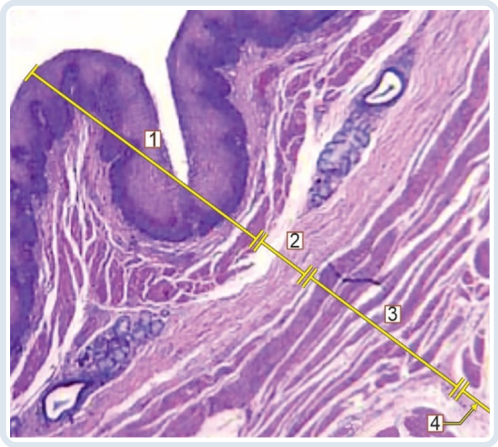
QUESTION

A young male child presents with failure to pass meconium and has been diagnosed with megacolon. Which of the following layers is affected by the absence of neurons?



A 1 and 2

B **2 and 3** CORRECT

C 3 and 4

D 1 and 4

RIGHT ANSWER

OK **B. 2 and 3**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which of the following about the enteric nervous regulation system is false?

1. Interstitial cells of Cajal are pacemakers
2. It can function independently of the brain and spinal cord
3. All the afferents are from the vagus nerve
4. Myenteric plexus is in the Lamina propria

A 1, 2

B 1, 2, 3

C 1, 3, 4

D **3, 4** CORRECT

OK RIGHT ANSWER

D. 3, 4

WHY THIS IS RIGHT

The Enteric Nervous System (ENS) is like a quiet maestro conducting digestion:
• Coordinates motility, secretion, and blood flow
• Works independently of CNS (but can be modulated by it)

Can function independently of CNS input. Interstitial cells of Cajal act as pacemakers generating slow waves. Afferents are not exclusively vagal; significant intrinsic and spinal afferents exist.

Feature	Myenteric (Auerbach)	Submucosal (Meissner)
Location	Between muscle layers	Submucosa
Function	Motility	Secretion, blood flow
Extent	Entire GI tract	Mainly small intestine

Clinical correlations

Disease	Defect
Hirschsprung disease	Absence of ganglion cells
Achalasia	Loss of inhibitory neurons
IBS	Dysregulated ENS

QUESTION

Which of the following sites has the least concentration of sodium ions in the fluid?

A **Salivary glands** CORRECT

B Stomach

C Duodenum

D Ileum

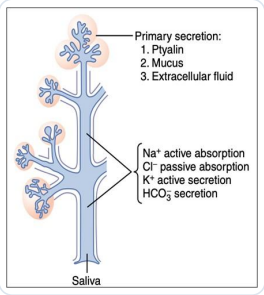
RIGHT ANSWER

OK **A. Salivary glands**

WHY THIS IS RIGHT

GI secretions are not static liquids. They get modified along ducts and segments like a river changing chemistry as it flows.
Saliva is the only GI fluid that becomes hypotonic
-> meaning Na□ gets reabsorbed more than water
Acinar cells -> Secrete isotonic fluid (Na□ high)
↓
Ductal cells -> Reabsorb Na□ & Cl□
↓
Final saliva -> Hypotonic (Na□ ↓↓↓, K□ ↑)
Ducts are impermeable to water
-> Na□ leaves, water cannot follow -> dilution happens

Site	Na□ Concentration	Key Feature
Saliva	Lowest	Hypotonic
Stomach	Moderate	HCl secretion dominates
Duodenum	Higher	Mixing with pancreatic juice
Ileum	Near isotonic	Absorption ongoing



QUESTION

A 54-year-old man with epigastric pain and positive occult blood test undergoes endoscopy, revealing hyperemic nodular mucosa. Biopsy shows submucosal glands producing alkaline mucus. Which part of the GI tract was biopsied?

- A **First part of the duodenum** — CORRECT
- B Mid-jejunum
- C Antrum of the stomach
- D Terminal ileum

RIGHT ANSWER

OK

A. First part of the duodenum

WHY THIS IS RIGHT

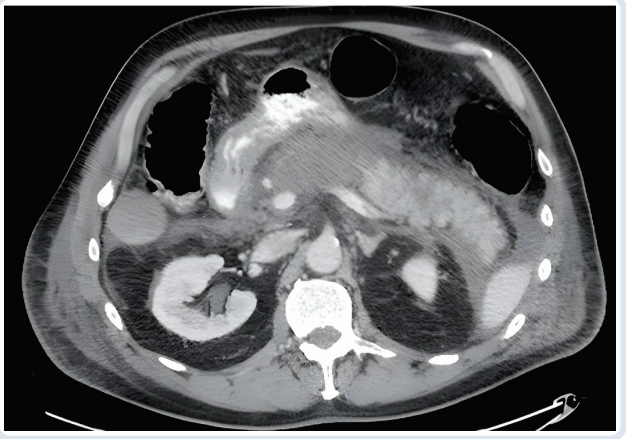
Brunner glands are submucosal glands located in the proximal (first part) of the duodenum that secrete alkaline mucus.

The alkaline mucus produced by Brunner glands neutralizes acidic chyme entering the duodenum from the stomach. This protects the duodenal mucosa and creates an optimal pH for pancreatic enzymes to function.

Key histologic feature:
Presence of mucus-secreting submucosal glands is characteristic of the first part of the duodenum and helps distinguish it from other segments of the small intestine.

QUESTION

A 38-year-old man comes to the emergency department with severe abdominal pain and vomiting. The patient is admitted to the hospital and treated with intravenous fluids and pain medication, but his condition fails to improve. An abdominal CT scan is shown below. The inappropriate activation of which of the following most likely initiated this patient's condition?



A Lipase

B Proelastase

C Prophospholipase

D **Trypsinogen** CORRECT

RIGHT ANSWER

OK **D. Trypsinogen**

WHY THIS IS RIGHT

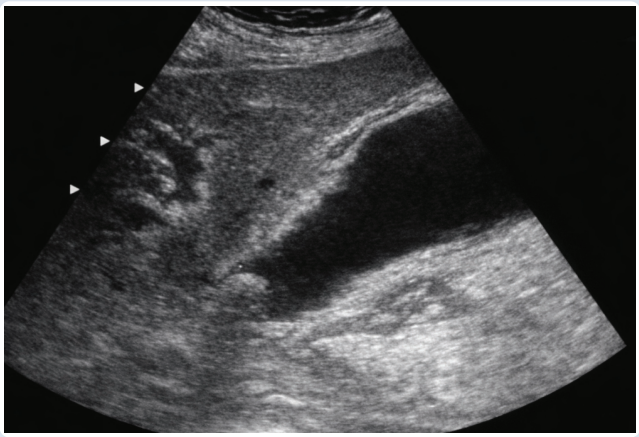
Acute pancreatitis is initiated by premature activation of pancreatic trypsinogen to trypsin within pancreatic acinar cells.

Pathophysiology:
Normally, pancreatic enzymes are secreted as inactive zymogens (eg, trypsinogen, proelastase, prophospholipase) and become activated only in the duodenum by enterokinase. In acute pancreatitis, trypsinogen is inappropriately activated inside the pancreas, forming trypsin.

Activated trypsin then triggers a cascade that activates other pancreatic enzymes (lipase, elastase, phospholipase), leading to:
• Autodigestion of pancreatic tissue
• Inflammation and edema
• Fat necrosis and vascular damage

QUESTION

A 64-year-old man comes to the OPD due to acute-onset right upper quadrant abdominal pain, nausea, and vomiting. The patient had an extensive small bowel resection due to bowel ischemia a year ago and has been receiving total parenteral nutrition since then. Abdominal ultrasonography is shown below. Which of the following is most likely factor responsible?



A **Decreased cholecystokinin release due to lack of enteral stimulation** CORRECT

B Decreased cholesterol conversion to bile acids due to liver dysfunction

C High cholesterol content of the nutritional fluids

D Inadequate supplementation of essential fatty acids

RIGHT ANSWER

OK **A. Decreased cholecystokinin release due to lack of enteral stimulation**

WHY THIS IS RIGHT

Patients receiving prolonged total parenteral nutrition (TPN) are at risk of developing gallbladder stasis and gallstones due to reduced enteral stimulation.
• Normally, fat and amino acids in the duodenum stimulate I cells to release cholecystokinin (CCK).
• CCK causes gallbladder contraction and bile release into the intestine.
• In patients on TPN, nutrients bypass the gastrointestinal tract, so CCK secretion decreases.

Consequences:
• Reduced gallbladder contraction
• Bile stasis within the gallbladder
• Formation of biliary sludge and gallstones
• Increased risk of acute cholecystitis

QUESTION

During a study on gastrointestinal hormones, a volunteer is administered the hormone secreted by S cells. Which of the following changes most likely represent the effect of this hormone on gastric and duodenal secretions?

A Gastric H+ increases, Duodenal HCO3 increased, Duodenal Cl- no change

B Gastric H+ decreases, Duodenal HCO3 decreased, Duodenal Cl- no change

C **Gastric H+ decreases, Duodenal HCO3 increased, Duodenal Cl- decreases** CORRECT

D Gastric H+ decreases, Duodenal HCO3 increased, Duodenal Cl- increases

RIGHT ANSWER

OK **C. Gastric H+ decreases, Duodenal HCO3 increased, Duodenal Cl- decreases**

WHY THIS IS RIGHT

S cells of the duodenum secrete secretin in response to acidic chyme entering the duodenum.

Major actions of secretin:
• ↓ Gastric acid (H□) secretion -> inhibits parietal cells and gastrin release
• ↑ Pancreatic and duodenal bicarbonate (HCO□□) secretion -> neutralizes gastric acid in the duodenum
• ↓ Chloride (Cl□) secretion in pancreatic/duodenal fluid because Cl□ is exchanged for HCO□□ via the Cl□/HCO□□ exchanger

Physiologic role:
• Protects the duodenal mucosa from acid
• Creates an optimal alkaline environment for pancreatic enzymes

QUESTION

Which of the following is correct treatment of peptic ulcer associated with H. pylori?

A Omeprazole only

B Omeprazole, Ciprofloxacin plus amoxicillin

C **Omeprazole, Clarithromycin and Metronidazole** CORRECT

D Metronidazole, Omeprazole and Sucralfate

RIGHT ANSWER

OK

C. Omeprazole, Clarithromycin and Metronidazole

WHY THIS IS RIGHT

H. pylori peptic ulcer requires triple therapy with a proton pump inhibitor plus two antibiotics.
• Colonizes gastric mucosa
• Produces urease -> ammonia -> mucosal damage
• Causes:
• Chronic gastritis
• Peptic ulcer

Standard Triple Therapy
• PPI (e.g., Omeprazole)
• Clarithromycin
• Amoxicillin *(or Metronidazole if penicillin allergy)*

Quadruple therapy	PPI + Bismuth + Tetracycline + Metronidazole	Resistant cases

Important Tests:
• Urea breath test
• Stool antigen test

Complications of H. pylori:
• Gastric carcinoma
• MALT lymphoma

QUESTION

An antiemetic was given to a chemotherapy patient, who then developed symptoms such as acute dystonia, bradykinesia, and tremors. Which of the following medications would have resulted in these symptoms?

A Ondansetron

B **Metoclopramide** CORRECT

C Meclizine

D Scopolamine

RIGHT ANSWER

OK

B. Metoclopramide

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which of the following drugs used in the treatment of irritable bowel syndrome (IBS) has a direct spasmolytic action on gastrointestinal (GI) smooth muscle?

A Dicyclomine

B Scopolamine

C **Mebeverine** CORRECT

D Racecadotril

RIGHT ANSWER

OK

C. Mebeverine

WHY THIS IS RIGHT

Mebeverine:
• A direct smooth muscle relaxant
• Works independently of autonomic receptors

Mechanism of Action
• Blocks sodium channels in smooth muscle
• Reduces calcium influx indirectly
• -> Decreases contractility
• -> Relieves intestinal spasm

Type of Drug	Example	Mechanism
Direct spasmolytic	Mebeverine	Acts directly on muscle
Anticholinergic	Dicyclomine, Scopolamine	Blocks M receptors
Antisecretory	Racecadotril	↓ intestinal secretion

Drug	Mechanism	IBS Role	Side Effects
Mebeverine	Direct muscle relaxant	Best for pain/spasm	Minimal
Dicyclomine	Anticholinergic	Useful	Anticholinergic effects
Scopolamine	Anticholinergic	Limited	CNS + anticholinergic
Racecadotril	↓ secretion	Diarrhea	Minimal

QUESTION

What is the rationale for the use of atropine in combination with diphenoxylate?

A Atropine decreases the adverse effect of diphenoxylate

B **To prevent abuse of diphenoxylate** CORRECT

C To enhance the antidiarrheal effect

D Atropine produces direct spasmolytic action

OK RIGHT ANSWER

B. To prevent abuse of diphenoxylate

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

Aprepitant is a drug used in the treatment of some cases of chemotherapy-induced nausea and vomiting. What is the mechanism of action of this drug?

A **NK1 antagonist** — CORRECT

B NK3 antagonist

C NK1 agonist

D NK2 agonist

RIGHT ANSWER

OK

A. NK1 antagonist

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

A 45-year-old man with a known history of gastroesophageal reflux disease (GERD) presents with persistent regurgitation and bloating despite several weeks of proton pump inhibitor therapy. To improve his symptoms, the physician plans to prescribe a medication that enhances lower esophageal sphincter tone and accelerates gastric emptying. Which of the following agents would be the most appropriate choice?

A Vonoprazan

B Omeprazole

C **Metoclopramide** CORRECT

D Sodium alginate

OK

RIGHT ANSWER

C. Metoclopramide

WHY THIS IS RIGHT

Metoclopramide acts like a traffic controller for the gut, clearing congestion and tightening the gate (LES).

Mechanism:
• D□ receptor antagonist
• ↑ Acetylcholine release in gut

Effects:
• ↑ Lower esophageal sphincter (LES) tone
• ↑ Gastric emptying
• ↓ Reflux

Drug	Class	Mechanism	Use
Omeprazole	PPI	↓ H□ secretion	First-line GERD
Vonoprazan	P-CAB	Strong acid suppression	Resistant GERD
Metoclopramide	Prokinetic	↑ LES tone + motility	Refractory GERD
Sodium alginate	Barrier	Floating raft	Mild symptoms

QUESTION

Which of the following statements about Lafutidine is incorrect?

1. Increases gastric mucosal blood flow
2. Acts as an H□ receptor antagonist
3. Decreases nitric oxide production
4. Increases somatostatin secretion+

A 1 and 2

B 2 and 4

C **3 only** CORRECT

D 1, 2 and 4

RIGHT ANSWER

OK **C. 3 only**

WHY THIS IS RIGHT

Lafutidine is a second-generation H□ receptor antagonist used for:
• Peptic ulcer disease
• GERD
• Gastritis
• Stress ulcer prophylaxis

What makes it special is that it has both acid-suppressing and mucosal-protective actions.
Correct answer is C because lafutidine is an H□ receptor antagonist that enhances mucosal defense by increasing gastric mucosal blood flow and stimulating protective mediators. It is known to promote nitric oxide-mediated mucosal protection rather than decrease nitric oxide production. Protective effects include:

Effect	Mechanism
↑ Gastric mucosal blood flow	via nitric oxide
↑ Prostaglandin synthesis	mucosal protection
↑ Somatostatin secretion	inhibits gastrin
↑ Mucus secretion	barrier protection

Thus it protects gastric mucosa beyond acid suppression.

QUESTION

Why is atropine mixed with diphenoxylate in combination medications?

