DIGESTIVE SYSTEM

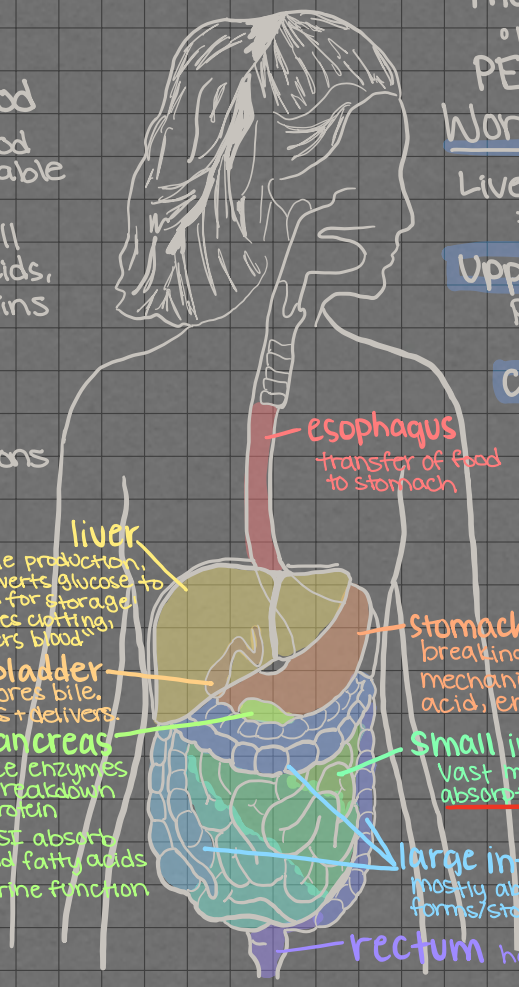
FUNCTION

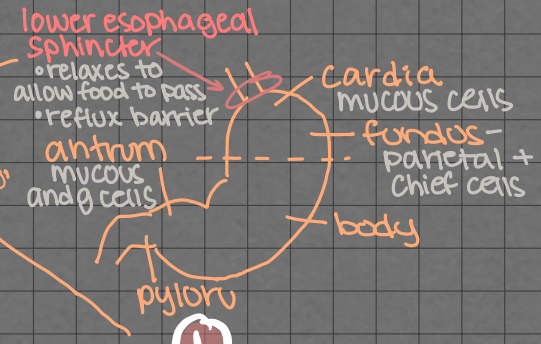
1. Digest/absorb food
   - breaks down food into small absorbable units
   - absorption of all protein, amino acids, fats, carbs, vitamins and minerals
2. Reabsorb fluid

Input → 2L diet, 7L GI secretions

Output → <200 mL stool/day

- GI tract absorbs 98% fluid





CLINICAL

History: OLDCHARTS
- relationship
- stool/urine changes

PE: start w/ least painful area

Workup

General → Stool studies, CBC, CMP

Liver/biliary → CMP/LFTs ± hepatitis panel, GGT, autoimmune

Upper endoscopy: esophagus, stomach, proximal duodenum
- diagnostic or therapeutic

Colonoscopy: entire colon, anus, distal ileum if needed
- diagnostic or therapeutic

Input
2L dietary
7L GI secretions

① enzymes in saliva begin to break down starch → simple sugar

gastric juice secreted by stomach wall. Acid and enzymes kill bacteria and break down proteins

② duodenum
bile and pancreatic enzymes break down fat

③ jejunem
enzymes breakdown carbs, protein, fat

GI tract absorbs 98% of fluid

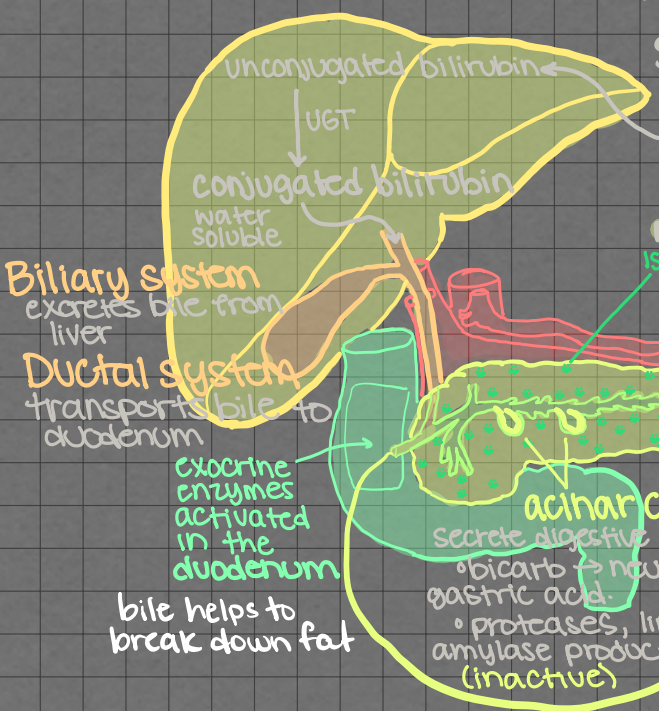
ileum
absorbs nutrients and bile

colon
absorption of water to form feces.

rectum
feces collects before excretion

Output
<200 mL
stool/day

SPLEEN breaks down old RBCs
- biliverdin ← heme
- unconjugated bilirubin



PHASES OF DIGESTION

① **Cephalic**: foods swallowed → gastric activity
- thought of food → brain → vagus nerve → stomach

② **Gastric**: stomach stretches, pH↑ → acid release → food breakdown (when pH<2, H^+ secretion inhibited)

③ **Intestinal**: food enters duodenum → enterogastric reflex ↓stomach stimulation → duodenum can process chyme before Stomach receives more

Esophagitis → Eosinophilic (EOE)

Pathophysiology: inflammation of esophagus
Causes: GERD, radiation, motility disorders, allergies (EoE), infections (CMV, HSV, HIV, candida → immunosuppressed)
Clinical Manifestations: odynophagia (painful swallowing), dysphagia, chest pain, pyrosis, regurgitation ↳ sensation of food being stuck

Mallory-Weiss tear

Pathophysiology: linear tear in the esophageal mucosa @ GEJ
Causes: forceful vomiting or retching
alcohol use
Clinical Manifestations: hematemesis
Diagnostics: endoscopy
Management: self limiting.
Can use PPI once bleeding has resolved
Rare - endoscopic injection of epinephrine or thermal coagulation

Eosinophilic (EOE)

Epidemiology: hx of asthma, eczema, psoriasis
Pathophysiology: eosonic infiltration of the esophagus
Clinical Manifestations: dysphagia to solid foods. GERD sx.
Physical exam: ask about speed of eating, chewing, drink clear fluids, etc
Diagnostics: endoscopy - exudates (white plaques), rings, edema, furrow, strictures (EREFS)
↳ ↓vascularity ↳ ↓lumen diameter
biopsy → >15 eosinophils per hpf
Management: omeprazole x 8 weeks → repeat endoscopy w/ biopsies
- esophageal dilation: serial dilations every 4-8 weeks
- swallowed fluticasone or budesonide
- elimination diets - 2, 4, 6 - dairy, gluten, eggs, nuts, soy, shellfish

Refer to GI

Esophageal strictures

Pathophysiology: Scarring in the esophagus
Causes: radiation, GERD, EoE, malignancy, injestions, meds
Clinical Manifestations: dysphagia
Diagnostics: barium swallow and endoscopy
Management: depends on underlying cause.
Esophageal dilations.

