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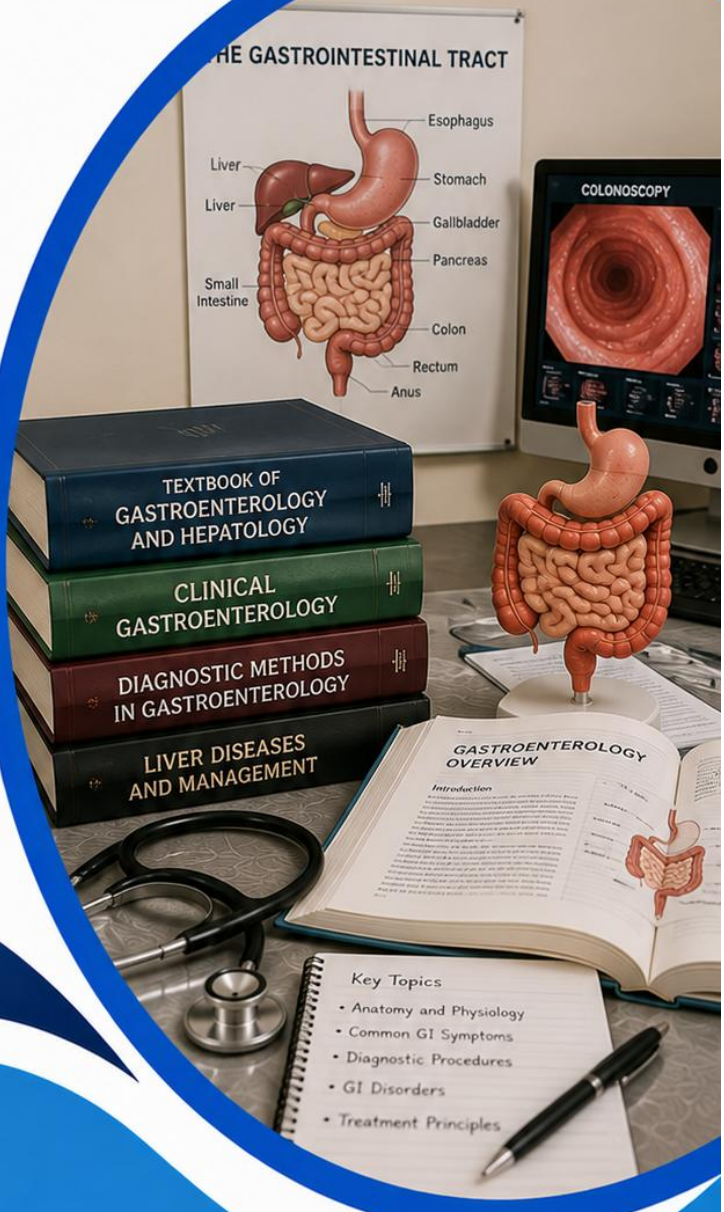
MED 404

GASTROENTEROLOGY I



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Course code: MED 404

Course title: Gastroenterology I

Course credit load: 2 Units

Series 1: Evaluation of Gastrointestinal Symptoms

- **Part 1.1: Approach to Abdominal Pain**
    - Sub Part 1.1.1: Mechanisms and classification of abdominal pain
    - Sub Part 1.1.2: History taking and physical examination
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    - Sub Part 1.2.1: Upper gastrointestinal haemorrhage
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- **Part 1.4: Approach to Weight Loss and Abdominal Swelling**
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    - Sub Part 1.4.3: Investigation of weight loss and abdominal swelling

Introduction

The ability to evaluate gastrointestinal symptoms accurately is one of the most important clinical skills in internal medicine and gastroenterology. In Nigeria, gastrointestinal complaints account for a significant proportion of outpatient consultations and emergency presentations. Conditions ranging from peptic ulcer disease and viral hepatitis to intestinal obstruction and colorectal malignancy often begin with the same cluster of symptoms, making a disciplined, systematic approach essential for reaching the correct diagnosis without delay.

The aim of this Series is to equip you with a structured clinical framework for evaluating the four major gastrointestinal symptom complexes encountered in practice. We shall commence this Series by examining abdominal pain, exploring its mechanisms, structured history, physical examination, and investigation pathway. Next, we will explore gastrointestinal bleeding, distinguishing upper from lower GI sources and describing the immediate management. Moving on, we will consider the clinical approach to jaundice, from its pathophysiological classification to the investigation pathway. Finally, we will conclude by addressing unintentional weight loss and abdominal swelling, including the assessment and investigation of ascites.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 1.1:** describe the mechanisms and classification of abdominal pain,
- **SLO 1.2:** conduct a systematic clinical assessment of a patient with abdominal pain,
- **SLO 1.3:** distinguish between upper and lower gastrointestinal bleeding and describe the clinical features of each,
- **SLO 1.4:** outline the investigation and initial management of gastrointestinal bleeding,
- **SLO 1.5:** classify jaundice according to its pathophysiological mechanism,
- **SLO 1.6:** describe the clinical assessment and investigation of the jaundiced patient,

- **SLO 1.7:** define clinically significant unintentional weight loss and identify its gastrointestinal causes in Nigeria, and
- **SLO 1.8:** describe the clinical approach to abdominal swelling and ascites including the serum-ascites albumin gradient.