A Atropine decreases the adverse effect of diphenoxylate

B **To prevent abuse of diphenoxylate** CORRECT

C To enhance the antidiarrheal effect

D Atropine produces direct spasmolytic action

OK RIGHT ANSWER

B. To prevent abuse of diphenoxylate

WHY THIS IS RIGHT

Diphenoxylate + Atropine (e.g., Lomotil)
• Diphenoxylate -> opioid derivative (antidiarrheal)
• Atropine -> anticholinergic (very low dose in combo)

Diphenoxylate is an opioid that can cause euphoria in high doses. A small amount of atropine is added to produce unpleasant anticholinergic effects if overdosed. This discourages intentional misuse while preserving antidiarrheal action. The combination is designed as an abuse-deterrent formulation.
Diphenoxylate taken in high dose
↓
Atropine also increases
↓
Anticholinergic toxicity
↓
Unpleasant symptoms
↓
Discourages abuse

Component	Role
Diphenoxylate	Antidiarrheal (opioid)
Atropine	Abuse deterrent

QUESTION

Which of the following statements about Palonosetron are incorrect:

A It is a second generation 5HT3 receptor antagonist

B It belongs to the class of most effective drugs for chemotherapy induced nausea and vomiting

C It is the longest acting drug amongst its class

D **High risk of QT prolongation as adverse reaction** CORRECT

RIGHT ANSWER

OK

D. High risk of QT prolongation as adverse reaction

WHY THIS IS RIGHT

Palonosetron is a second-generation 5-HT□ receptor antagonist used primarily for prevention of chemotherapy-induced nausea and vomiting (CINV).
• Higher receptor affinity and long half-life (\~40 hours) compared to first-generation agents (ondansetron, granisetron).
• Most effective for delayed CINV.
• Longest acting drug in the 5-HT□ antagonist class.

Safety profile:
• Unlike earlier 5-HT□ antagonists, palonosetron has minimal risk of QT interval prolongation.

QUESTION

A patient with a history of chronic liver disease presents with abdominal distension, jaundice, and pruritis. Ascitic fluid analysis revealed a neutrophil count > 650 per cubic mm. What is the most likely diagnosis?

A **Spontaneous bacterial peritonitis** CORRECT

B Malignant ascites

C Tubercular ascites

D Intestinal obstruction

OK RIGHT ANSWER

A. Spontaneous bacterial peritonitis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

A mother brought her 2-year-old child to the OPD with complaints of lack of weight gain, abdominal pain on eating, and poor eating habits. The child was advised to follow a gluten-free diet, after which there was an improvement in the general condition and weight gain. What is the probable diagnosis?

- A **Celiac disease** — CORRECT
- B Tropical sprue
- C Abetalipoproteinemia
- D Whipple disease

RIGHT ANSWER

OK

A. Celiac disease

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

A patient with diarrhoea and skin lesions was advised to change his diet, which resulted in the improvement of his symptoms. Which of the following is the likely antibody present in this patient?

A **Anti TTG antibody** CORRECT

B Anti Saccharomyces cerevisiae antibody

C p-ANCA

D Anti-nuclear antibody

RIGHT ANSWER

OK

A. Anti TTG antibody

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

A young boy with abdominal pain, diarrhea, and skin lesions was advised to change his diet, which resulted in the improvement of his symptoms. Which of the following is the likely diagnosis in this patient?

A Tropical sprue

B **Celiac sprue** CORRECT

C Whipple's disease

D Giardiasis

OK RIGHT ANSWER

B. Celiac sprue

WHY THIS IS RIGHT

The source quiz contains the placeholder "$1d" here and does not include a written explanation for this item.

QUESTION

Which of the following is not associated with celiac disease?

A Modified Marsh classification

B IgA transglutaminase positivity

C IgG antigliadin positivity

D **HLA DQ6 and HLA DQ11** CORRECT

RIGHT ANSWER

OK

D. HLA DQ6 and HLA DQ11

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1e” here and does not include a written explanation for this item.

QUESTION

A patient presents with abdominal pain, acute diarrhea, and is diagnosed with celiac disease. Which of the following statements is false regarding this condition?

A **Associated with HLA DQ6/DQ11** — CORRECT

B IgA Tissue Transglutaminase antibodies are present

C Marsh Criteria used for diagnosis

D IgG deamidated Gliadin antibodies are present

OK RIGHT ANSWER

A. Associated with HLA DQ6/DQ11

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1f” here and does not include a written explanation for this item.

QUESTION

An 18-month-old child was brought to OPD with a 3-day record of watery diarrhea and vomiting, displaying an altered sensorium. What possible conditions could be considered as the differential diagnoses in this case?
1. Severe dehydration
2. HUS
3. Cerebral Venous Thrombosis
4. Hyponatremia

A 1, 2, 3, 4

B 1, 2, 3

C **1, 3, 4** CORRECT

D 2, 3, 4

OK RIGHT ANSWER

C. 1, 3, 4

WHY THIS IS RIGHT

In children with acute gastroenteritis presenting with altered sensorium, common causes include severe dehydration, electrolyte disturbances (especially hyponatremia), and cerebral venous thrombosis due to dehydration-induced hypercoagulability.

Explanation:
• Severe dehydration can cause hypovolemia leading to decreased cerebral perfusion and altered mental status.
• Hyponatremia may occur due to excess free water intake or inappropriate fluid replacement, leading to cerebral edema and neurological symptoms.
• Cerebral venous thrombosis can develop in dehydrated children due to hemoconcentration and hypercoagulability, resulting in neurological manifestations such as altered consciousness or seizures.

Why not HUS:
Hemolytic uremic syndrome usually follows bloody diarrhea caused by Shiga toxin-producing bacteria and is characterized by hemolytic anemia, thrombocytopenia, and acute kidney injury, which are not typical in this presentation of watery diarrhea.

QUESTION

44-year-old man comes to the emergency department for exertional dyspnea, abdominal distention, and abdominal discomfort. He has a history of alcohol abuse, peptic ulcer disease, and acute pancreatitis. Physical examination shows a distended abdomen with shifting dullness and 1+ ankle edema. A diagnostic paracentesis is performed and shows the following:
Albumin-2.5 g/dL
Neutrophils-200/uL
Blood studies show the following:
Albumin-3.8 g/dL
Glucose-120 mg/dL
Aspartate aminotransferase-50 units/L
Alanine aminotransferase (ALT)-40 units/L
Which of the following is the most likely cause of this patient's ascites?

A Decreased liver synthetic activity

B **Increased hepatic capillary hydrostatic pressure** CORRECT

C GI malignancy

D Pancreatitis

OK RIGHT ANSWER

B. Increased hepatic capillary hydrostatic pressure

WHY THIS IS RIGHT

Ascites can be evaluated using the serum-ascites albumin gradient (SAAG), which helps determine whether portal hypertension is present.

SAAG = Serum albumin - Ascitic fluid albumin = 3.8 - 2.5 = 1.3 g/dL
• SAAG ≥ 1.1 g/dL indicates portal hypertension.
• SAAG < 1.1 g/dL suggests non-portal hypertensive causes (eg, malignancy, pancreatitis, tuberculosis).

In portal hypertension, ascites forms due to increased hydrostatic pressure in hepatic sinusoids, causing fluid to leak into the peritoneal cavity.
• Clinical context:
• History of alcohol abuse suggests alcoholic liver disease leading to portal hypertension.
• Neutrophil count <250 cells/μL excludes spontaneous bacterial peritonitis.

*

QUESTION

45-year-old man comes to the clinic for evaluation of chronic diarrhea. He has lost almost 7 kg over the past year. He has no blood in the stool. A 24-hour stool collection shows fecal fat content of 10 g/day (normal <6 g/day). Stool microscopy shows no pathogens and no leukocytes. Serum electrolytes and renal function are within normal limits. The patient is given 25 g oral D-xylose solution, and his urinary excretion of D-xylose at 5 hours is 1.2 g (normal 4.5-7.5 g). After 4 weeks of treatment with rifaximin, the D-xylose test is repeated, and the urinary excretion at 5 hours is 1.3 g. Based on these findings, which of the following is the most likely diagnosis in this patient?

A BOGS

B **Celiac disease** CORRECT

C Lactose intolerance

D Pancreatic insufficiency

RIGHT ANSWER

OK **B. Celiac disease**

WHY THIS IS RIGHT

The D-xylose test helps differentiate intestinal mucosal disease from pancreatic malabsorption.
• D-xylose is absorbed directly by the small intestinal mucosa and does not require pancreatic enzymes for absorption.
• Therefore:
• Low urinary D-xylose -> mucosal malabsorption
• Normal D-xylose -> pancreatic insufficiency

In this patient:
• Steatorrhea (fecal fat 10 g/day) -> fat malabsorption
• Low urinary D-xylose (1.2 g) -> impaired small intestinal absorption
• No improvement after rifaximin -> rules out bacterial overgrowth syndrome

Most likely cause: Celiac disease
• Immune-mediated damage to small intestinal villi
• Leads to malabsorption, weight loss, and steatorrhea

QUESTION

50 yr gentleman presented with progressive abdominal distension with shifting dullness on examination. Ascitic fluid analysis given below. What is the likely diagnosis?
S. Albumin - 3g/dl
S. Protein - 7g/dl
Ascitic Albumin - 1g/dl
Ascitic protein - 1.5g/dl
Cells - 125, 90%Lymphocytes

A Congestive heart failure

B Budd Chiari syndrome

C **Cirrhosis** CORRECT

D Tuberculosis

RIGHT ANSWER

OK **C. Cirrhosis**

WHY THIS IS RIGHT

Ascitic fluid analysis helps determine the cause of ascites using the Serum-Ascites Albumin Gradient (SAAG).

SAAG = Serum albumin - Ascitic fluid albumin = 3 - 1 = 2 g/dL
• SAAG ≥1.1 g/dL -> Portal hypertension
• Causes: Cirrhosis, Budd-Chiari syndrome, congestive heart failure
• SAAG <1.1 g/dL -> Non-portal hypertension
• Causes: Tuberculosis, malignancy, pancreatitis

Additional clue:
• Ascitic protein is low (1.5 g/dL) -> typical of cirrhotic ascites
• CHF and Budd-Chiari usually have high ascitic protein (>2.5 g/dL).

*

QUESTION

A 50-year-old man comes to the physician because of an 8-month history of intermittent watery diarrhea and abdominal pain. He has had a 12-kg weight loss during this period. He has also had episodic pain of the ankle, wrist, and knee joints during the past 5 years. An endoscopy with small bowel biopsy is performed. Histopathologic examination of a tissue specimen shows foamy macrophages in the lamina propria with periodic acid-Schiff (PAS)-positive inclusions. Further evaluation is most likely to show which of the following?

A Multinucleated trophozoites

B Anti-tissue translutaminase antibodies

C **Intracellular gram positive bacteria** CORRECT

D Anti-saccharomyces cerevisiae antibodies

OK RIGHT ANSWER

C. Intracellular gram positive bacteria

WHY THIS IS RIGHT

Whipple disease is a systemic infection caused by Tropheryma whipplei, a gram-positive intracellular bacterium that infects macrophages.
• Small bowel biopsy showing foamy macrophages in the lamina propria with PAS-positive inclusions

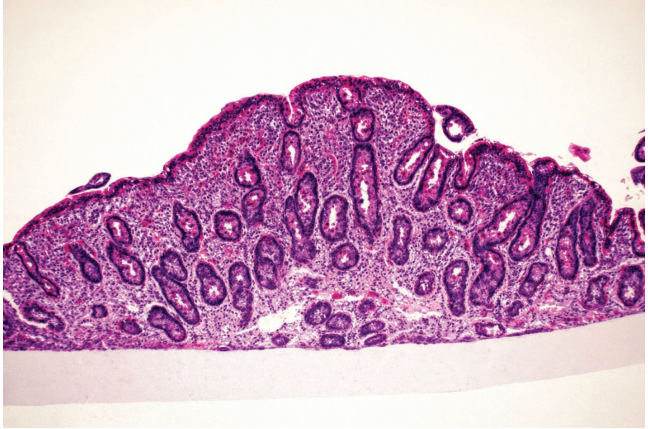
Clinical manifestations:
• Chronic watery diarrhea
• Weight loss and malabsorption
• Migratory arthralgia (often precedes GI symptoms by years)
• Fever, lymphadenopathy, hyperpigmentation, neurologic symptoms

Pathogenesis:
• Macrophages filled with intracellular gram-positive bacteria accumulate in the intestinal mucosa, impairing nutrient absorption and lymphatic transport.

Treatment:
• Prolonged antibiotic therapy (e.g., ceftriaxone followed by TMP-SMX).

QUESTION

A 19-month-old girl is brought to the OPD due to a 2-month history of diarrhea. Her parents report that she has 3- 5 loose, non-bloody bowel movements daily with occasional episodes of vomiting. She was breastfed exclusively until age 9 months and has since had a well-varied diet including whole milk, fruits, vegetables, bread, and meats. However, the girl has been less interested in food over the past several weeks After laboratory evaluation, duodenal biopsy findings are shown in the exhibit. Which of the following would most likely improve this patient's symptoms?



A Antibiotic therapy

B Enzyme supplementation

C Modified dairy diet

D **Modified grain diet** CORRECT

OK RIGHT ANSWER

D. Modified grain diet

WHY THIS IS RIGHT

The child’s chronic diarrhea after introduction of grains with duodenal biopsy showing villous atrophy, crypt hyperplasia, and lymphocytic infiltration is characteristic of celiac disease (gluten-sensitive enteropathy).

Pathophysiology:
• Immune-mediated reaction to gluten (gliadin) found in wheat, barley, and rye
• Leads to damage of small intestinal villi, resulting in malabsorption and chronic diarrhea

Management:
• Gluten-free diet -> eliminate wheat, barley, and rye
• Replace with rice, corn, millet, or other gluten-free grains

QUESTION

A 54-year-old man with alcoholic cirrhosis and ascites presents with fever, abdominal pain, and altered sensorium. Ascitic fluid analysis shows:
Total leukocyte count: 800 cells/mm³
Neutrophils: 320 cells/mm³
Protein: low
Culture: pending
What is the most appropriate diagnosis?

A Secondary bacterial peritonitis

B Tubercular peritonitis

C **Spontaneous bacterial peritonitis** CORRECT

D Pancreatic ascites

OK

RIGHT ANSWER

C. Spontaneous bacterial peritonitis

WHY THIS IS RIGHT

Spontaneous bacterial peritonitis (SBP) is a bacterial infection of ascitic fluid without an identifiable intra-abdominal source, most commonly seen in patients with cirrhosis and ascites.

Diagnostic criterion: Ascitic fluid polymorphonuclear (PMN) count ≥250 cells/mm³

Typical findings:
• Low ascitic fluid protein
• Fever, abdominal pain, altered mental status
• Culture may be positive (commonly E. coli, Klebsiella, Streptococcus) but treatment should start even if culture results are pending.

Management:
• Empiric third-generation cephalosporin (e.g., cefotaxime)
• Albumin infusion to reduce risk of hepatorenal syndrome.

QUESTION

A newborn male develops profuse, intractable watery diarrhea within the first few days of life, leading to severe dehydration and metabolic acidosis. The diarrhea does not improve with bowel rest or fasting.
Small intestinal biopsy shows:
Villous atrophy
Periodic acid-Schiff (PAS)-positive material within the apical cytoplasm of enterocytes
On electron microscopy, intracytoplasmic microvillous inclusions are seen.
Which of the following gene mutations is most likely responsible for this condition?

A CFTR

B SMAD4

C **MYO5B** CORRECT

D MTTP

OK RIGHT ANSWER

C. MYO5B

WHY THIS IS RIGHT

Microvillus inclusion disease (MVID) is a rare congenital enteropathy that presents in neonates with severe, intractable secretory diarrhea that persists despite fasting.
• Mutation in the MYO5B gene, which encodes myosin Vb, a protein involved in intracellular trafficking and maintenance of enterocyte polarity.

Pathologic findings:
• Villous atrophy
• PAS-positive material in the apical cytoplasm of enterocytes
• Intracytoplasmic microvillus inclusions on electron microscopy

Clinical consequence:
• Severe malabsorption and dehydration
• Often requires long-term parenteral nutrition or intestinal transplantation.

