GI Emergencies

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GI EMERGENCIES

I. ABDOMINAL PAIN

A. General

1. One of the most common causes for ED visits; 8-11% of test
   a. < 50 yo, Most common = nonspecific abdominal pain
   b. > 50 yo, Most common = biliary disease
2. Presentations extremely variable, KNOW THE CLASSICS!
3. Always consider extra-abdominal causes
4. High index of suspicion for children, elderly, and
   immunocompromised patients

II. DISORDERS OF THE GALLBLADDER / BILIARY TREE

B. Cholelithiasis (Gallstones)

C. Etiology (Gallstones)

1. 20% females, 8% males have asymptomatic gallstones
2. Risk factors
   a. Female, as high as 4:1
   b. Age
   c. Family history
   d. High estrogen state: female, fertile, forty, fat,
      contraceptives, clofibrate
   e. IBD: Crohn’s, ulcerative colitis
   f. Cystic fibrosis
   g. Hemoglobinopathies
   h. Sickle cell disease, thalassemia
   i. Rapid weight loss/starvation
   j. Elevated lipids/triglycerides
   k. TPN
   l. Post-op
3. Types of stones
   a. Cholesterol (pure or mixed)
      i. Most common! (> 75%)
      ii. Radiolucent (not visible) on X-ray
      iii. Due to delayed GB emptying and bile stasis
   b. Pigmented (black or brown)
      i. Unconjugated bilirubin + calcium salts
      ii. Radiopaque (due to calcium)
      iii. Black stones
         • Advanced liver disease
         • Hemolytic disorders
         • Found exclusively in the GB
iv. Brown stones
   - Associated with infection
   - Found throughout biliary system

4. History
   a. RUQ/epigastric pain – constant or intermittent
   b. Radiation to scapula or shoulder
   c. N/V
   d. Similar episodes in past

5. Physical exam
   a. RUQ/epigastric tenderness

6. Diagnosis
   a. Labs often within normal
   b. X-ray – only 10% of GB stones visualized
   c. US – test of choice
      i. Sensitivity for stones in GB nearly 100%

7. Treatment
   a. For asymptomatic patients
      i. No acute treatment needed
      ii. 15-20% becomes symptomatic
      iii. Outpatient elective surgery if:
          - Frequent or severe attacks
          - Diabetic
          - Large biliary calculi
   b. For symptomatic patients
      i. Pain control
      ii. Anti-emetics
      iii. Outpatient elective surgery
      iv. 90% have recurrent symptoms within 2 years
      v. 50% will develop acute cholecystitis

D. Acute cholecystitis

1. Etiology
   a. Sudden inflammation of GB
   b. Bacterial infection in 50-75%
   c. E. Coli, Klebsiella, Enterococci predominant

2. History
   a. RUQ or epigastric pain; radiation to tip of (scapula)
   b. Pain becomes constant!
   c. Nausea / vomiting
   d. +/- Fever

3. Physical exam
   a. RUQ tenderness
   b. Murphy’s sign (stops inspiratory breath upon deep palpation)
      i. Much lower sensitivity in elderly

4. Diagnosis
   a. CBC
b. Often elevated WBC with left shift
c. Alk Phos, Bilirubin, AST/ALT
   i. Mildly elevated or normal
   ii. Large elevations of Alk Phos, Bilirubin suggests stone in common bile duct (CBD)
d. KUB
   i. Poor sensitivity, stones seen ~ 10%
   ii. Air in biliary tree … rare, but serious! (Gangrenous infx)
e. Ultrasound: modality of choice
   i. Gallstones
   ii. Thickened GB wall
   iii. Pericholecystic fluid
   iv. Sensitivity 90-95%; specificity 75-85%
      • US can miss stones in cystic duct
f. CT scan
   i. Poor sensitivity for cholecystitis (nears 50%)
   ii. Can diagnose emphysematous cholecystitis
   iii. Excellent for diagnosing other intraabdominal disorders
g. HIDA scan – gold standard for this disease
   i. Positive = no visualization of GB
   ii. Identifies stones in cystic duct
   iii. Very sensitive; neg scan rules out diagnosis
5. Treatment
   a. Admit, NPO, IVFL
   b. Pain, emesis control (NGT if multiple emesis)
c. Antibiotics recommended
   i. Bugs = gram negatives, anaerobes and Enterococcus
   ii. Coverage with single broad-spectrum antibiotic (2-3rd generation Cephalosporin) adequate if not septic
   iii. Triple coverage if septic
d. Surgical consultation
   i. Surgery typically 24-72 hr after symptoms resolve
   ii. Immediate surgery if toxic/deterioration
6. Complications
   a. Perforation (3-5%)
   b. Emphysematous cholecystitis (1%)
      i. Gangrene due to gas forming bacteria
         • E. Coli, Clostridium perfringens, Klebsiella
      ii. Increased risk in diabetics, elderly
      iii. High mortality
      iv. Acalculous in 30% cases
c. Gallstone ileus
d. Pancreatitis (15-20%)
e. Ascending cholangitis
f. GB empyema
   i. Complete obstruction of cystic duct with bacteria
   ii. Abscess formation within GB wall
E. Acute acalculous cholecystitis

1. No stones present in GB
2. 5-10% of acute cholecystitis
3. **Rapid, malignant course, up to 40% mortality**
4. Bile stasis equivalent to common duct obstruction
5. Risk factors
   a. Elderly
   b. Post-surgery
   c. **Diabetics**
   d. Trauma, burns
   e. Sepsis
   f. AIDS (due to CMV or Cryptosporidium Infx)
   g. Systemic vasculitic states
6. Presentation
   a. Similar to acute calculous cholecystitis EXCEPT:
      i. Gravely ill on presentation
      ii. Frequently a complication of another process
7. Diagnosis
   a. **HIDA scan**
      i. Poor filling of gallbladder without gallstones
   b. **Ultrasound**
      i. No gallstone
      ii. Thickened GB wall, pericholecystic fluid
8. Treatment
   a. NPO, IVFL
   b. **Emergent surgical consultation** for cholecystectomy vs drain if unstable
   c. **Broad spectrum antibiotics**

F. Ascending cholangitis

1. Obstructed bile flow causes retrograde infection up biliary tree (gram negatives, enterococci, anaerobic)
2. Causes/risk factors
   a. **Choledocholithiasis (CBD stone): #1 cause**
   b. Diabetes
   c. Malignant strictures
   d. Sclerosing cholangitis
3. Presentation
   a. **Charcot’s Triad**
      i. RUQ pain
      ii. Fever/chills
      iii. Jaundice
   b. Reynolds’s Pentad = Charcot’s Triad plus…
      i. Sepsis
      ii. Mental status change
4. Diagnosis
   a. Labs
      i. Elevated WBC
      ii. Hyperbilirubinemia
      iii. Elevated alkaline phosphatase
   b. ERCP
   c. Transhepatic cholangiography
   d. Ultrasound
      i. Shows common and intrahepatic ductal dilatation
      ii. Often non-diagnostic

5. Treatment
   a. NPO, IVFL
   b. Broad spectrum antibiotics
   c. ERCP
      i. Removal of stones, tract decompression
   d. Emergency surgical consultation
   e. Mortality nears 100% if untreated

III. HEPATITIS

A. General
   1. Definition: general inflammation of liver
   2. Etiology: viral (Hep A, B, C or D, EBV), bacterial, alcoholic, immune, medications

B. Hepatitis A virus
   1. RNA virus
   2. Fecal-oral transmission
   3. 33% of US with immunity secondary to exposure
   4. Greatest infectivity before onset of symptoms of disease
   5. Usually self-limited
   6. Acute disease, no chronic carrier state

C. Hepatitis B
   1. DNA virus (3 major antigens)
      a. HepBsAg – surface antigen
      b. HepCcAg – core antigen
      c. HepBeAg – e antigen
   2. Effective vaccination has decreased prevalence
   3. Spread through parenteral/intimate exposure
   4. Highest among IVDA and homosexual males
   5. Onset of symptoms 60-90 days after exposure
   6. 10% infected adults (90% neonates) develop chronic hepatitis or carrier stage
D. Hepatitis C virus

1. RNA virus
2. Previously called non-A non-B
3. **Most common of blood borne infections in US**
   a. Occupational exposure (i.e. needle sticks)
   b. IVDA
   c. Transfusions
      i. Prior to screening, risk was 0.45% / unit
      ii. With screening, risk now 0.03% / unit
4. **Chronic HCV infection occurs in 85% of infected patients**
   a. 50% develop chronic hepatitis
   b. Cirrhosis develops in 20% of this group within 10 years

E. Hepatitis D virus

1. Defective RNA virus
2. Only **co-infects patients with chronic or current Hep B**
3. High incidence in IVDA, homosexual males
4. Increased risk of fulminant disease

F. History (Hepatitis in general)

1. Variable, many cases asymptomatic
2. Malaise, low-grade fever, anorexia (most common complaints)
3. Other complaints: N/V, abdominal pain, diarrhea
4. Jaundice most often reason for 1st physician visit
5. If fulminant hepatitis:
   a. Acute onset (days) of liver failure and encephalopathy
   b. Seen mostly in Hep B + Hep D
   c. **Hallmarks = altered mental status, mucosal bleeding**

G. Physical exam

1. Low-grade temperature
2. **Icterus (yellowing of sclera when bili >2.5 mg/dl)**
3. RUQ abdominal tenderness
4. Often tachycardic when vomiting is present
5. Hepatomegaly

H. Diagnosis

1. ALT > AST (Both 10 – 100 x normal!)
   a. AST > ALT seen in **alcoholic** hepatitis!
2. Bilirubin elevated (mild/severe)
3. Protime (PT)
   a. **Elevated PT is a sign of significant hepatic disfunction** and the first sign of a complicated course
4. Hepatitis A
   a. **Acute infection = presence of HAV-Ab (IgM antibody)**

5. Hepatitis B
   a. **Acute HBV infection = presence of HBsAg + HBeAb (IgM)**
   b. **High infectivity = presence of HBeAg**
   c. **Past infection or immunization = presence of HBsAb**

I. **Treatment**
   1. Treatment of viral hepatitis is primarily symptomatic
   2. Correct fluid / electrolyte imbalances
   3. Stop nonessential medications with potential hepatotoxicity
   4. Abstinence from alcohol
   5. Admit if altered sensorium or PT 5 seconds above normal

J. **Complications**
   1. Fluid and electrolyte imbalances (acute disease)
   2. GI bleed from multiple emesis
   3. Liver failure
   4. Hepatic encephalopathy
   5. Cirrhosis
   6. Death

K. **Prophylaxis**
   1. **Hepatitis A – provide to household or daycare contacts**
      a. **HAV immunoglobulin**
      b. Minimal risk of contacting to healthcare workers if universal precautions are followed (handwashing/hygiene)
   2. **Hepatitis B – provide to percutaneous or sexual contacts if previously unvaccinated**
      a. **HBIG + start HB vaccine**

