscopy (EGD), which showed a 3 cm polypoid mass within the duodenal bulb. The mass was removed endoscopically through snare polypectomy and Roth basket retrieval, leading to the resolution of his symptoms. Given his previous history, an EGD was obtained, which showed a 4 cm tan-pink polypoid mass prolapsing from the duodenal bulb through the pylorus into the stomach (Figure A). Endoscopic ultrasound revealed a mucosal mass on a stalk with a cystic component and no submucosal involvement (Figure B). He was evaluated by an advanced endoscopist and felt the mass was appropriate for endoscopic removal. Therefore, an Endoloop was placed around the mass initially with the base injected with epinephrine and then removed with cautery, placing a clip on the base afterward. Removal of the mass led to the resolution of his symptoms. The pathology report demonstrated Brunner gland hyperplasia, benign metaplastic cysts arising from Brunner gland ducts, and no dysplasia (Figure C; H&E stain medium magnification).
Discussion: Brunner's gland is located in the proximal part of the duodenum above the hepatopancreatic sphincter. The mechanism Brunneroma remains unclear. There is currently no consensus regarding surgical versus endoscopic management of Brunneroma. However, there is increasing endoscopic utilization avoiding a more invasive surgical procedure.



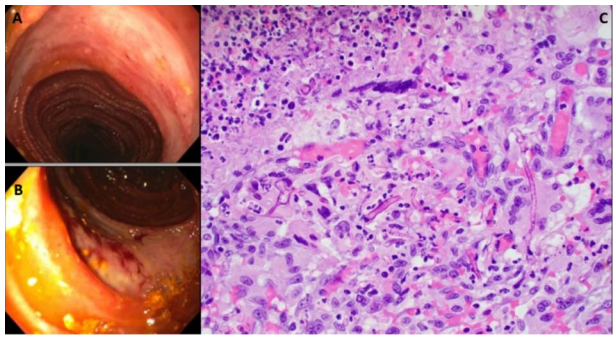
[3504] **Figure 1.**

S3505 Presidential Poster Award

A Case of Invasive Gastrointestinal Mold Infection

Tyson Amundsen, MD[1], Boris Zhong, DO, MS[2], James Risinger, BS[3], Mario A. Martinez, MD, MS[4], Hector Ramirez, MD[2].
[1]University of Tennessee Health Science Center, Bartlett, TN; [2]Baylor Scott & White, Temple, TX; [3]Texas A&M School of Medicine, Temple, TX; [4]Baylor Scott and White Health, Temple, TX.

IntroductionL: Invasive mold infections can be devastating and are mostly seen in patients with hematologic malignancies, profound neutropenia, solid organ or hematopoietic cell transplantation, and poorly controlled diabetes mellitus. Mucormycosis is caused by a group of ubiquitous molds called Mucormycetes and characterized by tissue infarction and necrosis that results from invasion of the vasculature by hyphae. We present a rare case of a patient with invasive gastrointestinal mold infection.
Case Description/Methods: A 40-year-old male with chronic inflammatory demyelinating polyneuropathy (CIDP) was admitted for autologous stem cell transplantation (ASCT). Initial hospitalization was complicated by neutropenic fever and C. difficile enteritis treated with Fidaxomicin. He was admitted one month later for nausea, vomiting and push enteroscopy revealed two circumferential deep small bowel ulcerations and a jejunal stricture too distal to be reached. Ulcer biopsies showed granulation tissue and was positive for Cytomegalovirus. He was treated with Valganciclovir and initiated on TPN. One week later, he had a third admission for intractable nausea and vomiting due to a small bowel obstruction. He failed conservative management and underwent diagnostic laparoscopy that revealed five strictures in the distal small bowel which were all resected with two small bowel resections and re-anastomosed. Histopathology of resected jejunum showed necrotizing granulomatous inflammation with aseptated fungal hyphae concerning for Mucormycosis, which was not identified on fungal PCR. He was treated with liposomal Amphotericin B then transitioned to oral Isavuconazole before making a full recovery. (Figure)
Discussion: The most common presentation of Mucormycosis is a rhino-orbital-cerebral infection. Gastrointestinal infection is rare in living patients and has been estimated to occur in around 7% of all Mucormycosis cases and carries mortality as high as 85%. Our patient had a history of refractory CIDP and underwent chemotherapy for ASCT that was complicated by development of C. difficile and CMV gastrointestinal infections and small bowel obstruction necessitating endoscopic intervention which led to exploratory laparoscopy for surgical resection and the diagnosis of invasive Mucormycosis. Although invasive GI Mucormycosis is rare, in severely immunocompromised patients with unexplained gastrointestinal symptoms and multiple risk factors as presented in our patient, this infectious organism can be considered in the differential.



[3505] **Figure 1.** (A, B) Circumferential ulcerations seen in the small bowel on push enteroscopy. (C) Mucosal ulceration with underlying necrotizing granulomatous inflammation in the deep muscularis propria/pericolonic adipose tissue with non-septated fungal hyphae present.

S3506

A Case of Jejuno-Jejunal Intussusception Caused by Underlying Metastatic Melanoma

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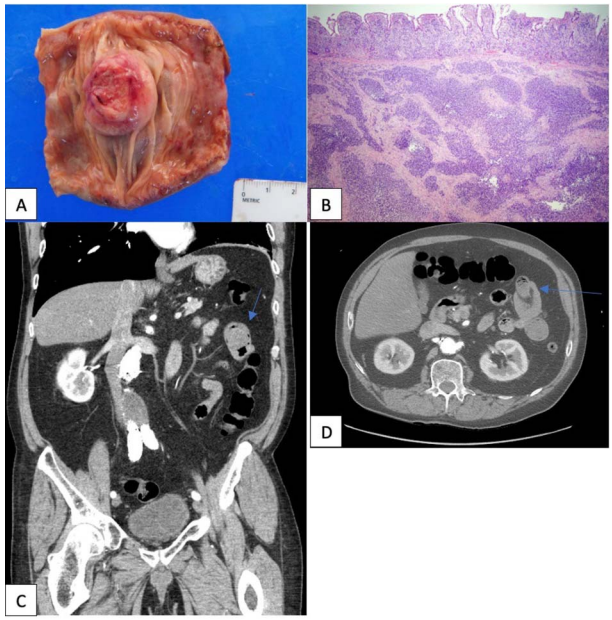
[1]Advocate Lutheran General Hospital, Chicago, IL; [2]Advocate Lutheran General Hospital, Park Ridge, IL.

Introducction: Intussusception in adults is a rare finding, with 95% of cases occurring in the pediatric population. If seen in adults, there should be a high degree of suspicion for a neoplastic process serving as the pathological lead point. Imaging is the primary modality for diagnosis, but exploratory laparotomy is often required posing an increased risk for morbidity and mortality.
Case Description/Methods: We present a 64-year-old male with history of alcoholism, 94 pack-year smoking, CAD on aspirin 81 mg daily, AAA repair, and Stage IV melanoma initially diagnosed in 2010 with mets to intestines/brain. He was treated with immunotherapy and brain radiation in 2015. He had been off medications for 5 years, with negative PET Scan within 2 years, who presented with 4 days of melanic stools and abdominal discomfort. Last colonoscopy 14 years prior demonstrated multiple tubular adenomas. On arrival, vital signs notable for mild tachycardia. Physical exam revealed conjunctival pallor and mild abdominal tenderness to palpation without rebound/guarding. Labs revealed hemoglobin 10.7 g/dL, platelet count 253,000/μL, AST 40, ALT 63, BUN/Cr 26, INR 1.0. An upper endoscopy showed mild duodenitis. Gastric biopsies were negative for H pylori and duodenal biopsies suggested gastric metaplasia and Brunner's glands hyperplasia. Given his vascular risk factors, CTA was obtained which revealed no evidence of active contrast extravasation but did demonstrate jejuno-jejunal intussusception with focal wall thickening for which an underlying lesion was difficult to exclude. He underwent exploratory laparotomy with findings of an 8cm intussuscepted bowel with a lead point mass that was resected with pathology revealing metastatic melanoma 3.1 x 2.7cm involving entire wall of small bowel. (Figure)
Discussion: Melanoma is one of the most rapidly progressive cancers. Metastasis of cutaneous malignant melanoma causing intussusception is a unique finding with few causes reported. Intussusception in adults can present with non-specific symptoms, and metastatic disease should be on the differential in any patient with history of melanoma. Unfortunately, about 50% of all patients treated for metastatic melanoma will eventually relapse, with about 20% being local, 50% in regional lymph nodes, and 30% with distant metastases [1]. Early detection increases the likelihood that the cancer is amenable to treatment (i.e., surgical resection) and exploratory laparotomy should be performed promptly in the right clinical context.



[3506] **Figure 1.** 1A: Opened small intestine resection specimen shows 3.1cm, umbilicated mass extending into lumen 1B: Nests of tumor cells diffusely invasive into mucosa (top), submucosa (mid), and muscularis propria (bottom) 1C: Arrow pointing to the metastatic lesion with surrounding air, viewed in coronal plane 1D: Arrow targeting the intussusception viewed in axial plane.

S3507

A Case of Duodenal Obstruction From Pancreatitis-Induced Duodenal Wall Hematoma

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[1]Wake Forest University School of Medicine, Winston-Salem, NC; [2]M. Islam Medical & Dental College, Winston-Salem, NC.

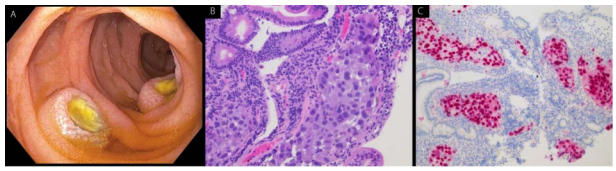
Introduction: Spontaneous duodenal wall hematomas are rare but well reported complications of pancreatitis. Patients can present with symptoms of small bowel obstruction and should initially be managed conservatively. Arterial embolization and laparoscopic drainage of the hematoma may be needed in patients who do not improve with supportive care.
Case Description/Methods: A 48-year-old male with a history of alcohol dependence and excessive non-steroidal anti-inflammatory drugs (NSAIDs) use presented with a one-month history of abdominal pain, nausea, and vomiting. The patient denied recent trauma or anticoagulant use. Two weeks prior, a computed tomography (CT) scan showed pancreatic head stranding and serum lipase was 172. Outpatient conservative management was planned but patient presented with worsening obstructive gastroenterology (GI) symptoms requiring a nasogastric tube. On examination, his temperature was 99.5, heart rate 112 and moderate diffuse abdominal tenderness was noted, greatest in the epigastric area. On labs, his lipase was 185. A CT abdomen showed a large descending duodenal wall hematoma with partial proximal obstruction and pancreatic head inflammatory changes related to pancreatitis. EGD showed collapsed 2nd portion of duodenum, without any mucosal lesion. The obstructive GI symptoms continued to worsen, and CT angiography showed enlarging duodenal wall hematoma, so evacuation was planned. In anticipation of evacuation, interventional radiology embolized the gastroduodenal and inferior pancreaticoduodenal artery to prevent further expansion of the hematoma followed by a laparoscopic drainage of the hematoma by surgery. Post-procedure, the patient's obstructive symptoms resolved.
Discussion: Blunt trauma, bleeding disorders, anticoagulant therapy, and iatrogenic factors are all potential causes of duodenal hematoma; however, our patient denied all these potential explanations. There have been multiple reports discussing how spontaneous duodenal hematomas may be a rare complication of pancreatitis. The mechanism is still unclear but it may be related to the release of proteolytic enzymes from an inflamed pancreas or ectopic pancreas, causing vascular erosions in the small bowel. Management includes supportive care with fluid resuscitation, pain control and NG decompression. A laparoscopic drainage of the hematoma may be required if hematoma is expanding with worsening obstructive symptoms and a prophylactic arterial embolization may help drainage by stabilizing the bleeding.

S3508

A Case of Metastatic Melanoma of the Duodenum

Eric Zhao, MD[1], Naveen Mallangada, MD[1], Kapil Gupta, MD[2], Mahmoud A. Ali, MBBCh[2], Anish Vinit Patel, MD[2].
[1]Rutgers Robert Wood Johnson Medical School, New Brunswick, NJ; [2]Rutgers Robert Wood Johnson University Hospital- New Brunswick, New Brunswick, NJ.

Introduction: Malignant melanoma can metastasize to the small bowel. Symptoms are non-specific, and most patients go un-diagnosed during their lifetime. When found, surgical resection significantly improves survival; however, overall prognosis remains poor.
Case Description/Methods: A 63-year-old male with history of metastatic melanoma with metastasis to the lungs and brain presented with abdominal pain and intermittent hematochezia. He was previously diagnosed with immune mediated colitis related to ipilimumab and nivolumab therapy that had resolved with steroid therapy and infliximab. Computerized tomography (CT) of the abdomen-pelvis did not reveal any bowel abnormalities. Endoscopic evaluation was pursued. Colonoscopy did not reveal any mucosal disease. However, upper endoscopy revealed five large and small-sized ulcerated masses in the duodenum (Image A). Biopsies of these masses revealed metastatic melanoma (Image B) with positive SOX-10 immunostaining (Image C). The patient was started on new immunotherapy with plans for further management with oncology.
Discussion: We present here a case of metastatic melanoma of the duodenum. Melanoma is the fifth most common cancer in the United States. The small bowel is the most common site of gastrointestinal (GI) tract metastasis. However, most patients with metastatic intestinal melanoma go un-diagnosed, as only 1.5% to 4.4% receive a clinical diagnosis. Common presentation of intestinal melanoma includes abdominal pain, anemia, obstruction and GI bleeding. Diagnostic modalities include abdominal ultrasound, contrast-enhanced CT, upper GI series, endoscopy, and capsule endoscopy. Imaging can reveal a polypoid mass with central ulceration, described as a target or a bulls-eye lesion. Endoscopic assessment typically reveals black colored ulcerated lesions. The treatment of choice for small bowel melanoma is complete resection. However, overall prognosis is still poor, as median survival for patients who undergo curative resection is 22 months. Immunotherapy is considered for multiple lesions. In conclusion, patients with known melanoma presenting with GI symptoms should be evaluated for small bowel involvement, either radiologically or endoscopically. These patients should be approached with a high index of suspicion, as early detection and complete resection can significantly improve survival. Prior history of immune mediated enterocolitis should not preclude repeat thorough endoscopic evaluation when patients have new onset of GI symptoms.



[3508] **Figure 1.** Image A: Endoscopic view of duodenal melanoma lesions Image B, C: Histologic view of duodenal melanoma lesions.

S3509

A Case of Diffuse Malignant Peritoneal Mesothelioma

Ariana R. Tagliaferri, MD[1], Gabriel Melki, MD[1], Walid Baddoura, MD[2].
[1]Saint Joseph's University Medical Center, Paterson, NJ; [2]St. Joseph's Regional Medical Center, Paterson, NJ.

Introduction: Malignant Mesothelioma occurs most commonly in the pleural space but can also occur in the peritoneum in those with an extensive history of asbestos exposure. Peritoneal mesothelioma (MPeM) is relatively rare accounting for 15% of all mesotheliomas. Most patients are asymptomatic until advanced stages but will present with nonspecific abdominal symptoms. We present a case of MPeM in a male who worked in a wire hanger factory for 20 years.
Case Description/Methods: An 81-year-old Arabic male presented with nausea, vomiting, abdominal distention and diarrhea lasting three weeks. His vitals were remarkable for hypertension (192/102 mmHg), and he had diffuse abdominal tenderness without peritonitis on physical exam. CT A/P with contrast showed fluid distending the small and large bowel and mild fat stranding and wall thickness of the ascending and descending colon. Ascites was also noted. Laboratory studies revealed elevated ALT (54 unit/L) with otherwise normal AST, ALP and bilirubin. CRP (87 mg/L), ESR (30 mm/hr) and WBC's (12.9 x10^3/mm3) were all elevated. A repeat CT A/P was suggestive of small bowel obstruction and he underwent a diagnostic laparoscopy with right hemicolectomy, ileocolonic anastomosis and omentectomy. The resected obstructing mass was diffuse MPeM, epithelioid type extensively involving the small bowel muscularis propria and submucosa and focally involving the lower mucosa of the ileocecal region (T0N0). Immunohistochemical analysis showed strong positive reactions with calretinin, CK 5/6, WT-1 and D2-40. Negative reaction was present with CK20, synaptophysin, chromogranin, and prostatic markers. He received 12 cycles of nivolumab, 7 cycles of ipilibumab and palliative radiation due to increasing tumor burden but was ultimately transferred to hospice approximately 2 years after initial diagnosis.
Discussion: MPeM as the primary source of mesothelioma is uncommon. Asbestos exposure confers a lower risk of MPeM in males. Patients with MPeM are typically younger at the time of diagnosis than those with pleural involvement and are usually women. Females have prolonged survival due to less aggressive histological subtypes, such as epithelial. The 3- year survival rate for MPeM after treatment is 39% for males. Our patient was 81 years-old at time of diagnosis and was a male who was likely exposed to asbestos for over 20 years. He outlived his life expectancy given his age, gender, location of mesothelioma, etiology and histological subtype.