QUESTION

A 52-year-old man presents with chronic diarrhea, weight loss, migratory arthralgia, and intermittent fever. He also has hyperpigmentation and generalized lymphadenopathy. Duodenal biopsy is performed. Which of the following statements regarding Whipple disease are CORRECT?
A. It is caused by Tropheryma whipplei
B. Duodenal biopsy shows PAS-positive macrophages in the submucosa
C. Migratory polyarthritis often precedes gastrointestinal manifestations
D. The organism is acid-fast positive on staining
E. Long-term antibiotic therapy is required for definitive treatment
F. Causes disturbance in lymphatic transport of absorbed lipids

A A,B,C,D

B A,B,C,D,E,F

C A,B,D,E

D **A,C,E,F** CORRECT

RIGHT ANSWER

OK **D. A,C,E,F**

WHY THIS IS RIGHT

Whipple disease is a rare systemic infection caused by Tropheryma whipplei that primarily affects the small intestine and can involve multiple organ systems.
• Chronic diarrhea and weight loss
• Migratory polyarthritis (often precedes GI symptoms)
• Fever, lymphadenopathy, hyperpigmentation
• Malabsorption

Pathophysiology:
• Infiltration of the intestinal mucosa by infected macrophages
• Leads to impaired lymphatic transport of absorbed lipids, causing malabsorption.

Diagnosis:
• Duodenal biopsy (classically PAS-positive macrophages, though not included in the correct set here)
• PCR for Tropheryma whipplei

Treatment:
• Requires prolonged antibiotic therapy (often ≥1 year) to prevent relapse and CNS involvement.

QUESTION

A 26-year-old man presents with acute diarrhea associated with fever and abdominal pain. The clinician wants to rapidly differentiate inflammatory diarrhea from non-inflammatory diarrhea at the bedside using a stool-based test that detects neutrophil-derived inflammatory markers and is more sensitive than direct microscopy. Which of the following tests is most appropriate for this purpose?

A Stool culture

B Fecal leukocyte examination

C **Fecal lactoferrin agglutination assay** CORRECT

D Stool osmotic gap

OK

RIGHT ANSWER

C. Fecal lactoferrin agglutination assay

WHY THIS IS RIGHT

-Fecal lactoferrin is a neutrophil-derived protein released during intestinal inflammation.
-Detection of lactoferrin in stool helps differentiate inflammatory diarrhea from non-inflammatory diarrhea.

Clinical significance:
• Positive fecal lactoferrin -> inflammatory diarrhea, seen in infections such as Shigella, Salmonella, Campylobacter, and in inflammatory bowel disease.
• It is more sensitive than stool microscopy for fecal leukocytes and provides a rapid bedside test.

Comparison:
• Stool culture: identifies specific pathogens but is slower.
• Fecal leukocyte exam: less sensitive and operator dependent.
• Stool osmotic gap: used for secretory vs osmotic diarrhea, not inflammation.

QUESTION

The presence of buccal mucosal melanosis and hamartomatous polyp is a feature of which of the following conditions?

A Lynch syndrome

B Familial adenomatous polyposis

C **Peutz-Jeghers syndrome** CORRECT

D Juvenile polyposis syndrome

OK RIGHT ANSWER

C. Peutz-Jeghers syndrome

WHY THIS IS RIGHT

Peutz-Jeghers syndrome is characterized by hamartomatous GI polyps and mucocutaneous melanotic pigmentation, especially on the buccal mucosa and lips. It results from STK11 mutation and carries increased cancer risk. The pigmentation is a classic clinical clue. Other polyposis syndromes lack this mucocutaneous feature.

Feature	Description
Pigmentation	Lips, buccal mucosa, fingers □
Polyps	Hamartomatous
Location	Small intestine (jejunum common)
Complication	Intussusception
Cancer risk	↑ GI + pancreatic + others

Feature	Peutz-Jeghers	FAP	Lynch	Juvenile Polyposis
Gene	STK11	APC	MMR genes	SMAD4/BMPR1A
Polyps	Hamartomatous	Adenomatous	Few	Hamartomatous
Pigmentation	Yes	No	No	No
Cancer risk	↑ multiple	100% CRC	↑ CRC	↑ CRC

QUESTION

Match the column:

Column A	Column B
A. Peetz Jegher polyp	1. APC
B. Familial adenomatous polyp	2. TSC 2
C. Juvenile polyposis	3. STK11
D. Tuberous sclerosis	4. SMAD4

A **A-3, B-1, C-4, D-2** CORRECT

B A-4, B-2, C-1, D-3

C A-3, B-1, C-2, D-4

D A-4, B-1, C-3, D-2

OK

RIGHT ANSWER

A. A-3, B-1, C-4, D-2

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which of the following statements regarding Peutz-Jeghers syndrome are true
1. Heterogeneous loss of function of the STK11 gene
2. Presents as multiple hamartomatous polyps in the GIT
3. Histology shows arborising smooth muscle bundles
4. It is an autosomal recessive condition
5. Extra-intestinal manifestations of Peutz-Jeghers syndrome include hypertrophy of the retinal pigment epithelium

A **1, 2, and 3** CORRECT

B 1, 3, and 5

C 2, 4, and 5

D 1, 2, 3, and 4

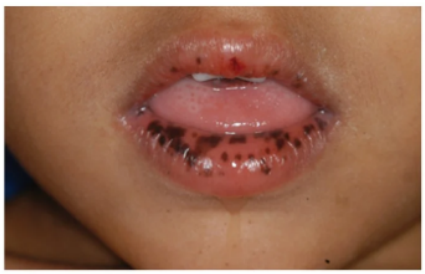
OK RIGHT ANSWER

A. 1, 2, and 3

WHY THIS IS RIGHT

Peutz-Jeghers syndrome is due to loss-of-function mutation of the STK11 tumor suppressor gene and is inherited in an autosomal dominant pattern. It presents with multiple hamartomatous GI polyps showing classic arborizing smooth muscle bundles on histology. Retinal pigment hypertrophy is seen in familial adenomatous polyposis, not Peutz-Jeghers.

|
Feature	Peutz-Jeghers	FAP
Gene	STK11	APC
Inheritance	AD	AD
Polyp type	Hamartomatous	Adenomatous
Histology	Arborizing smooth muscle	Dysplastic epithelium
Cancer risk	Moderate-high (extraintestinal)	Very high (colon)
Extra features	Mucocutaneous pigmentation	CHRPE (retinal lesions)



QUESTION

52-year-old man comes to the OPD for a painless mass in his right groin. He noticed the mass several weeks ago, and it has slowly enlarged. On examination, several enlarged, hard lymph nodes are palpated in the right inguinal area inferior to the inguinal ligament. An excisional biopsy is performed, and histopathology shows malignant cells. The malignant cells found in this patient most likely originated from which of the following sites?

A Dome of the bladder

B Lateral lobe of the prostate

C **Orifice of the anal canal** CORRECT

D Upper pole of the testes

OK RIGHT ANSWER

C. Orifice of the anal canal

WHY THIS IS RIGHT

Lymphatic drainage determines the pattern of metastasis to regional lymph nodes. Structures located below the pectinate line of the anal canal drain to the superficial inguinal lymph nodes. Key lymphatic drainage patterns:
• Anal canal below the pectinate line -> superficial inguinal lymph nodes
• Anal canal above the pectinate line -> internal iliac lymph nodes
• Testes -> para-aortic (lumbar) lymph nodes due to embryologic origin
• Prostate -> internal iliac and sacral lymph nodes
• Bladder -> external and internal iliac lymph nodes

Clinical significance:
A malignant mass in the superficial inguinal lymph nodes is most commonly associated with cancers of structures draining to these nodes, including the lower anal canal, perianal skin, external genitalia, and lower limb.

QUESTION

A 34-year-old woman comes to the physician with abdominal pain and melena. She also complains of progressive fatigue and a 5 kg (11 lb) weight loss over the last 2 months. She has a strong family history of colon, endometrial, and ovarian cancer. Colonoscopy shows a protuberant, friable mass in the ascending colon, and biopsy is diagnostic for colon adenocarcinoma. Genetic analysis confirms a mutation consistent with Lynch syndrome. Which of the following is most likely responsible for the development of colon cancer in this patient?

A **Nucleotide mismatches that escape repair** CORRECT

B Covalent bonds between adjacent pyrimidines

C Insertion of abnormal bases into DNA

D Double-strand breaks in DNA

OK RIGHT ANSWER

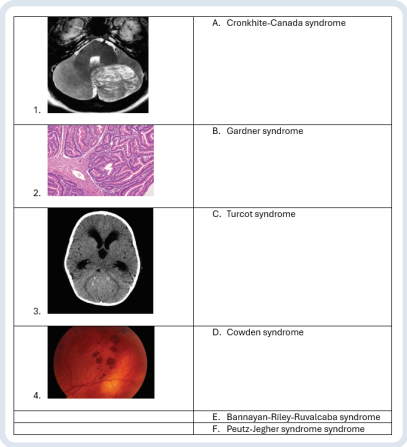
A. Nucleotide mismatches that escape repair

WHY THIS IS RIGHT

Lynch syndrome (hereditary nonpolyposis colorectal cancer) is caused by mutations in DNA mismatch repair (MMR) genes such as MLH1, MSH2, MSH6, and PMS2. These genes normally correct base-base mismatches and insertion-deletion errors that occur during DNA replication.
• Pathogenesis:
-Defective mismatch repair leads to accumulation of unrepaired nucleotide mismatches, particularly in repetitive DNA sequences called microsatellites.
-This results in microsatellite instability (MSI), which promotes mutations in genes controlling cell growth and apoptosis, leading to early-onset colorectal cancer and other malignancies.

QUESTION

Match the following images associated with GI polyps with the likely diagnosis:



A 1-A, 2-F, 3-B, 4-C

B **1-D, 2-F, 3-C, 4-B** CORRECT

C 1-D, 2-F, 3-C, 4-E

D 1-E, 2-B, 3-C, 4-A

OK RIGHT ANSWER

B. 1-D, 2-F, 3-C, 4-B

WHY THIS IS RIGHT

Syndrome	Key Feature	Gene
Cowden syndrome	Multiple hamartomas, cerebellar lesions (Lhermitte-Duclos disease)	PTEN mutation
Peutz-Jeghers syndrome	Hamartomatous polyps with arborizing smooth muscle, mucocutaneous pigmentation	STK11 (LKB1)
Turcot syndrome	Colon polyposis + brain tumors (glioblastoma or medulloblastoma)	APC or mismatch repair genes
Gardner syndrome	Variant of FAP with osteomas, epidermoid cysts, fibromas, retinal lesions	APC mutation

- 1 -> Cowden syndrome
- 2 -> Peutz-Jeghers syndrome
- 3 -> Turcot syndrome
- 4 -> Gardner syndrome

QUESTION

A 46-year-old man presents with intermittent rectal bleeding. Colonoscopy reveals 30-40 adenomatous polyps, predominantly in the proximal colon. Colorectal carcinoma is detected at 52 years of age. His father underwent colectomy for colon cancer at the age of 48. Upper GI endoscopy shows a few duodenal adenomas. Which of the following is the most likely diagnosis?

A MUTYH-associated polyposis

B Classic familial adenomatous polyposis

C **Attenuated familial adenomatous polyposis** CORRECT

D Lynch syndrome

OK RIGHT ANSWER

C. Attenuated familial adenomatous polyposis

WHY THIS IS RIGHT

Attenuated familial adenomatous polyposis (AFAP) is a milder variant of familial adenomatous polyposis caused by mutations in the APC gene.
• Fewer colorectal adenomas (10-100 polyps) compared to classic FAP (>1000)
• Polyps often predominantly in the proximal colon
• Later onset of colorectal cancer (usually in the 50s)
• May also have duodenal adenomas

Comparison:
• Classic FAP: hundreds to thousands of polyps, cancer in 30s-40s
• Lynch syndrome: few polyps but high risk of early colorectal cancer due to DNA mismatch repair defects

QUESTION

Which of the following are established prognostic factors in carcinoid (neuroendocrine) tumors, ALL EXCEPT:

A Mitotic rate

B Degree of histological differentiation

C Ki-67 proliferation index

D **Urinary 5-HIAA level** CORRECT

OK RIGHT ANSWER

D. Urinary 5-HIAA level

WHY THIS IS RIGHT

Prognosis of neuroendocrine (carcinoid) tumors is primarily determined by tumor proliferation and histologic features.

Established prognostic factors:
• Mitotic rate
• Degree of histological differentiation
• Ki-67 proliferation index (used for tumor grading: G1, G2, G3)

Not a prognostic factor:
• Urinary 5-HIAA (a serotonin metabolite) is mainly used to diagnose and monitor carcinoid syndrome, not to determine tumor aggressiveness or prognosis.

QUESTION

In contrast to Ulcerative Colitis, which of the following statements regarding Crohn's disease is true?

A There is no increased risk of colon cancer

B Continuous involvement rather than skip lesions

C **Perianal fistulas are frequent** CORRECT

D Seldom manifests with hematochezia

RIGHT ANSWER

OK **C. Perianal fistulas are frequent**

WHY THIS IS RIGHT

Crohn disease causes transmural inflammation, leading to fissures, abscesses, and frequent perianal fistulas. It shows skip lesions and has an increased colorectal cancer risk with long-standing colonic involvement.

Feature	Crohn	UC
Pattern	Skip lesions	Continuous
Depth	Transmural	Mucosal
Fistula	Common	Rare
Perianal disease	Common	Rare

QUESTION

Which JAK inhibitor drug is recommended for moderate to severe ulcerative colitis in a patient who is not responding well to TNF-alpha blockers?

A Ustekinumab

B Etanercept

C Vedolizumab

D **Tofacitinib** CORRECT

OK RIGHT ANSWER

D. Tofacitinib

WHY THIS IS RIGHT

Tofacitinib:
• Class: JAK inhibitor (Janus kinase inhibitor)
• Type: Oral small molecule (unlike biologics which are injectable)

Mild UC
↓
5-ASA (Mesalamine)
↓ (if not controlled)
Steroids
↓
Moderate-Severe UC
↓
Anti-TNF (Infliximab)
↓ (failure)
Tofacitinib / Vedolizumab / Ustekinumab

Drug	Class	Mechanism	Use in IBD
Infliximab	Anti-TNF	Blocks TNF-α	UC, Crohn
Etanercept	Anti-TNF	TNF receptor fusion protein	□ Not in IBD
Vedolizumab	Anti-integrin	Blocks gut lymphocyte entry	UC, Crohn
Ustekinumab	Anti IL-12/23	↓ Th1/Th17	UC, Crohn
Tofacitinib	JAK inhibitor	Blocks cytokine signaling	UC (oral)

QUESTION

The Truelove and Witt criteria are used for assessing which of the following conditions?

A Encephalitis

B **Acute severe ulcerative colitis** CORRECT

C ARDS

D Acute pancreatitis

OK RIGHT ANSWER

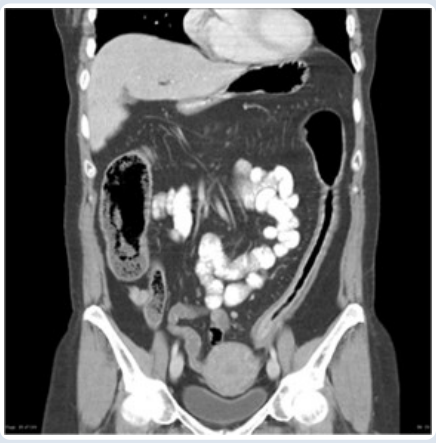
B. Acute severe ulcerative colitis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

A 26-year-old man presents with an 8-month history of chronic diarrhea. CT shows the following. Based on these findings, what is the diagnosis?



A Pseudomembranous enterocolitis

B **Ulcerative colitis** CORRECT

C Crohn's disease

D Intestinal TB

OK RIGHT ANSWER

B. Ulcerative colitis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

Onion skin fibrosis on histopathology is seen in which of the following conditions?

