Gastroenteritis

• Compare & contrast the clinical manifestations of viral and bacterial gastroenteritis

	Viral	Bacterial
Involved Site Diarrhea Vomiting	Small intestine Watery +++ or +	Colon Bloody, <u>mucoid</u> + or -
Abdominal pain/cramps	+ or -	+++ or ++
Tenesmus	-	++ or +
Anorexia	+ or -	+++ or ++
Fever	+ or -	++ or +
Fecal leukocytes	+ or -	++ or -
Systemic illness/ symptom	+ or -	+++ or ++

Does history contain EPIDEMIOLOGICAL CLUES that might favor a particular etiology (travel, antibiotic use)?

- Treatment for viral and bacterial (traveler's vs food borne)
- Treat viral diff than bacterial
 - o Management
 - Oral or IV fluid & electrolyte replacement (most important!)
 - Anti motility drug (loperamide) no more than 8 mg lomotil
 - Antidiarrheal (bismuth)
 - Antiemetic (ondansetron)
 - Empiric antibiotics (situational)
 - Food poisoning (severe sx >3 day)- ciprofloxacin, erythromycin base, metronidazole or Augmentin x 5-10 days
 - Traveler's diarrhea- azithromycin single high dose or daily lower dose x 3 days
 - P553 prevention bismuth 8 tablets a day
 - Bratty diet
- Common risk factors and findings in gastroenteritis
 - high risk for developing gastroenteritis include anyone traveling to a developing country, immunocompromised patients, anyone engaging in anal intercourse, residents of institutions or nursing homes, infants and children attending day-care centers, and individuals consuming raw shellfish and seafood.

Acute appendicitis

- Subjective and objective findings
 - o Subjective
 - acute mild to moderate colicky epigastric or periumbilical pain, can radiate to testes, nausea, vomiting, anorexia. bowels changes, mild fever
 - o Objective-
 - could be ill appearing, low grade fever, abdominal tenderness to palpation, muscle guarding, rebound tenderness, positive Rovsing's, Psoas, Obturator and McBurney's sign, HTN, tachycardia, variable bowel sounds- may be reduced

• How do they present common diagnostic things you would look for s/s. mc burneys point

Cefosporin and zosyn

Appendicitis

- I. Definition: inflammation and/or rupture of the appendix
- II. Signs and symptoms
 - A. Anorexia, N/V
 - B. Low-grade fever (possibly)
 - C. Stooped appearance when walking
 - D. Presence of one or more of these signs in adults increases likelihood
 - 1. Pain worsens with movement (e.g., walking or just moving around in bed), coughing, and deep breathing and is located in the RLQ.
 - 2. Pain migrates from the periumbilical region to the RLQ usually within 24 hr.
 - 3. Pain is noted before vomiting.
 - 4. There is positive rebound and guarding.
 - 5. Positive psoas sign, Markle sign, and Rovsing sign
 - E. In children <5 yr of age, incidence increases with
 - 1. Fever ≥ 100.5°F
 - 2. Diffuse abdominal pain with rebound tenderness and pain to percussion
 - 3. Irritability, grunting respirations
 - 4. Refusing to walk and may complain of right hip pain
 - 5. Pain with cough or jumping
 - 6. Anorexia, N/V
 - 7. WBCs >10,000/μL
 - 8. Neutrophils + bands ≥7500/μL

III. Diagnostic testing

A. CBC (may show leukocytosis), ESR/CRP, U/A (usually normal), urine pregnancy test if childbearing age

- B. Flat and erect abdominal x-ray (may show fecalith)
- C. Abdominal US for children (less invasive but the appendix may not be visualized)
- D. CT abdomen for appendicitis (see <u>Table 4.2</u>) is gold standard for diagnosis

IV. *Refer to a surgeon or ED* for further follow-up if any tests are positive or if the symptoms continue

Cholecystitis

- Subjective and objective findings
- Subjective-
 - RUQ pain; may radiate to right shoulder or subscapular area, Nausea and vomiting/anorexia, attack follows meal (especially high in fat) by 1-6 hours, Low-grade fever, light colored stools
- Objective
 - ill-appearing, febrile and tachycardic, Murphy's sign (classic), palpable RUQ mass, guarded movements, jaundice
- Diagnostic reasoning (priority imaging and lab values)
 - Initial- RUQ Abdominal Ultrasound, CBC, CRP, CMP
 - o Others to consider-, hepatobiliary (HIDA) scan, abdominal CT amylase
- s/s of and the point their in assessment fndngs or how they presents

Acute Pancreatitis

• Possible causes

Box 41.1 Causes of Pancreatitis

- Infection (mumps)
- Hyperlipidemia (particularly types I, IV, and V)
- Metabolic disorders (hyperparathyroidism, hypercalcemia)
- · Drugs (furosemide, valproic acid, sulfonamides, thiazides)
- Endoscopic retrograde cholangiopancreatography (ERCP)
- Structural abnormalities of the pancreatic duct (stricture, carcinoma, pancreas divisum)
- · Structural abnormalities of the common bile duct and ampullary region
- · Surgery (particularly of the stomach and biliary tract)
- · Vascular disease (atherosclerosis, severe hypotension)
- Trauma
 - Risk factors for AP
 - Acute pancreatitis is usually the result of some other process, such as passing of a gallstone, excessive alcohol intake, or other type of biliary tract disease. Drinking or lipids
 - Subjective reports
 - <u>Acute</u>- abrupt onset deep epigastric pain, radiating to the back, intractable nausea and vomiting, unable to lie supine, alcohol use, sweating, weakness or anxiety
 - <u>Chronic</u>- intractable abdominal pain, weight loss, diarrhea, dyspepsia, nausea, vomiting, bulky, foul smelling, fatty stools

Pancreatitis

I. Definition: inflammation of the pancreas, with leakage of pancreatic enzymes into the surrounding tissues. A. Can be caused by 1. Chronic ingestion of alcohol or obstructing gallstones 2. Strictures or tumors that block the duct of the pancreas 3. Hypertriglyceridemia (>1000 mg/dL) 4. Common medications that could be implicated a) Diuretics (hydrochlorothiazide) b) Sulfonamides (TMP-SMZ) c) Valproic acid (Depakene) d) Tetracycline e) GLP-1 agonists II. Signs and symptoms A. Mild to severe acute to chronic sharp pain in the upper abdomen that may radiate to the back or chest, abdominal distention B. N/V, fever, and possible scleral icterus; abdominal distention and/or hypoactive bowel sounds C. Signs of intra-abdominal bleeding with ecchymosis in the flanks (e.g., Grey-Turner sign) or periumbilical region (e.g., Cullen sign) D. Weight loss (unexplained) E. Hypotensive signs: tachycardia, sweating, tachypnea, hypoxemia III. Diagnostic testing A. Amylase and lipase >2 to 3 times the normal B. CBC (elevated WBC and HCT), elevated BUN and glucose, low calcium and magnesium C. Imaging findings (transabdominal US, CT abdomen, or MRI of abdomen) consistent with pancreatitis IV. Treatment A. ORefer to ED for possible admission. B. Recommendation after discharge 1. Patients should follow a low-fat diet; patients should limit foods high in trans fats or trans-fat substitutes (e.g., cookies, crackers, cakes, and donuts); this can limit recurrence and also help normalize triglycerides. 2. Encourage patients to eat foods high in antioxidants, such as green leafy vegetables, cold-water fish, beans, berries, tomatoes, and foods high in vitamin B and C, and iron. 3. Decrease or eliminate alcohol, caffeine, and tobacco. 4. Amylase, lipase, and glucose should be monitored every 3 mo until stable and then annually; if glucose does not normalize, may need to treat for DM (see Chapter 16). 5. Supplements for deficiencies related to pancreatitis: a) Multivitamin, omega-3 fatty acids b) Co-Q¹⁰ 100 to 200 mg at bedtime c) Probiotic supplement daily C. Suggest counseling for abusive habits leading to episodes of pancreatitis.

