GASTROENTEROLOGY

Diseases of the Esophagus

Achalasia

- Due to idiopathic dysfunction of distal esophageal neural structures leading to impaired relaxation of distal esophageal sphincter (LES) during swallowing
- 20-40 years old; males=females
- dysphagia for both solids and liquids
- weight loss
- nocturnal cough, recurrent aspiration, bronchitis, pneumonia
- X-ray (not needed) → air-fluid level in the distal esophagus
- Initial test → barium swallow showing dilated esophagus which tapers as a “bird’s beak” at the LES
- Next step → esophagoscopy with biopsies to rule out secondary causes of achalasia (cancer, Chagas’ disease)
- Most accurate test → esophageal manometry showing:
  1. Increased LES pressure
  2. Impaired relaxation of LES with swallowing
  3. Absence of normal peristaltic activity throughout the esophagus
- Initial therapeutic measure → pneumatic dilation
- If ineffective or patient refusal to undergo pneumatic dilation → botulinum toxin injection (everyone needs re-injection after some time)
- If both ineffective then proceed to Heller myotomy (usually done laparoscopically with a risk of post-op reflux development)

Diffuse Esophageal Spasm

- Intermittent chest pain relieved by nitroglycerin thus simulating myocardial infarction but unrelated to exertion or eating (ruling out myocardial infarction and odynophagia, respectively)
- Pain may be precipitated by drinking extremely cold or hot liquids
- Dysphagia, especially for liquids
- If can not differentiate clinically between Esophageal Spasm and MI proceed to EKG
- Initial test → barium swallow showing a “corkscrew” appearance
- Most accurate test → esophageal manometry showing:
  1. high amplitude, repetitive, simultaneous contractions
  2. nutcracker esophagus → high amplitude, prolonged contractions
- management → Ca+ channel blockers (nifedipine, etc.) or nitrates
- If severe and resistant to all forms of therapy proceed to longitudinal esophageal myotomy
Scleroderma

- Esophageal involvement due to atrophy and fibrosis of the esophageal smooth muscle (distal 2/3 of esophagus)
- Other manifestations of scleroderma (distal skin thickening, Raynaud’s phenomenon, visceral involvement)
- Dysphagia for both solids and liquids (not a constant feature in test questions)
- Severe heartburn (strictures develop in long-term survivors and patients report improvement of symptoms of reflux, but increased dysphagia; diagnosis made by endoscopy with biopsies; treated with tapered bougies or balloon dilatation)
- LES neither contracts nor relaxes, hence the presence of both dysphagia and heartburn
- Initial test (also the most accurate) ➔ esophageal manometry showing:
  1. decreased LES pressure
  2. low amplitude, weak peristaltic contractions in the distal 2/3 of the esophagus
- Management ➔ same as GERD (mainly proton-pump inhibitos, such as lansoprazole)

Esophageal Cancer

- Risk factors for squamous cell carcinoma (proximal 2/3 of esophagus) include older age, smoking and alcohol consumption (other risk factors such as dietary factors, lye ingestion, celiac sprue or achalasia are less important for the purpose of USMLE)
- Risk factor for the development of adenocarcinoma is long standing GERD with Barrett’s esophagus (distal 1/3 of esophagus)
- Progressive dysphagia (first for solids, then semi-solids, then liquids and finally one’s own saliva)
- Prominent weight loss
- Shorter duration than that of achalasia
- Other symptoms might include GI bleeding, cough, hoarsness or PTHrP secretion induced hypercalcemia
- Initial test ➔ barium swallow showing narrowing of the esophagus with irregular wall contours
- If you strongly suspect cancer proceed directly to esophagoscopy and biopsies, which is also the most accurate test
- CT scan and bronchoscopy used for assessment of tumor spread
- Management ➔ surgical resection if possible; use 5-fluorouracil-based chemotherapy combined with radiation for locally metastatic disease

Gastroesophageal reflux disease (GERD)

- Usually due to idiopathic dysfunction of the LES
- Secondary causes of decreased LES pressure include:
  1. pregnancy (due to smooth muscle relaxation from increased progesterone concentration)
  2. nicotine
  3. alcohol
  4. caffeine
  5. peppermint
  6. chocolate
  7. drugs (nitrates, ca+ channel blockers, anticholinergics, β-blockers)
• Risk factors → obesity, scleroderma, pregnancy, hiatal hernia
• Heartburn (substernal chest pain) after a heavy meal, when lying down, bending forward or wearing tight-clothing
• Sour-metallic taste in the mouth
• Regurgitation
• Recurrent laryngitis (hoarsness)
• Cough and wheezing (reflux-induced asthma)
• Stricture development (improved heartburn, but appearance of dysphagia for solids)
• Next step in management → if the clinical picture is conclusive, proceed directly to a trial of proton-pump inhibitors (PPIs)
• If diagnosis in question or for evaluation of recurrent laryngitis, chronic cough (especially if nocturnal) or unexplained asthma → perform 24 hour pH monitoring
• Indications for endoscopy include:
  1. GERD present for >5 years
  2. Alarming symptoms (dysphagia, weight loss, anemia, >45 years)
  3. GERD resistant to medical therapy
• Management → life-style changes (elevation of the head of bed, avoiding large and late-night meals, stopping using alcohol, nicotine, caffeine, etc. + PPIs (omeprazole, lansoprazole, rabeprazole, etc.)
• H2 blockers, antacids and metoclopramide are inferior to PPIs
• If resistant to therapy preform Nissen fundoplication (or any other surgical procedure that tightens the LES)
• Link to surgery → if you decide to perform surgery for GERD, perform all the basic diagnostic studies before operating on the patient (barium swallow, esophagoscopy, pH monitoring and manometry)

