

GI

- Pseudoachalasia, which is due to narrowing of the distal esophagus secondary to causes other than denervation (eg, esophageal cancer), can closely mimic achalasia. Clues pointing to pseudoachalasia include significant weight loss, rapid symptom onset, and presentation at age >60. Consequently, endoscopy is recommended to exclude malignancy in all patients with suspected achalasia

Acute liver failure	
Etiology	• Viral hepatitis (eg, HSV; CMV; hepatitis A, B, D & E) • Drug toxicity (eg, acetaminophen overdose, idiosyncratic) • Ischemia (eg, shock liver, Budd-Chiari syndrome) • Autoimmune hepatitis • Wilson disease • Malignant infiltration
Clinical presentation	• Generalized symptoms (eg, fatigue, lethargy, anorexia, nausea) • Right upper quadrant abdominal pain • Pruritus & jaundice due to hyperbilirubinemia • Renal insufficiency • Thrombocytopenia • Hypoglycemia
Diagnostic requirements	• Severe acute liver injury (ALT & AST often >1000 U/L) • Signs of hepatic encephalopathy (eg, confusion, asterixis) • Synthetic liver dysfunction (INR $\geq$1.5)

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- Chronic alcohol use is thought to potentiate acetaminophen hepatotoxicity by depleting glutathione levels and impairing the glucuronidation process.
- **Toxicity results from overproduction of the toxic metabolite N-acetyl-p-benzoquinone imine (NAPQI) which leads to hepatic necrosis.**
- **In ALF due to acetaminophen toxicity, LT is firmly indicated in patients with grade III or IV HE, PT >100 seconds, and serum creatinine >3.4 mg/dl (such as this patient). One year survival following LT for ALF is approximately 80%.**

Acute pancreatitis	
Etiology	• Chronic alcohol use (~40%) • Gallstones (~40%) • Hypertriglyceridemia • Drugs (eg, azathioprine, valproic acid, thiazides) • Infections (eg, CMV, <i>Legionella</i>, <i>Aspergillus</i>) • Iatrogenic (post-ERCP, ischemic/atheroembolic)
Clinical presentation	Diagnosis (requires 2 of the following) • Acute epigastric pain radiating to the back • ↑ Amylase or lipase >3 times normal limit • Abnormalities on imaging consistent with pancreatitis Other findings • ALT level >150 U/L suggests biliary pancreatitis • Severe disease: Fever, tachypnea, hypoxemia, hypotension

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- Amylase rises within 6-12 hours of symptom onset and may remain elevated for 3-5 days. Lipase rises within 4-8 hours of symptom onset but remains elevated longer than amylase (8-14 days). As a result, lipase is more useful and sensitive than amylase for diagnosis (especially in alcoholics and patients presenting later in the disease course).
- The serum triglyceride level generally must be >1,000 mg/dL to be considered a potential cause of pancreatitis.
- Cholesterol emboli : • Skin manifestations: Livedo reticularis (reticulated, mottled, discolored skin), blue toe syndrome • Kidney manifestations: Acute kidney injury • Gastrointestinal manifestations: Pancreatitis, mesenteric ischemia
- A ratio of AST to ALT >2 (thought to be due to hepatic deficiency of pyridoxal 5'-phosphate, an ALT enzyme cofactor) is very common in alcoholic liver disease (>90% in one study).

Ascites fluid characteristics	
Color	• Bloody: Trauma, malignancy, TB (rarely) • Milky: Chylous, pancreatic • Turbid: Possible infection • Straw color: Likely more benign causes
Neutrophils	• $<250/\text{mm}^3$: No peritonitis • $\geq 250/\text{mm}^3$: Peritonitis (secondary or spontaneous bacterial)
Total protein	• $\geq 2.5 \text{ g/dL}$ (high-protein ascites) ◦ CHF, constrictive pericarditis, peritoneal carcinomatosis, TB, Budd-Chiari syndrome, fungal (eg, coccidioidomycosis) • $<2.5 \text{ g/dL}$ (low-protein ascites) ◦ Cirrhosis, nephrotic syndrome
SAAG	• $\geq 1.1 \text{ g/dL}$ (indicates portal hypertension) ◦ Cardiac ascites, cirrhosis, Budd-Chiari syndrome • $<1.1 \text{ g/dL}$ (absence of portal hypertension) ◦ TB, peritoneal carcinomatosis, pancreatic ascites, nephrotic syndrome

Features of carcinoid syndrome	
Clinical manifestations	• Skin: flushing, telangiectasias, cyanosis • Gastrointestinal: diarrhea, cramping • Cardiac: valvular lesions (right > left side) • Pulmonary: bronchospasm • Miscellaneous: Niacin deficiency (dermatitis, diarrhea & dementia)
Diagnosis	• Elevated 24-hour urinary excretion of 5-HIAA • CT/MRI of abdomen & pelvis to localize tumor • OctreoScan to detect metastases • Echocardiogram (if symptoms of carcinoid heart disease are present)
Treatment	• Octreotide for symptomatic patients & prior to surgery/anesthesia • Surgery for liver metastases

Carcinoids are well differentiated neuroendocrine tumors found most commonly in the distal small intestine, proximal colon, and lung. with a strong propensity for metastasis to liver. These

tumors can secrete several products including histamine, serotonin, and vasoactive intestinal peptide that are metabolized in the liver. In patients with liver metastasis, these hormones are released directly into the systemic circulation, leading to carcinoid syndrome. Episodic flushing is the hallmark of carcinoid syndrome and occurs in almost 85% of patients. Severe flushing may be associated with hypotension and tachycardia. Secretory diarrhea may be accompanied by abdominal cramping. Other common features include cutaneous telangiectasias, bronchospasm, and tricuspid regurgitation. Pathognomonic plaque-like deposits of fibrous tissue occur most commonly on the endocardium on the right side of the heart, leading to tricuspid regurgitation and right-sided heart failure. Elevated 24-hour urinary 5-hydroxyindoleacetic acid can confirm the diagnosis in most patients.

- xylose is a monosaccharide that can be absorbed in the proximal small intestine without degradation by pancreatic or brush border enzymes. It is subsequently excreted in the urine. In the O-xylose test, the patient is given an oral dose of O-xylose, with subsequent assay of urine and venous blood. Patients with proximal small intestinal mucosal disease (eg, celiac disease) cannot absorb the O-xylose in the intestine, and urinary and venous O-xylose levels will be low. By contrast, patients with malabsorption due to enzyme deficiencies (eg, chronic pancreatitis) will have normal absorption of O-xylose (Choice 0). A false-positive O-xylose test (ie, decreased urinary excretion of O-xylose despite normal mucosal absorption) can be seen in patients with delayed gastric emptying or impaired glomerular filtration.