Jaundice

- Compare and contrast direct and indirect bilirubin results
 - o Unconjugated (indirect) not water soluble cant get out cytotoxic to CNS
 - Bilirubin in systemic circulation from breakdown of RBCs
 - Binds to albumin
 - Transported to the liver for conjugation and secretion
 - Conjugated (direct) water soluble and excreted
 - Liver converts unconjugated to conjugated bilirubin
 - Secreted in the bile to GI tract
 - Broken down to urobilinogen in gut
 - Secreted primarily in stool, minimally in urine

Treatment photo therapy

- ;'. What test would you do labs
 - o Initial
 - CBC, peripheral smear, direct and indirect bilirubin, CMP, UA, Hepatitis Panel, US RUQ
 - o Subsequent or as directed by exam findings
 - GGT, PT/INR, amylase, lipase, LDH, iron studies, HIDA Scan, CT/MRI
- Liver function labs obstructive hepatocellular

Hep b antigen antibody IgM means its active now

Hepatitis A, B, C

- Sources of infection
- Confirmatory lab values
- Look at slide for the diagnosis of which one. Look at graph
- Hep a
- Hep c
- And risk factors

Features	Hepatitis A Virus (HAV)	Hepatitis B Virus (HBV)	Hepatitis C Virus (HCV)	Hepatitis D Virus (HDV)	Hepatitis E Virus (HEV)
Transmission	Fecal–oral through sewage- contaminated water and shellfish; possibly through blood	Percutaneous and permucosal through infected blood and body fluids; sexual transmission	Percutaneous through infected blood and body fluids; community, many infected individuals have no known risk factors	Percutaneous, but must have co- infection with HBV	Fecal–oral
Incubation period (days)	15–50 (average 20–37)	25–160 (average 60–110)	42–49	Same as for HBV	10–56
Laboratory tests	Anti-HAV IgM (acute); anti-HAV IgG (resolving)	HBsAg (confirms), IgM anti-HBs (acute phase), IgG anti-HBs (resolving/immunity), HBeAg, anti-HBe, anti- HBc (persists in carriers)	Anti-HCV appears in 6–37 weeks	Anti-HDV appears late	Anti-HEV IgM detected within 26 days of jaundice; IgG antibody persists
Immunity/immunization	45% of United States population has antibodies against HAV; HAV vaccine available	5%–15% of U.S. population has anti- HBs; HBV vaccine available	Unknown; no vaccine available	People immune to HBV are also protected against HDV	Unknown
Prevalence	Increasing in adults	Decreasing in the United States	4% of post- transfusion hepatitis; 50% IV drug users	Common in IV drug abusers	Rare in United States; endemic in Southeast Asia, India, North Africa, Mexico
Course/mortality	Does not progress to chronic state; mortality is 0–0.2% with fulminant hepatitis	Chronic liver disease occurs in 1%–5% of adults and 80%–90% in children; mortality rate is 0.3%–1.5%	Chronic active hepatitis develops in 70%–90% of cases; 20% develop chronic liver disease; mortality rate is the same as for HBV	Chronic liver disease develops if present in chronic HBV; mortality rate is 2%– 20% for acute icteric hepatitis	Does not progress to chronic liver disease; mortality rate is 1%–2% but as high as 10%–15% in pregnant women

Cirrhosis

Clinical manifestations early and late signs

- Clinical presentation
 - Subjective- weakness, fatigue, anorexia, weight loss, upper GI bleeding, abdominal pain and distention, menstrual irregularities, low libido, impotence, sterility, bruise or bleed easily, hematemesis, melena, hematochezia, hemorrhoids, confusion, mental status changes, drowsiness
 - Objective-ascites, encephalopathy, gynecomastia, portal hypertension, esophageal varices, enlarged or difficult to palpate liver, spider nevi on anterior chest, pectoral alopecia, muscle wasting, Dupuytren's contracture, parotid gland enlargement, palmar erythema, hair loss, testicular atrophy, varicose veins, glossitis, cheilitis, peripheral neuropathy, jaundice, peripheral edema, splenomegaly, tremors, asterixis (hand flapping), hyperactive deep tendon reflexes,

GERD

- Clinical manifestations
 - Subjective- Heartburn, dysphagia, regurgitation, belching, sour taste in mouth, odynophagia, cough, hoarseness, wheezing at night, chest pain, weight loss, rectal bleeding,
 - \circ $\;$ Objective- Typically normal exam. May have occult blood on rectal exam
- Lifestyle changes
 - Lifestyle changes- weight loss, wait 2-3 hours after meals to lie down, avoid offending foods (chocolate, coffee, caffeine, acidic or spicy foods, ETOH see Fenstermacher pg. .231) and medicines (NSAIDS, ASA), elevate head of bed 6-8 inches, smoking cessation
- 1st and 2nd line pharmaceutical interventions
 - 1: 8 week Trial of any Proton Pump Inhibitor (e.g. omeprazole

• 2: With nocturnal component and incomplete response to PPI add a H2 antagonist (e.g. famotidine)

IX. Treatment

A. If there are no alarming symptoms and the clinical history indicates that GERD is the probable cause: initiate lifestyle and dietary modifications first.

- 1. Do not eat meals or drink carbonated beverages within 3 hr of bedtime.
- 2. Decrease the amount of fried, fatty, and spicy foods to decrease gastric acid production.
- 3. Raise the head of the bed using 4 to 6-inch blocks, especially if nocturnal symptoms are present.
- 4. Lose weight if indicated; avoid tight-fitting clothing, especially around the waist.
- 5. Decrease factors that aggravate reflux symptoms (see Box 10.8).
- 6. Avoid foods that relax the LES (see <u>Box 10.7</u>).
- 7. Decrease or eliminate NSAID use, nicotine, and alcohol.
- B. Drug therapy (also see <u>Table 10.2</u>, H₂ blockers/PPIs).
 - 1. Initial therapy for mild to moderate GERD
 - a) Use antacids with symptoms that occur < once a week
 - b) If symptoms are predictable, use antacids 1 hr before meals, after meals, and at bedtime; antacids have a rapid onset but short duration.
 - c) Use OTC H₂ blockers bid for 2 wk if needed; if symptoms continue, may need to initiate PPIs.
 - d) Bedtime dosing is useful with nocturnal reflux
 - 2. If symptoms are intense and predictable even with treatment,
 - a) Reevaluate lifestyle and dietary habits.
 - b) Use PPIs bid for 8 wk and then gradually decrease the dose.
 - i) If symptoms resolve, gradually taper dose off (sudden withdrawal may cause rebound acid reflux).
 - ii) If symptoms resolve but then recur, restart PPI at a lower dose for another 8 wk, then change to H_2 blocker qd for 4 wk and then stop treatment.
 - iii) If symptoms recur again (even without not-to-be-missed signs), *refer to gastroenterologist*.

PUD

- - -

• Clinical manifestations

•

- **Subjective** chronic, recurrent, epigastric pain, burning or gnawing, can be nocturnal pain, nausea, anorexia, melena or coffee ground emesis
- o **Objective** epigastric tenderness to palpation, occult blood in stool, abdominal rigidity
- RED flags- weight loss, recurrent or intractable vomiting, GI bleeding, iron deficiency anemia, dysphagia, odynophagia, palpable mass or lymphadenopathy, or new- onset dyspepsia, esp. > 60 years old

Hemorrhoids

- Compare and contrast the subjective and objective findings with internal vs external hemorrhoids
- Subjective and objective signs
 - **Subjective-** rectal pain or pressure, itching, bright red streaks of blood, or drips of blood in toilet
 - Objective- Rectal exam externally protruding or internally engorged veins, thrombosed veins, skin tags
- Book adequate fluid intake and increase fiber to 30-35
 - On physical examination, external hemorrhoids may not be visible at rest but usually protrude on standing or with the Valsalva maneuver. Thrombosed hemorrhoids may appear as shiny, blue masses located at the anus. Evidence of hemorrhoidal skin tags may appear at the site of resolved hemorrhoids; these skin tags are fibrotic and painless.
 - Internal hemorrhoids most often present with rectal bleeding described as bright red streaks on the toilet paper. Patients may report that blood drips into the toilet after a bowel movement.

Occasionally the bleeding is sufficient enough to cause anemia, which in any case merits further investigation.

