Board Review 2014

Catherine Kerschen DO, FACOI
Michigan State University
College of Osteopathic Medicine
GERD

• Etiology (most common)
  – Transient lower esophageal relaxation
  – Weak lower esophageal sphincter
  – Hiatal hernia
Diagnostic tests

• Not necessary for most pts with GERD
• Alarm symptoms warrant further testing
  – Dysphagia, hematemesis, anemia, wt. loss
• ~50% of pts with typical GERD symptoms have normal endoscopy
Diagnostic tests

• Endoscopy
  – Identifies complications of GERD
    • Ulcer, barretts, stricture, adenoca
  – UGI x-ray
    • Major usefulness: id strictures and large hiatal hernias
    • Sensitivity for GERD only 20%
  – pH monitoring
    • Indications: atypical symptoms, frequent atypical CP, refractory symptoms, Preop confirmation of GERD
Impedance testing

• When combined with pH testing
  – GERD independent or pH
    • Acid and non-acid reflux
Esophageal Question

What is the single most informative study in pts with medically refractory GERD?

a. EGD
b. pH monitor
c. Barium swallow
d. CT scan of chest
• 24 or 48 hour intraesophageal pH monitor while on antisecretory meds. A similar study off medication may be considered if the diagnosis of GERD is in doubt.
Factors associated with severe esophagitis

- Low LES pressure
- Esophageal motor abnormalities
- Recumbent reflux

**Most important determinants of severe endoscopic esophagitis**

- Presence of hiatal hernia also important
Treatment options

1. Lifestyle modifications--- healing rate 20-30%
2. Acid neutralization---- healing rate 20-30%
3. Acid suppression
   H2 blockers--- healing rate 50%
   PPI--- healing rate >80%
4. Prokinetics----healing rates 30-40%
5. Mechanical prevention of reflux
   Laparoscopic surgery----healing rate >80%
   Endoscopic therapies---healing rate >50%
GERD Atypical symptoms

• Evaluate for most common causes 1st
• Trial of high dose ppi
  – If symptoms resolve continue acid suppression at lowest possible dose
  – If symptoms do not resolve completely
    • Further evaluate with egd and/or ambulatory esophageal pH monitoring
Functional CP

- NERD “nonerosive reflux disease”
  - More common in females
  - Features of anxiety, panic, somatization may be present
  - Acid suppression may help, but rarely relieve symptoms completely
  - Reducing visceral hypersensitivity may be helpful
Barretts

• Columnar epithelium replaces the stratified squamous epithelium in the esophagus.
  – Due to years of gastric reflux into esophagus
  – Associated with reflux symptoms, advancing age, male sex, and white race.

• Pt require surveillance endoscopy due to increased risk of adenocarcinoma
  • Absolute risk of cancer is ~0.005 cancer/patient annually
AGA Guidelines 2011

• Screen for Barrett’s in pt with multiple risk factors
  – Male
  – >50 yrs old
  – White
  – Chronic GERD, hiatal hernia
  – Obesity (increased BMI), intra-abdominal distribution

• AGA is against screening general population with GERD
AGA cont...

- Pts with Barrett’s
  - GERD therapy to heal esophagitis
- Dysplasia should be confirmed by second expert pathologist
- Surveillance:
  - No dysplasia → 3 to 5 years
  - Low-grade dysplasia → 6 to 12 months
  - High-grade dysplasia in the absence of eradication therapy → 3 months
Esophagus question

31-year-old female presents with 6 months of dysphagia and recurrent chest pain. She has difficulty swallowing after every meal. Dysphagia has progressed to solids and liquids. She had a previous fundoplication. EGD showed it intact. Manometry showed no peristalsis, high LES pressure and no relaxation with wet swallows. The most likely diagnosis?

a. Failed reflux surgery
b. Nutcracker esophagus
c. Nonspecific motility disorder
d. achalasia
Answer ____________________

- Findings classic.
- EGD did not show malignancy (pseudoachalasia)
- May have been misdiagnosed before fundoplication
Achalasia

• Rare disease
• Loss of ganglion cells with the myenteric plexus
• Cause?
  – Increasing evidence suggests
    • Autoimmune process
      – Attributable to latent infection with herpes simplex 1 combined with genetic susceptibility
Differential

• DES (diffuse esophageal spasm)
• Chagas Disease
  – Protozoan *Trypanosoma cruzi*
• Classic xray: bird-beak
Esophagus question

