GI Review
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Diseases of the Esophagus
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10:45-11:15am

1. Gastroesophageal Reflux disease
   A. Reflux that produces frequent symptoms or results in damage to the esophageal mucosa or contiguous organs
   B. Etiologies
      i. Motility disorders
         1. transient lower esophageal relaxation**
         2. weak lower esophageal sphincter**
         3. weak esophageal peristalsis
         4. scleroderma and CREST
         5. delayed gastric emptying
      ii. Damaging factors
         1. increased gastric acid production
         2. bile and pancreatic juice
      iii. Resistance factors
         1. reduced saliva and HCO3 production
         2. diminished mucosal blood flow
         3. growth factors, protective mucus
      iv. Others
         1. hiatal hernia**
         2. obstructive sleep apnea
   **major/common factors

C. Epidemiology of GERD
   i. 40% of adult population in US has heartburn monthly
   ii. 18% report heartburn weekly
   iii. complications may be more common in males.

D. Symptoms
   i. esophageal: heartburn, acid regurgitation, odynophagia, dysphagia, chest-pain, water brash
   ii. Airway symptoms: cough, wheezing, hoarseness, throat clearing, globus, tracheal stenosis, aspiration pneumonia, pulmonary fibrosis

E. Diagnostic Tests
   i. Not necessary for most patients with typical GERD.
   ii. Alarm symptoms warrant further evaluation
   iii. About 50% of patients with typical GERD symptoms have normal endoscopy.
iv. Endoscopy will identify esophageal complications of GERD
   1. esophageal ulcer, Barrett’s esophagus, and esophageal adenocarcinoma.
   3. Higher risk for esophagitis and complications: male, middle age, and nocturnal heartburn.

v. Upper GI x-ray
   1. Major usefulness: identify strictures and large hiatal hernias.
   2. Insensitive: detecting erosions or superficial mucosal changes.
   3. sensitivity for GERD only 20%

vi. Ambulatory Esophageal pH
   1. Indications: atypical symptoms, frequent atypical chest pain, refractory symptoms in well-established GERD, preoperative confirmation of GERD.

F. Treatment Options
   i. lifestyle modifications---healing rate 20-30%
   ii. Acid neutralization----healing rate 20-30%
   iii. Acid suppression
      1. H2 blockers---healing rate 50%
      2. PPI---healing rate >80%
   iv. Prokinetics----healing rates 30-40%
   v. Mechanical prevention of reflux
      1. laparoscopic surgery----healing rate >80%
      2. Endoscopic therapies----healing rate >50%

Question: What factors are associated with severe esophagitis?
Answer: low LES pressure, esophageal motor abnormalities, and recumbent reflux are the most common determinants. Presence of hiatal hernia is also important.

Question: In what situation should you consider diagnostic testing in patients with suspected GERD?
   1. Answer: Uncertain diagnosis
   2. Atypical symptoms (chest pain, pulmonary, ENT)
   3. Complications: dysphagia, wt loss, bleeding, anemia
   4. Inadequate response to therapy
   5. Recurrent symptoms
   6. Prior to anti-reflux surgery

2. **Barrett’s Esophagus**
   A. Acquired disorder where columnar epithelium replaces the stratified squamous epithelium that is normal in the esophagus.
   B. Criteria
      i. salmon-colored mucosa in the tubular esophagus.
      ii. biopsy specimens must show intestinal metaplasia with goblet cells.
   C. Epidemiology—complication of chronic acid reflux
      i. risks: increasing age, male sex, white race.
   D. Cancer risk
i. 40- to 125-fold higher risk of adenocarcinoma of the distal esophagus.
ii. absolute cancer risk is ~ 0.005 cancer per patient annually (quite low)
iii. degree of dysplasia is more important than length of Barrett’s esophagus in predicting neoplastic progression.

E. Surveillance
i. American College of Gastroenterology recommendations
   1. Initial endoscopic biopsies:
      - Any suspicious lesion
      - 4-quadrant biopsies every 2 cm along the length of the segment.
   2. Dysplasia?
      No \(\rightarrow\) repeat in one year. If continued no dysplasia can repeat EGD at 3-5 years.
      Low grade dysplasia: endoscopy every 6 months x2 then annually x 2. If there is no progression then 3-5 years.
      High grade dysplasia: need confirmation with 2 expert pathologists. Standard care for patients able is esophagectomy.