Inguinal hernia

- Treatment recommendations
- Management when to refer and when to not book refer to surgeon
- Management Inguinal
 - Incarcerated, strangulated OR large OR symptomatic uncomplicated hernias- emergent surgical repair + prophylactic IV antibiotics
 - o Small asymptomatic or minimally symptomatic & reducible- watchful waiting

Chrons vs ulcerative colitis

Crohn's and Ulcerative colitis

- Compare and contrast the patho
- Patient education
- Red flag symptoms to report, when to seek care, medication adverse effects, life long treatment- no cure, smoking cessation, dietary advice, avoid NSAIDS, avoid live vaccines and generally avoid vaccinations for those on immunosuppressants per ACG, participate in regular exercise

Ulcerative Colitis

- Involves the colon, extends from rectum
- Continuous
- Involves mucosa and submucosa
- Steatorrhea absent
- Strictures and fistulas rare
- Remissions and relapses
- Malignancy common
- Book:UC commonly report four or fewer loose bowel movements per day associated with abdominal cramps that are relieved with defecation, small amounts of blood and mucus in the stool, and sometimes tenesmus.
- UC includes nutrition counseling. Patients should avoid caffeine, raw fruits, vegetables, and other foods high in fiber, which can cause trauma to the already inflamed mucosal surface. Some patients may benefit from a lactose-free diet

Ulcerative colitis

- I. Definition: inflammation that involves the colon and rectum
 - A. Primarily in young adults
 - B. Increased risk of colon cancer
- II. Signs and symptoms (may occur after infectious GE)
 - A. Frequent episodes of bloody mucus and pus from the rectum
 - B. Diarrhea can occur without warning
 - C. Cramping abdominal pain is usually mild
 - D. Anorexia, malaise, weight loss, and fatigue
 - E. Rectal tenesmus
 - F. Intolerance of dairy products periodically; consider avoiding during acute flares
- III. Diagnostic testing
 - A. CBC (may show microcytic anemia), CMP (may have low K+ and high BUN), ESR/CRP (usually elevated)
 - B. Stools positive for blood, stool cultures, culture for *C. difficile*, O&P, fecal calprotectin (marker for inflammation)
 - C. Abdominal/pelvic CT (see <u>Table 4.2</u>)
 - D. Sigmoidoscopy, colonoscopy (if symptoms are not severe)
- IV. Treatment

A. Refer to a gastroenterologist for management

- B. Stop smoking
- C. Avoid milk products with acute flares, increase intake of calcium/vitamin D supplements
- Acute mild UC
 - Proctitis or proctosigmoiditis- 1st line- mesalamine rectal enema or suppository preferred over mesalamine oral, 2ndl line- topical hydrocortisone per rectum
 - Left sided or extensive colitis- oral + rectal mesalamine with adjunct oral budesonide or prednisone if not responsive to mesalamine alone
- Acute moderate to severe UC-1st line Prednisone 40-60 mg/day or biologic agents (e.g. infliximab, adalimumab, etc.) + immunomodulators (e.g. azathioprine, methotrexate), 2nd line Tofacitinib and 3rd line colectomy
- Acute severe UC- hospital admission, IV corticosteroids, Blood transfusions, IV fluid and electrolyte replacement, LMWH to prevent thromboembolism +/- IV cyclosporine or infliximab or colectomy

Crohn's Disease

- Involves any part of the GI tract mouth to anus
- Segmental
- Involves all layers of mucosa
- Steatorrhea frequent
- Strictures and fistulas common
- Partial or complete obstructions
- Slowly progressive
- Malignancy rare
- Peyers patches and cobble stone appearance
- Book: The most common presenting symptoms of CD are abdominal cramping and tenderness, fever, anorexia, weight loss, spasm, flatulence, and right lower quadrant (RLQ) pain or mass. Individuals may report an increase in symptomatology during periods of stress or emotional upset or after meals consisting of poorly tolerated foods such as fatty or spicy foods or milk. Stools are soft or semiliquid. Observable blood is found in the stool intermittently; when present, it occurs in a larger amount than with UC. Because of the loss of healthy bowel mucosa, there may be insufficient resorption of bile salts, causing steatorrhea (foul-smelling, fatty stools). CD can involve the entire thickness of the bowel wall, causing microperforations and symptoms of acute localized peritonitis, which can mimic appendicitis or diverticulitis. If there is fistula formation, these symptoms may dominate the clinical picture.

Crohn disease (regional enteritis)

I. Definition: chronic recurring immune response and inflammation that may involve discontinuous segments of the GI tract from the mouth to the anus

A. Occurs at any age

B. Increased risk of colon cancer

II. Signs and symptoms

A. Intermittent diarrhea followed by constipation with recurrent episodes of mucoid diarrhea

B. Colicky or steady abdominal pain located in the RLQ or periumbilical region

C. Anorexia, flatulence, malaise, and weight loss

D. Perianal disease with fistula, superficial ulcers, and skin tags

E. Extraintestinal manifestations may occur in musculoskeletal and dermatologic sites about 10 yr after diagnosis

III. Diagnostic testing

A. CBC (may show macrocytic anemia), ESR/CRP, and CMP (to monitor albumin), iron, ferritin, and vitamin B12

B. Positive blood in stool, stool cultures, *C. difficile* culture, and O&P

C. Abdominal CT (see <u>Table 4.2</u>)

D. Endoscopy and colonoscopy

IV. Treatment

A. Refer to a gastroenterologist or surgeon for further management depending on the severity of symptoms

B. Stop smoking

C. Stay up to date with immunizations (especially flu and pneumonia)

D. Nonresidue diet with high protein, vitamins, and calories; avoid raw fruits and vegetables

Feature	Ulcerative Colitis	Crohn's Disease
History		
Age at onset	Age 10–40	Age 15–25; age 50–80
Etiology	Unknown	Unknown
Genetic tendency	Familial tendency	Familial tendency
Nicotine use	Nonsmoker	Smoker
Assessment Findings		
Serological	+ (positive) for antineutrophil cytoplasmic antibodies (pANCA)	– pANCA
Fever/malaise	With severe disease	Common
Weight loss	Uncommon	Common
Rectal bleeding	Common	Dependent on location of lesion; occurs in about 50% of cases
Abdominal pain	Usually mild	Can be moderate to severe
Abdominal mass	Negative	May be present
Perianal lesions	Absent	May develop fissures, abscesses
Fistulas	Absent	Common
Strictures	Uncommon	Common
Common		
Rectal involvement	Always	50% of the cases
Distribution	Confined to colon; continuous	Any portion of gastrointestinal tract; discontinuous, skipped lesions
Mucosa	Friable, granular	Cobblestone appearance
Ulceration	Crypt abscess development	Aphthous or linear ulcers
Inflammation	Surface involvement	Transmural involvement

- CD- Ranges from observation to management of extraintestinal complications- requires a multidisciplinary team
 - Therapies may include hospitalization, oral/IV antibiotics, immunomodulators, oral/IV/rectal corticosteroids, biologic therapy, immunosuppressants, surgical resection of intestines, dilation, fistulotomy drainage of perianal abscesses, medical nutritional therapy including PTN, PPIs, TNF-alpha inhibitors

Symptoms management

Celiac

- Clinical manifestations
 - **Subjective** asymptomatic or diarrhea, weight loss, dyspepsia, flatulence, fatigue, joint pain, depressed mood, amenorrhea, infertility, early menopause

- Objective- normal exam or muscle wasting, pallor, reduced subcutaneous fat, ataxia, peripheral neuropathy, dermatitis herpetiforms
- Management- Strict gluten free diet, vitamin D, B12, calcium, iron, and folate supplementation as labs suggest
- celiacs disease what to watch for and pt education

Diverticular Disease

- Clinical manifestations
- s/s/ of acute diverticulitits
- Clinical presentation
 - Subjective- LLQ abdominal pain, diarrhea or constipation, bloating, rectal bleeding, fever, chills, anorexia, nausea, vomiting, dysuria, or pneumoturia or fecaluria
 - Objective- Tenderness to palpation LLQ, firm fixed mass, rebound tenderness, involuntary guarding, rigidity, hypo or hyperactive bowel sounds, tenderness on rectal exam, occult blood
- Differential Diagnosis
 - IBS, colon cancer, irritable bowel disease, lactose intolerance, pelvic inflammatory disease, ovarian cyst, colitis, appendicitis, pyelonephritis
- Diagnostic Reasoning
 - Initial-CBC, CRP, UA/UC, abdominal X-ray, CT scan with oral & IV contrast
 - Subsequent- abdominal ultrasound, colonoscopy
- Management
 - Diverticulosis(asymptomatic)- lifestyle modification- high fiber diet, quit smoking, lose weight
 - Diverticular disease (symptomatic)- lifestyle modification, +/- amoxicillin/clavulanate for up to 10 days or ciprofloxacin +metronidazole for 7-10 days
 - Diverticulitis uncomplicated-outpatient- Tylenol or tramadol for pain and antibiotics as above, low residue diet, if not improvement in 72 hrs. admit
 - Diverticulitis complicated- admit to hospital
- Follow up
 - Close follow up in 72 hours for resolution and complications
 - Colonoscopy at some point
- Referral
 - ER- for signs of perforation or sepsis
 - o Gastroenterologist- for colonoscopy 6-8 weeks after treatment