- 25-year-old man presents with a history of intermittent food impaction for which he has had 2 previous EGDs. Esophagus is reported as normal appearing. Dysphagia is to solids only. He now presents with his 3\textsuperscript{rd} impaction. Your next step in management would be:
  a. Repeat EGD, with random biopsies
  b. Manometry testing
  c. Empiric trial of PPI
  d. Empiric trial of steroids
  e. Reassurance and dismiss
Answer:_________________

• History suggests eosinophilic esophagitis
• Manometry unlikely to yield a cause
• Some pts respond to steroids, but a diagnosis is needed 1st.
  – Topical steroid
  – Elimination diet
  – Caution with dilation
Eosinophilic Esophagitis

• Esophageal symptoms

• **Presence of 15-20 or more eosinophils/high-power field**

• Exclusion of GERD
  – PPI trial
A 62 yo man presents with reflux-predominant dyspepsia and no alarm signs or symptoms

• What would you do?
  a. EGD
  b. Test for *H. Pylori*
  c. Prescribe PPI
Answer:_____________

• According to guidelines:
  – Over the age of 55 or alarm symptoms
    • Unintended weight loss
    • Dysphagia
    • GI bleeding
    • Iron def anemia
    • Abnormal physical exam (?mass in abdomen)
Esophageal Question

• 16 yo presents with sudden onset of odynophagia after waking in the am. She is fairly healthy with hx of exercise induced asthma and acne. Her only meds are albuterol prn and doxycycline. What is the likely diagnosis?

  a. Schatki ring
  b. Hiatal hernia
  c. Pill esophagitis
  d. Infectious esophagitis
Answer: ________________

• Most common meds that can cause pill esophagitis:
  – Alendronate
  – Aspirin
  – Doxycycline
  – Indomethacin
  – Iron
  – Potassium
  – Quinidine
Malabsorption

• Carbohydrate
  – Increased gas, distention and possibly diarrhea
  – Get extensive dietary hx to r/o dietary causes
  – A fecal pH less than 6 is evidence of carbohydrate malabsorption
  – Hydrogen breath test
    • Increase of 20 parts/million → colonic fermentation of the lactose by bacteria
    • False + can occur in bacterial overgrowth
    • False – recent antibiotic use
Malabsorption

Fat

– 72-hour collection; normal is 6-8g/24 hours
– High fat intake (100g/d) necessary for accurate results

Protein

– Rare
– Alpha-1 antitrypsin clearance
Celiac disease “gluten enteropathy”

- genetically inherited associated with the HLA locus found on the short arm of chromosome 6. HLA-DQ2 is present in 95% of patients.
- pathology: flattening of the small bowel villi.
Diagnosis—screening tests

• Antigliadin antibody (AGA)
  - IgG good sensitivity (83-100%)
    - False + in cow milk protein intolerance and parasite infection.
  - IgA good specificity (72-100%)

• Antiendomysial antibody (AEA or EMA)
  - False – in IgA deficiency and kids <2 years.
  - Sensitivity 97-100%; specificity 98-99%
Diagnosis- Screening Tests

- **Tissue transglutaminase Ab (IgA)**
  - Single preferred test
  - Sensitivity 90-100%
  - Specificity 95-100%

- **Antireticulin Ab**
  - Not very sensitive (not used)

**check IgA**
Celiac diagnosis

• Biopsies should be performed in all pts who are suspected of having celiac, regardless of serologic evidence
  – Small # of people have + serology but normal biopsies
  – Negative serology does not preclude presence of disease
Complications

• Refractory sprue
  – Severe complications can develop
    • Ulcerative jejunitis, collagenous sprue, lymphoma

• T-cell lymphoma
  – High mortality rate
Pt with celiac has followed a gluten free diet for 6 months and was doing well but now diarrhea has returned. Review of her diet shows compliance. Endomysial antibody testing is now normal. What is the next step?

a. Repeat small bowel biopsy
b. SBFT
c. CT scan of abdomen
d. Colonoscopy with biopsies
e. Bacterial aspirate if small bowel contents
Answer:_________________

• Pt had responded to gluten-free diet
• Common cause of recurrent diarrhea is microscopic colitis, detected with random biopsies in a normal appearing colon.
• About 15% of time the 2 diseases coexist.
52 yo recently traveled to Puerto Rico for 3 months. He developed fatigue, malaise and abdominal cramps 1 week after returning, followed by diarrhea and dyspepsia. Stools are “oatmeal-like”. Lab Hgb 11.3 with MCV 103. Stool studies neg. Enteroscopy is performed. Likely diagnosis?

a. Celiac sprue
b. Giardia
c. Tropical sprue
d. Lactase def.
Answer: __________________