S3510

***Clostridium difficile* Phlegmonous Enteritis With Microperforation**

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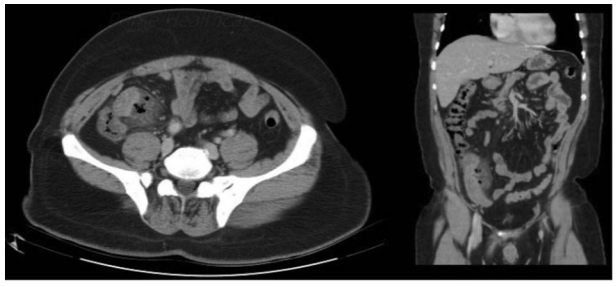
Introduction: Clostridium difficile infection (CDI) is typically associated with the colonic involvement preceded by leading risk factors such as antibiotics, old age, immunodeficiency and recent hospitalization. Diarrhea is the most frequent manifestation along with hypovolemia, sepsis and toxic megacolon however, to our knowledge, CDI causing phlegmonous ileum or enteritis leading to microperforation has never been reported in the literature.
Case Description/Methods: 51 y/o Hispanic male with no PMH presented with a 6-day history of left lower quadrant (LLQ) abdominal pain associated with 4 to 5 episodes of watery diarrhea per day. He denied any recent antibiotic use or hospitalizations. He lived with his sister who was a healthcare provider. Family history was unremarkable. Admission vitals were unremarkable. Physical exam was notable for RLQ tenderness and the digital rectal exam was normal. Labs showed leukocytosis of 11,400 cells/mm3 (4,400 - 11,000 cells/mm3), sed rate of 56 mm/hr (0-30mm/hr), CRP 6mg/dL (0-0.8 mg/dL) and stool for C. difficile PCR was positive. Stool ova & parasite, culture, ANCA panel and saccharomyces cerevisiae antibodies were negative. CT abdomen/pelvis showed phlegmonous changes in the RLQ with mesenteric extraluminal air and phlegmonous loop of terminal ileum. Patient was started on oral vancomycin and IV metronidazole and later on, metronidazole was discontinued and oral vancomycin was continued with complete resolution of his symptoms. (Figure)
Discussion: In a patient with Clostridium difficile colonization during autopsy, the bacterium was isolated in segments of the jejunum raising the possibility that the small bowel may be a reservoir for the pathogen. CDI presenting as phlegmonous enteritis (PE) has never been reported. PE is characterized by suppurative bacterial infection of the intestine affecting the submucosa. Also, CDI involving the small bowel is typically seen in patients with inflammatory bowel disease (in ~50% of cases), immunosuppression, prolonged bowel discontinuity and/or colectomy with ileoanal anastomosis. Our patient is unique in having none of the risk factors for CDI except for having household contact with a healthcare worker. It is imperative physicians consider this presentation of CDI as PE with microperforations.



[3510] **Figure 1.** CT Abdomen Pelvis showing phlegmonous changes in the right lower quadrant with a small amount of extraluminal air with phlegmonous loop of terminal ileum.

S3511

A Case of Mycobacterial Infection Causing Necrotizing Granulomas of the Gastrointestinal Tract and Retroperitoneal Lymphadenopathy

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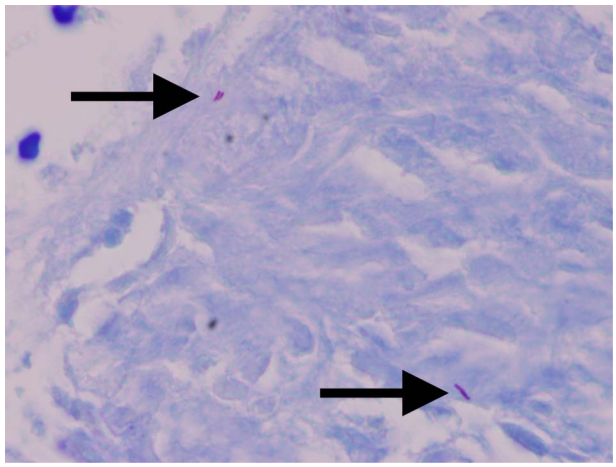
Introduction: Various species of *Mycobacterium*, best known for their role in causing pulmonary tuberculosis, have rarely been described in gastrointestinal disease. Historically, *Mycobacterium avium* and *Mycobacterium paratuberculosis* have been found in tissue samples in patients with terminal ileitis, and have previously been described in patients with Crohn's disease. Our study describes a rare case of mycobacterial infection with intestinal involvement.
Case Description/Methods: A 44-year-old female with a history of ankylosing spondylitis treated with adalimumab and travel to Mexico 2 years ago endorsed 5 weeks of worsening epigastric pain, unintentional weight loss, diarrhea, and arthralgias during an outpatient visit. Review of computed tomography scans 3 and 10 years prior described prominent retroperitoneal lymph nodes. She was referred to interventional radiology for lymph node biopsy which revealed necrotizing granulomatous lymphadenitis. She was admitted to the hospital and computed tomography was notable for mural thickening and

inflammatory stranding of the distal small bowel, particularly the terminal ileum. Esophagogastroduodenoscopy appeared normal. Colonoscopy displayed an edematous ileocecal valve and terminal ileum with ulcers as well as erosions of the cecum and proximal ascending colon. Biopsy of the ileum demonstrated necrotizing granulomatous inflammation and biopsy of the colon was consistent with focal poorly formed granulomatous inflammation. *Mycobacterium* PCR and staining of the duodenal biopsy was positive for acid-fast bacilli (see figure). The *Mycobacterium* polymerase chain reaction testing on the same duodenal sample was negative. Serum interferon-gamma release assay was positive. She was started on rifampin, isoniazid, pyrazinamide, and ethambutol to be taken for 2 months with plan to take an additional 7 months of rifampin and isoniazid. She experienced improvement in abdominal pain after initiation of antibiotic therapy.

Discussion: Granulomatous ileitis or other intestinal manifestations of *Mycobacterium* species are rare, and have been previously described in the Crohn's disease population. This case of *Mycobacterium* infection represents an unusual etiology for intestinal inflammation and erosions in a patient with an autoimmune condition but without known Crohn's disease. This case highlights the importance of including mycobacterial diseases within the differential for gastrointestinal tract lesions.



[3511] **Figure 1.** Acid-fast bacilli on duodenal biopsy.

S3512

A Case of Partial Small Bowel Obstruction due to a Rare Type of Intestinal T-Cell Lymphoma

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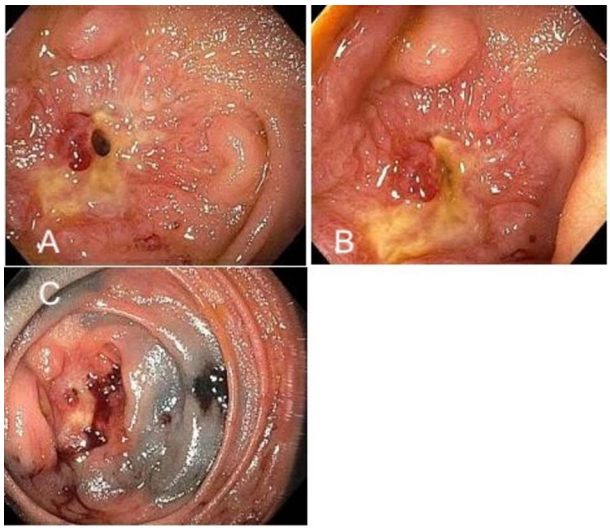
Introduction: Monomorphic epitheliotropic intestinal T-cell lymphoma (MEITL), formerly Type II enteropathy associated T-cell Lymphoma (EATL), is a rare and aggressive form of T-cell lymphoma. We present a case of MEITL presenting as a partial small bowel obstruction (SBO).

Case Description/Methods: 70-year-old Afro-Caribbean female with medical history of breast cancer, meningioma, gastroesophageal reflux disease and cholelithiasis presented to the emergency room with epigastric pain, non-bloody, non-bilious emesis, and inability to tolerate oral intake. She reported occasional odynophagia to solids and liquids and 30lb unintentional weight loss. The patient was admitted to the general medical floor for intractable nausea. CT abdomen and pelvis showed a thickened distal esophagus. Esophageal pathology was suspected, and the patient underwent an esophagogastroduodenoscopy (EGD) which revealed no gross lesions, however >700cc of bilious fluid and food content were removed. Small bowel series revealed a SBO with transition point at the proximal jejunum and was treated conservatively. Push enteroscopy showed severe stenosis in proximal jejunum with ulceration, and the colonoscope could not be advanced. The patient underwent exploratory laparotomy and small bowel resection. Pathology from the ex-lap revealed MEITL. PET scan revealed widely metastatic disease including lung, liver, pancreas, bowel, adnexa, and peritoneum. (Figure)

Discussion: EATL, formerly classified into Type I and Type II, makes up less than 5% of all gastrointestinal tumors and less than 1% of non-Hodgkin lymphomas. EATL, formerly Type I, is associated with celiac disease and typical presentation includes diarrhea with constitutional symptoms. MEITL is a primary intestinal T-cell lymphoma comprised of intraepithelial lymphocytes. Literature has proposed that MEITL is distinct from EATL due to lack of pre-existing enteropathy. MEITL has an increased incidence in Asian and Hispanic populations. The most common symptoms of MEITL are diarrhea and weight loss. Symptoms present late and metastatic disease to extra-gastrointestinal sites is common. MEITL is most common in the proximal jejunum, thus is often missed by both EGD and colonoscopy and therefore likely underdiagnosed. In our case, the lymphoma proliferated causing mass effect in the proximal jejunum resulting in SBO. No specific treatment has been proven to improve outcomes of MEITL, including chemotherapy, radiotherapy and/or surgery, thus prognosis remains poor with a median survival of 7 months.



[3512] **Figure 1.** Image A proximal jejunum Image B proximal jejunum Image C proximal jejunum.

S3513

A Challenging Case of Eosinophilic Gastroenteritis!

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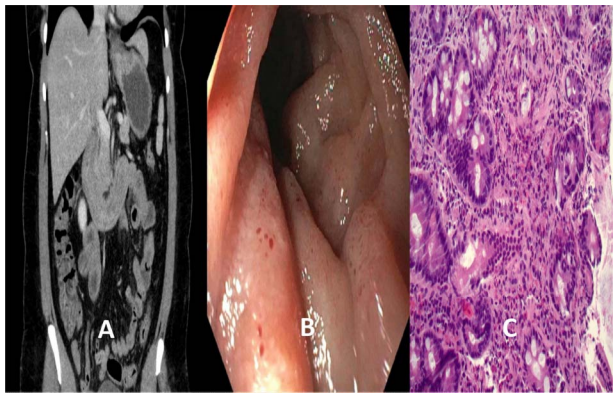
Introduction: Eosinophilic gastroenteritis (EGE) is an uncommon infiltrative eosinophilic disease. It is part of the spectrum of eosinophilic gastrointestinal disorders that also comprises eosinophilic esophagitis and eosinophilic colitis. Despite that EGE is described in the literature, its infrequent occurrence, unclear pathophysiology, and nonspecific symptoms result in substantial cases of missed and delayed diagnosis. Thus we report the case of a middle-aged woman who got diagnosed with EGE 15 years after the onset of her symptoms.
Case Description/Methods: A 43-year-old woman with a history of hypertension presented with worsening epigastric pain and postprandial vomiting for the last few weeks. She reports similar yearly episodes starting 15 years ago resolving spontaneously after a few weeks. Medications consisted of lisinopril and felodipine. Physical examination revealed epigastric tenderness. Lab tests showed a normal WBC of 8.4 ($x10^9$/L) with elevated eosinophil (Eo) count of 3.21 ($x10^9$/L) [normal range 0.00-0.70 ($x10^9$/L)] and high IgE level of 320 IU/mL [normal range 0-214 IU/mL]. Liver and kidney tests, blood smear, HIV serology, B12, tryptase levels, stool examination for ova and parasites were unrevealing. Abdominal CT scan showed wall thickening of the stomach and small intestine (Panel A). Upper endoscopy showed erythema and nodularity of the gastric and duodenal mucosa (Panel B). Biopsies showed 40 Eo/HPF in the gastric mucosa and 100 Eo/HPF in the duodenal mucosa suggestive of EGE (Panel C). Colonoscopy and colon biopsies were unremarkable. Prednisone 40 mg daily was started with noted rapid resolution of symptoms within a few days of therapy. The patient was discharged home with a prednisone taper over 8 weeks.
Discussion: EGE is a rare disorder with less than 300 reported cases. A personal or family history of atopy is frequently described. Pathogenesis is speculated to be related to a hypersensitivity response with eosinophilic infiltration of the bowel wall. Symptoms are nonspecific. Diagnosis relies on the presence of > 20 Eo/HPF on histopathological inspection of gastric and duodenal biopsies and exclusion of other pathologies potentially associated with bowel wall eosinophilia. Despite that spontaneous remission can be seen, the majority of patients require treatment. Current available therapeutic choices include dietary modifications and pharmacological options that mainly involve corticosteroids. Recurrence of symptoms is commonly reported requiring long-term treatment.



[3513] **Figure 1.** Panel A - CT Abdomen and pelvis showing wall thickening of stomach and small intestine Panel B - Upper endoscopy showing erythema and nodularity of the gastric and duodenal mucosa Panel C- Biopsy of the duodenal mucosa showing up to 100 eosinophils per high power field (Hematoxylin and eosin, original magnification 20 x).

S3514

A Case of Symptomatic Ileal Non-Secretory Neuroendocrine Tumor Successfully Treated With Octreotide

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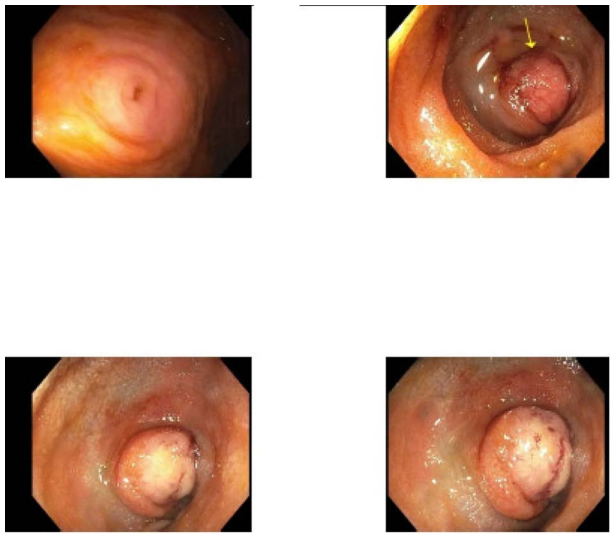
Introduction: Neuroendocrine tumor incidence is steadily increasing, likely from detection due to better imaging, routine colonoscopies, and endoscopies. Most NETs, 55%, are found in the GI tract and only 0.1-1% found on routine colonoscopy. About 40% of NETs are hormone producing leading to the clinical diagnosis of carcinoid syndrome. One of the most common symptoms of carcinoid syndrome is diarrhea occuring in 80% percent of cases, however diarrhea can be present in non-secretory NETs. Here we report a case of GNET presenting as diarrhea that responded to octreotide injections despite having normal 5-HIAA and chromogranin A levels.
Case Description/Methods: Our patient is an 80 year old female with past medical history of hypertension, renal stones, and IBS with baseline diarrhea of about 2-4 pasty stools a day. In November she developed two days of severe diarrhea with 10 watery stools a day and multiple episodes of incontinence which prompted patient to go to the ED. The inpatient workup was negative for acute causes, CT showed diverticulosis, and stools returned to baseline. The patient was discharged with outpatient GI follow up. At home the severe diarrhea retuned so outpatient GI planned for colonoscopy. Results of the procedure included a 2cm submucosal non-obstructing mass in the terminal ileum. Pathology showed a G1 well differentiated neuroendocrine tumor. Patient was referred to oncology and workup revealed no metastasis on PET/CT and negative 5-HIAA and chromogranin A levels. During initial oncology visit patient was started on octreotide injections which greatly improved the diarrhea. Even with negative carcinoid workup the decision was made to continue octreotide injections and not to pursue surgical options. (Figure)
Discussion: The terminal ileum is the most common GI location for NETs. Terminal ileum intubation on coloscopy is attempted 30-80% and greatly aids in diagnosis, as with our patient. Once diagnosed, there is limited guidance on treatment of diarrhea in patients with NETs and negative carcinoid workup. There are no abstracts or research on treatment of diarrhea with negative carcinoid syndrome. One treatment for carcinoid syndrome is somatostatin analogues but no guidelines with non-carcinoid NETs and therapeutic value in octreotide. However, with our patient, treatment with monthly octreotide injections resolved their symptoms. Our case emphasizes importance of ileal intubation in coloscopy and that somatostatin analogue treatment should be considered in non carcinoid NETs.