IBS 3 or more watery stools a day. Relief wth bm and flatus

- Clinical presentation
 - Subjective- Colonic abdominal pain relieved with flatus or a BM, constipation, diarrhea, urgency to defecate, bloating, gas, belching, dyspepsia, pyrosis, nausea, vomiting, passage of mucus with stool, increased stress, anxiety or depression
 - Objective- Abdominal tenderness to palpation, DRE- usually normal
- Ensure you do not miss red flag signs
 - Rectal bleeding and anemia, Men over 50 years of age, FH of colon cancer, weight loss, nocturnal diarrhea, any change in recent bowel pattern, and past antibiotic use
- Diagnostic testing
- Diagnostic Reasoning
 - o Predominantly a clinical diagnosis

- Management
- Diverticulosis(asymptomatic)- lifestyle modification- high fiber diet, quit smoking, lose weight
- Diverticular disease (symptomatic)- lifestyle modification, +/- amoxicillin/clavulanate for up to 10 days or ciprofloxacin +metronidazole for 7-10 days
- Diverticulitis uncomplicated-outpatient- Tylenol or tramadol for pain and antibiotics as above, low residue diet, if not improvement in 72 hrs. admit
- Diverticulitis complicated- admit to hospital
- Follow up
- Close follow up in 72 hours for resolution and complications
- Colonoscopy at some point
- Referral
- ER- for signs of perforation or sepsis
- Gastroenterologist- for colonoscopy 6-8 weeks after treatment
- Criteria -Dunphy table 40.1 and Rome IV criteria (link in module and Fenstermacher pg. 240)
- o Initial tests- CBC, CMP, TSH, CRP, stool for occult blood
- Other tests to consider-
 - For diarrhea predominant test for celiac disease- IgA human antitissue transglutaminase (anti- tTG) 48 hr. stool for bile acids, stool for ova and parasites and giardia, hydrogen breath test
 - Fecal calprotectin and lactoferrin flexible sigmoidoscopy (< 40), colonoscopy (> 50 or FH colon cancer < 60), plain abdominal x-ray, and food elimination diet

Treatment recommendations

V. Pharmacologic treatment is based on subtype

- A. Constipation-predominant symptoms 1. Increase fiber to 25 to 30 g/day with foods and supplemental psyllium products (see Appendix A)
 - Increase fiber to 25 to 30 g/day with foods as
 Ground linseeds 6 to 24 mg/day
 - 3. Polyethylene glycol 3350 (Miralax) 1 capful dissolved in liquid daily
 - 4. Bisacodyl (Dulcolax) 1 to 2 tabs as needed (prn)
 - 5. Probiotics daily
 - 6. Lubiprostone (Amitiza) 8 mcg bid
 - 7. Linaclotide (Linzess) 290 mcg qd
- B. Diarrhea-predominant symptoms
 - 1. Loperamide (Imodium) 2 capsules prior to meals on PRN basis
 - 2. Diphenoxylate/atropine (Lomotil) 5 to 20 mg qd prn (off label) for short-term use
 - 3. Increase fiber up to 25 to 30 g/day with foods and supplemental psyllium products (see Appendix A)
 - 4. Bile acid sequestrant (Cholestyramine) 4 to 36 g qd prn (off label)
 - 5. Probiotics (Bifidobacterium infantis) qd
- C. Abdominal pain and cramping
 - 1. Dicyclomine (Bentyl) 10 to 40 mg qid prn before meals
 - 2. Hyoscyamine (Levsin) 0.25 mg qid prn (max dose 1.5 mg/day)
 - 3. Peppermint oil caps after meals or prn

VI. *Refer to gastroenterologist* if symptoms continue or any not-to-be missed signs are present.

- General
 - Fiber25-30 gm/day (foods or psyllium products), probiotics, support groups, behavior modification, biofeedback, low FODMAP diet, avoid food triggers, stress reduction
- Constipation predominant
 - Polyethylene glycol (Miralax) daily, Bisacodyl (Dulcolax) prn, Lubiprostone (Amitiza) or Linaslotide (Linzess) daily or ground linseeds daily
- Diarrhea predominant

 Loperamide (Imodium) prn, & Bile acid sequestrant (Cholestyramine- more effective is cholecystectomy) [off label]; Lomotil no longer recommend

• Mixed

 1st Fiber, 2nd Loperamide prn in diarrheal phase, polyethylene glycol prn in constipation phase

• Abdominal pain

- 1st line Antispasmodics -Dicyclomine (Bentyl) or Hyoscyamine (Levsin) or peppermint oil caps (antispasmodic) prn
- 2nd line SSRIs (Paroxetine [Paxil] or Citalopram [Celexa]) more appropriate if constipated and TCAs (Desipramine [Norpramin], amitriptyline [Elavil] or doxepin [Silenor]) more appropriate if diarrhea. Can use either class in mixed presentations.

Table 1. Rome IV Criteria for the Irritable Bowel Syndrome.*		
Patie	nt has recurrent abdominal pain (≥1 day per week, on average, in the pre- vious 3 mo), with an onset ≥6 mo before diagnosis	
Abdo	minal pain is associated with at least two of the following three symptoms:	
Ρ	ain related to defecation	
C	hange in frequency of stool	
C	hange in form (appearance) of stool	
Patie	nt has none of the following warning signs:	
A	ge \geq 50 yr, no previous colon cancer screening, and presence of symptoms	
R	ecent change in bowel habit	
E	vidence of overt GI bleeding (i.e., melena or hematochezia)	
Ν	locturnal pain or passage of stools	
U	Inintentional weight loss	
F	amily history of colorectal cancer or inflammatory bowel disease	
Ρ	alpable abdominal mass or lymphadenopathy	
E	vidence of iron-deficiency anemia on blood testing	
Р	ositive test for fecal occult blood	

* The information is from Mearin et al.¹ GI denotes gastrointestinal.

Colorectal cancer Question!!!

- Screening recommendation
 - o colonoscopy Q 10 years at 45- 50 years old, FOBT or FIT tests periodically in between
 - \circ Hx of 1st degree relative > 60 yr. with CRC- start screen at 40 yr. of age, repeat Q 5 yr.
 - With 1st degree at 60 or 2 other +relatives -start screen at 40 or 10 yr. younger than when relative diagnosed
 - With personal hx of polyps follow up colonoscopy Q 3-5 years or per specialist

Bowel obstruction

- Clinical manifestations
- s/s of obstruction

- Subjective-
 - <u>Small bowe</u>l- sudden onset crampy intermittent abdominal pain, nausea, vomiting, failure to pass flatus or stool, lethargy
 - <u>Large bowel</u>- increasing colicky abdominal pain, constipation, lack of flatulence, hard or soft stool, tenesmus
- Objective-
 - <u>Small bowe</u>l-abdominal distention, tenderness to palpation with ischemia, hypotension, fever, tachycardia, potentially peritonitis
 - <u>Large bowel</u>- Distention, tympanic abdomen, DRE empty, impacted or with soft stool, increased frequency of bowel sounds, then absent in advanced stages, possible rectal or abdominal mass

Anemia mch vs mchc

	Iron Deficiency	B12 Deficiency Pernicious Anaemia	Anaemia of Chronic Disease
RBC Size	Microcytic	Macrocytic	Normocytic
MCV (82-99)	Decreased Ψ	Increased 🛧	Normal
MCH (27-32)	Low 🗸	Normal or High 🛧	Normal
TIBC	High 🛧	Normal	Normal or Low
Ferritin (10-291) iron iron	Low 🗸	High 🛧	Normal or High 🛧
B12 (130-700)	Normal	Low 🕈	Normal
Folate (200-650)	Normal	Normal	Normal
RBC (4-5)	Low	Low	Low
Нь (12-16) НЬ НЬ	Low	Low	Low © arthriticquaker 2009

Reticulocyte count question

• Reticulocytes are newly produced, relatively immature red blood cells (RBCs). A reticulocyte count helps to determine the number and/or percentage of reticulocytes in the blood and is a reflection of recent bone marrow function or activity. Recent bone marrow activity

Sickle, thaml gold standard electrophoresis

• Define it

Anemia Clinical Presentation

- Clinical presentation of microcytic and normocytic anemias
 - *Subjective* fatigue, palpitations, shortness of breath, dyspnea on exertion, dizziness or lightheadedness
 - Objective- pale mucous membranes, sallow-colored skin, tachycardia, tachypnea, capillary refill > 3 seconds, brittle nails, cheilosis, pica, systolic murmur, possible splenomegaly
- Clinical presentation of macrocytic anemias

- Subjective- mouth soreness, nausea, anorexia, diarrhea, peripheral neuropathies, malaise, cognition changes, memory changes. Can cause sores in mouth, NV, peripheral neuropathy
- Objective- variable Babinski, increased or decreased reflexes, pale or icteric mucosa, dry or cracked oropharynx, glossitis, stomatitis, tachycardia, systolic murmur, abdominal tenderness
- o to palpation, positive Romberg, diminished vibratory sensation in the lower extremities