IV. SPONTANEOUS BACTERIAL PERITONITIS

A. **Definition:** Acute bacterial infection of cirrhotic ascitic fluid

B. **Pathophysiology**
   1. Migration of enteric organisms through edematous bowel
      a. **E. coli (47 – 55% of cases)**
      b. Streptococcus species (18 – 26%)
      c. Klebsiella species (11%)
      d. Streptococcus pneumoniae (8 – 20%)
      e. **Anaerobes NOT common (<1%)**
C. History

1. Variable presentations
2. Abdominal pain (insidious or acute, mild or severe)
3. Fever/chills
4. Possibly hepatic encephalopathy and hemodynamic instability possible if septic

D. Physical exam

1. Variable
   a. Ranges from mild abdominal tenderness to rigidity / guarding with rebound

E. Diagnosis

1. Paracentesis
   a. WBCs > 500 cells/mm³
   b. Treat if neutrophil count > 250 cells/mm³
      i. Even if paracentesis culture negative!
   c. If coagulopathic (high PT) give FFP prior to procedure

F. Treatment

1. IV antibiotics
   a. Ampicillin plus aminoglycoside OR
   b. 3rd generation cephalosporin

V. PANCREATITIS

A. Pathophysiology

1. Release of activated digestive enzymes
2. Autodigestion of pancreatic / parapancreatic tissues
3. Edema and inflammatory cell infiltration

B. Etiology in the US

1. Alcohol abuse and/or biliary tract disease
   a. Together account for 80-90% of cases in U.S.
2. Idiopathic
3. Infectious
   a. Viral
      i. Mumps; coxsackie B (most common viruses)
      ii. EBV/ CMV
      iii. Hep A, B, C
      iv. HIV
   b. Bacterial
i. Mycoplasma  
ii. Legionella  
iii. M. avium complex

4. Trauma  
5. S/p ERCP  
6. Pregnancy  
7. Scorpion stings  
8. Hyperlipidemia (>500)  
9. Hypercalcemia (often from hyperparathyroidism)  
10. Drugs  
   a. Lasix  
   b. Tetracycline  
   c. Thiazides  
   d. HIV medications

C. History

1. Abdominal pain (classically “a boring pain”)  
   a. Epigastric/LUQ  
   b. Constant (colicky pain suggests alternate diagnosis)  
   c. Radiation to midback  
   d. May find pain improved with sitting forward  
2. Nausea / vomiting

D. Physical exam

1. Vital signs  
   a. Tachycardia common  
   b. Shock/hypotension in serious disease  
2. Epigastric/LUQ tenderness  
   a. Guarding common  
   b. Rebound tenderness uncommon because the pancreas is a retroperitoneal organ  
   c. Pain often out of proportion to exam  
3. Palpable mass (phlegmon, pseudocyst)  
4. Jaundice possible  
5. Grey-Turner sign  
   a. Ecchymosis at flank  
   b. Indicates severe hemorrhagic pancreatitis  
6. Cullen sign  
   a. Ecchymosis at umbilicus  
   b. Indicates severe hemorrhagic pancreatitis

E. Diagnosis

1. Amylase  
   a. Elevated in majority of acute pancreatitis  
   b. May be normal in chronic pancreatitis
c. Sensitivity 79 – 95%

**d. Poor specificity; differential includes:**
   i. Pancreatic/lung cancer
   ii. Bowel obstruction/perforation/infarction
   iii. Renal failure
   iv. Parotid disease
   v. Hepatitis
   vi. Ruptured ectopic pregnancy

**e. Specificity increased if 5x normal level used**

**f. Elevates within 6-24 hr, peak 48 hr, normal 5-7 days**

2. Lipase
   a. Found predominantly in pancreas
   b. **More specific than Amylase (> 90%)**
   c. Sensitivity equal to Amylase
   d. **Remains elevated 8 – 14 days**

3. Very high Amylase/Lipase suggests gallstone pancreatitis
   a. Amylase/Lipase levels do **not** correlate with prognosis/severity

F. **Ranson’s Criteria** – predicts outcome in acute alcoholic pancreatitis

1. **Criteria on admission**
   a. Age over 55 yo
   b. Blood glucose > 200mg/dl
   c. WBC > 16,000
   d. SGOT (AST) > 250
   e. LDH > 350

2. Criteria after 48 hours
   a. HCT decreases >10%
   b. BUN rises > 5 mg/dl
   c. Calcium < 8 mg/dl
   d. pO₂ < 60 mm Hg
   e. Fluid sequestration > 6L
   f. Base deficit > 4 mEq/L

3. Mortality criteria
   a. 0 – 2 criteria = 1% mortality
   b. 3 – 4 criteria = 15% mortality
   c. 5 – 6 criteria = 40% mortality
   d. 7 -8 criteria = 100% mortality

***Ranson’s criteria was developed at a time when we did not have the aggressive resuscitation measures that we do today. Mortality scores, therefore, are not as directly applicable to today’s practices. There are more recent scoring systems but their use in the ED is not very applicable.***

G. Imaging tests

1. Abdomen plain films: little utility
a. **Pancreatic calcifications = chronic disease**  
b. “Sentinel loop” seen if local ileus  
c. Left pleural effusion/atelectasis possible

2. Ultrasound  
   a. **Pancreas poorly imaged**  
      i. Retroperitoneal organ  
      ii. Seen in only 60% scans  
   b. Test of choice to evaluate if gallstone etiology  
   c. Good for pseudocyst, phlegmon, abscess  
   d. Overall inferior to CT for acute pancreatitis

3. **CT scan: contrast enhanced very useful**  
   a. Can diagnose enlarged pancreas, necrosis/hemorrhage, pseudocyst or abscess formation  
   b. Rule out other diseases in abdomen

H. Treatment

1. IV hydration (fluids, fluids, fluids!)  
   a. Consider CVP line if serious disease  
   b. Electrolyte replacement as needed  
2. NPO, pain control  
3. Antiemetics; NGT if persistent emesis  
4. Antibiotics if gallstone etiology, septic, or severe disease  
5. Surgical consultation for:  
   a. Gallstone etiology  
   b. Severe disease  
   c. Abscess/Hemorrhage/Pseudocyst  
6. ERCP if common duct stone

I. Complications

1. Pancreatic pseudocyst or phlegmon  
   a. 1 – 8% of patients  
   b. Approx. 2 – 4 wks after initial attack  
2. Hemorrhagic pancreatitis  
3. Pancreatic abscess, infected pseudocyst  
4. Pulmonary effusions (usually left-sided)  
5. ARDS (mortality 60%)  
6. Cardiovascular collapse  
7. Renal failure (ATN)  
8. Acute GI bleeding, DIC  
9. Hypocalcemia, hyperglycemia  
10. Pancreatic insufficiency – exocrine and endocrine  
    a. Most commonly with chronic pancreatitis  
    b. Fat malabsorption when 90% of pancreas is destroyed
VI. DISORDERS OF THE ESOPHAGUS

A. Esophageal Dysphagia

1. Obstructive disease (85%)
   a. Foreign body / food impaction
      i. Main areas of obstruction
         • Cricopharyngeus muscle (at C6) – most common!
         • Aortic arch
         • Left mainstem bronchus
         • Gastroesophageal junction
   b. Carcinoma
      i. Squamous cell - 95%
      ii. Risks factors
         • Alcohol
         • Smoking
         • Achalasia
         • Caustic lye ingestion
   c. Webs
      i. Mid – proximal esophagus
      ii. Plummer-Vinson syndrome
         • Chronic iron deficient anemia
   d. Schatzki’s ring
      i. Distal esophagus
      ii. Food impaction common
   e. Strictures
      i. Scarring from GERD or chronic inflammation
   f. Thyroid enlargement / goiter
   g. Diverticulum
      i. Can occur anywhere along esophagus
      ii. Zenker’s diverticulum
         • Proximal to upper esophageal sphincter (UES)
         • After age 50

2. Neuromuscular disorders (15%)
   a. Achalasia
      i. Impaired relaxation of LES
      ii. Odynophagia, CP, regurge, wt loss
   b. Peristaltic dysfunction
      i. “Nutcracker esophagus”
      ii. Strong, prolonged peristaltic contractions
   c. Scleroderma, Myasthenia Gravis, MS, Dermatomyositis
   d. Infectious
      i. Polio
      ii. Diphtheria
      iii. Botulism
      iv. Rabies
v. Tetanus  
e. ALS  
f. CVA  
g. Diabetic neuropathy  

3. History / physical exam  
a. Determine if acute, subacute or chronic  
b. Determine if obstructed to solids, liquids or both  
   i. Solids only = obstructive  
   ii. Solids and liquids = neuromuscular  
c. If complete obstruction  
   i. Inability to swallow  
   ii. Drooling  
   iii. Induced retching  
   iv. Pain from neck to epigastric region  

4. Diagnosis  
a. X-rays of neck/chest  
   i. Visualize FB  
   ii. Mediastinal air if perforated  
b. EGD – gold standard  
   i. Diagnosis and treatment  
c. Gastrografin / Barium contrast studies  
   i. Used if EGD unavailable  
   ii. Use Gastrografin if suspected perforation!  
      • Gastrografin is water soluble; barium is not  
      • Gastrografin is a pulmonary irritant…don’t use if aspiration risk.  
   iii. Barium may interfere with later EGD visualization.  