[3514] **Figure 1.** Small bowel (terminal ileum): well-differentiated neuroendocrine tumor (G1).

S3515

A Case of Symptomatic Lymphoid Hyperplasia

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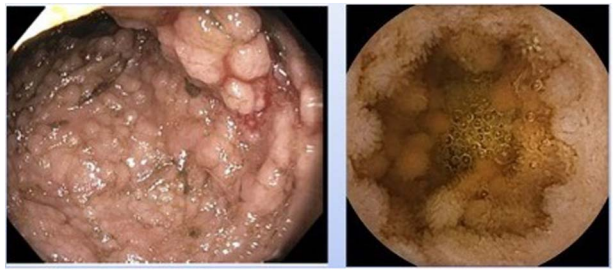
Introduction: Nodular lymphoid hyperplasia (NLH) is a rare condition whose pathogenesis is largely unknown. It is characterized by the presence of multiple small nodules usually along the small intestine. It can present with gastrointestinal bleeding, diarrhea, abdominal pain, or can be asymptomatic. Given its association with several diseases including immunodeficiency, it may represent functional compensation of immune tissue or even a transitional stage in the development of malignancy. Despite this, there are no clear guidelines on its management or surveillance.
Case Description/Methods: This is a case of a 21-year-old male patient who presented to the hospital with painless hematochezia. He reported negative work-up 3 years prior for iron deficiency anemia. He underwent Meckel's scan, upper endoscopy and colonoscopy as well as video capsule endoscopy which only demonstrated diffuse nodular mucosa in the terminal ileum which was biopsied. This revealed small intestinal mucosa with expansion of lamina propria by follicular lymphoid hyperplasia with germinal centers and small lymphocytic proliferation with negative immunohistochemical stains and flow cytometry. Work up also revealed hypogammaglobinemia concerning for Common Variable Immunodeficiency (CVID). He was ultimately managed supportively with a plan for close outpatient follow-up. (Figure)
Discussion: Clinically, it is important to recognize that the patient's presentation can be explained by the finding of NLH itself. Furthermore, NLH, though a benign condition, has been implicated as a risk factor for intestinal lymphoma. Reported cases demonstrate distinct pathologic diagnosis either simultaneously or over time after the diagnosis of NLH. Despite this, there is no consensus on surveillance or risk-mitigating strategies. Therefore, recognition of this association with careful follow-up is essential and further research on the topic should be considered.



[3515] **Figure 1.** NLH seen on colonoscopy and video capsule endoscopy.

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2. Matuchanksy C., Touchard G., Lemaire M. Malignant Lymphoma of the Small Bowel Associated with Diffuse Nodular Lymphoid Hyperplasia. N Engl J Medicine 1985. https://www.nejm.org/doi/10.1056/NEJM198507183130307

S3516

A Case of Recurrent Thromboangiitis Obliterans (Buerger's Disease) in a Patient With Abdominal Pain and Intestinal Transplant

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Introduction: We describe a case of abdominal pain and recurrent disease following intestinal transplant in a patient with a history of mesenteric ischemia and Thromboangiitis obliterans (Buerger's syndrome) that did not resolve with standard medical therapy.
Case Description/Methods: A wonderful 44 year old female presents with intestinal infarction. The patient required three prior revascularization procedures with acute on chronic mesenteric ischemia with superior mesenteric artery thrombosis. She was taken to the operating room for necrotic small bowel with resection 20 centimeters distal to the ligament of treitz and placed on parenteral nutrition. Following recurrent episodes of line related bacteremia and fungemia she was listed for intestinal transplantation. After being off tobacco for a year she received and small bowel and right colon transplant. She had initial anticoagulation which was restarted four year later after a deep venous thrombosis to the lower extremity. Seven years following transplant the patient had inconsistent use of the anticoagulation along with recurrent tobacco abuse and developed recurrent mesenteric ischemia with acute on chronic occlusion of the celiac trunk, superior mesenteric artery, and inferior abdominal aorta leading to generalized abdominal pain, ischemic colitis and septic shock. Given recurrent abdominal infections patient had enterectomy 8 years and six months following intestinal transplantation. The patient subsequently developed bacteremia, candidemia and line related thrombosis. Patient had eventual transition to palliative management and died.

Discussion: Buerger's disease (also known as thromboangiitis obliterans) affects blood vessels in the body, most commonly in the arms and legs. The use of tobacco is essential for both the initiation and progression of this disease. Blood vessels swell, which can prevent blood flow, causing clots to form. This can lead to pain, tissue damage, and even gangrene. It is typically treated with medications that dilate blood vessels, improve blood flow or dissolve blood clots, such as anticoagulants. Ongoing tobacco cessation is vital to success following rare intestinal transplantation. We present a case of intestinal transplant for Buerger's Disease with recurrent thrombosis seven years following intestinal transplant attributed to recurrent tobacco abuse.

S3517

A Rare Case of Large Brunner Gland Hyperplasia

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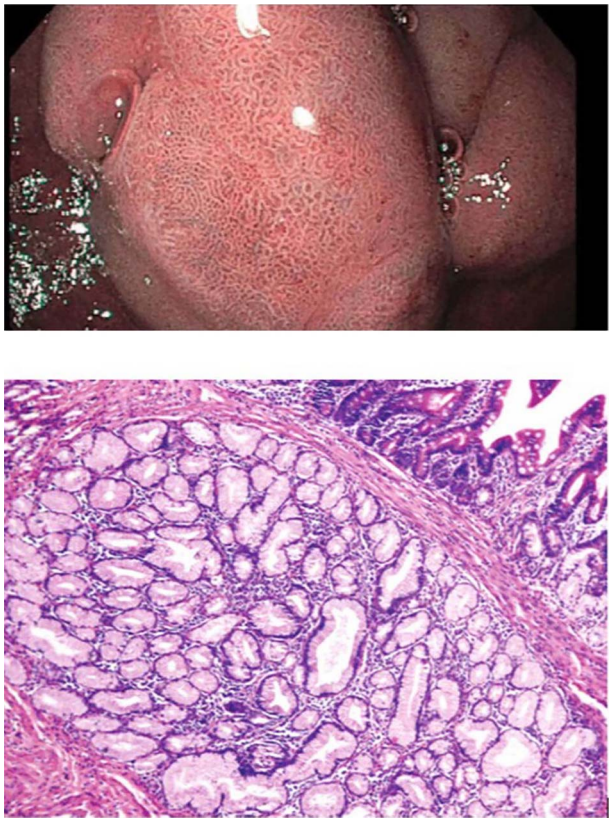
Introduction: Brunner's gland hyperplasia (BGH) is a rare, typically benign lesion of the small intestine, averaging 1-2 cm in size, and is usually diagnosed in the fifth or sixth decade of life with equal sexual predilection. It typically presents asymptomatically and is diagnosed incidentally during upper endoscopy, but can sometimes present in the setting of acute gastrointestinal hemorrhage, chronic anemia, or intestinal obstruction.
Case Description/Methods: We present a case of BGH that was measured to be 5 cm and diagnosed incidentally during upper endoscopy in the setting of investigating for causes of iron deficiency anemia (IDA) in a 67-year-old- male patient who also had a concomitant Helicobacter pylorus (H. pylori) infection. (Figure)
Discussion: BGH is caused by hyperplasia of the exocrine glands within the duodenum but its etiology still remains unknown. Brunner's glands are tubular mucus glands in the mucosa and submucosa of the duodenum that secrete mucus, which alkalinizes chyme from the stomach. Since Brunner's glands serve the purpose of increasing pH, it is postulated that increased acid secretion could cause these glands to undergo a state of hyperplasia. Other studies have suggested that H. pylori could possibly play an etiological role since most BGH cases are associated with concomitant H. pylori infection. BGH is typically diagnosed in asymptomatic patients and discovered incidentally on upper endoscopy, but clinical manifestations can consist of gastrointestinal bleeding, IDA, or duodenal obstruction. Diagnosis is made via endoscopic biopsy and histopathological analysis will reveal a lack of encapsulation and dysplasia. Management guidelines are nonexistent and although BGH is typically a benign lesion, few case reports have identified malignant transformation, which should elicit caution in presuming all lesions to be invariably benign. BGH lesions larger than 2 cm can cause more complications than smaller sizes and removal is recommended regardless if the patient is asymptomatic. Endoscopic polypectomy is typically the first-line treatment with surgical excision reserved for only massive lesions or polypectomy failure. This case highlights an example of a very large 5 cm, benign, pedunculated BGH lesion incidentally found within the duodenal bulb of a male patient, who presented for the purpose of being worked-up for IDA, and was also found to have a concomitant H. pylori infection, thereby further validating the purported hypothesis of H. pylori as an etiological factor in BGH.



[3517] **Figure 1.** Top: 5 cm polyp found within the duodenal bulb with biopsy results that revealed Brunner gland hyperplasia. Bottom: Example of photomicroscope of Brunner's gland hyperplasia showing hyperplastic lobules of Brunner's glands. Reference: Nandini D. Patel, Angela D. Levy, Anupamjit K. Mehrotra, and Leslie H. Sobin. American Journal of Roentgenology 2006 187:3, 715-722.

S3518

A Rare Case of Nivolumab-Induced Apoptotic Enteropathy

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Introduction: We report a case of Nivolumab induced apoptotic enteropathy in a patient presenting with abdominal pain and diarrhea. Nivolumab and Pembrolizumab are Programmed cell death protein 1 (PD-1) blocking agents. There are very few cases of Pembrolizumab-induced apoptotic enteropathy in the literature. This is a unique and first case of Nivolumab-induced apoptotic enteropathy diagnosed by small bowel enteroscopy.
Case Description/Methods: A 70-year-old male patient with stage IV Hodgkin lymphoma on treatment with Nivolumab in combination with AVD (Adriamycin, Vinblastine, and Dacarbazine) chemotherapy presented with abdominal pain, diarrhea, and hypotension. He received two cycles of chemotherapy, the last one was 2 weeks before admission. In the hospital, he was started on broad-spectrum antibiotics and pressors for sepsis and hypotension. Stool work-up for diarrhea was negative for infectious etiologies. CT abdomen showed diffuse small bowel wall thickening and mucosal hyperenhancement consistent with diffuse enteritis. Colonoscopy showed congested, inflamed, and ulcerated mucosa in the terminal ileum, and entire colon biopsies showed ischemic ileitis and colitis. Upper endoscopy showed erythema, mild

erosions, and inflammation in the duodenum, pathology showed nonspecific duodenitis. Fecal calprotectin was elevated at 1340 (normal < 49) and with a suspicion of ICI (Immune Check Point) inhibitor enteritis, he was started on intravenous steroids. With continued symptoms, a small bowel enteroscopy was performed which showed hemorrhagic mucosa with diffuse oozing, and linear erosions in the duodenum and jejunum. The jejunal biopsies showed cryptitis, crypt dilatation and distortion and rare apoptotic bodies suggestive of apoptotic enteropathy. The patient's course was complicated by pseudomonal pneumonia, hypoxic respiratory failure requiring intubation, and ventilator support. He was transitioned to comfort care and later passed away.
Discussion: ICI inhibitors like PD-1 blocking agents are novel immune therapeutics used for the treatment of advanced-stage malignancies. Apoptotic enteropathy can occur following treatment with antimetabolites and TNF inhibitors and rarely on Pembrolizumab. This is a unique case as the initial endoscopy showed non specific changes in the duodenum and diagnosis was confirmed on small bowel enteroscopy. This case highlights the importance of suspecting and identifying Nivolumab induced enteropathy by an enteroscopy and deeper biopsies.

S3519

A Rare Case of Epigastric Pain Caused by Duodenal Metastatic Cervical Cancer

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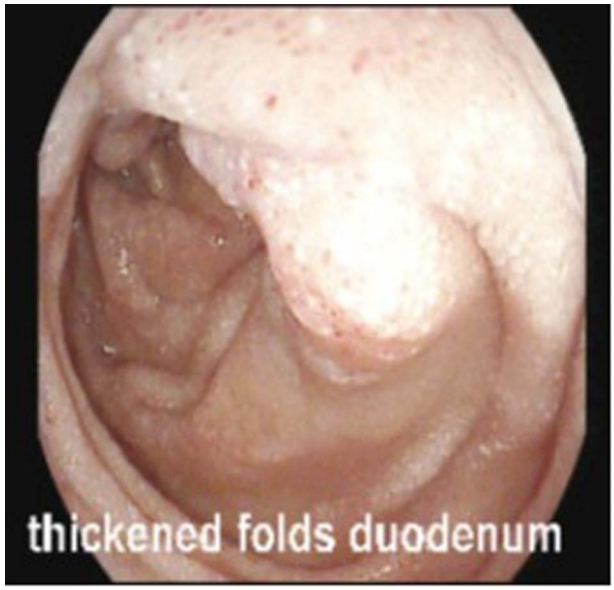
Introduction: Cervical cancer was previously one of the most common causes of death for women in the United States but survival has since improved with the use of screening and the HPV vaccine. Metastatic cervical cancer spread to the duodenum is rare and presents a diagnostic challenge due to vague symptoms. We present a case of epigastric pain caused by invasive squamous cell cervical cancer with duodenal metastasis.
Case Description/Methods: A 59-year-old female with a history of GERD and treated cervical cancer (2 years prior) and currently on pembrolizumab presented to clinic for 3 months of progressive upper abdominal pain and dyspepsia. Last upper endoscopy a year ago was normal. Physical exam was notable for epigastric tenderness without guarding. Laboratory data was remarkable for downtrending hemoglobin, normal liver function tests, normal lipase, and negative stool H pylori antigen. She was referred for EGD. A contrasted CT of the abdomen and pelvis performed revealed multiple enlarged lymph nodes of the mesentery. Upper endoscopy revealed thickened folds of the second portion of the duodenum and multiple biopsies taken of this site. It was an otherwise normal upper endoscopy. The duodenal biopsies were positive for metastatic squamous cancer of the cervix. The patient was transitioned to treatment with gemcitabine and carboplatin due to progressive disease on pembrolizumab. She unfortunately succumbed to her disease six months later. (Figure)
Discussion: There is an estimated 7.5 per 100,000 new cases of cervical cancer per year with death rate of 2.2 per 100,000 in the United States. Cervical cancer is usually locally invasive and spreads by lymphatics or hematogenously with most common metastatic sites of lungs, liver, and bone. In a previous review, liver metastases was in up to 12.5% of patients but did not mention a single case of luminal disease. It is very rare for most cancers to metastasize to the gastrointestinal tract, but when present, metastasis has a predilection for the stomach and duodenum. Few case reports are available for cervical cancer metastasis to the GI tract specifically, with about 16 identified. These patients generally presented with gastrointestinal bleeding, bowel perforation, nonspecific abdominal pain or obstructive symptoms. In conclusion, duodenal metastasis of cervical cancer is rare and can be frequently missed. A high degree of suspicion can expedite referral for upper endoscopy to diagnose and allow for treatment to extend patients' quality of life.



[3519] **Figure 1.** Thickened folds in the second portion of duodenum.

