**Gastrointestinal System/Nutrition**

### Inflammatory bowel disease (Crohn’s/UC)
- IBD → recurrent abd pain, fever, fatigue, diarrhea (bloody), weight loss, extra-intestinal sx
  - Antibodies: P-ANCA (UC), ASCA (Crohn dz)
  - Complications: toxic megacolon, obstruction, perf, abscess, fistulas, colon CA (>UC)
  - Tx: 5-ASA (sulfasalazine for colon, mesalamine for small bowel), glucocorticoids, IVF, immune modulators (azathioprine, MTX, cyclosporine, infliximab)

### Ulcerative colitis
- Chronic mucosal inflammation, almost always starts in rectum, inflammation extends continuously (no skip lesions) for variable extent
  - Types:
    - Ulcerative proctitis – bowel inflammation limited to rectum (usually <6in); mildest form
    - Proctosigmoiditis – inflammation involving rectum and sigmoid colon
    - Left sided colitis – continuous inflammation that begins at rectum and extends to splenic flexure of colon
    - Pancolitis – inflammation affecting entire colon
  - Clinical presentation: bloody, mucous diarrhea, fever, abd pain, tenesmus, weight loss
  - Dx:
    - Sigmoidoscopy, colonoscopy: mucosal erythema, granularity, friability, exudates, ulcers
  - Complications: toxic megacolon, colonic perforation, increased risk of colon CA (after 8yrs colonoscopy every 2yrs)

### Crohn’s disease
- Chronic mucosal inflammation affecting any part of GI tract w/skip lesions classically through affected terminal ileum/colon
  - Patterns: inflammatory (80%), structuring (10%), fistulizing (10%)
  - Clinical presentation: fever, abd pain, non-bloody diarrhea, fatigue, weight loss, anorectal fissures/fistulas/abscesses
  - Dx:
    - Sigmoidoscopy/colonoscopy (mucosal erythema w/nodularity or ulcers that are deep/longitudinal, cobblestoning, skip areas, strictures, fistulas)
    - Serologic markers (ASCA – ab against saccharomyces cerevisiae)
    - CT enterography (suspicion of small bowel involvement)
  - Complications:
    - Intestinal obstruction (often w/stricture formation)
    - Intestinal fistulas to bowel/bladder/vagina/skin + abscess formation
    - Increased risk of colon CA

<table>
<thead>
<tr>
<th><strong>ULCERATIVE COLITIS</strong></th>
<th><strong>CROHN’S DISEASE</strong></th>
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<tbody>
<tr>
<td>No skip lesions</td>
<td>Skip lesions</td>
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<tr>
<td>Affects colon only</td>
<td>Affects mouth to anus</td>
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<tr>
<td>Curative via colectomy</td>
<td>Not definitively curative</td>
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### Treatment of Crohn’s & UC
- Medications: 5 aminosalicylates (5-ASA), corticosteroids, immunosuppressants, abx (metronidazole (flagyl), cirprofloxacin), TNF inhibitors
- Surgery
  - Colectomy w/resulting ostomy
    - Curative in UC (limited to colon)
    - Non-curative in CD (skip-lesions → can have dz anywhere mouth to anus)
  - Colon resection
    - Common in CD pts w/stricture, recurrent fistulas and abscesses

### Irritable bowel syndrome
- → chronic abd pain, altered bowel habits, abd bloating, w/ no structural or biochem disorder

### Acute abdomen
- Peritonitis / ascites → perf, ruptured ulcer/appendix/diverticulitis, pancreatitis, SBP (cirrhosis)

### Bowel obstruction
- Obstruction → most commonly due to adhesions, malignancy, then hernia → xray, US, CT

### Appendicitis
- Periumbilical to RLQ pain, N/V, anorexia, fever, pain at McBurney point, (+) Rosving, Psoas, Obturator signs
- CT w/ contrast, US, can do MRI in pregnancy
- Appendectomy
- Antibiotics
### Gastrointestinal bleeding (Upper GI Bleed)

- **Etiologies:** PUD, erosive esophagitis/gastritis, esophageal varices, Mallory-Weiss syndrome, arteriovenous malformation, malignancy
- **Hx:** hematemesis, coffee-ground emesis, melena, hx of vomiting/retching, hx of salicylate, glucocorticoid use, NSAID, anticoagulant use, EtOH abuse, hematemochesia 14%

### Signs/sx’s of dz
- Shock – diaphoresis
- Liver dz – spider angiomas, palmar erythema, jaundice, gynecomastia
- Coagulopathy – petechiae, purpura

### Labs:
- CBC, BUN/Cr, CMP, glucose, coagulation studies, lactate, ECG (BUN/Cr ratio >30 suggests UGI)

### Imaging:
- XR studies limited value, Barium CI (may inhibit EGD)
- EGD for direct visualization of source of bleed, dx study of choice
- NG intubation and aspiration both dx and therapeutic
  - Visual inspection of bloody, maroon, coffee-ground aspirate verifies UGI
  - Negative NG does not exclusively exclude UGI source

### Risk stratification:
- **Low risk:** no major comorbidities, no hx of red hematemesis, no hematochezia, negative NG aspirate, hemodynamically stable at presentation, normal labs
- **High risk:** advanced age, comorbidities, red hematemesis, hematochezia or melena, positive NG aspirate, hemodynamically unstable, abnormal labs

### Initial tx:
- Stabilization

### Other tx:
- **Blood transfusion:** transfuse <7g/L (<9g/L in elderly pts)
- **Correct coagulopathy:** correct if INR elevated, platelets <50,000, severe bleed
- **Omeprazole:** 80mg IV bolus than infusion of 8mg/hr IV; labeled use for ulcer bleeding
- **Octreotide:** 50mcg bolus than 25-50mcg/hr IV; unlabeled use for lower range, use lower doses for elderly
- **Abx:** Ciprofloxacin 400mg IV or ceftriaxone 1g IV; use for cirrhotic pts
- **EGD tx:** used for direct visualization and administration of hemostatic therapy (clips, thermocoagulation, sclerosant injections, epi injections); should be done w/in 24hrs
- **Balloon tamponade:** short-term solution for life-threatening variceal bleeding, consider intubation before procedure
  - Sengstaken-Blakemore tube: 250cc gastric balloon, esophageal balloon, and single gastric suction port
  - Minnesota tube: has added esophageal suction port above esophageal balloon
  - Cx: mucosal ulceration, esophageal/gastric rupture, asphyxiation from tracheal compression
- **Surgery:** indicated for pts w/bleeds refractory to pharmacologic and EGD tx
  - Options: shunts, esophageal transection, gastro-esophageal junction devascularization, percutaneous embolization

### Prognosis
- Difficult to cure, most will die of recurrent disease

### Lactose intolerance
- Inability to digest lactose into glucose and galactose secondary to low levels of lactase enzyme in duodenal brush border
- **Prevalence:** very common (25% white Americans, 75-90% Asian Americans)
- **Clinical presentation:** loose stool, abd bloating and pain, flatulence, nausea, borborygmi
- **Dx:**
  - **Hydrogen breath test** – DX TEST OF CHOICE
    - Pts administered lactose after overnight fast
    - Expired air samples collected before and at 30min intervals for 3hrs
    - Assess rise in hydrogen gas concentrations (rise in H+ concentration = lactase deficiency)
  - Trial of dietary elimination
- **Tx:** dietary adjustment, lactase replacement (lactaid and lactrase)