Barrett’s esophagus

• Columnar metaplasia of the distal esophagus due to long-standing GERD
• Increased risk of adenocarcinoma, requiring endoscopic surveillance
• Management → endoscopy every 2-3 years; PPIs as for GERD
• Low-grade dysplasia on endoscopy and biopsy → repeat endoscopy every 3-6 months
• High-grade dysplasia → distal esophagectomy

Zenker’s diverticulum

• Mucosal herniation above the cricopharyngeal region (false diverticulum)
• Regurgiation of undigested food eaten several days ago
• Halitosis
• Dysphagia (especially on initiation of swallowing)
• The best initial and most accurate tests → barium swallow showing outpouching of the mucosa
• DO NOT perform endoscopy or NG tube placement → risk of perforation
• Management → cricopharyngeal myotomy; diverticulectomy for large lesions
## Esophageal Webs and Rings

<table>
<thead>
<tr>
<th>Esophageal Webs</th>
<th>Esophageal Rings (Schatzki’s rings)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal 1/3 of esophagus</td>
<td>Squamocolumnar junction</td>
</tr>
<tr>
<td>Plummer-Vinson (Paterson-Kelly) syndrome → Webs + Iron-deficiency anemia</td>
<td>No associated syndromes</td>
</tr>
<tr>
<td>(other features of the syndrome not relevant for the purpose of USMLE)</td>
<td></td>
</tr>
<tr>
<td>Occasional progression to cancer</td>
<td>No risk of cancer</td>
</tr>
</tbody>
</table>

- Both present with intermittent dysphagia, that is not progressive in nature
- Both diagnosed with barium esophagram (best initial and most accurate test)
- Management → bougienage to fracture the webs or rings, respectively. Iron supplementation for Plummer-Vinson syndrome
Esophagitis (Infectious)

- Most common cause is Candida albicans (other less common causes include Herpes and Cytomegaloviruses)
- Usually seen in patients with HIV infection and CD4 count <200/mm³
- Other, but less important risk factors include (less important for the purpose of USMLE):
  1. Diabetes
  2. Steroid treatment or chemotherapy
  3. Impaired emptying of the esophagus, as seen in achalasia
- Odynophagia or painful swallowing is the major manifestation
- If the patient is HIV positive → proceed directly to fluconazole trial (if effective it confirms the diagnosis of Candidal esophagitis)
- If fluconazole not effective or patient not HIV infected → perform endoscopy with biopsies to determine the exact etiology (suspect Herpervirus if you see intranuclear inclusions, or Cytomegalovirus if both intranuclear and intracytoplasmic inclusions are detected)
- As a general rule nearly all the more common causes of esophagitis are seen in patients with severe immunosuppression
- Management:
  1. Candida → fluconazole
  2. Herpes → acyclovir
  3. Cytomegalovirus → ganciclovir (or foscarnet)

Esophagitis (Pill-induced)

- Most common causes include:
  1. Oral bisphosphonates (alendronate, pamidronate, -dronates)
1. Iron sulfate
2. Potassium chloride
3. Aspirin and other NSAIDs
4. Tetracyclines
5. Vitamin C

- Diagnosis made by history of drug ingestion
- Management (prevention) ⇒ swallowing pills with plenty of water and remaining upright for a considerable period of time (e.g. at least 30 min for bisphosphonates)

Mallory-Weiss syndrome

- Linear mucosal tear at the squamocolumnar junction seen after vomiting, and less commonly after straining or coughing
- Hematemesis after an initial non-bloody vomitus
- May also present with melena if bleeding is >100ml, but not continued
- Next step in management ⇒ hemodynamic stabilization (IV fluid resuscitation with two large-bore needles, transfusion if low hematocrit, etc.)
- Best initial and most accurate test ⇒ esophageal endoscopy
- Management ⇒ usually resolves spontaneously with supportive treatment given only.
- If continued bleeding ⇒ direct epinephrine injection into the tear or cauterization (performed with the help of endoscopy)