S3520

A Rare Case of Gastrointestinal Amyloidosis Due to Monoclonal Gammopathy of Undetermined Significance

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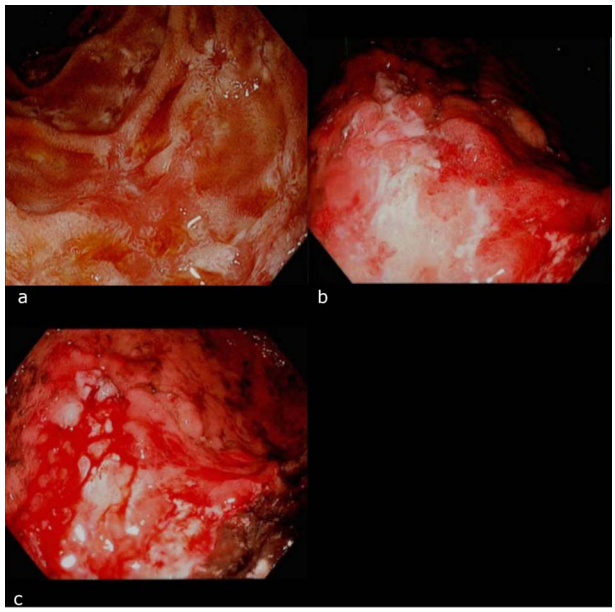
Introduction: Amyloidosis of the gastrointestinal (GI) tract is an uncommon disorder caused by extracellular deposition of fibrils made up of a variety of serum proteins. Symptomatic GI amyloidosis presents with one of four syndromes: GI bleeding, malabsorption, protein-losing gastroenteropathy, or GI dysmotility. Diagnosis of GI amyloidosis requires a tissue biopsy with positive Congo red staining or the presence of amyloid fibrils on electron microscopy. The prognosis of patients with amyloidosis and GI involvement appears to be worse than those without.
Case Description/Methods: A 64-year-old female with a past medical history significant for hypertension, systolic heart failure, depression, hyperlipidemia, and hypothyroidism presented with right lower quadrant abdominal pain. On examination, her lungs were clear, abdomen was benign, and vitals were stable. CT scan of the abdomen showed evidence of mesenteric, upper abdominal, and retroperitoneal adenopathy as well as omental findings concerning for metastatic disease or carcinomatosis. An EGD with biopsy revealed multiple gastric and duodenal ulcers. She presented five months later with hematemesis, weight loss, melanotic stool, and abdominal pain. EGD revealed findings suggestive of gastric malignancy involving the body and cardia of the stomach. CA 125 and LDH levels were elevated with increased adenopathy in the chest. Biopsy of gastric mucosa and duodenum showed findings suggestive of amyloidosis, with AL (Kappa) type amyloid deposition and positive Congo red staining. Kappa chain was detected in urine and serum as well, suggesting multiorgan involvement. Bone marrow biopsy revealed findings consistent with MGUS. A PET/CT demonstrated moderate irregular gastric wall thickening and infiltration of the omental fat with concern for intraperitoneal spread. She was started on CyBorD nine months after initial presentation. She died at home one month later. (Figure)
Discussion: Case reports concerning GI amyloidosis have reported on multiple myeloma, MGUS, or were not associated with a plasma cell dyscrasia. Two other case reports presented with MGUS at different stages, from an incidental finding to advanced disease with severe complications such as hematemesis, anemia, and weight loss. Four of these six patients improved with treatment while another two succumbed to the disease. This case illustrates that, while rare, it is important for clinicians to be aware of GI amyloidosis as early diagnosis is imperative for effective treatment and prognosis.



[3520] **Figure 1.** (a) Lining of the first part of the duodenum. (b-c) Active duodenitis in the first and second portions of the duodenum.

S3521

A Rare Case of Duodenal Leiomyoma

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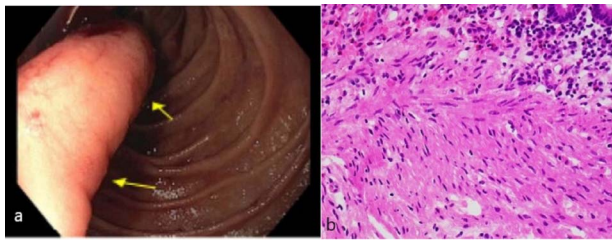
Introduction: Only 1–5% of gastrointestinal neoplasms are found in the small intestine. Adenocarcinomas, lymphomas, and neuroendocrine tumors are the most common malignant tumors seen in the small intestine, while gastrointestinal stromal tumors, leiomyosarcomas, and leiomyomas are uncommon. The jejunum, ileum, and duodenum are the three most common locations for leiomyomas. We are presenting a rare case of a female with duodenal leiomyoma.
Case Description/Methods: A 70-year-old female presented to the hospital with complaints of fatigue and weakness. Laboratory evaluation showed iron deficiency anemia with hemoglobin 7.5g/dl and hematocrit 25.4%. The patient reported an episode of black tarry stool about a week before the presentation. The patient received one packed red blood cell transfusion, and upper gastrointestinal endoscopy (EGD) was performed to evaluate anemia. EGD revealed non-erosive gastritis and gastric erosions without any current or recent bleeding stigmata. A single large pedunculated polyp was found in the duodenum's first portion, extending into the second portion of the duodenum(a). Biopsy from the polyp revealed submucosal leiomyoma without any evidence of dysplasia or malignancy(b). The next day, a colonoscopy showed significant diverticulosis and non-bleeding internal hemorrhoids. The patient remained hemodynamically stable and was discharged home the next day. The patient presented about a month later with iron deficiency anemia again. Repeat EGD and colonoscopy performed this time showed no obvious source of bleeding. We also conducted a pill cam study, which also came back negative. (Figure)
Discussion: It is unusual for leiomyomas, which are benign smooth muscle tumors, to develop in the gastrointestinal tract. Males are more likely than females to be diagnosed with duodenal leiomyomas in the 6th and 7th decades of life. The origin and pathology of these tumors in the gastrointestinal tract remain a mystery. Leiomyomas of the duodenum seldom cause symptoms. When a duodenal tumor is larger, it might cause more severe symptoms, such as bleeding, nausea, vomiting, and weakness. In our patient described above, we believe the leiomyoma caused bleeding, as no other definitive source of bleeding was found even after a complete evaluation. The case describes a rare duodenal tumor with an unusual presentation.



[3521] **Figure 1.** a. EGD showing a large pedunculated polyp extending from the first portion to the second portion of the duodenum. b. H&E stain showing submucosal benign spindle cell proliferation. No atypia, pleomorphism or mitotic activity.

S3522

A Rare Case of *Clostridium difficile* Enteritis in a Patient With Ileorectal Anastomosis and Loop Ileostomy

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Introduction: A subtotal colectomy with loop ileostomy is often performed in patients with colonic pathologies including IBD and colon cancer. Though Clostridium difficile colitis is prevalent and several cases of C. difficile enteritis have been reported, C. difficile enteritis after a subtotal colectomy with ileorectal anastomosis and loop ileostomy has never been reported and will be presented in the following case.
Case Description/Methods: A 72-year-old female with past medical history of subtotal colectomy, ileorectal anastomosis, and loop protecting ileostomy status post colonic perforation, dyslipidemia, end stage renal disease on hemodialysis, and diabetes mellitus presented to the hospital for hypotension during dialysis, dizziness, and lightheadedness causing her to fall. The patient admitted to multiple episodes of dizziness and non-traumatic falls in the previous month. She repeatedly became hypotensive during her dialysis sessions, during which she frequently presented at or below her dry weight. Patient denied any headaches, fever, chills, nausea, abdominal pain, chest pain, palpitations, shortness of breath, and blurry vision. Physical examination showed a right lower quadrant ileostomy bag. On admission, the patient was hypotensive, which normalized after fluid resuscitation. After extensive work-up, cardiac and renal causes of hypotension were ruled out. Throughout her hospital course, despite fluid resuscitation, the patient

recurrently became hypotensive and was given midodrine. The patient was found to have loose, high-output (approximately 2600 mL/day) from her ileostomy bag. C. difficile PCR returned positive. After an oral vancomycin regimen was begun, the patient's blood pressure improved markedly. The patient's symptoms of dizziness and weakness resolved.
Discussion: This case is important to emphasize the consideration of C. difficile infection in loop ileostomy patients presenting with hypotension. Though the patient had noted chronic high-output from her ileostomy bag, the patient did not have a recent prior course of antibiotics and was on a diet inappropriate for an ileostomy. Therefore, given the lack of risk factors, the limited cases of C. difficile enteritis, and no reported cases of C. difficile infection in ileorectal anastomosis and loop ileostomy, diagnosing the C. difficile infection causing hypotension in this patient was challenging. Further reporting of such cases will aid in guidelines, diagnosis, and treatment of C. *difficile* enteritis.

S3523

A Rare Case of Appendiceal Goblet Cell Adenocarcinoma With Peritoneal Carcinomatosis

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Introduction: Primary appendiceal cancers are an extremely rare entity, representing approximately 1% of gastrointestinal malignancies. Of those, appendiceal goblet cell carcinoma (GCC) is one of the most rare subtypes with a reported incidence of 0.05 cases per 100,000 population per year in the United States. These tumors consist of both glandular and neuroendocrine elements containing goblet cells. We present a unique case of appendiceal GCC with peritoneal carcinomatosis presenting as a small bowel obstruction.
Case Description/Methods: A 43-year-old male presented for 2 months of diffuse, intermittent abdominal pain which acutely worsened into a constant and colicky abdominal pain. On examination, he was afebrile with elevated blood pressure of 196/89 mmHg and had a distended, diffusely tender abdomen with sluggish bowel sounds. CT abdomen revealed small bowel obstruction and focal density in the right lower quadrant abutting the small bowel loops with associated edema and omental infiltration. Colonoscopy was performed which revealed an extremely tortuous colon and was complicated by a serosal tear and internal hemorrhage which required surgical intervention. Intraoperatively, omental caking and peritoneal carcinomatosis of the mesentery, peritoneal surfaces, distal jejunum, sigmoid colon and terminal ileum were noted. Biopsy and immunohistochemistry of the omentum and mesenteric nodules were consistent with a diagnosis of metastatic goblet cell adenocarcinoma with lymphovascular invasion. Once stabilized, he was started on FOLFOX chemotherapy regimen with outpatient heme/onc follow up.
Discussion: Appendiceal GCC is one of the rarest GI malignancies documented. It is often asymptomatic and diagnosed incidentally; however it can also present as chronic abdominal pain, acute appendicitis, and even bowel obstruction. Treatment consists of resection of the colon and chemotherapy in localized disease, and in cases of peritoneal carcinomatosis, cytoreductive therapy and heated intraperitoneal chemotherapy (HIPEC) are utilized. Not only is it rare, but it's also more aggressive than other appendiceal cancers. One study showed that the average survival rate of patients with peritoneal carcinomatosis treated with cytoreductive surgery and HIPEC was 18.5 months. In cases of acute appendicitis, it is crucial to biopsy the surgical specimen so this diagnosis is not missed. Our case highlights an interesting and complicated presentation of this rare disease.

S3524

A Rare Case of Autoimmune Enteropathy Misdiagnosed as Celiac Disease

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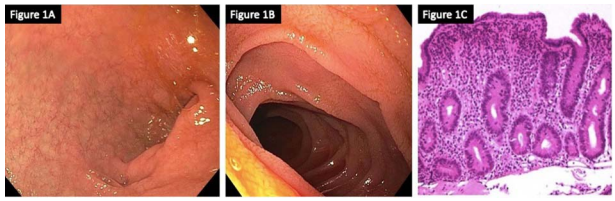
Introduction: Autoimmune enteropathy (AIE) is an increasingly rare condition characterized by immune mediated intestinal mucosal injury that manifests as intractable diarrhea. Originally thought to be a pediatric condition, increasing cases have been reported in the adult population. While AIE can affect all parts of the GI tract, the duodenum is the most common site of GI tract involvement. The management of AIE is centered around optimizing nutritional status in addition to steroids, immunosuppression and biologic agents.
Case Description/Methods: A 29-year-old man with a childhood diagnosis of celiac disease presented for weakness and electrolyte abnormalities in the setting of chronic watery diarrhea. Laboratory findings were significant for WBC 6k uL, Potassium 2.5 mmol/L, Chloride 117 mmol/L, Creatinine 2.43 mg/dL, and decreased levels of Vitamins A, B, E, Zinc. Further investigation showed negative Tissue Transglutaminase IgA and negative workup for C. diff, Giardia, Cryptosporidium, and Campylobacter. Endoscopic evaluation showed atrophic duodenitis with erythema but no endoscopic evidence of celiac disease (Figure A,B). Histological examination of duodenal biopsies showed loss of goblet cells and Paneth cells in addition to villous blunting, intraepithelial lymphocytosis and increased apoptotic bodies consistent with AIE (Figure C).
Discussion: Among the causes of chronic diarrhea and malabsorption, AIE is a rare disorder that is often misdiagnosed and mistaken for celiac disease or IBD. The pathophysiology of AIE is related to abnormal expression of self-antigens on mucosal epithelial cells, activating CD4 T lymphocytes, which leads to the destruction of enterocytes through cytotoxic effects and apoptosis. Immunohistological studies have shown that autoantibodies can be directed against enterocytes and goblet cells but are not pathognomonic for AIE. Endoscopic findings include mucosal hyperemia, ulcerations and mucosal atrophy. While difficult, the management of AIE is centered around managing malnutrition and treating the underlying autoimmune disorder. Initial recommended treatment includes corticosteroids, however, in refractory cases immunosuppressive therapy such as azathioprine, 6-mercaptopurine, and monoclonal antibodies have been shown to be beneficial. Highlighted by the case we present, AIE can be undiagnosed for years and establishing an early diagnosis is critical in reducing morbidity and improving long-term outcomes.



[3524] **Figure 1.** 1A: Duodenal bulb inflammation 1B: Erythema of 2nd portion of duodenum 1C: High power view of duodenal biopsy shows absent villi, expansion of the lamina propria with a lymphoplasmacytic infiltrate, and absence of goblet cells. Scattered intraepithelial lymphocytes and crypt apoptotic figures can be seen.

S3525

A Rare Case of *Campylobacter* and *Strongyloides stercoralis* Co-infection in an Immunocompetent Host

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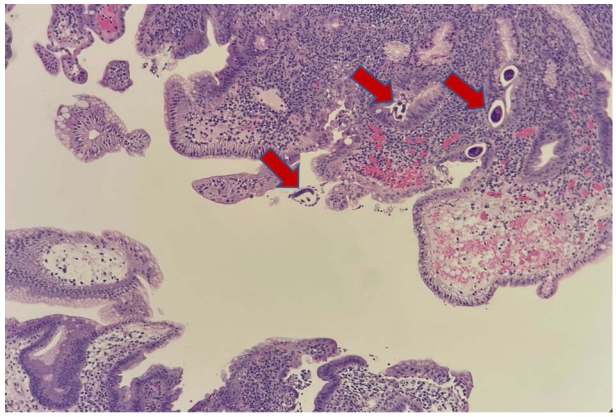
Introduction: *Strongyloides stercoralis* is a parasitic nematode mainly seen in immigrants and military personnel from endemic areas. Most cases in immunocompetent hosts are incidental findings or present with mild gastrointestinal (GI), respiratory or dermal symptoms. Co-infection with organisms like *Campylobacter* coupled with anatomical abnormalities such as a hiatal hernia can produce similar symptoms delaying diagnosis and treatment.
Case Description/Methods: A 75 year-old-woman presented with persistent watery diarrhea, nausea, dyspepsia, and non-bloody and non-bilious vomiting over the past five days. She also had an unintentional weight loss of 50 pounds over the past three months. She denied any recent travel, rashes, or fevers/chills. On presentation, she was hemodynamically stable with generalized abdominal tenderness on physical exam. CT of the abdomen revealed a moderately-sized hiatal hernia. Serologies were significant for normocytic anemia, thrombocytosis, and eosinophilia (white blood cell count 5300, eosinophils 6.6%). Stool studies were positive for *Campylobacter* and she was started on azithromycin. She initially felt better and was subsequently discharged on azithromycin. She then presented three days later with complaints of nausea, and bloody emesis. Upper endoscopy with biopsy was done which showed a Los Angeles grade D reflux esophagitis and duodenitis. Histological evaluation revealed *S. stercoralis* in the duodenal mucosa (Figure 1). Initial ova and parasite cultures from a week ago further confirmed the diagnosis. She was then started on an extended course of ivermectin with a resolution of her symptoms and was discharged home.
Discussion: *Campylobacter* is an enteric gram-negative bacterium that also affects the GI tract and can mimic *S. stercoralis* infection clinically. Co-infection with this organism occurs due to its similar mode of transmission, however, there have only been a few cases documented in literature. Diagnosis is made by stool testing and serology, however, stool tests are only positive in 50-60% of cases. Eosinophilia may or

may not be present. Clinicians should be aware that despite having underlying structural abnormalities such as a hiatal hernia, an EGD with biopsy is warranted to rule out infectious etiologies especially in the presence of eosinophilia as seen above. Early diagnosis and treatment are essential in preventing disseminated disease, especially in immunocompromised patients.



[3525] **Figure 1.** Hematoxylin and Eosin stain of the duodenum revealing acute duodenitis with associated Strongyloides stercoralis in the intestinal crypts and lumen.

