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Anatomy and physiology information about stomach and duodenum.

The stomach is an organ of the digestive system, specialized in the accumulation and digestion of food. Its anatomy is quite complex; it consists of four parts, two curvatures and receives its blood supply mainly from the celiac trunk. Innervation is provided via the vagus nerves and the celiac plexus.

The stomach is the most dilated part of the digestive system, lying between the esophagus and duodenum. More precisely, the stomach spans the region between the cardiac and pyloric orifices of the gastrointestinal tract. It is covered and connected to other organs by peritoneum. The lesser omentum connects the stomach to the liver and then extends around the stomach. The greater omentum then continues inferiorly from the stomach, hanging from it like a curtain.

The stomach is located inside the abdominal cavity in a small area called the bed of the stomach, onto which the stomach lies when the body is in a supine position, or lying face up. It spans several regions of the abdomen, including the epigastric, umbilical, left hypochondriac, and left flank regions. The stomach also has some precise anatomical relations and comes in contact with several neighboring structures.

The stomach consists of several important anatomical parts. The four main sections of the stomach are the cardia, fundus, body, and pyloric part. As the name implies, the cardia surrounds the cardiac orifice, which is the opening between the esophagus and the stomach. It is the first section that ingested food passes through, representing the inflow part. The fundus is the superior dilation of the stomach, which is located superiorly relative to the horizontal plane of the cardiac orifice.

The main function of the stomach involves mechanical and chemical digestion of ingested food. Ingested food enters the stomach from the esophagus via the cardiac orifice, falling into gastric juice produced by the stomach. Repetitive muscle contractions physically churn food particles, breaking them into smaller fragments which are mixed with the gastric juice. The various enzymes and hydrochloric acid (pH 1-2) in the gastric juice break food down even more, forming a semi-liquid substance called chyme. This ultimately passes into the duodenum through the pyloric orifice by a process called gastric peristalsis. Being a muscular organ, the stomach can distend quite a lot, accumulating anywhere between 2 and 3 liters of food.

The overall blood supply of the stomach originates from the abdominal aorta and is provided from two anastomotic systems along the curvatures and several direct branches. The anastomosis along the lesser curvature is created by the union of the right and left gastric arteries which originate from the common hepatic artery and celiac trunk respectively. The greater curvature anastomosis is formed by the union of the right and left gastroomental arteries (gastroepiploic), which originate from the gastroduodenal and splenic arteries respectively.

The splenic artery also sends out short and posterior gastric arteries, which directly supply the fundus and upper body of the stomach. The pyloric part receives arterial blood from the gastroduodenal artery, which stems from the common hepatic artery.The veins draining the stomach follow the course and nomenclature of the arteries very closely. They ultimately drain into three large vessels called the hepatic portal, splenic, and superior mesenteric veins.

The stomach receives involuntary innervation by the autonomic nervous system (ANS). Parasympathetic innervation originates from the anterior and posterior vagal trunks, which stem from the left and right vagus nerves (CN X), respectively. The anterior vagal trunk mainly supplies a portion of the anterior surface of the stomach, as well as the pylorus. The larger posterior vagal trunk innervates the remaining anterior surface, as well as the entire posterior surface. Parasympathetic innervation is responsible for inducing gastric secretion and motility, as well as relaxation of the pyloric sphincter during gastric emptying. The vagus nerves also carry sensations of pain, fullness, and nausea from the stomach.

In contrast, sympathetic innervation is provided by the celiac plexus. The nerve impulses originate from the fifth to twelfth thoracic spinal nerves (T5-T12) and travel to the celiac plexus via the greater splanchnic nerves. Sympathetic innervation is responsible for inhibiting gastric motility and constricting the pyloric sphincter, thus preventing gastric emptying.

Lymph is drained from the stomach by lymph vessels that empty into the gastric and gastroomental lymph nodes. They are located along the arteries of the lesser and greater curvatures of the stomach. The pyloric part is drained by the superior and inferior pyloric lymph nodes. Subsequently, lymph vessels drain these sets of lymph nodes into the celiac lymph nodes, which are located around the celiac trunk.

The duodenum is the first of the three parts of the small intestine that receives partially digested food from the stomach and begins with the absorption of nutrients. It is directly attached to the pylorus of the stomach. It has a C-shape, it is closely related to the head of the pancreas and consists of four sections: superior, descending, horizontal, and ascending parts. Histologically speaking, it consists of the typical three layers common to all hollow organs of the gastrointestinal tract, but it has Brunner's glands, which is the characteristic feature of the duodenum. The duodenum is about 25 to 30 cm long (“twelve fingers' length”), C-shaped and is located in the upper abdomen at the level of L1-L3. The head of the pancreas lies in the C loop. It may be subdivided into four sections: superior part, descending part, horizontal part and ascending part. He superior part lies intraperitoneally and is enlarged proximally (duodenal bulb). It is connected to the liver by the hepatoduodenal ligament. The superior part ends at the superior duodenal flexure and becomes the descending part.

The descending part and the rest of the duodenum lieretroperitoneally. The common bile duct and the pancreatic duct unify to a conjoint duct at the hepatopancreatic ampulla (ampulla of Vater) and empties into the descending part of the duodenum. At the opening there is an elevation of the mucosa, the major duodenal papilla (papilla of Vater). Many people have an accessory pancreatic duct which empties into an additional papilla, the minor duodenal papilla (papilla of Santorini). The transition from the descending to the horizontal part of the duodenum takes places at the inferior duodenal flexure.