### Gastric Cancer
- **Etiologies:**
  - High incidence in Korea, Japan, and China
  - Usually occurs after age 60
- **Signs & symptoms:**
  - Early disease is asymptomatic
  - Indigestion, nausea, early satiety, anorexia, and weight loss
  - Advanced: pleural effusions, SBO, bleeding
  - Palpable stomach, Hepatomegaly, Pallor
  - Virchow’s nodes or Sister Mary Joseph nodes
- **Workup:**
  - EGD, Endoscopic US, Barium swallow, CT/MRI
- **Management:**
  - Depends on stage ➔ Resection, chemo, radiation, adjuvants if needed
- **Prognosis:**
  - Difficult to cure, most will die of recurrent disease
Acute Diarrhea (Less than 2 weeks duration)
- Most common causes are infectious agents, bacterial toxins, or drugs
- Other causes to consider: Anal sex: Neisseria gonorrhoeae, syphilis, lymphogranuloma venereum, HSV - Noninfectious: drug reaction, UC, Crohn’s, ischemic colitis, fecal impaction, laxative abuse, radiation colitis, emotional stress
- Inflammatory
  - Invasive or toxin-producing bacteria
  - Causes: shigellosis, salmonellosis, Campylobacter, Yersinia, C. diff, EHEC, Entamoeba histolytica, Neisseria gonorrhoeae, Listeria
  - Blood, pus, fever = fecal leukocytes usually present
  - Diarrhea is typically smaller in quantity, associated LLQ cramps, urgency, tenesmus
  - Workup with stool cultures, C. diff, ova, parasites
- Noninflammatory
  - Viral or noninvasive bacteria
  - Infectious causes: virus, preformed toxin, toxin producing bacteria, protozoa (ETEC, Staph, Bacillus cereus, Clostridium, viruses, Giardia) - Watery, nonbloody = no fecal leukocytes - Diarrhea may be voluminous
  - May have periumbilical cramps, bloating, n/v - Prominence of vomiting suggests food poisoning or viral enteritis
  - Typically, only eval if persists beyond 7 days or worsens
- When to Evaluate Further
  - Signs of inflammatory diarrhea: fever > 101.3, bloody diarrhea, abdominal pain - Passage of > 6 loose stools in 24 hours
  - Profuse watery diarrhea and dehydration
  - Frail older patients, Immunocompromised patients, Hospital-acquired diarrhea
- Tests indicated: fecal leukocytes, routine stool culture, C. diff if recent hospitalization or antibiotics, 3x ova and parasites if > 10 d, travel, community water outbreak, HIV, MSM
- Management
  - Rehydration: 1/2 tsp salt, 1 tsp baking soda, 8 tsp sugar, 8 oz OJ diluted to 1 L with water
  - Antidiarrheals for mild to moderate illness
  - Loperamide (opioid) as long as there is no blood, high fever, or systemic toxicity
  - Bismuth subsalicylates (Pepto Bismol) good for traveler’s diarrhea as it is antibacterial and anti-inflammatory
  - Empiric antibiotic treatment only for immunocompromised, significant dehydration, mod-severe fever, tenesmus, bloody stools, or presence of fecal lactoferrin → cipro, Septra, or doxycycline
  - Send to ED for severe dehydration, severe or worsening bloody diarrhea, severe abdominal pain, signs of sepsis, or worsening diarrhea in patients > 70

Chronic Diarrhea (Greater than 4 weeks’ duration - not attributed to viruses or bacteria other than C. diff)
- Differential and signs/symptoms
  - Osmotic (lactose intolerance or other osmotic agents, factitious Mg overuse or laxative use): stool volume changes with fasting, ↑ stool osmotic gap
  - Secretory (hormonally mediated, factitious, villous adenoma, bile salt malabsorption, meds): > 1L stool per day, little change with fasting, normal stool osmotic gap, nonanion gap metabolic acidosis, hyponatremia
  - Inflammatory (UC, Crohn’s, microscopic colitis, malignancy, radiation): fever, hematochezia, abdominal pain, anemia, hypoalbuminia, ↑ ESR or CRP
  - Meds: SSRIs, cholinesterase inhibitors, NSAIDs, PPIs, ARBs, metformin, allopurinol - Malabsorption: weight loss, elevated fecal fat, anemia, hypoalbuminemia
  - Motility disorders (IBS): systemic disease or prior abdominal surgery - Chronic infections (parasites, AIDS-related)
- Workup
  - Ask if diarrhea occurs at nighttime or while fasting
  - Exclude causes of acute diarrhea, lactose intolerance, IBS, previous gastric surgery, parasitic infections, meds, systemic disease
  - Initial tests: CBC, CMP, Ca, P, albumin, TSH, vitamin A, vitamin D, INR, ESR, CRP, IgA for Celiac
  - Stool studies: ova, parasites, electrolytes, fat stain, occult blood, leukocytes or lactoferrin - Consider antigen detection for Giardia and Entamoeba
  - Consider acid stain for Crypto and Cyclospora
  - Refer for colonoscopy with biopsy
  - Further testing: 24-hour fecal fat, neuroendocrine tumors
- Management
  - Loperamide (Imodium)
  - Diphenoxylate with atropine
  - Codeine and deodorized tincture of opium: only for intractable chronic diarrhea - Clonidine
  - Octreotide: for neuroendocrine tumors and AIDS diarrhea
  - Cholestyramine
### Polyps
- Can be sessile or pedunculated and vary considerably in size.
- Polyps occur most commonly in the rectum and sigmoid and decrease in frequency toward the cecum.
- Multiple polyps may represent familial adenomatous polyposis.
- About 25% of patients with cancer of the large bowel also have satellite adenomatous polyps.
- Adenomatous (neoplastic) polyps are of greatest concern:
  - Tubular adenomas, tubulovillous adenomas (villoglandular polyps), or villous adenomas.
  - Likelihood of cancer is related to size, histologic type, and degree of dysplasia → a 1.5-cm tubular adenoma has a 2% risk of containing a cancer vs a 35% risk in 3-cm villous adenomas.
  - Serrated adenomas = more aggressive.
- Nonadenomatous (nonneoplastic) polyps:
  - Hyperplastic polyps, hamartomas (see Peutz-Jeghers Syndrome), juvenile polyps, pseudopolyps.
  - Inflammatory polyps and pseudopolyps occur in chronic ulcerative colitis and in Crohn disease of the colon.
  - Multiple juvenile polyps (but not sporadic ones) convey an increased cancer risk. The specific number of polyps resulting in increased risk is not known.

### Esophageal
#### Esophagitis
- Most common = GERD.
- 2nd most common = infections in immunocompromised - candida, CMV, HSV.
- Risk factors: pregnancy (GERD), smoking, obesity, ETOH, chocolate, spicy foods, meds (NSAIDs, beta blockers, CCBs).
- **Candida esophagitis:**
  - Linear yellow-white plaques.
  - Treat with PO fluconazole.
- **CMV esophagitis:**
  - Large superficial shallow ulcers.
  - Treat with ganciclovir.
- **HSV esophagitis:**
  - Small deep ulcers.
  - Treat with acyclovir.
- **Eosinophilic esophagitis:**
  - Atopic disease, endoscopy shows multiple corrugated rings +/- white exudates.
  - Treat with steroids via inhaler without spacer.
- **Pill induced esophagitis:**
  - Most commonly caused by prolonged pill contact with esophagus.
  - Common offenders: NSAIDs, bisphosphonates, iron, potassium-chloride, abx (doxycycline, tetracycline, clindamycin), BB’s, CCB’s, vitamin C.
- **Caustic (corrosive) esophagitis:**
  - Ingestion of corrosive substances: alkali, lye, bleach, acids, HCL → diagnose with endoscopy.
  - Treat with supportive measures, pain meds, IV fluids.

#### Hiatal Hernia
- Protrusion of upper portion of stomach into chest cavity due to diaphragm tear or weakness.
- **Type 1:**
  - Sliding hernia → GE junction and stomach slide into mediastinum.
  - Most common, increases reflux, treat the same as GERD.
- **Type 2:**
  - Rolling hernia → fundus of stomach protrudes through diaphragm with GE junction remaining in its anatomic location.
  - Surgical repair to avoid complications.

#### Obesity
- Overweight = BMI 25-29.9.
- Obesity = BMI > 30 → greater risk of DM, stroke, CAD, death.
- **Pharmacologic options** → Catecholaminergics (phentermine, diethylpropion, mazindol): short-term use only.
- **Bariatric surgery** → BMI > 40, or > 35 if obesity complications are present → BMI 35-40, or > 30 if obesity complications are present, surgical repair to avoid complications

#### Motility disorders – achalasia
- Sx: Dysphagia or both solids and liquids (also weight loss, dehydration, regurgitation).
- Dx: *Esophageal manometry = gold standard*, will show increased LES pressure >40mmHg and decreased peristalsis.
- Tx: Decrease LES pressure, botox injection for temporary relief (6-12 months), nitrates, CCBs, pneumatic dilation of LES, esophagomyotomy.
- Most common motility disorder presenting w/dysphagia.

#### Zenker’s Diverticulum
- Progressive out-pouching of pharyngeal mucosa just above upper sphincter.
- Distal diverticula assoc. w/motility disorders.
- Sx: Occur >50yo, transfer dysfunction, halitosis, feeling of neck mass.
- Dx: Barium swallow (collection of dye behind esophagus).
- Tx: Observation if small and asymptomatic, diverticulectomy, cricopharyngeal myotomy.