- Can mimic celiac sprue.
- Etiology unknown although it is suspected to be infectious.
- Tx: tetracycline 250mg QID and folate 5mg daily for 6-12 months

KEY:
Diarrhea+ tropics+ macrocytic anemia = Tropical sprue
Types of Lactase deficiencies

• congenital form: Present at birth, very rare.
• primary form: genetically determined and dependent on population (most common in Saharan and sub-Saharan Africa and East Asian and Pacific)
• secondary or acquired: occurs after intestinal injury.
A 35-year-old female presents for a second opinion concerning IBS

She states 2 years ago she was in Mexico and got severe diarrhea that got better with antibiotics. However, since that time she experiences increased abdominal bloating, cramps, and diarrhea after eating. She has kept a food diary and you notice her symptoms are worse after she has cereal, pizza, or chocolate. What is her likely diagnosis?

• a. Secondary lactase deficiency
• b. Congenital lactase deficiency
• c. Celiac Sprue
• d. Irritable bowel syndrome
Answer:_________________________________________
Bacterial Overgrowth

• A direct consequence of the presence of increased amounts of colonic-type bacterial flora in the small intestine.
• Can result in fat, carbo, and protein malabsorption
• Macrocytic anemia → cobalamin def.
Bacterial Overgrowth Etiology

• Small bowel diverticula
• Fistulas from crohn’s
• Bypass of intestine
  – Jejunoileal for obesity
• Functional stasis
  – Scleroderma
  – Diabetes
Bacterial Overgrowth diagnosis

• direct aspiration of aerobes and anaerobes from small bowel is the standard.
• Alternatives: carbon dioxide and hydrogen breath tests.

**Therapy:** If the cause is not correctable, can trial antibiotics.
largely empirical
Small Bowel Question
A 29 yo develops a mild watery diarrhea after camping in the Cascade Mountains of Washington. He drank water from the mountain streams. On PE he has no abdominal pain or masses. He is afebrile, +BS. Stool neg for occult blood. Diarrhea abates after 3 weeks. His children have similar symptoms. Which of the following infectious agents most likely caused his disease?
a. Rotavirus
b. *Shigella flexneri*
c. *Vibrio cholerae*
d. *Giardia lamblila*
e. *Salmonella enteritidis*
Answer:________________
Giardia

• Intestinal tract infection caused by protozoal parasite *Giardia lamblia*.

• Predominant age in US:
  – Preschool; especially daycare
  – Homosexual men

• Contaminates fresh water sources worldwide
  • (mountain streams)
Giardia cont.

- Clinical findings:
  - 70% have intestinal symptoms
    - Diarrhea
    - Flatulence
    - Cramps
    - Bloating
    - Nausea
  - Chronic diarrhea, malabsorption, and weight loss
  - 20-25% of infected pts are asymptomatic
Workup

• Stool
  – Immunoassays for Giardia antigen routinely used in labs

• r/o malabsorption
  – B12
  – Albumin
  – Stool fat test

• Tx:
  – Metronidazole, Nitazoxanide, Paromomycin
Small Bowel

45 yo has had malabsorption for the last year with low volume diarrhea, polyarthritis and occasional visual hallucinations. PE is neg. CT shows generalized lymphadenopathy. EGD shows broad flattened villi in the duodenum. Bx show numerous PAS + macrophages in the submucosa. Which therapy may be useful for this pt?

a. Gluten free diet
b. Steroids
c. Antibiotics
d. Antacids
Answer:_________________

• Whipples disease
  – *Trophera* *m*whippelii
  – May get a description of “foamy macrophages”

• Most common sx: arthralgias, weight loss, recurrent abd pain, diarrhea. CNS manifestations also classic
Water soluble vitamins

• B12
  – Cobalamin
    • Requires ingestion of animal products
    • Deficiencies:
      – Megaloblastic anemia and hyperhomocystinemia
        » Identical to folic acid def
      – Neuropsychiatric abnormalities
      – Posterior column of spinal cord degeneration
      – Loss of taste, anorexia, diarrhea
  – Serum methylmalonic acid levels
    • Normal in folate def
    • Increased in B12 def (or before)
B12 deficiency causes

- Achlorhydria in elderly
- Perncious anemia
  - Lack of IF and acid
- ZE syndrome
- Bacterial overgrowth
- Gastric bypass
Other water-soluble vitamins