The horizontal part runs from right to left ventrally from the abdominal aorta and inferior vena cava.

The ascending part runs cranially along the left side of the vertebral column. This last part of the duodenum joins the intraperitoneally lying jejunum at the duodenojejunal flexure. Here the duodenum is attached to the back of the abdominal wall through the suspensory ligament of duodenum (ligament of Treitz). Clinically the ligament of Treitz marks the border between the upper and lower gastrointestinal tract.

Blood supply

The supply of blood to the duodenum is carried by the anterior and posterior superior pancreaticoduodenal arteries (branches of the gastroduodenal artery) and the inferior pancreaticoduodenal artery (branch of the superior mesenteric artery) which form an arterial arcade. The correspondent veins are responsible for the venous drainage.
The sympathetic innervation is carried by nerves of the coeliac plexus, the parasympathetic innervation by the vagus nerve (cranial nerve X).

Etiology and pathogenesis of peptic stomach and duodenal ulcers.

The discovery of *Helicobacter pylori* has evinced great interest in the role played by this microbe. The eradication of this organism has been found to be of paramount importance to minimize the complications of peptic ulcers. The management of peptic ulcer disease and its complications remain a challenge. In addition, non-steroidal anti-inflammatory drugs (NSAIDs), low-dose aspirin, smoking, excessive alcohol use, emotional stress and psychosocial factors are increasingly important causes of ulcers and their complications even in *H. pylori*-negative patients. Other rare causes of peptic ulcer disease in the absence of *H. pylori*, NSAIDs, and aspirin also exist.

The pathogenesis of peptic ulcer disease may be considered as a combination scenario involving an imbalance between defensive factors (mucus-bicarbonate layer, prostaglandins, cellular regeneration, mucosal blood flow) and aggravating factors (hydrochloric acid, pepsin, ethanol, bile salts, drugs). NSAIDs play an important role in the pathogenesis.
The pathology can be divided in three broad categories, (1) *H. pylori* positive (2) *H. pylori* negative and non-NSAID associated (3) NSAID associated.

Ulcers can exist in the absence of *H. pylori* infection and non-NSAID group. Zollinger-Ellison syndrome, truly idiopathic ulcers, Cushing's ulcer, high-dose upper abdominal radiotherapy.

Clinical features, diagnosing and treatment of uncomplicated peptic ulcer

Dyspepsia. The most common symptoms of peptic ulcer are known collectively as *dyspepsia*. However, peptic ulcers can occur without dyspepsia or any other gastrointestinal symptom, especially when they are caused by NSAIDs. Dyspepsia may be persistent or recurrent and can lead to a variety of upper abdominal symptoms, including:

- Pain or discomfort
- Bloating
- A feeling of fullness - people with severe dyspepsia are unable to drink as much fluid as people with mild or no dyspepsia
- Hunger and an empty feeling in the stomach, often 1 - 3 hours after a meal
- Mild nausea (vomiting may relieve symptoms)
- Regurgitation (sensation of acid backing up into the throat)
- Belching (отрыжка)
- Occasionally, symptoms of GERD are present

Many patients with the above symptoms do not have peptic ulcer disease or any other diagnosed condition. In that case, they have what is called *functional dyspepsia*.

Ulcer Pain. Some symptoms are similar to those of gastric ulcers, although not everyone with these symptoms has an ulcer. The pain of ulcers can be in one place, or it can be diffuse (all over the abdomen). The pain is described as a burning, gnawing, or aching in the upper abdomen, or as a stabbing pain penetrating through the gut. The symptoms may vary depending on the location of the ulcer:

- Duodenal ulcers often cause a gnawing pain in the upper stomach area several hours after a meal, and patients can often relieve the pain by eating a meal.
- Gastric ulcers may cause a dull, aching pain, often right after a meal; eating does not relieve the pain and may even worsen it. Pain may also occur at night.

Ulcer pain may be particularly confusing or disconcerting when it radiates to the back or to the chest behind the breast bone. In such cases it can be confused with other conditions, such as a heart attack.

Because ulcers can cause hidden bleeding, patients may experience symptoms of anemia, including fatigue and shortness of breath.

Radiological and endoscopic examinations of patients with peptic ulcer. Studies of gastric secretion, motility, data evaluation.

ENDOSCOPY

Endoscopy is a procedure used to evaluate the esophagus, stomach, and duodenum using an endoscope – a long, thin tube equipped with a tiny video camera. When combined with a biopsy, endoscopy is the most accurate procedure for detecting the

presence of peptic ulcers, bleeding, and stomach cancer, or for confirming the presence of *H. pylori*.

In endoscopy, the doctor places a long, thin, flexible tube (called an endoscope) down the patient's throat and into the stomach and duodenum. A camera and light on the tip of the endoscope enables the doctor to check for abnormalities. Tiny samples may be taken to check for H. pylori bacteria, a cause of many peptic ulcers. If a bleeding ulcer is found, it may be sealed with a burning tool (cauterized) during the procedure.

Endoscopy is usually reserved for patients with dyspepsia who also have risk factors for ulcers, stomach cancer, or both.