A Primary biliary cirrhosis

B Chronic cirrhosis

C **Primary sclerosing cholangitis** CORRECT

D Extrahepatic biliary atresia

OK RIGHT ANSWER

C. Primary sclerosing cholangitis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

A patient with ulcerative colitis is experiencing three episodes of bleeding in stools per day but is otherwise stable. What is the appropriate treatment for this patient?

A **5-Aminosalicylic acid** CORRECT

B 5-Fluorouracil

C Budesonide

D 6-Mercaptopurine

OK RIGHT ANSWER

A. 5-Aminosalicylic acid

WHY THIS IS RIGHT

Mild ulcerative colitis with limited bleeding and stable vitals is treated with 5-aminosalicylic acid (5-ASA) as first-line therapy. It acts locally to reduce mucosal inflammation and induces remission. Steroids and immunomodulators are reserved for moderate-severe or refractory disease. 5-ASA is standard initial treatment in mild UC. Mechanism of Action-Inhibits:
• Cyclooxygenase (COX)
• ↓ Prostaglandins

Reduces local inflammation in colon

Severity	Clinical features	Treatment
Mild-moderate	≤4-6 stools/day, minimal systemic signs	5-ASA □
Moderate-severe	Frequent stools + systemic symptoms	Steroids
Severe	Toxic megacolon risk	IV steroids / biologics

QUESTION

26-year-old woman is evaluated for intermittent abdominal pain occurring over the last several years. The pain is crampy without radiation and graded 6/10 in intensity. She also has fluctuating diarrhea but has not seen blood in the stool. Temperature is 37.2 C (99 F), blood pressure is 115/70 mm Hg, and pulse is 90/min. On examination, the abdomen is tender without guarding or rebound. There is a draining fistula near her coccyx. Which of the following is the most likely diagnosis in this patient?

A **Crohn disease** CORRECT

B Diverticulitis

C Intergluteal pilonidal disease

D Irritable bowel syndrome

OK RIGHT ANSWER

A. Crohn disease

WHY THIS IS RIGHT

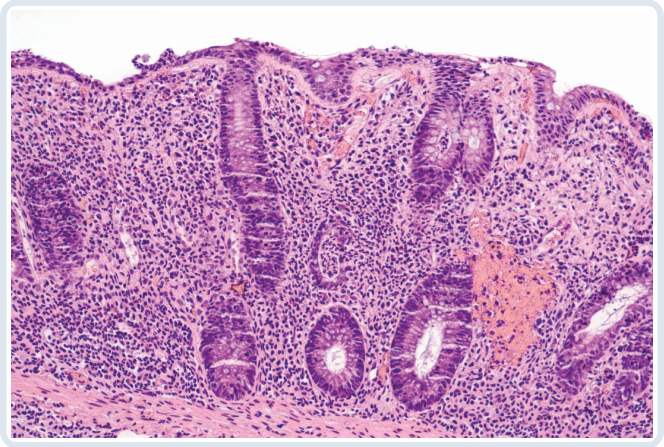
Crohn disease is a chronic inflammatory bowel disease characterized by transmural inflammation that can affect any part of the gastrointestinal tract and commonly leads to fistula formation.

Clinical features include:
• Chronic crampy abdominal pain
• Intermittent diarrhea (often without blood)
• Weight loss and fatigue
• Perianal disease such as fistulas, abscesses, or fissures
• Pathophysiology:
The transmural inflammation in Crohn disease can extend through the bowel wall, leading to complications such as fistulas, strictures, and abscesses.
Perianal fistulas are a characteristic finding and help differentiate Crohn disease from ulcerative colitis and irritable bowel syndrome.

*

QUESTION

A 45-year-old female patient presents to the OPD with intermittent rectal bleeding, tenesmus, and mucous discharge. Histological examination showed the following lesion. What is the first-line management of this condition?



A **Sulfasalazine** CORRECT

B Surgical resection

C NSAIDs

D Antibiotics

RIGHT ANSWER

OK **A. Sulfasalazine**

WHY THIS IS RIGHT

The patient's rectal bleeding, tenesmus, and mucus discharge with histology showing crypt distortion, inflammatory infiltrate, and crypt abscesses is characteristic of ulcerative colitis (UC).
• Continuous mucosal inflammation starting from the rectum
• Symptoms: bloody diarrhea, mucus discharge, tenesmus
• Histology:
• Crypt abscesses
• Mucosal inflammation
• Distorted crypt architecture

First-line treatment (mild-moderate disease): 5-amino salicylic acid (5-ASA) drugs
• Sulfasalazine
• Mesalamine
• Other options:
• Corticosteroids -> moderate-severe flares
• Immunomodulators/biologics -> refractory disease
• Surgery (colectomy) -> severe or complicated disease.

QUESTION

A 40-year-old woman develops chronic watery diarrhea after limited terminal ileal resection for Crohn disease. Stool examination shows normal fat content. Which of the following is the most appropriate treatment?

A Pancreatic enzyme supplementation

B Medium-chain triglyceride-rich diet

C **Cholestyramine** CORRECT

D Fat-restricted diet

RIGHT ANSWER

OK

C. Cholestyramine

WHY THIS IS RIGHT

After terminal ileal resection, bile acids are not adequately reabsorbed in the ileum. Excess bile acids enter the colon, where they stimulate water and electrolyte secretion, causing chronic watery diarrhea.
• Occurs after limited ileal resection
• Stool fat is normal (no significant fat malabsorption)
• Due to bile acid-induced secretory diarrhea

Treatment:
• Cholestyramine, a bile acid-binding resin, binds bile acids in the intestine and prevents their secretory effect in the colon.

Important distinction:
• Extensive ileal resection (>100 cm) -> bile acid deficiency -> fat malabsorption and steatorrhea, not bile acid diarrhea.

QUESTION

The Truelove-Witts criteria are used to define acute severe ulcerative colitis. All of the following are components of the Truelove-Witts criteria, EXCEPT:

A ≥ 6 bloody stools per day

B Hemoglobin < 10 g/dL

C Tachycardia with pulse ≥ 90 beats/min

D **Presence of jaundice and pruritus** CORRECT

RIGHT ANSWER

OK

D. Presence of jaundice and pruritus

WHY THIS IS RIGHT

The Truelove-Witts criteria are used to identify acute severe ulcerative colitis (ASUC) and guide the need for hospitalization and intensive therapy.
Criteria include:
• ≥6 bloody stools/day
• Plus ≥1 systemic sign of toxicity:
• Pulse ≥90/min
• Temperature >37.8°C
• Hemoglobin <10.5 g/dL
• ESR >30 mm/hr

Not included:
• Jaundice and pruritus, which are features of hepatobiliary disease (e.g., primary sclerosing cholangitis) rather than severity criteria for ulcerative colitis.

QUESTION

Sulfasalazine is commonly used in the treatment of ulcerative colitis and rheumatoid arthritis. Which of the following are recognized adverse effects of sulfasalazine?
A. Bone marrow suppression causing leukopenia
B. Stevens-Johnson syndrome
C. Reversible oligospermia
D. Ototoxicity
E. Hemolytic anemia in G6PD deficiency patient
F. Macrocytic anemia
G. pneumonitis

A All

B **all except D** CORRECT

C all except B,E,F

D A,B,C,D

OK RIGHT ANSWER

B. all except D

WHY THIS IS RIGHT

Sulfasalazine, a 5-aminosalicylic acid (5-ASA) derivative, is used in ulcerative colitis and rheumatoid arthritis and has several recognized adverse effects.
Important adverse effects include:
• Bone marrow suppression -> leukopenia
• Stevens-Johnson syndrome (rare hypersensitivity reaction)
• Reversible oligospermia (affects sperm motility and count)
• Hemolytic anemia, especially in G6PD deficiency
• Folate deficiency leading to macrocytic anemia
• Hypersensitivity reactions including pneumonitis

Not associated: Ototoxicity (seen with drugs like aminoglycosides or loop diuretics).

*

QUESTION

Laboratory investigations of a patient being evaluated for jaundice show elevated bilirubin and alkaline phosphatase levels. Levels of the remaining liver enzymes are normal. What is the likely diagnosis?

A **Obstructive jaundice** CORRECT

B Hemolytic jaundice

C Hepatic jaundice

D Prehepatic jaundice

OK RIGHT ANSWER

A. Obstructive jaundice

WHY THIS IS RIGHT

ALP is present in:
• Bile duct epithelium
• When bile flow is blocked:
• Pressure builds up
• ALP leaks into blood

So: ↑ ALP = cholestasis / obstruction

AST/ALT are Normal because-
• These are hepatocellular injury markers
• In obstruction:
• Liver cells are initially intact
• -> AST/ALT remain normal or mildly raised

Type of Jaundice	Bilirubin	ALP	AST/ALT
Obstructive	↑↑ (direct)	↑↑↑	Normal/mild ↑
Hepatic	↑	↑	↑↑↑
Hemolytic (Prehepatic)	↑ (indirect)	Normal	Normal

Parameter	Obstructive	Hepatic	Hemolytic
Bilirubin	Direct ↑	Mixed	Indirect ↑
ALP	↑↑↑	Mild ↑	Normal
AST/ALT	Mild ↑	↑↑↑	Normal
Urine bilirubin	Present	Present	Absent

QUESTION

A chronic alcoholic presented with abdominal pain, jaundice and weight loss. He has hypoglycemia and elevated alpha fetoprotein levels (600ng/ml) but has normal AST and ALT. Which of the following is a likely diagnosis of the patient?

A Liver cirrhosis

B Hepatitis

C **Hepatocellular carcinoma** CORRECT

D Cholangiocarcinoma

RIGHT ANSWER

OK **C. Hepatocellular carcinoma**

WHY THIS IS RIGHT

• Chronic alcoholic -> chronic liver disease risk
• Abdominal pain + jaundice + weight loss -> malignancy suspicion
• Hypoglycemia -> paraneoplastic clue
• AFP = 600 ng/ml -> tumor marker (normal: 10-25)
• Normal AST/ALT -> not active hepatitis

Put together -> Hepatocellular carcinoma (HCC)

Classic Triad of HCC:
• Weight loss
• Abdominal pain
• Liver mass

Other Paraneoplastic Features:
• Hypoglycemia
• Polycythemia (↑ EPO)
• Hypercalcemia

Imaging Hallmark:
• Arterial phase enhancement + venous washout (CT/MRI)

Feature	HCC	Cirrhosis	Hepatitis	Cholangiocarcinoma
AFP	↑↑	Normal	Normal	Normal
AST/ALT	Normal/slightly ↑	Mild ↑	↑↑	Mild ↑
Hypoglycemia	Yes	No	No	No
Cause	Cirrhosis	Alcohol, viral	Infection	Bile duct malignancy

QUESTION

In a patient who has undergone gastrectomy, vitamin supplementation required is:

A Vit C

B Vit D

C **Vit B12** CORRECT

D Vit A

RIGHT ANSWER

OK

C. Vit B12

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

A young lady presents with jaundice and pruritus. She is antimitochondrial antibody positive and ANA negative. Biopsy shows portal inflammation and ductular proliferation with lymphocytic infiltrate. What is the diagnosis?

A **Primary biliary cholangitis** — CORRECT

B Choledochal cyst

C Autoimmune hepatitis

D Nodular hyperplasia

RIGHT ANSWER

OK

A. Primary biliary cholangitis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

A patient with a history of consumption of 2-3 drinks per week comes with complaints of right upper quadrant discomfort. He has grade 2 steatosis. His BMI was 29 kg/m² and AST and ALT levels were slightly elevated. What would you advise him?

A **Lifestyle modification and weight loss** — CORRECT

B Ursodeoxycholic acid and lifestyle modification

C Lifestyle modifications and vitamin E

D Pioglitazone and lifestyle modifications

OK RIGHT ANSWER

A. Lifestyle modification and weight loss

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

Which of the following medications is most commonly associated with cholestasis in the absence of significant portal inflammation?

A Acetaminophen

B Amoxicillin-clavulanate

C Isoniazid

D **Estrogens/ Androgens** CORRECT

RIGHT ANSWER

OK **D. Estrogens/ Androgens**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

A patient with chronic liver disease presented with jaundice and melena. On examination, there is ascites and mild hepatosplenomegaly. Which of the following is not part of the management of his encephalopathy?

A **Beta blocker to stop variceal bleed** CORRECT

B Ascitic tap to rule out SBP

C Check for HRS

D IV Octreotide

RIGHT ANSWER

OK

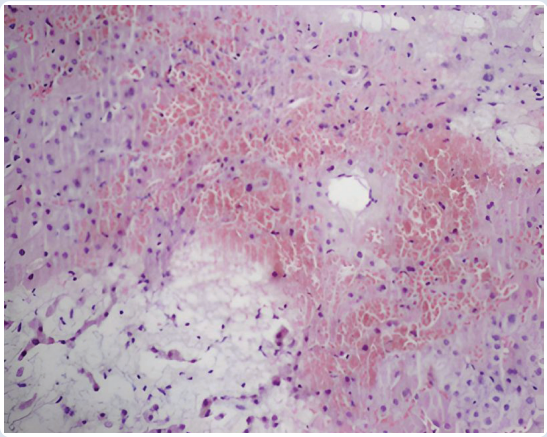
A. Beta blocker to stop variceal bleed

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1d” here and does not include a written explanation for this item.

QUESTION

A 70-year-old man comes to the emergency department due to shortness of breath, poor appetite, and abdominal distension for the past 3 months. His symptoms have progressively worsened, and he is now unable to perform his daily activities. The patient has a history of ischemic heart disease and underwent coronary artery bypass grafting 5 years ago. Physical examination shows jugular venous distension, abdominal distension, hepatomegaly, and bilateral lower extremity edema. A representative liver biopsy image is shown in the exhibit. Which of the following is the most likely cause of this patient's liver findings?



A Autoimmune biliary destruction

B Excessive deposition of iron

C Hepatitis virus infection

D **Passive hepatic congestion** CORRECT

OK RIGHT ANSWER

D. Passive hepatic congestion

WHY THIS IS RIGHT

Chronic right-sided heart failure causes passive hepatic congestion due to elevated central venous pressure transmitted to the hepatic veins and sinusoids.

Increased venous pressure leads to dilation of hepatic sinusoids, particularly around the central vein (zone 3). Reduced hepatic blood outflow causes centrilobular hypoxia and hepatocyte necrosis, with red blood cell congestion in the sinusoids.

Histologic and gross features:
Centrilobular congestion and hemorrhage with relatively preserved periportal hepatocytes produce the characteristic "nutmeg liver" appearance seen in congestive hepatopathy.

*

QUESTION

A 56-year-old man is brought to the emergency department by his wife because of increasing confusion and lethargy for the past 12 hours. He is oriented only to person. His temperature is 37.3°C (99.1°F), pulse is 109/min, respirations are 18/min, and blood pressure is 108/67 mm Hg. Examination shows abdominal distention and several erythematous, lacy lesions on the chest that blanch with pressure. His hands make a flapping motion when they are dorsiflexed. Which of the following is the most likely precipitating factor for this patient's symptoms?

A Elevated systemic vascular resistance

B Destruction of gut anaerobes

C Thiamine pyrophosphate deficiency

D **Accumulation of hemoglobin in the intestine** CORRECT

OK RIGHT ANSWER

D. Accumulation of hemoglobin in the intestine

WHY THIS IS RIGHT

Hepatic encephalopathy occurs in patients with advanced liver disease due to accumulation of neurotoxins, especially ammonia, that are normally detoxified by the liver.

Clinical clues:
• Confusion and lethargy
• Asterixis (flapping tremor)
• Spider angiomas on the chest
• Ascites indicating chronic liver disease

One common trigger is gastrointestinal bleeding. When blood enters the intestinal lumen, hemoglobin is digested into nitrogenous compounds that are metabolized by gut bacteria into ammonia. Because the diseased liver cannot adequately detoxify ammonia into urea, ammonia accumulates in the blood and reaches the brain, leading to hepatic encephalopathy.