Acute cholangitis	
Clinical presentation	• Fever, jaundice, right upper quadrant pain (Charcot triad) • Mental status changes, hypotension (Reynolds pentad) • Liver failure • Acute kidney injury
Diagnosis	• Biliary dilation on ultrasound or CT scan • ↑ Alkaline phosphatase, gamma-glutamyl transpeptidase, direct bilirubin • Leukocytosis, ↑ C-reactive protein
Treatment	• Biliary drainage: Endoscopic retrograde cholangiopancreatography with sphincterotomy or percutaneous transhepatic cholangiography • Broad-spectrum antibiotics: Beta-lactam/beta-lactamase inhibitor, third-generation cephalosporin + metronidazole

Acalculous cholecystitis	
Risk factors	• Severe trauma, extensive burns, recent surgery (eg, cardiopulmonary, aortic, abdominal) • Prolonged fasting or TPN • Critical illness (sepsis, ICU, mechanical ventilation)
Clinical presentation	• Unexplained fever, vague/RUQ abdominal discomfort, leukocytosis • Possible jaundice, RUQ mass, abnormal LFTs
Diagnosis	• Abdominal ultrasound (preferred) • Cholescintigraphy (HIDA scan) or abdominal CT scan if ultrasound not diagnostic

postcholecystectomy syndrome (PCS). PCS refers to persistent abdominal pain or dyspepsia (eg, nausea) that occurs either postoperatively (early) or months to years (late) after a cholecystectomy. PCS can be due to biliary (eg, retained common bile duct or cystic duct stone, biliary dyskinesia) or extra-biliary (eg, pancreatitis, peptic ulcer disease, coronary artery disease) causes. Patients usually notice the same pain they had prior to surgery, new pain just after surgery, or the same pain that never went away.

Pancreatic adenocarcinoma	
Risk factors	• Smoking • Hereditary pancreatitis • Nonhereditary chronic pancreatitis • Obesity & lack of physical activity
Clinical presentation	• Systemic symptoms (eg, weight loss, anorexia) (>85%) • Abdominal pain/back pain (80%) • Jaundice (56%) • Recent-onset atypical diabetes mellitus • Unexplained migratory superficial thrombophlebitis • Hepatomegaly & ascites with metastasis
Laboratory studies	• Cholestasis (↑ alkaline phosphatase & direct bilirubin) • ↑ Cancer-associated antigen 19-9 (not as a screening test) • Abdominal ultrasound (if jaundiced) or CT scan (if no jaundice)

Overview of chronic pancreatitis	
Etiology	• Alcohol use • Cystic fibrosis (common in children) • Ductal obstruction (eg, malignancy, stones) • Autoimmune
Clinical presentation	• Chronic epigastric pain with intermittent pain-free intervals • Malabsorption—steatorrhea, weight loss • Diabetes mellitus
Laboratory results/imaging	• Amylase/lipase can be normal & nondiagnostic • CT scan or MRCP can show calcifications, dilated ducts & enlarged pancreas
Treatment	• Pain management • Alcohol & smoking cessation • Frequent, small meals • Pancreatic enzyme supplements

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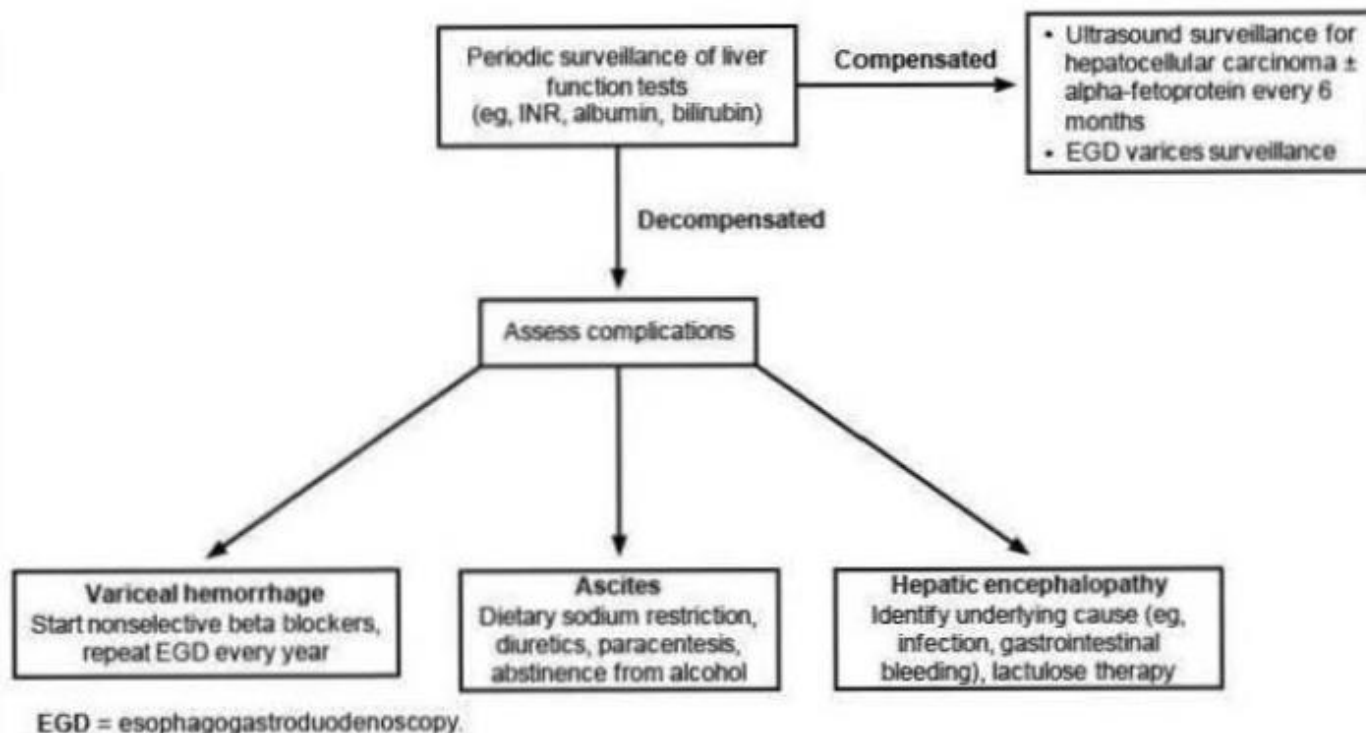
- In contrast to widespread Inflammation in acute pancreatitis, CP causes patchy inflammation and fibrosis (burned-out pancreas) and can present with normal or only slightly elevated serum amylase and lipase. Abdominal imaging showing pancreatic calcifications is the best way to confirm CP

Common causes of steatorrhea	
Pancreatic insufficiency	• Chronic pancreatitis due to alcohol abuse, cystic fibrosis, or autoimmune/hereditary pancreatitis • Pancreatic cancer
Bile salt-related	• Small-bowel Crohn disease • Bacterial overgrowth • Primary biliary cirrhosis • Primary sclerosing cholangitis • Surgical resection of ileum (at least 60-100 cm)
Impaired intestinal surface epithelium	• Celiac disease • AIDS enteropathy • Giardiasis
Other rare causes	• Whipple disease • Zollinger-Ellison syndrome • Medication-induced