Risk factors include the following:

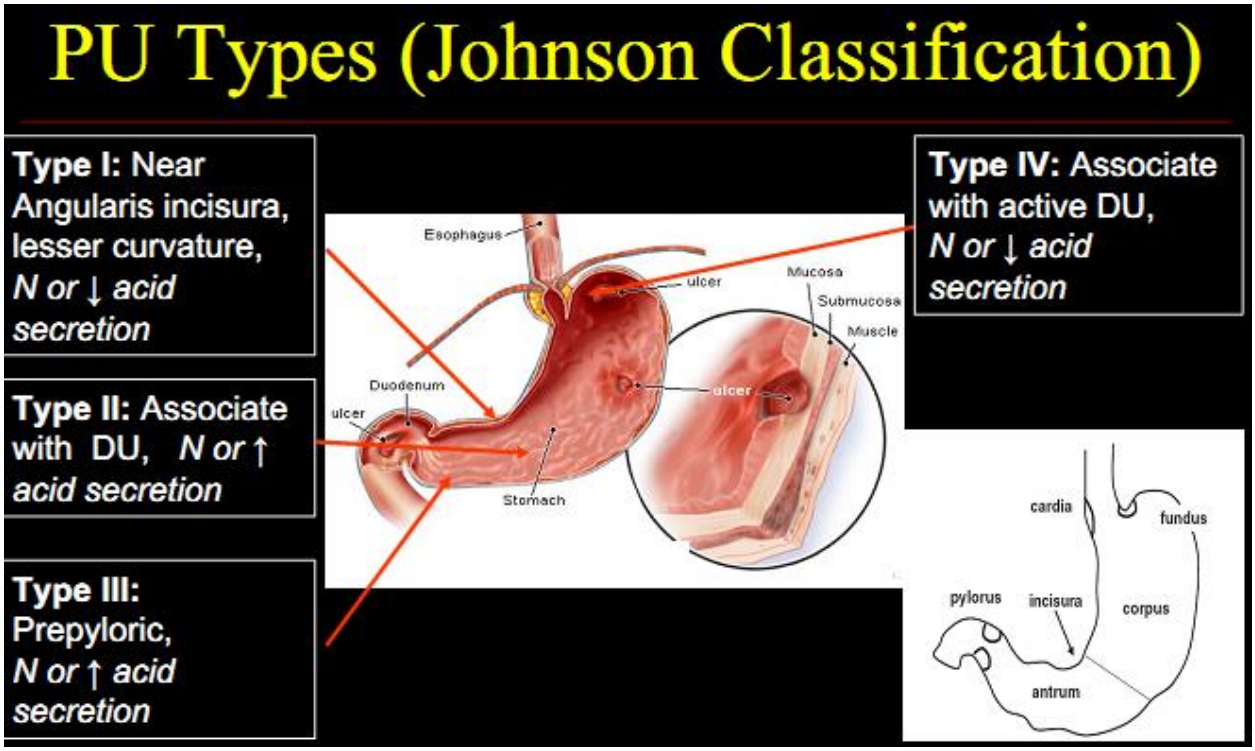
- "Alarm" symptoms (unexplained weight loss, gastrointestinal bleeding, vomiting, difficulty swallowing, or anemia). Patients with these symptoms generally have an endoscopy before treatment.
- Over age 55 (when the risk for stomach cancer increases)
- Failure to respond to medical treatment of *H. pylori*, if present

Experts disagree about whether endoscopy should be performed on all patients who do not respond to initial medication, unless there is evidence or suspicion of bleeding or serious complications, because it does not appear to add any useful information about treatment choices. There is also some debate about whether patients under age 45 who have persistent dyspepsia but no alarm symptoms should have an endoscopy.

Radiographic features In some patients, an abdominal X-ray may have been performed by emergency physician or primary medical team. It can show signs such as appearance of gas on both sides of the bowel wall (Rigler's sign), a large volume of free gas resulting in a large round black area (Football sign) and gas outlining soft tissue structures such as liver edge or falciform ligament. It is authors' practice not to perform an abdominal X-ray in patients with suspected PPU when chest X-ray does not show free air under the diaphragm. CT scan is recommended as it has a diagnostic accuracy as high as 98%. Besides, CT scan can exclude acute pancreatitis that would not need surgical intervention. CT scan is performed in supine position and free air is usually seen anteriorly just below the anterior abdominal wall. The falciform ligament can sometimes be visible when air is present on both sides. In resource poor healthcare facilities, oral gastrograffin can be used to diagnose PPU. Water-soluble contrast leaking into the peritoneal cavity can confirm the diagnosis of PPU. Absence of a leak does not exclude PPU as the perforation may have sealed off spontaneously. Barium study is contraindicated in gastrointestinal perforation and should be avoided as a tool to diagnose PPU. We consider lateral decubitis abdominal radiographs as obsolete and do not recommend. The traditional practice of instilling air *via* the nasogastric tube and repeating the erect chest X-ray after few minutes is not recommended except in resource poor facilities. It takes time and a repeat negative chest X-ray does not rule out the diagnosis of PPU and still a CT scan would be warranted. Rarely a CT scan is performed even when an erect chest X-ray reveals free air under diaphragm. The utility of this CT scan is justified when clinical presentation is not specific to upper gastrointestinal pathology or a malignancy is

suspected and patients' hemodynamics is not deranged. In patients with acute kidney injury, a non-contrast CT scan is adequate to see free air. Oral contrast with CT scan is a useful tool and if free leak is seen, diagnosis is certain.