1.1 Approach to Abdominal Pain

1.1.1 Mechanisms and classification of abdominal pain

Abdominal pain arises through three distinct mechanisms. **Visceral pain** is produced by distension, spasm, or ischaemia of a hollow viscus such as the bowel, gallbladder, or ureter. Because visceral afferent nerve fibres from both sides of the body converge at the same spinal level, this pain is poorly localised and felt diffusely in the midline, described as dull, cramping, or colicky in character. **Parietal pain** arises from direct irritation of the parietal peritoneum, which is richly supplied by somatic nerve fibres, making it sharp, well localised, and worsened by movement or coughing. **Referred pain** is perceived at a site distant from the diseased organ due to shared spinal nerve roots, as in right shoulder tip pain from diaphragmatic irritation in biliary disease.

Abdominal pain is further classified by onset and location. Sudden onset suggests a surgical emergency such as hollow viscus perforation or mesenteric vascular occlusion, while gradual onset suggests an inflammatory or obstructive process. Location provides important diagnostic clues: right hypochondrial pain points to biliary or hepatic pathology, epigastric pain to peptic ulcer disease or pancreatitis, periumbilical pain migrating to the right iliac fossa is the classic evolution of appendicitis, and left iliac fossa pain suggests colonic disease such as diverticulitis or colitis.

_____ pain is poorly localised and felt in the midline because visceral afferent fibres are bilateral, while _____ pain is sharp and well localised because the parietal peritoneum is supplied by somatic nerve fibres.

1.1.2 History taking and physical examination

A thorough history of abdominal pain follows the SOCRATES framework: Site (where the pain is worst), Onset (sudden suggests perforation or vascular catastrophe; gradual suggests inflammation), Character (colicky or constant), Radiation (to the back suggesting pancreatitis or aortic aneurysm; to the right shoulder suggesting biliary disease), Associations (nausea, vomiting, fever, change in bowel habit, jaundice, urinary symptoms), Timing (continuous or intermittent), Exacerbating and relieving factors, and Severity. The past medical and drug history must include previous abdominal surgery, peptic ulcer disease, inflammatory bowel disease, sickle cell disease, and use of non-steroidal anti-inflammatory drugs. In women of reproductive age, the gynaecological history is essential to exclude ectopic pregnancy and pelvic inflammatory disease.

Physical examination begins with general inspection for pallor, jaundice, and diaphoresis, followed by vital signs. Tachycardia, hypotension, and fever indicate severity and the need for urgent intervention. Abdominal palpation identifies **guarding** (voluntary resistance), involuntary **rigidity** (board-like firmness indicating generalised peritonitis), and **rebound tenderness** (pain on sudden release of pressure indicating peritoneal irritation). Specific signs include **Murphy's sign** (arrest of inspiration on right hypochondrial palpation, indicating cholecystitis), **Rovsing's sign** (right iliac fossa pain on left iliac fossa pressure, indicating appendicitis), and the **succussion splash** of intestinal obstruction. A digital rectal examination is mandatory in all patients with acute abdominal pain.

In the SOCRATES framework, radiation of abdominal pain to the _____ suggests pancreatitis, while radiation to the right _____ tip suggests biliary disease.

1.1.3 Investigation of abdominal pain

First-line investigations include a full blood count (leucocytosis in infection or inflammation), C-reactive protein and erythrocyte sedimentation rate, serum amylase and lipase (elevated in pancreatitis), liver function tests, urine dipstick, and a pregnancy test in all women of reproductive age. These investigations rapidly narrow the differential diagnosis and guide further imaging.

Imaging begins with a plain abdominal X-ray, which identifies dilated bowel in obstruction and, on an erect chest X-ray, free gas under the diaphragm in perforation. Abdominal ultrasound is the first-line modality for hepatobiliary and renal pathology. CT of the abdomen and pelvis is the most sensitive investigation for most acute abdominal emergencies and is used when the diagnosis remains uncertain after initial assessment.

Free gas under the right hemidiaphragm on an erect chest X-ray indicates bowel _____. Serum _____ and lipase are the key biochemical markers for pancreatitis.