• • *note splenomegaly in the presence of anemia requires further diagnostic evaluation Differentiate between microcytic, macrocytic and normocytic (lab values and common causes)

- Microcytic Iron deficiency anemia (IDA), anemia of chronic disease (ACD), thalassemia, sideroblastic anemia
- Macrocytic Vitamin B12 deficiency, folate deficiency, antimetabolite drugs, myelodysplasia, chronic liver disease
- Normocytic anemia of chronic disease, mixed anemias, iron and vitamin deficiencies, impaired bone marrow function, malignancies, bleeding, hemolysis, autoimmune hemolytic anemia (AIHA)

Initial labs on the test confirm diagnosis with 2 test

• CBC

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- Low hemoglobin, hematocrit or red blood cell counts
- Mean corpuscular volume (MCV)
 - Microcytic < 80 fL, normocytic 80-96 fL, macrocytic >96 fL
- Mean corpuscular hemoglobin (MCH)
 - Hypochromic < 28 pg., normochromic 28-34 pg., hyperchromic > 34 pg.
- Red cell distribution width (RDW) Normal is 10.5-14.5%
- Peripheral smear
 - Poikilocytosis (odd shape), anisocytosis (size variation) or target cells (nucleated)
- Reticulocyte count
- *note splenomegaly in the presence of anemia requires further diagnostic evaluation for hemolytic anemia
- Subsequent labs based on presentation and/or CBC results
- Microcytic presentation
 - Serum Iron, ferritin and total iron-binding capacity (TIBC) [R/O IDA]
 - Hemoglobin electrophoresis (R/O thalassemia)
 - Bone marrow aspirate (R/O sideroblastic anemia)
- Macrocytic presentation
 - Vitamin b12 and Folate (R/O b12 and folate deficiency)
- Normocytic presentation
 - o WBC with differential, platelet count, Serum creatinine, LDH. PT/PTT

Used to diagnose IDA, ACD, and thalassemia

- Serum ferritin- the body's major iron storage protein
- Serum iron- iron concentration in circulation
- Total iron binding capacity (TIBC) or transferrin- a measure of transferrin (a protein that combines with iron). Represents the available iron binding sites. Has a reciprocal relationship with serum iron.
 - Serum iron high = TIBC low

- Serum iron low = TIBC high (correlates with iron deficiency)
- See boxes in Fenstermacher on pg. 227-229 for patterns in each type of anemia
- VII. Treatment of iron-deficiency anemia (microcytic, hypochromic) (see <u>Box 10.1</u>)

A. All ages

- 1. Therapy is continued for 2 to 3 mo after Hgb and ferritin levels have returned to normal.
- 2. Recheck H/H, ferritin, and reticulocytes after 1 mo of therapy and then q6mo for a year.
- B. Adults
 - 1. Begin OTC oral iron replacement with goal of 60 to 100 mg of elemental iron *every other day*.
 - a) Ferrous sulfate (Feosol) extended-release 325 mg bid; liquid (Fer-in-Sol) 60 mg/5 mL bid
 - b) Ferrous gluconate (Fergon, Ferrotabs) 325 mg 2 tabs bid
 - c) Ferrous fumarate (Ferrimin) 150 mg qd
 - 2. Give vitamin C with iron supplement to enhance absorption of the iron product.
 - 3. Take the iron tablets 1 to 2 hr after the following:
 - a) Foods: tea, coffee, milk, and eggs
 - b) Medications
 - i) Bisphosphonates; levothyroxine
 - ii) Quinolone antibiotics, tetracycline
 - iii) Antacids, H₂ blockers, and PPIs

4. Consider stool softener (docusate 100 mg) 1 to 2 caps once or twice daily along with the iron replacement C. Children <12 yr of age

- 1. Screening for anemia at 12 and 24 mo of age with CBC (or H/H)
- 2. Limit cow's milk to about 20 oz/day
- 3. Evaluate diet; increase servings of iron-rich foods with each meal (see Appendix A)

4. Ferrous sulfate 3 to 6 mg/kg elemental iron every other day between meals with orange juice or vitamin C supplement

- a) May cause temporary staining of teeth, rinse well or brush after dose
- b) Monitor for constipation
- D. Adolescents

1. Screening for girls at \sim 12 yr of age (if menses have started) and then annually after menses; boys once and annually if heavily involved in sports

- 2. Ferrous sulfate 65 to 130 mg elemental iron every other day for a minimum of 3 mo; then repeat CBC
- 3. Encourage 3 servings per day of iron-rich foods (see <u>Appendix A</u>)
- 4. Monitor for GI effects (e.g., constipation or gastric pain)
- Important pt design
- Chronic disease that puts your patient at risk of anemia
- Describe rationale for initial diagnostic work up

IDA

- \circ Describe serum iron, ferritin and TIBC and what values would be expected in IDA
- Treatment see above

Sickle Cell

- $\circ \quad \text{What is it} \quad$
- **o** Clinical manifestations
- Awareness of crisis
- **Disease risk factors** Higher prevalence in those from Africa, Mediterranean, Middle Eastern and some areas of India
- Crisis risk factors- Infection, dehydration, overexertion, stress, extreme hot or cold temperature exposure, high altitudes
 - Subjective -acute, excruciating episodes of pain in long bones, pain in back, chest, abdomen that is unrelieved by rest or position, nausea, anorexia, lightheadedness, anxiety or panic, heart palpitations, shortness of breath in crisis, depression, priapism, frequent infections in general

- Objective- chronically ill appearing, possible jaundice, hepatomegaly, hot, tender, swollen joints, retinopathy, cardiomegaly, chronic lower leg ulcers, low grade fever, pin- point pupils, photophobia, tachycardia, systolic murmur, tachypnea,
- Differential Diagnosis- other forms of anemia, sickle thalassemia, hemoglobin C disease, nonspecific abdominal pain, UTI, poisoning, DM
- Diagnostic Reasoning
- sickle cell anemia
 - Initial CBC with peripheral smear for screening, then hemoglobin electrophoresis (HbS) for diagnosis
 - Other options- hemoglobin solubility test, and HPLC fractionation or a DNA based assay to confirm
 - Reticulocyte count
- In crisis
 - CBC, reticulocyte count, indirect bilirubin, haptoglobin, LDH, iron studies
- Management-per hematologist- hydration, pain management, supportive care, folic acid, O2, may need transfusions
- Refer to hospital for acute crisis
- Follow up per hematology

Polycythemia

- What is it elevated hematocrit increase in erythrocyte number or concentration, which results in an increase in blood viscosity. The disorder may be either relative or absolute. An Hct greater than 51% in women and 54% in men is characteristic of the condition.
- Common primary and secondary causes
- Initial management options of primary and secondary
- Changes in blood smear
 - Relative polycythemia—caused by dehydration, no true increase in circulating erythrocytes or RBC mass
 - Relative polycythemia: dehydration
 - o Absolute polycythemia- actual increase in circulating erythrocytes and mass of RBCs
 - Primary polycythemia vera or other myeloproliferative neoplasms- increased erythrocytes, leukocytes and thrombocytes caused by an acquired or inherited cell mutation
 - Secondary- chronic hypoxia (high altitude living, smokers, carbon monoxide exposure, renal disorders, or cardiopulmonary disease), erythropoietin secreting tumors, autologous blood transfusions (blood doping), anabolic steroid or androgen use
 - Subjective-
 - Nonspecific- fevers, fatigue, malaise, weakness, sweating
 - Volume depletion- N/V, diarrhea, anorexia, orthostatic symptoms, use of diuretics
 - CP disease symptoms- chronic cough, cyanosis, hypersomnolence, SOB, dyspnea on exertion, sleep apnea symptoms
 - PV symptoms- burning hands and feet, red extremities, HA, blurred vision, tinnitus, vertigo, dizziness, early satiety, epistaxis, itching after a warm shower, bone pain (ribs/sternum)
 - Tumors- weight loss, abdominal/pelvic pain, blood in urine

- Objective-splenomegaly, hepatomegaly, upper abdomen, rib and sternum tenderness, signs of thrombosis or hemorrhage, PUD, retinal vessel growth and engorgement, skin purple or cyanotic, ruddy fingers and toes, dark flushed mucous membranes, facial plethora (ruddy cyanosis), breathing patterns, murmurs, bruits, lung sounds, heart sounds
- Differential Diagnosis- polycythemia vera, secondary polycythemias, spurious polycythemia (relative), hemoglobinopathy, chronic myeloid leukemia, myelofibrosis, essential thrombocytosis