QUESTION

A 12 year old male child brought by his parents with complaints of, his school performance has declined, and he has been moodier and more impulsive. His parents also note that he has been sleeping more than usual and has had a poor appetite for the past 2 months. Approximately 3 weeks ago, the patient developed a tremor in both hands that is most prominent when he reaches for an object or tries to write. The tremor has worsened significantly over the past week, and he has developed an unsteady, broad-based gait. Laboratory studies reveal elevated serum transaminases and 24-hour urinary copper excretion. The patient is prescribed penicillamine therapy. This medication primarily works through which of the following mechanisms?

A Decreasing intestinal absorption of copper

B **Increasing urinary excretion of copper** CORRECT

C Repletion of intracellular glutathione

D Stimulation of defective gene expression

RIGHT ANSWER

OK **B. Increasing urinary excretion of copper**

WHY THIS IS RIGHT

Penicillamine is a copper-chelating agent used in the treatment of Wilson disease that increases urinary excretion of copper.

Wilson disease is caused by a mutation in the ATP7B gene, leading to impaired biliary excretion of copper and accumulation of copper in the liver, brain, and other tissues. This results in hepatic dysfunction and neuropsychiatric manifestations such as tremor, behavioral changes, and gait abnormalities.

Penicillamine binds free copper in tissues and blood, forming a soluble complex that is excreted in the urine. This reduces total body copper levels and prevents further tissue damage.

QUESTION

Which of the following is incorrect about obstructive jaundice?

A Increased direct bilirubin

B Increased alkaline phosphatase

C Presence of bilirubin in the urine

D **Increased urobilinogen** CORRECT

RIGHT ANSWER

OK

D. Increased urobilinogen

WHY THIS IS RIGHT

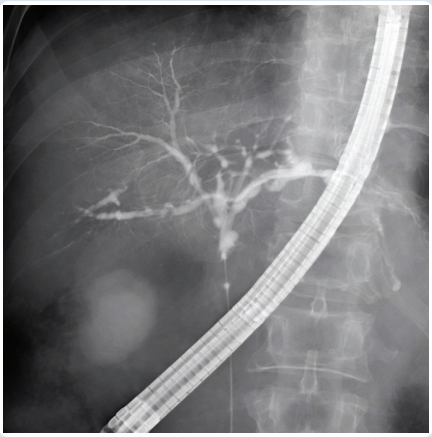
Obstructive (post-hepatic) jaundice occurs due to blockage of bile flow, commonly from gallstones, pancreatic carcinoma, or cholangiocarcinoma. Typical laboratory findings:
• Increased conjugated (direct) bilirubin
• Increased alkaline phosphatase and GGT indicating a cholestatic pattern
• Presence of bilirubin in urine because conjugated bilirubin is water soluble

Urobilinogen is decreased or absent because bile does not reach the intestine, so bilirubin cannot be converted into urobilinogen by intestinal bacteria.

Clinical stool and urine findings:
• Pale or clay-colored stools due to absence of stercobilin
• Dark urine due to excretion of conjugated bilirubin.

QUESTION

A 35-year-old patient is being evaluated for jaundice. ERCP and lab values are shown below.
Aspartate Aminotransferase (AST) - 55 U/L
Alanine Aminotransferase (ALT) - 56 U/L
Alkaline phosphatase (ALP) - 213 U/L
Gamma-glutamyl transferase (GGT) - 75 U/L
Total Bilirubin - 1.2 mg/dL
Which of the following statement is false about this condition?



A It is associated with ulcerative colitis

B It is a premalignant condition

C **Biopsy shows florid duct lesion** CORRECT

D There is no effective medical therapy

OK RIGHT ANSWER

C. Biopsy shows florid duct lesion

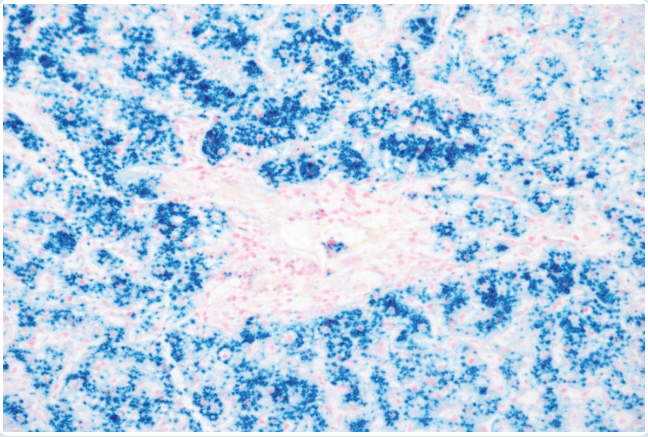
WHY THIS IS RIGHT

The ERCP image showing multifocal strictures and dilatations (“beading”) of intra- and extrahepatic bile ducts with a cholestatic pattern of liver enzymes (↑ALP, ↑GGT) is characteristic of Primary Sclerosing Cholangitis (PSC).
• Chronic inflammatory fibrosis of bile ducts
• Beading appearance on ERCP/MRCP
• Strong association with ulcerative colitis
• Premalignant condition -> increased risk of cholangiocarcinoma
• No effective medical therapy; definitive treatment is liver transplantation

Important distinction:
• Florid duct lesion is seen in Primary Biliary Cholangitis (PBC), not PSC.

QUESTION

A 63-year-old woman dies of congestive heart failure. Autopsy shows a dilated heart with brownish pigmentation of the myocardium. Light microscopy of her liver after Prussian blue staining is shown in the image below. The patient's brother died of profuse upper gastrointestinal bleeding at age 43. Assuming this patient's disorder is hereditary, which of the following most likely contributed to the delayed onset of her disease compared to her brother?



A Heterozygosity for HFE gene mutation

B High-dose vitamin C intake

C Incomplete penetrance of homozygotic HFE mutations

D **Premenopausal menstrual bleeding** CORRECT

RIGHT ANSWER

OK

D. Premenopausal menstrual bleeding

WHY THIS IS RIGHT

This patient likely had hereditary hemochromatosis, an autosomal recessive disorder due to HFE gene mutation causing excess intestinal iron absorption and iron deposition in organs (liver, heart, pancreas).
• Prussian blue staining showing iron deposition (hemosiderin) in hepatocytes

Clinical manifestations:
• Cirrhosis
• Cardiomyopathy
• Diabetes mellitus (“bronze diabetes”)
• Skin hyperpigmentation

Why women present later: Premenopausal menstrual blood loss and pregnancy reduce iron accumulation, delaying iron overload compared with men.

QUESTION

Noninvasive test to detect risk of hepatic fibrosis include?
1. Aspartate aminotransferases /platelet ratio
2. Fibro scan
3. Forns index
4. Serum laminin & serum hyaluronidase

A **1, 2, 3 , 4** CORRECT

B 2, 4

C 1, 3

D 2 only

OK RIGHT ANSWER

A. 1, 2, 3 , 4

WHY THIS IS RIGHT

Several noninvasive tests are used to assess hepatic fibrosis, helping reduce the need for liver biopsy in chronic liver diseases such as hepatitis B, hepatitis C, and NAFLD. Common noninvasive fibrosis assessment tools:

1. AST-to-Platelet Ratio Index (APRI)
• Uses AST level and platelet count
• Helps estimate significant fibrosis and cirrhosis
• 2. FibroScan (Transient Elastography)
• Measures liver stiffness using ultrasound-based elastography
• Widely used for staging fibrosis
• 3. Forns Index
• Uses age, cholesterol, platelet count, and GGT
• Helps predict significant hepatic fibrosis
• 4. Serum fibrosis markers
• Includes laminin and hyaluronic acid
• Reflect extracellular matrix deposition in fibrosis

*

QUESTION

A 46-year-old man is brought to the emergency department by paramedics after an episode of large-volume hematemesis. Physical examination reveals a palpable spleen. Endoscopy shows bleeding esophageal varices. A liver biopsy performed 2 days later shows no abnormalities. Which of the following is the most likely cause of this patient's condition?

A Long-term alcohol consumption

B Budd-Chiari syndrome

C Constrictive pericarditis

D **Portal vein thrombosis** CORRECT

RIGHT ANSWER

OK

D. Portal vein thrombosis

WHY THIS IS RIGHT

Portal vein thrombosis causes prehepatic portal hypertension, leading to splenomegaly and esophageal varices, while the liver parenchyma remains normal on biopsy.

Key features of prehepatic portal hypertension:
• Normal liver histology
• Splenomegaly
• Esophageal/gastric varices -> hematemesis
• May occur due to portal vein thrombosis or external compression

Contrast with other causes:
• Alcoholic cirrhosis -> abnormal liver biopsy with fibrosis and cirrhosis
• Budd-Chiari syndrome -> hepatic vein obstruction with liver congestion and necrosis
• Constrictive pericarditis -> causes congestive hepatopathy

*

QUESTION

Match the Following
Column A - Antibodies
i) Anti-LKM-1
ii) Anti-LKM-2
iii)Anti-LKM-3
iv) Anti-SMA
Column B - Associated Disease
A. Autoimmune hepatitis type 2
B. Drug-induced hepatitis
C. Chronic hepatitis D infection
D. Autoimmune hepatitis type 1

A i-A , ii-C, iii- B, iv- D

B i-B, ii-A, iii-D, iv- C

C i-C ,ii- B, iii- A, iv- D

D **i-A, ii-B, iii-C, iv-D** CORRECT

RIGHT ANSWER

OK **D. i-A, ii-B, iii-C, iv-D**

WHY THIS IS RIGHT

Certain autoantibodies are associated with specific types of autoimmune or viral liver diseases.

Key associations:

Antibody	Associated Disease
Anti-LKM-1	Autoimmune hepatitis type 2
Anti-LKM-2	Drug-induced hepatitis
Anti-LKM-3	Chronic hepatitis D infection
Anti-SMA (Smooth muscle antibody)	Autoimmune hepatitis type 1

Clinical relevance:
• Autoimmune hepatitis type 1: ANA and anti-SMA
• Autoimmune hepatitis type 2: Anti-LKM-1
• LKM antibodies may also appear in drug-induced or viral hepatitis.

*

QUESTION

A 17-year-old boy presents with progressive tremors, dysarthria, personality changes, and jaundice. Slit-lamp examination reveals Kayser-Fleischer rings. Liver biopsy shows copper accumulation. A diagnosis of Wilson's disease is made.Which of the following biochemical findings is most consistent with this diagnosis?

A. Increased ceruloplasmin level in blood with decreased urinary copper excretion

B. **Decreased ceruloplasmin level in blood with increased urinary copper excretion** — CORRECT

C. Increased ceruloplasmin level in blood with increased urinary copper excretion

D. Normal ceruloplasmin level in blood with decreased urinary copper excretion

RIGHT ANSWER

OK

B. Decreased ceruloplasmin level in blood with increased urinary copper excretion

WHY THIS IS RIGHT

Wilson disease is an autosomal recessive disorder caused by mutation in the ATP7B gene, leading to impaired copper excretion into bile and defective incorporation of copper into ceruloplasmin.
• ↓ Serum ceruloplasmin
• ↑ Free copper in blood
• ↑ Urinary copper excretion
• Copper accumulation in liver, brain (basal ganglia), and cornea (Kayser-Fleischer rings)

Clinical features:
• Hepatitis, cirrhosis
• Neurologic symptoms (tremor, dysarthria, psychiatric changes)
• Kayser-Fleischer rings on slit-lamp exam.

Treatment:
• Copper chelation (penicillamine or trientine)
• Zinc therapy to reduce copper absorption.

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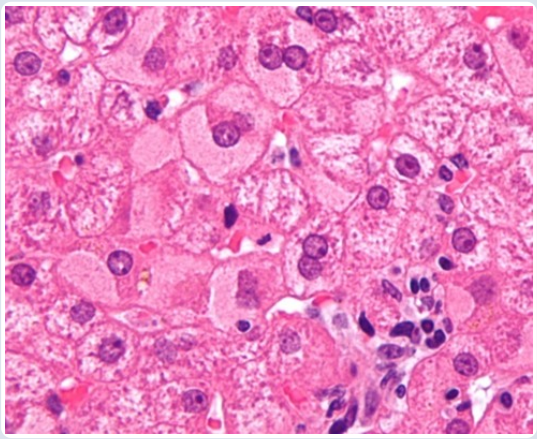
QUESTION

58-year-old male with a history of multiple sexual partners, presented with Anorexia and Jaundice. Biopsy shows ground-glass hepatocytes. What is the most probable diagnosis?



A **Hepatitis B** CORRECT

B Hepatitis A

C Hepatitis C

D Hepatitis D

RIGHT ANSWER

OK **A. Hepatitis B**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which of the following anti-HIV drugs is effective in treatment of hepatitis B?

A Dolutegravir

B Ritonavir

C **Lamivudine** CORRECT

D Saquinavir

RIGHT ANSWER

OK

C. Lamivudine

WHY THIS IS RIGHT

Lamivudine is a nucleoside reverse transcriptase inhibitor active against both HIV and hepatitis B virus. It inhibits HBV DNA polymerase and is used in chronic HBV therapy. Protease inhibitors and integrase inhibitors have no activity against HBV. Dual antiviral activity is the key exam clue for lamivudine.

Drug	Class	HBV Activity
Lamivudine	NRTI	□
Tenofovir	NRTI	□
Emtricitabine	NRTI	□
Entecavir	HBV drug	□ HIV

QUESTION

A patient presents to you with fever, jaundice, and malaise. What is the most likely diagnosis based on the serology reports given below?

- **Anti-HBc (IgM): Positive**
- **HBsAg: Positive**
- **Anti-HBs: Negative**
- **Anti-HCV antibodies: Negative**

A **Acute hepatitis B** — CORRECT

B Acute hepatitis C

C Chronic hepatitis B

D Chronic hepatitis C

RIGHT ANSWER

OK **A. Acute hepatitis B**

WHY THIS IS RIGHT

Marker	Meaning
HBsAg (+)	Current infection present
Anti-HBc IgM (+)	Acute infection
Anti-HBs (-)	No immunity yet
Anti-HCV (-)	Not hepatitis C

HBsAg positivity with anti-HBc IgM positivity is diagnostic of acute hepatitis B. IgM anti-HBc indicates recent infection and active hepatocellular injury. Absence of anti-HBs confirms the patient has not yet recovered or developed immunity. This serologic pattern is classic for acute HBV infection.

Option B) Acute Hepatitis C
• Would show:

o Anti-HCV positive

Option C) Chronic Hepatitis B
• Would show:

o Anti-HBc IgG, not IgM
o Duration > 6 months

Option D) Chronic Hepatitis C
• Requires:

o Anti-HCV positive

QUESTION

A 30-year-old male is found to be positive for HBsAg and HBeAg and is diagnosed with chronic hepatitis B. The viral load of the patient was 2 x 10^5 IU/mL and ALT is found to be doubled. What is the appropriate management?

A Lamivudine

B **Tenofovir** CORRECT

C Peg IFN for 52 weeks

D Combined peg IFN with lamivudine

RIGHT ANSWER

OK **B. Tenofovir**

WHY THIS IS RIGHT

Chronic HBV (HBsAg > 6 months)
↓
Check HBeAg + HBV DNA + ALT
↓
High DNA + ↑ ALT
HBV DNA >2000IU/ml
↓
Start antiviral therapy
↓
Tenofovir + Entecavir x 12wks

Tenofovir:
□ Class: Nucleotide analog (reverse transcriptase inhibitor)
□ Action:
• Inhibits HBV DNA polymerase
• -> Stops viral replication

QUESTION

A 30-year-old man had an episode of jaundice due to HBV one year back. On follow-up now, he has normal levels of liver enzymes. His current profile is shown.