5. Treatment  
a. Food impaction  
   i. Glucagon 1 mg IV (up to 2 mg)  
      • Relaxes lower esophageal smooth muscle  
      • Do NOT use if history of pheochromocytoma, may precipitate hypertensive crisis.  
   ii. Carbonated beverages  
      • Avoid if complete obstruction or obstruction of > 24 hrs due to possible perforation  
   iii. Nifedipine 10 mg SL  
      • Relaxes smooth muscle  
   iv. Nitroglycerin  
      • Relaxes smooth muscle  
      • Avoid if hypotensive  
   v. Meat tenderizers  
      • Not recommended!!  
   vi. EGD if readily available or all else fails  
      • Gold standard  

b. Foreign bodies  
   i. If able to visualize, remove with forceps
ii. Esophageal FBs need EGD
  iii. FBs passed into stomach can follow-up as outpatient (if not sharp/long)

  c. Strictures/Webs/Rings
     i. Dilatation by gastroenterologist

B. Esophagitis

1. Etiology (many!)
   a. Infectious
      i. Usually immunocompromised
         • Chemotherapy pts
         • HIV
         • Transplant
         • DM
         • ETOH
   ii. Organisms
      • **Candida (most common)**
      • HSV
      • CMV

   b. Inflammatory

   c. GERD
      i. May lead to ulcers, strictures, Barrett’s esophagus

   d. **Medications** – pills stick to esophagus
      i. **Doxycycline (most common)**
      ii. NSAIDS
      iii. KCl supplements
      iv. Ferrous sulfate

2. History
   a. Odynophagia
   b. Dysphagia (liquids and solids)
   c. CP, heartburn
   d. Fever/bleeding in some immunocompromised patients

3. Physical exam
   a. Oropharynx may appear normal

4. Diagnosis
   a. EGD
      i. **White plaques seen in Candida**
      ii. Ulcerative lesions seen in HSV
      iii. Definitive diagnosis is with biopsy
   b. Air-contrast barium swallow
      i. Reveals ulcerations/plaques
      ii. Not definitive
      iii. Bad idea if any perforations
   c. X-rays not helpful

5. Treatment
   a. Candida infection
i. Oral Fluconazole (other –azoles) 3-4 wks
b. HSV
   i. Acyclovir (Fam/Vancyclovir) 1-2wks
c. CMV
   i. Ganciclovir or foscarnate for 2-3 wks
d. Admit for severe disease, dehydration
e. Topical anesthetics, antacids, Sucralfate

C. Boerhaave’s Syndrome

1. Spontaneous rupture of esophagus
   a. Most commonly during forceful emesis
   b. Classically seen in alcoholic patients
   c. Also seen in severe coughing, straining during childbirth & weightlifting
2. Esophageal perforation generally has highest mortality of all perforated GI viscus
3. History
   a. Episode of severe, violent emesis
   b. Chest pain
4. Physical
   a. Hamman’s sign
      i. “Crunching” on auscultation of heart due to mediastinal emphysema
   b. Neck crepitans, subcutaneous emphysema
   c. Septic shock (late presentation)
5. Diagnosis
   a. CXR
      i. Mediastinal air, subcutaneous emphysema, pneumothorax, air-fluid levels, pleural effusion (Rt > Lt)
   b. Gastrografin swallow study – Do NOT use barium!
      i. Shows extravasation through perforation
6. Treatment
   i. Broad spectrum antibiotics
   ii. Fluid resuscitation
   iii. Emergent surgical repair

D. GERD (Gastroesophageal Reflux Disease)

1. Affects up to 25% adult population; higher in elderly
2. Pathophysiology
   a. Intermittent decrease in LES pressure
   b. Decreased esophageal motility
   c. Prolonged gastric emptying time
3. History
   a. Burning sensation / discomfort / pressure in chest, neck
   b. May radiate to arms, shoulders, back
   c. Regurgitation (bitter or acid material in mouth)
d. Symptoms worse after meals
e. Relief with antacids (often brief)
f. Worse when lying down
g. May be aggravated with activity, better at rest
h. Asthma (reflux gastric contents into lungs)

4. Physical exam
   a. Not very helpful
   b. History is key

5. Diagnosis
   a. Clinical diagnosis (history) in ED
   b. R/O cardiac cause of pain
   c. EGD for definitive diagnosis

6. Treatment
   a. Lifestyle modifications
      i. Sleep at angle (30°), not recumbent
      ii. Do not eat within 3hr of bedtime
      iii. Avoidance of:
         • Smoking / nicotine
         • Alcohol, caffeine, fatty foods, citrus
         • Tomato based foods, chocolate
   b. Antacids
   c. H2 blockers, PPIs
   d. Surgery last resort

7. Complications
   a. Changes in esophageal mucosa lead to inflammation, ulcers, erosions and strictures
   b. Barrett’s esophagus
      i. Metaplasia from normal stratified squamous epithelium to metaplastic columnar epithelium
      ii. Strong correlation with adenocarcinoma of esophagus
   c. Asthma exacerbations
   d. Dental erosions
   e. Chronic cough

VII. DISORDERS OF THE STOMACH

A. Acute gastritis

   1. Pathophysiology
      a. Superficial gastric erosions (not into submucosa)
      b. Localized or generalized

   2. Causes
      a. Helicobacter pylori
      b. Drugs
         i. ASA/NSAIDs
         ii. Iron
         iii. Potassium
         iv. ETOH
c. Infectious (viral, bacterial, parasitic)
d. Corrosive agents
   i. Acids
   ii. Alkali
e. Ischemia from severe illness
   i. Shock, trauma, severe burns, organ failure
f. Crohn’s
g. Sarcoidosis

3. Presentation
   a. Epigastric pain – burning, midline
   b. Pain **increases** with eating in 50%
   c. Vomiting often relieves pain that occurs after eating

4. Diagnosis
   a. Usually clinical diagnosis in ED
   b. Endoscopy is test of choice
   c. Rule out other diseases

5. Treatment
   a. Antibiotics for H. pylori
      i. Not usually started in ED
   b. H2 receptor antagonists
   c. Antacids, sucralfate
   d. Discontinue contributing factors
      i. NSAIDs, ETOH
      ii. Stress

6. Complications
   a. **Pernicious anemia:**
      i. Macrocytic anemia, memory loss, peripheral neuropathy
      ii. Pathophysiology
         • Destruction of gastric parietal cells, leads to…
         • Loss of intrinsic factor production, which leads to…
         • Malabsorption of vit B12 (pernicious anemia)
   b. Bleeding
   c. Gastric outlet obstruction
   d. Perforation

B. Peptic Ulcer Disease (PUD)

1. Erosions into submucosal layer of stomach or duodenum
2. **Duodenal 80%; gastric ulcers 20%**
3. Risk factors
   a. H. pylori
   b. NSAIDs
   c. **Zollinger-Ellison syndrome**
      i. Gastrin secreting tumor
      ii. Causes up to 1% of PUD cases
   d. Smoking
   e. Alcohol
   f. Family history of PUD
4. Duodenal ulcers
   a. Common, seen in up to 10% of population
   b. Recurs in up to 90% within 2 years
   c. **H. pylori present in 95% of patients**
   d. H. pylori eradication reduces the recurrence rate to 15%

5. Gastric ulcers
   a. 95% are within 2 cm of pylorus
   b. Up to 20% also have duodenal ulcer disease
   c. **H. pylori present in 80-90% of patients**

6. Presentation
   a. Epigastric pain seen in both types of ulcers
      i. Duodenal: food relieves pain
      ii. Gastric: classically worse with food
   b. Constant, gnawing or aching pain
      i. Pain may awaken the patient at night (increased gastric output)
      ii. Pain may radiate to the back
      iii. Relief often with antacids
   c. Weight loss

7. Physical exam
   a. Vague epigastric tenderness
   b. Heme positive stools (not sensitive)

8. Diagnosis
   a. Labs (primarily to rule out other diseases)
   b. Test for H. pylori
      i. Not usually done in ED
      ii. Biopsy, CLO test during EGD
      iii. Urea breath test
      iv. Antibody testing
         • Antibodies remain positive for years after eradication
   c. EGD: gold standard
   d. Radiographic studies (upper GI)

9. Treatment
   a. General measures
      i. **Stop smoking**
      ii. Frequent small, bland meals
      iii. Avoid alcohol, NSAIDs
      iv. Decrease stress
   b. Medications
      i. Antibiotics for H. pylori
         • Not usually started in ED
         • Pylori antibiotic regimens often changing
      ii. Antacids
         • May decrease absorption of warfarin, digoxin, some anticonvulsants/antibiotics
      iii. PPI (preferred for NSAID related ulcers)
iv. H2-receptor antagonists
v. Sucralfate
vi. Misoprostol (Prostaglandin E analog)
c. Surgery indicated in medical failures

10. Complications
a. Hemorrhage
i. Occurs in up to 20% patients
ii. **Accounts for 60% of UGI bleeds**
iii. Factors predicting higher risk of death
   - Older age
   - Comorbid illnesses
   - Large initial bleed
   - Hemodynamic instability
   - Continued or recurrent bleeding
   - Failure to clear NG with lavage
iv. Emergency treatment
   - IV (2 large bore)
   - Resuscitate with isotonic fluid/PRBCs
   - **Transfuse with screened whole blood if pt deteriorating (don’t wait for cross-matched)**
   - NG lavage
   - IV H2 blockers
   - Emergent EGD if uncontrolled bleeding for sclerotherapy, coagulation, or laser therapy
b. Perforation
i. Less common (7% patients)
ii. Most often on anterior surface of the duodenum/pyloris or lesser curve of stomach
iii. Abrupt onset of severe pain!
   - Localized pain, quickly spreads to entire abdomen
   - Elderly with less pain, less impressive exam
iv. **Duodenal ulcers**
   - Perforate posteriorly into pancreas, retroperitoneum
v. **Gastric ulcers**
   - Perforate anteriorly into abdomen
vi. Upright Abd X-ray in anterior perforation shows subdiaphragmatic air
   - May insufflate 200 – 400cc air through NG tube to accentuate – this is controversial
vii. Treatment of perforation with antibiotics, NGT, **surgery**
c. **Gastric outlet obstruction**
   i. 2% patients
   ii. Edema and scarring near gastroduodenal ulcers
   iii. Presents as weight loss, reflux, pain, vomiting, early satiety
   iv. Physical exam: abdominal distention, succussion splash
v. Treatment: IVFL, NGT, admit
   • Some obstructions will open as edema subsides
   • Others need surgical procedure

VIII. PERFORATED VISCUS

A. Pathophysiology

1. Results from acutely or chronically diseased viscus
   a. Rare to occur in normal bowel (except trauma)
   b. Small bowel perforation
      i. Mid-gut perforations are rare
   c. Large bowel perforation
      i. Usually due to
         • Diverticulitis
         • Carcinoma
         • Trauma
         • Colitis
         • Foreign bodies
      ii. Rapid onset due to bacterial contamination
2. Most perforations occur freely into peritoneum
3. High mortality rate

B. Presentation

1. Abrupt onset of abdominal pain
   a. Elderly may have little or no pain
2. Frank peritoneal signs (mild to severe)
3. Vomiting
4. +/- Fever
5. Hypovolemic, toxic late in course
6. Severity depends on
   a. Site of perforation
      i. Gastric = chemical peritonitis
      ii. Intestinal = bacterial peritonitis
   b. Time from onset of perforation
   c. Age
   d. Comorbid diseases, immunocompetency

C. Physical exam

1. Decreased bowel sounds
2. Tympany
3. **Rigid abdomen**
4. Tachycardia
D. Diagnosis

1. X-ray
   a. Upright chest
      i. **Air under diaphragm**
         - Can increase yield by adding 200-400 ml of air into NGT and retaking film – this is controversial
   b. Left lateral decubitus abdominal film
      i. Air over liver
   c. Abdominal flat plate
      i. Air around bowel wall
2. CT scan - very sensitive for air!
3. Causes of pneumoperitoneum seen on X-ray or CT scan **NOT** from perforated viscus
   a. Tracheostomy
   b. S/p DPL, abdominal surgery
   c. Pneumothorax
   d. Infection from gas forming organisms
   e. Urogenital insufflation during sex