SOLID and LIQUID DYSPHAGIA

ESOPHAGEAL MOTILITY DISORDERS

difficult to diagnose w/ endoscopy

Achalasia

Progressive.
Bland regurg, weight loss

Pathophysiology: lack of esophageal body peristalsis. Non-relaxation of LES
Causes: Chagas disease - kissing bugs seen in S. America, C. america, mexico
Clinical Manifestations: Weight loss, Solid AND liquid dysphasia
- regurgitated contents are foamy/frothy

Diagnostics: barium swallow → bird beak
endoscopy → dilated, fluid filled
confirm w/ eHRM (manometry)
Management: EGD w/ esophageal dilation and botox injection (repeated x 6 months)
Pneumatic balloon dilation - risk of esophageal perf.
POEM: per-oral endoscopic myotomy (↑ risk of reflux)
Heller Myotomy w/ toupet fundoplication

Esophageal Spasm

intermittent chest pain

Clinical Manifestations: chest pain, dysphagia ± GERD
Diagnostics
barium swallow → tertiary contractraction
endoscopy → "corkscrew" esophagus
Management: botox

Barrett's esophagus

Pathophysiology: intestinal metaplasia - change in esophageal epithelium from stratified squamous (esophageal) → simple columnar (intestinal)

Clinical Manifestations: typically asymptomatic

Diagnostics: histopathologic

Management: PPI qd-bid indefinitely to prevent progression to cancer

- Non-dysplastic - surveillance endoscopy every 3 yrs w/ biopsies
- Dysplastic - radiofrequency ablation, cryotherapy, EMR
  - surveillance endoscopy every 6 months after treatment completed then yearly

Esophageal Cancers

Adenocarcinoma - associated w/ Barretts in distal 2/3 of the esophagus

Risk factors: GERD, obesity, tobacco

SCC - proximal esophagus

Risk factors: tobacco, alcohol, caustic injestion, HPV, achalasia

Clinical Manifestations: progressive SOLID food dysphagia, anemia/GI bleeding, weight loss, GERD symptoms

Diagnostics

- barium swallow → obstructive process
- egd w/ biopsies to confirm
- EUS/CT → staging

Management

- endoscopic: mucousal resection, cryotherapy, radiofreqeuncy ablation
- surgical resection: esophagectomy +chemo and/or radiation
- chemo ± radiation can shrink tumor enough to perform endoscopic therapy
- palliative: cryotherapy, stents.

Esophageal varices

Pathophysiology: dilated veins typically in distal third of esophagus

Causes: portal hypertension (high pressure in portal circulation)

- Cirrhosis - alcohol, hepatitis
- Budd-Chiari syndrome - thromboses of the portal vein

Clinical Manifestations: Painless UGIB

Diagnostics: endoscopy

Management: directed at prevention - b-blockers, endoscopic banding

- avoid hepatotoxic agents

hemodynamic support - high volume fluid replacement, vasopressors

GERD

Epidemiology: most common digestive disease in the US

Pathophysiology: stomach contents reflux back into the esophagus leading to sx

Causes: dysfunction of lower esophageal sphincter (LES)
- food - caffeine, alcohol, chocolate, citrus, tomato, vinegar
- lifestyle - weight gain, smoking, pregnancy, eating prior to recumbency

Clinical Manifestations
- esophageal - heartburn, acid regurgitation, chest pain
- extra-esophageal - chronic cough, laryngitis, asthma, sinusitis, aspiration pneumonia, tooth decay

Prominent weight loss, recurrent vomiting, bleeding, dysphagia, jaundice, mass → referral

Diagnostics: PPI trial unless alarm sx → endoscopy: direct visualization of esophagus, stomach, duodenum
- rule out other pathology

24 hr pH impedence - determine if sx associated w/ acid

Management
- Lifestyle: ↓ acidic food, tobacco, alcohol, weight loss, elevate head of bed
- OTC antacids: neutralize stomach acid
  Tums, rolaids, gaviscon, maalax, mylanta
- H2RAs: blocks histamine 2 → first stimuli for acid production. Duration: an hour
  Pepcid, tagamet, zantac. Effective: within 1 hr
- PPIs: blocks proton pump inside paricetal cell to suppress acid. Duration: 24-72 hrs
  Prilosec, prevacid, nexium, zegeride (omeprazole/sodium bicarb) Effective: within 4 days
- Surgery: wrap stomach around esophagus
  Side effects - gas/bloat syndrome, nausea, post-op dysphagia.

Gastritis

inflammation of gastric mucosa → dyspepsia (stomach ache), N/V
Chronic is typically asymptomatic

Causes

Vascular: portal hypertensive gastropathy
Infectious: viral, h. pylori
Neoplastic: Zollinger-Ellison syndrome
Drugs: NSAIDs, EtOH, bisphosphates
Inflammatory: "non-specific" eosinophilic
Congenital
Allergic/autoimmune: atrophic gastritis
Trauma: NG tubes
Endo/metabolic: ménétrier disease

H. pylori

typically contract during childhood → fecal-oral transmission
Sx: burning abdominal pain, ↓ appetite, belching, weight loss, bloating
Complications: gastritis (80-90%) peptic ulcer disease and adenocarcinoma
Diagnosis: HISTOLOGY gold standard
- Rapid urease testing

Active tests: UBT, stool antigen
Treatment: PPI w/ 2 abx (10-14d)
- amox + clarithromycin (resistance)
- metronidazole + tetracycline

Gastrinoma

gastrin secreting tumor that causes hypergastrinemia
- pancreas or duodenum
- refractory PUD
- ± diarrhea

Diagnosis: fasting gastrin >150
- secretin to confirm

Treatment: PPI, surgery

Gastric Adenocarcinoma

Epidemiology: 2nd LC of cancer mortality
Risk factors: men, diet, >40, tobacco, h. pylori
Sx: dyspepsia, weight loss, anemia, GI bleed, dysphagia, postprandial vomiting
Metastatic → left supraclavicular LA (Virchow node) umbilical nodule (sister mary joseph node)
Diagnosis: iron def. anemia, endoscopy + biopsy, CT
Treatment: Surgery (palliative or curative). Chemo or radiation

Peptic Ulcer Disease

an ulcer is a defect in the mucosal surface penetrating through the muscularis mucosa

Gastric ulcer: WORSENS with food **Duodenal ulcer**: IMPROVES with food
- anorexia, weight loss

Causes: H. pylori, NSAIDs, alcohol, smoking

Clinical Manifestations

Abdominal pain - burning, gnawing, often radiates to back
Dyspepsia - bloating, belching, heartburn
Nausea

Diagnostics: endoscopy can localize

Management

Uncomplicated - PPI x 8 weeks, eradicate h.pylori, STOP NSAIDs and smoking

Complicated -

Bleeding → endoscopic therapy
melena, hematemesis, hematochezia

Perforation → surgery
toxic appearance, peritoneal signs

Obstruction → NG suction, dilation, surgery
vomiting, succussion splash

PYLORIC STENOSIS

: pyloric hypertrophy causing gastric outlet obstruction
- males > females

Symptoms: OLIVE MASS (palpable)
- progressive, nonbilious, projectile vomiting in children
- constant hunger
- presents between 4-6 wks of age
- weight loss
- dehydration

Diagnosis: ultrasound, barium swallow → string sign and delayed emptying

Treatment: Surgery

ACUTE

- **acute gastroenteritis**

Cause: bacterial, viral, parasitic
- rotavirus, enteric adeno, noro, S.aureus

Sx: diarrhea and/or vomiting

- **post-operative**: 1/3 of surgical patients after receiving anesthesia
  - female, nonsmoker, past hx, post-op opioids
- **vestibular neuritis**: acute labyrinthine disorder characterized by rapid onset of severe vertigo w/ N/V and gait instability
- **chemotherapy**

anticipatory antiemetic therapy

- **drugs**: abx, antidepressants, aspirin, NSAIDs, opioids, anti-retrovirals, vitamins, minerals

Cannabinoid hyperemesis syndrome

Compulsive bathing

CHRONIC

- **pregnancy** - almost always begins in first 9wks

risk factors: low education/income, AA, female fetus, ↑gravidity, multiple gestation, fetal triploidy, gestational trophoblastic

severe: hyperemesis gravidarum

- **Chronic nausea vomiting syndrome**: dx requires presence of sx for **>3 months**
  - bothersome nausea
  - not self-induced
  - no sign of organic, systemic, metabolic disease
- **Cyclic vomiting syndrome**: idiopathic disorder recurrent, stereotypical bouts of vomiting with intervening periods of normal health

Diagnosis: ROME criteria
1. stereotypical episodes of vomiting
2. 3+ episode in past year, 2 in last 6 months
3. Absence of vomiting between episodes
4. Sx onset 6 months prior to diagnosis

Treatment:

Abortive - 10% IV dextrose, IV ondansetron, sedate

Supportive - IV fluids, antiemetics

Prophylactic - triptans (anti-migraine), others

gastroparesis

"Stomach paralysis" is a syndrome of objectively delayed gastric emptying in the absence of mechanical obstruction/cardinal symptoms

Causes:

idiopathic → most common. Can develop following viral infection

diabetes → T1 > T2. usually develops after DM present >10yrs and end stage organ damage

Post-surgical → fundoplication, bariatric surgery, partial gastrectomy

Symptoms: highly non-specific and not well correlated with gastric emptying

nausea, vomiting, early satiety, postprandial fullness

- rate of emptying depends on: physical nature, particle size, fat/caloric content

↑volume = ↑ rate of liquid emptying

Diagnosis: Scintigraphy - nuclear medicine gastric emptying study
- Wireless motility capsule (UNLESS known stricture)

Treatment:	mild	moderate	severe
Homogenized food	if symptomatic	if symptomatic	routine liquid supplement
Nutritional supplement	rarely need	Caloric liquids PO	may need PEJ tube
Pharmacologic	metoclopramide (10mg) + dimenhydrinate (50mg)	metoclopramide OR domperidone ± erythromycin and dimenhydrinate	± tegaserod ± IV 5-HT-receptor antagonist (zofran)
Non-pharmacologic	none	none	Gastrostomy-tube decomp, parenteral nutrients or gastric electrical stimulation

Diet: 4-6 small meals, low fat, ↓fiber, ↑liquids, soft food

Celiac Disease

Epidemiology: COMMON. Any age.
- Risk factors: 1st degree relative, T1DM, IgA deficiency, Down's synd.