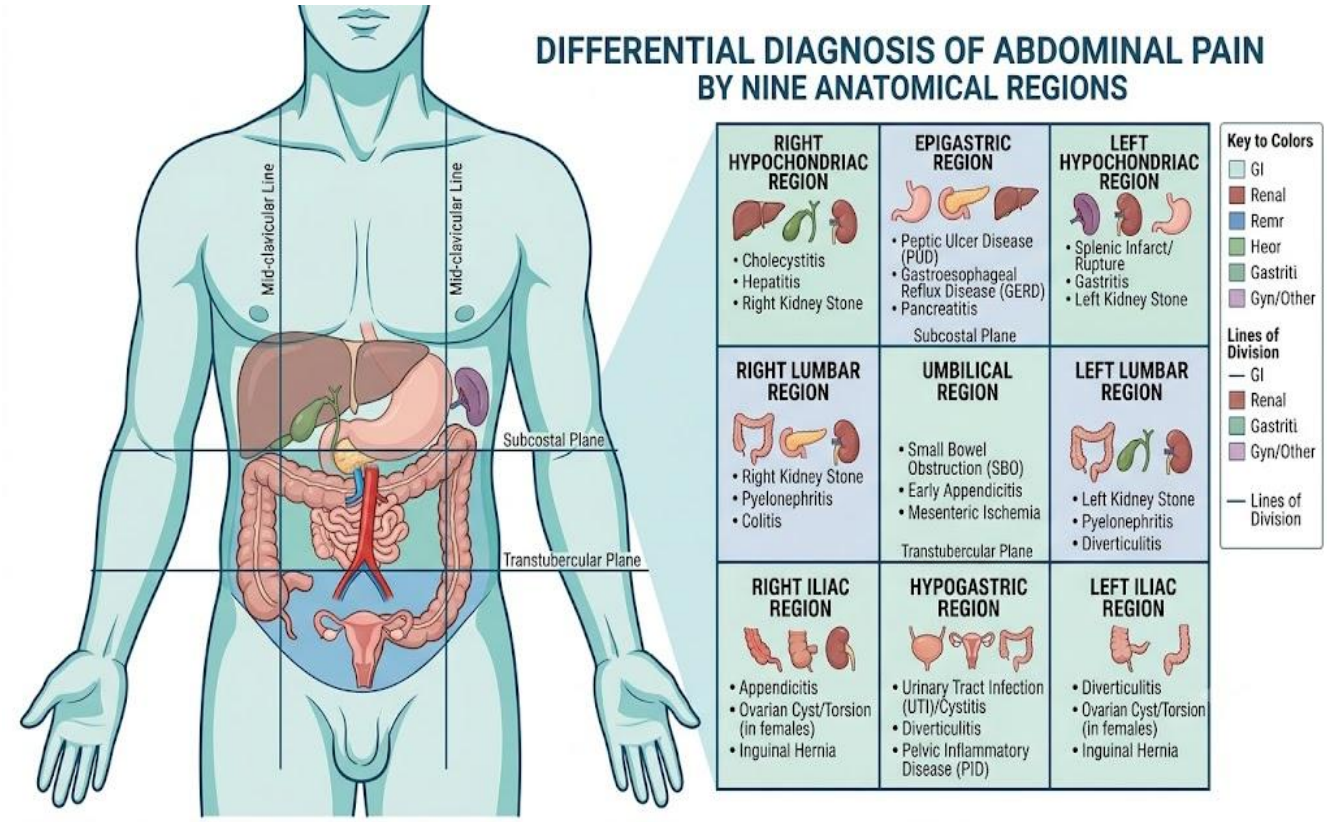

Figure 1.1: Regional differential diagnosis of abdominal pain

Pilot questions 1.1

1. Describe the three mechanisms of abdominal pain. (Linked to SLO 1.1)
2. State two features that distinguish parietal pain from visceral pain. (Linked to SLO 1.1)

3. Describe four components of the SOCRATES framework as applied to abdominal pain. (Linked to SLO 1.2)
4. State the significance of Murphy's sign and Rovsing's sign. (Linked to SLO 1.2)
5. Name two radiological findings that indicate a surgical emergency in acute abdominal pain. (Linked to SLO 1.2)

1.2 Approach to Gastrointestinal Bleeding

1.2.1 Upper gastrointestinal haemorrhage

Upper gastrointestinal (UGI) haemorrhage is defined as bleeding from a source proximal to the ligament of Treitz. It presents as **haematemesis**, which is the vomiting of blood that is bright red when bleeding is active or coffee-ground when blood has been partially digested, or as **melaena**, which is the passage of black, tarry, foul-smelling stools from degradation of blood during GI transit. The most common causes in Nigeria are **peptic ulcer disease** (the most frequent cause overall), **oesophageal varices** from portal hypertension in liver cirrhosis (the most life-threatening cause), Mallory-Weiss tears, oesophagitis, and gastric malignancy.

The clinical assessment must rapidly identify haemodynamic compromise: a heart rate above 100 beats per minute, systolic blood pressure below 100 mmHg, and postural dizziness indicate significant blood loss requiring immediate resuscitation. The Glasgow-Blatchford score, using blood urea, haemoglobin, blood pressure, pulse rate, and clinical features, stratifies risk and guides the urgency of endoscopy. A markedly elevated blood urea to creatinine ratio is a biochemical clue to upper GI bleeding, as digested blood is absorbed and converted to urea.

Upper GI haemorrhage presents as _____ or melaena. The most life-threatening cause in a patient with chronic liver disease is bleeding from oesophageal _____.