3. Esophageal Motility
   A. Achalasia “failure to relax”
      i. Pathophysiology
         1. a decreased number of nonadrenergic noncholinergic inhibitory ganglion cells.
         2. Similar disease pathologically: Chagas’ disease due to infection by *Trypanosoma cruzi*.
      ii. Clinical Presentation
         1. dysphagia to solids and frequently liquids.
         2. regurgitation
         3. heartburn from fermentation of food, not acid reflux.
         4. chest pain
         5. weight loss
         6. cough, pulmonary symptoms from aspiration.
      iii. Diagnosis
         1. Barium swallow: bird’s beak appearance: dilated esophagus with a tight LES.
         2. Manometry: aperistalsis, incomplete relaxation of LES after swallow, increased LES pressure.
      iv. Differential
         1. pseudoachalasia: from other diseases or malignancy.
            Need endoscopy to exclude malignancy.
      v. Management:
         1. drugs: limited benefit (calcium channel antagonists, nitrates)
         2. Botulinum toxin: injected into LES
         3. Pneumatic dilatation: major disadvantage: perforation rate is 3%.
         4. surgical myotomy
* patients with achalasia have a 2-7% increased risk of squamous cell carcinoma.

Esophageal Motility Disorders Summary:
Inadequate LES relaxation: achalasia (aperistalsis), atypical disorders
Uncoordinated contractions: diffuse esophageal spasm
Hypercontractile: nutcracker esophagus, isolated hypertensive LES
Hypocontractile: ineffective motility (nonspecific motility disorder)

4. **Eosinophilic Esophagitis**
a. unexplained solid food dysphagia or intermittent food impaction
b. common in young, typically men. Usually don’t have symptoms of GERD.
c. Endoscopy
   i. corrugated (ringed) esophagus, particularly in the mid esophagus.
   ii. biopsy: increased intraepithelial eosinophils (>20/high-power field)
d. Most patients respond to topical corticosteroids, but no treatment is evidence based at this point. Research is ongoing.
1. Malabsorption
   A. Carbohydrate Malabsorption
      i. increased gas, distention, and sometimes diarrhea.
      ii. fecal pH <6 or the presence of reducing substances in the stool is evidence.
      iii. Lactose: hydrogen breath test, an increase of 20parts/million is indicative of colonic fermentation
   B. Protein Malabsorption
      i. isolated disorders rare
      ii. alpha-1-antitrypsin clearance can be measured in protein-losing enteropathies
   C. Fat malabsorption
      i. 72-hour fecal fat excretion (24-48 hour may be adequate if stool frequency is great)
         normal excretion: 6-8 g/24hrs

Question: What are common signs and symptoms of malabsorptive disorders?
Answer: large volume diarrhea and associated weight loss. May also see fatigue from anemia, bloating and flatulence, bruising from Vit K deficiency, skin rashes, muscle wasting and paresthesias.

2. Small-bowel disease and Bacterial Overgrowth
   A. Celiac disease “gluten enteropathy”
      i. genetically inherited associated with the HLA locus found on the short arm of chromosome 6. HLA-DQ2 is present in 95% of patients.
      ii. pathology: flattening of the small bowel villi.
      iii. serology:
         Gliadin Ab: not very specific and may lead to unnecessary testing
         Endomysial Ab: more specific
         Tissue transglutaminase: similar specificity to endomysial.
         * a compatible biopsy with serologic confirmation indicates the need for treatment.
      iv. Treatment
         gluten restricted diet: wheat, rye, barley. Oats don’t contain gluten but may be contaminated.
         Pts with continued diarrhea and compliance to diet should be evaluated for microscopic colitis.
   v. Complications
      refractory sprue
      T-cell lymphoma
      Ulcerative jejunitis
      Collagenous sprue
Pts are at increased risk of esophageal cancer and oropharyngeal cancer and other autoimmune diseases.

**Question:** Describe the classic endoscopic appearance of the small intestine in celiac sprue.
**Answer:** scalloping, nodularity and/or absence of the small intestinal plicae (circular folds).