Boerhaave syndrome

- Esophageal wall rupture secondary to forceful vomiting (most common location is the left posterolateral wall of lower 1/3 of esophagus)
- More commonly esophageal perforation follows instrumentation, such as pneumatic dilation or endoscopy (iatrogenic esophageal rupture)
- Most common risk factor is excessive alcohol intake
- Sudden onset of lower thoracic/upper abdominal pain following a period of retching and vomiting (or instrumentation of the esophagus)
- Pain aggravated with swallowing
- Shortness of breath and cough are also common at presentation
- Cracking sound on auscultation coinciding with heartbeat (Hamman’s crunch)
- Pleural effusion (low pH, high amylase content, presence of food particles or gastric juice)
- Pneumomediastinum and subcutaneous emphysema
- Best initial test (if not already done in the test question) ⇒ chest X-ray showing left pleural effusion, pneumomediastinum and subcutaneous emphysema
- Most important diagnostic test (and usually the correct answer on test questions) ⇒ Gastrograffin swallow (water-soluble contrast)
- Most accurate diagnostic test (although rarely needed) ⇒ CT scan
- Management ⇒ fluid resuscitation, broad-spectrum antibiotic administration, NG suction and immediate referral to the surgical unit
- Most important step in management ⇒ early diagnosis (within 24 hours of onset)
Diseases of the stomach

Hiatal Hernia

- Herniation of a part of the stomach through the diaphragm
- Two major types:

<table>
<thead>
<tr>
<th>Sliding hiatal hernia (Type I)</th>
<th>Paraesophageal hernia (Type II)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Both GE junction and portion of stomach displaced above the diaphragm</td>
<td>GE junction remains below the diaphragm with only gastric fundus herniating</td>
</tr>
<tr>
<td>Asymptomatic to GERD</td>
<td>Usually asymptomatic until complicated</td>
</tr>
<tr>
<td>Rx similar to GERD (lifestyle modification, PPIs; possible fundoplication)</td>
<td>Surgical gastropexy to prevent volvulus and strangulation of the stomach</td>
</tr>
</tbody>
</table>

- Both may be detected incidentally on routine chest X-ray
- Best initial test (and the one most likely to be tested for on the exam) → barium swallow

Gastritis (Acute)

- Acute inflammation of the gastric mucosa that may be erosive (acute erosive gastritis) but not ulcerative, hemorrhagic (acute hemorrhagic gastritis) or focal to diffuse
- Commonly implicated etiologic factors include (but are not limited to):
  1. Drugs, such as aspirin and other NSAIDs
  2. Excessive alcohol consumption
  3. Infection (H. pylori, phlegmonous gastritis due to Strep or Staph)
  4. Caustic injury
  5. Severe burns (Curling’s ulcer)
  6. Head trauma (Cushing’s ulcer)
  7. Prolonged mechanical ventilation
- Painless upper GI bleeding (hematemesis or melena) is the most common presentation
- Severe forms of gastritis may also present with nausea, vomiting and epigastric pain
- Most accurate and best initial diagnostic test → Esophagogastroduodenoscopy (EGD), or simply upper endoscopy
- Best initial step in management (of course if we are dealing with hemorrhagic form of gastritis) → Fluid resuscitation + PPIs or H2-receptor blockers and removal of any offending drug (Start treatment before definite diagnosis)
Gastritis (Chronic)

<table>
<thead>
<tr>
<th>Type A</th>
<th>Type B</th>
</tr>
</thead>
<tbody>
<tr>
<td>10% of cases</td>
<td>90% of cases</td>
</tr>
<tr>
<td>Fundus and body of the stomach</td>
<td>Antral region of the stomach</td>
</tr>
<tr>
<td>Autoimmune (parietal cell antibodies)</td>
<td>H.pylori infection; reflux gastritis</td>
</tr>
<tr>
<td>Achlorhydria, increased gastrin levels</td>
<td>Increased acid secretion, decreased gastrin levels</td>
</tr>
<tr>
<td>Pernicious anemia (B12 vitamin deficiency),</td>
<td>Associated with Peptic ulcer disease (H.Pylori infection) or gastric surgery such as Billroth II (reflux gastritis)</td>
</tr>
<tr>
<td>hypothyroidism, diabetes mellitus, vitiligo are associated features</td>
<td>Increased risk of gastric adenocarcinoma and gastric MALToma (a type of lymphoma)</td>
</tr>
<tr>
<td>Increased risk of gastric adenocarcinoma</td>
<td>Lifelong vitamin B12 supplementation</td>
</tr>
<tr>
<td></td>
<td>Eradication of H.Pylori (Clarithromycin + Amoxicillin + PPIs)</td>
</tr>
</tbody>
</table>

- Most accurate diagnostic test for chronic gastritis \(\rightarrow\) EGD (mandatory if patient has epigastric distress unresponsive to empirical PPIs, vomiting, dysphagia, bleeding and anemia or is >45 years old)
- Best initial test to detect H.Pylori infection (Hint: if you have already done EGD than it can also be used to detect H.Pylori infection with no need to perform other tests) \(\rightarrow\) Serology or urea-breath test (serology remains positive for lifetime; urea-breath test changes with treatment)
- Most accurate test for H.Pylori \(\rightarrow\) EGD with biopsy
- Best initial test to detect pernicious anemia \(\rightarrow\) Vitamin B12 level or methylmalonic acid level (more sensitive than vitamin B12 level)
- Confirmatory test for pernicious anemia \(\rightarrow\) Anti-parietal cell antibodies or anti-intrinsic factor antibodies