#### Mallory Weiss Tear
- A tear of the distal esophagus at the gastroesophageal junction, typically occurring after a bout of vomiting.
- Major cause of upper GIB.
- **Risk Factors** → Underlying portal HTN.
- Sx → Middle aged male presenting with hematemesis, may have recent h/o alcohol ingestion.
- Dx → Endoscopy is test of choice.
- Tx → Most will resolve bleeding spontaneously.
  - May require injection or thermal coagulation.
- Prognosis:
  - Risk of rebleeding.
**Esophageal Neoplasms**
- Squamous cell (*90-95% worldwide*): associated with tobacco & ETOH, achalasia, hot beverages, exposure to noxious stimuli, men, nitrates (decreased risk: NSAIDs/coffee)
  - Peaks age 50-70, more common: African Americans
  - Assume in >49yo w/new onset dysphagia
  - MC found in upper 1/3
- Adenocarcinoma (*50-80% in the US*): presents in younger patients and presents early, usually a complication of:
  - GERD/Barret’s, obesity
  - MC found in lower 1/3
- Sx → Solid food dysphagia, weight loss/chest pain, anorexia, cough, hematemesis, Virchow’s node (left supra-clavicular)
- Dx → Upper endoscopy with biopsy
- Tx:
  - Esophageal resection, radiation, chemo (5-FU)
  - Commonly spreads to the mediastinum
- Prevention: *Screen patients with Barret’s every 3-5 years*

**Esophageal Webs**
- Thin membranes of mucosa and submucosa located in mid to proximal esophagus
- Can occur w/Plummer-Vinson syndrome
- Dx: Barium swallow
- Tx: dilation

**Schatzki ring**
- Stricture near GE junction
- Most common cause of intermittent dysphagia with solid food
- Commonly associated with hiatal hernia
- Sx: Most asymptomatic, presents with food impaction
- Dx: Barium swallow
- Tx: Dilatation

**Plummer-Vinson Syndrome**
- TRIAD: Dysphagia + esophageal webs + iron deficiency
- Sx → Atrophic glossitis, angular cheilitis, koilonychia, splenomegaly
- Most common: in Caucasian women 40-70yo
- Iron repletion may lead to resolution of the dysphagia, dilation may also be required if significant obstruction,
- Recognition is important due to risk of developing esophageal or pharyngeal squamous cell carcinoma

**Esophageal Strictures**
- Thin membranes (mid/upper esophagus)
- Congenital or acquired (eosinophilic esophagitis)
- Leads to dysphagia
- Develops d/t scarring from GERD, inflammation
- Occur in distal esophagus proximal to GE junction
- Sx: → Build over years, typically noted w/solids only
- Dx: → Barium esophagram/swallow, r/o malignancy w/EGD
- Tx: → Endoscopic dilation of the area

**Esophageal Varices**
- Dilation of gastroesophageal collateral submucosal veins
- Complication of *portal vein HTN*
- 5-11% UGIB (can lead to hypovolemia if severe)
- Risk factors
  - Cirrhosis - mortality rate is 30-50% in first bleed
  - 90% pts w/cirrhosis develop varices, 30% of varices bleed, 70% of bleeding varices re-bleed in 1st year
  - Mortality rate 1st bleed – 30-50%; mortality rate re-bleed – 33%
  - Portal vein thrombosis in kids
- Sx’s: UGIB (5-11% present as UGIB, hematemesis, melena, hematochezia), sx’s of hypovolemia (d/t blood loss)
- Dx:
  - Upper endoscopy (enlarged veins)
  - Red wale markings & cherry red spots
- Tx:
  - Stabilize patient (large bore IV/transfusion, vitK, FFP)
  - Endoscopic ligation – tx of choice, +/- sclerotherapy
  - Pharmacologic: octreotide (vasoconstriction), vasopressin
  - Balloon tamponade → indicated for bleeding refractory to EGD ligation or pharmacologic tx, fast bleeding, temporary management before surgical decompression
    - Increased risk of esophageal perforation, ulceration, aspiration pneumonia
  - Surgical decompression - trans jugular intrahepatic portosystemic shunt (*TIPS*) if endoscopic ligation and pharm does not work,
    - Contraindicated with infection or hepatic encephalopathy
  - Devascularization and embolization – indicated for severe bleeds or cases of thrombosis
- Prevention of re-bleed
  - 70% re-bleed within 1st year
  - Beta-blockers - treatment of choice in primary prophylaxis of re-bleeds (Propranolol, nadolol)
  - Isosorbide - long acting vasodilator
  - Abx prophylaxis: floroquinolones (norfloxicin), ceftriaxone

**Esophageal Perforation**
- Most commonly: left posteroalteral wall of distal esophagus
- Causes of perforation:
  - Iatrogenic
  - Boerhaave’s syndrome: full thickness rupture
  - Mc cause sudden, forceful emesis d/t bulimia, EtOH
  - Trauma: penetrating more common than blunt
  - FB, Infection, Tumor
  - Aortic pathology: aneurysm, aberrant right subclavian artery
  - GI pathology: Barrett’s esophagus, Zollinger-Ellison syndrome
- Sx’s: acute may radiate to back and shoulders
- PE: Abd rigidity w/hypotension, fever, tachycardia, tachypnea
  - Cervical subQ emphysema, Pleural effusions
  - Hamman’s crunch (air being moved by heart beat)
- Dx: XR can suggest dx, CT chest or emergent EGD confirms
- Tx: prompt detection, parenteral abx, emergent surgery
### GERD
- Reflux of gastric contents into esophagus via LES \(\rightarrow\) heartburn, dysphagia, cough, laryngitis, asthma, chest pain
- **Etiologies:**
  - Decreased pressure of LES: high-fat food, nicotine, ethanol, caffeine, meds (nitrates, CCB, estrogen anticholinergics, progesterone) pregnancy
  - Decreased esophageal motility: achalasia, scleroderma, presbyesophagus, DM
  - Prolonged gastric emptying: medicines (anticholinergics), outlet obstruction, diabetic gastroparesis, high-fat food
  - Hiatal hernia
- **Sx:**
  - Pain, heartburn, odynophagia, dysphagia, regurgitation, hyper-salivation, pain and discomfort with meals, diaphoresis, pallor, n/v
  - Symptoms can be exacerbated by head-down position or increase in intra-abd pressure; may be temporarily relieved by antacids
  - Radiation may mimic cardiac pain and may occur with exertion or rest; pain may radiate to one or both arms, neck, shoulders, back
- **Less common presentations:** asthma exacerbation, sore throat, ear/nose/throat symptoms, dental erosion, vocal cord ulcers and granulomas, laryngitis w/hoarseness, pain and discomfort with meals, diaphoresis, pallor, n/v
- **Dx:**
  - EGD, manometry (decreased LES pressure), 24h ambulatory pH monitoring (gold standard)
- **Complications:**
  - Strictures, dysphagia, inflammatory esophagitis, Barrett’s esophagus \(\rightarrow\) adenocarcinoma
- **Tx:**
  - Rx = avoid triggers, antacids (Tums), PPIs (omeprazole), H2 blockers (Ranitidine/Zantac)
  - Lifestyle modifications – elevation of head of bed, avoid lying down 3hrs after eating, eat small meals, avoid trigger foods, decrease fat and EtoH intake, weight loss, smoking cessation
  - Antacids and OTC H2RA’s PRN
  - H2RA’s, PPI’s, prokinetic agents
  - Nissen fundoplication if refractory

### Peptic Ulcer Dz
- **PUD mc cause of UGIB**
- **Gastric ulcer \(\rightarrow\)** decreased protective factors (mucus, bicarbonate, prostaglandin, blood flow)
  - GU more common in elderly, 4% caused by gastric cancer or become malignant
  - GU: food-provoked dyspepsia/indigestion; pain 1-2h post-meals, weight loss
- **Duodenal ulcer \(\rightarrow\)** increased damaging factors (acid, pepsin)
  - DU 4x more common than gastric ulcers, >90% in first portion, typically benign
  - DU: ulcer-like; acid provoked dyspepsia; relief w/food, antacids, anti-secretory agents; worse before meals or 2-5h post-meals, nocturnal sx’s
- **Etiologies:**
  - H pylori infection
  - NSAIDs: ASA, indomethacin, ketorolac
  - Zollinger Ellision Syndrome
  - Other: EtOH, smoking, stress (burns, trauma, surgery), elderly, malignancy, steroids
- **Sx:**
  - Asymptomatic, dyspepsia 80% (burning, gnawing epigastric pain), sx’s usually worse at night, +/- n/d
- **Dx \(\rightarrow\)** EGD gold standard
  - Biopsy r/o malignancy \(\rightarrow\) used in pts with alarm sx (>50yo, anorexia, weight loss, anemia, dysphagia)
  - Barium swallow option for pts unable to do EGD
- **H pylori testing:**
  - EGD w/bx + rapid urease test = gold standard
  - Urea breath test – H pylori converts urea to CO2, can be used to confirm eradication after therapy
  - H pylori stool antigen \(\rightarrow\) >90% specific
  - Serologic antibodies – only useful in dx, not confirming eradication
- **Complications:**
  - Bleeding: melena, hematemesisis, dizziness, hematomaesthesia, pallor, weakness, dyspnea, anemia
  - Perforation: sudden onset severe diffuse abdominal pain, rigid abdomen, rebound tenderness (DU)
  - Penetration: pain radiation to back, not relieved by food/antacids (GU)
  - Obstruction: vomiting hallmark, cramping abdomen pain, nausea
- **Tx:**
  - H pylori (+): Clarithromycin + amoxicillin + PPI (CAP)
  - H pylori (-): PPI, H2RA, misoprostol, antacids, bismuth, sucralfate
  - Parietal cell vagotomy if refractory, bilroth II associated w/dumping syndrome
### Gastric Neoplasms
- **Adenocarcinoma** MC worldwide (90%), 4% lymphomas and leiomyosarcomas
- **Risk factors**
  - H. pylori = biggest
  - Salt, cured/smoked/pickled foods containing nitrites (converted by H. pylori), pernicious anemia, chronic atrophic gastritis, achlorhydria, smoking, EtOH, blood type A
  - Most common in males >40, presents late
- **Sx:**
  - Indigestion, weight loss, early satiety, abdominal pain/fullness, nausea, post-prandial vomiting, dysphagia, melena, hematemeses
  - Pts often have iron deficiency anemia
- **Signs of metastasis:**
  - Virchow’s node - supraclavicular
  - Sister Mary Joseph’s node - umbilical
  - Ovarian mets = krukenberg tumor
  - Palpable node on DRE = blumer’s shelf
  - Left axillary lymph node = irish sign
- **Dx**
  - Upper endoscopy w/ biopsy
    - Linitis plastica = diffuse thickening of stomach wall due to infiltration (worst kind)
- **Tx:**
  - Gastrectomy, XRT & chemotherapy
  - Prognosis → poor