Pathophysiology: abnormal, exaggerated immune response to gluten protein → damage to small intestine → inflammation w/ loss of villi

Clinical Manifestations: diarrhea, weight loss (malabsorption), abdominal discomfort
- Rare - gluten ataxia and dermatitis herpetiformis
- Non-classic - iron deficiency anemia

Diagnostics: test if unexplained GI sx
- First line → serum TTG IgA AND IgA total antibody level
- Confirm dx → endoscopy with duodenal biopsy
  - needed if TTG in positive
  - patient must be on gluten containing diet for 2-4 wks

Management: strict avoidance of gluten
- monitor serum antibodies to monitor response

Gluten Intolerance
gluten sensitivity

Discomfort w/ gluten ingestion due to high osmotic load in the bowel

Symptoms: abdominal bloating, pain, discomfort, diarrhea

Carbohydrate malabsorption

diarrhea, bloating, flatulence

Lactose Intolerance

Epidemiology: lactase deficiency is common in many population.
native american > asian > african > jewish > mexican > caucasian

Pathophysiology: deficiency of lactase - enzyme responsible for lactose breakdown. Lactose enters colon, where it's fermented by colonic bacteria causing symptoms.

Clinical Manifestations: Osmotic diarrhea

Diagnostics
1. Empiric trial of lactose free diet for two weeks
2. ~~Hydrogen breath test~~
   patient ingests 50mg lactose → 90 minutes → ↑breath hydrogen → bacterial fermentation indicating poor lactose absorption in gut

Management
1. reduce ingestion of milk products
2. lactase enzyme supplements
   - lactaid tablets or milk w/ lactaid

Bacterial overgrowth (SIBO)

Risk factors: anything that impedes flow of bowel (slow motility, bowel obstructions, strictures)
Pathophysiology: excess bacteria cause **fermentation** of food products, deconjugation of **bile acids**, and possible damage to small intestine
Clinical Manifestations: **bloating**, **loose stools/diarrhea**
Diagnostics: **breath test** → increased acidity. Not very accurate.
Management: **antibiotics** for ~10 days (empiric)

Mesenteric ischemia

Epidemiology: atherosclerosis, older, arrhythmias, CHF, hypovolemia, malignancy
Pathophysiology: impaired blood flow to the bowel → **ischemia**, pain
- GI tract supplied by **Celiac** trunk, **SMA**, and **IMA**

Causes: anything that causes **poor blood flow** in the mesenteric vessels
- atherosclerosis, **↓CO**, **thrombus**.

Clinical Manifestations
- ACUTE: **arterial clot** leads to acute occlusion of vessel
  - **abrupt** onset of **intense** abdominal pain → can lead to bowel necrosis and perforation
- CHRONIC: usually due to **atherosclerotic plaques** (SMOKING!)
  - **intense, post-prandial** generalized abdominal pain
    30-60 min after eating, "doubled-over", may avoid eating

Diagnostics: **vascular imaging** - CT angiography, US w/ mesenteric dopplers
Management
- Acute: **Surgical emergency**
- Chronic: revascularization
  often percutaneous stenting
  Vascular surgery → challenging, high risk

Intussusception

"telescoping" of the bowel itself
Epidemiology: **CHILDREN** (1-3 years old)
Pathophysiology: 25% lead point. 75% **idiopathic**
Causes: **lead point** - an abnormality of the bowel that gets trapped by bowel peristalsis and pulls bowel segment forward. ex. **meckel's diverticulum**
Clinical Manifestations: **classic triad** (however, seen infrequently)
1. Sudden onset, intense abdominal pain
2. palpable abdominal pain
3. bloody "currant jelly" stools

Diagnostics: **IMAGING** - Ultrasound, X-ray, CT
Management
- Stable: high pressure **enema** to reduce water/air
- Unstable - **urgent surgery**

Inflammatory bowel disease ≠ IBS

Chronic ↑risk of colon cancer

Multifactorial immune-mediated group of disorders characterized by chronic recurring episodes of inflammation in the GI tract (w/ skin, joint, eye manifestations)

Pathogenesis: vulnerable individual → trigger → immune response

Chrohn's Disease

Epidemiology: peak onset teens - 20s + another peak 40 - 60s

Pathophysiology: deep (transmural) inflammation in ANY part of GI tract

Clinical Manifestations: diarrhea, abdominal pain, malnutrition, systemic sx.

Complications: strictures (bowel obstruction), fistulas, abscess (perianal involvement)

Diagnostics: endoscopy → "cobblestoning"

Ulcerative Colitis

Epidemiology: peak onset teens - 20s

Pathophysiology: superficial (mucosal) inflammation in COLON only.
- always includes rectum

Clinical Manifestations: DIARRHEA (±blood), urgency.

Complications: fulminant colitis (toxic megacolon)

Diagnostics: endoscopy – continuous and involves rectum

Skip lesions	diffuse, continuous disease
Variable rectal involvement	rectal involvement always present
not necessarily worse distally	usually worse distally
fissures and fistulas	no fissures
transmural lymphoid aggregates	no transmural lymphoid aggregates
can involve any part of GI tract	only involves colon
Surgery → Palliative	Surgery → curative

Workup: Colonoscopy is hallmark

MANAGEMENT of IBD: aims to induce and maintain remission

Meds:

Mild disease, no abd tenderness, painful mass/obstruction

Aminosalicylates – NO immune suppression. Use to induce and maintain remission in UC.
- Sulfasalazine, Mesalamine
- MOA: topical anti-inflammatory agent in GI tract.
- Toxicities: nausea, HA, pancreatitis, interstitial nephritis (rare)

Thiopurine – better for maintenance
- Azathioprine, 6-Mercaptopurine, Methotrexate
- Toxicities: bone marrow suppression, hepatotoxic, ↑cancer risk, GI upset

Severe disease, extraluminal manifestations, abscess, high fever, diffuse tenderness (rebound)

Biologics: monoclonal antibody (Ab) → target specific immune pathway

TNFα inhibitors – adalimumab, infliximab, certolizumab, golimumab
- Significant immune suppression
- Gold standard since 2000

Interleukin inhibitors – ustekinumab, tofacitinib

Integrin inhibitors – vendolizumab gut targeted → minimal systemic immune suppression but less effective

Steroids: used short-term for flares to temporarily control symptoms
- Prednisone or other corticosteroids. NOT USED LONG TERM
- Side effects: bones (osteoporosis), immune suppression / infection risk, poor wound healing, adrenal suppression, weigh gain

Surgery if medically refractory disease, complications (stricture, obstruction), or cancer/dysplasia
- Proctocolectomy + J-pouch → surgical removal of colon and rectum.