Menetrier’s disease (low yield for the USMLE)

- Hypertrophic gastritis with massive enlargement of gastric folds
- Diff. diagnosis \(\rightarrow\) Zollinger-Ellison syndrome, amyloidosis, lymphoma and cancer
- Presents with \(\rightarrow\) abdominal pain, weight loss and peripheral edema (protein-losing gastropathy)
- Diagnosis made with EGD and biopsies (also to exclude other causes of gastric hypertrophy)
- Hypochlorhydria differentiates Menetrier’s disease from hypersecretory, hypertrophic form of gastritis which is associated with increased acid secretion
- Management \(\rightarrow\) Anticholinergics (to close intercellular tight junctions), H2-receptor blockers and steroids
- Surgery \(\rightarrow\) only for very severe, resistant cases
Peptic Ulcer Disease

- A group of disorders of the gastrointestinal tract characterized by ulceration, mainly of the stomach or proximal duodenum and formerly (by mistake) thought to be caused by increased acid secretion and enhanced pepsin activity.
- Actually it is caused by imbalance between defensive (blood flow, prostaglandin production, bicarbonate secretion) and offensive (acid, digestive enzymes) factors (hence relative, not absolute increase in acidity and pepsin activity).
- H. pylori is the most common cause of peptic ulcer disease (most gastric and nearly all duodenal ulcers).
- Other etiologic factors include:
  1. NSAIDs (through inhibition of Prostaglandin production → decreased protective mucus secretion)
  2. Zollinger-Ellison syndrome
  3. Crohn’s disease
  4. Gastric cancer
  5. Head trauma, burns and prolonged mechanical ventilation (Stress ulcers)
- Risk factors include (note these are not etiologic factors as they do not cause PUD by themselves):
  1. Smoking
  2. Alcohol consumption
  3. Chronic steroid therapy, as in asthma or rheumatoid arthritis
  4. Male gender
  5. COPD, cirrhosis, hyperparathyroidism and chronic renal disease (not as important for the USMLE)

<table>
<thead>
<tr>
<th>Gastric Ulcer</th>
<th>Duodenal Ulcer</th>
</tr>
</thead>
<tbody>
<tr>
<td>25% of PUD cases</td>
<td>75% of PUD cases</td>
</tr>
<tr>
<td>Median age 50 years at presentation</td>
<td>Median age 40 years at presentation</td>
</tr>
<tr>
<td>Epigastric pain worse with eating; nausea, vomiting and weight loss more common</td>
<td>Epigastric pain improves with meals, only to worsen 2-3 hours after eating; Pain that awakens patient from sleep</td>
</tr>
<tr>
<td>Normal to low acidity; High gastrin levels</td>
<td>High acidity; normal to low gastrin levels</td>
</tr>
<tr>
<td>Most cases due to H. pylori infection, but higher percentage associated with cancer or NSAID use</td>
<td>Nearly always due to H. pylori infection</td>
</tr>
<tr>
<td>Epigastric tenderness not common</td>
<td>Epigastric tenderness not common</td>
</tr>
</tbody>
</table>

- The only way to differentiate between gastric and duodenal ulcers is by direct visualization with the help of EGD or upper gastrointestinal series.
- Best initial test in suspected PUD → Hematocrit or CBC to rule out bleeding (usually already done in most if not all test questions).
- If patient presents only with epigastric pain and is <45 years old → next step in management → empiric treatment with PPIs (without any diagnostic studies).
- If patient >45 years old, or symptoms persist despite PPIs, or if there are associated symptoms of weight loss, GI bleeding with anemia, dysphagia or severe vomiting → best initial and most accurate diagnostic test → EGD with biopsies (can detect both ulcers and H. pylori infection and rule out cancer at the same time).
- No cancer, no H. pylori infection → PPIs (omeprazole, lansoprazole, etc.) or H2-receptor blockers (cimetidine, ranitidine, etc.).
• No cancer, H.Pylori present → Triple therapy with Clarithromycin, Amoxicillin and PPIs; If symptoms persist do an urea breath test to evaluate for possible H.Pylori persistence;
• If H.Pylori still present → change antibiotics to tetracycline (doxycycline), metronidazole and bismuth subsalicylate
• If no H.Pylori → do a serum gastrin level to evaluate for possible Zollinger-Ellison syndrome
• Misoprostol → To prevent PUD in patients with chronic NSAID usage (e.g. rheumatoid arthritis) *COX-2 inhibitors, such as celecoxib (-coxibs) have a lower chance of inducing PUD and also have no effect on platelet function
• Refractory cases require surgery → parietal cell vagotomy being the preferred procedure (most selective with least complications) or distal antrectomy with resection of any gastric ulcer if present (classic scenario for surgery as the answer would be a gastric ulcer not responsive to therapy in which cancer has been ruled out with EGD and biopsies)
• Classic surgical procedures such as Billroth I or Billroth II are rarely if ever performed nowadays