B. Whipple’s disease
   i. rare infectious disease
   ii. periodic acid-Schiff-positive macrophages in the gut mucosa
   iii. *Tropheryma whippelii*

C. Lactase Deficiency – 3 syndromes
   i. congenital form: Present at birth, very rare.
   ii. primary form: genetically determined and dependent on population (most common in Saharan and sub-Saharan Africa and East Asian and Pacific)
   iii. secondary or acquired: occurs after intestinal injury.

D. Bacterial overgrowth
   i. cause: proliferation of bacteria caused by interference with normal protective mechanisms (examples: afferent loop of gastrojejunostomy, scleroderma, gastrocolic fistula, diabetic autonomic neuropathy, chronic pancreatitis)
   ii. manifestations: malabsorption of fats, carbohydrates, and protein.
   iii. diagnosis: direct aspiration of aerobes and anaerobes from small bowel is the standard. Alternatives: carbon dioxide and hydrogen breath tests.
   iv. treatment: if cause is not correctable then antibiotics. No regimen has been proven better than others. Typical antibiotics: metronidazole, tetracycline, amoxicillin-clavulanic acid.

**Question:** What medications may result in malabsorption of vitamins and other nutrients?
**Answer:** antacids, mineral oil, cholestyramine, methotrexate, chemotherapeutic agents, phenytoin, orlistat, sulfasalazine, and sucralfate.
1. **Alcoholic Hepatitis**
   A. Risk factors for alcoholic liver disease.
      - Amount of alcohol consumed
      - Duration of alcohol consumption
      - Gender
      - Viral hepatitis
      - Nutrition
      - Iron overload
      - Genetics
   B. ALD- Drug Metabolism
      a. Alcoholics get increased liver injury from drugs
      b. Chronic alcohol ingestion alters metabolism and clearance of a variety of drugs
      c. Drug-Alcohol Interactions
         - Antibiotics: *Metronidazole*
         - Ulcer Meds: *Cimetidine*
         - Antidepressants: *Tricyclics*
         - CV drugs: *Nitro, propranolol*
         - Narcotics: *Morphine, meperidine*
         - Pain relievers: *Acetaminophen*
   C. 3 Types of Liver Damage
      a. Fatty Liver
         - Seen in moderate drinker and alcoholics
         - > 60g of alcohol/day (even for short periods)
         - Usually benign
         - About 10% will develop cirrhosis if they continue to drink
         - Other causes of fatty liver
            i. Symptoms
               - Frequently none.
               - If severe:
                  - Fatigue, weakness, malaise, anorexia, nausea, abdominal discomfort.
                  - Hepatomegaly very common-- 70%
            ii. treatment
               - Abstinence
               - Histologic changes return to normal within 2-4 weeks.
               - Good nutrition
      b. Alcoholic hepatitis
         - Typically seen in malnourished patients
         - Frequently precipitated by a period of binge drinking
         - Prodrome: (2-3 weeks)
            - Anorexia, Nausea, Fatigue, Weight loss
Persistence of Alc. Hep. is associated with relentless progression to cirrhosis over months to years. Complications can be identical to those of cirrhosis.

i. Poor prognostic signs:
   - Advanced age, jaundice, azotemia, and coagulopathy

ii. Clinical manifestations
   - Hepatomegaly, mild fever, jaundice
   - More severe cases: ascites, encephalopathy

iii. Lab
   - Increased AST&ALT \( \rightarrow \) not more than 10x normal
   - **Increased AST/ALT ratio (2-3:1)**
   - Decreased albumin
   - Prolonged PT

iv. Histology
   - Inflammation and necrosis
   - Varying degrees of fibrosis
   - Mallory bodies

v. Treatment
   - Abstinence
   - Bed rest
   - Nutrition
   - +/- steroids

c. Cirrhosis

Question: What % of alcoholics has antibody positivity to hepatitis C?
Answer: 40%

Question: What 3 variables independently increase the risk of progression of alcohol-related hepatic steatosis to cirrhosis?
Answer: continued alcohol consumption, severity of initial histologic injury and female gender.

2. NAFLD vs. NASH
   **Nonalcoholic fatty liver disease (NAFLD)**
   - Spectrum of liver disease, from simple fatty liver progressing to steatohepatitis, fibrosis, and cirrhosis

   **Nonalcoholic steatohepatitis (NASH)**
   - Subset of NAFLD involving inflammation and cellular injury.
   - Marked by histologic features resembling the pattern of injury seen in alcoholic liver disease.