Indications for surgical treatment of gastric ulcer and duodenal ulcer.



Indications for surgical treatment of gastric ulcer and duodenal ulcer.

Perforated ulcer

Profuse gasroduodenal bleeding

Pylorostenosis

Malignant ulcer

Penetration

Type 1-4 Johnsons classification

Types of operations for peptic ulcer and duodenal ulcer (gastric resection, ablative surgery).

The appropriate surgical procedure for peptic ulcer disease must be tailored to the specific needs of the individual patient. During emergency operations for hemorrhage from duodenal ulcer, recommended suture ligature of the bleeding vessel and vagotomy-pyloroplasty for high-risk patients, or vagotomy-antrectomy for the lower-risk patient. Bleeding gastric ulcers should be resected, if possible. For massive hemorrhage from stress ulceration requiring surgery, near-total or total gastrectomy should be performed. Perforated duodenal ulcers are best managed by closure and a definitive ulcer operation,

such as vagotomy-pyloroplasty. Perforated gastric ulcers are best excised but may be simply closed if conditions do not favor resection. In these situations, biopsy should be performed. Recommended truncal vagotomy-antrectomy for patients presenting with obstruction. Vagotomy (truncal or proximal gastric) with drainage is an acceptable alternative in this situation. For patients with intractable ulcer disease or for those who are noncompliant, proximal gastric vagotomy is the preferred operation. However, other operations may need to be considered, depending on the specific situation. Recurrent ulceration needs appropriate work-up to determine the possible cause. Although patients with ulcer recurrence initially may be placed on medical treatment, about 50% will require reoperation. The most effective procedure for peptic ulcer disease is truncal vagotomy-antrectomy, which has a recurrence rate of less than 1%. The procedure with the least morbidity and the fewest undesirable side effects is proximal gastric vagotomy. Ulcer recurrence after proximal gastric vagotomy or truncal vagotomy-pyloroplasty is in the range of 10% to 15%.

The standard major surgical approach uses a wide abdominal incision and standard surgical instruments. Laparoscopic techniques use small abdominal incisions, through which are inserted tubes that contain miniature cameras and instruments. Laparoscopic techniques are increasingly being used for perforated ulcers. Research finds that laparoscopic surgery for a perforated peptic ulcer is comparable in safety with open surgery, and results in less pain after the procedure.

There are a number of surgical procedures aimed at providing long-term relief of ulcer complications. These include:

Vagotomy, in which the vagus nerve is cut to interrupt messages from the brain that stimulate acid secretion in the stomach. This surgery may impair stomach emptying. A recent variation that cuts only parts of the nerve may reduce this complication.

Antrectomy, in which the lower part of the stomach is removed. This part of the stomach manufactures the hormone responsible for stimulating digestive juices.

Pyloroplasty, which enlarges the opening into the small intestine so that stomach contents can pass into it more easily.

Antrectomy and pyloroplasty are usually performed with vagotomy.

Clinical features, diagnosing and treatment of callosus, penetrating, malignant ulcers.

This type of ulcers has very bad or no- result after the conservative treatment. Patients have permanent abdominal pain with small effect medical therapy. Endoscopically this type of ulcers has deep damaging with fibrosis of the wall. Must do biopsy.

Clinical features, stages, diagnosing and treatment of perforated gastric ulcer and duodenal ulcer.

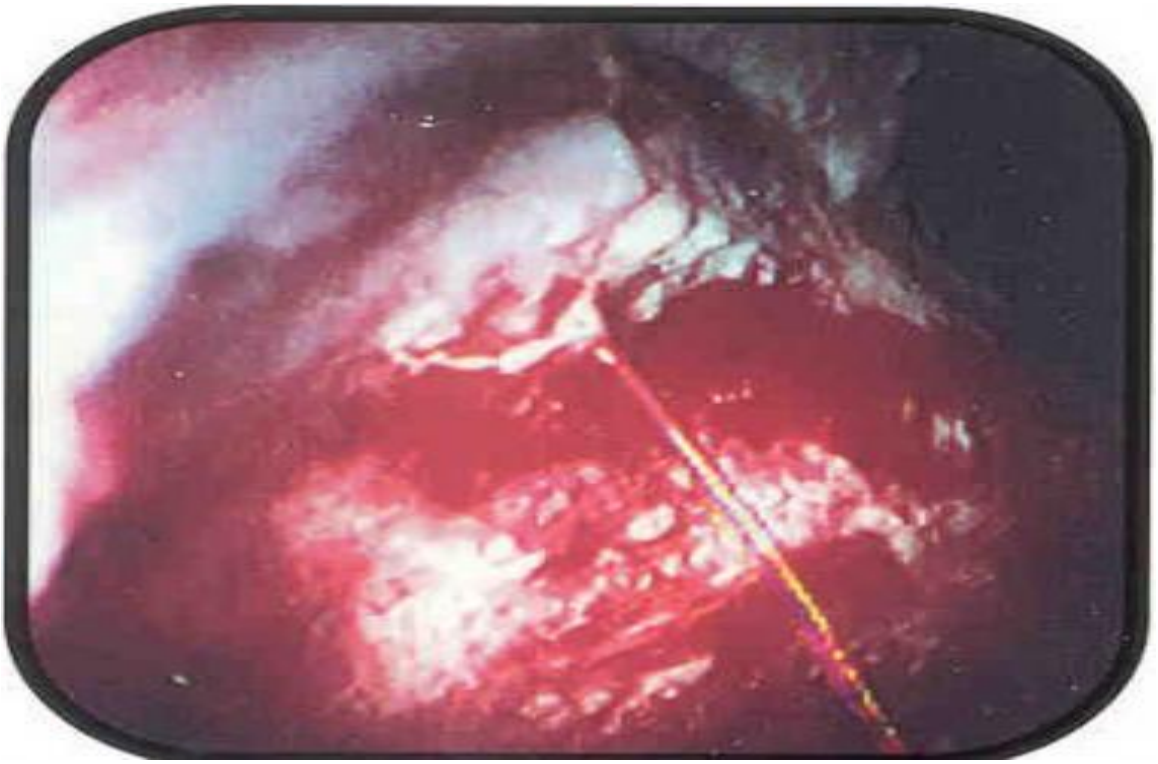
Patients with perforated peptic ulcer disease usually present with a sudden onset of severe, sharp abdominal pain. Most patients describe generalized pain; a few present with severe epigastric pain. As even slight movement can tremendously worsen their pain, these