S3526

A Red Herring? Duodenal Vasculitis in the Setting of Post-COVID-19 Infection

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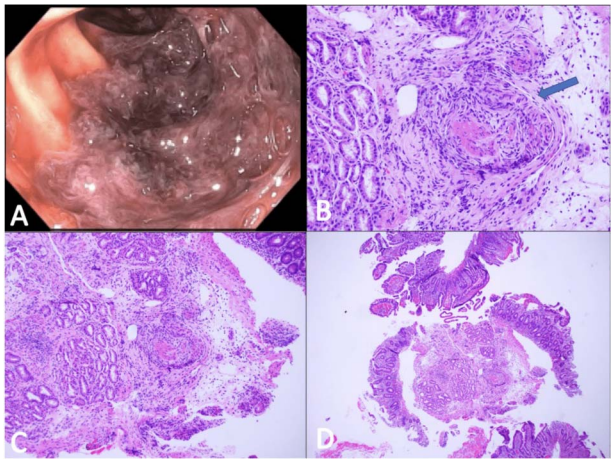
Introduction: While the respiratory symptoms of coronavirus disease 2019 [COVID-19]) are well-defined, the gastrointestinal implications continue to be explored with increased experience. Herein we describe a unique case illustrating duodenal and cutaneous leukocytoclastic vasculitis (LCV) as a manifestation of COVID-19.
Case Description/Methods: A 55-year-old female with a medical history significant for triple-negative left breast cancer status post mastectomy presented with a two-day history of intermittent epigastric pain associated with nausea and acute anemia. One week prior, the patient was admitted for a GI bleed, initially suspected to be NSAID - induced due to COVID-19 infection. Symptoms resolved without endoscopic intervention and outpatient follow up was arranged. Exam was significant for epigastric tenderness and bilateral lower extremities with a non-tender, non-pruritic maculopapular rash. Labs revealed leukopenia, negative ANA and C-ANCA, low C4 (< 2), and elevated CRP (25). CT imaging demonstrated small bowel thickening, suggestive of enteritis. Endoscopy revealed mucosal changes involving the duodenum and jejunum with fibrinoid-like necrosis and mild inflammatory cell infiltrates, consistent with vasculitis-like changes (Figure A). Dermatological biopsy revealed negative immunofluorescence studies (IgA, IgG, IgM, and C3) in the basement membrane and perivascular regions consistent with LCV, excluding IgA vasculitis (Figure B-D). Cryoglobulin level was not diagnostic for cryoglobulinemia. Angiography studies of her chest, abdomen, and pelvis excluded large- or medium-vessel vasculitis. We suspected symptoms may be due to COVID-19-induced cutaneous and duodenal LCV. Treatment comprised of an oral steroid taper and PPI therapy with a plan for repeat outpatient endoscopy.
Discussion: This presentation of duodenal vasculitis is a suspected consequence of her recent COVID-19 infection. Literature demonstrates postmortem analysis of direct viral mononuclear cell infiltration in the vascular intima and lumen of vessels causing complications such as coagulopathy and vasculitis. COVID-19 may be a possible trigger for an immune complex hypersensitivity reaction. Literature also supports COVID-19 vasculitis with some cases associated with vaccination. The gastrointestinal effects of COVID-19 continue to be explored. COVID-19 vasculitis should be considered on the differential in presentation of abdominal pain, melena and dermatological findings in the setting of recent COVID-19 diagnosis.



[3526] **Figure 1.** A. Endoscopic images of the second part of duodenum suggestive of vasculitis. B. Active duodenitis with hemorrhage and focal changes in blood vessels (blue arrow). There are blood vessels in the submucosa showing fibrinoid like necrosis and mild inflammatory cells infiltrates. C. There is active and chronic duodenitis with hemorrhage. D. Further inflammatory cells and fibrinoid like necrosis around blood vessels.

S3527

A Rare Cause of Enteritis in an Adult Male

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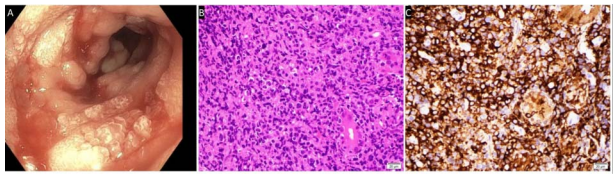
Introduction: IgA vasculitis is more commonly seen in the pediatric population than in adults. Rarely IgA vasculitis is associated with malignancy, most commonly solid tumor malignancies, although there are case reports of association with hematologic malignancies. We report a case of large B-cell lymphoma mimicking IgA vasculitis in a 33-year-old immunosuppressed male with a prior history of IgA vasculitis.

Case Description/Methods: A 33-year-old Caucasian male post renal transplant from reflux nephropathy on chronic immunosuppression was hospitalized for postprandial epigastric abdominal pain, nausea, vomiting and diarrhea. Two years prior, he was admitted for the same symptoms, palpable purpura of the lower extremities and elevated serum IgA. Enteroscopy had shown duodenal and jejunal ulceration with biopsies staining positive for IgA, confirming IgA vasculitis. He had complete resolution with a steroid taper. His current presentation had resulted in multiple hospital admissions, but empiric trial of steroids failed to alleviate symptoms. Vitals were normal and exam was notable for epigastric tenderness. Labs were notable for WBC 19.00 x103/cmm with normal differential, hemoglobin 9.2 gm/dL (prior 11.0 gm/dL), CRP 20.7 mg/L, serum creatinine 2.7 mg/dL (prior 1.5 mg/dL), and urinalysis with proteinuria, sterile pyuria, and hematuria. CTA abdomen/pelvis revealed thickening of the duodenum with shotty mesenteric lymph nodes without ischemia. Enteroscopy revealed an erythematous duodenum and jejunum (figure A). Jejunal biopsy (figure B) revealed CD20 positive cells consistent with DLCBL (figure C). He was seen by oncology and treated with R-CHOP but later unfortunately expired due to COVID-19 complications.
Discussion: Non small cell lung cancer and renal cell carcinoma are most commonly associated with IgA vasculitis. It may also be seen in both Hodgkin and Non-Hodgkin lymphomas in adult patients. If IgA vasculitis occurs after a malignancy is diagnosed, it may indicate that metastasis has occurred. Malignancy associated IgA vasculitis is more likely to have an incomplete response to steroids and requires treatment of the underlying malignancy to achieve remission. Our case illustrates posterior probability error and premature closure cognitive biases. We should consider alternative diagnoses rather than anchor on prior diagnoses even when presentations are similar. Our case also highlights the importance of considering occult malignancy in adults with diagnosis of IgA vasculitis.



[3527] **Figure 1.** A) Enteroscopic view of erythematous jejunal mucosa Figure B) Histopathologic imaging (hematoxylin and eosin coloring, 40x) of jejunum demonstrating diffuse atypical lymphoid infiltrate Figure C) Immunohistochemical imaging staining of jejunum (CD20, 40x) confirming diffuse large cell B lymphoma.

S3528

A Rare Presentation of Small Bowel Metastasis From Renal Cell Carcinoma: A Case Report and Review

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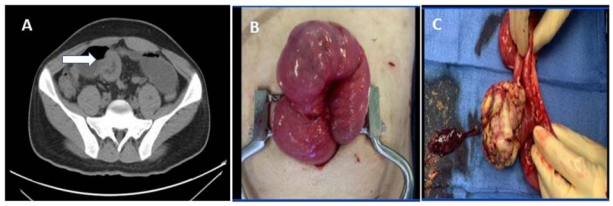
Introduction: At the time of diagnosis, around 25–30% of patients with renal cell carcinoma (RCC) had metastatic disease. The most common locations for metastasis of renal cancer in order of frequency include lung, bone, liver, lymph nodes and brain. RCC metastases to the small intestine are extremely rare, with limited reports in the literature (Table).
Case Description/Methods: A 20-year-old male presented with a 3-week history of dull epigastric pain, nausea, and vomiting. Past medical history was significant for iron deficiency anemia, and metastatic RCC was diagnosed in 2021 status post right open radical nephrectomy. The biopsy showed Xp11.2 translocation associated renal cell carcinoma WHO grade 4. The patient had lost follow-up previously. His laboratory workup this admission showed persistent iron deficiency anemia with mild leukocytosis and thrombocytosis and elevated liver enzymes. CT chest/abdomen/pelvis showed concern for intussusception in the distal jejunum causing an obstruction. Surgery team was consulted who took patient for diagnostic laparoscopy. Intraoperatively intussusception was partially reduced laparoscopically but could not be completely reduced since there was a big mass acting as a lead point. The decision was then made to convert to a mini-laparotomy. Small bowel resection with primary anastomosis was performed. Pathology from the specimen confirmed metastatic RCC. Oncology was consulted who plan to start pembrolizumab plus axitinib for systemic therapy. (Figure)
Discussion: RCC intraluminal metastases in the small intestine are unusual reported as 2-4% incidence. Bowel metastases from RCC can manifest in several different forms including intussusception, bowel perforation, gastrointestinal bleeding and symptoms of intestinal obstruction due to the presence of a mass within the intestinal wall. This case presented a young male with metastatic RCC to small bowel which has not been reported so far. Clinicians should be aware that, in patients presenting with anemia, clinical symptoms of bowel obstruction and a history of RCC, intestinal tumor involvement should be considered. Metastasectomy may extend patient survival and surgical resection of the involved intestinal segment has been recommended as the treatment of choice.



[3528] **Figure 1.** A: Axial-enhanced CT scan of the abdomen showing Intussusception in the distal jejunum causing the obstruction. B: Intraoperative pictures of intussusception. C: A resected segment of the small intestine with a mass found at the lead point.

Table 1. Published reports of intussusception from renal cell carcinoma

No.	Author, Year	No. of pts	Stage of RCC (TNM)	Gender	Age (years)	Histological subtype	Outcome
1	Vani et al., 2017	2	NA	M, F	65, 68	NA	Pt. 1: Death 3 months after surgery. Pt. 2: Death 6 months after surgery.
2	Mishra et al., 2015	1	NA	M	57	Clear cell	NA
3	Wan Kyu Eo et al., 2008	1	pT1aN0M1	M	47	Clear cell	NA
4	Venugopal et al.,2017	1	NA	NA	NA	NA	NA
5	Ekbote et al., 2015	1	NA	M	52	Clear cell	Alive after ten months
6	Bellio et al., 2016	1	pT1bN0	M	75	Clear cell	NA
7	Deguchi et al., 2000	1	NA	M	58	NA	NA
8	Ogiso et al., 2005	1	pT1aN0M1	F	57	NA	NA
9	Hegde R J et al., 2014	1	NA	M	52	Clear cell	NA
10	Budmiger et al., 2015	1	NA	M	61	NA	NA
11	Kerkeni B et al., 2013	1	pT2bNxM1	F	32	Clear cell	Not interpretable
12	Khan AB et al., 1991	1	NA	NA	NA	NA	NA
13	Trojaneillo et al., 2017	1	NA	M	75	NA	Pt. died 9 months after surgery.
14	Tutar et al., 2008	1	NA	M	59	Clear cell	NA
15	Aissa et al., 2012	1	NA	M	64	Tubulo- Papillary	NA

Table 1. (*continued*)

No.	Author, Year	No. of pts	Stage of RCC (TNM)	Gender	Age (years)	Histological subtype	Outcome
16	Longo et al., 2013	1	NA	F	52	Clear cell	NA
17	Budmiger et al., 2015	1	NA	M	61	Clear cell	NA
18	Sasaki et al., 2006	1	NA	M	64	Clear cell	Pt died after few months after jejunal resection
19	Roviello et al., 2006	1	NA	M	48	Clear cell	Alive at follow up.
20	Rampersad et al., 2006	1	NA	F	69	NA	NA
21	Collet et al., 2001	1	NA	F	77	NA	NA

Abbreviations: NA, not applicable; F, Female; M, Male.

S3529

A Small Bowel Stricture Revealing Intestinal Lymphoma: A Rare Presentation

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Introduction: Lymphoma originating from the gastrointestinal tract is rare. Presenting symptoms are vague and even with the use of advanced imaging and endoscopic techniques, diagnosis is challenging.
Case Description/Methods: A 43-year-old male with a history of gastroesophageal reflux disease, sarcoidosis (in remission), and hypertension presented with three months of worsening periumbilical pain, a 30-pound weight loss, and repeated emesis. Prior outpatient workup including inflammatory bowel disease (IBD) serology, esophagogastroduodenoscopy, and colonoscopy were unrevealing. Video capsule endoscopy (VCE) demonstrated localized inflammation in the ileum. The capsule was unable to pass beyond this point. On presentation, physical exam was notable for tenderness in the periumbilical area. Routine labs and chest radiograph were normal. CT abdomen/pelvis revealed a partial small bowel obstruction (images 1 and 3). Small bowel enteroscopy (SBE) demonstrated congested mucosa in the proximal ileum, jejunal inflammation, erythematous duodenopathy, and gastritis. Biopsies showed nonspecific chronic inflammation of the ileum and jejunum. CT enterography (CTE) disclosed the presence of a stricture in the mid-ileum (images 2 and 4). The capsule was still seen lodged at the stricture (image 5) but later passed without intervention. ACE levels, calprotectin, and CEA were normal. Stool ova, parasites, and culture for enteric bacteria were negative. Small bowel resection with side-to-side anastomosis was performed. Pathology revealed diffuse large B-cell lymphoma of the small bowel and the patient was started on chemotherapy.
Discussion: A small bowel stricture is rarely attributed to lymphoma and often raises suspicion of an inflammatory process. The stricture in this case was initially thought to be inflammatory given the lack of a bulky mass or lymphadenopathy and evidence of chronic inflammation on biopsies. However, the inflammatory workup for IBD was negative and sarcoidosis was stable, thus further evaluation was required. VCE, SBE with biopsies, and CTE have improved the evaluation of small intestinal pathologies but were nondiagnostic in this case. Due to the patient's worsening clinical condition and lack of etiology of pathology, more invasive exploration and surgical intervention were warranted and provided the definitive diagnosis. While imaging and endoscopy are crucial in the workup of primary intestinal lymphoma, lack of findings does not rule it out and persistent evaluation is necessary.

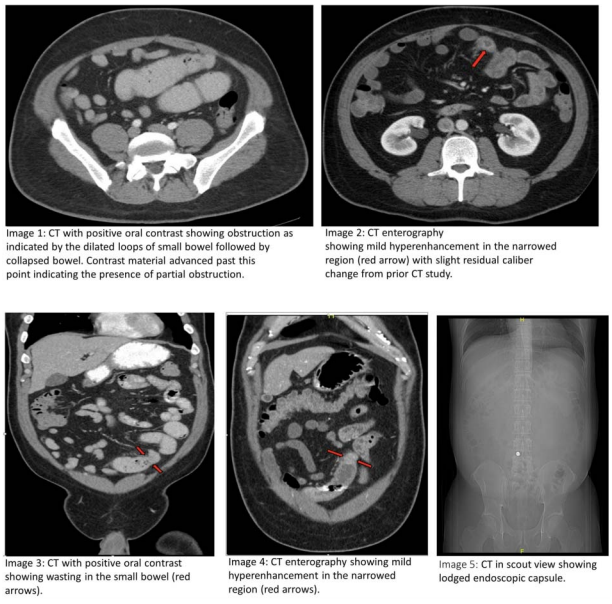

Image 1: CT with positive oral contrast showing obstruction as indicated by the dilated loops of small bowel followed by collapsed bowel. Contrast material advanced past this point indicating the presence of partial obstruction.

Image 2: CT enterography showing mild hyperenhancement in the narrowed region (red arrow) with slight residual caliber change from prior CT study.

Image 3: CT with positive oral contrast showing wasting in the small bowel (red arrows).

Image 4: CT enterography showing mild hyperenhancement in the narrowed region (red arrows).

Image 5: CT in scout view showing lodged endoscopic capsule.

[3529] **Figure 1.** CT abdomen and pelvis with partial small bowel obstruction and CT enterography showing a stricture in the mid-ileum.