### Gastritis
- **Etiologies**
  - H. pylori = most common
  - NSAIDs = 2nd most common - disrupts mucosal barrier via prostaglandin inhibition
  - Acute stress (critically ill patients)
  - ETOH, bile salt reflux, meds, radiation, trauma, corrosives, ischemia, pernicious anemia, portal HTN
- **Sx**
  - Usually asymptomatic, can cause UGIB (hematemesis & melena)
  - Epigastric pain
  - Nausea, vomiting, anorexia
- **Dx**
  - Endoscopy - shows thick edematous erosions <0.5cm, h. pylori testing
  - Tx
    - H. pylori → *clarithromycin + amoxicillin + PPI*, metronidazole if allergen to PCN
    - NOT h. pylori -- PPI, antacids
- **Prophylaxis:** PPI, H2 blockers

### Gastroenteritis
- Inflammation of the lining of the intestines caused by a virus, bacteria or parasites.
- Viral gastroenteritis is the second most common illness in the U.S., often a norovirus infection → It spreads through contaminated food or water, and contact with an infected person. The best prevention is frequent hand washing.
- **Sx:**
  - Diarrhea, abdominal pain, vomiting, headache, fever and chills. Most people recover with no treatment.
  - The most common problem = dehydration
  - Dehydration is most common in babies, young children, the elderly and people with weak immune systems.

### Liver
#### Acute Hepatic Failure
- **Characteristics:** acute liver injury, hepatic encephalopathy, impaired synthetic function (elevated INR, prolongation of PT)
- **Prognosis** determined by presence of hepatic encephalopathy and jaundice
  - Hyperacute: <7days (good prognosis 36%)
  - Acute: 7-21 days (good prognosis 7%)
  - Subacute: 21 days – 26wks (good prognosis 14%)
- **Etiology**:
  - Clinical Etiology – infectious, vascular, toxic, metabolic
  - Morphologic classification – massive hepatic necrosis, chronic liver dz, hepatic dysfunction w/out necrosis
  - Most commonly acetaminophen toxicity (39-46%), indeterminate (14%), drug rxn (12%), Hep B (7%), Hep A (3%)

<table>
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<tr>
<th>Acetaminophen</th>
<th>Idiosyncratic drug reactions</th>
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<td>Viral Hepatitis</td>
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<tr>
<td>Autoimmune hepatitis</td>
<td>Wilson disease</td>
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<tr>
<td>Ischemic hepatopathy</td>
<td>Budd-Chiari syndrome</td>
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<tr>
<td>Veno-occlusive disease</td>
<td>Acute fatty liver of pregnancy and HELLP</td>
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<td>Sinusoidal obstructive syndrome</td>
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<tr>
<td>Malignant infiltration</td>
<td>Partial hepatectomy</td>
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<tr>
<td>Toxin, mushrooms</td>
<td>Sepsis</td>
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<tr>
<td>Heat Stroke</td>
<td>Hemophagocytic lymphohistiocytosis</td>
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<table>
<thead>
<tr>
<th>Grade</th>
<th>Symptoms</th>
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</thead>
<tbody>
<tr>
<td>I</td>
<td>AMS, mild confusion, slurred speech, disordered sleep</td>
</tr>
<tr>
<td>II</td>
<td>Lethargy, moderate confusion</td>
</tr>
<tr>
<td>III</td>
<td>Marked confusion (stupor), incoherent speech, awakes with stimulation</td>
</tr>
<tr>
<td>IV</td>
<td>Coma, unresponsive</td>
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</tbody>
</table>
• **Symptoms of Acute Hepatic Failure:**
  - 80-90% destruction before sx
  - Initially symptoms are nonspecific → fatigue/malaise, lethargy, anorexia, N/V, RUQ pain, jaundice, abdominal distention due to ascites
• **Complications:**
  - Cerebral edema, coagulopathy, hemodynamic instability, respiratory or renal failure, metabolic disturbances, sepsis
• **Dx:**
  - **Labs:**
    - Markedly elevated AST and ALT (>5000 units/L)
      - Micro-vesicular steatosis (fatty liver of pregnancy) >300 units/L
      - Will trend downward if condition improving
    - Elevated bilirubin
    - Low platelet counts
    - INR/PT will improve/worsen depending on disease progression
    - AKI can occur 30-50%
  - Labs based on disease process →
    - Acetaminophen: very high AST and ALT, modest increase in bilirubin, high INR
    - Ischemic hepatic injury: very high AST and ALT, elevated LDH
    - Hep B: high AST and ALT, ALT>AST
    - Wilson’s disease: coombs negative, hemolytic anemia, high AST and ALT, low alk phos and bilirubin, rapidly progressive RF, low uric acid levels
    - Acute fatty liver of pregnancy/HELLP: high AST and ALT, elevated bilirubin, low platelets, hemolytic
    - HSV: very elevated AST and ALT, leukopenia, low bilirubin
  - **Imaging**
    - CT abd: heterogeneity of liver parenchyma, hepatomegaly, ascites, malignant infiltration, vein occlusion
    - Doppler US: Budd Chiari, portal HTN, hepatic steatosis, hepatic congestion, underlying cirrhosis
    - Head CT/MRI: cerebral edema
    - CXR: pulmonary edema
    - Echo: cardiac dysfunction
  - **Biopsy** (indicated if serology unclear) → performed transjugular, can identify malignancy, autoimmune hepatitis, Wilson, acute fatty liver of pregnancy
• **Treatment**
  - Tx generally supportive
    - Admit to ICU, correct metabolic/electrolyte abnormalities, nutritional support
  - Tx underlying cause
  - Transplantation
• **Prognosis**
  - Survival >60%
  - 40% will survive without need of transplant
  - Highest cause of death sepsis and cerebral edema
  - Mortality rate for fulminant hepatic failure w/severe encephalopathy approx. 80%

**Viral Hepatitis – Rapid Review**
- **HAV:** fecal-oral, shellfish, alone (no carrier), asymptomatic, acute
- **HBV:**
  - HBsAg: active and chronic infection
  - Anti-HBs: recovered or immunized
  - Anti-HBc IgM: early marker of infection, positive in window period
  - Anti-HBc IgG: best marker for prior HBV
  - HBeAg: high infectivity
  - Anti-HBeAb: low infectivity
- **HCV:** IVDA, chronic, cirrhosis, carcinoma, carrier
- **HDV:** dependent on HBV coinfection
- **HEV:** fecal-oral (enteric) high mortality rate among pregnant (expectant) patients, epidemics,
  - HAV and HEV are fecal-oral: “The vowels hit your bowels”
- **Autoimmune hepatitis:** young females
- **Alcoholic hepatitis:** moderate transaminase elevation, AST>ALT
### Hepatitis A
- **Transmission**: fecal-oral from ingestion; person-person
- **Incidence**: declined 90% between 1995-2006 most likely due to vaccination (2 vaccines children 12-23mo 6mo apart)
- **RF's**: institutionalization, daycare, MSM, foreign travel
  - Booster 2-4wks before traveling to endemic areas (India, Africa, Central/South America)
- **Incubation period**: 2-6wks
- **Clinical presentation**:
  - Prodrome – mild flu-like illness
  - Icteric phase – dark urine, pale stool, jaundice, pain
- **Dx**: NAT (detection of anti-HAV IgM ab's)
- **Tx**: supportive care, post-exposure prophylaxis (if >40yo, pooled gamma-globulin given w/in 2wks of exposure, also given to household contacts)
- **Prognosis**: excellent long-term immunity; rarely lasting sx