- Folic acid: Macrocytic anemia, glossitis, increased risk colon ca and CV disease
- Vit C: scurvy
- Thiamine (b1): beriberi with cardiac or neurologic disorders. May be exacerbated by glucose administration to thiamine-def patients
- Riboflavin (b2): angular stomatitis, cheilosis, dermatitis, visual impairment
- Niacin (b3): pellagra (diarrhea, dermatitis, dementia)
- Pyridoxine (b6): def cause of increased ratio of AST to ALT in alcoholic hepatitis
Fat-soluble vitamins

- A: night blindness
- D: nausea, vomiting, weakness
- E: neurologic symptoms, hemolysis
- K: excessive bruising and bleeding
- Absorption requires luminal bile salts and pancreatic esterases, assembly into chylomicrons and lymphatic transport
- Excess can cause toxicity
Minerals

• Iron
  – Microcytic hypochromic anemia

• Zinc
  – Required as a cofactor for many enzymes
  – Def impairs growth, development and reproductive and immune functions

• Copper
  – Microcytic hypochromic anemia, leukopenia, neutropenia, diarrhea and bony changes
Minerals

• Selenium
  – Cardiomyopathy
  – Myositis

• Manganese
  – Night blindness, tachycardia, tachypnea, HA

• May develop in pts receiving long-term TPN or TF
Matching Vitamin/Mineral def with symptoms/signs

- Vitamin A
- Vitamin C
- Vitamin K
- Zinc
- Chromium
- Copper
- Glucose intolerance
- Night blindness
- Hypogonadism
- Impaired wound healing
- Menkes (kinky hair) syndrome
- Easy bruising
Answers

• Vitamin A: night blindness
• Vitamin C: impaired wound healing, bleeding gums, depression
• Vitamin K: easy bruising, bleeding
• Zinc: hypogonadism, growth arrest, poor wound healing
• Chromium: glucose intolerance
• Copper: Menkes syndrome, microcytic anemia
Small bowel question

• What is the most consistent laboratory abnormality that may occur in small bowel bacterial overgrowth?
  a. Vitamin K malabsorption
  b. Gapped PT/INR
  c. Vitamin C Malabsorption
  d. Vitamin B12 malabsorption
  e. Iron deficiency anemia
Answer:_________________________
Alcoholic Liver disease

• Risk Factors for Alcoholic Liver Disease
  – Amount of alcohol consumed
  – Duration of alcohol consumption
  – Gender
  – Viral hepatitis
  – Nutrition
  – Iron overload
  – Genetics
3 Types of Liver Damage

1. Fatty Liver
2. Alcoholic hepatitis
3. Cirrhosis
Alcoholic Hepatitis

• Typically seen in malnourished patients
• Frequently precipitated by a period of binge drinking
• Prodrome: (2-3 weeks)
  – Anorexia
  – Nausea
  – Fatigue
  – Weight loss
Alcoholic Hepatitis

- Persistence of Alc. Hep. is associated with relentless progression to cirrhosis over months to years.
- Complications can be identical to those of cirrhosis.
- Poor prognostic signs:
  - Advanced age, jaundice, azotemia, and coagulopathy.
Alcoholic Hepatitis

• Clinical manifestations
  – Hepatomegaly, mild fever, jaundice
  – More severe cases: ascites, encephalopathy

• Lab
  – Increased AST&ALT ➞ not more than 10x normal
  – Increased AST/ALT ratio (2-3:1)
  – Decreased albumin
  – Prolonged PT
Alcoholic hepatitis-treatment

• Abstinence

• Bedrest

• Nutrition

• +/- steroids
Liver question

• What is most commonly used to assess the prognosis of patients with alcoholic hepatitis?
Answer: Maddrey discriminant function analysis

• Discriminant function = 4.6(prothrombin time-control) + serum bilirubin (mg/dL)

• Discriminant function >32 effectively identifies patients whose risk of death is higher than 50%
  – Consider steroids
Nonalcoholic Fatty Liver Disease

• Clinical
  – Nonalcoholic (<20g alcohol/day)
  – Exclusion of viral, autoimmune, genetic, and drug-induced liver disease.