patients assume a fetal position. Abdominal examination usually discloses generalized tenderness, rebound tenderness, guarding, and rigidity. However, the degree of peritoneal findings is strongly influenced by a number of factors, including the size of perforation, amount of bacterial and gastric contents contaminating the abdominal cavity, time between perforation and presentation, and spontaneous sealing of perforation.

These patients may also demonstrate signs and symptoms of septic shock, such as tachycardia, hypotension, and anuria. Not surprisingly, these indicators of shock may be absent in elderly or immunocompromised patients or in those with diabetes. Patients should be asked if retching and vomiting occurred before the onset of pain.

Surgical therapy is first line treatment for an ulcer with an associated perforation. The operation of choice for a duodenal perforation is the Graham patch repair. In this maneuver, a portion of the omentum is placed over the perforation and is secured in place with interrupted silk sutures. The sutures should be placed quite wide of the ulceration to prevent tearing through the friable tissue. Perforated gastric ulcers can also be treated with a Graham patch or excision with repair of the defect as done for a bleeding duodenal ulcer. Again, the specimen of the gastric ulcer should be sent for pathology to rule out a malignancy. Postoperatively, patients should be treated with acid suppression therapy and for H. Pylori infection, if positive. They should also be counseled on peptic ulcer disease.

Bleeding gastric ulcer and duodenal ulcer: pathological anatomy, pathogenesis, clinical features, diagnosing, treatment.



Gastric and duodenal ulcers are the most common cause of upper GI bleeding and occur in 50-70% of patients. However, bleeding is the presenting symptom in only 10% of patients with peptic ulcers. Bleeding from duodenal ulcers is four times more common than from

gastric ulcers. As described above, posterior duodenal ulcers are the most likely to bleed based on proximity to branches of the GDA. Significant bleeding occurs in 10-15% of peptic ulcers while 20% of these require surgical therapy for control. Acute bleeding is sudden and can sometimes be severe. Chronic bleeding is slight bleeding that can last a long time or may come and go.

Symptoms of the upper gastro-intestinal bleeding are black or tarry stool, bright red blood in vomit or coffee-like vomiting, dizziness or faintness, feeling tired, paleness, shortness of breath, weakness

Symptoms of shock include, a drop in blood pressure, little or no urination, a rapid pulse, unconsciousness

Chronic bleeding symptoms Chronic bleeding may develop anemia. Symptoms of anemia may include feeling tired and shortness of breath, which can develop over time. Some people may have occult bleeding. Occult bleeding may be a symptom of inflammation or a disease such as colorectal cancer . A simple lab test can detect occult blood in stool.

Diagnosis

Signs of anemia or dehydration including pallor, lethargy, dry mucous membranes, skin tenting, or flat neck veins. A thorough abdominal exam should be performed to locate any tenderness, rebound, guarding, or other signs of peritonitis. Pay attention to signs of cirrhosis including spider angiomata, prominent abdominal veins, caput medusa, and ascites. A rectal exam MUST be performed in patients presenting with a GI bleed in order to identify perianal causes of bleeding in addition to the presence of blood in the rectal vault. Perform an occult blood test at time of rectal exam. In women include a vaginal examination.
Workup includes the following:
Orthostatic blood pressure, common blood analyses (Er, Hb level), coagulogramm, group type of blood and Rh-factor, nasogastric lavage, endoscopy, chest radiography, calcium level, gastrin level,

To diagnose gastrointestinal (GI) bleeding, at first find the site of the bleeding based on medical history, family history, a physical exam, and diagnostic tests. All observation must be in the horizontal position of the patient to prevent orthostatic collapse. In situation of profuse bleeding patients (shock-symptoms) observation firstly, at the way to / or in the operation room in short time.