A. Nonalcoholic Fatty Liver Disease
   a. Clinical
      - Nonalcoholic (<20g alcohol/day)
      - **One drink (12 ounces of beer, 5 ounces of wine, or 1.5 ounces of spirits) delivers about 12 to 14 g of alcohol**
Exclusion of viral, autoimmune, genetic, and drug-induced liver disease.
b. Nonalcoholic Steatohepatitis (NASH)
   i. Chronic inflammatory condition in people who don’t have significant alcohol history.
      *Characteristics: steatosis, hepatocellular necrosis, and inflammation.*
   ii. Should be suspected in people with persistently elevated aminotransferases in whom fat is present in the liver on imaging and viral serology is negative.
   iii. Pathogenesis
      Cause: unknown
      Risk factors: obesity, diabetes, hyperlipidemia, drugs, toxins, TPN, protein-calorie malnutrition, rapid weight loss, IBD jejunoileal bypass, HTN
      Many individuals with NASH can lack risk factors.
   iv. 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
   v. Lab
      Elevated aminotransferase (<300UI/L)
      AST/ALT ratio <1
      Mild elevation alkaline phosphatase and GGTP
   vi. 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.
   vii. Histologic finding
      Steatosis-macrovesicular mild to severe
      Inflammation
      Hepatocyte injury– focal necrosis and ballooning
      Hepatocyte degeneration– mallory hyaline
      Fibrosis– varying degree
   viii. Natural history
      Still being defined
      Long-term studies
      15-50% develops severe fibrosis
      7-16% develops cirrhosis
   ix. 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)

Question: What drug can result in the histologic picture of non-alcoholic steatohepatitis?
Answer: Amiodarone

3. Viral Hepatitis
A. Hepatitis B: A DNA virus.
   a. Risks in US: sexual promiscuity and IVDA
      Many immigrants likely contracted at birth or young childhood
   b. 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.
   c. Lab
      **HBV Antigens**
      HBsAg→1st serologic marker to appear after infection. General
      marker of infection.
      HBeAg→ indicates active replication of virus
      **HBV Antibodies**
      Anti-HBs→ Documents recovery from or immunity to HBV.
      Detectable after immunity conferred by HBV vaccine
      Anti-HBe→ marker of reduced level of replication.
      Anti-HBc (IgM)→ Marker of acute or recent HBV infection
      Anti-HBc (IgG)→ marker of previous infection
   d. Treatment
      If pt at increased risk of progression: LFTs >2x normal, active
      viral replication (HBV DNA increased), and active disease
      identified in liver biopsy specimens

B. Hepatitis C
   A factor in 40% of all cases of chronic liver disease and is the leading
   indication for liver transplantation.
   a. 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)
   b. 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.

c. treatment
Should be given to pts at highest risk of developing cirrhosis. Pegylated interferon with ribavirin is the standard of care. New, non-interferon treatments developing.

d. prevention: currently no vaccine is available.

Question: What is the major side effect of oral ribavirin therapy for HCV?
Answer: A dose dependent hemolytic anemia occurs within 2-6 weeks after beginning treatment with 15% of hemoglobin tested falling 4 grams when 1200mg/d is administered.

4. Cirrhosis
A. Portal hypertension: an increase in hepatic venous pressure gradient.
   i. in cirrhosis occurs through an increase in resistance to portal venous outflow.
   ii. hyperdynamic circulation and peripheral vasodilation maintain portal hypertension.
      a. octreotide and vasopressin reduce splanchnic hyperemia and portal venous inflow.
   iii. development of collateral circulation may decrease portal pressure.
   iv. risk factors for hemorrhage from esophageal varices: radius of varix, thickness of varix wall, and the pressure gradient between the varix and the esophageal lumen.
   v. Recommendations for treatment of Esophageal varices
      a. 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
      b. Control of bleeding: best managed by endoscopic means preferable band ligation.
         - begin octreotide, continue for up to 5 days.
         - 2nd line therapy: TIPS
      c. 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

B. Ascites: most common major complication in cirrhosis.
   i. Pathogenesis: renal retention of sodium and movement of this extra fluid into the peritoneal space.
ii. 80% of patients with ascites have cirrhosis, 10% is from malignancy, 5% have heart disease, 4% have mixed reasons for ascites, 1% have it due to conditions like TB, pancreatitis, chylous ascites.

iii. Diagnostic test
   a. 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).

iv. Management
   a. low sodium diet
   b. fluid restriction: only necessary if serum sodium is <125mEq/L
   c. 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.
   d. large volume paracentesis. Performed in pts with tense ascites.
      - usually 6-8 grams of albumin is given for each liter of fluid removed.