• Nonalcoholic Steatohepatitis (NASH)
  – Chronic inflammatory condition in people who don’t have significant alcohol history.
    • Characteristics: steatosis, hepatocellular necrosis, and inflammation.
Nonalcoholic Fatty Liver Disease

- clinical manifestations
  - Central obesity (apple shaped not pear-shaped)
    - Abd. Obesity (waist >40” in men and 34.5” for women)
  - NIDDM
  - +/- hyperlipidemia
  - Most patients are asymptomatic
  - Occasional RUQ discomfort, malaise, fatigue
  - Hepatomegaly→ 75% of patients
Nonalcoholic Fatty Liver Disease

• Lab
  – Elevated aminotransferase (<300UI/L)
  – AST/ALT ratio <1
  – Mild elevation alkaline phosphatase and GGTP
Nonalcoholic Fatty Liver Disease

• Diagnosis
  – Findings of fatty infiltrate on imaging studies.
  – Exclusion of other liver diseases by history, physical, and serology.
  – Alcohol consumption should be <40g/week.
  – Liver biopsy is the definitive method of diagnosis. Not indicated in asymptomatic patients with normal AST, ALT.
Nonalcoholic Fatty Liver Disease

• Histologic finding
  – Steatosis-macrovesicular mild to severe
  – Inflammation
  – Hepatocyte injury—focal necrosis and ballooning
  – Hepatocyte degeneration—mallory hyaline
  – Fibrosis—varying degree
Nonalcoholic Fatty Liver Disease

• Management
  – Directed at associated risk factors.
  – Gradual weight loss.
  – Control of hyperglycemia and hyperlipidemia.
  – Discontinue suspected meds.
  – Alcohol use <20g/day. Alcohol abstinence if significant fibrosis
  – HAV and HBV vaccination
  – Avoid drugs that may promote steatohepatitis (amiodarone, tamoxifen)
Viral Hepatitis
Hepatitis B

A DNA virus.

• Risks in US: sexual promiscuity and IVDA
  – Many immigrants likely contracted at birth or young childhood

• Prevention:
  – Hep B immune globulin should be given to household and sexual contacts of patients with acute hepatitis B.
  – Infants and previously unvaccinated should receive hep B vaccine.
Hep B Serologic Markers

1. HBsAG
2. Anti-HBs
3. IgM anti-HBc
4. IgG anti-HBc
5. HBeAg and/or HBV DNA > 10^5 viral copies/mL

1. Current infection
2. Immunity (immunization or resolved infection)
3. Recent infection, occasionally reactivation
4. Remote infection
5. Active viral replication
Interpretation of Hep B serologic panel- examples

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<th>Chronically infected</th>
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Hep B

• Treatment –when?
  – If pt at increased risk of progression:
    • LFTs >2x normal,
    • active viral replication (HBV DNA increased),
    • and active disease identified in liver biopsy specimens
Hep B treatment

• Interferon
  – Pegylated—once weekly and better efficacy

• Oral agents
  – Lamivudine, Adefovir, Entecavir
  – Become popular for treatment of chronic hepB
  – Few side effects
    • Adefovir → nephrotoxicity
  – Useful in pts with decompensated cirrhosis
Hepatitis C

Leading indication for liver transplantation

• Diagnostic tests
  – anti-HCV: indicates current infection or previous exposure with clearance.
  – “gold standard” presence of HCV RNA by PCR—now the preferred test, bypassing RIBA.

• Level of RNA does not correlate with severity of disease.

• Genotyping: genotype 1 less likely to respond to treatment (most common in US)
Hepatitis C

Natural history and clinical presentation

• 60-85% develops chronic disease.
• Rarely do pts present with acute hepatitis
• Some pts have fatigue and mild RUQ pain
• 20% of pts with chronic Hep C will progress to cirrhosis.
Hepatitis C

Subgroup of pts likely to develop progressive liver disease

1. duration of infection
2. alcohol intake >50g/d
3. coinfection with HIV or HBV
4. male sex

Pts with cirrhosis due to HCV generally have disease >20 years.
Hepatitis C

Treatment

• Should be given to pts at highest risk of developing cirrhosis.

• Pegylated interferon with ribavirin is the standard of care
  – 24-48 weeks depending on genotype

(new and evolving treatments—too early for boards)
Hepatitis D

• A defective virus
  – Requires the presence of HBsAg to replicate
Hepatitis E

- Single stranded RNA
- The highest incidence of HEV infection is in Asia, Africa, Middle East, and Central America.
- HEV is the second most common cause of sporadic hepatitis in North Africa and the Middle East.
Hepatitis E

• HEV is spread by fecally contaminated water in endemic areas

• Person-to-person transmission is uncommon

• HEV can be transmitted by blood transfusion, particularly in endemic areas
Portal hypertension

• an increase in hepatic venous pressure gradient.