Endoscopy

Endoscopy procedures involve a doctor examining upper gastrointestinal tract using a special optical fibroscope. An endoscopy procedure may help to see if and where have GI bleeding and the bleeding's cause. Is main method to diagnose and treatment of the upper GIB. Endoscopy is typically the next step as it is both diagnostic and therapeutic. Esophagogastroduodenoscopy (EGD) evaluates the esophagus, stomach, and duodenum. Several different interventions can be performed during EGD to control the bleeding.

These include direct pressure, injection with vasoconstrictive properties (epinephrine, vasopressin), sclerotherapy, electrocautery, ligation, and clipping. Of note, ligation has been found to be as effective as sclerotherapy but with fewer complications and is practiced more commonly in the acute setting. Failed therapy and re-bleeding occurs in 55% of patients found to have active pulsatile bleeding or oozing at the time of endoscopy. A nonbleeding, visible vessel has 43% risk of rebleeding. Adherent clot carries a 22% risk of re-bleed, and a clean based ulcer has 0-5% chance of re-bleeding. Endoscopic intervention in patients with known cirrhosis and liver failure needs to be well planned given the inability to synthesize clotting factors. For acute bleeding, initial therapy should include ligation and vasopressin. The use of a Sengstaken-Blakemore tube which provides balloon tamponade can help temporize bleeds in unstable patients or those in whom EGD has not been successful.

Treatment

Management should begin with the ABCs (airway, breathing, circulation). The airway needs to be secured if sensorium is altered or the patient is unable to protect the airway. Simultaneously, two large bore IVs (16 gauge or larger) should be placed for arterial line in those patients with deteriorating clinical status for dynamic blood pressure monitoring. Fluid resuscitation should start with a 1L bolus of crystalloids, either NS or LR. If the patient responds well with improvement in hemodynamic parameters, a second 1L crystalloid bolus can be administered. If the patient remains unstable with a suspected GI bleed, resuscitation should be continued with packed RBCs. Fluid balance should be monitored with strict ins and outs and a Foley catheter may be placed for monitoring urine output. An NGT should also be placed and gastric lavage performed. Bloody return indicates a gastric or upper GI bleed.

Elective surgeries for gastric and duodenal ulcers have significantly decreased in frequency due to improved medical therapy with H2 blockers and proton pump inhibitors. However, the number of urgent or emergent surgeries for bleeding duodenal ulcers has remained somewhat stable. Indications for surgical intervention include uncontrolled bleeding in a patient with a known ulcer after failure of endoscopic treatment of the bleed. Pre-operative preparation includes adequate fluid resuscitation with either crystalloid or blood pending the status of the patient. The operative approach for an upper GI bleed is via an exploratory laparotomy through an upper midline incision. For a duodenal ulcer, dissection is carried out to expose the pylorus and first part of the duodenum. An anterior longitudinal duodenotomy is made extending through the pyloric channel to the distal stomach. Bleeding from the GDA complex is controlled with a three-vessel ligation technique. This consists of a superior suture, inferior suture, and a horizontal mattress suture creating a “U” stitch for the transverse pancreatic artery. A HeinekeMikulicz closure of the duodenotomy is then performed by closing the horizontal incision in a vertical fashion. Bleeding gastric ulcers are best treated with surgical excision of the ulcer and repair of the remaining gastric defect. A truncal vagotomy can be added to the operation for long-term ulcer control. However, this is not appropriate in an unstable or under resuscitated patient. Additionally, this should only be performed if the patient was

on adequate medical therapy prior to surgery. If a truncal vagotomy is performed, a 1 cm portion of each vagus nerve (anterior and posterior) is resected. It is necessary to send both of the vagal trunk specimens to pathology to document the vagotomy was performed successfully.

Mallory-Weiss syndrome: etiology, pathological anatomy, clinical features, diagnosing, treatment.

Mallory-Weiss syndrome is caused by prolonged or severe retching which results in a partial thickness mucosal tear at the gastroesophageal junction. This is in contrast to Boerhaave syndrome in which a full thickness tear results in mediastinitis. Mallory-Weiss tears account for about 5-10% of all upper GI bleeds. Classically, patients will present with an episode of vomiting without blood. Further emesis leads to pain and the development of hematemesis due to a tear in the esophagogastric mucosa. Bleeding due to a Mallory-Weiss tear resolves spontaneously 90% of the time with no further intervention required. Endoscopic therapy with epinephrine injection or balloon tamponade may be necessary in the other 10% that don't resolve spontaneously.

Pyloric and duodenal stenosis of ulcer etiology: pathogenesis, stages, diagnosing, preoperative preparation, types of operations.

Gastric outlet obstruction (GOO, also known as pyloric obstruction) is not a single entity; it is the clinical and pathophysiological consequence of any disease process that produces a mechanical impediment to gastric emptying.

PUD manifests in approximately 5% of all patients with GOO. Ulcers within the pyloric channel and first portion of the duodenum usually are responsible for outlet obstruction. Obstruction can occur in an acute setting secondary to acute inflammation and edema or, more commonly, in a chronic setting secondary to scarring and fibrosis. *Helicobacter pylori* has been implicated as a frequent associated finding in patients with GOO, but its exact incidence has not been defined precisely.