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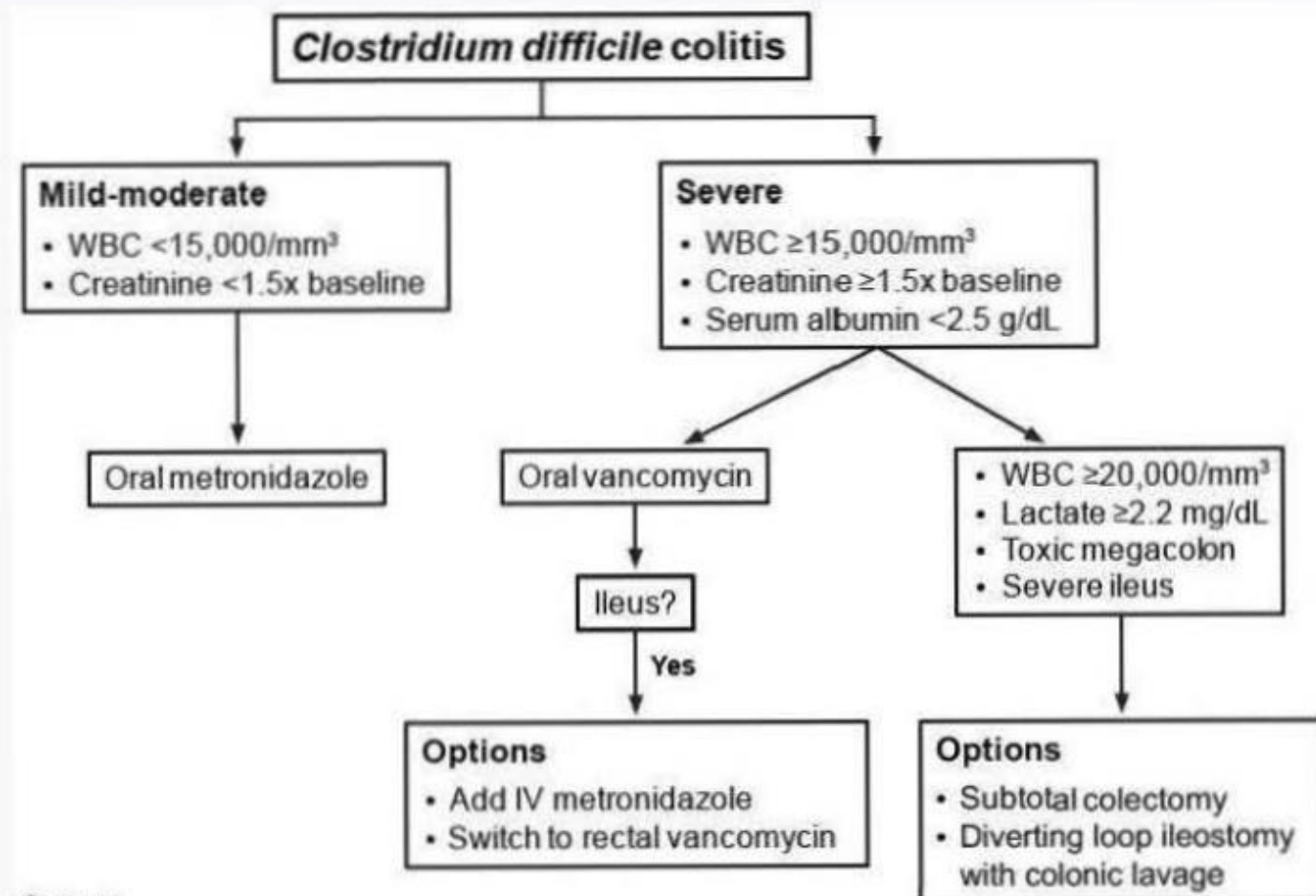
- In patients with cirrhosis, spider angioma and palmar erythema both arise from hyperestrogenism due to impaired hepatic metabolism of circulating estrogens, a process that begins in the cytochrome P450 system. Circulating estrogens affect vascular wall dilation. Spider angioma consists of a central, dilated arteriole surrounded by smaller radiating vessels. Palmar erythema is a result of increased normal speckling of the palm due to increased vasodilation, especially at the thenar and hypothenar eminences. Other manifestations of hyperestrogenism in patients with cirrhosis include gynecomastia, testicular atrophy, and decreased body hair in males.
- Dupuytren's contracture occurs when the palmar fascia thickens and shortens, deforming the hand. It is usually most notable initially in the fourth and fifth digits and occurs due to fibroblast proliferation and collagen deposition, which are likely worsened by oxidative stress from increased free radical production.

Management of cirrhosis



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- There are 2 subtypes of HRS. Type 1 is rapidly progressive: most patients die within 10 weeks without treatment. Type 2 progresses more slowly, with an average survival of 3-6 months. The most common causes of death are infection and hemorrhage.



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<i>Clostridium difficile</i> colitis	
Risk factors	• Recent antibiotics • Hospitalization • PPI
Pathogenesis	• Disruption of intestinal flora → <i>C difficile</i> overgrowth • Exotoxins cause mucosal inflammation/injury
Clinical presentation	• Watery diarrhea (most common) • Fulminant colitis/toxic megacolon
Diagnosis	• Stool PCR
Treatment	• Oral metronidazole or vancomycin

- A polyp is a grossly visible protrusion from the flat mucosal surface of the intestine. Most polyps are benign. Polyps can be classified as follows:
- 1. Hyperplastic polyps: These are the most common non-neoplastic polyps in the colon. These arise from hyperplastic mucosal proliferation. No further work-up is needed.
- 2. Hamartomatous polyps: These include juvenile polyp (a non-malignant lesion, generally removed due to the risk of bleeding) and Peutz-Jeghers polyp (generally non-malignant).
- 3. Adenoma: This is the most common type of polyp found in the colon. It is present in approximately 30-50 % of elderly people. These polyps are potentially premalignant; however, <1% of such polyps become malignant. Most polyps are asymptomatic; less than 5% of patients have positive occult stool tests.
- The probability of an adenomatous polyp progressing into cancer can be judged clinically by the lesion's gross appearance, histology, and size.
- 1. Adenoma can be sessile or stalked (pedunculated). Cancer is seen more commonly in sessile polyps.
- 2. Histologically, adenoma is divided into tubular, tubulovillous, and the villous variety. Villous adenomas, which are most commonly sessile, are most likely (three times more likely than tubular adenoma for malignant transformation) to become malignant among all three varieties. Second on the list is tubulovillous, followed by tubular adenoma with the least risk of malignant transformation. Therefore, as the villus component increases, the risk of malignancy increases.
- 3. The likelihood of an adenomatous lesion containing invasive cancer also depends on the size of the polyp. The risk is negligible (<2%) with < 1.5 cm polyp, intermediate (2-10%) with 1.5--2.5 cm size polyp, and substantial (10%) with polyps >2.5 cm in size.
- The American Cancer Society and the United States Preventive Services Task Force (USPSTF) strongly encourage routine colon cancer screening in all patients age ≥50. Screening can be performed using high-sensitivity fecal occult blood testing (FOBT) annually, flexible sigmoidoscopy every 5 years combined with FOBT every 3 years, or colonoscopy every 10 years. All 3 strategies decrease colon cancer mortality. Colonoscopy is the most sensitive and specific test, but it is also the most costly and expertise dependent.

- Patients with a history of colon cancer in a first degree relative should be screened at age 40 or 10 years before the age of the relative's diagnosis. This patient's father was diagnosed with colon cancer at age 50, and he should begin screening at age 40.