• In cirrhosis it occurs through an increase in resistance to portal venous outflow
  – Due to distortion of liver
  – ~30% of the increase is thorough potentially reversible vascular factors---where pharmacotherapy targets
Esophageal varices

- risk factors for hemorrhage from esophageal varices:
  - radius of varix,
  - thickness of varix wall
  - pressure gradient between the varix and the esophageal lumen.
Esophageal varices

- Recommendations for treatment of Esophageal varices
- Primary prophylaxis: all patients with cirrhosis should have EGD for screening.
  - If no varices repeat endoscopy in 2-3 years.
  - 1st line therapy: nonselective beta blockers (propranolol or nadolol)
  - 2nd line therapy: endoscopic band ligation
- Control of bleeding: best managed by endoscopic means preferable band ligation.
  - begin octreotide, continue for up to 5 days.
  - 2nd line therapy: TIPS
Esophageal varices

• Secondary prophylaxis: prevent rebleeding. Essential—80% of patients who bleed will have a rebleed within 2 years.

  1st line therapy: endoscopy and beta blockers.

  other: transplantation
Ascites

• Pathogenesis: renal retention of sodium and movement of this extra fluid into the peritoneal space.
• diagnostic paracentesis is essential for patients who present with ascites.
  – the difference between serum albumin and ascitic albumin help determine portal hypertension (1.1g/dL or greater). Could be liver or heart disease.
  – A protein of 2.5g/dL or more favors heart disease.
• cell count of more than 250 neutrophils/mm3 is spontaneous bacterial peritonitis (SBP).
Management of Ascites

- low sodium diet
- fluid restriction: only necessary if serum sodium is <125mEq/L
- diuretic therapy:
  - urinary sodium excretion is used to determine the efficacy of therapy.
    - If urinary sodium excretion is more than 30mEq/d, spironolactone alone may be used.
    - If urinary sodium excretion is between 10-30mEq/L then a combination of spironolactone and furosemide is used.
    - If urinary sodium excretion is < 10mEq/L then large volume paracentesis is usually required.
Hepatic encephalopathy

• Pathogenesis:
  – Ammonia and manganese considered etiologic factors for encephalopathy.

• Clinical features:
  – range from 0—no overt encephalopathy to IV patient in a coma.

• Precipitating factors:
  – GI bleed, infection, large protein meal, use of sedatives, electrolyte abnormalities or hypoxia, constipation, and hypoglycemia.
Hepatic encephalopathy - Management

• dietary: limit protein based on level of encephalopathy.
  – Long-term restriction of dietary protein of < 1g/kg daily should be avoided.

• Nonabsorbable disaccharides:
  – Lactulose, may help remove dietary and endogenous ammonia.
    • Pt should have 2-3 semifomed stools/day.

• Antibiotics:
  – neomycin, metronidazole, and rifaximin have been used for treatment.
Liver question

A definite indication for antibiotic prophylaxis with norfloxacin for spontaneous bacterial peritonitis in pts with cirrhosis is:

a. Pts with ascites undergoing endoscopic sclerotherapy
b. Gi bleeding without ascites
c. Ascites with gram- urinary infection
d. Ascites with albumin concentration <1g/dL
e. Pts with ascites undergoing esophageal dilatation
• 2 clear indications of SBP prophylaxis
  – Pts with GI bleed with or without ascites
  – Pts with previous SBP
  – In up to 50% of pts with bleeding, gram- infections occur at 1 week and are associated with rebleeding risk and mortality.
  – Ascitic fluid albumin concentration <1g/dL is not a definite indication for antibiotic prophylaxis.
Hemochromatosis

- autosomal recessive disorder with increased intestinal absorption of iron.
- Excess iron is deposited in the liver, pancreas, and other organs.
- About 1 in every 250 white persons in the US is homozygous for the mutation.
Hemochromatosis

- Suspect in pts with elevated iron sat, ferritin, or family hx.
- Most pt asymptomatic
- Cirrhosis, heart failure, hypogonadism, and arthritis
- HFE gene mutation
  - Autosomal recessive dz
    - 85% homozygous for C282Y mutation
Hemochromatosis-Treatment

• reserved for patients with evidence of iron overload, indicated by an increase in the serum concentration of ferritin.
  – therapeutic phlebotomy: simple, relatively inexpensive and effective.
  – avoid supplements with iron
  – avoid raw fish due to risk of *Vibrio vulnificus* infection
  – avoid alcohol

• If diagnosed and treated before diabetes and cirrhosis develops survival rate is normal
Wilson’s disease

• Diagnosis
  – Reduced ceruloplasmin
  – Increased urinary excretion of copper
  – Presence of K-F rings
  – Elevated hepatic copper level

• Treatment
  – Copper-chelating medications
Liver question
A 38 yo female presents with increased LFTs. She has no history of alcohol, drugs use or transfusions. She takes Tylenol once a month for headaches. Her exam is negative and she has no pain.