For chrohns, stricture resection, fistula can be challenging (try meds but often surgery). Fistula can lead to abscess → abx and drainage (tubes or surgery)

Microscopic Colitis

distinct from IBD → colon visibly normal

Epidemiology: middle age. STRONG female predominance

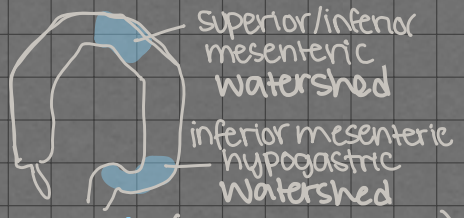
Clinical Manifestations: intense, watery diarrhea. ± urgency

Diagnostics: Colonoscopy → colon visibly normal
- Biopsy need to diagnose → microscopic inflammation
- Lymphocytic colitis → WBCs infiltrate lumen
- Collagenous colitis → thick collagenous band

Management: start ileocolonic release budesonide
- ± imodium adjunct
- start at 9mg daily x 1 month
  - ↳ taper slowly over 1-2 months

Ischemic Colitis

hypoperfusion of colon



Risk factors: older, diabetes, CAD/MI hx, smoking, cocaine

Causes: global hypotension (ex. shock) or atherosclerotic disease

Clinical Manifestations: Crampy LLQ pain (on exam), hematochezia (mucosal sloughing)

Diagnostics: CT abdomen/pelvis → nonspecific colitis/colon edema (segmental)
- Colonoscopy → ischemic changes in a watershed area → Biopsy to confirm

Treatment: Supportive (IV fluids) and bowel rest

Diverticular disease

Diverticulum: outpouching of colonic wall where vessels penetrate into the colon

① Diverticulosis: benign and asymptomatic. 90% will never progress

② DIVERTICULITIS: inflammation/infection of diverticulum pockets

Symptoms: acute abdominal pain. Fever, poor appetite, constipation, leukocytosis
- ↳ left lower quadrant (diverticula typically in sigmoid colon)

25%

75%

Complicated: due to severe inflammation often with a microperforation (± macro)

Uncomplicated: just inflammation/infection

Management: supportive care, clear diet (2-3 days) → advance diet if improving, ± antibiotics

If severe (high fever, difficulty eating, ↑WBC, peritonitis)
- ↳ CT abdomen/pelvis

- no resolution
- resolution → routine colonoscopy
- abscess/complication → Management: hospitalization, IV abx, surgical consult, ± drain
- normal → abx, clear diet ± hospitalization

antibiotics: amox/clav or metro + cipro (7-10 days)

surgery: avoid unless multiple recurrances or complication (fistula, abscess, stricture)

③ Diverticular bleeding: brisk, acute lower GI bleeding

Epidemiology: elderly. Leading cause of lower GI bleeding

Clinical Manifestations: brisk, painless hematochezia. Bright red stools.

Management: Admit to hospital, supportive (IVF), usually self-resolves
- Colonoscopy → try to find and treat bleeding diverticulum

CONSTIPATION

Pathophysiology: impaired defecation

Types:

① Slow transit

- **CIC** chronic idiopathic
  - bothersome
  - no structural or functional cause
- **metabolic**
  - hypothyroid
  - diabetes
  - pregnancy
  - hypercalcemia
- **meds**
  - diuretics, anticholinergics, TCA, opiates
- **neurogenic**
  - stroke/CVA
  - Parkinsons
  - multiple sclerosis
- **OIC** opioid induced
  - same criteria as chronic but opioid use
  - ↳ ± treatment w/ selective agents
    - PAMORAS

② Obstructed defecation

- structural/muscle dysfunction
- **pelvic floor dyssynergia**: uncoordination of pelvic floor muscles. Don't relax normally.
  - pt will push but muscles don't allow passage of stool

Management: done by GI
- specialized physical therapy
- meds → limited effect

Anorectal manometry → diagnosis

- **rectocele/enterocele** → small bowel protrusion
  - anterior rectal protrusion

Sx - frequency, pelvic pressure, sexual dysfunction, splitting
- may require **surgical repair**
  - meds may not help much

③ Hirschsprung's disease rare

congenital neuromuscular disorder
- aganglionic segment of colon fails to relax → functional obstruction
  - megacolon

First Line Therapy:

1. Fiber supplement + PO water
   - psyillum husk, methylcellulose
2. Osmotic laxatives
   - polyethylene glycol, lactulose
3. Stimulant laxatives
   - bisacodyl, senna
4. Secretory agents
   - lubiprostone, linaclotide, plecanitide

Clinical Manifestations: ↓stool frequency, straining, passage of hard stool, incomplete evacuation

Diagnostics: ROME III criteria - must have at least 2 of following

Sx must have started at least 6 months prior to diagnosis.

① fewer than 3 BMs per week
② straining with at least 25% of BMs
③ hard, lumpy stools
④ sense of incomplete evacuation
⑤ sense of anorectal blockage
⑥ manual assistance

Management

Interview + physical exam
↓
metabolic and structural evaluation. Baseline labs.
↓
therapeutic trial fiber
↓ ± laxatives
inadequate response
↓
refer to GI
↓
Anorectal manometry and balloon expulsion test

Complications

Fecal Impaction

hard/constipated stool → impacted in lower colon/rectum
- discomfort, bloating, pain, obstructive sx.
- oral laxatives are often not enough
  - ↳ need rectal therapy and frequent enemas

Overflow diarrhea

impacted stool → watery component flows around
- pt complains of diarrhea
- risk factors: old, meds that slow bowel, inactivity, anything that slows motility

IBS chronic disorder affecting large intestine

Epidemiology: younger, female more likely to be diagnosed

Pathophysiology: Unclear

Clinical Manifestations:

Abdominal pain - crampy, can be severe. Often relieved by defecation.
- may be worsened by stress/anxiety

Diarrhea - preceding crampy pain. No alarm features.

Constipation - hard to pass, lumpy. Precedes crampy pain.

Upper GI symptoms: bloating, ↑gas production, reflux, early satiety, intermittent dyspepsia, nausea, non-cardiac chest pain

Extraintestinal symptoms: impaired sexual function, dysmenorrhea, dyspareunia, increased urinary urgency/frequency, fibromyalgia sx.

IBS-C: constipation
IBS-D: diarrhea
IBS-M: mixed
IBS-U: unsubtyped (insufficient abnormality in stool consistency

ALARM symptoms → pain associated w/ anorexia/weight loss, pain that's progressive, wakes from sleep, large volume diarrhea, nocturnal stools, bloody stools, and greasy stool

Diagnostics: primarily Clinical based on ROME criteria - recurrent abdominal pain, 1 day/week in last 3 months + two:
- related to defecation
- change in stool frequency
- change in form (appearance) of stool
- criteria fulfilled for last 3 mon. Sx for 6.

- Stool tests, celiac panel, CBC, H. pylori
  - ↳ O/P, pathogen panel, FOBT

Management

① Dietary Management

Traditional diet: regular meal pattern, avoid large meals, ↓consumption of fat, insoluble fiber, caffeine, gas-producing food (beans, cabbage, onions)

FODMAP's diet: taking out highly processed foods

GF diet? Food allergy testing?

② Lifestyle Modifications

Physical activity → lowers stress/anxiety. Improves overall health.

③ Medications

IBS-C - fiber (20-35 mg daily). Avoid FODMAPS (↑gas production)
osmotic laxatives (milk of magnesia, miralax, lactulose
IBS-D - imodium, bile acid seaquestrants, anticholinergics (help cramping)
rifampin (non-absorbed antibiotic) ↳ Dicyclomine, hyoscyamine
viberzi (contraindicated if pancreatitis, damaged/absent gallbladder)
tricyclic antidepressants (slows intestinal transit time)
IBS-C - secretory agents (Amitiza ↑fluid secretion, linzess, trulance)

Dyspepsia: pain or discomfort centered in epigastrum ± abdominal fullness, early satiety, bloating, nausea, retching, vomiting

FUNCTIONAL DYSPEPSIA

postprandial fullness, early satiety, Postprandial distress syndrome
epigastric pain/burning, epigastric pain syndrome
AND no evidence of structural disease to explain

Hemorrhoids

Pathophysiology: hemorrhoids are NORMAL → considered pathological when they become enlarged → pain, bleeding
- Causes: increased rectal pressure

Clinical Manifestations: bright red blood on TP while wiping.
- can become thromboses → clot develops in plexus → ACUTE PAIN

Physical exam: external visible on bedside exam
- based on dentate line internal could prolapse or be seen on bedside anoscopy

Diagnostics: Clinical

Management

Recurrent hemorrhoidal Bleeding
1. conservative tx: avoid constipation/straining. use of fiber, laxatives, softeners
2. Hemorrhoid banding
3. Endoscopic coagulation
4. Surgical hemorrhoidectomy

Thrombosed (acutely painful) hemorrhoids: Sitz baths, anti-inflammatories, treat constipation
- If pain persists → consider Surgical hemorrhoidectomy

Anal fissure

tear in the anoderm below the dentate line.