### Hepatitis C
- **Transmission**: percutaneous blood exposure, STD (<5%)
- **RF's**: transfusion prior to 1992; IV DU, hemodialysis, healthcare worker, screen if born between 1945-1965
- **Clinical presentation**: often asymptomatic
  - **Dx**:
    - Serologic markers →
      - HCV RNA (viral load) – usually seen in serum 7-21 days after exposure
      - Anti-HCV ab (anti-HCVab) – at 1-3-months
    - Genotypes
      - Genotype 1a/b – most common (70%); best response to medications and prognosis
      - Genotype 2 and 3 more difficult to treat
  - **Tx**:
    - Alpha IFN + ribavirin for 24-48wks
    - New therapy → achieve SVR (undetectable viral load at 12wks) 95-97%
    - Harvoni (Ledipasvir/Sofosbuvir) – approved for Genotype 1a/b → Combination pill = X12wks
    - Zepatier (Grazoprevir/Elbasvir) – approved for Genotype 1a/b and 4
    - Solvaldi + ribavirin – approved for Genotypes 2/3
- **Course of infx**: subclinical → icteric → acute → asx chronic
  - **Tx**:
    - Goals –prevention of progression to cirrhosis, liver failure, HCC
    - Begin tx if HBV DNA >20,000 IU/mL and ALT 2X upper limit of normal
    - PEG-IFN 180mcg subQ injection weekly X48wk → SE's: depression, suicidal thoughts, poor compliance
    - Entacavir 0.5mg PO daily X 48wks
  - **Vaccine recommendations** → 3-part vaccination series at 0, 1, 6mo, all healthcare workers revaccinated if titers neg
  - **Post-exposure prophylaxis**: HEP B immunoglobulin (HBIG) if no previous HEP B vaccination or inadequate response
- **Prognosis**: 95-99% healthy, immunocompetent adult pts have favorable course and recovery completely if compliant w/meds

### Hepatitis D – must have concurrent infection with Hep B
- **Transmission**: percutaneous blood exposure, STD
- **RF's**: IV DU, multiple sexual partners
- **Course of infx**: Strong association w/HEP B → almost always co-infected (93-95%)
- **Dx**: serologic markers
  - HBV DNA = first marker for positive infectivity
  - HbsAg & HbeAg → chronic infx = HbsAg >6mo
    - HbsAg pos. w/exposure
    - Anti-HBc IgM follows elevation of HBV DNA, SVR → recovery → seroconversion to antibodies
    - PEG-IFN 180mcg subQ injection weekly X48wk → SE's: depression, suicidal thoughts, poor compliance
    - Entacavir 0.5mg PO daily X 48wks
- **Prognosis**: 95-99% healthy, immunocompetent adult pts have favorable course and recovery completely if compliant w/meds

### Hepatitis E
- **Transmission**: fecally – contaminated water within endemic areas (China, Nepal, Southwest Frances, North African)
  - Can also be transmitted by animals
- **Incubation period**: 15-60 days
- **Clinical presentation**:
  - Prodrome – mild flu-like symptoms
  - Icteric phase – dark urine, pale stools, jaundice
- **Dx**: serologic markers
  - Positive HEV RNA, HEV ag, anti-HEV ab (IgM)
- **Tx**:
  - Mainly preventative – clean drinking water, good sanitation, proper personal hygiene
  - Supportive care
  - Self-limited, will resolve within 5-6wks
- **Vaccination**: given 2-4wks before traveling to endemic areas
- **Prognosis**: excellent; no risk cirrhosis/fulminant hepatic failure; long-term immunity following infection

### Hepatitis B
- **Transmission**: body fluids (blood, saliva, semen, vaginal secretions) → Perinatal, sexual, IV DU, blood transfusions
- **Incidence**: worldwide problem → 1/3 global population infected, #1 cause of cirrhosis and HCC worldwide
- **RF's**: multiple sex partners, IV DU
- **Incubation period**: 1-6mo
- **Clinical presentation**:
  - Subacute phase – anorexia, N/V, low-grade fever, myalgia, fatigue, RUQ pain
  - Icteric phase – dark urine, pale stools, jaundice
  - Chronic phase – flu-like sx’s (same as Subacute)
  - Extra-hepatic manifestations – uticaria, joint pain, polyarteritis, nodosa, glomerulonephritis
- **Course of infx**: subclinical → icteric → acute → asx chronic
  - **Hep B infx** → cirrhosis → HCC
  - **Dx**: serologic markers
    - HBV DNA = first marker for positive infectivity
    - HbsAg & HbeAg → chronic infx = HbsAg >6mo
      - HbsAg pos. w/exposure vaccine as well
    - Anti-HBc IgM → HbsAg seroconversion
    - HBV DNA X3, HBV RNA X100
    - HBV DNA undetectable at 48wks
    - Entecavir 0.5mg PO daily X 48wks
  - **Post-exposure prophylaxis**: HEP B immunoglobulin (HBIG) if no previous HEP B vaccination or inadequate response
- **Prognosis**: 95-99% healthy, immunocompetent adult pts have favorable course and recovery completely if compliant w/meds

### Hepatic carcinoma
- Chronic asymptomatic carriers of HepB are at increased risk
- Most cases of HCC are because of either a viral hepatitis infection (Hepatitis B or C), metabolic toxins such as alcohol or aflatoxin, conditions like hemochromatosis and alpha 1-antitrypsin deficiency
- Occurs most commonly in countries where hepatitis B infections are common
### Alcoholic hepatitis

- **Progressive, inflammatory liver injury assoc. w/long-term heavy ETOH intake**
  - >6 drinks/day for men; >4 drinks/day for women
  - Most common precursor of cirrhosis
- **Clinical presentation:**
  - Often asx
  - Severe cases have acute onset of jaundice, hepatomegaly, portal HTN
  - Impairment of liver function → *elevated AST (marker malnutrition), low serum albumin, thrombocytopenia, elevated INR (coagulopathy)*
- **PE:** hepatomegaly/splenomegaly, peripheral edema and ascites (fluid wave), asterixis (hand flapping), spider angiomas
- **Dx:** clinical dx, no imaging necessary
  - Clinical presentation:
    - heavy ETOH intake
    - Progressive, inflammatory liver injury assoc. w/long-term heavy ETOH intake
  - Onset usually 5 days
- **Tx:**
  - Abstinence of alcohol
  - Supplemental vitamins and minerals
    - Folate, thiamine, MVT (banana bag in ER)
    - Oral supplementation can be given in office
    - Prevents Wernicke’s encephalopathy
    - Neurological disorder caused by thiamine deficiency (alcoholics)
    - Characterized by confusion, abnormal eye movements, wobbly gait
  - Medication reconciliation
  - Onset usually 5-90 days of starting drugs
  - Asx ALT elevation (if ALT >3X ULN should repeat test w/in 72hrs)

### Drug-induced hepatitis

- **Incidence:** >900 drugs, toxins, herbs implicated; drugs account for 20-40% fulminant hepatic failure
- **Most common hepatotoxic meds:**
  - Acetaminophen (Tylenol)
  - Abx – amoxicillin, cipro, erythromycin, TMP/SMX
  - Anti-arrhythmics – amiodarone
  - Antifungals – fluconazole, terbinafine (always cause LFT elevation; start topical for toenail fungal infxn)
  - Anticonvulsants – carbamazepine, phenytoin, valproic acid
  - TB meds – INH, rifampin
  - OCP’s
  - Statins (check LFTs at 3mo and 6mo → if not-elevated don’t need to recheck, if elevated will continue to rise; change to Ctrisor – has least chance of elevation)
  - Herbal meds/weight loss pills – Japanese, Asian populations
  - Recreational drugs – ecstasy, cocaine
- **Dx:**
  - Medication reconciliation
  - Onset usually 5-90 days of starting drugs
  - Asx ALT elevation (if ALT >3X ULN should repeat test w/in 72hrs)

### Autoimmune hepatitis

- **Chronic liver dz of unknown cause; characterized by continued hepatocellular inflammation and necrosis**
  - 20% tendency to progress to cirrhosis
- **Incidence:** most commonly occurs in women (15-25yo and 45yo); strong assoc. w/other autoimmune conditions (RA, UC)
- **Clinical presentation:** dark urine, pale stools, anorexia
- **PE:** hepatomegaly/splenomegaly, scleral icterus, spider angiomas, ascites, encephalopathy
- **Diagnosis:**
  - ALT/AST >5X normal
  - +ANA or +ANCA, +anti-SMA (smooth muscle Ab)
- **Treatment:**
  - Corticosteroids (chronic prednisone therapy 20-40mg/day)
  - Immunosuppressant therapy (often added for flares; azathioprine (6MP) 50-100mg/day)

### Wilson Disease

- Autosomal recessive disorder of copper metabolism that causes excessive deposition of copper in liver, brain
- **Incidence:** usually occurs between 12-23yo
- **Clinical presentation:**
  - Fatigue, ascites, jaundice (neurologic: drooling, gait disturbance, tremor, mask-like faces)
  - Ophthalmologic – Kayser-Fleischer rings (pathopneumonic of Wilson’s – copper deposition in cornea)
  - Encephalopathy