Complications of PUD

<table>
<thead>
<tr>
<th>Hemorrhage</th>
<th>Perforation</th>
<th>Outlet obstruction</th>
<th>Penetration</th>
</tr>
</thead>
<tbody>
<tr>
<td>20% of PUD cases</td>
<td>5-10% of PUD cases</td>
<td>5-10% of PUD cases</td>
<td>Usually caused by a posterior duodenal ulcer</td>
</tr>
<tr>
<td>“coffee-ground” emesis; melena; hematemesis, decreased hematocrit or hypovolemic shock may all be presentations of this complication</td>
<td>Signs of peritoneal irritation (rigid abdomen, intense pain, rebound tenderness, decreased bowel sounds)</td>
<td>Early satiety, epigastric fullness, nausea vomiting and possible weight loss; succussion splash on abdominal auscultation</td>
<td>Most common organ of penetration is the pancreas</td>
</tr>
<tr>
<td>Manage the same way as any other GI bleed (volume resuscitation, type and cross-match, blood transfusion as needed) + IV PPIs</td>
<td>More common with duodenal (especially anterior) than gastric ulcers</td>
<td>More common with duodenal than gastric ulcers</td>
<td>Sudden onset of epigastric pain that radiates straight to the back</td>
</tr>
<tr>
<td>Reserve surgery for refractory bleeding</td>
<td>10% may simultaneously bleed</td>
<td>Diagnosis suggested by history and confirmed by aspiration of &gt;300 ml of gastric contents &gt;3 hours after a meal</td>
<td>Elevated serum amylase and lipase levels</td>
</tr>
<tr>
<td>Most accurate test → EGD (but perform after patient stabilization)</td>
<td>Best initial test and next step in management → X-ray (chest or abdomen) looking for free air under the diaphragm</td>
<td>NG suction and replacement of fluid and electrolytes as needed</td>
<td>Arrange for surgical intervention</td>
</tr>
<tr>
<td>Usually results from posterior duodenal ulcer eroding into the gastroduodenal artery</td>
<td>Management → arrange for immediate exploratory laparotomy</td>
<td>Need to perform an EGD to rule out obstructing cancer</td>
<td></td>
</tr>
</tbody>
</table>
Complications of PUD surgery

<table>
<thead>
<tr>
<th>Dumping syndrome</th>
<th>Alkaline gastritis</th>
<th>Afferent loop syndrome</th>
<th>Anemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early dumping syndrome → dizziness, flushing, sweating and palpitations about 30 min after a meal</td>
<td>Asymptomatic to clinical features resembling other forms of gastritis or PUD (epigastric pain, nausea, vomiting, weight loss)</td>
<td>Bloating and/or vomiting approximately 1 hour after a meal</td>
<td>Factors that may lead to iron-deficiency anemia include: chronic blood loss from alkaline gastritis, decreased conversion of Fe2+ to Fe3+ due to increased gastric pH, and diversion of iron from duodenum which is the preferred site of absorption</td>
</tr>
<tr>
<td>Late dumping syndrome → dizziness, weakness, sleepiness, palpitations, diaphoresis</td>
<td>Due to reflux of duodenal contents into the stomach</td>
<td>May become complicated with bacterial overgrowth leading to malabsorption</td>
<td>Patients with extensive gastric resection may also develop vitamin B12 deficiency anemia due to lack of intrinsic factor production (and this is what most test questions ask for)</td>
</tr>
<tr>
<td>Early → due to rapid osmotic shift of fluid in the duodenum, leading to decreased circulating volume (caused by rapid entry of chyme into the duodenum)</td>
<td>Diagnosis confirmed with HIDA scan showing reflux HIDA-labeled bile into the stomach</td>
<td>Diagnosis confirmed with the help of HIDA scan showing failure of radiolabeled material to enter the GI tract after visualization of the afferent loop (which is duodenum)</td>
<td>Diagnosis → CBC, blood smear, reticulocyte count; Iron studies; Vitamin B12 or methylmalonic acid level</td>
</tr>
<tr>
<td>Late → due to reactive hypoglycemia (rapid glucose absorption leads to excessive insulin production culminating in hypoglycemia several hours after a meal)</td>
<td>Mild cases may be managed conservatively with sucralfate or bile acid binding resins, such as cholestyramine</td>
<td>Mild cases require no specific therapy, but severe disease needs surgical revision of the afferent loop</td>
<td>Management → supplementation of the deficient nutrient</td>
</tr>
</tbody>
</table>

Management → frequent, small meals (usually self-limited over time) | Severe cases require surgical correction (Roux-en-Y anastomosis) |