1.2.2 Lower gastrointestinal bleeding

Lower gastrointestinal (LGI) bleeding originates distal to the ligament of Treitz and presents as **haematochezia**, the passage of bright red or maroon blood per rectum. In young adults in Nigeria, the most common causes are **haemorrhoids** (the most common cause overall), anal fissures, and infectious colitis from bacterial dysentery or amoebic colitis. In patients above 40 years, **colorectal carcinoma** must always be excluded, alongside diverticular disease and angiodysplasia.

Blood coating the outside of the stool suggests an anal or low rectal source, whereas blood mixed within the stool suggests a colonic source. Massive upper GI bleeding may also present as haematochezia when colonic transit is rapid, so haemodynamic status and the urea to creatinine ratio help distinguish lower from upper GI bleeding in ambiguous presentations.

The most common cause of lower GI bleeding in young Nigerian adults is _____. Blood _____ the outside of the stool suggests an anal or low rectal source.

1.2.3 Investigation and initial management

Immediate management follows the ABCDE approach. Two large-bore intravenous cannulae are inserted and blood is taken for full blood count, coagulation screen, group and crossmatch, urea and electrolytes, and liver function tests. Intravenous crystalloid resuscitation is commenced while awaiting blood products. Packed red cell transfusion is given when haemoglobin falls below 70 grams per litre (or 80 grams per litre in cardiovascular disease), fresh frozen plasma when the INR exceeds 1.5, and platelets when the count falls below 50 times ten to the nine per litre.

Upper GI endoscopy performed within 24 hours is the investigation of choice for UGI haemorrhage, allowing therapeutic intervention in the same procedure: adrenaline injection and haemoclip placement for peptic ulcer bleeding, and band ligation for oesophageal varices. Colonoscopy is the first-line investigation for stable LGI bleeding, while CT angiography is used when active bleeding is suspected and endoscopy is not immediately feasible.

Upper GI endoscopy is performed within _____ hours of presentation. Packed red cell transfusion is given when haemoglobin falls below _____ grams per litre.

Feature	Upper GI Bleeding	Lower GI Bleeding
Source	Proximal to ligament of Treitz: **esophagus, stomach, duodenum**	Distal to ligament of Treitz: **small intestine, colon, rectum, anus**
Presentation	- **Hematemesis** (vomiting blood, bright red or coffee-ground) - **Melena** (black, tarry stools) - Elevated **BUN:Cr ratio** due to protein absorption	- **Hematochezia** (fresh red or maroon blood per rectum) - Occasionally melena if right-sided colonic bleed - Normal **BUN:Cr ratio**
Common Causes in Nigeria	- **Peptic ulcer disease** (most frequent, linked to H. pylori and NSAID use) - **Esophageal varices** (from portal hypertension due to cirrhosis, often hepatitis B/C related) - **Gastritis/esophagitis** - **Mallory-Weiss tear**	- **Diverticular disease** (increasing with aging population) - **Colorectal cancer** (rising incidence in Nigeria) - **Hemorrhoids** (very common locally) - **Inflammatory bowel disease** (less common but present) - **Angiodysplasia, ischemic colitis**
Investigation	- **Esophagogastroduodenoscopy (EGD)** within 24 hours - **CT angiography** if ongoing brisk bleeding - Labs: CBC, coagulation profile, liver function tests	- **Colonoscopy** (diagnostic and therapeutic) - **CT angiography** or **capsule endoscopy** for small bowel source - Labs: CBC, coagulation profile, stool occult blood

Table 1.1: Comparison of upper and lower gastrointestinal bleeding

Pilot questions 1.2

1. Define haematemesis and melaena and state which part of the GI tract each originates from. (Linked to SLO 1.3)

2. State three causes of upper GI haemorrhage in Nigeria in order of frequency. (Linked to SLO 1.3)
3. State three causes of lower GI bleeding in young adults in Nigeria. (Linked to SLO 1.3)
4. Describe two clinical features of haemodynamic compromise in GI bleeding. (Linked to SLO 1.4)
5. State the investigation of choice for upper GI haemorrhage and two therapeutic procedures it allows. (Linked to SLO 1.4)

1.3 Approach to Jaundice

1.3.1 Classification and pathophysiology of jaundice

Jaundice is the yellow discolouration of the skin, sclerae, and mucous membranes from an elevated serum bilirubin, which becomes clinically apparent above approximately 35 to 50 micromoles per litre. It is classified into three types. **Pre-hepatic jaundice** results from excessive bilirubin production due to haemolysis that overwhelms the liver's conjugating capacity: serum unconjugated bilirubin is elevated, urine is normal (unconjugated bilirubin is not water-soluble), and stools are dark from excess urobilinogen. Common causes in Nigeria include sickle cell disease, malaria, and glucose-6-phosphate dehydrogenase deficiency.

Hepatic (hepatocellular) jaundice results from liver cell damage impairing both conjugation and excretion, elevating both conjugated and unconjugated bilirubin. Common causes include viral hepatitis, alcoholic liver disease, and drug-induced liver injury. **Post-hepatic (obstructive) jaundice** results from obstruction of bile flow: conjugated bilirubin accumulates in blood and is excreted in the urine (dark urine), while reduced bile in the bowel produces pale or acholic stools. Important causes include gallstones in the common bile duct, carcinoma of the head of the pancreas, cholangiocarcinoma, and biliary stricture.