### Non-alcoholic fatty liver disease

- **Condition caused by fatty infiltration of liver (steatosis) not related to alcohol - can result in liver failure**
- **Incidence:** increasing prevalence due to rise in obesity → 90% BMI 38-39 or greater; affects 10-20% Americans; leading cause of elevated LFTs in adults
- **RFs:** metabolic syndrome → obesity, glucose intolerance/DM, HTN, HLD
- **Clinical presentation:** typically asx, usually incidental finding
- **Dx:** dx of exclusion → Other causes of hepatitis
- **Tx** → Lifestyle modifications – weight loss
  - Avoidance hepatotoxins – alcohol, Tylenol
  - Tx HLD – statins, fibrates
  - Tx DM – A1c goal <7% (glucose >126 but normal A1c put on metformin 500mg 1X daily for prevention)
  - Hepato-protective – betaine, vitamin E, ursodeoxycholic acid

### Non-alcoholic steatohepatitis

- **Condition characterized by steatosis + inflammation and hepatocellular ballooning/other evidence of hepatocyte injury**
- **Incidence:** affects 2-5% of Americans
- **RFs:** metabolic syndrome → obesity, glucose intolerance/DM, HTN, HLD; NAFLD dx
- **Clinical presentation:**
  - Typically asx, often incidental finding
  - May present w/fatigue, mild RUQ pain
  - Tx: only definitive dx = liver bx (invasive, complications)
  - Steotest = newer test; calculates fat necrosis score
- **Tx:** same as NAFLD
- **Prognosis:** 15% will progress to cirrhosis
Cirrhosis

- Late stage of hepatic fibrosis with changes in hepatic architecture (generally considered irreversible)
- Usually hard/shrunken liver, collagen spreads throughout liver
- ETOH may stimulate cells to produce more collagen
- 2 most common causes in the US are ETOH and HepC, also can be caused by hemochromatosis and NASH
- Symptoms:
  - Anorexia, weight loss, weakness, jaundice, pruritis, UGIB, ascies, spider angioma, splenomegaly, digital clubbing, asterixis, systemic hypotension
- Clinical findings:
  - Fecotor hepaticus – musty, “sweat and sour” smell or urine and breath
  - Impaired estrogen metabolism – palmar erythema, spider angioma, hypogonadism/gynecomastia
- Diagnosis:
  - Presence of ascites, thrombocytopenia, spider angioma predictive of cirrhosis
  - Predictive scales → Bonacini, Lok
  - Can be made clinically
  - Definitive dx made w/bx (gold standard)
  - Determine underlying cause (will affect tx)
  - AST/ALT moderately elevated
  - Alk Phos – elevated
  - GGT – elevated
  - Can be made clinically given constellation of symptoms, also can use CT
  - When diagnosing cirrhosis one needs to determine the underlying cause because it may change treatment
  - Hepatology should be involved early
  - Imaging →
    - Abd imaging may show shrunken, irregular, nodular liver; evidence portal HTN
      - US most common, usually not sp/sn enough alone
- Management
  - ABSTAIN FROM EOTH
  - Treat underlying cause
    - Anti-virals for Hepatitis C
    - Abstain from ETOH
  - Vaccinations for Hepatitis A and B if needed
  - Avoid hepatotoxins
  - Adjust medications, Avoid APAP
- Complications: variceal hemorrhage, portal HTN, ascites, spontaneous bacterial peritonitis, hepatic encephalopathy, hepatocellular carcinoma, hepatorenal syndrome, hepatopulmonary syndrome, hepatoperoxyroax, portal vein thrombosis, cirrhotic cardiomyopathy
- Transplant
  - Definitive treatment for decompensated cirrhosis is liver transplantation (refer early)
  - Criteria for transplant
    - Irreversible fatal disease
    - No absolute contraindications
      - No active drinking, extrahepatic CA, advanced chronic disease, active sepsis, or unfavorable anatomy
    - Compliance with long-term care
    - Financial security
  - Many specialties involved in listing for transplant
  - Allocation by UNOS (United Network of Organ Sharing)
- Prognosis
  - Median survival for compensated pts >12yrs
  - Median survival for decompensated pts <6mo
  - Complications + ongoing drinking <50% survival rate at 5yrs
# Gallbladder/biliary

## Cholelithiasis/Colecystitis/Cholangitis

- **Cholelithiasis** → biliary colic w/ radiation to back or R shoulder (80% are cholesterol stones)
  - Meperidine (Demerol) or NSAIDs, elective cholecystectomy
- **Cholecystitis** → RUQ pain, fever, N/V, fatty food intolerance, jaundice, radiation to back or shoulder, (+) Murphy sign, ↑ leukocytosis w/ L shift
  - US* (stones, gallbladder wall thickening, dilated ducts), HIDA scan, CT
  - Refer to hospital → NPO, IVF, opioids or ketorolac, empiric abx, cholecystectomy
- **Cholangitis** → Charcot Triad = fever, jaundice, RUQ pain
  - + hypoTN and AMS = Reynold Pental → suppurative cholangitis

### Overview

- Two types: cholesterol vs. pigmented
  - Cholesterol (75%)
  - Pigmented (25%): black/brown; calcium bilirubinate, other Ca salts
- 5 F’s: fat, female, forty, fertile, flatulence

### Clinical presentation:

- Obstruction at any level by stones/sludge can causes sxs
  - Possible locations: common hepatic duct, cystic duct, common bile duct, ampulla of vater
  - 50% asymptomatic
  - Other 50%: 35% develop biliary colic, 15% develop acute dz
  - **Biliary colic**: contraction of gallbladder during transient obstruction of cystic duct by gallstones
    - **Non-specific sxs**: dyspepsia, fatty food intolerance, bloating/flatulence, heartburn, belching
  - **Acute cholelithiasis**: persistent obstruction of cystic duct leading to inflammation or infection
    - **Sxs**: acute onset RUQ pain for several hrs, gradual increase in severity, typically localizes to RUQ/epigastrum, radiation to right lumbar, scapula, shoulder possible, N/V, anorexia, no spontaneous resolution
    - **PE**: (+) Murphy sign, fever (mild jaundice, palpable gallbladder less common)
    - **Complications**: emphysematous cholecystitis (DM, elderly, immunocompromised), empyema, gangrene, gallbladder perforation
  - **Cholangitis**: infection of biliary tract
  - **Pancreatitis**: may result from obstruction of distal common bile duct

### Diagnosis:

- **Asx** → LFTs: “cholestatic pattern”: increased Alk Phos, increased direct bili, normal/mildly elevated AST & ALT
- **Biliary colic**
  - Imaging → **US** (95% sn/sp, 20% if CBD) – stones have bright surface echo and acoustic shadow, EUS and MRCP (accuracy of 90-95% for cholelithiasis and CBD stones)
- **Acute cholecystitis**
  - US – presence of gallstones, pericholecystic fluid, gallbladder wall thickening, (+) US Murphy sign, CBD size (>7mm dilated in adults)
  - HIDA scan – radionucleotide scan; if gallbladder fills w/isotope AC highly unlikely (not blocked)

### Management:

- **Asx** → Monitor
  - Prophylactic cholecystectomy if increased risk of complications
    - DM → increased risk of m/m from acute cholecystitis
    - Calcified (porcelain) gallbladder or large polyps → increased risk for gallbladder carcinoma
    - Sickle cell: hepatitis crisis
    - Children
    - Native Americans → increased risk of gallbladder cancer
- **Biliary Colic**
  - Surgical → cholecystectomy (open if adhesions, obesity, cirrhosis)
    - Most pts need to undergo, performed in 24-48hrs following presentation most commonly
  - **Non-surgical**
    - Ursodeoxycholic acid - dissolves gallstone, reserved for non-surgical candidates, life-long administration
    - Abx → Indicated if fever or leukocytosis
      - Cover Gram (-) rods and anaerobes, add pseudomonas coverage if previous infx → Zosyn + Flagyl
### Toxic Megacolon
- Extreme dilation and immobility of colon
- True emergency
- **Etiologies:**
  - Newborns: Hirschsprung dz
  - Adults: UC, C. diff, Crohn’s, pseudomembranous colitis, shigella infx, campylobacter infx

### Signs & Symptoms
- Fever, prostration, localized/diffuse/rebound abd tenderness
- Dx: CXR → colonic dilation
- Tx: surgical decompression of colon; may also require colostomy or complete colonic resection
  - Management
    - May reduce complications of diverticulosis with fiber supplements

### Diverticulitis
- Inflammation of the diverticula caused by obstructing
- **Signs & Symptoms**
  - Cases range from mild to severe
  - Acute abdominal pain and fever
  - LLQ tenderness and mass
  - Perforation → generalized peritonitis
  - Constipation or diarrhea, n/v

### Differential:
- Perforated colonic carcinoma, Crohn’s, appendicitis, ischemic colitis, C. diff, ectopic pregnancy, ovarian cyst, ovarian torsion
- Workup
  - CBC may show mild leukocytosis
  - FOBT may be positive
- Management
  - Mild disease → empiric therapy first (Augmentin, or metronidazole + cipro, or Septra, want to cover anaerobes)
  - Clear liquid diet followed by referral for imaging (CT colonography or barium enema, don’t want to do this right away due to risk of causing perforation)
  - Advance diet after 3 days or as tolerated
  - Send patients with increasing pain, fever, or inability to tolerate fluids to ED, might need bowel resection
  - Severe disease → send to ED for abdominal CT