Pathophysiology: trauma → stretch/tear anal mucosa → repeat injury + healing → deeper fissure → causes spasm in anal sphincter
- this can eventually lead to chronic anal fissure

Causes
- Primary: passage of hard stool, prolonged diarrhea, vaginal delivary, anal sex
- Secondary: IBD/crohn's, malignancy, infection (ex HIV, syphilis), previous anal procedure

Clinical Manifestations: typically VERY PAINFUL. Acute, sharp pain in anal region
- Physical exam: careful external exam. DRE may not be tolerated.

Diagnostics: history - painful defecation, sharp anal pain

Management: initially, sitz baths, treat constipation, topical analgesic
- Meds: topical nifedipine or nitroglycerine (relax anal sphincter)
- Refractory → botox injection to relieve spasm
  sphincterotomy surgery also considered

Abscess

infected pocket of pus in perineal region.
- intense, constant anal pain, difficulty sitting → urgent drainage

Fistula

an epithelialized tract from anus or rectum to perianal skin
- can form after abscess → Surgery

Causes: CROHNS (treat w/ meds)

Anal Cancer

usually squamous cell. Distinct from rectal cancer.

Risk factors: HPV, female, genital warts, smoking, anal intercourse, HIV

Etiology: HPV infection → premalignant lesions (AIN)

Treatment: topical (trichloroacetic acid).
Immune modulation (intra-anal imiquimod)
infrared coagulation, anoscopy-directed lesion ablation (electrocautery)

DIARRHEA

>200-300 gm stool/day

ACUTE
<4 weeks
usually self-limiting
typically 2-3 week duration

NON-INFLAMMATORY
non-invasive
Watery, involves small bowel, typically due to enterotoxin

VIRAL GASTROENTERITIS
Rotovirus: 2 day incubation
Sx: low grade fever, emesis, diarrhea x4-8d
Outbreaks → children, elderly
Tx: rehydration Prevent: vaccine

Norovirus: 12-48 hr incubation
Adolescents/adults → college campuses, cruise ships
SX: nausea, emesis, cramps, diarrhea, low grade fever, malaise, HA, myalgia
- lasts 2-3d → resolves

Highly contagious

BACTERIAL
S. Aureus — eggs, mayo, dairy
Bacillus cereus — fried rice!
heat stable enterotoxin - fast 6 hrs
SX: N/V ± diarrhea
TX: supportive care

E. coli: enterotoxin (not as stable)
Longer incubation → 1-3 days
Traveler's diarrhea - contaminated food/drinking water
SX: watery diarrhea, crampy pain
TX: supportive ± abx (quinolones)

Vibrio cholerae: gram- rod
inhibits sodium/chloride transporter
Dx: rapid PCR on stool sample or culture
SX: watery diarrhea, electrolyte abn.
TX: supportive ± abx
└ fluid/electrolyte repletion

INFLAMMATORY
Invasive
pts are ILL
└ fever
mucus, blood, pus
Often involves colon ± SB

BACTERIAL
CAMPYLOBACTER JEJUNI: most common
bacterial enteritis
CAUSES: undercooked poultry, cattle, milk, water, pets
SX: fever + periumbilical pain
TX: supportive, abx if severe (doxy, quinolones)

SHIGELLA: fecal-organ transmission
SX: lower abd pain, bloody/mucoid stool
RISKS: seizures, ↑↑↑ WBC, HUS
TX: supportive, abx if severe (quinolones, azithro, ceph)

E. COLI: enterohemorrhagic → verotoxin → GI bleed
CAUSES: undercooked beef, unpasturized milk, contaminated water, day care centers
SX: watery diarrhea → bloody. Abd pain, vomiting but usually no/mild fever. HUS risk
TX: supportive

YERSINIA ENTEROCOLITICA: less common
CAUSES: undercooked pork, milk, water, tofu
SX: fever, abd pain, mesenteric lymphadenopathy
- can cause subacute/chronic infection

TX: supportive, quinolones/bactrim

SALMONELLA

	Typhoid (uncommon in US)	Non-typhoid (common in US)
transmission	fecal-oral	foodborne, contact w/ reptiles
clinical	HA, fever, abd pain, fatigue, malaise, ± bradycardia, hepatosplenomegaly, rose spots on skin	N/V, fever, cramping
PE		
Tx	abx - quinolones	supportive

CHRONIC
due to chronic condition
typically requires more workup and management

WATERY
OSMOTIC: large amount of poorly absorbed, osmotically active solute is in GI lumen → pulls water into lumen
- Carbohydrate malabsorption
- Mg, PO4 ingestion
- osmotic laxative abuse

SECRETORY: something is forcing fluid into lumen
- drugs (interfere w/ transporters)
- bacteria
- stimulant laxative abuse
- vasculitis
- cancer
- neuroendocrine tumors → release peptide signal causing secretion

Stool osmotic gap = 290 - [2x(stool Na + stool K)]
↓ 24 hr collect
gap < 50 | 50-100 inconclusive | gap > 100
↓ tx based on etiology | ↓ eliminate offending agent

FATTY
malabsorptive
greasy, oily, malodorous, float
- Chronic pancreatitis
- Celiac disease

Stool studies → fecal fat
blood work → fat soluble vitamins
likely refer to GI

MIXED
multiple or hard to distinguish
- Irritable bowel syndrome
- hyperthyroidism
- diabetic autonomic neurop.
- post vagotomy diarrhea

clinical
refer if dx unclear or severe sx

INFLAMMATORY
mucoid (pus) or bloody, systemic sx
- IBD, ischemic and radiation colitis
- malignancy
- some infections

refer to GI
Stool studies → fecal leuks to rule out infection
± colonoscopy → IBD?

C. DIFF
spore forming, gram+, produces toxin. RF → older, PPI use, hospital/healthcare contact

Pathophysiology: disruption of normal flora → overpopulation of pathogenic C. diff.
Clinical Manifestations: severe diarrhea, can be bloody and lead to dehydration, toxic megacolon, occ death
Diagnostics: endoscopy → pseudomembranes. Workup → stool test PCR (or toxin in stool)
Management: oral vanc 125mg PO QID. second line → fidoxamicin ($). severe → fecal transplant

Upper GI bleeding

associated with hematemesis, coffee ground emesis, melena, and hematochezia

esophageal varicoses: large, dilated veins
- Prone to brisk bleeding.
- Often life threatening
- *Usually patients w/ cirrhosis

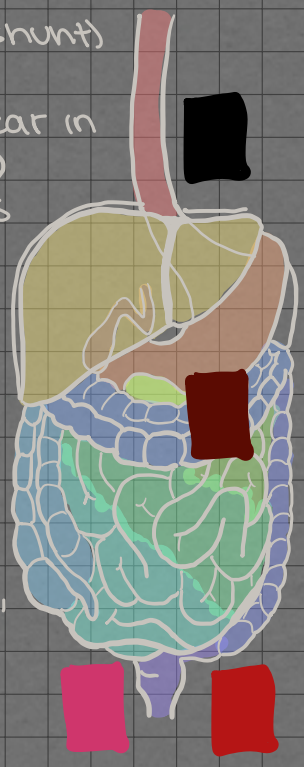
resuscitation → IV Octreotide drip → endoscopy to stop bleeding
→ rescue therapy
- blakemore tube
- TIPS (transhepatic portal shunt)

Mallory-Weiss tear: linear tear in the esophageal mucosa (GEJ)
- forceful vomiting, hematemesis
- Alcohol use!

bleeding peptic ulcer ulcer erodes through mucosa exposing an arterial vessel
- can bleed briskly and be life threatening
- PPI can rapidly heal ulcer and stop bleeding
- endoscopic - clips, cautery, argon coagulation, epinephrine injection

Other:
- erosive esophagitis/gastritis
  - ↳ acid, H.pylori, EtOH, NSAIDs
  - ↳ acid reflux → acid-induced mucosal injury

Treat w/ PPI x 6-8 wks



Others
- Arteriovenous malformations
  aberrant blood vessels prone to oozing/bleeding
  ↳ endoscopic treatment
- Upper GI malignancies
  microcytic anemia, dysphagia, weight loss. EtOH/smoking hx.
  ↳ endoscopy + biopsy for dx

Mid bowel bleeding

Small bowel bleeding
difficult to diagnose and manage
- AVMs, small bowel diverticula, small bowel Crohn's, tumors, vasculitis, atypical infections

Endoscopic: capsule, "push", single or double balloon enteroscopy

Imaging: CT-angio, tagged red cell scan, angiography (diagnose AND embolize)

Lower GI bleeding

associated with hematochezia and bright red blood per rectum (BRBPR)

Diverticular bleeding: acute, painless lower GI bleeding. Bright red stools.
Admit to hospital, supportive (IVF), usually self-resolves
Colonoscopy → try to find and treat bleeding diverticulum (clip)

Hemorrhoids: bright red blood on TP is classic presentation.
- typically benign. Not a large volume of blood.