E. Treatment

1. Fluid resuscitation
2. IV antibiotics
3. NGT
4. Emergent surgical consultation / immediate surgery

IX. DISORDERS OF THE SMALL AND LARGE INTESTINE

A. Intestinal obstruction (general)

1. Common cause of abdominal pain in ED
   a. SBO represents 20% of hospital admissions for acute abdominal pain
2. Overall mortality
   a. 3-5 % for SBO
   b. 20% for LBO

B. Mechanical small bowel obstruction (SBO)

1. Etiology
   a. Adhesions – post surgical
      i. **Most common cause!** (more than 50%)
      ii. Typically months to years after surgery, however may occur within the first few weeks
   b. Hernias - incarcerated
      i. **Second most common cause!**
c. Neoplasms
   i. Adenocarcinoma
   ii. Polyps
   iii. Lymphoma

d. Intussusception
   i. Often lymphoma at the lead point in adults

e. Bezoars
   i. In patients s/p pyloroplasty or pyloric resection

f. Crohn’s disease

g. Infection/abscess

h. Radiation enteritis

2. Pathophysiology, H&P, diagnosis & treatment … included below under Large Bowel Obstruction

C. Functional small bowel obstruction (adynamic ileus)

1. Etiology: No structural abnormality
   a. Abdominal trauma, s/p laparotomy
   b. Infection with local inflammation
   c. Metabolic (hypokalemia!)
   d. Drugs (opiates)
   e. Others: MI, scleroderma, hypothyroidism

2. Pathophysiology, H&P, diagnosis & treatment ..... included below under Large Bowel Obstruction

D. Large bowel obstruction

1. Etiology
   a. Tumor (most common cause!)
      i. Tumors on left = obstruction
      ii. Tumors on right = bleeding
   b. Diverticulitis (second most common)
   c. Volvulus (see next section for more detail)
      i. Sigmoid: 80 – 85% of cases
      ii. Cecal: 10 – 15% of cases
   d. Fecal impaction
      i. Elderly, nursing home patients
      ii. Narcotic and laxative abusers
      iii. Mentally delayed
   e. FB
   f. Crohn’s
   g. Radiation or ischemic colitis

2. Pathophysiology
   a. Proximal bowel distends
   b. Lymphatic and venous compromise with edema and stasis
   c. Third spacing of fluids in distended loops of bowel
   d. Bacteria propagate in distended loop
   e. Increasing intra-luminal pressure impairs vascular supply
f. Bowel wall becomes ischemic, develops gangrene, perforates
g. Peritonitis, sepsis, shock

3. History
   a. Crampy, colicky abdominal pain
   b. Vomiting
   c. Obstruction suggests complete obstruction (late finding)

4. Physical exam
   a. Abdominal distension
   b. High pitched bowel sounds, “rushes”
   c. Tenderness (variable): minimal / diffuse or severe / localized
   d. Peritoneal signs seen in gangrenous or perforated bowel
   e. Fever, hypotension, tachycardia suggest sepsis

5. Diagnosis
   a. Obstructive series: flat & upright Abd, upright CXR, lateral
decubitus film
      i. SBO confirmed by XRY in 50-60% cases
         • “Step ladder” air fluid levels
         • Absence of large bowel gas
      ii. Dilated loops of bowel
         • Large bowel >10 cm = imminent perforation
         • Small bowel >3 cm = distention
   b. Laboratory (very nonspecific)

6. Treatment
   a. IV fluids!!
   b. Correct electrolyte abnormalities
   c. NG suctioning
   d. Broad spectrum antibiotics if strangulation, perforation or
      peritonitis
   e. Emergent surgical consultation

E. Sigmoid Volvulus – **most common volvulus (80 – 85%)**

1. Epidemiology
   a. **Elderly, bedridden**
   b. Psychiatric patients on anticholinergics
   c. Neurologic disorders
   d. **History of constipation**

2. Pathophysiology
   a. Lengthening of chronically distended colon
   b. Leads to redundant sigmoid loop which twists on itself

3. History
   a. Crampy lower abdominal pain
   b. N/V/dehydration
   c. Constipation
   d. SOB due to abdominal distention
   e. Similar episodes in past that resolved with passage of flatus
      and stool
4. Physical exam  
   a. Diffuse abdominal tenderness  
   b. Distension  
   c. Fever suggests possible peritonitis due to bowel strangulation or perforation in older patients  

5. Diagnosis  
   a. Must have high index of suspicion in incapacitated patients with abdominal pain  
   b. **KUB confirms diagnosis in 80% cases**  
      i. Dilated loop of colon on left  
      ii. *“Bent inner tube”*  
   c. Barium enema = “Bird’s beak”  
      i. Use Gastrografin if perforation suspected  
   d. WBC > 20,000 suggests strangulation present  
   e. CT scan often useful  

6. Treatment  
   a. IV fluids  
   b. Antibiotics if peritonitis / strangulation  
   c. Surgery consult  
      i. **Decompression with sigmoidoscopy and rectal tube 85 – 95% success rate**  
         - Recurrence rate 90%  
      ii. Surgery  
         - If unable to decompress  
         - If signs of peritonitis  
         - Elective surgical resection often after decompression  

F. Cecal Volvulus  

1. **Most common in ages 25-35**  
2. **NO history of chronic constipation**  
3. Pathophysiology  
   a. Hypo fixation of bowel to the posterior abdominal wall  
      i. Often congenital  
   b. Previous abdominal surgery, disrupting the fixation of cecum to posterior abdominal wall  
   c. Rotation of bowel leads to closed loop obstruction  
4. History  
   a. Severe, colicky abdominal pain  
   b. N/V/dehydration  
5. Physical exam  
   a. Diffusely tender abdomen with large distention  
6. Diagnosis  
   a. KUB  
      Large dilated loop of colon in midabdomen  
      i. Empty distal bowel  
      ii. *“Coffee bean” deformity*  
      iii. Free air indicates bowel perforation
7. Treatment
   a. Surgery is treatment of choice!
   b. Non-operative decompression is unsuccessful
   c. Mortality rate is high!
      i. 10 – 15% if bowel viable
      ii. 30 – 40% if bowel gangrenous

G. Hernias

1. Types
   a. Inguinal (most common)
      i. 75% of all U.S. hernia operations are inguinal
      ii. Indirect (50%) = through inguinal ring, failure of
          obliteration of processus vaginalis
      iii. Direct (25%) = through Hesselbach’s triangle, weakness
           of transversalis fascia
      iv. Men > women
      v. High risk of incarceration in kids
   b. Femoral (5%)
      i. Women >> men
   c. Incisional (10%)
   d. Umbilical
      i. Common in newborns
         • Incarceration very rare
         • 80% spontaneously close by 3-4 yo
      ii. In adults women > men
      iii. Higher risk in obese, pregnant, ascites
   e. Other: epigastric, spigelian, obturator

2. Definitions
   a. Incarcerated = unable to reduce
   b. Strangulated = incarcerated with vascular compromise

3. Clinical presentation
   a. Vast majority asymptomatic
   b. Pain at site if incarceration
   c. Infants may just be irritable
   d. Leads to SBO symptoms
   e. Strangulation and necrosis may present as peritonitis and
      shock

4. Treatment
   a. Reduce if recent, non-tender incarceration
      i. Trendelenburg, sedation, warm compresses
      ii. Do NOT reduce if prolonged incarceration or possible
          dead bowel present!
   b. IV, NGT, labs, supportive care
   c. Antibiotics if perforation or dead bowel suspected
   d. All non-reducible, acutely incarcerated hernias or
      strangulated hernias need emergent surgical repair
H. Mesenteric vascular disease

1. Rare, but catastrophic; mortality rate of 70 – 90%
2. Occurs primarily in elderly (not exclusively)
3. Etiology
   a. **Arterial embolus 50%**
   b. Non-occlusive disease 20%
      i. CHF, hypovolemia, sepsis, shock
   c. Arterial thrombus 15%
   d. Venous occlusion 5%
   e. Chronic ischemia
4. Risk factors
   a. Increased age!
   b. CAD, vascular disease, valvular heart disease
   c. Dysrhythmias (Afib!)
   d. Drugs: vasopressors, diuretics, digitalis, cocaine
   e. Bleeding, dehydration, low cardiac output
   f. Hypercoagulable states
5. Pathophysiology
   a. Impaired blood supply from SMA, IMA, and celiac trunk
   b. Adynamic Ileus due to lack of blood supply
   c. Mucosal infarction/sloughing (occult or gross bleeding)
   d. Third-spacing of fluid in affected bowel
   e. Bacterial invasion of bowel wall/ blood supply
6. Presentation
   a. Acute, severe colicky pain, poorly localized in pt > 50 yo with significant underlying vascular disease (i.e., Afib)
   b. Recent h/o postprandial pain (intestinal angina), weight loss, altered bowel habits in non-occlusive or arterial thrombus disease
   c. N/V/D
   d. 25% with predominant complaint of abdominal distention with rectal bleed (but no abd pain)
7. Physical exam
   a. **Pain out of proportion to exam** (classic!)
   b. Nonspecific exam
   c. Abdominal distension
   d. Heme positive stools (more than half of all cases)
   e. Tenderness progresses with time
   f. Late findings = peritonitis, shock, hypothermia
8. Diagnosis
   a. Clinical suspicion is most useful!
   b. Tachycardia
   c. WBC often > 15,000 (nonspecific)
   d. Metabolic acidosis (lactatic acidosis)
      i. High sensitivity (but nonspecific)
   e. Elevated amylase, phosphate, LDH (nonspecific)
   f. **Angiography is gold standard!**
i. Diagnostic and therapeutic
ii. Contraindicated if patient in shock or receiving vasopressor therapy – each mimics disease

g. CT scan of abdomen
i. Edema of bowel wall and mesentery
ii. Demonstrates abnormal gas patterns
iii. Occasionally see mesenteric thrombus
iv. **Normal CT scan does not rule out ischemia**

h. Plain X-rays
i. Nonspecific
ii. Only late findings may be seen:
   - Ileus
   - Bowel wall edema
   - Gas in portal venous system
   - Gas in bowel wall - “pneumatosis intestinalis” (classic)

i. Barium studies
i. Thumb printing (thick mucosal folds)
ii. Contraindicated because barium can limit visualization during angiography

9. Treatment
a. Aggressive IV fluid hydration
b. Correct precipitating causes
c. NG tube
d. IV antibiotics – broad spectrum
e. Interarterial papaverine (reduces arterial spasms)
f. Emergent surgical and radiographic consult
i. **Laparotomy should be avoided in nonocclusive mesenteric ischemia.** Anesthesia & operative manipulation can cause ischemia to progress to necrosis.
ii. Angioplasty
iii. Surgical resection of necrotic bowel