Pre-hepatic jaundice produces _____ urine because unconjugated bilirubin is not water-soluble. Post-hepatic jaundice produces dark urine and _____ stools because bile cannot reach the bowel.

1.3.2 Clinical assessment of the jaundiced patient

The history in jaundice explores onset and progression (acute in viral hepatitis and haemolysis; gradual and painless in malignancy), fever and rigors (which together with right upper quadrant pain and jaundice form Charcot's triad of ascending cholangitis), dark urine and pale stools (indicating obstructive jaundice), and pruritus from bile salt deposition in the skin. Weight loss and progressive jaundice raise strong suspicion for malignancy. The history must also cover alcohol intake, use of conventional drugs and traditional herbal medicines, blood transfusions, sexual history, and travel history.

Examination in hepatocellular jaundice reveals tender hepatomegaly and **stigmata of chronic liver disease** including spider naevi, palmar erythema, leuconychia, gynaecomastia, and caput medusae. In obstructive jaundice, **Courvoisier's law** applies: a palpable, non-tender, smooth gallbladder in the presence of jaundice suggests malignant obstruction of the common bile duct rather than gallstones, because chronic gallstone disease causes a fibrosed, shrunken gallbladder that cannot distend. Scratch marks from pruritus confirm cholestasis.

The combination of fever, right upper quadrant pain, and jaundice is called _____ triad and indicates ascending cholangitis. A palpable non-tender gallbladder with jaundice is explained by _____ law and indicates malignant biliary obstruction.

1.3.3 Investigation and differential diagnosis

Liver function tests reveal the pattern of jaundice: markedly elevated alanine aminotransferase and aspartate aminotransferase with mildly elevated alkaline phosphatase suggests a hepatocellular pattern, while markedly elevated alkaline phosphatase and gamma-glutamyl transferase with mildly elevated transaminases suggests a cholestatic pattern. Bilirubin fractionation, urine bilirubin and urobilinogen, full blood count with blood film (haemolysis,

sickle cells, malaria parasites), coagulation screen, and viral hepatitis serology complete the initial investigation.

Abdominal ultrasound is the first-line imaging investigation, identifying dilated bile ducts in obstruction, gallstones, liver parenchymal changes, and masses. Magnetic resonance cholangiopancreatography (MRCP) is the investigation of choice for suspected bile duct obstruction, providing detailed non-invasive imaging of the biliary tree. Endoscopic retrograde cholangiopancreatography (ERCP) is reserved for therapeutic biliary decompression such as stone extraction or stent insertion.

A markedly elevated alkaline phosphatase and gamma-glutamyl transferase suggests a _____ pattern of jaundice. The non-invasive imaging of choice for suspected bile duct obstruction is _____.

Type	Mechanism and Causes	Urine and Stool Changes & Key Investigation
Pre-hepatic	- Excessive hemolysis → ↑ unconjugated bilirubin - Causes: Hemolytic anemias, malaria (common in Nigeria), sickle cell disease, G6PD deficiency	- Urine: Normal (no bilirubin, ↑ urobilinogen) - Stool: Darker (↑ stercobilin) - Key Investigation: Peripheral blood smear, reticulocyte count, LDH
Hepatic	- Impaired conjugation or excretion of bilirubin due to hepatocellular dysfunction - Causes: Viral hepatitis (HBV, HCV), alcoholic liver disease, drug-induced hepatitis, cirrhosis	- Urine: Dark (↑ conjugated bilirubin) - Stool: Variable (may be pale if severe cholestasis) - Key Investigation: Liver function tests (ALT, AST, ALP, bilirubin), viral serology
Post-hepatic	- Obstruction of bile flow → cholestatic jaundice - Causes: Gallstones, biliary strictures, pancreatic cancer, cholangiocarcinoma	- Urine: Dark (↑ conjugated bilirubin, absent urobilinogen) - Stool: Pale/clay-colored (↓ stercobilin) - Key Investigation: Abdominal ultrasound, MRCP/ERCP, ALP markedly raised

Table 1.2: Classification of jaundice by mechanism, causes, and clinical and investigative features

Pilot questions 1.3

1. Define jaundice and state the serum bilirubin level at which it becomes clinically apparent. (Linked to SLO 1.5)
2. Distinguish pre-hepatic from post-hepatic jaundice in terms of bilirubin type elevated and urine and stool changes. (Linked to SLO 1.5)
3. State three causes of hepatocellular jaundice common in Nigeria. (Linked to SLO 1.6)
4. Explain Courvoisier's law and state what it indicates clinically. (Linked to SLO 1.6)
5. State the investigation of choice for suspected bile duct obstruction and explain why it is preferred. (Linked to SLO 1.6)

1.4 Approach to Weight Loss and Abdominal Swelling

1.4.1 Evaluation of unintentional weight loss

Unintentional weight loss is defined as the loss of more than 5 percent of usual body weight over 6 to 12 months without deliberate dietary restriction or increased physical activity, and is always clinically significant. Gastrointestinal causes in Nigeria include **tuberculosis** (the most important infective cause to exclude, presenting with night sweats, cough, and haemoptysis), **HIV-related enteropathy** and opportunistic GI infections, **malabsorption syndromes** (tropical sprue, coeliac disease, chronic pancreatitis causing steatorrhoea), and **gastrointestinal malignancy** affecting the oesophagus, stomach, or colorectum.