C. Hepatorenal syndrome
   i. Type 1: progression to renal dysfunction more rapid than type 2. Most patients have alcoholic hepatitis, SBP, or other infections.
   ii. Type 2: Pts with ascites have a decrease in GFR to less than 40mL/min or an increase in serum creatinine to more than 1.5mg/dL. Pt also have to have advanced liver disease and portal hypertension.
   iii. Evaluation: initial step → measure fractional excretion of sodium.
      a. if the value is < 1, the pt has prerenal azotemia, hepatorenal syndrome, or glomerulonephritis. Once prerenal and glomerulonephritis is ruled out the diagnosis can be made.

D. Encephalopathy
   i. Pathogenesis: Ammonia and manganese considered etiologic factors for encephalopathy.
   ii. Clinical features: range from 0—no overt encephalopathy to IV patient in a coma.
   iii. Precipitating factors: GI bleed, infection, large protein meal, use of sedatives, electrolyte abnormalities or hypoxia, constipation, and hypoglycemia.
   iv. Management
a. dietary: limit protein based on level of encephalopathy. Long-term restriction of dietary protein of < 1g/kg daily should be avoided.
b. Nonabsorbable disaccharides: Lactulose, may help remove dietary and endogenous ammonia. Pt should have 2-3 semiformal stools/day.
c. Antibiotics: neomycin, metronidazole, and rifaximin have been used for treatment. No benefit has been shown for long-term treatment with antibiotics.

E. Management of Spontaneous bacterial peritonitis
   i. cefotaxime 2 grams every 12 hours for at least 5 days. After 48 hours may repeat paracentesis, if neutrophil count decreases by more than 25%, then pt is having good response and the treatment is continued for a total of 5 days. If they are not responding then a CT scan should be performed to evaluate for secondary bacterial peritonitis.
   ii. albumin 1.5g/kg body weight on day one and 1g/kg on day 3. This has shown improved survival.
   iii. Once pt has had SBP they should receive prophylaxis long-term. Norofloxacin 400mg/d is recommended.

Question: How frequently do esophageal varices occur in patients with cirrhosis?
Answer: Nearly 80% with alcoholic cirrhosis develop varices within 10 years. The risk is somewhat less with Hep C.

Question: What is Budd-Chiari syndrome?
Answer: Obstruction of the hepatic veins. The blockage may be due to thrombosis or presence of an intraluminal membrane.

5. Hemochromatosis
   A. 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.
   B. the gene is HFE and located on the short arm of chromosome 6.
      i. 2 point mutations have been designated “C282Y” and “H63D.”
   C. Clinical features: usually not seen until 5th to 6th decade.
      i. classic description: cutaneous hyperpigmentation, diabetes, and cirrhosis.
      ii. most pts today are diagnosed when asymptomatic.
   D. Diagnosis: combination of clinical, lab, and pathologic criteria.
      i. lab: increase serum transferrin saturation and an increase in the serum ferritin.
         a. transferrin sat more than 45% is the earliest phenotype abnormality in hereditary hemochromatosis (HH).
         b. HFE gene test is most useful for surveillance of adult 1st degree relatives of an identified proband.
ii. Liver biopsy: iron is assessed with an iron stain such as Perl’s Prussian blue. In HH iron initially accumulates in periportal hepatocytes and eventually distributes throughout the liver.

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

F. If diagnosed and treated before diabetes and cirrhosis develops survival rate is normal
   i. 1/3 of pts with HH and cirrhosis develop liver cancer.

Remember not all iron overload is hemochromatosis.