I. Acute appendicitis

1. General
a. Common cause of abdominal pain
b. 250 – 300,000 new cases yearly in US
c. Approximately 6% of people in their lifetime will develop appendicitis
d. Low mortality
i. 0.1% in simple appendicitis
ii. 2-6% with perforation (general public)
iii. 9% with perforation (elderly)
e. Can affect all ages (peaks at 10 – 30 yo)
f. Difficult to diagnose in young, old, pregnant and immunocompromised

2. Pathophysiology
a. Appendiceal luminal obstruction leads to inflammation, bacterial invasion, infection, vascular compromise, appendiceal edema
b. May progress to gangrene, perforation, sepsis, and death

3. History
a. **Abdominal pain (98% of cases)**
   i. “Classic” presentation (much less often)
      - Vague epigastric, periumbilical pain later migrating to RLQ
      - Occurs in 20 – 60% young adults, 30% elderly
      - < 48 hrs to localize to RLQ
b. Anorexia (70%)
c. Nausea, vomiting (67%)
d. **Kids, elderly and pregnant perforate more often**
   i. Delay in diagnosis is greatest risk
   ii. Nonspecific complaints and exam
   iii. 70% of child perforations are < 9 years old
   iv. 70% of elderly perforations are > 60 years old
   v. 50% of kids diagnosed with appendicitis previously seen by physician with misdiagnosis
      - Gastroenteritis, UTI most common misdiagnoses
e. **Pregnant patients often with RUQ tenderness**
   i. Appendix rises as uterus grows

4. Physical exam
a. **RLQ to Right Mid tenderness seen in nearly 95%**
b. Rovsing sign: RLQ pain on palpating LLQ
c. Psoas sign: pain on elevation & extension of right hip
d. Obturator sign: flexion & internal rotation causes pain
e. Palpable mass felt in <5%
f. Rebound tenderness is late finding
g. Pelvic appendix may lead to rectal / vaginal tenderness

5. Diagnosis
a. Laboratory
   i. WBC > 10K (75% of patients)
      - Normal value does NOT rule out diagnosis
      - Left shift common
   ii. Urinalysis
      - Sterile pyuria common, especially if pelvic appendix
      - > 20 WBCs/hpf more likely to have genitourinary tract infection
b. X-ray
   i. KUB not helpful, may help to r/o other diseases
c. Ultrasound
   i. Sensitivity 75 – 90%, specificity 95%
   ii. Best for kids, pregnant women
   iii. Visualization of immobile, non-compressible appendix of greater than 6mm diameter
iv. Difficult to visualize if retrocecal appendix  
v. Results are user dependent  
d. CT scan  
i. **Test of choice!**  
ii. Oral and IV contrast best  
iii. Sensitivity 96%, specificity 95%  
iv. Large appendix > **6mm diameter**  
v. Pericecal inflammation  
vi. Appendicolith  

6. Treatment  
a. IV fluids  
b. Antibiotics to cover abdominal flora  
c. **Surgery** is definitive treatment  

7. Complications  
a. Perforation, abscess  
b. Localized wound infection, especially if perforated  
c. Low complication rate if not perforated  
d. SBO  

J. Irritable Bowel Syndrome  

1. Approx. 15% population with symptoms consistent with IBS  
2. Young to middle-aged adults  
3. **Female: male = 2:1**  
4. Pathophysiology  
a. Precipitating cause **unknown**  
b. Alteration in intestinal motility and sensation  
c. Thought to be neurophysiologic disorder  

5. History  
a. Abdominal pain  
i. Often relieved with passage of stools/flatus  
b. **Altered defecation**  
i. Constipation, diarrhea, or both  
ii. Incomplete evacuation  
iii. Mucus in stools  
c. Many extracolonic symptoms  
i. Bloating, belching, nausea, weakness  
ii. **Significant wt loss is UNUSUAL**  

6. Physical exam  
a. Nonspecific  
b. Anxiety  
c. Mild, diffuse, vague lower abdominal tenderness  
d. No stool on rectal exam  

7. Diagnosis  
a. No specific tests  
b. **Diagnosis of exclusion**  
c. Outpatient work-up often includes:  
i. Sigmoidoscopy
ii. Stool examination
   • Occult blood
   • Bacteria, parasites (Giardia)
iii. Food allergies
iv. Testing for lactose intolerance

8. Treatment
   a. Dietary
      i. Increase stool bulk
      ii. Avoid milk products
      iii. Avoid caffeine
   b. Anti-diarrheals prn
   c. Reassurance
      i. Avoid implying that problem is all psychological!
   d. Consider anticholinergics
   e. Consider anxiolytics, antidepressants. Usually reserved for primary physician
   f. Avoid narcotic analgesics!
   g. Regular appointments and therapeutic relationship with primary physician most helpful

K. Diverticular disease

1. Pathophysiology (general)
   a. Herniation of mucosa/submucosa through defects in muscular wall of bowel
   b. Most common in sigmoid colon (not exclusively)
   c. Diverticulosis
      i. Presence of one or more diverticulum
   d. Diverticulitis
      i. Inflammation of a diverticulum which is almost always symptomatic

2. Acute diverticulitis
   a. Inflamed diverticulum with microperforation
      i. Left > right
      ii. Incidence increases with age (2-4% under 40 yo)
   b. History
      i. Pain, often in LLQ, worsened with bowel movements
      ii. Change in bowel habits, often constipation
      iii. N/V
   c. Physical exam
      i. Low grade fever
      ii. Abdominal distension
      iii. Heme positive stools >50%
      iv. LLQ abdominal tenderness
         • May have localized peritoneal finding
   d. Diagnosis
      i. Often clinical
      ii. KUB nonspecific
iii. Sigmoidoscopy may show diverticula
   • Not done with acute diverticulitis for fear of perforation
iv. Barium enema often shows diverticula
   • Not done with acute diverticulitis for fear of perforation
v. CT scan
   • **Test of choice**
   • Show inflamed diverticula and local irritation to bowel/fat
   • Abscess
   • Evaluates for other disease processes
e. Treatment
i. If pain only
   • Clear liquid diet
   • Stool softeners
   • Pain meds
   • Oral antibiotics
   • Can manage as outpatient if no comorbidities
ii. If systemic symptoms, significant inflammation or abscess
   • Admit
   • Bowel rest (NPO, NG)
   • IV antibiotics
   • Surgical consultation
   • After 2 episodes of acute diverticulitis, consider elective surgery for resection
f. Complications
i. Fistula
ii. Abscess
iii. Obstruction
iv. Perforation
3. Painful diverticular disease
a. Pathophysiology
i. Increased muscular contraction leads to increased intraluminal pressure and stretching of bowel wall and pain
b. History
i. Recurrent, intermittent LLQ pain
   • Pain often post meals
   • Often relieved with defecation or flatus
ii. Diarrhea or constipation
   • May be increased with emotional stimuli
c. Physical exam
i. Mild LLQ tenderness
   • No rebound
   • No guarding
ii. No fevers
d. Diagnosis
   i. Suspect if > 40 years old with above symptoms
   ii. WBC wnl
   iii. Barium enema
   iv. Colonoscopy
e. Treatment – goal is to decrease bowel spasm
   i. Anticholinergics
   ii. Local heat
   iii. High fiber diet
   iv. Anxiolytics
   v. Laxatives/stool softeners
   vi. No antibiotics
4. Diverticular bleeding
   a. Most common cause of significant LGI bleed
   b. Most patients asymptomatic until bleed
   c. Prevalence increases with age
d. Diverticula cause stretching of vasa recta vessels
   i. Bleeding occurs when the vasa recta rupture into the diverticulum
e. Presentation
   i. Mild hematochezia to massive lower GI bleeding
   ii. Most bleeds are painless (classic)
   iii. Many with previous episodes of diverticulitis / pain
f. Diagnosis
   i. Sigmoidoscopy/colonoscopy
      • Proctosigmoidoscopy can r/o hemorrhoidal bleeding
   ii. Tagged red blood cell studies
      • Need bleeding of 0.1-0.2 ml/min
   iii. Selective arteriography
      • Need bleeding of 0.5 ml/min
g. Treatment
   i. Resuscitation
      • 2 large bore IVs
      • Isotonic fluid
   ii. Transfuse PRBCs if needed
   iii. Surgical consultation
   iv. Sigmoidoscopy/colonoscopy
      • Sclerotherapy
   v. Arterial catheterization at bleeding site
      • Inject vasopressor to slow bleed
L. Colonic tumors
1. Presentation
   a. Weight loss
   b. Change in stool character
   c. Rectal bleeding
d. Obstructive symptoms
2. Adenocarcinoma most common tumor
3. Lower GI
   a. **Apple core lesion** (typically left sided)
4. Colonoscopy for definitive diagnosis

M. Crohn’s Disease
1. Pathophysiology
   a. Chronic inflammatory disease of the GI tract that can involve any part from mouth to anus
   b. Segmental granulomatous inflammation **“skip lesions”**
      i. **Transmural** inflammation
      ii. Ileum is most common site
   c. **“Cobblestoning”** by criss-crossing longitudinal ulcers
   d. Confined to colon in 20%
   e. Perianal complications 90%
2. General
   a. Peak incidence **15 – 22 yo**
   b. Secondary peak **55–60 yo**
   c. Prevalence: 10 – 100 cases / 100,000
   d. **Increasing** in U.S. over 20 years
   e. Women increased risk by 20 – 30%
   f. More common in **Caucasians**; 4x more prevalent in Jewish
   g. Family history in 10 – 15%
   h. Diagnosed by upper GI, air-contrast barium enema and colonoscopy
   i. Recurrence rate 25–50% in 1 year for those that have responded to medical management
   j. 75% require surgery within 20 years of onset
   k. Presentation (Crohn’s exacerbation)
      i. Abdominal pain/cramps
      ii. Diarrhea
         • May be bloody, but less common than ulcerative colitis
      iii. Anorexia, weight loss
      iv. Low grade fever
      v. Anemia, malabsorption
      vi. Approximately ½ patients c/o perianal disease
3. Treatment
   a. IV fluids, NPO
   b. Symptomatic care (analgesics, antipyretics, antiemetics)
   c. **Steroids**
   d. **Sulfasalazine**
   e. Nutritional support
   f. Broad-spectrum antibiotics if fulminant colitis
   g. Evaluate for possible complications (below)
   h. Surgery may be needed for complications
4. Complications (lifetime)
a. Perianal abscess / fissure / fistula seen in 1/3 patients
b. Internal abscess / fissure formation in 30%
c. Toxic megacolon – 6%
d. Extraintestinal manifestations in 25-30%
   i. Gastrointestinal
      • Perforation
      • Peritonitis
      • Obstruction
      • Reason for surgery in 33% cases
   ii. Rheumatologic
      • Arthritis
      • Ankylosing spondylitis
      • Sacroiliitis
   iii. Dermatologic
      • Erythema nodosum
      • Pyoderma gangrenosum
   iv. Hepatobiliary
      • Gallstones
      • Pericholangitis, cholangiocarcinoma
      • Pancreatitis
   v. Ophthalmologic
      • Uveitis
      • Episcleritis
   vi. Vascular
      • Vasculitis
      • Thromboembolic disease
   vii. Cancer
      • Risk increased 3-5x as compared to the general population