Recommendations for lung cancer screening	
Recommended test	Low-dose chest CT
Recommended interval	Yearly
Age for screening	55-80
Eligibility for screening based on smoking history	- Patient has ≥ 30-pack-year smoking history AND - Patient is a current smoker or quit smoking within the last 15 years
Termination of screening	- Age > 80 OR - Patient successfully quit smoking for ≥ 15 years OR - Patient has other medical problems that significantly limit life expectancy or ability/willingness to undergo lung cancer surgery

Diffuse esophageal spasm	
Pathophysiology	Uncoordinated, simultaneous contractions of esophageal body
Symptoms	- Intermittent chest pain Dysphagia for solids & liquids
Diagnosis	- Esophagram: "Corkscrew" pattern - Manometry: Intermittent peristalsis, multiple simultaneous contractions
Treatment	- Calcium channel blockers - Alternate: Nitrates, tricyclics

Clinical features of acute diverticulitis	
Clinical presentation	• Abdominal pain (usually lower left quadrant) • Fever, nausea & vomiting • Ileus (peritoneal irritation)
Diagnosis	• Abdominal CT (oral & intravenous contrast)
Management	• Bowel rest • Antibiotics (eg, ciprofloxacin, metronidazole)
Complications	• Abscess, obstruction, fistula, perforation

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Clinical features of colovesical fistula	
Etiology	• Diverticular disease (sigmoid most common) • Crohn disease • Malignancy (colon, bladder, pelvic organs)
Clinical presentation	• Pneumaturia (air in urine) • Fecaluria (stool in urine) • Recurrent urinary tract infections (mixed flora)
Diagnosis	• Abdominal CT with oral or rectal (not intravenous) contrast • Colonoscopy to exclude colonic malignancy

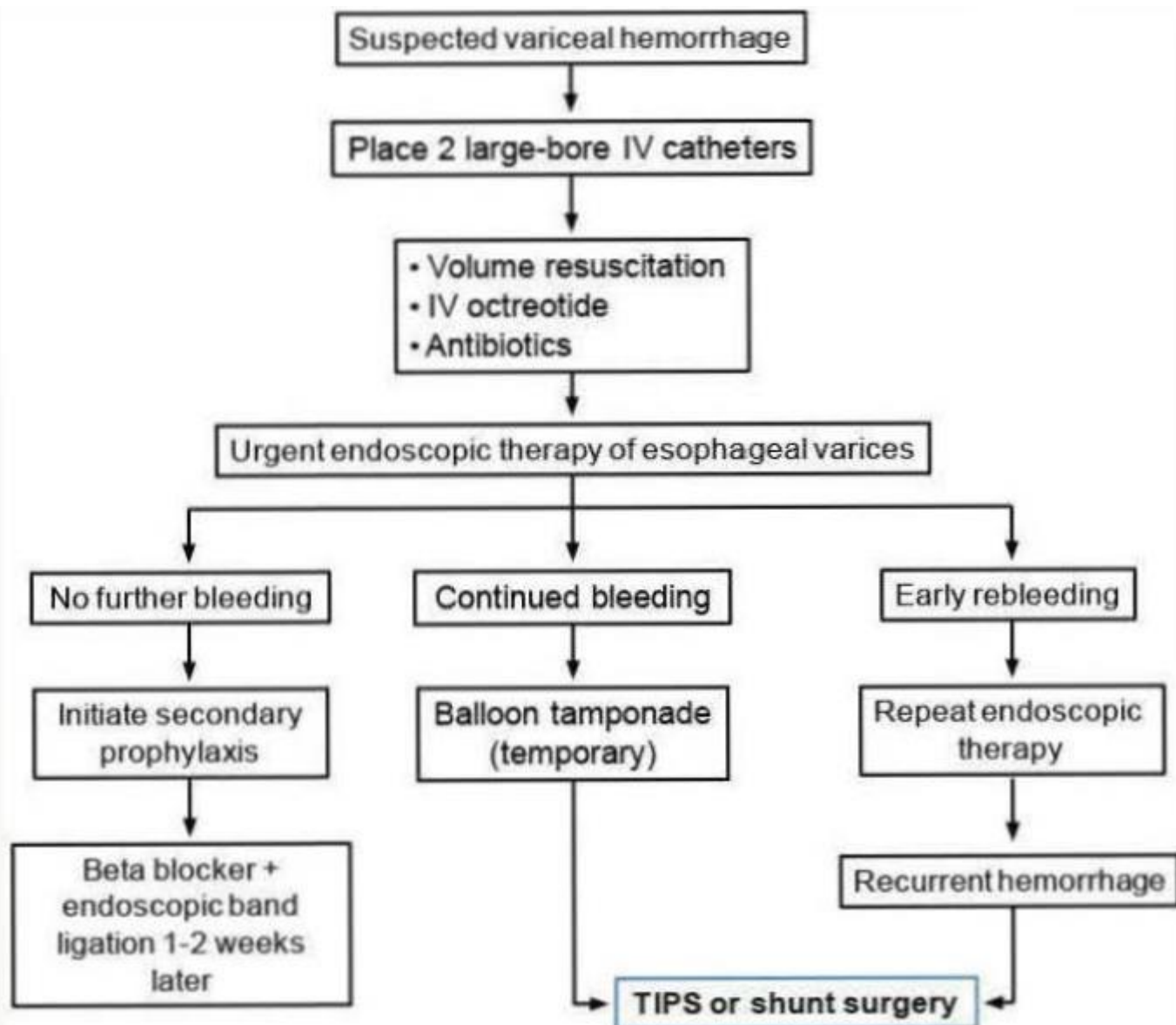
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- Abdominal CT scan with oral or rectal (not intravenous) contrast can confirm the diagnosis by showing contrast material in the bladder with thickened colonic and vesicular walls. Colonoscopy is usually recommended in patients diagnosed with colovesical fistula to exclude colonic malignancy. Treatment is typically surgical after resolution of the Infection.
- The impact of dietary fiber on the Initial formation of diVerticulosis is unclear. However the incidence of acute diverticular complications is lower in individuals with a high intake of fruit and vegetable fiber. Physical activity is also inversely correlated with the risk of complications. Factors associated with an increased risk of complications Include heavy meat consumption, aspirin or nonsteroidal anti-inflammatory drug use (Choice F) obesity, and possibly smoking.
- Drug-induced liver disease can also be broadly categorized according to morphology:
 - 1) cholestasis, which is caused by medications such as chlorpromazine, nitrofurantoin erythromycin, and anabolic steroids;
 - 2) fatty liver, which is caused by medications such as tetracycline, valproate, and anti-retrovirals;
 - 3) hepatitis, which is caused by medications such as halothane, phenytoin, isoniazid, and alpha-methyldopa;