Note an increased risk of gastric cancer in patients with previous Billroth II gastric resection
Zollinger-Ellison syndrome

- Gastrin producing tumor, found in descending order of frequency within the duodenum, pancreas and stomach, the so called “gastrinoma triangle”. (Bet for a pancreatic rather than intestinal location on your exam)
- 10-20% of cases are associated with MEN-1 (3Ps → pituitary adenomas, hyperparathyroidism, pancreatic islet cell tumors)
- Suspect MEN-1 if the patient has hypercalcemia in the test question or presents with signs and symptoms of hyperprolactinemia in addition to those of Zollinger-Ellison syndrome
- Suspect that you are dealing with Zollinger-Ellison syndrome if:
  1. Patient has multiple ulcers
  2. Patient has large (>1cm) ulcers
  3. Patient has ulcers located in the esophagus, distal duodenum, jejunum, etc.
  4. Patient has ulcers resistant to therapy (nonresponsive and/or recurrent)
  5. Patient has diarrhea/steatorrhea in addition to pain from PUD
- Diarrhea/Statorrhea is caused by:
  1. Inactivation of pancreatic lipase and precipitation of bile acids by increased acidity of chyme passed into the duodenum (gastrin stimulates acid production)
  2. Increased volume of gastric secretions
  3. Increased intestinal motility and incomplete absorption of sodium and water due to high levels of gastrin
- Gastrinomas are usually malignant neoplasms with 50% developing metastasis disease
- Best initial test → serum gastrin levels (stop all antisecretory medications for several days before measuring gastrin)
- If serum gastrin is within normal limits and you still suspect Zollinger-Ellison syndrome → perform a secretin stimulation test showing increase (rather than decrease) in gastrin levels after injecting secretin
- Rarely performed (and nearly never tested on the exam) tests:
  1. Calcium stimulation test (showing marked increase in serum gastrin)
  2. Basal-to-stimulated acid output ratio >0.6 (which means increased basal rate of secretion with only minimal increase after a meal)
- Next best step in management (after diagnosis confirmed) → localization of tumor mass with the help of CT scan, MRI or ultrasound
- Most sensitive test to detect gastrinoma → endoscopic ultrasound
- Other tests that may aid in localization → angiography and somatostatin-receptor scintigraphy
- Management of localized disease → surgical resection of the tumor
- Management of disseminated disease → high-dose PPIs to decrease acid output (H2-receptor blockers not as effective); May also need to perform total gastrectomy
### Gastric Malignancies

<table>
<thead>
<tr>
<th>Gastric Adenocarcinoma</th>
<th>Gastric lymphoma</th>
<th>Gastric stromal tumors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Two morphologic variants: Intestinal (arising from intestinal metaplasia of gastric mucosa and associated with the risk factors mentioned below) and Diffuse (no known risk factors)</td>
<td>Most common location for extranodal non-Hodgkin lymphomas (NHL) is the stomach. Hodgkin’s lymphoma of the stomach is very uncommon</td>
<td>Stomach is the most common location for GI stromal tumors, with small intestine being second in order. Tumor is derived from interstitial cells of Cajal, which function as pacemaker cells</td>
</tr>
<tr>
<td>Risk factors include: diet high in nitrates and salted and preserved foods and low in antioxidant vitamins and minerals; smoking; older age; male gender; genetic factors (e.g. blood group A); H.Pylori infection; Japanese ancestry; gastric adenomas; atrophic gastritis; post-Billroth II procedure</td>
<td>Two of the most common types of gastric NHL that merit consideration are MALTomas (associated with H.Pylori) and diffuse histiocytic non-Hodgkin lymphomas (never assume a MALToma in the test question on gastric lymphoma until it specifically tells you that you are dealing with MALTOMA)</td>
<td>Patients may present with GI bleeding, abdominal pain or abdominal mass detected on physical exam. May also have early satiety, epigastric fullness, vomiting or weight loss.</td>
</tr>
<tr>
<td>Macroscopically there are three major variants: ulcerative, fungating and superficial spreading (involving mucosa and submucosa only) * linitis plastica is an advanced form with all layers involved thick, non-elastic wall</td>
<td>Clinical presentation is similar to other types of gastric malignancy with early satiety, nausea, vomiting, epigastric fullness, etc.</td>
<td>Tumors are c-kit positive, a protein with considerable tyrosine kinase activity (used both diagnostically and therapeutically)</td>
</tr>
<tr>
<td>Asymptomatic (early stages) to dull, epigastric pain; epigastric fullness; early satiety; weight loss; anorexia, nausea, vomiting; GI bleeding (melena, anemia, etc.) enlarged left supraclavicular lymph nodes (Virchow’s node), acanthosis nigricans or severe eruption of seborrheic keratoses known as sign of Leser-Trelat</td>
<td>Diagnosis rests on EGD with biopsies and special immunohistochemical staining for surface markers specific for each type of NHL (Very important to know for the USMLE step 1 but for the purpose of USMLE Step 2 CK you only need to differentiate between NHL and Hodgkin’s disease and not between various NHLs)</td>
<td>Benign versus malignant nature is determined by the number of mitotic figures (&gt;5 per 10 high power fields) and/or size (&gt;4cm)</td>
</tr>
<tr>
<td>Initial test → upper GI series (barium studies) showing an ulcer, mass or “leather bottle” nondistensible stomach</td>
<td>Next step (after tissue diagnosis) → Imaging studies (e.g. CT scan) to evaluate for dissemination outside the stomach</td>
<td>Diagnosis made with EGD with biopsies and by special immunohistochemical staining for c-kit (CD 117)</td>
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<tr>
<td>Most accurate test → EGD with biopsies (used to screen high-risk individuals in Japan, but not in USA)</td>
<td>Nearly all the test questions on gastric lymphomas will ask about management (with all the diagnostic studies already performed)</td>
<td>Management → Imatinib mesylate (Gleevec), a tyrosine kinase inhibitor, most commonly used for Chronic Myelogenous Leukemia</td>
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</tbody>
</table>
Gastroparesis