S3530

A Rare Presentation of Adult Onset IgA Vasculitis With Gastrointestinal Manifestations Without Typical Skin Manifestations

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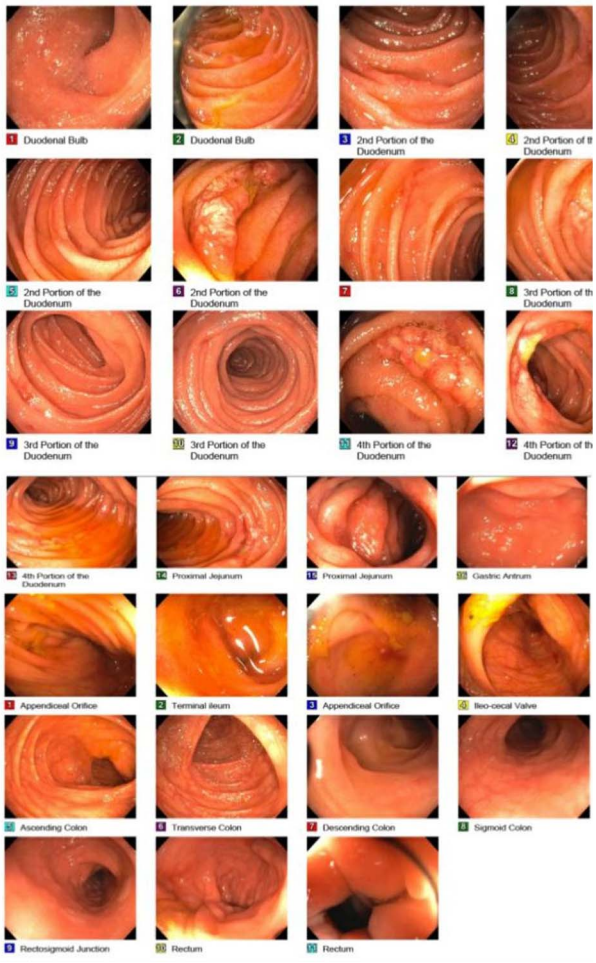
Introduction: IgA Vasculitis is a small vessel vasculitis that is commonly seen in children but rarely affects adults. IgA Vasculitis can present as arthralgia, rash, kidney involvement, and gastrointestinal symptoms. We present a case of a 56 years old Male that presented with abdominal pain without the characteristic purpuric rash or any other features. Imaging findings showed enteritis, colonoscopy showed ulcers in the terminal ileum, rectum, sigmoid and descending colon. The Kidney biopsy showed IgA deposition. Thus, adult-onset IgA vasculitis can present with gastrointestinal symptoms without the typical skin manifestations and imaging and endoscopy can guide in diagnosis and management of the same.
Case Description/Methods: We present a case of a 56 years old Male who presented to the ED with abdominal pain and arthralgia without the characteristic skin purpura. Abdominal imaging showed severe diffuse wall thickening of multiple loops of small bowel with adjacent fat stranding. Small bowel enteroscopy was performed that showed many non-bleeding cratered ulcers in the duodenum and jejunum, and

colonoscopy showed ulcers in the terminal ileum, rectum, sigmoid and descending colon. Biopsy from Duodenal and Jejunal ulcers showed focal acute and chronic inflammation and ulceration. A kidney biopsy showed IgA deposition. The patient's abdominal pain rapidly resolved after steroid administration and supportive care. (Figure)

Discussion: IgA Vasculitis is a small vessel vasculitis that affects various organs, including the skin, gastrointestinal (GI) tract, joints, and kidneys and commonly occurs in children. The incidence of IgA Vasculitis in adults is as low as 0.8-1.8/100 000 for adults. Approximately 53% of patients with adult IgA vasculitis have Gastrointestinal involvement. Gastrointestinal manifestations range from mild (nausea, vomiting, abdominal pain, paralytic ileus) to severe findings (gastrointestinal bleeding, intussusception, bowel ischemia with secondary necrosis, bowel perforation). Abdominal pain is the most common gastrointestinal symptom which is attributed to mesenteric vasculitis leading to bowel ischemia and edema of the gastrointestinal tract. The small bowel, and in particular, the duodenum is the most commonly affected site. Vasculitis is not commonly observed in GI biopsies. While the clinical and histologic findings may mimic early inflammatory bowel disease, the presence of predominant small bowel involvement, especially erosive duodenitis, should raise suspicion for IgA vasculitis.



[3530] **Figure 1.** Endoscopic and Colonoscopic Findings in IgA Vasculitis.

S3531

A Rare Cause of Obstructive Jaundice: Afferent Limb Syndrome

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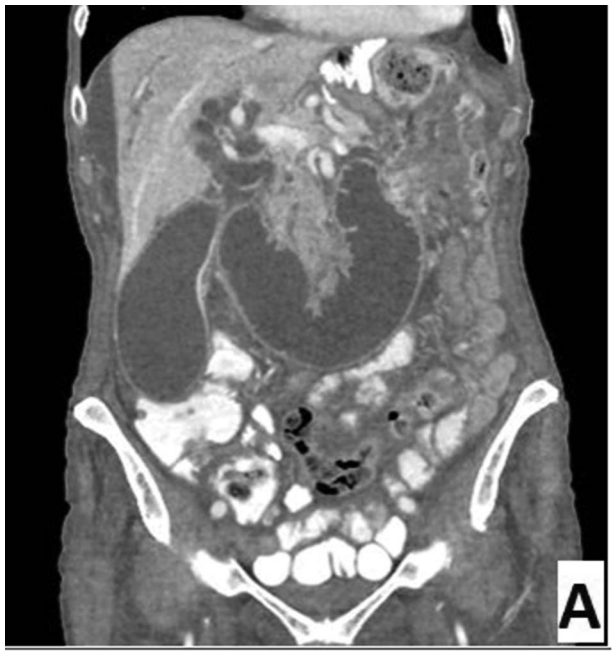
Introduction: Afferent limb syndrome (ALS) is a rare but potentially life-threatening complication of upper gastrointestinal (GI) surgery and reconstruction causing distal obstruction of the biliopancreatic limb with subsequent accumulation of bile, pancreatic fluid, and other small bowel secretions upstream afferent limb dilation. It is imperative to recognize this complication postoperatively as buildup in the small bowel can result in bowel perforation requiring emergency surgery. We present a patient who developed ALS after upper gastrointestinal reconstruction with esophagojejunostomy for the treatment of gastric adenocarcinoma.

Case Description/Methods: An 84 year-old female with a past medical history of gastric adenocarcinoma with total gastrectomy and esophagojejunostomy presented to the emergency department with a subacute history of upper abdominal pain and new-onset jaundice. Initial labs were notable for elevated liver chemistries including alkaline phosphate level of 435 U/L (normal 39-117 U/L), alanine transaminase of 62 U/L (normal 6-45 U/L), and aspartate transaminase of 36 U/L (normal 10-25 U/L). Abdominal ultrasound demonstrated markedly dilated common bile and intrahepatic ducts and gallbladder distention without stones. CT scan of the abdomen confirmed the ultrasound findings, but also identified a dilated duodenum with an abrupt transition point in the proximal jejunum suggestive of ALS shown in Figure A. The patient underwent open laparotomy which demonstrated multiple tumor implants. A large implant at the site of the previous jejunojejunostomy was identified as the cause of afferent limb obstruction. A palliative bypass was performed. The patient had a complicated post-operative course. Ultimately, due to the extensive disease burden and ongoing patient discomfort, the patient and their family decided to pursue hospice care.

Discussion: ALS is a post-operative complication of upper GI tract surgery that involves mechanical obstruction of the biliopancreatic limb. Patients often present with jaundice. CT scan is the preferred diagnostic modality. First-line treatment involves surgery to relieve the obstruction with revision of the prior anastomosis. Although rare, this diagnosis should be considered in appropriate patients.



[3531] **Figure 1.** A: CT scan with markedly dilated intrahepatic and extrahepatic biliary ductal dilation, distended gallbladder, and markedly dilated duodenum with abrupt transition point in proximal jejunum.

S3532

A Rare Presentation of a Neuroendocrine Tumor

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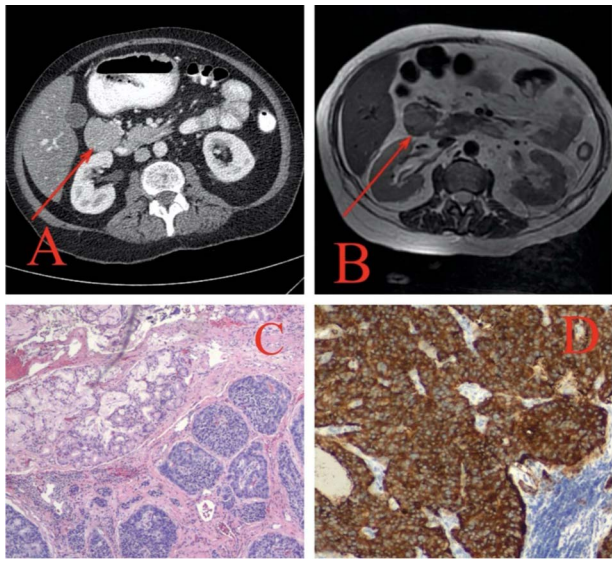
Introduction: Neuroendocrine tumors (NETs) are rare types of tumors, only contributing to 0.5% of all malignancies. They are defined as epithelial cells with predominantly neuroendocrine differentiation and consist of a spectrum of tumors emerging from stem cells throughout the body and can occur anywhere in the body. While they are rare, the incidence over the past few decades has increased. The primary locations for NETs is the gastrointestinal tract (62-67%) and pulmonary tract (22-27%) but duodenal NETs only contribute to 2-3% of all gastrointestinal tract NETs. Here we present a case of a 64 year old female who was incidentally found to have a duodenal neuroendocrine tumor.

Case Description/Methods: A 64-year old female presented to the emergency department secondary to syncope and collapse. During her trauma evaluation, an incidental lobulated soft tissue mass inferior to the distal stomach was seen on complete computed tomography (CT) scans. The surgery team was consulted for resection of the mass and an octreotide scan was performed prior to resection to further evaluate the mass and to check for any signs of metastatic disease. The octreotide scan demonstrated intense radiotracer accumulation within the duodenal mass consistent with a neuroendocrine tumor and no areas of abnormal radiotracer accumulation suspicious for metastatic disease. Pathology of the resected mass was positive for a well differentiated neuroendocrine tumor with an organoid pattern and homogenous oval-round neoplastic cells with salt-pepper nuclear and pseudoglandular arrangement that was well circumscribed and partially encapsulated with negative margins. Immunohistochemistry was positive for AE ⅓, CD56, Synaptophysin, and chromogranin and negative for CD117, DOG-1, CD34 and CD45. (Figure)

Discussion: The prevalence of NETs has increased over the years due to the improvement in diagnostic tools, such as upper gastrointestinal endoscopy. The duodenum is a rare location for such tumors, accounting only for 4% of all GI tumors overall. Neuroendocrine tumors are also typically found in those under 50 years old. However, our patient was found to have both a duodenal mass and was over the age of 50 at the time of presentation and diagnosis. To date, a consensus on a conclusive treatment of D-NETs has not been reached. This case brings to light the importance of further research in diagnosing and treating neuroendocrine tumors and also raises awareness for clinicians to have this in their differential.



[3532] **Figure 1.** A) CT abdomen and pelvis with contrast displaying a lobulated soft tissue mass inferior to the pylorus measuring approximately 3.0 x 3.3 x 3.5 cm with possible connection with the adjacent bowel (red arrow). B) MRI of the abdomen showing a paraduodenal mass (red arrow). C) Histological slide demonstrating the surface duodenal mucosa and underlying organoid pattern of neuroendocrine tumor. D) An Immunohistochemistry slide demonstrating the positivity of synaptophysin of the neoplastic cells.

S3533

A Unique Case of Progressive Disseminated Histoplasmosis With Significant Gastrointestinal Involvement

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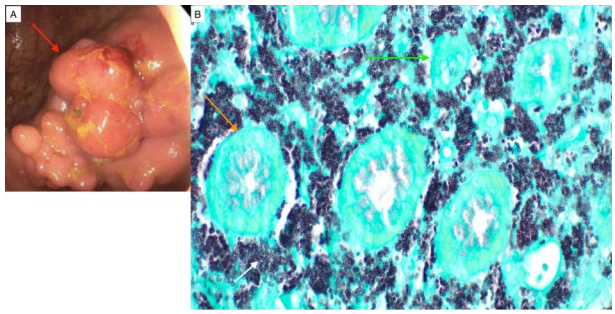
Introduction: Histoplasmosis is a predominantly pulmonary fungal infection, caused by inhalation of Histoplasma Capsulatum. Immunocompetent individuals typically have self-limiting disease, while immunosuppressed individuals are at risk for severe disease complications, including extrapulmonary manifestations. We present a rare case of a transgender woman with disseminated histoplasmosis complicated by severe gastrointestinal manifestations including small bowel obstruction, splenic infarction, hepatopathy, ileitis and large colonic masses.
Case Description/Methods: A 43-year-old transgender woman with AIDS was first identified to have disseminated histoplasmosis in a bone marrow biopsy done in 2018 for anemia. Since then, her long history of medication noncompliance had resulted in diffuse gastrointestinal manifestations. In 2019, she developed a small bowel obstruction due to excessive fungal infiltration of Peyer's patches. She underwent an exploratory laparotomy and partial small bowel resection with primary anastomosis and splenectomy due to an incidentally noted splenic infarct on abdominal CT. Spleen pathology revealed extensive necrosis, as well as positive GMS and PAS stains for fungal budding yeasts within histiocytes. Amphotericin infusions with eventual transition to maintenance oral itraconazole dosing led to initial suppression of her infection, until the latter was no longer covered by her insurance. In early 2022, after being off of antifungal treatment for about two months, a repeat colonoscopy revealed multiple large, polypoid, non-obstructing cecal masses (Figure). Pathology reports revealed histiocytic aggregates (GMS+, PAS+), and she was restarted on 4-6 weeks of IV amphotericin.
Discussion: This unique case emphasizes the varied GI manifestations of disseminated histoplasmosis and emphasizes the importance of access to care. It is important to keep this atypical presentation in mind as clinical improvement is often achieved with prompt antifungal treatment. However, recurrence is seen in 50% of patients, despite adherence to therapy. As a member of the LGBTQ community, our patient has well known personal and institutional barriers to healthcare. It is unclear if her recurrence was from failed therapy or due to barriers in consistent care. If left untreated, disseminated histoplasmosis has a mortality rate of >90% within one year. Thus, it is imperative to identify patients with potential barriers to health care to reduce morbidity and mortality.



[3533] **Figure 1.** A: Polypoid histoplasmosis mass found in cecum on colonoscopy (Red Arrow) Figure B: Histology slide of the terminal ileum biopsy using GMS stain. Intestinal crypts of the ileum (orange arrow) are surrounded by densely packed Histoplasma capsulatum (white arrow). Some crypts are formed while others appeared destroyed by this fungus (green arrow).

S3534

A Unique Presentation of Gastrinoma Complicated by Duodenal Perforation

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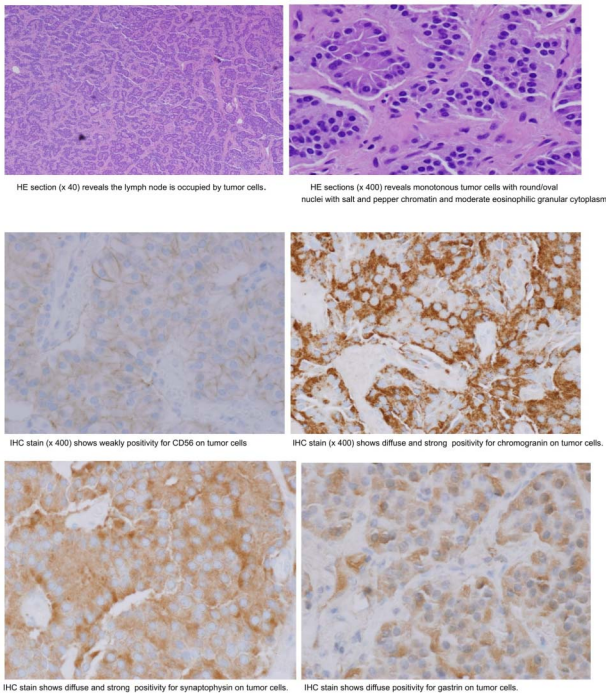
Introduction: Neuroendocrine tumors are uncommon neoplasms with multiple different characteristics. They can be benign but also proliferate into malignant tumors with different pathological scenarios such as intestinal obstruction/ intraluminal mass. Gastrinomas are type of NET originating from pancreatic islet cells but can also arise from gastrin-producing cells in the duodenum. We present a case of gastrinoma, in which clinical period was complicated by small bowel perforation.
Case Description/Methods: A 55-year-old lady with no significant past medical presented with complaints of abdominal pain, diarrhea and nausea/vomiting that was uncontrolled with symptomatic management. The patient underwent an EGD that showed esophagitis, gastritis, and duodenitis, and she was transferred to our facility for further work-up. She underwent a workup for Zollinger Ellison

Syndrome in view of persistent symptoms. Her gastrin level came back elevated at 4,180 pg/ml after her medications were stopped to get the correct lab readings. A nuclear scan was scheduled for the patient to localize the gastrinoma lesion, but suddenly the patient became altered, hypotensive, and developed an acute abdomen. On further investigation with CT scan of the abdomen, patient was found to have pneumoperitoneum, secondary to bowel perforation. She underwent an emergent exploratory laparotomy, was found to have large perforations of the 3rd, 4th portion of the duodenum and several small perforations throughout the small bowel. A Para duodenal lymph node was taken and sent for pathology. Pathology revealed a well-differentiated neuroendocrine tumor: histological subtype gastrinoma. The patient underwent duodenal resection, gastrojejunostomy, cholecystectomy and feeding tube placement. Post-op period was complicated by patient having persistent dysphagia, initially to solids and progressed to have dysphagia to liquids as well. The patient underwent an EGD again which showed presence of esophageal stricture and had to undergo dilatation 6 times in a span of 2 months for symptom relief. She follows up with oncology and gastroenterology regularly and is scheduled to get a PET scan soon to rule out metastasis. (Figure)

Discussion: Gastrinomas manifest with peptic ulcer disease symptoms but some patients present with diarrhea. Serum gastrin levels of $>$ 1000 pg/ml with symptoms is diagnostic of gastrinoma. A secretin provocative test is used for diagnosis if gastrin levels are $<$ 1000 pg/ml. As clinicians, we must be aware of the complications of gastrinoma.