Diagnostics

- Initial test- CBC with peripheral smear, CMP
 - HGB > 16.5 Female and > 18.5 Males or HCT > 48% suspicious for polycythemia
 - Absolute polycythemia HCT > 55% Female and > 60% Males
 - RBC count elevated
 - WBC 10-20,000
 - Platelet count elevated (thrombocytosis)
 - o Peripheral Smear- RBC morphology WNL unless underlying myeloproliferative neoplasm
- Secondary tests- based on suspected pathology/exam findings
 - Red blood cell mass- elevated in all absolute polycythemias (primary and secondary) but not widely available in the US
 - Polycythemia vera (primary type)- if splenomegaly, low MCV, evidence of IDA- get abdominal ultrasound, JAK 2 gene mutation screen (mutation present), serum erythropoietin [EPO] (low to normal)
 - Carbon monoxide exposure –ABGs for PaO2, carboxyhemoglobin level
 - Hypoxia- (smoking and sleep apnea, RA O2 sats < 92%) screen for underlying lung disease, PFTs, overnight oximetry, Sleep study ABGs, erythropoietin level (normal to elevated)

Labs

0

- Relative Polycythemia
 - Increased HCT
 - Normal RBC mass
 - Elevated BUN/CRT or ratio of > 20:1
 - Electrolyte evidence of volume depletion
- Polycythemia Vera
 - HCT > 55%
 - RBC mass elevated
 - Serum erythropoietin (EPO) normal
 - JAK2 mutations positive
 - 2nd to cardiopulmonary disease
 - Increased HCT
 - RBC mass elevated
 - EPO level elevated
 - Arterial O2 sats. low
- Smoker's or CO polycythemia
 - Increased HCT
 - RBC mass elevated
 - EPO level elevated
 - Arterial O2 sats. WNL
 - Carboxyhemoglobin elevated
- Other Causes
 - Increased HCT
 - RBC mass elevated

- EPO level elevated
- Arterial O2 sats. low
- Carboxyhemoglobin normal
- Search for tumor or renal source of EPO (CT head, abdomen, pelvis, renal ultrasound)

Management

- Relative polycythemia- medication adjustments (if cause) and oral or IV rehydration
- Absolute polycythemia (primary and secondary) phelobotomy
 - Low risk patients (<u><</u> 60 yrs. *, <u>and</u> no hx of thrombosis)*
 - If Hct > 55%- therapeutic phlebotomy weekly until the Hct is < 45%
 - No iron supplementation!
 - Unless contraindicated give low dose ASA
 - Assess and mange CV risk factors- HTN control, weight loss, physical activity and smoking cessation
- High risk patients (> 60 yrs, *or* hx of thrombosis)
 - As above + cytoreductive therapy 300 ml
 - Preferred- Hydroxyurea, alternatives- interferon or busulfan
 - Treat PV associated symptoms (pruritis, gout, hyperuricemia, erhythromelalgia, bleeding)
- Treat underlying cause in secondary polycythemia

Thrombocytopenia

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- Clinical manifestations
- Laboratory findings
- Clinical symptoms
- Subjective- asymptomatic to fatigue, bleeding, epistaxis, or bruising
- Objective- petechiae, nonpalpable purpura, mucous membrane hemorrhagic blisters, bruising, signs of GI bleeding, evaluate for lymphadenopathy, and hepatosplenomegaly (should be absent)
- Differential Diagnosis- Heparin induced, vaccine induced, antiphospholipid syndrome, DIC, thrombotic microangiopathy, paroxysmal nocturnal hemoglobinuria, bone marrow disorders, liver disease, hypersplenism, dilution, pregnancy, congenital platelet disorders, infection, HIV, parasites, drug induced, alcohol, malignancy, nutrient deficiencies, autoimmune disorders, & pseudothrombocytopenia

• Diagnostic Reasoning

- o Initial- CBC & peripheral smear
 - CBC & WBC typically WNL **except** Platelet count < 100,000
 - Peripheral smear- large platelets, no abnormal morphology
- Testing to eliminate other causes- HIV, Hep C, LFTs, other causes as above
- Management principles
 - Plt < 20,000 glucocorticoids to increase count</p>
 - Plt. < 50, 000 consider avoidance of extreme sports
 - +/- anticoagulants & antiplatelets based on risk for thrombosis
 - For nonurgent presentations refer to hematology for confirmation of dx and management
 - For urgent presentations (very low platelet count, evidence of bleeding or thrombosis) refer to ER

Educate patients on red flag symptoms

Leukemia

Classic presentation of leukemia difference

- Population specific
 - Acute Lymphoblastic Leukemia (ALL)- cancer of the immature lymphocytes- childhood disease, rare in adults
 - Chronic Lymphocytic Leukemia (CLL) -cancer of the mature lymphocytes- over age 60 on TEST
- Middle age and older
 - Acute Myeloid Leukemia (AML)- cancer of the bone marrow- affects immature cells (WBCsmyeloblasts, monoblasts, RBCs -erythroblasts and platelets)
 - Chronic Myelogenous Leukemia (CML)- cancer of immature granulocytes
- Subjective
 - Fatigue, infections, bleeding or easy bruising- all
 - Fever- ALL, CLL
 - Weight loss- ALL, CLL, CML
 - Night sweats- ALL , CLL , CML
 - Bone pain- ALL, CML
 - Dyspnea- ALL, CML
 - Early satiety- CLL, CML
 - Angina, headache & confusion- ALL
- Objective
 - Hepatosplenomegaly- all
 - Asymptomatic- CLL
 - Lymphadenopathy, AML, CLL, CML
 - Pallor- ALL, AML, CLL
 - Petechiae- ALL, CLL
 - Increased intracranial pressure- AML
 - Retinal hemorrhages, epistaxis, cranial nerve palsies, meningeal signs, abnormal testicular exam-ALL
 - Skin involvement- ALL, CLL
- Differentials- polycythemia, lymphoma, aplastic anemia, viral diseases, mononucleosis, paroxysmal nocturnal hemoglobinuria, myelodysplastic syndromes

• Diagnostic Reasoning-

- Initial CBC w/Diff, peripheral smear, CMP, US abdomen
- Follow up tests by suspected leukemia type or refer to hematology
 - ALL- Hep B/C, HIV, CMV Ab, LDH, uric acid, CT or MRI head, US scrotum, lumbar puncture, bone marrow & lymph node biopsy
 - AML- ESR, LDH, coagulation profile bone marrow biopsy, lumbar puncture for leukemic cells
 - CLL- immunophenotyping, hemolysis labs, CT scan chest/abdomen/pelvis, PET scan, lymph node biopsy
 - CML- Genetic testing on bone marrow biopsy(Philadelphia chromosome), LDH, uric acid

Type 1 reaction anaphylaxis

- Allergic Reactions Immune-mediated reaction to foreign environmental allergens
 - **Type I** Hypersensitivity IgE mediated reactions
 - Includes anaphylaxis and angioedema
- Subjective

- Atopic disease- exposure to an antigen/allergen followed by fatigue, malaise irritability, itchy and watery eyes, sneezing, rhinorrhea, nasal congestion, nonproductive cough, pruritus, wheezing
- Anaphylactic- exposure to an allergen followed by difficulty breathing, wheeze, fainting or dizziness, may become unresponsive, hives, itching, red face/neck, swollen lips, tongue, throat, & eyes, sensation of throat closure or choking, abdominal pain/cramping, N/V, diarrhea, heart racing

• Objective

- Atopic- exam findings for dermatitis, rhinitis, wheeze, allergic conjunctivitis,
- Anaphylactic- dyspnea, stridor, wheeze, bronchospasm, facial edema (lips, tongue, conjunctiva, periorbital area, and uvula), change in voice quality, hypoxemia, reduced peak expiratory flow, hypotension, hives, facial flushing, syncope, confusion, unconsciousness, hypotonia, incontinence, tachycardia