N. Ulcerative colitis

1. Epidemiology (similar to Crohn’s)
   a. All age groups; peak in 2nd - 3rd decades of life
   b. Industrialized nations; increasing in U.S.
   c. Family history in 10 – 15% cases
   d. Caucasians 4x higher risk
   e. Males slightly more prevalent
2. Pathophysiology
   a. Chronic inflammatory disease of colon and rectum
   b. Continuous from rectum proximally
   c. Limited to mucosa and submucosa, shallow ulcers
   d. Bloody diarrhea with mucoid stools due to friable, ulcerated mucosa -“crypt abscesses”
3. Presentation
   a. Broken into mild, moderate, severe disease
      i. Mild (60%), < 4 BMs per day, few extraintestinal
manifestations
ii. Moderate (25%)
iii. Severe (15%), > 6 BMs per day, anemia, fever, wt. loss, low albumin, multiple extraintestinal manifestations

b. 10% usually present with
i. Low grade fever
ii. Crampy abdominal pain
iii. Tenesmus
iv. Bloody, mucopurulent stool

c. Remainder with
i. Insidious onset
ii. Recurrent fevers
iii. Abdominal pain
iv. Anorexia
v. Mild diarrhea

4. Diagnosis
   a. Sigmoidoscopy / colonoscopy and biopsy

5. Treatment
   a. IV fluids, correct electrolyte abnormalities
   b. Steroids or ACTH
   c. Sulfasalazine
   d. Enemas
      i. Topical glucocorticoid
      ii. 5-aminosalicylic
   e. Antibiotics controversial, used primarily in severe dz
   f. If mild disease: discharge on low residue diet, close medical F/U, possible medication adjustments
   g. Admission if severe symptoms or toxic megacolon

6. Complications
   a. Toxic megacolon
      i. Seen in 5% cases
      ii. Etiology: Loss of neuromuscular tone leads to dilatation of transverse colon
      iii. KUB = long segment of air-filled colon dilated more than 6cm; possible thumbprinting
      iv. Risks
         • Drugs that decrease bowel motility
         • Cathartics
         • Enemas
      v. Presentation
         • Systemic toxicity
         • Marked abdominal distension
         • Peritonitis
      vi. Treatment
         • IVFL, NG, steroids, antibiotics
         • Surgery if not improved 24-48 hr
   b. Obstruction
c. GI bleed
  d. Perforation (25%)
    i. 50% mortality
  e. Perianal abscesses not as common as in Crohn’s
  f. Iron deficiency
  g. Stricture
  h. Extracolonic complications
    i. Same as those seen with Crohn’s

X. ANORECTAL DISORDERS

A. Anal fissure

1. General
   a. Most common cause of painful rectal bleeding
2. Pathophysiology
   a. Tear in squamous epithelium of anal canal
3. Presentation
   a. Pain on defecation!
   b. Hematochezia – not massive!
   c. Often initiated by passage of a hard or sharp stool
4. Physical exam
   a. “Fissure triad”
   i. Deep ulcer
   ii. Sentinel pile
   iii. Enlarged anal papillae
   b. Sentinel pile
   i. 90% posterior midline
   ii. Central linear ulcer
   iii. Hypertrophied papilla internally
   iv. Hypertrophied skin externally, chronic inflammation
   c. Treatment
   i. Sitz baths
   ii. Stool softeners
   iii. Bulking agents
   iv. Non-narcotic pain medications
   v. Topical lidocaine
   vi. Antispasmodic agents – nitroglycerin or calcium-channel blocker gel

B. Hemorrhoids

1. Pathophysiology
   a. Dilated venules of hemorrhoidal plexus
   b. Precipitating factors
   i. Low bulk diet
   ii. Erect posture
   iii. Increased abdominal pressure (straining)
iv. Chronic constipation
v. Pregnant
c. Can occur internally or externally
d. Anoscopy to evaluate for internal hemorrhoids
e. Dentate (pectinate) line
   i. Defines junction of squamous and columnar epithelium
   ii. Defines internal from external hemorrhoids
f. External hemorrhoids
   i. Below dentate line
   ii. **Covered with skin**
   iii. Painful
g. Internal hemorrhoids
   i. Above dentate line
   ii. **Covered with mucosa**
   iii. Painless

2. External hemorrhoids
   a. Presentation
      i. Itching
      ii. Burning
      iii. Rectal bleeding with defecation
      iv. **Thrombosis** (pain!)
   b. Treatment
      i. NSAIDS for pain
      ii. Sitz baths
      iii. Stool bulking agents/stool softening agents
      iv. Avoidance of straining
      v. **Excision of thrombosed hemorrhoid**

3. Internal hemorrhoids
   a. Presentation
      i. Painless, bright-red rectal bleed with defecation
         • Common cause of rectal bleeding
      ii. Prolapse of internal hemorrhoid
         • Often while straining
         • May become permanent
         • May appear like thrombosed external hemorrhoid
         • Often painful
   b. Treatment
      i. Non-prolapsed
         • Hot sitz baths TID
         • Analgesics
         • Prevent prolonged sitting / straining
         • Bulk laxatives / stool softener
         • High fiber/bran diet
      ii. Prolapsed
         • **Manual reduction!**
         • **No excision or I&D!**
         • Surgery may be needed (band ligation) if continued
bleeding or irreducible prolapsed hemorrhoid

C. Rectal foreign body

1. Seen in assault, self-administration, psych patients
2. Fever, abdominal pain suggest perforation
3. Diagnosis
   a. Abdominal X-ray
      i. See foreign body, position, shape
      ii. Evaluate for free air / perforation
4. Treatment
   a. Soft, low lying (< 10 cm from anal verge)
      i. Remove safely in ED
         • Sedation/muscle relaxant
         • Local anesthesia
         • Have patient bear down
         • Forceps
         • May try to pass a Foley beyond obstruction then inflate balloon and provide gentle traction
   b. Do not try to remove large, fragile, hard object especially if > 10 cm from verge
   c. Surgical consultation/general anesthesia often needed
   d. Do not use cathartics!
   e. Indications for admission
      i. Signs of perforation
      ii. Rectal bleeding
      iii. Torn sphincter

D. Anorectal abscess

1. Presentation
   a. Pain!
      i. Worse with defecation, sitting
   b. Fever +/-
   c. Mucopurulent discharge
2. Physical exam
   a. Localized external tenderness
      i. Painful swelling at anal verge = perianal
      ii. Lateral pain, swelling = perirectal abscess
         • Perirectal abscesses may extend into surrounding potential spaces
         • Fluctuance
         • Painful digital exam with tender mass / induration
         • Anoscopy with pus and anal crypts
3. Complications
   a. Fistulas
   b. Gas gangrene
c. Necrotizing fasciitis
d. Sepsis

4. Treatment
   a. I & D necessary in virtually all cases
      i. Simple perianal abscess = in ED
      ii. Pilonidal cyst = in ED
      iii. Perirectal abscess = in OR
   b. Pack all drained abscesses
   c. Antibiotics only if:
      i. Immunocompromised
      ii. Toxic appearing
      iii. Diabetic
      iv. Marked cellulitis
   d. Culture if antibiotics are given
   e. Surgical consultation often necessary

E. Infectious proctitis

1. Seen in patients who have had receptive anal intercourse
2. Initially: itching, seepage, mild pain / irritation
3. Untreated: significant pain, bleeding, discharge

4. Organisms
   a. Gonococcus
   b. Chlamydia
   c. Syphilis
   d. Condylomata acuminata (papillomavirus)
   e. Pinworms
   f. Herpes simplex virus
   g. Atypical organisms seen in AIDS
      i. Mycobacterium avium
      ii. CMV

5. Treatment
   a. Take cultures prior to treatment
   b. Gonorrhea
      i. Multiple choices of antibiotics
      ii. 50% concomitant Chlamydia infection
   c. Chlamydia
      i. Doxycycline first line
   d. Syphilis
      i. PCN
   e. Herpes
      i. Acyclovir

F. Radiation proctitis

1. Presentation
   a. Immediate or delayed after irradiation
   b. Diarrhea, tenesmus, urgency
c. Mild to moderate rectal bleeding
d. Constipation and obstruction
   i. Usually late, secondary to strictures
e. Edematous, friable mucosa in anal canal
f. Fistulas, abscesses

2. Diagnosis
   a. Made clinically in one with diarrhea or rectal bleeding with h/o pelvic irradiation

3. Treatment
   a. Steroid enemas helpful in acute stages
   b. Bulk forming agents
   c. Antimotility agents
   d. Iron supplements

XI. INFECTIOUS DISORDERS OF THE GASTROINTESTINAL TRACT

A. Diarrhea

1. Leading cause of death worldwide due to dehydration
2. Second to “common cold” for lost days from work/school in U.S.
3. Dysentery = diarrhea with blood, pus, mucous
4. Etiology
   a. Infectious #1
      i. Viral (50-70%)
      ii. Bacterial (15-20%)
      iii. Parasitic (10-15%)
   b. Drugs
   c. Diet
   d. Food allergies
   e. Inflammatory bowel disease
   f. Other
      i. Malabsorption
      ii. Malignancy
      iii. Laxatives
      iv. Obstruction
5. Pathophysiology
   a. Invasive
      i. Alteration of GI mucosa
   b. Enterotoxin
      ii. Causes mucosal hypersecretion
         • Isotonic diarrhea in adults
         • Hypotonic diarrhea in children
6. Presentation (variable)
   a. History of ill contacts
   b. New meds or diet
   c. Diarrhea (qualitative, quantitative)
      i. Watery
      ii. Bloody
iii. Mucus
d. Abdominal pain
   i. Generalized
   ii. Crampy
e. Low grade fevers, myalgias, fatigue

7. Diagnosis
   a. **Stool WBC > 5/hpf suggests invasive infection**
   b. Stool cultures
      i. Who needs them?
         - Public health concerns
         - Immunocompromised
         - Travel history
         - Prolonged symptoms
      ii. Fecal leukocytes
         - **Seen in invasive diarrhea**; not seen with toxigenic diarrhea
      iii. AM stool preferable
   c. Stool O & P and special strains