[3534] **Figure 1.** Histopathological slides.

S3535

A Unique Case of HER2 Positive Adenocarcinoma of the Small Bowel

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Introduction: 59 yo male with history of Crohn's disease underwent partial small bowel resection due to obstruction with lysis of adhesions. A 3.5 cm jejunal mass with lymphovascular invasion and negative margins was identified and frozen section showed findings of adenocarcinoma. His symptoms before surgery were abdominal pain and obstructive symptoms, but no unintentional weight loss. His only therapy at the time was anti-TNF therapy. After various therapies he has shown positive results with Herceptin and Perjeta.

Case Description/Methods: Preliminary pathology was consistent with T3NxM0 adenocarcinoma of the small intestine. Capecitabine was recommended for a 6 month course, but the patient developed significant diarrhea and hand-foot syndrome after his second cycle and therapy was unable to continue following 2 cycles despite dose reduction. Patient was followed closely with CEA and CT. Thirteen months following his surgery, recurrent disease was found due to increased CEA of 16.6 and PET scan showed focal thickening of the small bowel in the left lower quadrant and mesenteric masses and lymph nodes extending toward the central root of the mesentery. Upper endoscopic ultrasound was performed with fine needle biopsy was positive for metastatic adenocarcinoma. Patient then began FOLFOX 7 chemotherapy and then transitioned to maintenance 5-FU with leucovorin. Due to disease progression he was transitioned to FOLFIRI (irinotecan, leucovorin, and fluorouracil). He then underwent next generation sequencing with ERBB2 amplification identification. He was then initiated on Enhertu. Due to GI side effects, he transitioned to Herceptin and Perjeta every 3 weeks. Imaging shows a complete clinical response to therapy. His CEA has normalized from a peak of 30.9. These results have continued over 6 months has been responding well with most recent level 1.5. Lifestyle has improved with energy and appetite gradually returning to normal.

Discussion: In this case, we report an ongoing complete response to Her2-directed therapy in a patient with an ERBB2 mutation. HER2 positive adenocarcinoma of the small bowel is uncommon, and using next generation sequencing should be considered in all patients with metastatic disease to allow for targeted therapy.

S3536

A Unique Case of Paroxysmal Anxiety Leading to the Diagnosis of a Rarely Seen Neuroendocrine Tumor at the Ampulla of Vater

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Introduction: We present a rare case of worsening anxiety, leading to the work-up and diagnosis of a well differentiated neuroendocrine tumor of the ampulla of Vater.

Case Description/Methods: A 37-year-old black female with no significant past medical history was being evaluated for complaints of worsening anxiety. She stated that her anxiety was not situational and was paroxysmal in nature without an obvious stressor, and the duration of her attacks varied. She denied any skin flushing, diarrhea, frequent urination, increased thirst, dizziness, shakiness, hoarseness, cough, or jaundice She was noted to have a history of elevated liver function tests and alkaline phosphatase on laboratory testing. Her total bilirubin, prothrombin time and partial thromboplastin time were within normal limits. Extensive work-up was performed and found to be negative, including but not limited to; immunoglobulin A, celiac panel, hepatitis panel, human immunodeficiency virus, ceruloplasmin, AFP tumor marker, anti-mitochondrial antibody and anti-smooth muscle antibody. Ultrasound of the abdomen revealed a mildly dilated bile duct. Further evaluation with a magnetic resonance cholangiopancreatography study was performed and revealed prominent dilatation of the intrahepatic, extrahepatic and common bile ducts with a possible lesion at the ampulla of Vater. Endoscopic ultrasound with biopsy of the ampulla of Vater revealed a well differentiated neuroendocrine tumor. She was referred to surgery for removal of the malignancy.

Discussion: Neuroendocrine tumors involving the ampulla of Vater are rarely seen in clinical practice. The clinical presentation of this patient was relatively benign, however her labs consistently showed elevations which could not otherwise be explained. Due to the rarity of this malignancy in this location, it's imperative for physicians to be aware of how a patient could possibly present to avoid missing a

potential diagnosis or ordering an imaging test which could lead to a diagnosis. This case highlights the lack of symptoms which can be exhibited by these patients; however, the random variable anxiety attacks she experienced can now be attributed to her underline malignancy.

S3537

Adult Intussusception: Keep a Close Eye on the Telescope

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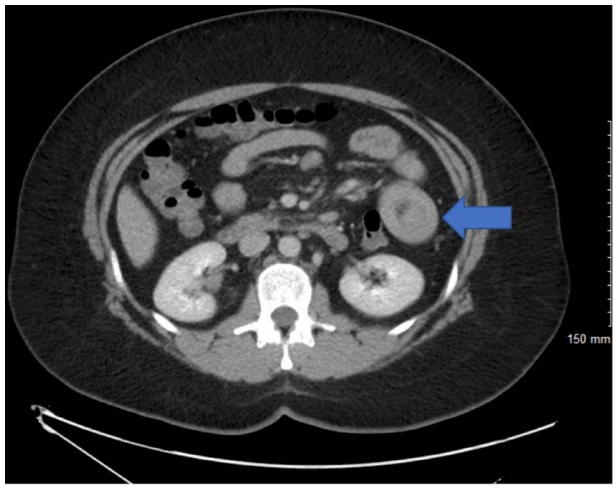
Introduction: Intussusception is when part of the bowel invaginates or telescopes into itself; commonly seen in infants 3 months to 2 years old. The overall incidence of adult intussusception is 2-3 cases/ 1,000,000 population/year. It is considered a medical abdominal emergency as it may result in a small bowel obstruction, peritonitis or bowel perforation.
Case Description/Methods: A 26 year old female with no medical history presented with vomiting, colicky abdominal pain, and watery diarrhea for two days. Despite being recently treated for gastroenteritis, she continued to have symptoms. Initial vitals were significant for tachycardia and fever. Laboratory results were unremarkable. She appeared to be in acute distress; dry heaving, rigors, extreme nausea and diffuse abdominal tenderness. Suspicious for appendicitis, a CT abdomen/pelvis with contrast was obtained. Results revealed mild sclerosing mesenteritis and short segment jejuno-jejunal intussusception measuring up to 4.4cm in length. Stool culture, blood culture, c. difficile antigens and HIV were negative. She was observed for self-resolution of the intussusception. Serial abdominal exams were performed along with bowel rest. She was started on a clear liquid diet 48 hours later and her symptoms of nausea and vomiting returned despite antiemetic medication. A repeat CT abdomen/pelvis to rule out obstruction demonstrated interval resolution of the previously visualized jejuno-jejunal intussusception. After 72 hours, with no intervention, her abdominal pain improved and was tolerating a diet. (Figure)
Discussion: Intussusception is commonly overlooked in adults as the diagnosis is unusual in this patient population. The majority of cases in adult intussusception are due to a pathological lead point within the bowel. Common risk factors include endometriosis, bowel adhesions, malignancy, polyps, and AIDS. Presenting symptoms include intermittent abdominal pain (most common), partial mechanical bowel obstruction, nausea, vomiting, and fever. Adult intussusception is diagnosed with CT abdomen. A 'target sign' (bowel appears thickened as it has two layers of bowel) may be seen on the sagittal view, while on axial or coronal view, it will appear as a sausage-shaped mass. Intussusception in adults is rare, representing 5% of all intussusceptions. However, as demonstrated in our patient, can occur without any obvious lead point. Manouras A, Lagoudianakis EE, Dardamanis D et al Lipoma induced jejunojejunal intussusception. World J Gastroenterol 2007; : 3,641–3,644.



[3537] **Figure 1.** Enteroenteric intussusception. Axial CT image demonstrating a round mass with "target" pattern (arrow).

S3538

An Atypical Case of Suspected Small Bowel Metastasis From a Gastric Primary Site

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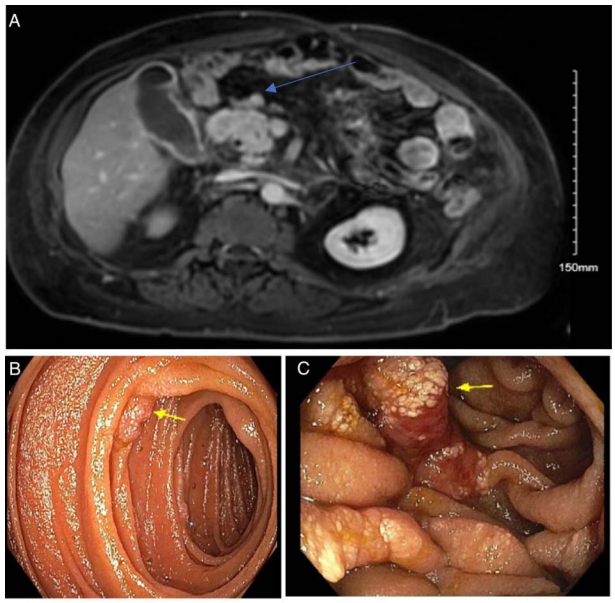
Introduction: Small bowel neoplasms are significantly rarer in comparison to their colonic and gastric counterparts, with primary and secondary lesions accounting for a combined estimated 1-2% of all gastrointestinal malignancies. Primary lesions of the small bowel are most commonly adenocarcinomas, followed by small bowel neuroendocrine tumors, lymphomas, and sarcomas. Metastases to the small intestine may be associated with contiguous spread, lymphatic spread, extraluminal invasion in the setting of peritoneal carcinomatosis, or hematogenous spread often from melanoma, breast, or lung primaries. Given the infrequency with which small intestinal cancer is observed, such a diagnosis may easily be missed.
Case Description/Methods: We report on the case of a 68-year-old male with a history of gastric adenocarcinoma 2 years status post subtotal gastrectomy and extensive lymphadenectomy followed by adjuvant chemotherapy, who presented for evaluation after 1 episode of melena. Initial laboratory workup was notable for a hemoglobin of 7.8 g/dL. The patient had been admitted twice in the previous months with symptomatic anemia requiring multiple transfusions. MRI from these recent prior hospitalizations revealed regions of retroperitoneal lymphadenopathy with the most notable conglomerate measuring 4.0 x 1.3 cm anterior to the pancreatic head. Serial EGDs/push enteroscopies at that time revealed friability/bleeding at the gastrojejunal anastomosis requiring clip placement. There were also several small flat hypopigmented nodules scattered in the efferent limb; biopsy from one of these sites returned positive for adenocarcinoma. Repeat push enteroscopy with biopsy was done. Anastomotic biopsies taken demonstrated only mild foveolar hyperplasia, while biopsies from 5 separate jejunal nodules were positive for poorly differentiated adenocarcinoma with extensive lymphovascular invasion. (Figure)
Discussion: Metastatic small bowel cancer originating from a gastric primary site without evidence of either direct extension or peritoneal implants - as is seen in this patient - is a phenomenon not described in the current literature. Given the multifocality of this patient's jejunal disease, the present working diagnosis is metastatic recurrence of known gastric adenocarcinoma. Follow up immunostaining is pending to confirm these suspicions and to rule out the presence of a primary multifocal small bowel malignant process.



[3538] **Figure 1.** A) Lymphadenopathy anterior to pancreatic head seen on MRI from a previous admission I B & C) Regions of jejunal nodularity visualized on push enteroscopy during most recent admission.

S3539

ANCA-Associated Leukocytoclastic Vasculitis With Mesenteric Involvement

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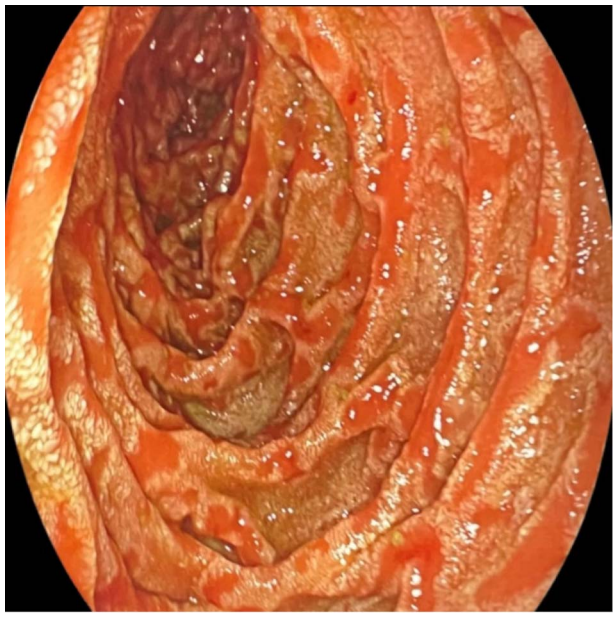
Introduction: Leukocytoclastic vasculitis (LCV) is a usually self-limiting, idiopathic small-vessel vasculitis with cutaneous symptoms. Extracutaneous manifestations should raise suspicion for underlying autoimmune conditions such as ANCA-associated vasculitis, of which LCV may be the initial manifestation. Mesenteric involvement is uncommon and usually represents advanced disease. Delay in recognition of mesenteric vasculitis can lead to progression and perforation. We present a case of gastrointestinal (GI) hemorrhage caused by ANCA-associated LCV.
Case Description/Methods: A 69-year-old male with a history of bilateral inguinal hernia repair was admitted for post-surgical groin wound infection. He was started on antibiotics and underwent mesh removal, washout, and debridement. He developed shock, renal failure, small vessel ischemic cerebrovascular infarction, and upper extremity papular eruptions. Work-up was notable for elevated inflammatory markers, decreased C3, negative ANA, positive C-ANCA and negative P-ANCA. He met Sapporo criteria for antiphospholipid syndrome given small vessel arterial thrombosis and positive lupus anticoagulant. Shave biopsy of skin lesions confirmed LCV. He was started on IV methylprednisolone and heparin infusion. He subsequently developed melena and anemia. EGD showed grade C esophagitis, gastropathy, and diffuse erosive duodenopathy (Figure). He was diagnosed with ANCA-associated LCV with mesenteric involvement. Bleeding resolved after holding heparin and treatment with pantoprazole. He was transitioned to prednisone and started on rituximab with significant improvement.
Discussion: Though uncommon, mesenteric vasculitis merits consideration in patients with abdominal pain or GI bleeding and clinical evidence of systemic autoimmune disease. Symptoms arise from intestinal ischemia which can progress to infarction and perforation if untreated. Endoscopy should be pursued with great caution due to increased perforation risk. Histopathology of superficial mucosal biopsies usually shows mucosal damage but does not yield a definitive diagnosis. Classic endoscopic findings include mucosal pallor, edema, or necrosis dispersed between normal mucosa. Ulcers and bleeding can be seen in severe cases. Timely recognition of GI manifestations of ANCA-associated LCV and initiation of therapy are pivotal in ensuring reduction of morbidity, mortality, and the need for surgery. A high index of suspicion for infarction or perforation warrants early surgical intervention.



[3539] **Figure 1.** Endoscopic evidence of diffuse erosive duodenopathy interspersed between normal mucosa in the setting of mesenteric vasculitis.