- **HBsAg: Positive**
- **HBeAg: Negative**
- **Anti-HBe: Positive**
- **Anti-HBc IgG: Positive**
- **Anti-HBc IgM: Negative**
- **HBV DNA: Low**
- **ALT: Normal**

A Chronic Hepatitis B

B **Chronic Hepatitis B** CORRECT

C Resolved infection

D Acute Hepatitis B

RIGHT ANSWER

OK **B. Chronic Hepatitis B**

WHY THIS IS RIGHT

Key interpretation of markers:
• HBsAg positive (>1 year) -> Chronic infection
• HBeAg negative + Anti-HBe positive -> Low replication
• Anti-HBc IgG positive, IgM negative -> Chronic (not acute)
• HBV DNA low + ALT normal -> Inactive carrier state

Why not others?
• Resolved infection: HBsAg would be negative, Anti-HBs positive
• Acute HBV: Anti-HBc IgM positive
• Chronic Hep B (active): ↑ ALT, high HBV DNA, often HBeAg +

QUESTION

The blood investigation of a patient is given below. What is the probable diagnosis?

- **HBsAg - NR,**
- **Anti HBs - NR,**
- **Anti HBc IgM - NR,**
- **Anti HBc total - positive,**
- **HBeAg - NR,**
- **Anti HBe - NR (NR - Non reactive)**

A Acute HBV infection in window period

B Immune with recombinant HBV Vaccine

C **HBV infection in the remote past, completely recovered** CORRECT

D Chronic HBV infection inactive carrier

RIGHT ANSWER

OK **C. HBV infection in the remote past, completely recovered**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

Which of the following viruses spreads predominantly through water?

A HCV

B **HAV (Hepatitis A Virus)** CORRECT

C HDV

D HBV

RIGHT ANSWER

OK **B. HAV (Hepatitis A Virus)**

WHY THIS IS RIGHT

Hepatitis A virus spreads by the fecal-oral route, classically through contaminated water and food. It causes acute, self-limited hepatitis and is linked to poor sanitation and outbreaks. In contrast, HBV, HCV, and HDV are transmitted parenterally via blood or body fluids. Waterborne transmission is a key epidemiologic hallmark of HAV.

Feature	HAV	HBV	HCV	HDV	HEV
Transmission	Feco-oral	Blood/sexual	Blood	Blood	Feco-oral
Chronicity	□	□	□ (high)	□	□ (except pregnancy severe)
Envelope	□	□	□	□	□
Vaccine	□	□	□ (via HBV)	□
Special note	Waterborne outbreaks	Vertical transmission	Silent chronic	Needs HBV	Severe in pregnancy

• HAV & HEV = Enterically transmitted
• HBV, HCV, HDV = Parenteral
• HAV:
• No carrier state
• No chronic hepatitis
• Lifelong immunity after infection

QUESTION

A man presents with deranged LFT. The serum viral markers are given below:

Anti-HAV IgM - Non-reactive
Anti-HAV-IgG - Reactive
HBsAg - Non-reactive
Anti-HBc total/IgG - Reactive
Anti-HBc IgM - Non-reactive
Anti-HCV IgM - Non-reactive
Anti-HCV IgG - Reactive
Anti-HEV IgM - Non-reactive
Anti-HEV IgG - Non-reactive

Which of the following statements is not correct about his condition?

A **Chronic Hepatitis B infection, HBV DNA titres should be done by real time PCR** — CORRECT

B HBV infection in the remote past

C HCV DNA titres should be estimated by RT-PCR

D Past HAV infection or received vaccine for HAV

OK RIGHT ANSWER

A. Chronic Hepatitis B infection, HBV DNA titres should be done by real time PCR

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

A health worker had a needle stick injury. While handling a chronic Hepatitis B case. She has taken 2 complete schedules (6 doses) of HBV vaccination. She was tested and was diagnosed as a non-responder to vaccination 3 months back, What is the most appropriate management in this case:

A **Give 2 doses of HBIG one now and another 4 weeks later** CORRECT

B Give 2 doses of HBIG now (stat)

C Give HBV vaccination starting now, after 4-6 weeks, check HBV titres and decide on giving HBIG

D HBIG single dose with HBV vaccination (first dose beginning now)

OK RIGHT ANSWER

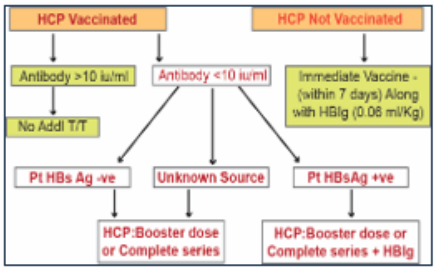
A. Give 2 doses of HBIG one now and another 4 weeks later

WHY THIS IS RIGHT

A documented vaccine non-responder exposed to an HBsAg-positive source requires passive immunization only. Two doses of HBIG are given: one immediately and another at 1 month, as further vaccination is ineffective in proven non-responders. HBIG provides immediate anti-HBs protection after high-risk exposure. Revaccination strategies are used only in unknown or incomplete responders, not confirmed non-responders.

Criteria	Meaning
Anti-HBs <10 mIU/mL	No protective immunity
After 2 full vaccine series	True non-responder

Non-responder : anti-HBs < 10 after 2 full series
What to do: HBIG × 2 doses: 4 weeks apart; No further vaccine



QUESTION

A 42-year-old male patient presented with jaundice. His AST was 48 U, ALT was 51 U, ALP, GGT were normal. Ultrasound of liver was suggestive of cirrhosis. Viral markers were done and the following results were obtained. What is the next best step?
Anti-HAV: Negative
Anti-HBeAg: Negative
Anti-HBcAg IgG: Positive
Anti-HBcAg IgM :Negative
HBsAg: Negative
Anti-HbsAg: Positive
Anti-HCV: Positive

A Start interferon therapy

B Liver biopsy

C **RT-PCR for hepatitis C virus** CORRECT

D PCR for HBV-DNA

OK RIGHT ANSWER

C. RT-PCR for hepatitis C virus

WHY THIS IS RIGHT

-Anti-HCV positivity indicates exposure to hepatitis C virus but does not confirm active infection.
-The next step is to detect viral RNA using RT-PCR to determine whether there is ongoing viral replication.
• Anti-HAV negative -> no evidence of hepatitis A infection.
• HBsAg negative with anti-HBs positive and anti-HBc IgG positive -> past hepatitis B infection with immunity.
• Anti-HCV positive -> prior exposure to hepatitis C.

Since hepatitis C antibodies can persist even after viral clearance, confirmation of active infection requires detection of HCV RNA.

QUESTION

34-year-old woman comes to the OPD with recent onset of malaise. The patient works as a nurse at a local hospital and lives at home with her husband and 2-year-old son. Physical examination is notable for hepatomegaly. Laboratory results are as follows:
Anti-HAV IgM Positive
Anti-HAV IgG negative
HBsAg negative
HBeAg negative
Anti-HBs positive
Anti-HBc negative
Anti-HBe negative
Anti-HCV negative
Which of the following is most likely to be elicited on further history taking?

A Had an accidental needlestick exposure at work

B Had a blood transfusion

C **Had steamed oysters at a neighborhood restaurant** CORRECT

D Had unprotected sexual intercourse with a new partner

OK RIGHT ANSWER

C. Had steamed oysters at a neighborhood restaurant

WHY THIS IS RIGHT

Hepatitis A virus (HAV) is transmitted via the fecal-oral route, commonly through contaminated food or water.
• Anti-HAV IgM positive indicates an acute hepatitis A infection.
• Anti-HAV IgG negative indicates the patient has not yet developed long-term immunity.
• Anti-HBs positive with negative HBsAg and anti-HBc suggests immunity to hepatitis B due to vaccination.

Common sources of HAV infection include:
• Contaminated food or water
• Raw or undercooked shellfish (especially oysters) from contaminated water
• Close household or daycare contact with infected individuals
• Poor sanitation or travel to endemic areas

QUESTION

Two weeks after returning from vacation, a 21-year-old man comes to the emergency department because of malaise, nausea, vomiting, fever, and abdominal pain. He has no history of serious illness and takes no medications. Physical examination shows scleral icterus and right upper quadrant tenderness. The liver is palpated 1.5 cm below the right costal margin. A biopsy specimen of this patient's liver would most likely show which of the following findings?

A Concentric periductal fibrosis

B **Ground glass hepatocytes and periportal inflammation** CORRECT

C Mallory Denk bodies

D Necrosis with neutrophilic infiltrate

OK RIGHT ANSWER

B. Ground glass hepatocytes and periportal inflammation

WHY THIS IS RIGHT

Ground-glass hepatocytes are characteristic of hepatitis B infection and occur due to accumulation of hepatitis B surface antigen (HBsAg) within the endoplasmic reticulum of hepatocytes.
• Viral hepatitis commonly presents with fever, malaise, nausea, vomiting, jaundice, and hepatomegaly.
• Hepatitis B infection shows hepatocytes with finely granular eosinophilic cytoplasm on histology, giving a “ground-glass” appearance.
• There is also periportal inflammation due to immune-mediated hepatocyte injury.
• Differential histologic clues:
• Concentric periductal fibrosis -> primary sclerosing cholangitis
• Mallory-Denk bodies -> alcoholic hepatitis or nonalcoholic steatohepatitis
• Necrosis with neutrophilic infiltrate -> severe alcoholic hepatitis.

*

QUESTION

A 33-year-old woman, gravida 2, para 1, at 24 weeks' gestation is brought to the emergency department by her husband for lethargy, nausea, and vomiting for 4 days. She returned from a trip to Thailand 2 weeks ago. Her immunizations are up-to-date and she has never received blood products. Her temperature is 38.9°C (102°F). She is not oriented to person, place, or time. Examination shows jaundice and mild asterixis. Her prothrombin time is 18 sec (INR=2.0), serum alanine aminotransferase is 3911 U/L, and serum aspartate aminotransferase is 3724 U/L. This patient's current condition is most likely associated with increased titers of which of the following serum studies?

A Anti-HBe IgM

B HBsAg

C **Anti-HEV IgM** CORRECT

D Anti-HCV IgG

OK RIGHT ANSWER

C. Anti-HEV IgM

WHY THIS IS RIGHT

Hepatitis E virus (HEV) infection is a major cause of acute viral hepatitis in developing regions (e.g., South and Southeast Asia) and is transmitted via the fecal-oral route through contaminated water.
• Pregnant woman (especially 2nd-3rd trimester) with acute liver failure
• Travel to endemic area
• Very high AST/ALT (>3000 U/L)
• Encephalopathy and coagulopathy -> acute fulminant hepatitis

Diagnosis: Anti-HEV IgM antibodies -> marker of acute HEV infection

HEV infection during pregnancy carries high risk of fulminant hepatic failure and mortality.

Contrast:
• HBV -> HBsAg positive
• HCV -> Anti-HCV IgG (usually chronic)
• Anti-HBe IgM is not a typical marker.

QUESTION

A previously healthy 25-year-old woman comes to the physician because of a one-week history of diffuse abdominal pain. Her temperature is 39.1°C (102.3°F). Physical examination shows numerous scars and excoriations along both arms, scleral icterus, and tender hepatomegaly. Serum studies shown below:
Which of the following is least likely to be seen in the liver biopsy of this patient?

Alanine Aminotransferase	927 U/L
Aspartate Aminotransferase	796 U/L
Hepatitis B Surface Antigen	Positive
Hepatitis B Surface Antibody	Negative
Anti-Hepatitis B Core Antibody	Negative
Hepatitis C Antibody	Negative

A Ballooning degeneration

B **Ground glass hepatocytes** CORRECT

C Focal or spotty necrosis

D Confluent necrosis of hepatocytes

RIGHT ANSWER

OK **B. Ground glass hepatocytes**

WHY THIS IS RIGHT

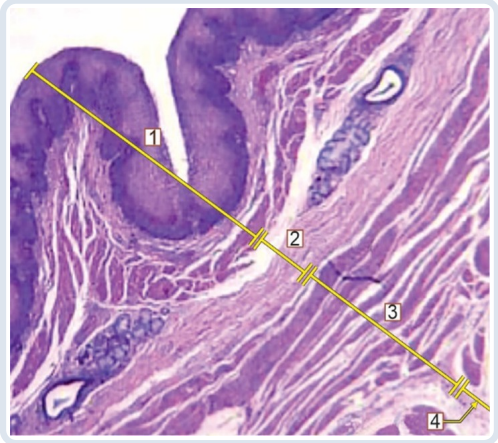
This patient has acute hepatitis B infection (HBsAg positive with very high AST/ALT and acute symptoms).

Histologic findings in acute viral hepatitis include:
• Ballooning degeneration of hepatocytes
• Focal or spotty necrosis (apoptosis)
• Confluent necrosis in severe cases
• Inflammatory infiltrates in the lobules

Ground-glass hepatocytes are not typical of acute infection.
They are characteristic of chronic hepatitis B, caused by accumulation of hepatitis B surface antigen (HBsAg) in the endoplasmic reticulum of hepatocytes.

QUESTION

A young male child presents with failure to pass meconium and has been diagnosed with megacolon. Which of the following layers is affected by the absence of neurons?



A 1 and 2

B **2 and 3** CORRECT

C 3 and 4

D 1 and 4

OK RIGHT ANSWER

B. 2 and 3

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which of the following about the enteric nervous regulation system is false?

1. Interstitial cells of Cajal are pacemakers
2. It can function independently of the brain and spinal cord
3. All the afferents are from the vagus nerve
4. Myenteric plexus is in the Lamina propria

A 1, 2

B 1, 2, 3

C 1, 3, 4

D **3, 4** CORRECT

OK RIGHT ANSWER

D. 3, 4

WHY THIS IS RIGHT

The Enteric Nervous System (ENS) is like a quiet maestro conducting digestion:
• Coordinates motility, secretion, and blood flow
• Works independently of CNS (but can be modulated by it)

Can function independently of CNS input. Interstitial cells of Cajal act as pacemakers generating slow waves. Afferents are not exclusively vagal; significant intrinsic and spinal afferents exist.

Feature	Myenteric (Auerbach)	Submucosal (Meissner)
Location	Between muscle layers	Submucosa
Function	Motility	Secretion, blood flow
Extent	Entire GI tract	Mainly small intestine

Clinical correlations

Disease	Defect
Hirschsprung disease	Absence of ganglion cells
Achalasia	Loss of inhibitory neurons
IBS	Dysregulated ENS

QUESTION

Which of the following sites has the least concentration of sodium ions in the fluid?

A **Salivary glands** CORRECT

B Stomach

C Duodenum

D Ileum

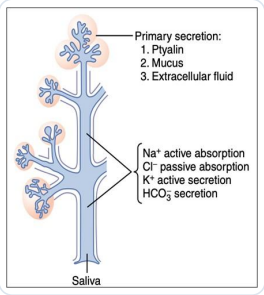
RIGHT ANSWER

OK **A. Salivary glands**

WHY THIS IS RIGHT

GI secretions are not static liquids. They get modified along ducts and segments like a river changing chemistry as it flows.
Saliva is the only GI fluid that becomes hypotonic
-> meaning Na□ gets reabsorbed more than water
Acinar cells -> Secrete isotonic fluid (Na□ high)
↓
Ductal cells -> Reabsorb Na□ & Cl□
↓
Final saliva -> Hypotonic (Na□ ↓↓↓, K□ ↑)
Ducts are impermeable to water
-> Na□ leaves, water cannot follow -> dilution happens

Site	Na□ Concentration	Key Feature
Saliva	Lowest	Hypotonic
Stomach	Moderate	HCl secretion dominates
Duodenum	Higher	Mixing with pancreatic juice
Ileum	Near isotonic	Absorption ongoing



QUESTION

Which of the following is correct treatment of peptic ulcer associated with H. pylori?