The history distinguishes reduced caloric intake (from anorexia, dysphagia, or odynophagia), increased caloric loss (from diarrhoea, steatorrhoea, or vomiting), and increased metabolic demand (from fever, infection, or malignancy). Red flag symptoms demanding urgent investigation include dysphagia (suggesting oesophageal carcinoma), haematemesis or melaena

(suggesting gastric malignancy), a change in bowel habit in a patient above 40 years (suggesting colorectal carcinoma), haemoptysis and night sweats (suggesting tuberculosis), and a palpable abdominal mass.

Unintentional weight loss is clinically significant when it exceeds _____ percent of body weight over 6 to 12 months. The most important infective cause to exclude in Nigeria is _____.

1.4.2 Abdominal swelling and ascites

Abdominal swelling has multiple causes recalled by the mnemonic of the five Fs: Fat (obesity), Fluid (ascites), Flatus (gaseous distension from obstruction or ileus), Faeces (faecal loading from constipation or large bowel obstruction), and Foetus (pregnancy, which must be considered in all women of reproductive age). Ascites is confirmed clinically by shifting dullness on percussion and a fluid thrill in large collections.

The serum-ascites albumin gradient (SAAG) is calculated by subtracting the ascitic fluid albumin from the serum albumin. A SAAG of 11 grams per litre or above indicates **portal hypertension**: the most common cause in Nigeria is liver cirrhosis from chronic hepatitis B or C or alcoholic liver disease, followed by right heart failure and Budd-Chiari syndrome. A SAAG below 11 grams per litre indicates an exudative cause such as **peritoneal carcinomatosis**, **tuberculous peritonitis**, or nephrotic syndrome.

A serum-ascites albumin gradient of _____ grams per litre or above indicates portal hypertension. The most common cause of ascites in Nigeria is liver _____.

1.4.3 Investigation of weight loss and abdominal swelling

Investigation of unintentional weight loss includes a full blood count (anaemia of chronic disease; eosinophilia for parasitic infection), erythrocyte sedimentation rate and C-reactive protein, sputum for acid-fast bacilli and a chest X-ray (tuberculosis), thyroid function tests, blood glucose, and stool microscopy and culture. Upper and lower GI endoscopy with biopsy is indicated when malignancy or malabsorption is suspected.

Diagnostic paracentesis is the most important investigation in ascites: approximately 10 to 20 mL of ascitic fluid is aspirated from the left iliac fossa under ultrasound guidance and sent for albumin (to calculate the SAAG), total protein, cell count (a **neutrophil count above 250 cells per microlitre** indicates spontaneous bacterial peritonitis requiring immediate antibiotics), cytology (for malignant cells), Gram stain and bacterial culture, and **adenosine deaminase (ADA)** with AFB culture for tuberculous peritonitis, in which ADA is elevated above 30 to 40 units per litre.

A neutrophil count above _____ cells per microlitre in ascitic fluid indicates spontaneous bacterial peritonitis. Adenosine deaminase is measured to investigate for _____ peritonitis.

Clinical Assessment	Diagnostic Paracentesis
- Five Fs: Fat, Fluid, Flatus, Feces, Fetus (differential for abdominal distension) - Clinical confirmation of ascites: shifting dullness, fluid thrill, puddle sign - SAAG classification (Serum-Ascites Albumin Gradient): • High SAAG (>1.1 g/dL) → portal hypertension causes (cirrhosis, cardiac failure, Budd–Chiari syndrome) • Low SAAG (<1.1 g/dL) → non-portal causes (peritoneal carcinomatosis, tuberculosis, pancreatitis, nephrotic syndrome)	- **Technique**: bedside paracentesis under aseptic conditions, preferably with ultrasound guidance; needle insertion in left lower quadrant avoiding vessels - **Investigations and significance**: • **Cell count & differential** → neutrophils >250/mm³ suggest spontaneous bacterial peritonitis • **Protein concentration** → helps differentiate transudate vs exudate • **Culture & sensitivity** → guides antibiotic therapy • **Cytology** → detects malignant cells • **Amylase** → elevated in pancreatic ascites • **ADA (adenosine deaminase)** → elevated in tuberculous ascites

Table 1.3: Clinical assessment of ascites and investigations performed on ascitic fluid

Pilot questions 1.4

1. Define clinically significant unintentional weight loss. (Linked to SLO 1.7)
2. State three gastrointestinal causes of unintentional weight loss in Nigeria. (Linked to SLO 1.7)