6. **Wilson’s Disease**
   A. an autosomal recessive disorder characterized by abnormal intrahepatic copper metabolism and deposition of excess copper in the liver, brain, cornea, and other organs.
   B. Genetics
      i. gene is *ATP7B* located on chromosome 13
   C. Clinical features: median age at presentation is 12-23 years old. The oldest age 58 years.
      i. 5 main categories of clinical presentation: hepatic, neurologic, psychiatric (may be dramatic), hematologic (hemolytic anemia), and ophthalmologic (Kayser-Fleischer rings)
   D. Diagnosis: consider in any young person with liver disease.
      i. need at least 2 of the following: Kayser-Fleischer rings, low levels of ceruloplasmin, typical neurologic symptoms, and liver copper concentration greater than 250ug/g dry weight.
      ii. tissue analysis is “gold standard” for diagnosing Wilson’s disease.
   E. Treatment:
      Decoppering Agents
      i. Penicillamine
         a. can deplete B6 levels
      ii. Trientine—1st choice for treatment
   Inhibition of Copper Absorption
      i. Zinc acetate
   F. Compliance: if a patient stops treatment the probability of death from fuliment liver failure within 1-2 years is very high, even if the patient was initially asymptomatic.

7. **Autoimmune Hepatitis:**
   inflammation of liver of unknown cause.
   A. General: associated with interface hepatitis (piecemeal necrosis), hypergammaglobulinemia, and autoantibodies.
      i. female to male ratio: is 3.6:1.
ii. affected all ages, even infants.

B Clinical features: 70% of cases are women. 50% are younger than 40. 40% have abrupt onset of symptoms.
   i. fatigue most common initial symptom (85%).
   ii. jaundice (46%).
   iii. polymyalgias (30%).
   iv. physical findings: hepatomegaly (78%).
   v. 30-48% of patients with autoimmune hepatitis will have other immune diseases.

C. Lab
   i. has to have increased transaminase
   ii. increased gamma globulin and IgG
   iii. autoantibodies
      a. antinuclear antibody (ANA)
      b. smooth muscle antibody (SMA)
      c. antibodies to liver/kidney microsome type 1 (anti-LKM1)

D. Treatment
   i. prednisone alone or with azathioprine is effective.

8. Cholestatic Liver Disease
A. Primary biliary cirrhosis: AMA + in 95% of patients. 90% cases are women.
   i. presentation: frequently asymptomatic with abnormal lab work. Fatigue most common, then pruritis (40%).
   ii. Lab: increased alkaline phosphatase. May see increased cholesterol and IgM. AMA (antimitochondrial antibody)
   iii. treatment: ursodiol 13 to 15mg/kg daily.

B. Autoimmune cholangitis: AMA-negative primary biliary cirrhosis.
   i. treatment: most pts respond to ursodiol.

C. Primary sclerosing cholangitis
   i. about 70% of the patients have inflammatory bowel disease.
   ii. more common in men than women.
   iii. diagnosis requires cholangiography.
   iv. treatment: ursodiol has inconsistent effects.
   v. pt at risk for cholangiocarcinoma

9. Alpha 1- anti-trypin deficiency
   i. Autosomal codominant disorder with lung and liver injury
   ii. Can cause premature emphysema and liver disease
   iii. Pt with cirrhosis due to AAT have a significant increased risk of HCC up to 30%
   iv. Diagnosed by phenotyping. Liver damage does NOT correlate with serum AAT levels (unlike lung). Diagnosis confirmed with biopsy
   v. No effective medical treatment for the liver manifestations of AAT deficiency.
GI Surgeries
March 26, 2014
3:30-400p

Surgery for GERD
Indications for GERD
   Failed optimal medical management
   Noncompliance with medical therapy
   High volume reflux
   Severe esophagitis by endoscopy
   Benign stricture
   Barretts without severe dysplasia
Non GERD indications (not first line treatment)
   Hoarseness
   Laryngitis
   Wheezing
   Nocturnal asthma
   Cough/aspiration
   Dental erosions
Complications
   Dysphagia
   Gas bloat syndrome
   Revisional surgery needed in 5-10% of pts after laparoscopic
   fundoplication

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)
Postgastrectomy syndromes
   Postvagotomy diarrhea
   Dumping syndrome
   Alkaline reflux gastritis
   Early satiety
Operation for gastric ulcer. Difference from duodenal is that gastric ulcer may
   harbor malignancy and therefore must be excised or generously biopsied.

Acute Cholecystitis
Cholecystectomy
   Indications
   Complication
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.