### Signs & Symptoms
- Many cases of colonic diverticula are asymptomatic and discovered incidentally -Chronic constipation, abdominal pain, fluctuating bowel habits
- May have mild LLQ tenderness

### Management
- Reduce complications of diverticulosis with fiber supplements

### Colonic CA
- Incidence:
  - 1st most common GI malignancy
  - 3rd most common cancer in US
  - 2nd leading cause of neoplastic death
  - Highest incidence in North America, Europe, Australia, New Zealand
  - Incidence increasing in US, but increasing worldwide
  - Overall lifetime risk 1/20
  - Avg. age of dx = 73yo
  - Most common in men, more common in AA vs. white
  - Nearly 98% adenocarcinoma
  - Most frequently affects sigmoid colon

### Pre-disposing conditions: age, gender, race, DM, FHx CRC, inherited CRC syndromes, IBD
- IBD (incidence 60% higher)
- Adenocarcinoma 10-20X more common in US
- Crohn’s w/extensive dz (>1/3 colon) 6-8X increased risk
- Screening every 1-2yrs, 8-10yrs after IBD onset

### RF’s: obesity, low physical activity, tobacco, excessive alcohol, high-fat diet, lack of dietary fiber

### Polyposis syndromes
- **FAP: familial adenomatous polyposis**
  - Autosomal dominant (chrn 5)
  - Development of 100-1000’s adenomatous polyps/CRC by age of 40
  - Polyps distrusted fairly evenly throughout colon (slight increase in distal colon)
  - Tx: total proctocolectomy with ileostomy

- **HNPCCC: hereditary non-polypsis colon CA (lynch syndrome)**
  - Autosomal dominant → MLH1, MSH2, MSH6, PMS2
  - Multiple polyps (not as extensive as FAP)
  - Most common hereidatry CRC (% of cases)
  - Higher risk for other malignancies – endometrial, ovarian, gastric, SB, hepatobiliary, ureter, pancreatic
  - Screening - colonoscopy over 2yrs from 21yo, annually after 40yo
    - Women – pelvic exams every 1-3yrs from 1yo, annual pelvic exams, transvaginal US, endometrial bx after 25yo, consider prophylactic TAH and BSO
  - Tx – subtotal colectomy

### Clinical presentation: BI bleeding, change in BM habits, weight loss, intestinal obstruction, abd pain, iron deficiency

### Dx/Screening: SCREENING KEY (early dx = best prognosis)
- Colonoscopy → Screening at 50yo, or 10 years before dx of relative
- Flexible sigmoidoscopy, stool DNA

### Tx:
- Chemoprevention: NSAIDs (ASA), Calcium pills
- Lifestyle modification: less fat, more fruit, more fiber
- Surgery: primary tx modality →
  - Colonic resection w/neg. margins, 12+ LNS for examination/staging
  - Radiation therapy (esp, rectal cancer)
- Chemo/combo therapies

### Prognosis: incurable metastisic dz median survival 6mo – 2yrs
**Acute Pancreatitis**

- **Etiology**
  - Gallstones, chronic ETOH use (2/3 of cases)
  - **ETOH use most common cause in US; gallstones most common cause worldwide**
    - Hypertriglyceridemia, chylomicronemia
    - Hypercalcemia, viruses (mumps, CMV, HSV, HIV, varicella, coxsackie)
    - Trauma: blunt, bleeding, retroperitoneal, procedural (ERCP), malformed ducts, ischemia, vasculitits, SLE

- **Clinical presentation:**
  - Epigastric pain, frequent radiation to back
  - Worse lying supine, with movement
  - N/V, sweating, worse lying supine, with movement
  - Epigastric pain, frequent anorexia, nausea, back pain, diarrhea, steatorrhea

- **Lab findings:**
  - Serum lipase and amylase elevation
    - Elevation does not correlate w/severity
  - Hgb & Hct (with dehydration Hgb may go up)
  - Leukocytosis
  - BUN/Cr elevation (dehydration), LFTs elevated, Alk Phos elevation, triglycerides (over 1000)

- **Imaging:**
  - Confirms dx, determines etiology, defines prognosis
  - **US – best for evaluating biliary tree**
  - **CT w/contrast – evaluate pancreas for necrosis, pseudocyst, stranding, r/o other abd etiologies**
    - Stranding – inflammation around pancreas
    - Pseudocyst – mass in pancreas due to inflammation; if infx needs to be drained; can rupture causing bleeding and ascites; not a true cyst (lacks epithelial layer, tends to grow and cause complications)
  - **MRI - magnetic resonance cholangiopancreatography (MRCP) used to evaluate stone in CBD**

- **Prognosis → Ranson’s criteria**
  - 5 markers evaluated at admission
    - Age >65, WBC > 15,000, glucose >200, LDH >350, AST >250
  - 6 markers evaluated over next 48hrs
    - Hct drop >10%, serum Ca <8%, base deficit >4.0, BUN increase >5, fluid sequestration >6L, arterial PO2 <60
  - Increased mortality w/higher score
    - 5% mortality risk w/<2signs, 40% mortality risk w/5-6 signs

- **Tx:**
  - Remove offending agent and supportive therapy
    - IVF, NPO, NG tube if ileus develops, opioid analgesia, abx if indicated
  - Biliary duct dilation → MRCP, ERCP
  - Surgical tx
    - Remove gallbladder
    - Placement of percutaneous drain if cholangitis (infx biliary tree)
    - Debridement of necrotic tissue
    - Drain pseudocyst

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<th>Neoplasms/pseudocysts</th>
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- Typically develop 4+wks after episode of acute pancreatitis
- Due to necrosis or liquidfication, disruption of pancreatic duct w/leakage
- Can be seen in chronic pancreatitis, pancreatic trauma
- Often asx, abd pain, biliary, obstruction, infx, bleeding
- May resolve spontaneous
- If large/sx → percutaneous/surgical/endoscopic drainage

**Chronic Pancreatitis**

- **Recurrent episodes of pancreatic inflammation most commonly due to ETOH**

- **Pathophysiology:**
  - Change in inflammatory environment → shift to chronic inflammation → fibrosis and calcification
  - Self-perpetuating (anxiety/stress can cause more pain/inflammation leading to more anxiety/stress)

- **Etiologies:**
  - ETOH abuse
  - Hereditary –autosomal dominant, high risk for pancreatic adenocarcinoma
  - Autoimmune, Hypertriglycerides, 20% no clear cause

- **Lab findings: No elevation → mild elevation**

- **Treatment**
  - Remove offending agent (ETOH cessation)
  - Pain control (non-narcotic and narcotic)
  - Pancreatic enzymes: reduces pain by decreases amount of work of pancreases
    - Decreases enzymes secreted through neg. feedback, Supply w/every meal
  - Steroids for autoimmune
    - Needs to be prescribed by GI specialist; can impair glucose control (difficult to prescribe in diabetics)
  - Nerve blocks via EUS to celiac plexus
    - Mixed results, indicated for pts refractory to tx

- **Prognosis: Difficult condition to manage, no cure**

**Adenocarcinoma**

- 4th leading cause of CA death in US
- Most common in males and AA’s >65yo, and in the west
- Poor prognosis – overall 5yr survival rate >5%
- RF’s: hereditary pancreatitis, lynch syndrome, chronic pancreatitis, tobacco, obesity

- **Clinical presentation:** abd pain, jaundice, weight loss, anorexia, nausea, back pain, diarrhea, steatorrhea

- **Dx → biopsy**
  - Tumor markers: CA 19-9 (more accurate for larger tumors)
  - US → better for larger tumors, CT
  - MRI/MRCP – usually better than CT
  - ERCP → bx and stent placement can occur in same procedure

- **Tx → Surgery = curative**
  - Only 15-25% candidates, can’t do it metastatic dz, malignant ascites, invasion into blood vessels
  - May use chemo, radiation, combo therapy for pre-operative shrinkage, post-operatively, unresectable tumors
**Rectum**

**Hemorrhoids**
- Affect 50% population, M>F
- Varices of hemorrhoidal plexus
- RF's: pregnancy, obesity, long-term sitting, genetics, older age

**Internal Hemorrhoids**
- Painless bleeding after defecation
- Visible during anoscopy
- Not palpable or painful on DRE
- Management → 1% hydrocortisone
- Refer to GI for rubber band ligation if prolapsed (bulging out of anus)

**External Hemorrhoids**
- Rarely bleed but are extremely painful, especially if thrombosed (blueish perianal nodule)
- Visible externally on perianal exam
- Management → Sitz bath, 1% hydrocortisone
  - Stool softeners
  - May need to remove thrombosed clot

**Proctitis**
- Inflammation of the lining of the rectum (caused by radiation)
- Proctitis can cause rectal pain and the continuous sensation that you need to have a bowel movement