- Lab: AST 980
- ALT 1030
- Alk phos 185
- Total Bilirubin 1.0
What test will help confirm her diagnosis?

a. MRI
b. ERCP
c. ANA (antinuclear Antibody)
d. CBC
e. PT (prothrombin time)
Answer: ________________

• Pt likely has autoimmune hepatitis
• Interface hepatitis (periportal hepatitis or “piecemeal necrosis”) is the histologic hallmark
• Cause unknown
• 70% are women and 40% are under age of 40
Autoimmune Hepatitis: Typical lab

- Increased AST and Alt 100%
- Increased gamma globulin and IgG 90%
- Mild hyperbilirubinemia 83%
  - <3 mg/dL
- Alkaline phosphatase increase 67%
  - <2x normal
- ANA, SMA, or anti-LKM1 87%
Primary Biliary Cirrhosis

- Cholestatic liver disease
- 90% women
- 95% will be AMA + (anti-mitochondrial Ab)
- Fatigue common
- Pruritis 30-50%
- Frequently being picked up in pts with asymptomatic lab abnormalities
- IgM high
DDx for cholestasis without biliary obstruction

- Drug-induced cholestasis
- Primary biliary cirrhosis
- Primary sclerosing cholangitis
- Idiopathic adulthood ductopenia
- Idiopathic biliary ductopenia
- Cholestasis of pregnancy
- Cystic fibrosis
- HIV-associated cholestasis
- Sarcoidosis
- Granulomatous hepatitis
Alpha1-antitrypsin (AAT) deficiency

- Autosomal codominant disorder with lung and liver injury
- Can cause premature emphysema and liver disease
- Pt with cirrhosis due to AAT have a significant increased risk of HCC up to 30%
- Diagnosed by phenotyping. Liver damage does NOT correlate with serum AAT levels (unlike lung). Diagnosis confirmed with biopsy
- No effective medical treatment for the liver manifestations of AAT deficiency.
GI Surgery Review
Surgery for GERD

• Reserved for pts:
  – Complications from GERD
  – Refractory esophagitis**
  – Stricture
  – Barretts
  – Persistent “reflux symptoms” despite acid suppression
  – Asthma

** most frequent
Preop Evaluation

• No consensus
• Useful tests in making surgical decisions
  – Egd
  – Esophageal manometry
  – 24-48 hour pH probe
Antireflux surgery

• For most pts with GERD laparoscopic Nissen fundoplication
  – Several advantages with similar efficacy and safety as an open procedure
Post-op Symptoms

• Dysphagia
  – Occurs in most pts
  – dilatation

• Gas bloat
  – Most pt improve over time
  – Mild → simethicone or charcoal tablets, avoid carbonation
  – Trial of metoclopramide
  – Persistent symptoms consider gastroparesis
Long-term efficacy

• Laparoscopic fundoplication
  – 90-95% of patients satisfied with the results
    • Experienced surgeons
Surgery for PUD

Indications

• Failure of nonoperative management of ulcer complication

• Suspicion of malignancy (usually gastric ulcer)
Operation for duodenal ulcer

• based of reduction of acid secretion
  – Sectioning of vagus (vagotomy)
  – Eliminating hormonal stimulation from the antrum (antrectomy)
  – Decreasing the number of parietal cells (gastric resection)
Operation for gastric ulcer

• Difference from duodenal is that gastric ulcer may harbor malignancy and therefore must be excised or generously biopsied.
Postgastrectomy syndromes

- Postvagotomy diarrhea
- Dumping syndrome
- Alkaline reflux gastritis
- Early satiety
Post vagotomy diarrhea

- 30% of pts
- Most self limiting
- Pathogenesis poorly understood
  - Rapid passage of unconjugated bile salts
- Oral cholestyramine
Dumping syndrome

- ~20% pts after gastrectomy or vagotomy and drainage

- Symptoms:
  - Postprandial GI discomfort
  - +/- nausea, vomiting, diarrhea and cramps
  - Vasomotor symptoms
    - Diaphoresis
    - Palpitations
    - flushing
Dumping syndrome

• Precise mechanism not completely understood

• Attributed to rapid emptying of hyperosmolar chyme (particularly carbs) into the small bowel
  – Leads to net fluid retention
  – Leads to vasoactive hormone release
    • Serotonin and VIP
Dumping syndrome

• Treatment
  – Dietary changes
  – Rarely operative therapy needed
  – Octreotide may help with severe symptoms
Gallbladder
Acute cholecystitis