Patients present with intermittent symptoms that progress until obstruction is complete. Vomiting is the cardinal symptom. Initially, patients may demonstrate better tolerance to liquids than solid food. In a later stage, patients may develop significant weight loss due to poor caloric intake. Malnutrition is a late sign, but it may be very profound in patients with concomitant malignancy. In the acute or chronic phase of obstruction, continuous vomiting may lead to dehydration and electrolyte abnormalities.

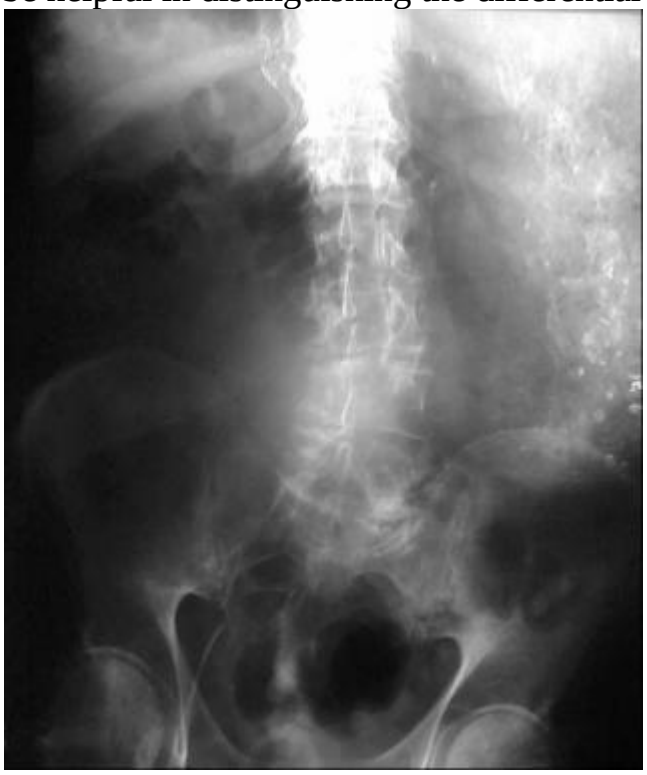
When obstruction persists, patients may develop significant and progressive gastric dilatation. The stomach eventually loses its contractility. Undigested food accumulates and may represent a constant risk for aspiration pneumonia.

Physical examination often demonstrates the presence of chronic dehydration and malnutrition. A dilated stomach may be appreciated as a tympanitic mass in the epigastric area and/or left upper quadrant.

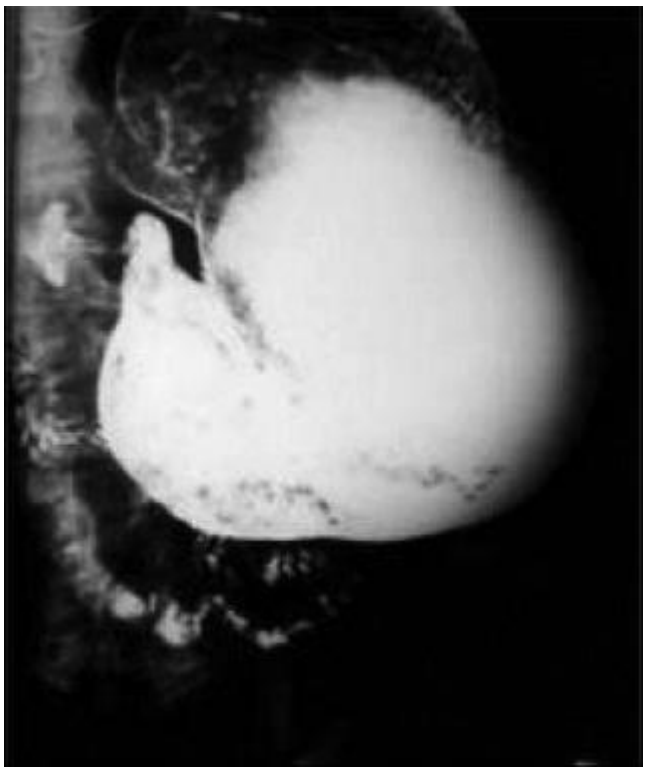
Dehydration and electrolyte abnormalities can be demonstrated by routine laboratory examinations. Increases in blood urea nitrogen (BUN) and creatinine are late features of dehydration.

Prolonged vomiting causes loss of hydrochloric acid and produces an increase of bicarbonate in the plasma to compensate for the lost chloride and sodium. The result is a hypokalemic hypochloremic metabolic alkalosis. Alkalosis shifts the intracellular potassium to the extracellular compartment, and the serum positive potassium is increased factitiously. With continued vomiting, the renal excretion of potassium increases in order to preserve sodium. The adrenocortical response to hypovolemia intensifies the exchange of potassium for sodium at the distal tubule, with subsequent aggravation of the hypokalemia.

Plain abdominal radiography, contrast upper gastrointestinal (GI) studies (Gastrografin or barium), and computed tomography (CT) with oral contrast are helpful. (See the images below.) Plain radiographs, including the obstruction series (ie, supine abdomen, upright abdomen, chest posteroanterior), can demonstrate the presence of gastric dilatation and may be helpful in distinguishing the differential diagnosis.