8. Treatment
   a. Proper hydration without heavy osmotic loads (sugar)
   b. Antiemetics
   c. Antidiarrheals – controversial!
   d. Antibiotics
      i. High suspicion for invasive diarrhea
      ii. Toxic appearing
      iii. Positive culture
      iv. **Ciprofloxacin or TMP-SMX** most common

XII. BACTERIAL GASTROINTESTINAL DISEASE

A. Salmonella (Invasive)

1. Most common cause of bacterial gastroenteritis in US
2. **Invasive** bacteria
3. Food and water borne, also transmitted by
   a. Domestic pets (10%)
   b. Eggs
   c. Unpasteurized milk
   d. Pet turtles
   e. Chickens / turkeys
   f. 8 – 48 hour incubation
4. Presentation
   a. **Bradycardic with fever**
   b. Colicky abdominal pain
   c. Watery diarrhea, often with mucous/blood
   d. Mild nausea or vomiting
   e. Can cause **osteomyelitis, especially in sickle cell patients**
f. Can cause septic arthritis in HIV and splenectomized patients

5. Treatment
   a. Uncomplicated
      i. Rarely needs antibiotics
      ii. Increases carrier state and resistance with antibiotics
   b. Antibiotics (Ciprofloxin) in typhoid fever (serotype D)

B. Shigella (Invasive)

1. Food and water borne
2. Oral/fecal transmission
3. As little as 50 – 100 bacilli can cause infection
4. **Invasive and exotoxin** meditated disease
   a. Seen in confined population
   b. Nursing homes
   c. Penal institutions
   d. Indian reservations
5. 24 – 48 hour incubation
6. Presentation
   a. Severe abdominal cramping
   b. Mild watery & often bloody diarrhea
   c. Dysentery possible
   d. Fever
   e. **Seizures/neurologic effects** (rare)
7. Treatment
   a. No antibiotics needed unless severe disease or culture positive for Shigella dysenteriae
   b. Consider Ciprofloxacin, TMP-SMX

C. Yersinia (Invasive)

1. Food (milk, pork) and water borne
2. Oral/fecal transmission
3. **Invasive** bacteria
4. Domesticated animals
5. Presentation
   a. Fever
   b. Watery and sometimes bloody diarrhea
   c. Colicky abdominal pain
   d. Anorexia and vomiting
   e. Usually lasts 10 – 14 days
   f. Cause of mesenteric adenitis, “pseudoappendicitis”
   g. Can cause **erythema nodosum**
6. Treatment
   a. Supportive care
   b. No antibiotics usually necessary
      i. Reserved for refractory disease or immunocompromised
D. Campylobacter (Invasive)

1. Overview
   a. **Most common bacterial causing diarrhea**
   b. Frequent cause of “Backpacker’s Diarrhea”
   c. Water borne (streams)
   d. Raw milk, chicken, pigeons
   e. **Invasive** bacteria
   f. 2 – 5 day incubation
2. Presentation – symptoms of rapid onset!
   a. Crampy abdominal pain
   b. Bloody/loose/watery diarrhea 8-10/day
   c. Constitutional symptoms are the rule
      i. Anorexia
      ii. HA
      iii. Myalgias, malaise
      iv. Fever
3. Treatment
   a. Ciprofloxacin
   b. Erythromycin
   c. Resistance pattern to TMP-SMX

E. Vibrio parahaemolyticus (Invasive)

1. General
   a. Found in temperate coastal sea waters
      i. Very common in Japan, US
   b. **Invasive** bacteria
   c. **Raw fish/shellfish common source**
      i. Cruise ships!
2. Presentation
   a. Incubation often 8-12 hr (range 4 - 48 hr)
   b. Diarrhea
   c. Moderately severe abdominal cramping
   d. Fever
   e. HA
   f. Vomiting not predominant
3. Treatment
   a. Most are self-limited (usually lasts 24-48 hr)
   b. Treatment with Cipro or TMP-SMX shortens duration

F. E. coli serotype 0157:H7 (Cytotoxin)

1. General
   a. **Toxin** mediated
      i. Cytotoxic to intestinal wall
   b. Contaminated food
      i. **Undercooked meats**
ii. Raw milk
iii. Apple cider
iv. Raspberries
c. Person-to-person spread, especially:
   i. Day care centers
   ii. Nursing homes

2. Presentation
   a. Incubation 4 – 9 days
   b. Severe abdominal cramps
   c. Watery stools initially
   d. Becomes grossly bloody in 95% patients

3. Treatment
   a. Avoid anti-motility agents
   b. Antibiotics often indicated

4. Complications
   a. **Hemolytic-uremic syndrome (HUS)**
      i. Seen in 20 – 25 % patients
   b. **Thrombotic thrombocytopenic purpura (TTP)**

G. Traveler’s Diarrhea (Enterotoxin)

1. **Enterotoxic E. coli** most common cause (50 – 75%)
2. Seen throughout the world
3. Contaminated food/water
4. 18 – 72 hour incubation
5. Presentation
   a. Watery diarrhea **without fecal leukocytes** (classic)
   b. Vomiting seen in less than 50%
   c. Mild abdominal pain
   d. Possible bloody stools
6. Treatment
   a. Bismuth – large doses
   b. TMP-SMX or tetracycline
   c. Yogurt (natural “good” bacteria, i.e., lactobacillus)

H. Bacillus cereus (Enterotoxin)

1. Aerobic, spore forming gram (+) rod
2. Two forms:
   a. Emetic form – **contaminated fried rice < 6 hours prior**
   b. Diarrheal – meats or vegetables
3. Presentation
   a. Emetic form
      i. Incubation 2 – 3 hours
      ii. Abdominal cramps
      iii. Diarrhea in 25% patients
      iv. < 10 hours duration
   b. Diarrheal form
i. Incubation 6 – 14 hours  
ii. Diarrhea 100%  
iii. Abdominal cramping 75%  
iv. Vomiting 20%  
v. Duration 20 – 36 hours  

4. Treatment  
a. Mild and self limited  
b. No antibiotics  
c. Antiemetics as needed  

I. Staphylococcal (Enterotoxin)  

1. Grows in protein rich foods (picnic foods!)  
a. Mayonnaise  
b. Potato salad  
c. Eggs  
d. Ham  

2. Only few hours needed for bacteria to grow / make enterotoxin  

3. Presentation  
a. **Explosive onset within 6 hours**  
b. Crampy, abdominal pain  
c. Multiple vomiting  
d. Diarrhea mild  
e. Fever occasionally  
f. **Lasts 6 – 8 hours**, possibly as long 24 hours  

4. Treatment  
a. Antiemetics  
b. IV fluids  
c. No antibiotics  
d. Rapid uncomplicated recovery is rule  

J. Clostridium perfringens (Enterotoxin)  

1. Probably #1 cause of acute food poisoning in U.S.  
2. Undercooked meat / poultry (esp. after sitting out > 24 hours)  

3. Presentation  
a. Onset after 6-12 hr, possibly up to 24 hr  
b. Watery diarrhea  
c. Moderate – severe abdominal cramping  
d. Rarely fevers, N/V  

4. Treatment  
a. Self-limited  
b. **Lasts < 24 hr**  

K. Clostridium difficile (Enterotoxin)  

1. **Pseudomembranous colitis**  
2. **Overgrowth of flora bacteria due to antibiotic use**
3. Toxin mediated – beware of toxin A
4. All classes of antibiotics implicated, especially:
   a. Clindamycin
   b. Ampicillin
   c. Cephalosporins
5. Increased risk if using constipating agents
6. Presentation
   a. During or up to 3 weeks after antibiotic use
   b. Watery, profuse diarrhea
      i. Occasionally bloody
      ii. Up to 30 stools/day
   c. Crampy abdominal pain
   d. Fecal WBC common
   e. High WBC counts
7. Diagnosis
   a. Difficile toxin in stool
      i. Takes lab 48 - 72 hr
8. Treatment
   a. Stop antibiotic!
   b. Flagyl PO or IV first line
   c. Vancomycin PO only if second course needed
   d. 25% relapse after 7 – 14 days

XIII. VIRAL GASTROINTESTINAL INFECTIONS

A. Rotavirus

1. Most common cause of gastroenteritis in young children
2. Double stranded RNA virus
3. Seasonal in winter
4. Severe diarrhea and dehydration
5. Vomiting early then subsides
6. High fever may be seen
7. Self-limited
8. Immunity develops

B. Norwalk Agent

1. Adults, older children
2. Single stranded RNA virus
3. Acute onset diarrhea and mild abdominal cramps
4. Usually no vomiting
5. Possible low-grade fever
6. Myalgias common
7. Self-limited, rarely severe
8. No immunity
XIV. PARASITIC GASTROINTESTINAL INFECTIONS

A. Giardia lamblia

1. **Most common parasitic cause of diarrhea**
2. Most common waterborne cause of diarrhea
3. Water supplies infected with cystic-infected human or animal feces
   a. Beavers
   b. Dogs
   c. Raccoon
4. “**Backpack diarrhea**”; foreign travel with bad water
5. Giardia trophozoites infect small bowel
6. **Presentation**
   a. Colicky abdominal pain
   b. Bloating and flatulence characteristic
      i. Often audible borborygmi sounds
   c. **Explosive, foul smelling stool**
7. **Treatment**
   a. Metronidazole
   b. Quinacrine

B. Entamoeba histolytica

1. In U.S., **chronic disease more common** than acute illness
   a. Incubation 1 week – 1 year
2. Fecal/oral transmission
3. Vague abdominal cramping
4. Flatulence
5. Bloody diarrhea of gradual onset
6. Can cause **liver abscess**
7. O & P may take up to 6 stools to obtain cysts
8. **Treatment**
   a. Metronidazole plus tetracycline, or metronidazole plus iodoquinol for 5 – 10 days
   b. If carrier: Iodoquinol 650 mg TID for 20 days

XV. GASTROINTESTINAL TOXINS

A. Scromboid

8. Heat stable toxin
   a. Produced by bacterial action in certain types of **dark-meat** fish (improperly refrigerated)
      i. Dark meat of tuna
      ii. Bluefish (mackerel, swordfish)
      iii. Mahi-mahi
9. Presentation
   a. **Histamine-like reaction** (key!)
      i. Flushing (facial sunburn)
      ii. Urticarial at times
   b. Diarrhea
   c. Headache
   d. Abdominal cramping
   e. Peppery, metallic taste may be noted
   f. Onset within 20 – 30 minutes of ingestion
   g. Resolves in 5 – 6 hours