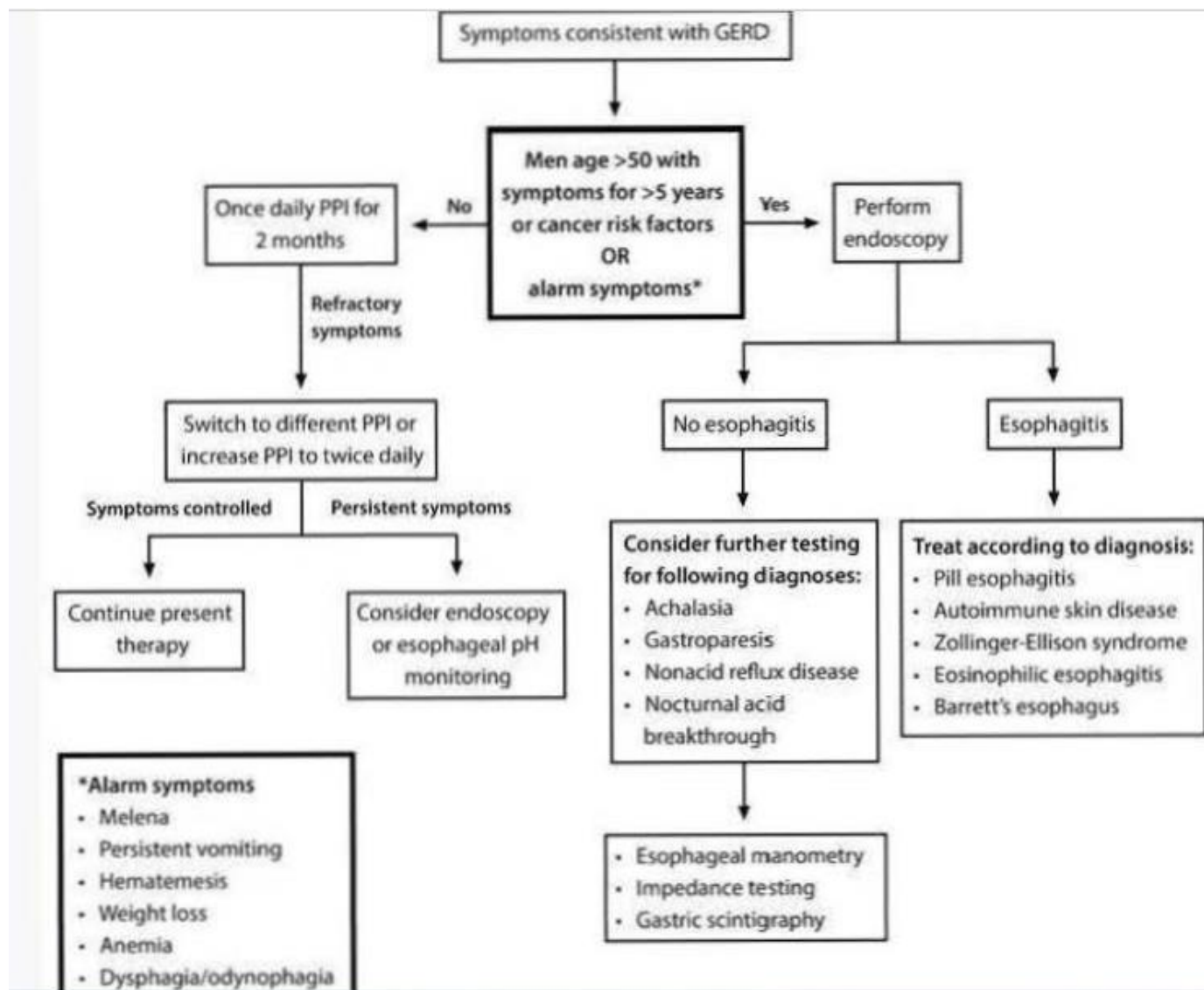
- 4) toxic or fulminant liver failure, which is caused by medications such as carbon tetrachloride and acetaminophen; and
- 5) granulomatous, which is caused by medications such as allopurinol and phenylbutazone.
- Since this patient is on isoniazid, idiosyncratic liver injury with a histological picture similar to viral hepatitis must be considered. While extrahepatic hypersensitivity manifestations like rash, arthralgias, fever, leukocytosis, and eosinophilia are common in patients with drug-induced liver injury, they are characteristically absent in cases of Isoniazid-induced hepatic cell injury
- Definitive diagnosis of dyspepsia often requires endoscopy, a procedure that is invasive and relatively expensive, especially given the benign nature of most cases. Endoscopy should be considered in patients age >55 or with alarm symptoms (eg, weight loss, bleeding, anemia, dysphagia, persistent vomiting). In those without alarm symptoms, evaluation for H pylori infection (eg, urea breath testing, stool antigen testing) can be performed. Patients who fail to improve after initial management (eg, antibiotics if positive for H pylori, proton pump inhibitor) should undergo endoscopy.

Clinical features of esophageal perforation	
Etiology	• Spontaneous rupture (Boerhaave syndrome) • Instrumentation (eg, endoscopy) • Esophagitis (infectious/pills/caustic) • Esophageal ulcer
Clinical presentation	• Chest & abdominal pain, systemic findings (eg, fever) • Subcutaneous emphysema in the neck • Hamman sign (crunching sound on chest auscultation)
Diagnosis	• CXR or CT scan: Wide mediastinum, pneumomediastinum, pneumothorax, air around paraspinal muscles, pleural effusion (late) • CT scan: Esophageal wall thickening, mediastinal air fluid level • Water-soluble contrast esophagogram: Leak at perforation site
Management	• Antibiotics & supportive care for all patients • Surgical repair for significant leakage with systemic inflammatory response