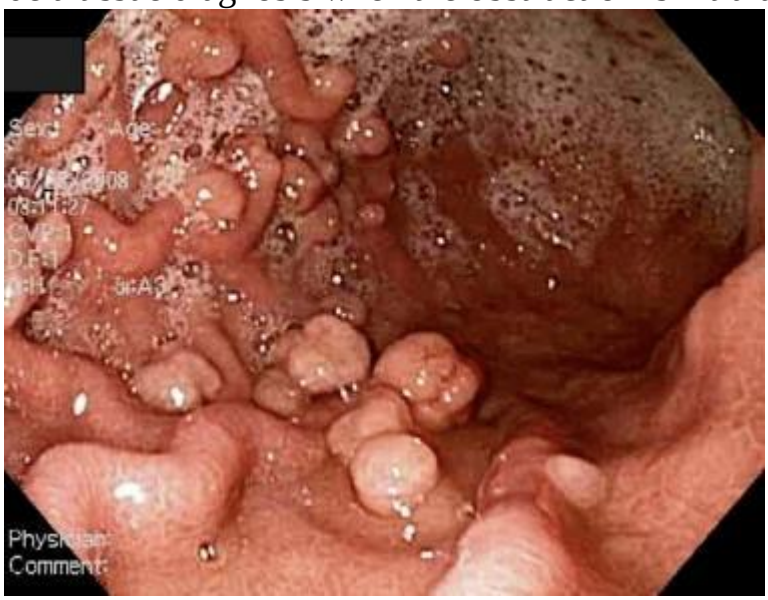


Plain radiograph of the abdomen. Enlarged stomach with calcified content.



Contrast study demonstrating an enlarged stomach. The point of obstruction is visualized at the pyloric-duodenal junction (string sign).

Upper endoscopy (see the image below) can help visualize the gastric outlet and may provide a tissue diagnosis when the obstruction is intraluminal.



Upper endoscopy showing multiple gastric polyps. Such polyps are a major cause of gastric outlet obstruction.

Barium upper GI studies are very helpful because they can delineate the gastric silhouette and demonstrate the site of obstruction. An enlarged stomach with a narrowing of the pyloric channel or first portion of the duodenum helps differentiate GOO from gastroparesis.

GOO due to benign ulcer disease may be treated medically if results of imaging studies or endoscopy determine that acute inflammation and edema are the principal causes of the outlet obstruction (as opposed to scarring and fibrosis, which may be fixed).
If medical therapy conducted for a reasonable period fails to alleviate the obstruction, then surgical intervention becomes appropriate. Typically, if resolution or improvement is not seen within 48-72 hours, surgical intervention is necessary. The choice of surgical procedure depends upon the patient's particular circumstances; however, vagotomy and antrectomy should be considered the criterion standard against which the efficacy of other procedures is measured.
Contraindications for surgery relate to the underlying medical condition. Most patients benefit from an initial period of gastric decompression, hydration, and correction of electrolyte imbalances. In patients who are severely malnourished, postponing surgical intervention until the nutritional status has been optimized may be wise. In selective cases, some patients may benefit from total parenteral nutrition (TPN) or distal tube feeding (eg, placed via a percutaneous jejunostomy).

One of the relative contraindications for surgery is the presence of advanced malignancy; in these cases, in which life expectancy may be limited to a few months, palliation via endoscopically placed stents should be considered.

Preparation for surgery

Perform standard preoperative evaluation in these patients. Correct fluid and electrolyte abnormalities prior to surgery. Perform gastric decompression by NG tube and suction and alert the anesthesiologist to the potential risk for aspiration upon induction.
Perform a preoperative nutritional evaluation and initiate appropriate nutritional therapy (TPN or enteral feedings via a percutaneous jejunostomy placed distal to the obstruction) as soon as possible. Maximizing preoperative nutrition can greatly reduce or eliminate postoperative complications related to delayed healing.
Surgical intervention usually provides definitive treatment of GOO, but it may result in its own comorbid consequences. Operative management should offer relief of obstruction and correction of the acid problem.
The most common surgical procedures performed for GOO related to PUD are vagotomy and antrectomy, vagotomy and pyloroplasty, truncal vagotomy and gastrojejunostomy, pyloroplasty.
Vagotomy and antrectomy with Billroth II reconstruction (gastrojejunostomy) seem to offer the best results. Vagotomy and pyloroplasty and pyloroplasty alone, although used with some success, can be technically difficult to perform due to scarring at the gastric outlet. A combination of balloon dilatation and highly selective vagotomy has been described, but it is associated with gastroparesis and a high recurrence rate.
Placement of a jejunostomy tube at the time of surgery should be considered. This provides temporary feeding access in already malnourished patients. Also, in chronically dilated partial obstructions, the stomach may be slow to recover a normal rate of emptying.
The role of the laparoscopic approach in the treatment of GOO is under investigation and may represent a valid form of therapy with low morbidity.
If a gastric reconstruction is performed, NG intubation is recommended. How long the tube should remain in place is controversial; however, it is important to remember that a

previously dilated stomach, the performance of a vagotomy, and the presence of metastatic cancer may all contribute to decreased gastric motility. An anatomically patent gastrojejunostomy may fail to empty for days. This syndrome of delayed gastric emptying is a well-known entity and requires surgical patience. Again, preoperative planning for feeding access becomes important during this immediate postoperative period.

Aggressive pulmonary toilet, prophylaxis for gastritis and deep vein thrombosis (DVT), and early ambulation are advisable.