Ischemic colitis: hematochezia, cramping. elderly. Usually self limiting
- relatively acute

MANAGEMENT - stabilization and resuscitation

1. Assess patient for instability
2. Get IV access
3. Resuscitation → IV fluids!
4. Labs → CBC, INR, type and screen
5. Meds → IV PPI (treat ulcers quickly). IV Octreotide (if suspicion for cirrhosis)
6. Order blood if hgb < 7 or symptomatic anemia
7. GI consult → endoscopic evaluation

PHENYLKETONURIA

Epidemiology: autosomal recessive. Presents early.

Pathophysiology: defect in hepatic enzyme phenylalanine hydroxylase leads to accumulation of phenylketones in urine and blood.

Clinical Manifestations: cognitive delay, intellectual disability, vomiting, irritability, seizures, light color skin, musty urine

Diagnostics: measure serum phenylalanine, molecular studies
In US → newborn screening is standard

Management: lifelong dietary restriction of phenylalanine foods → milk, cheese, nuts, eggs, fish, chicken
- protein supplements
- tyrosine supplementation

Short bowel Syndrome

patient does not have enough bowel to maintain adequate nutrition

Causes: anything that causes large portions of bowel to be removed
- chrohns w/ multiple resections
- acute mesenteric ischemia w/ large section of bowel necrotic/resected
- trauma
  - less than 100-200 cm → high risk

Clinical Manifestations: difficulty maintaining weight and hydration.
profound/persistant electrolyte or nutrient deficiencies

Diagnostics: CLINICAL

Management

Supportive - anti-diarrheals (slow motility)
- loperamide (Imodium) and diphenoxylate-atropine (Lomotil)

supplement vitamins/electrolytes
rehydration solution, high caloric density food

Parenteral nutrition — IV fluids
IV potassium, IV magnesium
TPN (total parenteral nutrition) → give complete caloric needs through central line. HIGH RISK

Medication - tedeglutide → stimulates small bowel hypertrophy to increase small bowel surface area
GLP2 analog

Intestinal Transplantation - high risk. Survival is often only a few years

VITAMIN DEFICIENCIES/TOXICITIES

Common vitamins

Fat soluble		If deficient →	Water soluble	
Stored in fat → ↑risk of toxicity	Vitamin A	LOSS OF VISION	SCURVEY	Vitamin C
	Vitamin D	RICKETS, OSTEOMALACIA	BERIBERI	Vitamin B1 - thiamin
	Vitamin E		PELLAGRA	Vitamin B3 - niacin
	Vitamin K			Vitamin B6 - pyridoxine
				Vitamin B12 - cobalamin

Use same transporters as fatty acids → fat malabsorption → vitamin deficiency

VITAMIN	SOURCE	DEFICIENCY	TOXICITY
FAT SOLUBLE			
A vision, growth, immune function, spermatogenesis/ embryonal development, epithelial differentiation	**Retinoids** egg yolk, dairy **Carotenoids** yellow/red fruits/ veggies, dark leafy greens	• GI/fat malabsorption disorders • chronic nephritis • intestinal parasite • alcoholism ← bitot's spots Signs: ↑keratinization → ocular - ↓nyctalopia, xerophthalmia Derm - rough, dry scaly skin follicular hyperkeratosis (bumps)	Bone/muscle pain, cirrhosis, dry itchy skin, conjunctivitis ↑beta carotene → orange skin (not toxic)
D calcium transport, bone health, immune, CNS,	**UV light** - photolytic action produces VD from sterols in body **Food** - fish, egg yolk, fortified foods	**Bone health** - weakening Adults: osteomalacia/↑fractures due to bone weakening Children: rickets Risks: breastfed, raised in poverty, >50 yo, limited sun exposure, alcoholics, fat malabsorption	Hypercalcemia and calcification of soft tissue from supplementation NOT sun exposure
E antioxidant, DNA repair, immune function	vegetable oils, nuts, leafy greens, cereal	Rare. Neurologic problems - nerve degeneration in hands/feet. ↑RISK: fat malabsorption disorders alcoholics, preemies, genetic defect	↑bleeding and impaired blood coagulation
K blood coagulation	dark leafy greens, broccoli, cabbage • small amount made in GI tract tract via bacterial synthesis	Problems w/ blood coagulation ex. bruising, nose/GI bleeds life threatening bleeds in newborns ↳ in/around brain ↓bone density → ↑risk of fractures	May reduce effectiveness of anticoagulants → ↑clot risk (Warfarin)
WATER SOLUBLE			Readily excreted less toxicities
C potent antioxidant co-factor for enzyme involved with synthesis of norepi, collagen, carnitine	Citrus fruits, Papaya, cantalope' broccoli, strawberry ↓intake → Smoking ↑need → to repair damage	Hyperkeratosis corkscrew hairs Hemorrhage bleeding gums, poor wound healing Hematologic anemia, ↑bleed time **Severe → scurvey** 3 H's	
B **B1 Thiamin** Coenzyme in Carb metabolism	enriched, fortified, Whole-grain products	**Alcoholics** - Wernicke-Korsakoff affects nervous system • Wernickes encephalopathy • Korsakoff syndrome → irreversible Beriberi: wet affects CVS → HF, SOB, ↑HR, edema dry affects NS → confusion, tingling TX: IV thiamine → PO thiamine	
B3 Niacin required for energy metabolism	meat, fish, poultry grains - enriched, and whole-grains	Pellagra: 4 D's seen - diarrhea, dermatitis, dementia, death Signs/sx - scaly skin sores, inflamed mucus membranes	Supplements → flushing, GI distress
B6 Pyridoxine >enzymes, glucoreogen, RBC metabolism	Steak, bananas, navy bean	Causes: alcoholism, meds (isoniazid) Sx: peripheral neuropathy, anemia, glossitis, dermatitis	Neuropathy
B12 Cobalamin lipid metabolism, dna synthesis, nerve cells, RBCs	meat, fish, poultry, milk	Risk factors: Vegan, alcoholism, Crohn's, pernicious anemia Sx: megaloblastic anemia, peripheral neuropathy (can be permanent) glossitis, diarrhea, fatigue	

ACUTE PANCREATITIS

Pathophysiology: acute inflammation resulting from premature activation of digestive enzymes → enzymes digest pancreas

Causes: alcohol (direct toxin) and gallstones (backup of fluid)
- hypertriglyceridemia
- meds (thiazide diuretics)

↳ "band like"

Clinical Manifestations: intense, acute deep abdominal pain. N/V, fever, chills
- multiorgan involvement

Physical exam: Cullen sign (bruising around umbilicus) and grey-turner sign (bruising around flanks)
- SIRS criteria, tachy, hypotensive, ± febrile

Acute → Severe Acute → Necrotizing

Classic SX, often self-limiting → progression to extensive tissue destruction and organ failure

Diagnostics: need 2 or 3 "band-like"
1. acute onset severe epigastric pain
2. elevated amylase or lipase >3x ULN
3. CT imaging (or MRI) consistent

Other features: hypocalcemia, high BUN/creatinine, ↑WBC, hypoxia, ↑LFTs, ↑glucose

Scoring: BISAP score → BUN, mental status, SIRS, age >60, pleural effusion (2+ = ↑mortality)

Management: **IV fluid resuscitation**
- Liter boluses (2 in first hour) and monitor ins/outs and symptoms
- Symptomatic → pain control, anti-emetic
- EARLY enteral nutrition as soon as pt can tolerate → low fat diet

search for underlying cause:
- Alcohol → EtOH cessation
- Gallstone → early cholecystectomy
- Gallstone + Cholangitis → ERCP
- meds → stop meds