10. Treatment
    a. Self-limited
    b. **Antihistamines** (diphenhydramine)
    c. H2 blockers

B. Ciguatera

1. Most common cause of fish-related poisoning in US
2. Ciguatoxin
   a. Heat stable neurotoxin made by dinoflagellate (small plankton that sit within algae)
   b. Accumulates in tissue of fish (larger, older fish)
      i. Red snapper
      ii. Grouper
      iii. Sea bass
      iv. Sturgeon
      v. Barracuda
      vi. Moray eels
3. Presentation
   a. 2 – 6 hour incubation
   b. Predominantly **GI and neurologic** findings
   c. GI
      i. N/V
      ii. Profuse diarrhea
      iii. Abdominal cramping seen early
   d. Neurologic
      i. Throat and perioral dysesthesias, paraesthesias
      ii. Distorted temperature sensation “**Hot–Cold reversal**”
   e. **Worsened by alcohol**
   f. Lasts 1 – 2 weeks on average
4. Treatment
   a. Supportive
   b. IV mannitol may be helpful
      i. 1g/kg of 20% solution over 30 minutes
   c. Abstain from alcohol until all symptoms resolve
XVI. GI BLEED

A. Overview

1. Overall mortality 10%
2. Upper vs lower/ligament of Treitz
3. Causes upper GI bleed
   a. PUD (60%, most common etiology!)
   b. Gastritis/esophagitis (15%)
   c. Varices
      i. Portal hypertension, liver disease
   d. Mallory-Weiss tear
      i. Mucosal tear in lower esophageal region
      ii. Classic: repeated retching, followed by bright red hematemesis
   e. Aortoenteric fissure
      i. History of aortic graft (AAA repair)
      ii. Classic: small self-limited “herald” bleed, subsequent massive hemorrhage
   f. Others
      i. Stress ulcers
      ii. Malignancy
      iii. AV malformation
   g. ENT emergencies can masquerade as GI bleeds
4. Causes of primary lower GI bleed
   a. Hemorrhoids
      i. Most common overall LGI bleed
   b. Diverticulosis
      i. Most common severe LGI bleed
   c. Angiodysplasia
      i. Usually of right colon
      ii. Undiagnosed
   d. Polyps/cancer
   e. Rectal disease
   f. IBD

B. History

1. Hematemesis seen in approximately 50% UGI bleed
2. Melena
   a. 150 – 200 ml blood in GI tract for prolonged period
   b. Seen in 70% patients with UGI bleed
   c. Seen in 30% patients with LGI bleed
   d. Iron, Bismuth can simulate (stool guaiac = Neg)
3. Hematochezia
   a. Bright red/maroon stools
   b. Most likely LGI bleed
c. Can be UGI bleed if rapid transit time
4. Ask about NSAIDs, ASA, ETOH
5. Weight loss, changes in bowel habits
   a. Malignancy
6. May complain solely of:
   a. Weakness
   b. SOB
   c. Dizziness
   d. ICP
   e. Abdominal pain

C. Physical exam

1. Vital signs, orthostatics
2. Skin: warmth, diaphoresis, color, rashes
   a. Cool, clammy skin is sign of shock
   b. Spider angiomata, palmer erythema, jaundice, bruises suggest underlying liver disease
3. Abdomen
4. Rectal

D. Diagnosis

1. Hemoccult
   a. Can have guaiac positive stool up to 14 days after UGI bleed
   b. False positives
      i. Red fruit or meat
      ii. Iodide
      iii. Methylene blue
2. Labs
   a. CBC
      i. Hgb/Hct—traditionally transfuse acute bleed if Hgb < 8
      ii. Platelets
   b. PT/PTT
      i. Increased PT/INR may be from liver failure, vitamin K deficiency, Coumadin
   c. Type & screen / type & cross
      i. Type specific blood in 10-15 minutes
      ii. Fully crossmatched blood in 45-60 minutes
   d. Electrolytes
      i. Decreased potassium; metabolic acidosis if vomiting
      ii. Pre-renal azotemia
         • Reabsorption of blood from GI tract increases BUN
         • Hypovolemia increases BUN
3. EKG
   a. Obtain when
      i. >50 yo
      ii. Preexisting CAD
iii. Patients with significant anemia
iv. CP/SOB
v. May have asymptomatic MI

4. X-rays
   a. Not helpful
   b. Barium studies limit use of endoscopy or angiograms

5. Treatment
   a. If unstable, initial resuscitation
      i. 2 large bore IVs, O₂, monitor
      ii. Blood products
      iii. GI consult for UGI bleed, surgery consult for LGI bleed
   b. NGT and lavage
      i. May help identify UGI vs LGI bleed
      ii. Evaluate for active UGI bleed
      iii. Neg NG does not r/o UGI bleed
      iv. Note: 14% of bright red/maroon blood per rectum unexpectedly from UGI
   v. OK with varices
      vi. Done with room temperature water
   c. Anoscopy/proctosigmoidoscopy
      i. For mild rectal bleeding
      ii. Check for hemorrhoids
      iii. Check if stool above rectum with blood
   d. Colonoscopy
      i. Diagnose diverticulosis, AV malformations
      ii. Able to ligate or sclerose
   e. EGD
      i. Most accurate diagnostic tool for UGI bleeds
      ii. Identifies lesions up to 95% patients
      iii. Emergently at bedside if unstable, otherwise if stable may be done in 12-24 h
      iv. Band ligation or sclerotherapy for varices
   f. Octreotide / Somatostatin
      i. Patients with varices, Peptic ulcer disease
      ii. IV infusion at 50 mcg/hr for 24 hours in ICU
   g. Vasopressin
      i. Patients with varices
      ii. Many catecholamine side effects
      iii. Consult with GI before using
   h. Balloon tamponade (Sengstaken-Blakemore tube)
      i. Controls variceal hemorrhage in 40- 80%
      ii. Many serious side effects
      iii. Use has decreased significantly
   i. Surgery indications
      i. Hemodynamically unstable
      ii. Unresponsive to volume resuscitation, endoscopy and correction of coagulopathy
      iii. Transfused > 5 units within 4 – 6 hours, OR 2 units every
4 hours after replacing initial loss
iv. Mortality approaches 23% when taken emergently to operating room
j. May discharge home if
i. Currently asymptomatic
ii. No comorbidities
iii. Normal vital signs, normal hgb/hct
iv. NG aspirate negative
v. Heme negative or trace heme positive stools
vi. Patient with understanding of disease with good follow-up within 24 hours and ability to return if worse
vii. Admit all others
GI EMERGENCIES

PEARLS

1. Appendicitis is the most common cause of acute abdominal pain requiring emergency surgery (37%).

2. Elderly patients are more prone to perforation from appendicitis (44-70% compared to 10-17% for adults under 60 years) due to the poor blood supply to the appendix and the thin appendix wall. Children under 6 years also have a high appendix perforation rate (65%) due to atypical presentation and inability to communicate.

3. Intermittent abdominal pain and vomiting with a prior surgery or trauma - think obstruction.

4. The most common causes of small bowel obstruction are postoperative adhesions (50%), hernia (15%) and malignancy (15%).

5. The most common causes of large bowel obstruction (excluding fecal impaction) are carcinoma (50-69%), volvulus (10-25%) and diverticular disease (10-15%).

6. Adynamic (paralytic) ileus is the most common cause of ileus overall.

7. Acalculous cholecystitis due to biliary stasis and obstruction has an atypical presentation with higher morbidity and mortality than calculous cholecystitis.

8. Charcot’s triad for ascending cholangitis is fever, jaundice and RUQ pain.

9. Air in the biliary tree is associated with emphysematous cholecystitis and gallstone ileus.

10. There are many causes of pancreatitis but the most common by far are alcoholism and biliary tract disease.

11. No clinical features are pathognomonic for pancreatitis.

12. Know Ranson’s criteria (on admission):
   - Age over 55
   - Blood sugar greater than 200 mg/dl
   - WBC greater than 16,000 mm$^3$
   - SGOT greater than 250 SF units/L
   - LDH greater than 700 IU/L
   - (during initial 48 hours):
HCT fall > 10%
BUN rise > 5 mg/dL
Serum calcium < 8 mg/dL
Base deficit > 4 mEq/L
Estimated fluid sequestration > 6L
Arterial pO₂ < 60 mmHg

13. Risk factors for peptic ulcer disease include cigarettes, alcohol, coffee, cola, aspirin, NSAIDS, family history, Helicobacter pylori infection. Posterior duodenal ulcers penetrate into the pancreas while anterior ulcers perforate into the peritoneum.

15. A normal viscus will not perforate.

16. The most common cause of visceral perforation overall is ulcers. The most common cause of colonic perforations is diverticular disease.

17. The classic presentation for perforated ulcer is sudden onset of severe pain with a predisposing cause for perforation. Pneumoperitoneum is found in up to 50-80%.

18. Pain out of proportion to physical findings suggests mesenteric ischemia, particularly in the elderly with an arrhythmia.

19. Weight loss, change in bowel habits and abdominal pain—think mesenteric ischemia or cancer.

20. Avoid digoxin, beta blockers and vasopressors in a patient with mesenteric ischemia as they all decrease splanchnic blood flow.

21. The sigmoid colon is involved in 90% of diverticular disease; 80% of patients with diverticular disease are asymptomatic.

22. Bleeding diverticula are painless, usually from the right colon, and are the most common cause of massive LGI bleed overall.

23. Crohn’s disease has segmental granulomatous lesions involving all layers of the intestinal wall with skip areas of normal intestine in between; 75% involve the ileum and perianal disease is common.

24. Ulcerative colitis and Crohn’s disease have similar systemic symptoms and both can result in toxic megacolon.

25. Inguinal hernias are the most common hernias for both sexes.

26. Gastrointestinal “Flags”:
   - Pet turtles, fever but bradycardic, osteomyelitis
     - Salmonella
• Fever, diarrhea, seizure
  - Shigella
• Mesenteric adenitis, erythema nodosum
  - Yersinia
• Cruiseship, raw fish / shellfish
  - Vibrio
• Bad meat, crampy pain, H-U Syndrome
  - E. Coli 0157:H7
• Contaminated fried rice
  - Bacillus Cereus
• Picnic, potato salad, explosive diarrhea
  - Staphalococcal
• Antibiotic use, diarrhea
  - C. Diff
• Child, winter, severe watery diarrhea
  - Rotavirus
• Backpacker, bloating/gas, foul-smelling diarrhea
  - Giardia
• Fish, flushing / urticaria
  - Scromboid
• Fish, paraesthesias, hot / cold reversals
  - Ciguterra
REFERENCES

Abdominal Pain


Acute Appendicitis


Anorectal Disease


Colonic Disease


Gall Bladder Disease


Gastroenteritis


Gastrointestinal Bleeding


Hepatic Disease


Inflammatory Bowel Disease


Ischemic Bowel Disease and Bowel Infarction


Intestinal Obstruction


Pancreatitis

Peptic Ulcer Disease / GERD


Small Intestinal Disease


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