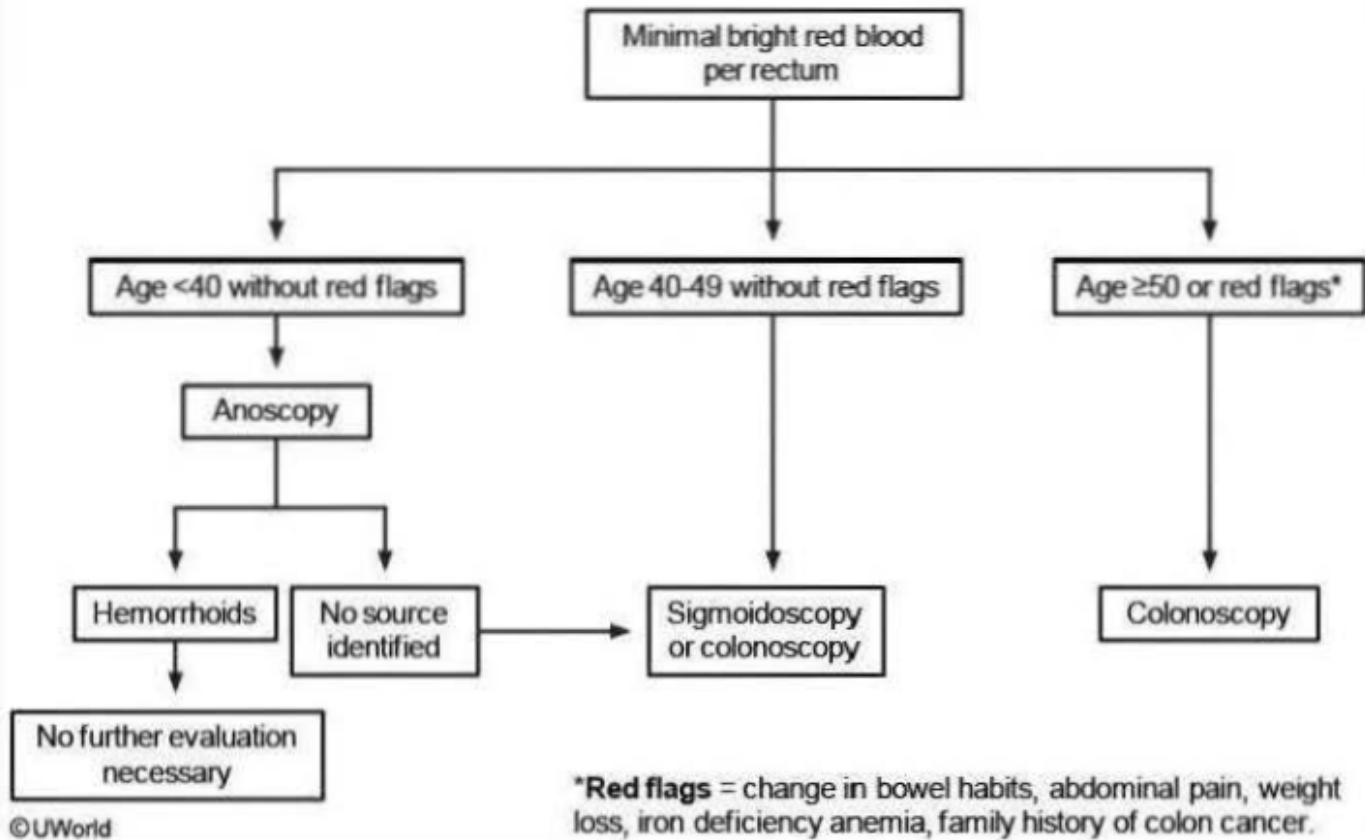
Characteristics of gastroesophageal mural injury		
	Mallory-Weiss tear	Boerhaave syndrome
Etiology	• Upper gastrointestinal mucosal tear • Caused by forceful retching (↑pressure) • Submucosal arterial or venule plexus bleeding	• Esophageal transmural tear • Caused by forceful retching (↑pressure) • Esophageal air/fluid leakage into nearby areas (eg, pleura)
Clinical presentation	• Vomiting, retching • Hematemesis • Epigastric pain	• Vomiting, retching, chest & upper abdominal pain • Odynophagia, fever, dyspnea & septic shock can ensue • Subcutaneous emphysema may be seen
Laboratory/imaging	• EGD confirms diagnosis	• CT or contrast esophagography with Gastrografin confirms diagnosis • Chest x-ray: Pneumomediastinum & pleural effusions • Pleural fluid analysis: Exudative, low pH, very high amylase (>2,500 IU/L)
Treatment	• Most tears heal spontaneously • Endoscopic therapy for continual bleed	• Surgery: For thoracic perforations • Conservative measures (eg, antibiotics): For cervical perforations



Clinical features of chronic hepatitis C	
Clinical presentation	• Can be asymptomatic or develop fatigue (most common) • Other nonspecific symptoms (eg, nausea, anorexia, myalgia, arthralgia, weakness, weight loss) • Serum transaminases can be elevated or normal (up to 1/3 of patients) • Can progress to cirrhosis in up to 20% of patients • Increased risk of hepatocellular carcinoma
Extrahepatic manifestations	• Heme: Essential mixed cryoglobulinemia • Renal: Membranoproliferative glomerulonephritis • Skin: Porphyria cutanea tarda, lichen planus • Endocrine: Increased risk of diabetes



Evaluation of minimal bright red blood per rectum



- Patients with upper (but not lower) GI bleeding often have an elevated blood urea nitrogen (BUN) and elevated BUN/creatinine ratio. Possible causes include increased urea production from intestinal breakdown of hemoglobin and increased urea reabsorption in the proximal tubule due to associated hypovolemia.

Red blood cell transfusion thresholds	
Hemoglobin (g/dL)	Recommendation
<7	• Generally indicated
7-8	• Cardiac surgery • Oncology patients in treatment • Heart failure
8-10	• Symptomatic anemia • Ongoing bleeding • Acute coronary syndrome • Noncardiac surgery
>10	• Not generally indicated

Gilbert syndrome	
Epidemiology	• More common in males • Most common inherited disorder of bilirubin glucuronidation
Pathogenesis	• AR or AD mutation in <i>UGT1A1</i> gene • ↓ UDP-glucuronosyltransferase activity → ↑ unconjugated bilirubin
Clinical findings	• Intermittent episodes of mild jaundice • Provoked by stress (eg, infection, fasting, vigorous exercise, surgery)
Diagnosis	• Unconjugated hyperbilirubinemia on repeat testing • Normal CBC, blood smear, reticulocyte count • Normal AST, ALT, alkaline phosphatase
Treatment	• No specific treatment

- All patients with chronic liver disease should be immunized against hepatitis A and B unless they are already immune, as they are at high risk for acute hepatic failure or cirrhosis upon infection with viral hepatitis

Solid liver masses	
Focal nodular hyperplasia	• Associated with anomalous arteries • Arterial flow & central scar on imaging
Hepatic adenoma	• Women on long-term oral contraceptives • Possible hemorrhage or malignant transformation
Regenerative nodules	• Acute or chronic liver injury (eg, cirrhosis)
Hepatocellular carcinoma	• Systemic symptoms • Chronic hepatitis or cirrhosis • Elevated alpha-fetoprotein
Liver metastasis	• Single/multiple lesions • Known extrahepatic malignancy

- Needle biopsy is not recommended for suspected hepatic adenoma due to the risk of bleeding, and surgical excision is preferred. Possible long-term complications include progressive growth, rupture, and malignant transformation.

- Hypokalemia. which can exacerbate HE as the resultant intracellular acidosis) excreted Intracellular potassium replaced by hydrogen ions to maintain electroneutrality) causes increased NH₄⁺ production (glutamine conversion) in renal tubular cells
- Metabolic alkalosis (elevated bicarbonate), which can also exacerbate HE as It promotes conversion of ammonium (NH₄⁺), which cannot enter the CNS, to NH₃ which can As a result, patients

Colon cancer screening in high-risk patients	
Family history of adenomatous polyps or CRC • First-degree relative at age <60	• Colonoscopy at age 40 or 10 years before the age of diagnosis in the relative (whichever comes first) • Repeat every 3-5 years
Inflammatory bowel disease • Ulcerative colitis • Crohn colitis	• Begin 8 years post diagnosis (12-15 years if disease only in left colon) • Colonoscopy with biopsies every 1-2 years
Familial adenomatous polyposis	• Begin at age 10-12 • Colonoscopy every year
Hereditary nonpolyposis CRC (Lynch syndrome)	• Begin at age 20-25 • Colonoscopy every 1-2 years

Toxic megacolon typically presents with total or segmental nonobstructive colonic dilation, severe bloody diarrhea, and systemic findings (eg, fever, tachycardia). IBD patients are at the highest risk of developing toxic megacolon early in the disease, within 3 years of diagnosis in most cases. Other causes of toxic megacolon include ischemic colitis, volvulus, diverticulitis, infections (eg, *Clostridia difficile*), and obstructive colon cancer (less common). Diagnosis is confirmed by plain abdominal x-rays and ≥3 of the following: fever >38 C (100.4 F), pulse >120/min, white blood cells >10,500/μL, and anemia. Other findings can include volume depletion, altered mental status, hypotension, and electrolyte abnormalities. Plain films usually reveal dilated right or transverse colon (>6 cm), possible multiple air-fluid levels, and thick haustral markings that do not extend across the entire lumen (white arrows in above image).