• Typical presentation
  – RUQ pain
  – Fever
  – Leukocytosis

• Associated with gallbladder inflammation,
  – Usually due to gallstone disease

• Complications (can be life-threatening)
  – Gangrene
  – Gallbladder perforation
Acute cholecystitis-treatment

• Supportive

• Antibiotics
  – Secondary infection from cystic duct obstruction and bile stasis
  – Guidelines
    • Start antibiotics if infection suspected based on:
      – Lab (WBC >12,500)
      – Clinical (temp >38.5C)
      – Radiographic findings (air in gallbladder or wall)
      – Advanced age, diabetes, immunodeficiency

Infectious Diseases Society of America
Timing of surgery

• Asymptomatic gallstones should not be treated

• Low risk pts with clinical improvement
  – Elective cholecystectomy same hospitalization

• Low risk pts with deterioration
  – Emergent cholecystectomy

• High risk (ASA 3 and >) mortality 5-27%
  – Clinical deterioration– percutaneous cholecystostomy
Complication of Laparoscopic cholecystectomy

- Serious complications
  - Result in part from patient selection
  - Surgical inexperience
  - Technical constraints of minimally invasive approach
Bile duct injury

• Classified A-E based on type of injury
• Repair should always be approached by an experienced multidisciplinary team
  – Surgeon
  – Diagnostic radiologist
  – Interventional gastroenterologist
  – Interventional radiologist
Biliary leakage

- Suspect in pts with fever, abdominal pain, bilious ascites
- Large loculated collections
  - Percutaneous drainage, with catheter left in place for drainage
  - ERCP: define leak and place stent
- Severe pain, progressive intraabdominal sepsis
  - Operative exploration and washout
Other complications

• Bleeding
• Bowel injury
• Postcholecystectomy syndrome
  – Complex of symptoms including
    • Abdominal pain
    • dyspepsia
Bariatric Surgery

Early Complications
- Bleeding
- Wound infection
- Leaks
- PE/DVT
- CV complications
- Pulmonary complications

Late Complications
- Roux-en-Y
- gastric remnant distension
- stomal stenosis
- marginal ulcers
- cholelithiasis
- ventral incisional hernia
- internal hernia
- short bowel syndrome
- Dumping syndrome
Post Op Ileus
Definition

• Transient inhibition of normal GI motility in the post op setting.
• Presumably, the muscle of the bowel wall is transiently impaired and fails to transport intestinal contents.
• Typically lasts 3-5 days.
Clinical Consequences

- Worse pain
- Nausea and vomiting
- Delay in enteral nutrition
- Prolonged hospitalization
- Increased risk of complications
- Increased health care costs
Pathophysiology

- Poorly understood

1. Neural reflexes involving the sympathetic nervous system may inhibit motility
   1. Epidural anesthetic agents decreased duration of post op ileus.
   2. Due to blockade of neural reflexes at the spinal cord level.
Pathophysiology

2. Local and systemic inflammatory mediators may play a role.
   1. NSAIDs decrease POI
3. Exacerbating factors
   1. Opioid analgesics
   2. Intraperitoneal surgery
   3. Degree of bowel manipulation
   4. Open vs. laparoscopic surgery
   5. hypokalemia
Clinical Presentation

- Abdominal pain
- Nausea/vomiting
- Anorexia
- Abdominal bloating/distension
- Absent bowel sounds
- Lack of passage of flatus or stool
- Tympanic abdomen
- No visible peristalsis
Clinical Presentation

• Pain is typically mild and constant
  – Mechanical obstruction usually severe
Physical exam

- Lack of bowel sounds
- Increase abdominal girth
- Lack of visible peristalsis
- Tympanic abdomen
- Xray: air-fluid levels or nonspecific patterns
Treatment

• Most cases
  – Watchful waiting and supportive care
  – Hydration
  – If nausea and vomiting--NGT
Treatment-Pharmacologic

• Metoclopramide, cisapride, erythromycin
  – RCT don’t show benefit

• Laxatives
  – Possible benefit

• Opiate antagonists
  – May show benefit, but more studies needed
Treatment-Pharmacologic

• Epidural anesthesia
• NSAIDs
  – Probable benefit
  – Need to be cautious of SE
• Multimodality therapy
Treatment-Nonpharmacologic

- Nasogastric tube
  - No evidence of benefit, may increase pulmonary complication.
- Early enteral nutrition
  - Appears safe and well tolerated.
- Early mobilization
  - No change, but may decrease other complication
- Chew gum
- OMM