3. List the five Fs of abdominal swelling. (Linked to SLO 1.8)
4. Explain the SAAG and state what a value above 11 grams per litre indicates. (Linked to SLO 1.8)
5. Name three investigations performed on ascitic fluid and state the significance of each. (Linked to SLO 1.8)

Series Summary

This Series has provided you with a structured clinical framework for evaluating the four major gastrointestinal symptom complexes. We began by examining abdominal pain, exploring its three mechanisms of visceral, parietal, and referred pain, and applying the SOCRATES framework to history taking, followed by the key physical signs and the investigation pathway including plain X-ray, ultrasound, and computed tomography. Next, we explored gastrointestinal bleeding, distinguishing upper GI haemorrhage presenting as haematemesis or melaena from lower GI bleeding presenting as haematochezia, and described the immediate resuscitation approach and the role of endoscopy. Moving on, we examined jaundice, classifying it as pre-hepatic, hepatic, and post-hepatic, describing the clinical assessment including Charcot's triad and Courvoisier's law, and outlining the investigation pathway from liver function tests to MRCP. Finally, we addressed unintentional weight loss and abdominal swelling, including the five Fs mnemonic, the SAAG and its diagnostic significance, and the investigations performed on ascitic fluid.

By completing this Series, you should now be able to describe the mechanisms and classification of abdominal pain and conduct a systematic clinical assessment, distinguish between upper and lower gastrointestinal bleeding and describe the immediate management, classify jaundice and outline the appropriate investigation pathway, and describe the clinical approach to unintentional weight loss and ascites including the SAAG. These abilities align with the Series Learning Outcomes (SLOs 1.1 to 1.8) and equip you with the clinical reasoning skills needed to evaluate the gastrointestinal patient systematically in the Nigerian clinical setting.

Glossary of Terms

- **Adenosine deaminase (ADA):** an enzyme measured in ascitic fluid that is elevated above 30 to 40 units per litre in tuberculous peritonitis, making it a useful diagnostic marker for this condition.

- **Ascites:** an abnormal accumulation of fluid in the peritoneal cavity, confirmed clinically by shifting dullness and a fluid thrill on abdominal examination.
- **Charcot's triad:** the combination of fever, right upper quadrant pain, and jaundice, which is the classical presentation of ascending cholangitis.
- **Courvoisier's law:** a palpable, non-tender gallbladder in the presence of jaundice suggests malignant obstruction of the common bile duct rather than gallstones, because gallstone disease causes a fibrosed, shrunken gallbladder that cannot distend.
- **Haematemesis:** the vomiting of blood from a source in the upper gastrointestinal tract proximal to the ligament of Treitz.
- **Haematochezia:** the passage of bright red or maroon blood per rectum, usually indicating a lower gastrointestinal source of bleeding.
- **Melaena:** the passage of black, tarry, foul-smelling stools from degradation of blood during transit through the upper gastrointestinal tract.
- **Murphy's sign:** arrest of inspiration on deep palpation of the right hypochondrium, indicating acute cholecystitis.
- **Parietal pain:** sharp, well-localised abdominal pain from irritation of the parietal peritoneum, worsened by movement or coughing.
- **Pre-hepatic jaundice:** jaundice from excessive bilirubin production due to haemolysis, characterised by elevated unconjugated bilirubin and normal urine colour.
- **Serum-ascites albumin gradient (SAAG):** the difference between serum albumin and ascitic fluid albumin; a value of 11 grams per litre or above indicates portal hypertension as the cause of ascites.
- **Visceral pain:** dull, poorly localised abdominal pain from distension, spasm, or ischaemia of a hollow viscus, felt in the midline because visceral afferent fibres are bilateral.

Concept Check Questions [Series 1]

Objective questions

1. The type of abdominal pain that is poorly localised and felt in the midline because visceral afferent fibres are bilateral is called _____ pain. (Linked to SLO 1.1)
2. The passage of black, tarry, foul-smelling stools from digested blood in the upper GI tract is called _____. (Linked to SLO 1.3)
3. Post-hepatic jaundice produces dark urine because _____ bilirubin, which is water-soluble, spills into the urine. (Linked to SLO 1.5)
4. A serum-ascites albumin gradient of 11 grams per litre or above indicates _____ hypertension as the cause of ascites. (Linked to SLO 1.8)
5. A neutrophil count above 250 cells per microlitre in ascitic fluid indicates spontaneous _____ peritonitis. (Linked to SLO 1.8)

Short-answer questions

6. Describe the difference between visceral pain and parietal pain. (Linked to SLO 1.1)
7. State two clinical features that indicate haemodynamic compromise in a patient with GI bleeding. (Linked to SLO 1.4)
8. Explain Courvoisier's law and state its clinical significance. (Linked to SLO 1.6)
9. Define clinically significant unintentional weight loss. (Linked to SLO 1.7)
10. State two investigations performed on ascitic fluid and what each detects. (Linked to SLO 1.8)

Long-answer questions

11. Describe the classification of jaundice, the clinical assessment of the jaundiced patient, and the investigation pathway. (Linked to SLOs 1.5 and 1.6)