Complications

1. **PANCREATIC NECROSIS** - often develop multi organ failure
   - VERY serious - may need endoscopic, radiologic, or surgical debridement
2. **PANCREATIC PSEUDOCYSTS** - walled off sterile fluid collection that develops weeks after acute pancreatitis.
   - No tx if asymptomatic ± drainage

CHRONIC PANCREATITIS

Epidemiology: Smoking and alcohol have synergistic effect

Pathophysiology: progressive fibroinflammatory disorder associated with a loss of pancreatic parenchyma and function

Causes: alcohol, idiopathic, genetic - cystic fibrosis, autoimmune, tumor, recurrent severe acute

Clinical Manifestations: intense epigastric pain, fatty diarrhea, weight loss, malabsorption

Complications:

exocrine insufficiency - malnutrition, steatorrhea (fat malabsorption)
- loss of fat soluble vitamins
- need to lose 90% of pancreatic function

Treat w/ Pancrealipase

endocrine insufficiency - diabetes, ↑risk of pancreatic cancer

Diagnostics: no single gold standard amylase/lipase are NOT helpful

Relies on clinical impression:
- low weight, malnutrition

Labs: low fecal elastase, low albumin

Imaging: endoscopic ultrasound

CT/MRI - atrophy, dilated duct, calcifications

Management: Underlying cause

exocrine insufficiency → pancreatic enzyme replacement, low fat diet

pain → neuromodulatory meds (gabapentin, amitryptalline) or opiates
- occasionally, celiac plexus block

endoscopic

If there's a structural issue, consider ERCP to break up stone/open duct

surgical

resections to relieve symptoms typically if dilated or obstructed.
- total pancreatectomy + islet cell AUTO-transplant is an emerging therapy

PANCREATIC CANCER

Epidemiology: age > 55. Males > females. Presents late → high mortality

Risk factors: Chronic pancreatitis, hereditary pancreatitis, african american, tobacco, diet (fat, meat)
- fruits and vegetables are protective

Pathophysiology: typically adenocarcinoma

Clinical Manifestations: Painless jaundice, pruritis, weight loss, malaise, early satiety, palpable gallbladder (Courvoisier's sign), depression, diabetes

Diagnostics: difficult to pick up clinically

Imaging often suggestive → CT/MRI

Ultrasound → can see pancreas, lymph nodes and do biopsy if needed
- ERSP is less helpful

Blood test → Ca19-9 tumor marker, but not very accurate

Management: Cure → surgery

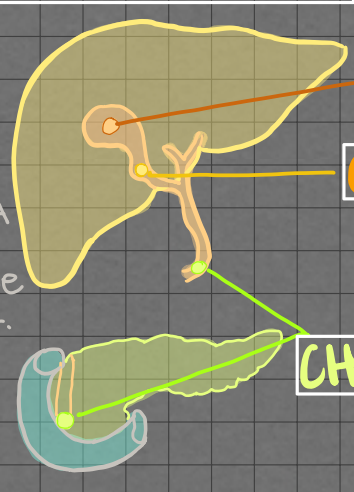
Curative: Whipple surgery usually combined with chemo.
- major surgery, quite morbid
- Sometimes not feasible due to anatomy (tumor wrapped around vessels)

Palliative: biliary stent to relieve bile duct obstruction (Sx relief)
± palliative chemo (5FU, gemcitabine)

POOR PROGNOSIS
- 5 yr survival
- local recurrance common
- mets: liver, lungs, occassionally bone
- without treatment → 6-9 month survival

BILE DUCT OBSTRUCTION

Clinical presentation - biliary colic, jaundice, clay-colored stool, dark urine



Cholesterol stone
Most common
Risk factors: older, female, obesity, multiparity, race Indian > caucasian > AA

Pigmented stone
seen on X-ray due to calcium content.
Black → calcium bilirubinate
- Chronic hemolysis

Brown: recurrent

CHOLELITHIALIS - typically asymptomatic
Biliary colic: RUQ pain w/ gallbladder contraction

CHOLECYSTITIS: RUQ pain, fever, Murpheys sign. NORMAL LFTs but ↑WBC.
- no biliary dilation. Normal bilirubin level.

Treatment: abx and cholecystectomy (indicated for biliary colic)

CHOLEDOCHOLITHIASIS - stones in bile duct
Cholestatic hepatitis
Imaging: biliary dilation
↑LFTs - alkaline phosphatase + Tbili

CHOLANGITIS - bile stasis from obstruction can lead to infection

Charcots triad: jaundice, RUQ pain, fever

Medical emergency → abx and decompression w/ ERCP

Acute suppurative: presence of PUS in the biliary ducts that may result in → Reynold's phenomenon: triad plus hypotension and confusion.

CHRONIC LIVER DISEASE

FIBROSIS ⟶ CIRROSIS
F0/F1 ⟶ F2 → F3 → F4 irreversible

VIRAL

HEPATOTROPHIC VIRUSES

A
Epidemiology: mostly under-developed countries
Transmission: fecal-oral from contaminated food
- shellfish common. Water near infected farms
- 4-6 wk intubation period

Symptoms: Jaundice, dark urine
- flu-like sx. Abdominal pain w/ N/V/D

Diagnostics: Hep A IgM positive
- IgG indicitive of prior exposure/vaccine

Management: supportive care w/ fluids, anti-emetics, anti-diarrheals
DOES NOT CAUSE CHRONIC HEPATITIS
Prevention: Vaccine

B DNA Virus
Epidemiology: highest rates in Africa and SE Asia
Transmission: Vertical → SE Asia and Africa
Sexual or IVDU → western world
- major source of cirrhosis

Clinical Manifestations: if symptomatic → flu-like illness, jaundice, N/V ± abdominal pain

Diagnostics
+HBsAg → acute/chronic infection
+HBsAb → infection cleared or vaccine
+HBcAb → IgM → acute infection
IgG → chronic/resolved
+HBeAg → active, rapid viral replication
+HBeAb → active, slow viral replication
Hepatitis B viral load

Management: Antivirals with high barriers of resistance
tenofovir or entecavir
- initiated on anyone that has cirrhosis
- typically on antivirals for LIFE

HBV DNA still remains in liver cells after clearance

C causes chronic hepatitis in 85% of patients. SSRNA virus.
Transmission: blood (parenteral)
Clinical Manifestations: usually asymptomatic
fatigue and ↓QOL
- those w/ cirrhosis have higher risk of developing hepatocellular carcinoma

Diagnostics
① HCV Ab → exposed
② HCV RNA viral load → ⊕ infection
⊖ cleared

Treatment: harvoni, epclusa, mavyret

D only seen in Chronic HBV infection
Defective RNA virus that requires HBsAg to replicate

E acute, icteric, self-limited hepatitis similar to hepatitis A
HIGH MORTALITY IN PREGNANCY

TOXINS

ALCOHOL → AST/ALT >1
Chronic use → steatosis → fibrosis → cirrhosis

alcohol associated hepatitis
severe inflammation secondary to binge drinking (daily use >40g f, 60g m >6mon)
SX: jaundice, tender hepatomegaly
- portal HTN, ascites, fever

DX: bili >3, ↑INR, leukocytosis → steroids

MEDs: methotrexate, tylenol, statins
most commonly - ABX, NSAIDs

NON-ALCOHOLIC FATTY LIVER DISEASE

Hepatic steatosis WITHOUT hepatocellular injury
↓
NASH non-alcoholic steatohepatitis
>5% hepatic steatosis WITH hepatocellular injury
↳ fibrosis → cirrhosis → death/transplant

Diagnosis: liver biopsy demonstrating steatosis ± inflammation and fibrosis PLUS ⊖ serologic workup
- ALT > AST

Treatment: weight loss → >10% (improves fibrosis)
- do NOT stop statins

CIRRHOSIS

process by which normal architecture of the liver is replaced by regenerative hepatic nodules separated by bands of fibrosis.

END-STAGE LIVER DISEASE - takes years to develop
10 year mortality of 35-70%.

