Gastrointestinal System

United States Medical Licensing Examination (USMLE) Step 1

6 to 10% of Step 1 · 8 sections · content outline and specifications

This system covers the mouth to the anus, the liver, the biliary tree and the pancreas. Step 1 questions often begin with a patient's symptoms and liver or electrolyte results, then ask for a mechanism, a hormone, an organism or an embryologic error. Physiology items test GI hormones, digestion, absorption and bilirubin handling. Pathology items test ulcers, inflammatory bowel disease, polyps and cancer syndromes, hepatitis, cirrhosis and pancreatitis. Microbiology and pharmacology items test toxin mechanisms, parasites and drug injury to the liver and gut.

You should be able to: After this guide you should be able to link GI symptoms and liver or electrolyte results to the disorder, its mechanism and the organism, gene or drug behind it.

Every section: the key point and the trap

1 GI physiology and hormones

Gastrin (G cells, antrum): increases acid. It rises with protein, stomach stretch and vagal input; acid in the stomach lowers it.

Trap: Secretin and cholecystokinin both act on the pancreas. Secretin brings bicarbonate (water); cholecystokinin brings enzymes.

2 Esophagus and stomach

Achalasia: loss of inhibitory neurons (nitric oxide, VIP) in the myenteric plexus; the lower sphincter does not relax; bird's beak on barium swallow.

Trap: A high gastrin also occurs with proton pump inhibitor use and atrophic gastritis. In those, stomach acid is low, not high.

3 Small and large intestine: absorption, inflammation and motility

Celiac disease: gluten, HLA-DQ2/DQ8, IgA anti-tissue transglutaminase, villous atrophy in the duodenum, dermatitis herpetiformis.

Trap: Fistulas and perianal disease point to Crohn disease. Continuous disease from the rectum and toxic megacolon point to ulcerative colitis.

4 GI infections and toxins

Preformed toxin (vomiting within 1 to 6 hours): Staphylococcus aureus, Bacillus cereus (fried rice).

Trap: Fever and blood point to invasion of the mucosa. Large watery stool without fever points to a toxin acting on enterocytes.

5 Polyps and GI cancers

Adenoma-carcinoma sequence: APC loss, then KRAS activation, then TP53 loss.

Trap: Many polyps in a young person does not always mean APC. Hamartomas with lip pigment point to Peutz-Jeghers, and recessive inheritance points to MUTYH.

6 Liver: bilirubin, hepatitis and cirrhosis

Unconjugated bilirubin rises with hemolysis, Gilbert syndrome (mild UGT1A1 reduction) and Crigler-Najjar syndrome (absent or very low UGT1A1).

Trap: Do not call every high bilirubin liver failure. First decide if it is conjugated or unconjugated, then check the enzymes.

7 Biliary tract and pancreas

Cholesterol stones form when bile has too much cholesterol for its bile salts and phospholipids; risks are female sex, obesity, rapid weight loss and pregnancy.

Trap: Primary biliary cholangitis and primary sclerosing cholangitis are easy to mix up. Small ducts and antimitochondrial antibodies: biliary. Large beaded ducts and ulcerative colitis: sclerosing.

8 Congenital and mechanical disorders and drug effects

Pyloric stenosis: projectile nonbilious vomiting at 2 to 6 weeks, olive mass, hypochloremic hypokalemic metabolic alkalosis.

Trap: Bilious vomiting in a newborn points below the ampulla (atresia, malrotation). Nonbilious projectile vomiting at a few weeks points to pyloric stenosis.

Key terms

Enterohepatic circulation: The cycle in which bile salts leave the liver, are absorbed in the ileum and return to the liver.

Conjugated bilirubin: Bilirubin joined to glucuronic acid in the liver so it can dissolve in bile and urine.

SAAG: Serum albumin minus ascitic fluid albumin; 1.1 g/dL or more points to portal hypertension.

Steatorrhea: Greasy, bulky stool with too much fat.

Hamartoma: A growth made of normal tissue types in a disorganized pattern.

Osmotic gap: Measured stool osmolality minus twice the stool sodium and potassium.

Transmural: Through the whole thickness of the bowel wall.

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