Toxic megacolon is a medical emergency that can progress rapidly and result in colonic perforation. Treatment includes intravenous fluids, broad-spectrum antibiotics, and bowel rest. Intravenous corticosteroids are preferred for treating IBD-induced toxic megacolon. Emergency surgery (subtotal colectomy with end-ileostomy as the procedure of choice) may be required if the colitis does not resolve.

The most common cause of iron deficiency anemia in the elderly is gastrointestinal blood loss. The next step in evaluation would be colonoscopy and endoscopy. A single negative occult blood test does not exclude the possibility of gastrointestinal bleeding.

Clinical features of irritable bowel syndrome	
Rome diagnostic criteria	Recurrent abdominal pain/discomfort ≥ 3 days/month for the past 3 months & ≥ 2 of the following: • Symptom improvement with bowel movement • Change in frequency of stool • Change in form of stool
Warning signs/symptoms	Signs/symptoms suggesting etiologies other than IBS • Rectal bleeding • Nocturnal (awakens from or prevents sleep) or worsening abdominal pain • Weight loss • Abnormal laboratory findings (eg, anemia & electrolyte disorders)

The hallmark of ischemic hepatopathy is a rapid and massive increase in the transaminases with modest accompanying elevations in total bilirubin and alkaline phosphatase. In patients who survive the underlying cause of their hypotension (e.g. septic shock, heart failure), liver enzymes typically return to normal within one to two weeks.

Pellagra ("rough skin" in Italian vernacular) is due to **niacin deficiency** and is characterized by the "3 Ds": **dermatitis, diarrhea, and dementia**:

- Dermatitis is primarily on sun-exposed areas of the body and is characterized by rough, hyperpigmented, scaly skin.
- Diarrhea is often associated with abdominal pain, nausea, and loss of appetite.
- Dementia is due to neuronal degeneration in the brain and spinal cord and can lead to memory loss, affective symptoms (eg, depressed mood in this patient), and psychosis.

Niacin is present in a broad variety of foods and can be synthesized endogenously from **tryptophan**. In developing countries, niacin deficiency is seen in populations that subsist primarily on corn products (niacin in corn occurs in a bound, unabsorbable form). In developed countries, it is primarily seen in patients with impaired nutritional intake (eg, alcoholism, chronic illness). Pellagra can also be seen occasionally in those with carcinoid syndrome (due to depletion of tryptophan) or Hartnup disease (congenital disorder of tryptophan absorption). Prolonged isoniazid therapy can interfere with metabolism of tryptophan and occasionally lead to pellagra.

Nonalcoholic fatty liver disease	
Definition	• Hepatic steatosis on imaging or biopsy • Exclusion of significant alcohol use • Exclusion of other causes of fatty liver
Clinical features	• Mostly asymptomatic • Metabolic syndrome • +/- Steatohepatitis (AST/ALT ratio <1) • Hyperechoic texture on ultrasound
Treatment	• Diet & exercise • Consider bariatric surgery if BMI ≥35

Major risk factors for pancreatic cancer	
Hereditary	• First-degree relative with pancreatic cancer • Hereditary pancreatitis • Germline mutations (eg, BRCA1, BRCA2, Peutz-Jeghers syndrome)
Environmental	• Cigarette smoking (most significant) • Obesity, low physical activity • Nonhereditary chronic pancreatitis

Primary biliary cholangitis	
Pathogenesis	• Autoimmune destruction of intrahepatic bile ducts
Clinical features	• Affects middle-age women • Insidious onset of fatigue & pruritus • Progressive jaundice, hepatomegaly, cirrhosis • Cutaneous xanthomas & xanthelasmas
Laboratory findings	• Cholestatic pattern of liver injury (↑↑ alkaline phosphatase, ↑ aminotransferases) • Antimitochondrial antibody • Severe hypercholesterolemia Mainly HDL , No Cardiovascular Risk
Treatment	• Ursodeoxycholic acid (delays progression) • Liver transplantation for advanced disease
Complications	• Malabsorption, fat-soluble vitamin deficiencies • Metabolic bone disease (osteoporosis, osteomalacia) • Hepatocellular carcinoma

Primary sclerosing cholangitis	
Clinical features	• Fatigue & pruritus • Majority of patients asymptomatic at time of diagnosis • About 90% of patients have underlying inflammatory bowel disease, mainly ulcerative colitis
Laboratory/imaging	• Cholestatic liver function test pattern (serum aminotransferases typically <300 U/L) • Multifocal stricturing/dilation of intrahepatic &/or extrahepatic bile ducts on cholangiography
Liver biopsy	• Fibrous obliteration of bile ducts with concentric replacement by connective tissue in an "onion-skin" pattern
Complications	• Intrahepatic &/or extrahepatic biliary stricture • Cholangitis & cholelithiasis (cholesterol &/or pigment stones) • Cholangiocarcinoma (10%-15% lifetime risk) • Cholestasis (eg, ↓ fat-soluble vitamins, osteoporosis) • Colon cancer

This patient's history of anticoagulation, symptoms of weakness and dizziness, and evidence of anemia and tachycardia should immediately raise concern for internal hemorrhage. The risk of bleeding while on warfarin therapy is greatest in patients with risk factors such as diabetes, age > 60, hypertension and alcoholism. A supratherapeutic INR also increases one's risk of hemorrhage, although in the case of retroperitoneal hematomas, even therapeutic-range INRs confer significant risk. This patient's back pain should put retroperitoneal hematoma high on the differential, and warrants an abdominal CT. The above CT image reveals a large isodense collection in the right retroperitoneum, which lies anterior to the psoas muscle and displaces the right kidney anteriorly. The CT findings are consistent with a spontaneous retroperitoneal hematoma (**Choice C**).

Small intestinal bacterial overgrowth	
Etiology	• Anatomical abnormalities (eg, strictures, surgery) • Motility disorders (eg, diabetes mellitus, scleroderma) • Other causes (eg, end-stage renal disease, AIDS, cirrhosis, advanced age)
Signs/ symptoms	• Abdominal pain, diarrhea, bloating, excess flatulence, malabsorption, weight loss, anemia, & nutritional deficiencies
Diagnosis	• Endoscopy (gold standard) with jejunal aspirate showing $> 10^5$ organisms/mL • Glucose breath hydrogen testing
Common organisms	• Streptococci, <i>Bacteroides</i>, <i>Escherichia</i>, <i>Lactobacillus</i>
Treatment	• 7-10-day course of antibiotics (eg, rifaximin, amoxicillin-clavulanate) • Avoid antimotility agents (eg, narcotics) • Dietary changes (eg, high-fat, low-carbohydrate) • Trial of promotility agents (eg, metoclopramide)

Spontaneous bacterial peritonitis	
Clinical presentation	• Temperature ≥ 37.8 C (100 F) • Abdominal pain/tenderness • Altered mental status (abnormal connect-the-numbers test) • Hypotension, hypothermia, paralytic ileus with severe infection
Diagnosis from ascitic fluid	• PMNs $\geq 250/\text{mm}^3$ • Positive culture, often gram-negative organisms (eg, <i>Escherichia coli</i>, <i>Klebsiella</i>) • Protein < 1 g/dL • SAAG ≥ 1.1 g/dL
Treatment	• Empiric antibiotics - third-generation cephalosporins (eg, cefotaxime) • Fluoroquinolones for SBP prophylaxis

Hypotension, hypothermia, or paralytic ileus (dilated loops of bowel on x-ray) Indicate severe SBP

Whipple's disease is a rare multi-systemic illness. It is an infectious disease caused by the bacillus *Tropheryma whippelii*. It is most commonly seen in white men in the fourth-to-sixth decades of life, and often presents with weight loss. Gastrointestinal symptoms include abdominal pain, diarrhea, and malabsorption with distension, flatulence, and steatorrhea. Extraintestinal manifestations include migratory polyarthropathy, chronic cough, and myocardial or valvular involvement leading to congestive failure or valvular regurgitation. Later stages of the disease may be characterized by dementia and other central nervous system findings, such as supranuclear ophthalmoplegia and myoclonus. Intermittent low-grade fever, pigmentation and lymphadenopathy may also be occasionally seen. PAS-positive material in the lamina propria of the small intestine is a classical biopsy finding.