- Disorder of gastric emptying caused by “weakness” of stomach and not related to obstruction
- Usually due to diabetes mellitus
- Other causes to consider include (but are not limited to):
  1. electrolyte abnormalities (potassium, calcium, magnesium)
  2. systemic sclerosis
  3. postvagotomy state
- Early satiety, epigastric fullness, bloating and postprandial nausea/vomiting are the usual presenting symptoms
- Patients with diabetes also have evidence of diabetic neuropathy (“stocking-and-glove” peripheral neuropathy, orthostatic hypotension, impotence, etc.), nephropathy or retinopathy
- Best initial test  EGD to rule out obstruction
- Most accurate test (rarely needed in the face of a patient with long-standing diabetes)  nuclear solid-phase gastric emptying study
- Management  Erythromycin (increases motilin levels leading to enhanced Migratory Motor Complex activity which is lost in diabetic gastroparesis) or metoclopramide (prokinetic agent)
Diseases of the Small Intestine, Colon and Anorectal Region

Intestinal Obstruction

- Failure of passage of intestinal contents due to mechanical obstruction
- Common causes of small bowel obstruction (SBO) include:
  1. Adhesions from prior abdominal/pelvic surgery (most common cause in the USA)
  2. Incarcerated hernias (most common cause worldwide, in those with no history of surgery and children)
  3. Neoplasms
  4. Gallstone ileus (complication of acute cholecystitis with fistula formation between the gallbladder and duodenum; stone lodges at the ileocecal valve causing SBO; CT scan shows pneumobilia; dilated loops of small bowel and stone at the ileocecal valve; Suspect it if there is SBO with air in the biliary system on routine abdominal X-ray)
  5. Strictures (either congenital or acquired), e.g. Crohn’s disease
  6. Intussusception
  7. Volvulus
- Common causes of large bowel obstruction (LBO) include:
  1. Colon cancer (until proven otherwise)
  2. Volvulus (sigmoid; cecal)
  3. Diverticulitis
  4. Fecal impaction

<table>
<thead>
<tr>
<th>SBO</th>
<th>LBO</th>
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<tbody>
<tr>
<td>Cramping, crescendo-decrescendo pain; Early vomiting (nonfeculent, but may become feculent with distal SBO); abdominal distention (more severe with distal obstruction); Failure to pass flatus or feces (complete obstruction) or ability to pass flatus but not feces (partial obstruction)</td>
<td>Deep, crampy abdominal pain; Massive abdominal distention; Nausea with no vomiting; (or late feculent vomiting); No passage of flatus or feces;</td>
</tr>
<tr>
<td>High-pitched bowel sounds with rushes and tinkles; visible peristaltic waves;</td>
<td>High-pitched bowel sounds; abdominal tenderness; possible palpable abdominal mass;</td>
</tr>
<tr>
<td>Fever, hypotension, tachycardia, increased WBC count and signs of peritonitis (absent bowel sounds, rebound tenderness, guarding, etc.)  (\rightarrow) stanglulated bowel (emergency surgery needed)</td>
<td>As is SBO, Fever, signs of dehydration and/or peritonitis signal bowel strangulation with the need to perform immediate explotatory laparotomy</td>
</tr>
</tbody>
</table>