Rheumatoid Arthritis RA

- What is it
- Clinical manifestations
- Common laboratory and imaging findings that support the diagnosis
- Test to diagnose
- **Subjective** gradual onset over weeks/months with morning stiffness and edema of > 1 hr. in jointsthis gets better with movement, pain, redness, warmth, & swollen smaller joints, low grade fever, generalized body aches, fatigue, weight loss, anorexia. Have major depression
- **Objective** symmetrical involvement- hands, wrists, elbows, ankles, feet, knees, or shoulders, joint edema and tenderness to palpation with limited mobility Chronic disease- boutonniere deformity (hyperextended DIP joint with flexed PIP joint), swan neck deformity (flexed DIP with hyperextended PIP), rheumatoid nodules Extra articular signs- cardiac or pulmonary friction rub, A-fib, HF signs, diminished respiratory excursion, dry crackles, injected sclera, neuropathy, ecchymoses
- **Differential Diagnosis** osteoarthritis, psoriatic arthritis, infectious arthritis, reactive arthritis, gout, Lyme disease, SLE, human parvovirus B19, Sjogren's syndrome, polymyalgia rheumatica, sarcoidosis, various neoplasms
- Diagnostic Reasoning
 - Initial- disease activity score, CBC, CMP, sed rate or CRP, Rheumatoid factor (RF), Anticyclic citrullinated peptide antibody (Anti-CCP), Xray, US
 - Subsequent- Quantitative antinuclear antibody (ANA), uric acid, UA
 - Before treatment- Hep B & C testing, PPD

• Management

- First line- monotherapy with DMARDS (methotrexate preferred)
- +/- biologic agent, TNF- alpha inhibitor, interleukin-6 inhibitor or targeted DMARD such as a JAK inhibitor if needed
- +/- corticosteroids and/or NSAIDS for symptom relief in early disease or with flares
- Avoid live vaccines if taking biologic DMARDS (e.g. Enbrel, Humira)
- Paraffin baths for hand and foot pain
- Give daily folic acid with methotrexate because of anemia
- $\circ \quad \text{Oral corticosteroids for flares}$
- Joint replacement surgery

- See Dunphy Table 62.1. pgs. 1007-1008- Disease-modifying Antirheumatic Drugs (DMARDS)
- Patient education
 - Don't stop treatment without consultation
 - Support groups
 - Stay active
 - Discuss adverse medication side effects
- Follow up
 - Monitor for signs of HF, CAD, interstitial lung disease, skin cancers, infections, depression, and flares
 - o Frequency per managing provider
- Referral
 - Rheumatology at diagnosis for management
 - o Other specialist as dictated by findings of disease involvement
 - OT or PT for functional limitations
 - Psychotherapy as needed

Fibromyalgia

- Clinical manifestations
- Diagnostic criteria
- Risk factors- female, ages 20-60, FH of fibromyalgia, autoimmune rheumatologic disorders (e.g. rheumatoid, lupus) or chronic pain conditions (e.g. osteoarthritis)
- **Subjective** chronic diffuse muscle pain, fatigue unrelieved by rest, sleep and mood disturbance (depression or anxiety), cognitive dysfunction, headaches, paresthesia, stiffness, sensitivity to sensory stimuli (bright lights, odors, noises, medications), symptoms may worsen with lack of sleep, increased anxiety, changes in weather conditions.
- **Objective** tenderness to palpation on exam with hyperalgia, allodynia, and sensory hyperresponsiveness to most things
- Risk factors- female, ages 20-60, FH of fibromyalgia, autoimmune rheumatologic disorders (e.g. rheumatoid, lupus) or chronic pain conditions (e.g. osteoarthritis)
- Differential Diagnosis-chronic fatigue syndrome (CFS), myofascial pain syndrome, vitamin D deficiency, RA, systemic lupus erythematosus, osteoarthritis, hypothyroidism, IDA, chronic liver disease, myositis, polymyalgia rheumatic, ankylosing spondylitis, **Polymyalgia rheumatica**

• Diagnostic criteria

- Clinical diagnosis
- Chronic widespread pain > 3 mos. + associated symptoms (fatigue, sleep disturbance)
- Diffuse tenderness to palpation and no evidence of systemic disease as cause (see Fenstermacher pg 357)
- Widespread pain index (WPI) \geq 7 (out of 19) and symptom severity scale (SSS) \geq 5
- Tests to rule out other diagnoses- CBC, CMP, ESR, TSH, RF, Anti CCP, ANA, Vit. D level
- Management 3 months pain
 - o First line pharmacotherapy- amitriptyline or cyclobenzaprine at HS
 - o 2nd line- duloxetine, milnacipran, pregabalin or gabapentin
 - +/- Analgesics- naproxen or tramadol

- Non pharmacologic therapy- exercise, yoga, T'ai Chi, cognitive behavior therapy and biofeedback, education about disorder
- Education-
 - \circ $\;$ patient and family education about condition and management
 - o sleep hygiene and behavior therapy, adherence to exercise
- Follow up- based on treatment, remissions and exacerbations
- Referral
 - Psychologist/ Psychiatrist at dx onset

Sjogren's Syndrome

- Clinical manifestations
- Symptoms
- Risk factors- female, peaks 20-30's or after menopause, hx of other autoimmune disorders, scleroderma (systemic sclerosis)
- **Subjective** dry eyes, red eyes, burning and itching, and dry mouth, dental caries, fatigue, sleep disturbance, decreased physical capacity, loss of taste and smell, dysphagia, vaginismus, rectal bleeding, joint swelling, pain, low grade fever
- **Objective** vasculitic skin rash, severe dental caries, bad breath, pale and dry mucosa, beefy red tongue, inflammation of the small joints of the hands, assess cardiopulmonary, renal, GI and CNS for other possible manifestations as listed in Dunphy pg. 1014
- Differential diagnosis- hypothyroidism, SLE, Hep C, RA systemic sclerosis, fibromyalgia, depression, HIV, sarcoidosis, drug induced sicca, idiopathic or age related sicca

• Diagnostic reasoning

- Initial tests- Schirmer test, Anti-Ro Anti-La & Anti-60 KD
- Additional tests to consider- CBC, CMP, RF, ANA, sialometry, salivary gland biopsy, specialized eye testing
- Diagnosis with **4** initial criteria
 - Subjective dry eyes > 3 months
 - Dry mouth , waking from sleep to drink > 3 months
 - Schirmer test decreased tear production < 5 mm in 5 min.
 - + antibody testing (anti-Ro and/ or anti- La)
- If reasonable suspicion remains and the above criteria is not met and other disorders are ruled out, the next step is get sialometry. If confirmation still needed, proceed to get a salivary gland biopsy.
- Refer to ophthalmology for specialized eye testing to confirm dx as well.

• Management

- Symptomatic treatment based on symptoms/associated conditions
 - Dry eyes- 1st line -artificial tears or 2nd line cyclosporine drops or 3rd line oral cholinergics + spectacle eye shields, humidifiers, punctal plugs or thermal punctal occlusion
 - Dry mouth- 1st line salivary substitutes or saliva-stimulating products, 2nd line cholinergics
 +humidifiers and moisturizers
 - Fatigue- treat comorbid conditions
 - MSK- acetaminophen or NSAIDs, short courses of corticosteroids for inflammatory arthritis
 - Vasculitis- 1st line corticosteroids 2nd line intravenous immunoglobulin (IVIG)
 - Renal Tubular acidosis- potassium repletion and alkali by mouth
 - Neuropathy- IVIG

- Education- Avoid anticholinergics, antihistamines, diuretics, artificial tears with preservatives, use spectacle eye shields for create a humid environment, take breaks when reading, use room humidifiers, moisturize lips, use products to relieve mouth dryness, sip water frequently, get regular dental checkups
- Follow up- monitor for complications
- Referral optometry, rheumatology, dentist

SLE Systemic Lupus Erythematosus

- Clinical manifestations
- Diagnostic criteria

Lupus symptoms and labs

- Subjective malaise, fatigue, anorexia unexplained weight loss, blurred vision, sleeplessness, depression, painful and swollen joints, vague abdominal pain, or cramping, possibly SOB and inspiratory pain, musculoskeletal joint pain
- Objective- malar "butterfly" rash, photosensitvities, alopecia, scalp exanthema, splinter hemorrhages, periungual erythema, fingertip lesions, lymphadenopathy, Raynaud's phenomenon, asymmetrical, nondeforming, migratory arthritis, swollen joints, impaired cognitive thought processes, peripheral paresthesia and diminished DTRs, systolic murmur, JVD, oral and nasal ulcers, RUQ or RLQ tenderness
- Differential Diagnosis- RA, vasculitis, scleroderma, systemic sclerosis, active hepatitis, drug reactions, or drug induced lupus, polyarteritis, hypothyroidism
- Diagnostic Reasoning- Initial- CBC, activated PTT, CMP or BMP, ESR or CRP, antinuclear antibodies (ANA), double stranded (ds) DNA, Smith antigen, UA, CXR
- Others to consider if symptoms are associated with other manifestations of disease OR refer to specialty providers
 - coombs test, complement levels, antiphospholipid antibodies, joint x-rays, 24 hr urine for protein or spot urine for protein/creatinine ratio, renal ultrasound, ECG