Wilson's disease is a rare, autosomal recessive disease most often identified in younger individuals aged 5 to 40 years. The genetic mutations associated with Wilson's disease hinder copper metabolism by reducing the formation and secretion of ceruloplasmin and by decreasing the secretion of copper into the biliary system. Copper is a pro-oxidant, and as it accumulates in greater quantities within the liver, it causes damage to the hepatic tissue through the generation of free radicals. Eventually copper leaks from injured hepatocytes into the blood to be deposited in various tissues, including the basal ganglia (hepatolenticular degeneration) and cornea.

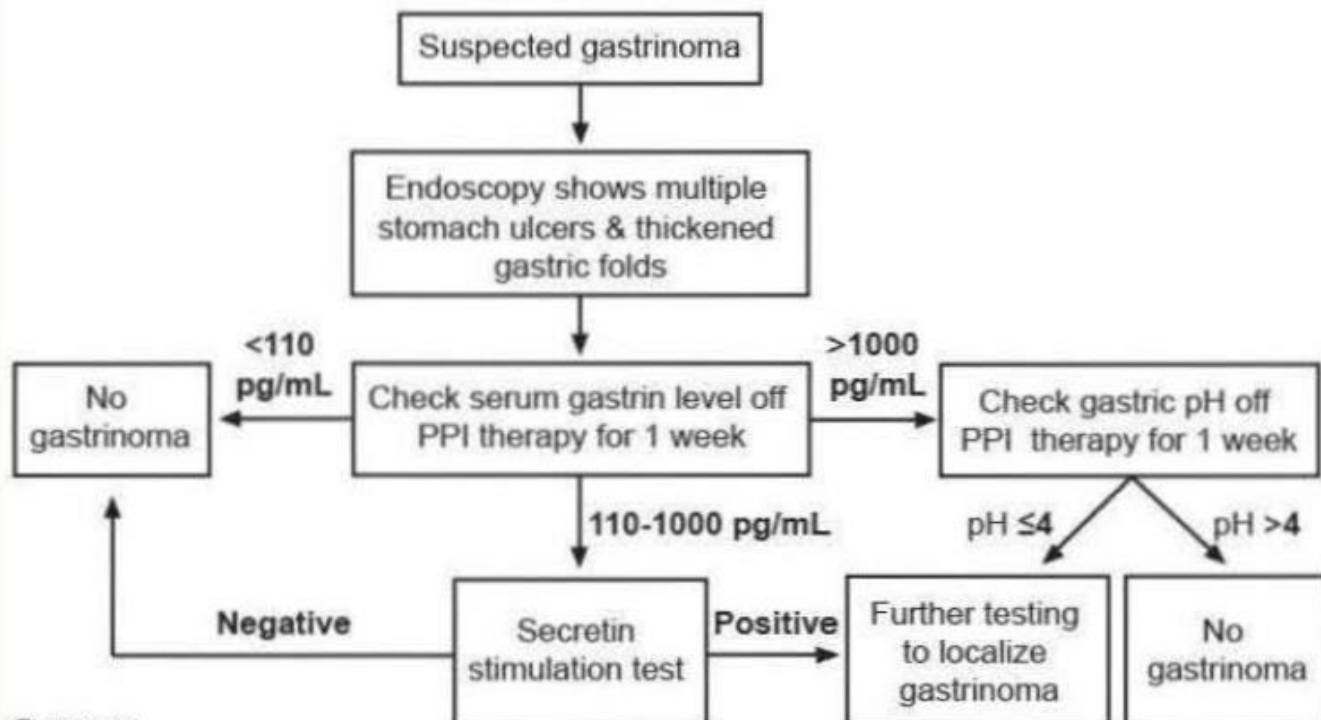
Liver involvement in Wilson's disease may present as asymptomatic liver function abnormalities, chronic hepatitis, fulminant hepatitis, portal hypertension, or macronodular cirrhosis. Neuropsychiatric symptoms can include Parkinsonian-like tremor, rigidity, ataxia, slurred speech, drooling, personality changes, depression, paranoia, and catatonia. Wilson's disease is also associated with Fanconi syndrome, hemolytic anemia, and neuropathy. The gold standard for diagnosis is liver biopsy that demonstrates a quantitative hepatic copper level >250 mcg/gram dry weight. More commonly, diagnosis is confirmed by the presence of low serum ceruloplasmin (particularly <20 mg/dL) in conjunction with increased urinary copper excretion or Kayser-Fleischer rings.

Treatment must be adhered to for the patient's lifetime and focuses on removing accumulated copper in the tissues and preventing re-accumulation. First-line medications include copper chelators like d-penicillamine or trientine. Oral zinc is also recommended as it prevents copper absorption. Liver transplantation may be the only option for those with fulminant hepatic failure or decompensated liver disease that does not respond to pharmacotherapy.

Zenker diverticulum	
Clinical features	• Usually $\geq$ age 60 • More common in males • Dysphagia • Halitosis • Regurgitation & aspiration • Variable neck mass
Diagnosis	• Barium esophagram • Esophageal manometry
Management	• Open/endoscopic surgery • Cricopharyngeal myotomy

Clinical manifestations of trace mineral deficiencies	
Chromium	• Impaired glucose control in diabetics
Copper	• Brittle hair • Skin depigmentation • Neurologic dysfunction (eg, ataxia, peripheral neuropathy) • Sideroblastic anemia • Osteoporosis
Iron	• Microcytic anemia
Selenium	• Thyroid dysfunction • Cardiomyopathy • Immune dysfunction
Zinc	• Alopecia • Pustular skin rash (perioral region & extremities) • Hypogonadism • Impaired wound healing • Impaired taste • Immune dysfunction

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Zollinger-Ellison syndrome	
Epidemiology	• Age 20-50 • 80% Sporadic/20% MEN1
Clinical features	• Multiple & refractory peptic ulcers • Ulcers distal to duodenum • Chronic diarrhea
Diagnosis	• Markedly elevated serum gastrin (>1000 pg/mL) in the presence of normal gastric acid (pH <4)
Workup	• Endoscopy • CT/MRI & somatostatin receptor scintigraphy for tumor localization

● **MEN1** = multiple endocrine neoplasia type 1

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