**Pilonidal cyst**
- Infection of skin & subQ tissue at top of intergluteal fold
- Pilonidal cavity may be asymptomatic
- Can become infected and a sinus tract can develop → an acute subcutaneous abscess develops, spreads along the tract, and may discharge its contents through a pilonidal sinus in the skin cephalad to the natal cleft

**Treatment**
- Abscess → I & D
- Recurrent → excise sinus & tracts
- Cephalalexin, Dicloxacillin, Clinda

**Anal abscess**
- Infection of small anal glands
- Perianal abscess is most common → painful boil-like swelling near the anus
- Surgical I&D = treatment for all types of anal abscesses
- About 50% of abscesses → fistula, can lead to recurrent anal abscesses
  - Surgery is needed to cure almost all anal fistulas
- Etiologies of an abscess
  - Infection of an anal fistula, STI, blocked anal glands
- Risk factors:
  - Colitis, IBD (Crohn’s/UC), DM, diverticulitis, PID
  - Being the receptive partner in anal sex
  - Use of medications such as prednisone

**Fistula**
- Occur due to ulcers, rectal abscesses
- Clinical presentation:
  - Perirectal/perianal: MC, painful swelling at anus, painful defecation → erythema, tenderness
  - Deeper abscesses: buttock/coccyx pain, rectal fullness → fever more likely
- Tx:
  - Surgical drainage, warm-water cleansing, analgesics, stool softeners, high-fiber diet
  - Fistulectomy

**Fissures**
- Posterior most common
- Lateral more pathologic (AIDs, cancer, syphilis, Crohn’s)
- Excruciating pain during defecation, assoc. w/ BRBPR
- Tx: bulking agents, inc. fluids, sitz baths, topical NTG

**Fecal impaction**
- Risk factors → immobility, nervous system dysfunction that damages GI nerves
  - Medications → Anticholinergics, antiarrheals, opiates
- Symptoms
- Common symptoms include:
  - Abdominal cramping and bloating
  - Leakage of liquid or sudden episodes of watery diarrhea in someone who has chronic (long-term) constipation
  - Rectal bleeding, small, semi-formed stools, straining when trying to pass stool
- Other possible symptoms include:
  - Bladder pressure or loss of bladder control, lower back pain
  - Rapid heartbeat or lightheadedness from straining to pass stool
- Diagnosis
  - Hard mass of stool in the rectum
  - Colonoscopy to screen for cancer
- Treatment
  - Removal of the impacted stool
  - Warm mineral oil enema is used to soften and lubricate the stool
  - The mass may have to be broken up by hand. This is called manual removal
  - Suppositories inserted into the rectum may be given between attempts to help clear the stool
  - Surgery is rarely needed to treat a fecal impaction
  - An overly widened colon (megacolon) or complete blockage of the bowel may require emergency removal of the impaction
<table>
<thead>
<tr>
<th>Celiac Disease</th>
<th>Malabsorption syndromes – vitamin/nutritional deficiencies</th>
</tr>
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<tbody>
<tr>
<td>• T-cell mediated gluten intolerance</td>
<td>• Global malabsorption results from diseases associated with either diffuse mucosal involvement or a reduced absorptive surface.</td>
</tr>
<tr>
<td>• Incidence: frequency low (1/3000 persons)</td>
<td>• An example is celiac disease in which diffuse mucosal disease can lead to impaired absorption of almost all nutrients.</td>
</tr>
<tr>
<td>• Clinical presentation: weight loss, diarrhea, excess flatus, abdominal cramps</td>
<td>• Classic manifestations:</td>
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<tr>
<td>o Rare neurologic symptoms: brain fogs, anxiety/depression</td>
<td>o Diarrhea with pale, greasy, voluminous, foul-smelling stools and weight loss despite adequate food intake.</td>
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<tr>
<td>• Diagnosis</td>
<td>o However, the majority of patients have relatively mild gastrointestinal symptoms: anorexia, flatulence, abdominal distension and borborygm</td>
</tr>
<tr>
<td>o Serologic markers: IgA anti-tissue transglutaminase test, IgA antiendomysial ab test</td>
<td>o Clinical manifestations related to a specific micronutrient deficiency can predominate in some patients</td>
</tr>
<tr>
<td>o EGD with biopsy = test of choice</td>
<td>• Example → iron deficiency anemia or osteopenia may be a clue towards celiac disease</td>
</tr>
<tr>
<td>▪ Mucosal intestinal biopsy of distal duodenum or proximal jejunum (will see blunted villi/flat mucosal surface)</td>
<td></td>
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<tr>
<td>• Treatment ➔ Dietary elimination (wheat, barley, rye)</td>
<td></td>
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<tr>
<td>• Complications:</td>
<td></td>
</tr>
<tr>
<td>o Nutrient deficiency (iron, folate, Ca), osteopenia/osteoporosis, increased risk of CA (gut lymphoma, non-Hodgkin’s lymphoma X15%), skin involvement (dermatitis herpetiformis)</td>
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<tr>
<td>• Follow up:</td>
<td></td>
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<tr>
<td>o Serologic testing 6-8mo after starting gluten free diet (Ab’s should be negative after cessation of ingesting gluten)</td>
<td></td>
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<tr>
<td>• Non-celiac gluten sensitivity: no true celiac disease but have sx’s of gluten intolerance; abstain from gluten just as with celiac disease</td>
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<tr>
<th>Thiamine deficiency</th>
<th>B12 deficiency (Cobalamin)</th>
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<td>• Thiamine is involved in many cellular metabolic activities and participates in initiation of nerve impulse propagation.</td>
<td>• B12 needed to convert homocysteine to methionine for DNA synthesis</td>
</tr>
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<td>• Thiamine is found in foods such as yeast, legumes, pork, brown rice, and cereals made from whole grain</td>
<td>• Stores last for yrs, absorption occurs in ileum</td>
</tr>
<tr>
<td>• Thiamine deficiency causes clinical phenotypes of beriberi and Wernicke-Korsakoff syndrome</td>
<td>• Etiologies</td>
</tr>
<tr>
<td>o Beriberi ➔ has two clinical phenotypes, described as &quot;dry&quot; or &quot;wet.&quot; Dry beriberi is the development of a symmetrical peripheral neuropathy characterized by both sensory and motor impairments, mostly of the distal extremities. Wet beriberi includes signs of cardiac involvement with cardiomegaly, cardiomyopathy, heart failure, peripheral edema, and tachycardia, in addition to neuropathy</td>
<td>o Malabsorption ➔ pernicious anemia MC, EtoH, Crohn’s, meds (H2RA, PPls)</td>
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<td>o Wernicke-Korsakoff syndrome ➔ often associated with alcoholism but can also occur in other situations including malabsorption, (poor dietary intake, increased metabolic requirement, and in dialysis patients)</td>
<td>o Decreased intake (found in animal products)</td>
</tr>
<tr>
<td>▪ Untreated, it leads to coma and death</td>
<td>• Sx ➔ Pallor, Glossitis, Stomatitis, GI sx, Psych sx, Hyperhomocysteinemia, Neurologic sx’s (peripheral neuropathy LE, abnormal Babinski)</td>
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<td>▪ Prognosis is improved by immediate administration of parenteral thiamine</td>
<td>• Dx ➔ Peripheral smear: MCV &gt;115, hyper-segmented neutrophils, macroovalocytosis</td>
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<td>▪ Thiamine supplementation, along with other multivitamin supplementation, is recommended for patients at risk for thiamine deficiency</td>
<td>o Increased serum homocysteine &amp; methylmalonic acid (decreased B12 &lt;170 (normal = 240)</td>
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<td>• Pallor, Glossitis, Stomatitis, GI sx, Psych sx, Hyperhomocysteinemia, Neurologic sx’s (peripheral neuropathy LE, abnormal Babinski)</td>
<td>o Pernicious anemia: (+) intrinsic factor ab, parietal cell ab, inc. gastrin levels, (+) Schilling test</td>
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<td>• Tx ➔ B12 replacement (IM), watch for hypokalemia (reticulocytes produced during tx take up large amounts of K)</td>
<td>• Complications ➔ Spinal cord demyelination &amp; degeneration (ataxia, weakness, vibratory, sensory, and perception deficits, dec. DTR)</td>
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<td>• Hemolysis (inc. cell turnover deplete folate stores), Meds (MTX, bactrim)</td>
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<td>• Sx ➔ Pallor, Glossitis, Stomatitis, GI sx, Psych sx, Hyperhomocysteinemia, No neurologic sx’s</td>
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<td>• Dx ➔ Peripheral smear: MCV &gt; 115, hyper-segmented neutrophils, Decreased folate, Normal B12</td>
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<td>• Folate Deficiency</td>
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<td>• Required for DNA synthesis, absorption occurs in jejunum</td>
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<td>• Etiologies ➔ Malabsorption, Pregnancy, Hemolysis (inc. cell turnover deplete folate stores), Meds (MTX, bactrim)</td>
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<td>• Tx ➔ Folic acid 1mg PO qid</td>
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<td>• Complications ➔ Fetal neural tube deficits if during</td>
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