S3540

An Extremely Rare Case of Primary Duodenal Bulb Adenocarcinoma Presenting as Epigastric Abdominal Pain

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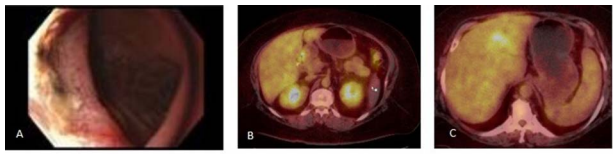
Introduction: Adenocarcinoma of small the bowel is a rare entity with less than 3% of gastrointestinal cancers. The duodenum is the most involved segment of the small bowel followed by the jejunum and ileum. Duodenal bulb adenocarcinoma is, however, an extremely rare finding with very few cases reported. In this case, we present primary duodenal bulb adenocarcinoma that manifested as epigastric pain with nausea.
Case Description/Methods: 55 y/o female with history of Diabetes that presented for GI consultation for two months of epigastric pain and nausea. Her endoscopy showed a single 3 cm cratered ulcer in the posterior duodenal bulb. Duodenal bulb biopsy showed poorly differentiated adenocarcinoma. PET CT scan showed a 2.9x1.8cm eccentric mass of the first portion duodenum and non-measurable left lobe hepatic hypodensity. CT Chest Abdomen and Pelvis showed unchanged 3.1cm soft tissue density in the duodenal bulb and interval increase of an hypoehancing mass of the left hepatic lobe. Her biopsy of the left hepatic lesion showed poorly differentiated adenocarcinoma. In the interim, she developed jaundice and PTC showed stenosis in the central region of the posterior branches of the right biliary tree and had internal-external biliary drain placed. Multidisciplinary tumor discussion was conducted with Medical Oncology, Surgical Oncology, Radiation Oncology, Radiology and Pathology with final recommendations proceed with FOLFOX chemotherapy. (Figure)
Discussion: Duodenal adenocarcinoma is rare with a very aggressive trajectory. A large population study indicates that majority of Duodenal adenocarcinoma arise in the D2 followed by D3/D4. Presentation in the first portion of the duodenum especially at the duodenal bulb is extremely rare. Patients tend to present with advanced disease due to nonspecific symptoms such as abdominal pain which is the most common presenting symptom. Endoscopy is the favored approach for visualization and biopsy. Contrast enhanced cross-sectional imaging (CT) is a valuable tool for assessing involvement and planning for surgery. Interventions ranges from radical resection (pancreaticoduodenectomy) which has the best outcome to palliative chemotherapy. Primary duodenal bulb adenocarcinoma is an extremely rare finding with only few cases reported. This is an intriguing phenomenon and raises the question if the duodenal bulb mucosa is inherently advantaged environmentally or physiologically to resist to malignant transformation.



[3540] **Figure 1.** A: Endoscopic imaging finding of 3 cm cratered ulcer in the posterior duodenal bulb Figure B: PET CT scan showing 2.9x1.8cm eccentric mass of the first portion duodenum Figure C: PET CT scan showing non-measurable left lobe hepatic hypodensity.

S3541

Ampullary Somatostatinoma: Diagnostic Challenge and Associations,

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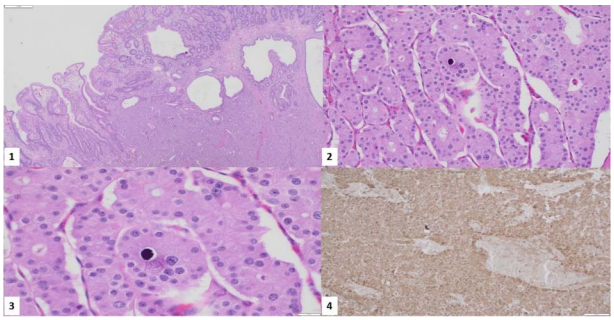
Introduction: Duodenal somatostatinoma is an exceedingly rare neuroendocrine tumor (NET) originating from the delta cells of the pancreas or enterochromaffin cells of the duodenum. While pancreatic tumors mostly produce clinical symptoms due to excess somatostatin production, duodenal tumors are often asymptomatic or can present with biliary obstruction symptoms such as abdominal pain, jaundice, or cholelithiasis. We report a subtle case of somatostatinoma arising from an ampulla.
Case Description/Methods: 64-year-old male started experiencing sudden onset shortness of breath, melena, intermittent abdominal pain, and anorexia a few months after being involved in an uneventful motor vehicle collision. Imaging displayed 2 cm ampullary nodule protruding into the duodenum. The upper endoscopy revealed non-obstructing mass, biopsy of which showed well differentiated NET. The Whipple resection unveiled 2.5 cm subepithelial lesion, infiltrating the sphincter of Oddi and duodenal muscularis propria. Histologically, lesions were composed of small nests of cells with stippled chromatin arranged in a glandular pattern. Focal psammoma bodies within the glandular lumina were appreciated. Immunohistochemically tumor cells were positive for synaptophysin, chromogranin, CAM-5.2, and somatostatin, confirming the diagnosis of somatostatinoma (Figure). Three peripancreatic lymph nodes were positive for metastatic carcinoma.
Discussion: According to The World Health Organization criteria, most duodenal somatostatinomas are malignant, and hence prompt diagnosis is crucial, but can be challenging particularly in non-functional ones as in our case. Seeing psammoma bodies in histology are diagnostic clues. Somatostatin analogs such as Octreotide and Lanreotide are used as first-line agents after confirmation. Ampullary tumors may require genetic evaluation since their Neurofibromatosis type 1 and other syndromic associations.



[3541] **Figure 1.**

S3542

An Uncommon Cause of Abdominal Obstruction: A Jejunal GIST

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Introduction: Gastrointestinal Stromal Tumors (GISTs) are rare neoplasm representing only 1% of all primary GI tumors. Yet they are the most common mesenchymal tumor (80%) of the GI tract with an age of onset usually between the seventh decade of life and a similar male-to-female ratio. GISTs can originate at any site from the esophagus to the anus and in fewer cases outside the GI tract. Therefore, having a wide range of presentation from asymptomatic to signs of acute abdomen. We report a case of a jejunal GIST causing chronic obstruction of the small intestine.
Case Description/Methods: Case of a 54-year-old male, inmate with medical history of Hepatitis C arrives to the emergency department with complaints of intractable emesis and abdominal discomfort. The patient reports 8-10 episodes/day of gastric content vomiting that eventually turned bilious for the past 2 weeks. Furthermore, he refers having epigastric discomfort associated with fatigue, anorexia, and weight loss of 20 lbs since approximately 2 months ago. The patient denied any family history of gastrointestinal disease. On evaluation, he appeared chronically ill with signs of hypovolemic shock. Physical exam was remarkable for a peri-umbilical mass with no tenderness to palpation. Abdominal CT showed an exophytic soft tissue mass arising from the mid ileum that measured approximately $4.5 \times 5.3 \times 4.7$ cm with associated slight swirling of the mesentery and upstream bowel loops, resulting in transition point and upstream dilatation of the small bowel compatible with a partial high grade small bowel obstruction. He underwent percutaneous biopsy with pathology report resulting in a spindle cell lesion, high risk, most consistent with GIST. Immunohistochemistry was only positive for C-KIT with a mitotic rate $> 5/5$ mm^2. The patient had surgical excision of jejunal mass via small bowel resection and was discharged on tyrosine kinase inhibitor. At 6 months follow up, the patient was found disease free.
Discussion: Occasionally, GISTs are found incidentally on imaging, predominantly in the stomach and small intestine, respectively. In our case, the patient presented with a KIT-positive jejunal GIST causing abdominal obstruction. Neoadjuvant therapy with Imatinib was given due to its elevated mitotic rate and high risk for progression. Early detection of these tumors requires a high level of suspicion hence the necessity of additional investigation to improve the prognosis and survival rates in this population.

S3543

An Unusual Complication of Peptic Ulcer Disease

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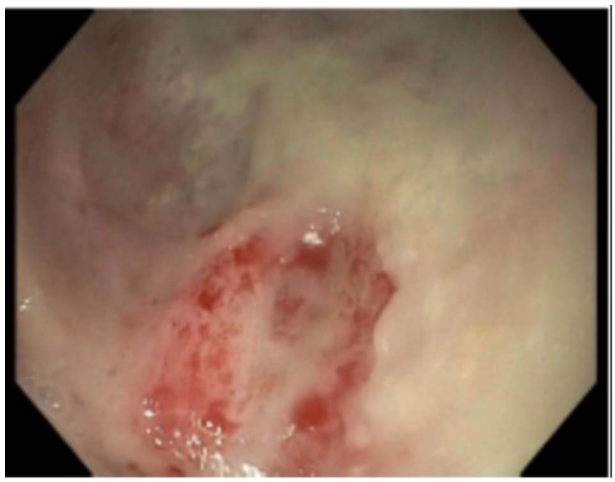
Introduction: In patients with severe peptic ulcer disease (PUD), perforation is a serious complication, often presenting with acute abdominal pain. Perforation occurs in approximately 5% of patients with PUD over their lifetime. Although fistulas have been reported as a potential complication of postoperative perforated peptic ulcer disease, we present a unique case of antral-abscess fistula complicated by duodenal adenocarcinoma.
Case Description/Methods: A 57-year-old female presented to the hospital with a chief complaint of epigastric pain. The patient had a history of perforated gastric ulcer status post graham patch repair. Computed Tomography (CT) of abdomen revealed remote gastric perforation with chronic fistulous connection between the gastric antrum and an abscess along the superior margin of the gastric antrum, underneath the left liver. Upper GI endoscopy revealed diffuse mildly erythematous mucosa without bleeding in the stomach as well as non-bleeding large cratered chronic appearing duodenal ulcer with a clean ulcer base (Forrest Class III) in the duodenal bulb involving more than 90% of the duodenal circumference, causing narrowing at the duodenal sweep and 2nd portion of duodenum. There were multiple ulcer/mucosal changes spots of bluish/reddish concerning for non-bleeding visible vessel and fistula given the severity of the ulcer/mucosal changes. Subsequent duodenal ulcer biopsy revealed necrotic tissue with infiltrating cells which were significant for adenocarcinoma of the duodenum (Figure).
Discussion: To our knowledge, this is the first report demonstrating antral stomach-abscess fistula with concomitant evidence of duodenal adenocarcinoma after perforated peptic ulcer disease. With this report, we not only aim to shed light on this unique complication but also to spark the scientific community to further investigate potential associations of duodenal adenocarcinoma including peptic ulcer disease.



[3543] **Figure 1.** Endoscopy of duodenum demonstrating ulcerations concerning for non-visible bleeding vessels and fistula.

S3544

An Important Syncopal Event: Syncope as the Presenting Sign of Primary Small Bowel Diffuse Large B Cell Lymphoma

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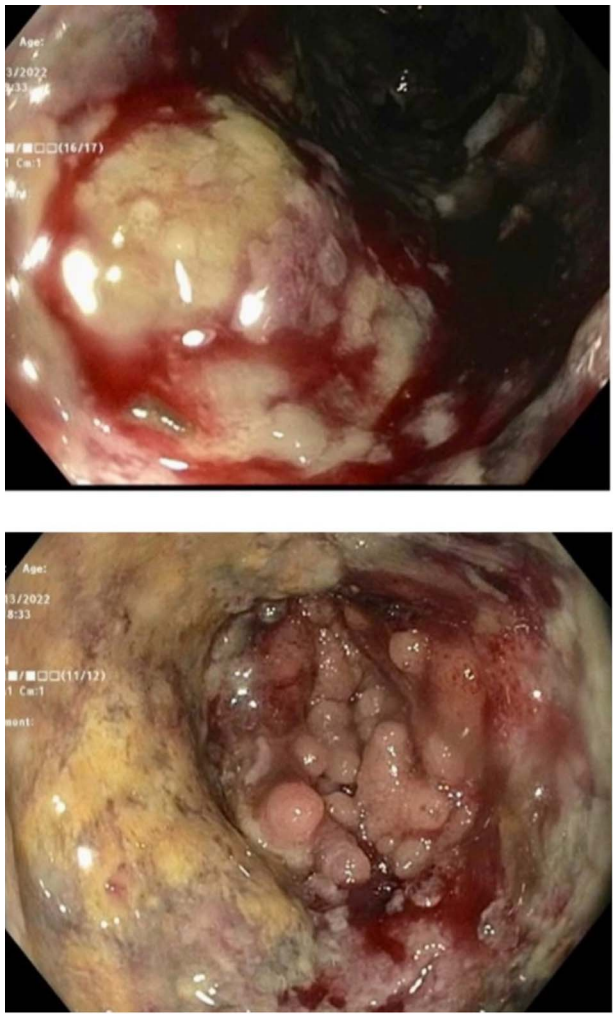
Introduction: Primary tumors of the small intestine are very rare and account for less than 2% of all gastrointestinal (GI) malignancies. Due to their rarity and non-specific symptoms, they are often missed on initial evaluation. We present a case of syncope leading to the diagnosis of primary small bowel lymphoma in the absence of predisposing risk factors or other GI-related symptoms.
Case Description/Methods: A 58-year-old man with history of GERD on PPI therapy presented for further evaluation following a syncopal episode. Cardiac workup including nuclear stress test was unremarkable, but he was found to have iron deficiency anemia with a hemoglobin of 10.4 in the absence of overt GI bleeding. With suspected symptomatic anemia and no prior endoscopic evaluation, he underwent EGD which was unrevealing, but his colonoscopy was significant for fresh blood throughout the entire colon and a 10 cm frond-like/villous, non-obstructing, circumferential mass in the terminal ileum. Subsequent CT scan of the abdomen/pelvis confirmed a non-obstructive, 8 cm distal ileal mass with innumerable lobulated and irregular lymph nodes suggesting mesenteric lymphadenopathy either from small bowel lymphoma or carcinoma but no features of peritoneal carcinomatosis. Endoscopic biopsy of the mass was consistent with Diffuse Large B cell Lymphoma (DLBCL). During his hospitalization, he underwent a right hemicolectomy and ileocecectomy with primary ileocolonic anastomosis. Surgical pathology revealed DLBCL (non-germinal center subtype). All regional lymph nodes were benign. He was started on first-line chemotherapy with R-CHOP. On presentation, he denied B-symptoms, abdominal pain, nausea, vomiting, melena or hematochezia (Figure).
Discussion: Lymphoma involving the GI tract are mostly secondary to widespread nodal disease. However, primary GI lymphomas constitute 1-4% of all GI malignancies. Primary small intestine lymphomas account for 20-30% of all GI lymphomas and can have varied presentations. The most common pathology subtype is DLBCL. Clinical presentations include abdominal pain, nausea, vomiting, weight loss, obstructive symptoms, and perforation, while B symptoms and syncope are rare. Risk factors include celiac disease, HIV/AIDS and EBV infection. Although there have been notable advances in diagnosis and treatment of GI lymphomas, they portend poor prognosis due to advanced disease at the time of diagnosis. Given their non-specific presentation, it is important to always consider small bowel lymphoma as a differential diagnosis.



[3544] **Figure 1.** Endoscopic views of large friable villous mass in Terminal Ileum.

S3545

An Unusual Case of Alveolar Hemorrhage Responding to a Gluten-Free Diet

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Introduction: Idiopathic pulmonary hemosiderosis (IPH) is a rare cause of hemoptysis in children. The association of IPH with celiac disease is infrequent and has been described in the pediatric population as Lane-Hamilton syndrome (LAH). We present a case of an adult patient with suspected LAH that responded to a gluten-free diet.
Case Description/Methods: A 47-year-old male presented with cough, and shortness of breath, associated with abdominal bloating and diarrhea. Laboratory workup was significant for a low hemoglobin level. High-resolution computed tomography of the chest (HRCT) revealed a diffuse, non-specific inflammatory process with reactive hilar lymphadenopathy. The patient underwent bronchoscopy with endobronchial ultrasound (EBUS), and bronchoalveolar lavage (BAL) was performed; however, an attempt to obtain a biopsy was unsuccessful after the patient had oxygen desaturation during the procedure. BAL showed abundant hemosiderin-laden macrophages, three times more than normal. Tissue transglutaminase and gliadin-A antibodies were positive suggestive of celiac disease. Given the high suspicion of LAH, the patient was started on a gluten-free diet, and his symptoms, hemoglobin level, and CT imaging significantly improved after a 3-month follow-up.
Discussion: The combination of IPH and celiac disease, known as Lane-Hamilton syndrome, is extremely rare. The association between these two conditions remains unclear and is presumed immune-mediated. Very few cases have been reported in adults with limited literature on management. Screening for celiac disease is recommended for suspected IPH, and a gluten-free diet trial is suggested even in the adult population.