Etiologies:
- **viral** - hepatitis B and C
- **toxic** - alcohol, chronic drugs
- **metabolic** - NAFLD
- **biliary** - PSC, PBC
- **genetic/hereditary** - hemochromatosis, Wilson's, A1antitrypsan deficiency
- **others** - autoimmune, Congestive hepatopathy, cystic fibrosis, sarcoidosis

Pathophysiology → Manifestations
- unable to process bile → jaundice, scleral icterus, itching
- cant produce clotting factor → ↑risk of bleeding, ↑INR
- low protein production → ascites, edema
- reduced capacity to metabolize drugs → drug toxicity
- less gluconeogenesis → hypoglycemia
- portal hypertension → ascites, varices

Physical exam: ascites, scleral icterus, caput medusa, spider telangiectasia, edema, palmer erythema, asterixis. Gynecomastia, dark urine, splenomegaly

Cirrhosis

- → **hepatic encephalopathy**
- → **HCC**
- → jaundice
- → coagulopathy
  - → portal venous thrombosus
  - → GI bleeding
  - → Thrombocytopenia
- → edema
- → **portal HTN**
  - → Hypersplenism
  - → **varices**
  - → **Ascites**
  - → Hepatic hydrothorax
  - → **HRS**
  - → **SBP**

hepatic encephalopathy
disturbance in CNS function due to hepatic insufficiency
- Ammonia is neurotoxin.

Covert: alterations in psychomotor speed → ↓awareness, attention
Overt: lethargy, disoriented (time), asterixis → confused, somnolence, still responsive → coma
Management: support
lactulose and rifaximin

Portal HTN
- ↑portal venous inflow due to mesenteric arteriolar dilation
- ↑resistance through cirrhotic hepatic sinusoids

Forms pressure gradiant btwn systemic and portal circulations
HVPG > 10 mmHg

varices - portal HTN causes dilation of intra-abdominal veins
↳ **hemorrhage** is most devastating complication
- mortality ~50% w/out tx

Treatment: Acute bleed → banding, "Blakemore" tube, TIPs (Shunt), injection sclerotherapy
Prevention: b-blockers ↓portHTN
Pt w/ cirrhosis + PLT < 150 → Screening EGD

Ascites due to portal HTN gradient
Presentation: abdominal distension
- can lead to abdominal pain and SOB

Diagnosis: paracentesis
can help find etiology
serum ascites-albumin gradient
1.1 > SAAG ≥ 1.1 cirrhosis
Management: low sodium diet (<2g/d), diuretics
furosemide + spironolactone

SBP spontaneous bacterial peritonitis
peritoneal fluid w/ >250 PMNs
Presentation: ↓liver function, renal failure, GI bleed, or encephalopathy
Diagnosis: paracentesis rule out in each pt w/ ascites
Treatment: 3rd gen cephalosporin or quinolone + albumin
secondary prophylaxis → CIPRO 500 mg daily

HRS hepatorenal Syndrome
development of AKI in a patient w/ cirrhosis/fulminant hepatic failure
Presentation: low urine Na, oliguria, hypotension
Treatment: liver transplant.
Midodrine and octreotide.
Norepi infusion. vasopressin analogs

ESOPHAGEAL CANCER

1% of all cancers.

Epidemiology: Adeno or squamous. Men > women. Age > 55 yo

Symptoms: dysphagia, weight loss, upper GIB, anemia

Causes: **Barrett's esophagus**, tobacco, alcohol, obesity, GERD, achalasia
- metaplastic change in the distal esophagus
  - often develops as a consequence of chronic GERD (10-15%)
- does NOT cause sx. Predisposes to esophageal carcinoma

Pathophysiology: rate of progression is slow
GERD → barrett's → low grade dysplasia → high grade dysplasia → cancer
└ most will not progress

Screening: patients w/ chronic (>5 yrs) or frequent (≥ weekly) GERD sx AND 2+ risk factors → age >50, caucasian, smoking (current or past), fam hx of barrett's or esophageal cancer, central obesity.
- females not typically screened due to lower risk

Management: ALL patients w/ barrett's should be on once daily PPI therapy.
- No dysplasia → surveillance w/ EGD every 3-5 yrs
- Low grade dysplagia → endoscopic ablation (alternative is close surveillance)
- High grade dysplagia → endoscopic ablation

LIVER MASSES

BENIGN

1. **Simple hepatic cysts** thin walled cysts filled w/ fluid density that is consistant throughout
   - asymptomatic → occassionally grow large enough to rupture or compress organs → surgery
   - Warning signs: multiple internal septations, calcifications, refer to surgery/GI
   - Polycystic liver disease: genetic disorder - defect in ADPK1 gene
     - massive hepatomegaly → portal HTN → liver transplant (usually kidney too)
2. **Hepatic hemangioma** vascular malformations or hamartomas. 30-50yo most common
   - Clinical: rarely hemorrhage or grow → resection, embolization
   - Diagnosis: multiphase MRI → progressive filling
3. **Focal nodular hematoma** hyperplastic/regenerative response to hypo/hyperperfusion by anomalous arteries in center of nodules. 20-50yo female.
   - Clinical: solitary, usually <5cm, asymp. no malignant risk
   - Diagnosis: central stellate scar
4. **Hepatic Adenoma** large plates of atypical hepatocytes that don't function. 20-44 yo
   - Risk factors: OCP use, obesity, pregnancy
   - Clinical: solitary, right lobe. Potential for rupture if >5cm. Can undergo malignant transformation
   - Management: refer to GI. symptomatic or male → resect (↑ risk of malignancy)
     asymptomatic → stop OCP, MRI Q6 mon w/ alpha-feto protein
     - resect if >5cm, exophytic protrusion, or desire pregnancy

MALIGNANT

1. **Metastatic Tumors** most common mets
2. **Hepatocellular carcinoma** most common primary malignancy of liver
   - Etiology: associated with chronic liver disease → HBV, HCV, cirrhosis most risk
   - Diagnosis: ↑alpha-fetoprotein. MRI → arterial enhancement w/ "washout", pseudocapsule
   - Management: hepatology, surgery, interventional radiology, oncology
     - screen ALL cirrhosis pts w/ RUQ US x6m

COLORECTAL CANCER

Epidemiology: 4% lifetime risk. 3rd most common cancer

Risk factors
- non-modifiable → age, AA race, fam hx, IBD, hereditary CRC syndromes
- modifiable → obesity, diabetes, smoking, alcohol, red meat, low fiber, sedentary

Pathophysiology: normal mucosa → mutations → early adenoma (small polyp) → carcinoma sequence

normal (0 years) → early adenoma (3 years) → immediate (7y) → late (8y) → carcinoma (10 years)

Other causes: hereditary colorectal cancer syndromes

Lynch syndrome (HNPCC): autosomal dominant germline mutation in a mismatch repair (MSH2, MLH1, MSH6, PMS2, EPCAM)
- Lifetime risk of CRC → 70-80%. average age: 44

3-2-1 rule: 3+ family members w/ a Lynch associated cancer
Amsterdam criteria: 2+ generations
1 dx before age 50

Management: early and frequent cancer screenings. Colonoscopy starting @ 20-25 then every 1-2 years

FAP syndrome: autosomal dominant germline mutation in APC gene
Patients get dozens if not hundreds of polyps throughout colon
- Lifetime risk of CRC ~100%.

Management: begin screening at age 12 until Colectomy in late teens/20s

Clinical Manifestations: large bowel obstruction, iron deficiency anemia
- change in stool, lower GI bleeding, fatigue, weight loss

Physical exam: usually found incidentally

Diagnostics: Colonoscopy + biopsy for definitive diagnosis
Imaging → "apple core" lesion

Screening: start at age 45 until 75. If 76-86 → selectively screen

Stool based only DX — cheaper, less invasive, no bowel prep
- Hemoccult
- FIT - fecal immunohistochemical. Newer, more accurately detects microscopic heme
  ↳ annually, do not need to modify diet
- Stool DNA test (↑accuracy but expensive)
  Cologuard → fecal DNA + FIT
  ↳ every 3 years

If any of these positive → Colonoscopy → DX and TX

Direct Visualization — high sensitivity, less frequent
- CT Colonography: x-ray technology. Requires complete bowel prep and distension of colon. No sedation, less complications

If positive finding → Colonoscopy → DX and TX

+fam hx OR hx of IBD
↳ start screening at 40 or 10y before dx age
- repeat x 10yrs (5 if fam ↓60 at dx)

Pre-Op: pt must stop blood thinning/anti-platelet meds ~5days before. Low fiber diet for 1 week. Drink "bowel prep".

Procedure: anesthesia → 20-30 min procedure → home 30 min later (~3hr total)
- back to regular activity next day

Recommendations

Tier 1: Colonoscopy q10 yrs
FIT annually
- can consider Cologuard every 3 years
- CT colonography and capsule colonoscopy rare
  ↳ reserved for atypical cases or failed colonoscopy