• Management

- General- hydroxychloroquine, +/- NSAIDS, corticosteroids
- With nephritis- + DMARD or immunosuppressant
- With neuropsychiatric illness- may add IVIG, plasmapheresis, antidepressants, antipsychotics, anticonvulsants or antimigraine meds
- Education- provide resources such as the Lupus Foundation of America, avoid a sedentary lifestyle, smoking cessation, eat healthy diet, sun protection, good oral care
- Follow up based on specialty providers who will manage this disorder
- Referral
 - Rheumatology
 - Nephrology
 - Cardiology
 - Neurology
 - Hematology

Mononucleosis

- Clinical manifestations
- Diagnostic work up
- Patient education

- Diag Findings
- Return to work history
- **Subjective-** fever, sore throat, tender, enlarged lymph nodes, very fatigued, nausea, vomiting, anorexia, headache, sometimes a rash
- **Objective** fever > 102.5 F, tender posterior cervical, axillary and inguinal, lymphadenopathy, nuchal stiffness, enlarged tonsils +/- exudate, posterior pharynx erythema, splenomegaly, possible hepatomegaly, fine maculopapular rash posterior cervical lymph nodes
- Differential diagnosis- strep pharyngitis, peritonsillar abscess, acute HIV, leukemia, or toxoplasmosis
- Diagnostic Reasoning
 - Clinical symptoms + CBC, CMP, heterophile antibody test (Monospot), Epstein Barr virus (EBV) specific antibodies (VCA IgM, VCA IgG, EA, EBV EBNA)
 - Can consider- real-time polymerase chain reaction (PCR) test (EBV DNA), US Abdomen, CT Abdomen
- Management
 - Supportive care- OTC antipyretics and analgesics
- Education- no strenuous activity or contact sports for at least 3-4 weeks due to possibility of splenic rupture, rest, stay hydrated, no blood donations with recent illness
- Follow up
 - For 1-2 months to ensure resolution of symptoms
 - Monitor for post-infective chronic fatigue syndrome and other complications listed in Dunphy pg .1025
 - Consider a repeat ultrasound prior to return to sports in athletes
- Referral
 - ER for upper airway obstruction, hemolytic anemia, or thrombocytopenia

Lyme Disease

- Risk factors
- Clinical manifestations Erythema Migrans bulls eye rash
- Subjective- early disease- rash that occurs within 1-2 weeks of a tick bite, may burn or itch, fever, chills, myalgia, later disease- headache, fatigue, neck pain and stiffness progressing to joint pain, memory loss, mood changes
- **Objective** erythema migrans (bull's- eye) rash spreads from source of bite, regional or organ specific abnormalities, joint edema and tenderness to palpation, gait disturbance, encephalopathy, peripheral neuropathy, nuchal rigidity, paresthesias, dysrhythmias, facial palsy
- Differential Diagnosis- tick bite allergy, cellulitis, erythema multiforme, rickettsiosis, ehrlichiosis, babesiosis, tick-borne encephalitis, southern tick associated rash illness (STARI), chronic fatigue syndrome
- Diagnostic Reasoning- 2 step approach
 - If enzyme immunoassay (EIA) or immunofluorescence assay (IFA) positive, follow up with standardized Western blot assay or a second EIA per new FDA clearance (2019)
 - Other tests to consider-
 - For early Lyme disease(within 4 weeks) test for Lyme specific IgM and IgG immunoblot assays in the 2nd confirmatory step
 - ECG
- Management

- Presumptive-for known tick bite postexposure prophylaxis with a single 200 mg dose of doxycycline
- With erythema migrans- 1st line doxycycline 100 mg BID x 10 days or amoxicillin, cefuroxime or pen VK for 14 days
- NSAIDS for arthritis
- Education avoid tick exposure and how to dress when walking in foliage (see Dunphy pg. 1029)
- Follow up
 - In 1-2 weeks for resolution of symptoms
 - in 2-4 weeks during convalescent period with uncertain initial diagnosis
 - For 30 days for those who have removed ticks themselves and received prophylactic antibiotics
- Referral
 - ER for carditis symptoms (syncope, dyspnea or chest pain, AV block or prolonged PR interval)
 - Cardiology
 - Neurology

HIV/AIDS

- Diagnostic reasoning (laboratory findings)
- Diagnostic criteria
- What is the difference between HIV and aids
- Risk factors- > 90% unprotected, high risk anal or vaginal sex, the rest is from IV drug use, maternal to child transmission, blood transfusions before 1985 and needle stick injury
- See Dunphy box pg. 1033 Focus on History: Evaluating Risk of HIV
- **Subjective-** fever, chills, night sweats, weight loss, fatigue, enlarged lymph nodes, diarrhea, oral sores, mouth pain, sinusitis, visual changes, skin rash, neurologic symptoms, cough, SOB or DOE skin rash
- **Objective** Generalized lymphadenopathy, chronic vaginal candidiasis, genital STIs, shingles, seborrheic dermatitis, pruritic popular rashes, tinea corporis or unguium, oral ulcers, angular cheilitis, oral thrush or oral hair leukoplakia, Kaposi sarcoma, encephalopathy, depression, anxiety, retinal lesions, hepato or splenomegaly
- **Differential Diagnosis** Burkitt lymphoma, candidiasis, CMV, coccidiolmycosis, cryptococcus, EBV, herpes simples, influenza, lymphoma, mononucleosis, TB, toxoplasmosis
- Laboratory Diagnosis Options:
 - Rapid oral test or noninvasive buccal swab home test kits
 - Serum HIV enzyme-linked immunosorbent assay (ELISA) + supplemental Serum p 24 antigen
 - 4th gen antibody (ELISA) and antigen (p24) HIV1/HIV2
 - Confirm positive or indeterminate ELISAs or rapid tests with Western Blot
 - Serum p 24 antigen can be used with older generation ELISA tests as the protein is present with viral replication in acute infection
 - Serum HIV RNA PCR- most sensitive test in the window period

*Disease detection tests are subject to window periods where false negatives are possible before antibodies to HIV have occurred- patients may need to retest to ensure a true negative result

- AIDS criteria is a CD4 count < 200 or the presence of an opportunistic infection
 - Pneumocystis jiroveci (carini) pneumonia

- Cryptococcal meningitis
- Recurrent bacterial pneumonia
- Candida esophagitis
- CNS toxoplasmosis,
- **TB**
- Non-Hodgkin or Hodgkin lymphoma
- Progressive multifocal encephalopathy,
- HIV nephropathy
- Kaposi sarcoma
- Invasive cervical cancer
- Late infections with advanced HIV- CMV & disseminated Mycobacterium avium complex
- Labs prior to treatment
 - HIV RNA (viral load)
 - Lymphocyte subset panel, including CD4 count
 - HIV genotype resistance assay
 - Pregnancy test
 - TB test &/ or CXR
 - Comorbid infections- Serum Hepatitis A, B and C serology, STIs (gonorrhea, chlamydia, syphilis), toxoplasma IgG
 - Human leukocyte antigen B*5701 testing
 - CMP, CBC with dif
- Treatment depends on CDC or WHO stage of disease, pregnancy status and concurrent medical conditions
- Antiretroviral therapy (ART) initiated for all patients with detectable HIV RNA regardless of CD4 cell count
- Selected regimen based on resistance testing and pregnancy status
- Tenofovir/emtricitabine (Truvada) is FDA approved for PrEP in high risk adults
- Immunizations Pneumococcal, meningococcal, influenza, Hep B, HPV, TDAP
- Avoid live vaccines (MMR, varicella, zoster, oral polio, typhoid, and yellow fever) with CD4 count < 200
- Prophylactic antimicrobial agents for opportunistic infections *P.jiroveci, M. tuberculosis, T. gondii, M. avium & S,pneumoniae*
- See Dunphy table 63.2, pgs. 1030-1031 CDC HIV Classification System
- Education most important is adequate counseling and advice, avoid unprotected , high risk sex and sharing needles, good nutrition, avoid raw eggs and unpasteurized dairy products, medication education and drug interactions
- Follow up
 - HIV RNA viral load 2-8 weeks after tx initiation and Q 3-6 months until suppressed
 - $^\circ$ HIV RNA, CD4 and CBC Q 3-4 mos. During 1st two years of ART & when CD4 < 300
 - When viral load stable and undetectable CD4, HIV RNA, & CBC, UA annually
 - Annual cervical cytology until 3 negative screens, then every 3 years
 - Annual lipids, fasting glucose and CMP
 - Every 6- 12 months, UA, total/direct bilirubin
- Referral- Infectious Disease, Hepatology, Hematology, Pulmonology, Dermatology