A Omeprazole only

B Omeprazole, Ciprofloxacin plus amoxicillin

C **Omeprazole, Clarithromycin and Metronidazole** CORRECT

D Metronidazole, Omeprazole and Sucralfate

RIGHT ANSWER

OK **C. Omeprazole, Clarithromycin and Metronidazole**

WHY THIS IS RIGHT

H. pylori peptic ulcer requires triple therapy with a proton pump inhibitor plus two antibiotics.
• Colonizes gastric mucosa
• Produces urease -> ammonia -> mucosal damage
• Causes:
• Chronic gastritis
• Peptic ulcer

Standard Triple Therapy
• PPI (e.g., Omeprazole)
• Clarithromycin
• Amoxicillin *(or Metronidazole if penicillin allergy)*

Quadruple therapy	PPI + Bismuth + Tetracycline + Metronidazole	Resistant cases

Important Tests:
• Urea breath test
• Stool antigen test

Complications of H. pylori:
• Gastric carcinoma
• MALT lymphoma

QUESTION

An antiemetic was given to a chemotherapy patient, who then developed symptoms such as acute dystonia, bradykinesia, and tremors. Which of the following medications would have resulted in these symptoms?

A Ondansetron

B **Metoclopramide** CORRECT

C Meclizine

D Scopolamine

RIGHT ANSWER

OK

B. Metoclopramide

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

Which of the following drugs used in the treatment of irritable bowel syndrome (IBS) has a direct spasmolytic action on gastrointestinal (GI) smooth muscle?

A Dicyclomine

B Scopolamine

C **Mebeverine** CORRECT

D Racecadotril

OK RIGHT ANSWER

C. Mebeverine

WHY THIS IS RIGHT

Mebeverine:
• A direct smooth muscle relaxant
• Works independently of autonomic receptors

Mechanism of Action
• Blocks sodium channels in smooth muscle
• Reduces calcium influx indirectly
• -> Decreases contractility
• -> Relieves intestinal spasm

Type of Drug	Example	Mechanism
Direct spasmolytic	Mebeverine	Acts directly on muscle
Anticholinergic	Dicyclomine, Scopolamine	Blocks M receptors
Antisecretory	Racecadotril	↓ intestinal secretion

Drug	Mechanism	IBS Role	Side Effects
Mebeverine	Direct muscle relaxant	Best for pain/spasm	Minimal
Dicyclomine	Anticholinergic	Useful	Anticholinergic effects
Scopolamine	Anticholinergic	Limited	CNS + anticholinergic
Racecadotril	↓ secretion	Diarrhea	Minimal

QUESTION

What is the rationale for the use of atropine in combination with diphenoxylate?

A Atropine decreases the adverse effect of diphenoxylate

B **To prevent abuse of diphenoxylate** CORRECT

C To enhance the antidiarrheal effect

D Atropine produces direct spasmolytic action

RIGHT ANSWER

OK

B. To prevent abuse of diphenoxylate

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

Aprepitant is a drug used in the treatment of some cases of chemotherapy-induced nausea and vomiting. What is the mechanism of action of this drug?

A **NK1 antagonist** CORRECT

B NK3 antagonist

C NK1 agonist

D NK2 agonist

RIGHT ANSWER

OK **A. NK1 antagonist**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

A 45-year-old man with a known history of gastroesophageal reflux disease (GERD) presents with persistent regurgitation and bloating despite several weeks of proton pump inhibitor therapy. To improve his symptoms, the physician plans to prescribe a medication that enhances lower esophageal sphincter tone and accelerates gastric emptying. Which of the following agents would be the most appropriate choice?

A Vonoprazan

B Omeprazole

C **Metoclopramide** CORRECT

D Sodium alginate

OK RIGHT ANSWER

C. Metoclopramide

WHY THIS IS RIGHT

Metoclopramide acts like a traffic controller for the gut, clearing congestion and tightening the gate (LES).

Mechanism:
• D□ receptor antagonist
• ↑ Acetylcholine release in gut

Effects:
• ↑ Lower esophageal sphincter (LES) tone
• ↑ Gastric emptying
• ↓ Reflux

Drug	Class	Mechanism	Use
Omeprazole	PPI	↓ H□ secretion	First-line GERD
Vonoprazan	P-CAB	Strong acid suppression	Resistant GERD
Metoclopramide	Prokinetic	↑ LES tone + motility	Refractory GERD
Sodium alginate	Barrier	Floating raft	Mild symptoms

QUESTION

Which of the following statements about Lafutidine is incorrect?

1. Increases gastric mucosal blood flow
2. Acts as an H□ receptor antagonist
3. Decreases nitric oxide production
4. Increases somatostatin secretion+

A 1 and 2

B 2 and 4

C **3 only** CORRECT

D 1, 2 and 4

OK RIGHT ANSWER

C. 3 only

WHY THIS IS RIGHT

Lafutidine is a second-generation H□ receptor antagonist used for:
• Peptic ulcer disease
• GERD
• Gastritis
• Stress ulcer prophylaxis

What makes it special is that it has both acid-suppressing and mucosal-protective actions.
Correct answer is C because lafutidine is an H□ receptor antagonist that enhances mucosal defense by increasing gastric mucosal blood flow and stimulating protective mediators. It is known to promote nitric oxide-mediated mucosal protection rather than decrease nitric oxide production. Protective effects include:

Effect	Mechanism
↑ Gastric mucosal blood flow	via nitric oxide
↑ Prostaglandin synthesis	mucosal protection
↑ Somatostatin secretion	inhibits gastrin
↑ Mucus secretion	barrier protection

Thus it protects gastric mucosa beyond acid suppression.

QUESTION

Why is atropine mixed with diphenoxylate in combination medications?

A Atropine decreases the adverse effect of diphenoxylate

B **To prevent abuse of diphenoxylate** CORRECT

C To enhance the antidiarrheal effect

D Atropine produces direct spasmolytic action

OK RIGHT ANSWER

B. To prevent abuse of diphenoxylate

WHY THIS IS RIGHT

Diphenoxylate + Atropine (e.g., Lomotil)
• Diphenoxylate -> opioid derivative (antidiarrheal)
• Atropine -> anticholinergic (very low dose in combo)

Diphenoxylate is an opioid that can cause euphoria in high doses. A small amount of atropine is added to produce unpleasant anticholinergic effects if overdosed. This discourages intentional misuse while preserving antidiarrheal action. The combination is designed as an abuse-deterrent formulation.
Diphenoxylate taken in high dose
↓
Atropine also increases
↓
Anticholinergic toxicity
↓
Unpleasant symptoms
↓
Discourages abuse

Component	Role
Diphenoxylate	Antidiarrheal (opioid)
Atropine	Abuse deterrent

QUESTION

A patient with a history of chronic liver disease presents with abdominal distension, jaundice, and pruritis. Ascitic fluid analysis revealed a neutrophil count > 650 per cubic mm. What is the most likely diagnosis?

A **Spontaneous bacterial peritonitis** CORRECT

B Malignant ascites

C Tubercular ascites

D Intestinal obstruction

RIGHT ANSWER

OK **A. Spontaneous bacterial peritonitis**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1d” here and does not include a written explanation for this item.

QUESTION

A mother brought her 2-year-old child to the OPD with complaints of lack of weight gain, abdominal pain on eating, and poor eating habits. The child was advised to follow a gluten-free diet, after which there was an improvement in the general condition and weight gain. What is the probable diagnosis?

A **Celiac disease** CORRECT

B Tropical sprue

C Abetalipoproteinemia

D Whipple disease

OK RIGHT ANSWER

A. Celiac disease

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1e” here and does not include a written explanation for this item.

QUESTION

A patient with diarrhoea and skin lesions was advised to change his diet, which resulted in the improvement of his symptoms. Which of the following is the likely antibody present in this patient?

A **Anti TTG antibody** CORRECT

B Anti Saccharomyces cerevisiae antibody

C p-ANCA

D Anti-nuclear antibody

RIGHT ANSWER

OK

A. Anti TTG antibody

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1f” here and does not include a written explanation for this item.

QUESTION

A young boy with abdominal pain, diarrhea, and skin lesions was advised to change his diet, which resulted in the improvement of his symptoms. Which of the following is the likely diagnosis in this patient?

A Tropical sprue

B **Celiac sprue** CORRECT

C Whipple's disease

D Giardiasis

OK RIGHT ANSWER

B. Celiac sprue

WHY THIS IS RIGHT

The source quiz contains the placeholder "$20" here and does not include a written explanation for this item.

QUESTION

Which of the following is not associated with celiac disease?

A Modified Marsh classification

B IgA transglutaminase positivity

C IgG antigliadin positivity

D **HLA DQ6 and HLA DQ11** CORRECT

RIGHT ANSWER

OK

D. HLA DQ6 and HLA DQ11

WHY THIS IS RIGHT

The source quiz contains the placeholder “$21” here and does not include a written explanation for this item.

QUESTION

A patient presents with abdominal pain, acute diarrhea, and is diagnosed with celiac disease. Which of the following statements is false regarding this condition?

A **Associated with HLA DQ6/DQ11** CORRECT

B IgA Tissue Transglutaminase antibodies are present

C Marsh Criteria used for diagnosis

D IgG deamidated Gliadin antibodies are present

OK RIGHT ANSWER

A. Associated with HLA DQ6/DQ11

WHY THIS IS RIGHT

The source quiz contains the placeholder “$22” here and does not include a written explanation for this item.

QUESTION

The presence of buccal mucosal melanosis and hamartomatous polyp is a feature of which of the following conditions?

A Lynch syndrome

B Familial adenomatous polyposis

C **Peutz-Jeghers syndrome** CORRECT

D Juvenile polyposis syndrome

RIGHT ANSWER

OK

C. Peutz-Jeghers syndrome

WHY THIS IS RIGHT

Peutz-Jeghers syndrome is characterized by hamartomatous GI polyps and mucocutaneous melanotic pigmentation, especially on the buccal mucosa and lips. It results from STK11 mutation and carries increased cancer risk. The pigmentation is a classic clinical clue. Other polyposis syndromes lack this mucocutaneous feature.

Feature	Description
Pigmentation	Lips, buccal mucosa, fingers □
Polyps	Hamartomatous
Location	Small intestine (jejunum common)
Complication	Intussusception
Cancer risk	↑ GI + pancreatic + others

Feature	Peutz-Jeghers	FAP	Lynch	Juvenile Polyposis
Gene	STK11	APC	MMR genes	SMAD4/BMPR1A
Polyps	Hamartomatous	Adenomatous	Few	Hamartomatous
Pigmentation	Yes	No	No	No
Cancer risk	↑ multiple	100% CRC	↑ CRC	↑ CRC

QUESTION

Match the column:

Column A	Column B
A. Peetz Jegher polyp	1. APC
B. Familial adenomatous polyp	2. TSC 2
C. Juvenile polyposis	3. STK11
D. Tuberous sclerosis	4. SMAD4

A **A-3, B-1, C-4, D-2** CORRECT

B A-4, B-2, C-1, D-3

C A-3, B-1, C-2, D-4

D A-4, B-1, C-3, D-2

OK

RIGHT ANSWER

A. A-3, B-1, C-4, D-2

WHY THIS IS RIGHT

The source quiz contains the placeholder “$23” here and does not include a written explanation for this item.

QUESTION

Which of the following statements regarding Peutz-Jeghers syndrome are true
1. Heterogeneous loss of function of the STK11 gene
2. Presents as multiple hamartomatous polyps in the GIT
3. Histology shows arborising smooth muscle bundles
4. It is an autosomal recessive condition
5. Extra-intestinal manifestations of Peutz-Jeghers syndrome include hypertrophy of the retinal pigment epithelium

A **1, 2, and 3** CORRECT

B 1, 3, and 5

C 2, 4, and 5

D 1, 2, 3, and 4

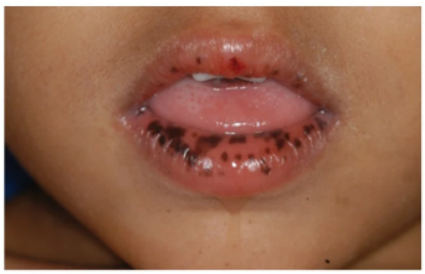
OK RIGHT ANSWER

A. 1, 2, and 3

WHY THIS IS RIGHT

Peutz-Jeghers syndrome is due to loss-of-function mutation of the STK11 tumor suppressor gene and is inherited in an autosomal dominant pattern. It presents with multiple hamartomatous GI polyps showing classic arborizing smooth muscle bundles on histology. Retinal pigment hypertrophy is seen in familial adenomatous polyposis, not Peutz-Jeghers.

|
Feature	Peutz-Jeghers	FAP
Gene	STK11	APC
Inheritance	AD	AD
Polyp type	Hamartomatous	Adenomatous
Histology	Arborizing smooth muscle	Dysplastic epithelium
Cancer risk	Moderate-high (extraintestinal)	Very high (colon)
Extra features	Mucocutaneous pigmentation	CHRPE (retinal lesions)



QUESTION

In contrast to Ulcerative Colitis, which of the following statements regarding Crohn's disease is true?

A There is no increased risk of colon cancer

B Continuous involvement rather than skip lesions

C **Perianal fistulas are frequent** CORRECT

D Seldom manifests with hematochezia

RIGHT ANSWER

OK

C. Perianal fistulas are frequent

WHY THIS IS RIGHT

Crohn disease causes transmural inflammation, leading to fissures, abscesses, and frequent perianal fistulas. It shows skip lesions and has an increased colorectal cancer risk with long-standing colonic involvement.

Feature	Crohn	UC
Pattern	Skip lesions	Continuous
Depth	Transmural	Mucosal
Fistula	Common	Rare
Perianal disease	Common	Rare

QUESTION

Which JAK inhibitor drug is recommended for moderate to severe ulcerative colitis in a patient who is not responding well to TNF-alpha blockers?

A Ustekinumab

B Etanercept

C Vedolizumab

D **Tofacitinib** CORRECT

OK RIGHT ANSWER

D. Tofacitinib

WHY THIS IS RIGHT

Tofacitinib:
• Class: JAK inhibitor (Janus kinase inhibitor)
• Type: Oral small molecule (unlike biologics which are injectable)

Mild UC
↓
5-ASA (Mesalamine)
↓ (if not controlled)
Steroids
↓
Moderate-Severe UC
↓
Anti-TNF (Infliximab)
↓ (failure)
Tofacitinib / Vedolizumab / Ustekinumab

Drug	Class	Mechanism	Use in IBD
Infliximab	Anti-TNF	Blocks TNF-α	UC, Crohn
Etanercept	Anti-TNF	TNF receptor fusion protein	□ Not in IBD
Vedolizumab	Anti-integrin	Blocks gut lymphocyte entry	UC, Crohn
Ustekinumab	Anti IL-12/23	↓ Th1/Th17	UC, Crohn
Tofacitinib	JAK inhibitor	Blocks cytokine signaling	UC (oral)

QUESTION

The Truelove and Witt criteria are used for assessing which of the following conditions?

A Encephalitis

B **Acute severe ulcerative colitis** CORRECT

C ARDS

D Acute pancreatitis

OK RIGHT ANSWER

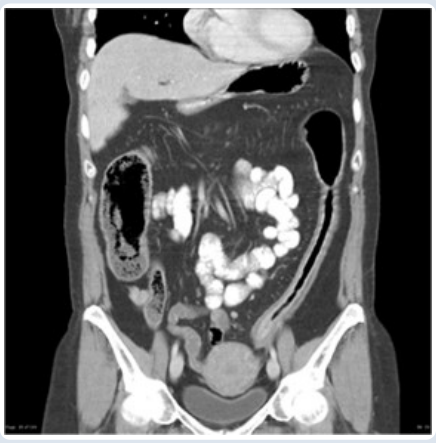
B. Acute severe ulcerative colitis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$24” here and does not include a written explanation for this item.

QUESTION

A 26-year-old man presents with an 8-month history of chronic diarrhea. CT shows the following. Based on these findings, what is the diagnosis?



A Pseudomembranous enterocolitis

B **Ulcerative colitis** CORRECT

C Crohn's disease

D Intestinal TB

OK RIGHT ANSWER

B. Ulcerative colitis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$25” here and does not include a written explanation for this item.