- Best initial test  \(\rightarrow\) abdominal X-ray (standing and supine) showing:
  1. “ladder-like” distention of small bowel loops with air-fluid levels and absence of colonic gas  \(\rightarrow\) SBO
  2. Distention of large bowel proximal to obstruction (LBO)
- Other studies to consider for the diagnosis of intestinal obstruction include:
  1. barium enema (especially for LBO)
  2. CT scan
  3. Sigmoidoscopy or colonoscopy (only in hemodynamically stable patients)
• Management of SBO → Hospitalization; NPO + NG suction + fluid and electrolyte replacement + monitoring of hemodynamic status
• Indications for surgery in the management of SBO include:
  1. Complete SBO (even though complete SBO requires surgery, best initial step in management would still be NG suction, NPO and IV hydration)
  2. Signs of strangulation (peritonitis, fever, leukocytosis, signs of dehydration, increased lactic acid)
  3. Obstruction lasting >3 days
• Management of LBO → Hospitalization; NPO + IV hydration; Usually requires surgical intervention, but first try colonoscopic decompression or rectal tube placement as it might help some patients with LBO
• Management of Strangulated Bowel → Immediate exploratory laparotomy with resection of all necrotic bowel with a second-look operation after 18-36 hours to assess bowel viability

Paralytic ileus
• Failure of passage of intestinal contents due to absence of intestinal peristalsis and unrelated to obstruction
• Risk factors for Ileus include:
  1. Recent abdominal/pelvic surgery (should last <5 days)
  2. Electrolyte imbalances (e.g. hypokalemia)
  3. Diabetes mellitus
  4. Drugs that decrease GI motility (e.g. opioids, anticholinergics)
  5. Hypothyroidism
  6. Any severe intraabdominal inflammatory condition
• Abdominal distention; bloating, nausea and vomiting; failure to pass flatus or gas
• No bowel sounds, no visible peristaltic waves
• No signs of peritonitis (in that case we would call it acute peritonitis and not paralytic ileus)
• No fecal impaction present on rectal exam (usually this is what you do first in an elderly patient with failure to pass intestinal contents)
• X-ray → Distended loops of both small and large bowel with air in the rectum
• Management → discontinue any contributing medications (e.g. opioids); make patient NPO, perform NG suction; correct any electrolyte abnormalities present; wait for spontaneous resolution
• If there is no resolution consider colonoscopic decompression

Sigmoid Volvulus
• Rotation of bowel on its mesentery leading to obstruction and possible strangulation
• Consider sigmoid volvulus in the elderly who live in nursing homes, have some form of CNS disease (e.g. Alzheimer’s disease) or have chronic constipation
• Clinical presentation similar to LBO with abdominal distention, crampy pain, obstipation and feculent vomiting
• Physical examination may show an abdominal mass
• Best initial test → abdominal X-ray showing a large air-filled loop of bowel in the RUQ tapering into the LLQ (inverted “U” shape)
• If diagnosis doubtful after plain X-ray perform a barium enema showing a “bird’s beak” appearance
• To evaluate for ischemic changes perform a CT scan
• Management → colonoscopic/sigmoidoscopic decompression with rectal tube placement afterwards; If no resolution proceed to surgery (e.g. laparoscopic derotation);
• Consider elective sigmoidopexy (endoscopic or surgical) or resection to prevent recurrence
• A few words about cecal volvulus → Signs of SBO or LBO + X-ray showing a bowel loop in the LUQ pointing to the RLQ; Surgical resection is the correct answer on test questions about management

Intestinal Pseudo-obstruction (Ogilvie syndrome)

• Recurrent episodes of large bowel obstruction with no demonstrable source of obstruction, thought to be due to interruption of sacral parasympathetic nerves leading to adynamic distal colon (similar to Hirschprung’s disease but with normal ganglion cells observable on autopsy)
• Most severely affected segment is the cecum (according to LaPlace law, surface tension equals transmural pressure multiplied by radius; as cecum has the largest diameter in the colon, equal transmural pressures lead to increased surface tension in the cecum relative to other parts of the large bowel, increasing the risk of cecal perforation)
• Suspect Ogilvie syndrome if a test question describes an elderly patient, usually living in a nursing home, who possibly has some form of chronic disease of the CNS, Cardiovascular or Pulmonary systems and who undergoes surgery for unrelated reasons (e.g. prostatectomy, hip replacement, etc.) → This is followed by development of clinical symptoms and signs of large bowel obstruction (distention, abdominal pain, obstipation, etc.) and plain abdominal X-rays show dilated loops of large intestine, most severely affecting the cecum
• One should always exclude mechanical causes of large bowel obstruction with the help of CT scan and/or colonoscopy (both diagnostic and therapeutic)
• You are most likely to encounter a test question asking you to recognize Ogilvie syndrome or intestinal pseudo-obstruction as the cause of the patient’s complaints, but if they ask you how to treat the patient, answer Colonoscopic decompression with/without placement of a rectal tube (of course preceded by NPO status, NG suction, correction of fluid and electrolyte abnormalities and discontinuation of any contributing medications, as you would do with any form of intestinal obstruction)
• Risk of perforation is increased if colon diameter is >10cm, or >4 days have passed since the onset
• If cecum is about to blow, perform immediate tube cecostomy
• If perforation has already developed, perform subtotal colectomy with temporary ileostomy
• Some authors recommend using cholinergic agents such as neostigmine (but mechanical obstruction has to be ruled out with contrast enema and air must be present throughout the colon and rectum to start medical management)