QUESTION

Onion skin fibrosis on histopathology is seen in which of the following conditions?

A Primary biliary cirrhosis

B Chronic cirrhosis

C **Primary sclerosing cholangitis** CORRECT

D Extrahepatic biliary atresia

RIGHT ANSWER

OK

C. Primary sclerosing cholangitis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$26” here and does not include a written explanation for this item.

QUESTION

A patient with ulcerative colitis is experiencing three episodes of bleeding in stools per day but is otherwise stable. What is the appropriate treatment for this patient?

A **5-Aminosalicylic acid** CORRECT

B 5-Fluorouracil

C Budesonide

D 6-Mercaptopurine

OK RIGHT ANSWER

A. 5-Aminosalicylic acid

WHY THIS IS RIGHT

Mild ulcerative colitis with limited bleeding and stable vitals is treated with 5-aminosalicylic acid (5-ASA) as first-line therapy. It acts locally to reduce mucosal inflammation and induces remission. Steroids and immunomodulators are reserved for moderate-severe or refractory disease. 5-ASA is standard initial treatment in mild UC. Mechanism of Action-Inhibits:
• Cyclooxygenase (COX)
• ↓ Prostaglandins

Reduces local inflammation in colon

Severity	Clinical features	Treatment
Mild-moderate	≤4-6 stools/day, minimal systemic signs	5-ASA □
Moderate-severe	Frequent stools + systemic symptoms	Steroids
Severe	Toxic megacolon risk	IV steroids / biologics

QUESTION

Laboratory investigations of a patient being evaluated for jaundice show elevated bilirubin and alkaline phosphatase levels. Levels of the remaining liver enzymes are normal. What is the likely diagnosis?

A **Obstructive jaundice** CORRECT

B Hemolytic jaundice

C Hepatic jaundice

D Prehepatic jaundice

RIGHT ANSWER

OK **A. Obstructive jaundice**

WHY THIS IS RIGHT

ALP is present in:
• Bile duct epithelium
• When bile flow is blocked:
• Pressure builds up
• ALP leaks into blood

So: ↑ ALP = cholestasis / obstruction

AST/ALT are Normal because-
• These are hepatocellular injury markers
• In obstruction:
• Liver cells are initially intact
• -> AST/ALT remain normal or mildly raised

Type of Jaundice	Bilirubin	ALP	AST/ALT
Obstructive	↑↑ (direct)	↑↑↑	Normal/mild ↑
Hepatic	↑	↑	↑↑↑
Hemolytic (Prehepatic)	↑ (indirect)	Normal	Normal

Parameter	Obstructive	Hepatic	Hemolytic
Bilirubin	Direct ↑	Mixed	Indirect ↑
ALP	↑↑↑	Mild ↑	Normal
AST/ALT	Mild ↑	↑↑↑	Normal
Urine bilirubin	Present	Present	Absent

QUESTION

A chronic alcoholic presented with abdominal pain, jaundice and weight loss. He has hypoglycemia and elevated alpha fetoprotein levels (600ng/ml) but has normal AST and ALT. Which of the following is a likely diagnosis of the patient?

A Liver cirrhosis

B Hepatitis

C **Hepatocellular carcinoma** CORRECT

D Cholangiocarcinoma

RIGHT ANSWER

OK

C. Hepatocellular carcinoma

WHY THIS IS RIGHT

• Chronic alcoholic -> chronic liver disease risk
• Abdominal pain + jaundice + weight loss -> malignancy suspicion
• Hypoglycemia -> paraneoplastic clue
• AFP = 600 ng/ml -> tumor marker (normal: 10-25)
• Normal AST/ALT -> not active hepatitis

Put together -> Hepatocellular carcinoma (HCC)

Classic Triad of HCC:
• Weight loss
• Abdominal pain
• Liver mass

Other Paraneoplastic Features:
• Hypoglycemia
• Polycythemia (↑ EPO)
• Hypercalcemia

Imaging Hallmark:
• Arterial phase enhancement + venous washout (CT/MRI)

Feature	HCC	Cirrhosis	Hepatitis	Cholangiocarcinoma
AFP	↑↑	Normal	Normal	Normal
AST/ALT	Normal/slightly ↑	Mild ↑	↑↑	Mild ↑
Hypoglycemia	Yes	No	No	No
Cause	Cirrhosis	Alcohol, viral	Infection	Bile duct malignancy

QUESTION

In a patient who has undergone gastrectomy, vitamin supplementation required is:

A Vit C

B Vit D

C **Vit B12** CORRECT

D Vit A

OK RIGHT ANSWER

C. Vit B12

WHY THIS IS RIGHT

The source quiz contains the placeholder “$27” here and does not include a written explanation for this item.

QUESTION

A young lady presents with jaundice and pruritus. She is antimitochondrial antibody positive and ANA negative. Biopsy shows portal inflammation and ductular proliferation with lymphocytic infiltrate. What is the diagnosis?

A **Primary biliary cholangitis** — CORRECT

B Choledochal cyst

C Autoimmune hepatitis

D Nodular hyperplasia

RIGHT ANSWER

OK

A. Primary biliary cholangitis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$28” here and does not include a written explanation for this item.

QUESTION

A patient with a history of consumption of 2-3 drinks per week comes with complaints of right upper quadrant discomfort. He has grade 2 steatosis. His BMI was 29 kg/m² and AST and ALT levels were slightly elevated. What would you advise him?

A **Lifestyle modification and weight loss** — CORRECT

B Ursodeoxycholic acid and lifestyle modification

C Lifestyle modifications and vitamin E

D Pioglitazone and lifestyle modifications

OK RIGHT ANSWER

A. Lifestyle modification and weight loss

WHY THIS IS RIGHT

The source quiz contains the placeholder “$29” here and does not include a written explanation for this item.

QUESTION

Which of the following medications is most commonly associated with cholestasis in the absence of significant portal inflammation?

A Acetaminophen

B Amoxicillin-clavulanate

C Isoniazid

D **Estrogens/ Androgens** CORRECT

RIGHT ANSWER

OK **D. Estrogens/ Androgens**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$2a” here and does not include a written explanation for this item.

QUESTION

A patient with chronic liver disease presented with jaundice and melena. On examination, there is ascites and mild hepatosplenomegaly. Which of the following is not part of the management of his encephalopathy?

A **Beta blocker to stop variceal bleed** CORRECT

B Ascitic tap to rule out SBP

C Check for HRS

D IV Octreotide

RIGHT ANSWER

OK

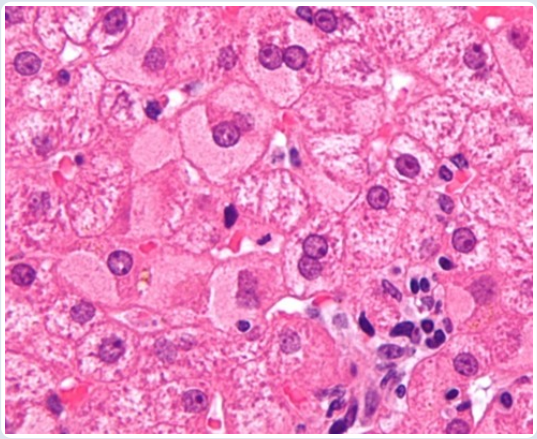
A. Beta blocker to stop variceal bleed

WHY THIS IS RIGHT

The source quiz contains the placeholder “$2b” here and does not include a written explanation for this item.

QUESTION

58-year-old male with a history of multiple sexual partners, presented with Anorexia and Jaundice. Biopsy shows ground-glass hepatocytes. What is the most probable diagnosis?



A **Hepatitis B** — CORRECT

B Hepatitis A

C Hepatitis C

D Hepatitis D

RIGHT ANSWER

OK **A. Hepatitis B**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$2c” here and does not include a written explanation for this item.

QUESTION

Which of the following anti-HIV drugs is effective in treatment of hepatitis B?

A Dolutegravir

B Ritonavir

C **Lamivudine** CORRECT

D Saquinavir

RIGHT ANSWER

OK

C. Lamivudine

WHY THIS IS RIGHT

Lamivudine is a nucleoside reverse transcriptase inhibitor active against both HIV and hepatitis B virus. It inhibits HBV DNA polymerase and is used in chronic HBV therapy. Protease inhibitors and integrase inhibitors have no activity against HBV. Dual antiviral activity is the key exam clue for lamivudine.

Drug	Class	HBV Activity
Lamivudine	NRTI	□
Tenofovir	NRTI	□
Emtricitabine	NRTI	□
Entecavir	HBV drug	□ HIV

QUESTION

A patient presents to you with fever, jaundice, and malaise. What is the most likely diagnosis based on the serology reports given below?

- **Anti-HBc (IgM): Positive**
- **HBsAg: Positive**
- **Anti-HBs: Negative**
- **Anti-HCV antibodies: Negative**

A **Acute hepatitis B** — CORRECT

B Acute hepatitis C

C Chronic hepatitis B

D Chronic hepatitis C

RIGHT ANSWER

OK **A. Acute hepatitis B**

WHY THIS IS RIGHT

Marker	Meaning
HBsAg (+)	Current infection present
Anti-HBc IgM (+)	Acute infection
Anti-HBs (-)	No immunity yet
Anti-HCV (-)	Not hepatitis C

HBsAg positivity with anti-HBc IgM positivity is diagnostic of acute hepatitis B. IgM anti-HBc indicates recent infection and active hepatocellular injury. Absence of anti-HBs confirms the patient has not yet recovered or developed immunity. This serologic pattern is classic for acute HBV infection.

Option B) Acute Hepatitis C
• Would show:

o Anti-HCV positive

Option C) Chronic Hepatitis B
• Would show:

o Anti-HBc IgG, not IgM
o Duration > 6 months

Option D) Chronic Hepatitis C
• Requires:

o Anti-HCV positive

QUESTION

A 30-year-old male is found to be positive for HBsAg and HBeAg and is diagnosed with chronic hepatitis B. The viral load of the patient was 2 x 10^5 IU/mL and ALT is found to be doubled. What is the appropriate management?

A Lamivudine

B **Tenofovir** CORRECT

C Peg IFN for 52 weeks

D Combined peg IFN with lamivudine

RIGHT ANSWER

OK

B. Tenofovir

WHY THIS IS RIGHT

Chronic HBV (HBsAg > 6 months)
↓
Check HBeAg + HBV DNA + ALT
↓
High DNA + ↑ ALT
HBV DNA >2000IU/ml
↓
Start antiviral therapy
↓
Tenofovir + Entecavir x 12wks

Tenofovir:
□ Class: Nucleotide analog (reverse transcriptase inhibitor)
□ Action:
• Inhibits HBV DNA polymerase
• -> Stops viral replication

QUESTION

A 30-year-old man had an episode of jaundice due to HBV one year back. On follow-up now, he has normal levels of liver enzymes. His current profile is shown.

- **HBsAg: Positive**
- **HBeAg: Negative**
- **Anti-HBe: Positive**
- **Anti-HBc IgG: Positive**
- **Anti-HBc IgM: Negative**
- **HBV DNA: Low**
- **ALT: Normal**

A Chronic Hepatitis B

B **Chronic Hepatitis B** CORRECT

C Resolved infection

D Acute Hepatitis B

RIGHT ANSWER

OK **B. Chronic Hepatitis B**

WHY THIS IS RIGHT

Key interpretation of markers:
• HBsAg positive (>1 year) -> Chronic infection
• HBeAg negative + Anti-HBe positive -> Low replication
• Anti-HBc IgG positive, IgM negative -> Chronic (not acute)
• HBV DNA low + ALT normal -> Inactive carrier state

Why not others?
• Resolved infection: HBsAg would be negative, Anti-HBs positive
• Acute HBV: Anti-HBc IgM positive
• Chronic Hep B (active): ↑ ALT, high HBV DNA, often HBeAg +

QUESTION

The blood investigation of a patient is given below. What is the probable diagnosis?

- **HBsAg - NR,**
- **Anti HBs - NR,**
- **Anti HBc IgM - NR,**
- **Anti HBc total - positive,**
- **HBeAg - NR,**
- **Anti HBe - NR (NR - Non reactive)**

A Acute HBV infection in window period

B Immune with recombinant HBV Vaccine

C **HBV infection in the remote past, completely recovered** CORRECT

D Chronic HBV infection inactive carrier

OK RIGHT ANSWER

C. HBV infection in the remote past, completely recovered

WHY THIS IS RIGHT

The source quiz contains the placeholder “$2d” here and does not include a written explanation for this item.

QUESTION

Which of the following viruses spreads predominantly through water?

A HCV

B **HAV (Hepatitis A Virus)** CORRECT

C HDV

D HBV

RIGHT ANSWER

OK **B. HAV (Hepatitis A Virus)**

WHY THIS IS RIGHT

Hepatitis A virus spreads by the fecal-oral route, classically through contaminated water and food. It causes acute, self-limited hepatitis and is linked to poor sanitation and outbreaks. In contrast, HBV, HCV, and HDV are transmitted parenterally via blood or body fluids. Waterborne transmission is a key epidemiologic hallmark of HAV.

Feature	HAV	HBV	HCV	HDV	HEV
Transmission	Feco-oral	Blood/sexual	Blood	Blood	Feco-oral
Chronicity	□	□	□ (high)	□	□ (except pregnancy severe)
Envelope	□	□	□	□	□
Vaccine	□	□	□ (via HBV)	□
Special note	Waterborne outbreaks	Vertical transmission	Silent chronic	Needs HBV	Severe in pregnancy

• HAV & HEV = Enterically transmitted
• HBV, HCV, HDV = Parenteral
• HAV:
• No carrier state
• No chronic hepatitis
• Lifelong immunity after infection

QUESTION

A man presents with deranged LFT. The serum viral markers are given below:

Anti-HAV IgM - Non-reactive
Anti-HAV-IgG - Reactive
HBsAg - Non-reactive
Anti-HBc total/IgG - Reactive
Anti-HBc IgM - Non-reactive
Anti-HCV IgM - Non-reactive
Anti-HCV IgG - Reactive
Anti-HEV IgM - Non-reactive
Anti-HEV IgG - Non-reactive

Which of the following statements is not correct about his condition?

A **Chronic Hepatitis B infection, HBV DNA titres should be done by real time PCR** — CORRECT

B HBV infection in the remote past

C HCV DNA titres should be estimated by RT-PCR

D Past HAV infection or received vaccine for HAV

RIGHT ANSWER

OK **A. Chronic Hepatitis B infection, HBV DNA titres should be done by real time PCR**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$2e” here and does not include a written explanation for this item.

QUESTION

A health worker had a needle stick injury. While handling a chronic Hepatitis B case. She has taken 2 complete schedules (6 doses) of HBV vaccination. She was tested and was diagnosed as a non-responder to vaccination 3 months back, What is the most appropriate management in this case:

A **Give 2 doses of HBIG one now and another 4 weeks later** CORRECT

B Give 2 doses of HBIG now (stat)

C Give HBV vaccination starting now, after 4-6 weeks, check HBV titres and decide on giving HBIG

D HBIG single dose with HBV vaccination (first dose beginning now)

OK RIGHT ANSWER

A. Give 2 doses of HBIG one now and another 4 weeks later

WHY THIS IS RIGHT

A documented vaccine non-responder exposed to an HBsAg-positive source requires passive immunization only. Two doses of HBIG are given: one immediately and another at 1 month, as further vaccination is ineffective in proven non-responders. HBIG provides immediate anti-HBs protection after high-risk exposure. Revaccination strategies are used only in unknown or incomplete responders, not confirmed non-responders.

Criteria	Meaning
Anti-HBs <10 mIU/mL	No protective immunity
After 2 full vaccine series	True non-responder

Non-responder : anti-HBs < 10 after 2 full series
What to do: HBIG × 2 doses: 4 weeks apart; No further vaccine