12. Discuss the approach to a patient with acute upper gastrointestinal haemorrhage, including the immediate management and the role of endoscopy. (Linked to SLOs 1.3 and 1.4)
13. Describe the clinical assessment of abdominal pain, including the mechanisms of pain, the structured history, and the key physical signs. (Linked to SLOs 1.1 and 1.2)
14. Explain the serum-ascites albumin gradient, state what different values indicate, and describe the investigations performed on ascitic fluid. (Linked to SLO 1.8)
15. Describe the approach to a patient with unintentional weight loss in Nigeria, including the definition, the differential diagnoses, and the investigation pathway. (Linked to SLO 1.7)

Answers to Concept Check Questions [Series 1]

Objective questions

1. Visceral.
2. Melaena.
3. Conjugated.
4. Portal.
5. Bacterial.

Short-answer questions

6. Visceral pain arises from distension, spasm, or ischaemia of a hollow viscus and is dull, cramping, and poorly localised to the midline because visceral afferent fibres from both sides of the body converge at the same spinal level. Parietal pain arises from irritation of the parietal peritoneum, is sharp and well localised to the site of irritation, and is worsened by movement or coughing because the parietal peritoneum is supplied by somatic nerve fibres.
7. A heart rate above 100 beats per minute is an early sign of significant blood loss and indicates the need for urgent resuscitation. A systolic blood pressure below 100 millimetres of mercury, particularly with postural dizziness and cold peripheries, indicates established haemodynamic compromise requiring immediate blood transfusion.
8. Courvoisier's law states that a palpable, non-tender, smooth gallbladder in the presence of jaundice suggests malignant obstruction of the common bile duct rather than gallstones. It is clinically significant because it directs investigation towards a malignant cause such as carcinoma of the head of the pancreas or cholangiocarcinoma, which require entirely different management from gallstone disease.
9. Clinically significant unintentional weight loss is the loss of more than 5 percent of the patient's usual body weight over 6 to 12 months without deliberate dietary restriction or increased physical activity.

10. Cell count detects a neutrophil count above 250 cells per microlitre, which diagnoses spontaneous bacterial peritonitis and requires immediate intravenous antibiotics. Cytology of the fluid is examined for malignant cells, which confirm peritoneal carcinomatosis from an underlying primary tumour.

Long-answer questions

11. **Basic response:** Jaundice is classified as pre-hepatic, hepatic, and post-hepatic. Pre-hepatic jaundice results from haemolysis causing elevated unconjugated bilirubin with normal urine and dark stools. Hepatic jaundice results from liver cell damage such as viral hepatitis or alcoholic liver disease, elevating both conjugated and unconjugated bilirubin and producing stigmata of chronic liver disease. Post-hepatic jaundice results from bile duct obstruction, producing elevated conjugated bilirubin, dark urine, and pale stools, with a palpable non-tender gallbladder in malignant obstruction by Courvoisier's law. Clinical assessment uses the history to identify onset, Charcot's triad of ascending cholangitis, pruritus, alcohol use, and drug history, and examination to identify stigmata of chronic liver disease or a palpable gallbladder. Investigation begins with liver function tests, viral hepatitis serology, and abdominal ultrasound, followed by MRCP for suspected bile duct obstruction.

    Value-added response: The distinction between hepatocellular and obstructive jaundice is critical because management differs completely: hepatocellular jaundice from viral hepatitis requires antiviral therapy and avoidance of hepatotoxins, while obstructive jaundice requires biliary decompression by ERCP with stone extraction or surgical bypass for malignant obstruction. Ascending cholangitis is a medical emergency requiring simultaneous intravenous antibiotics and urgent biliary drainage, because septic shock can develop rapidly if drainage is delayed.

12. **Basic response:** Immediate management follows the ABCDE approach with two large-bore intravenous cannulae, blood taken for full blood count, coagulation screen, group and crossmatch, urea and electrolytes, and liver function tests. Intravenous crystalloid

resuscitation is commenced, packed red cell transfusion is given when haemoglobin falls below 70 grams per litre, and coagulopathy is corrected with fresh frozen plasma and platelets. A proton pump inhibitor is given for suspected peptic ulcer bleeding, and terlipressin for suspected variceal bleeding. Upper GI endoscopy within 24 hours identifies the bleeding source and allows adrenaline injection and haemoclip placement for peptic ulcer bleeding, and band ligation for oesophageal varices.

Value-added response: Risk stratification using the Glasgow-Blatchford score guides urgency: a score of zero identifies patients suitable for outpatient management. In patients with variceal bleeding, broad-spectrum antibiotics such as cefotaxime are essential because spontaneous bacterial peritonitis develops in up to 50 percent of cirrhotic patients admitted with variceal haemorrhage, significantly increasing mortality. Patients who rebleed after endoscopy should be considered for transjugular intrahepatic portosystemic shunting, which reduces portal pressure and controls haemorrhage in the majority of cases.

13. **Basic response:** Abdominal pain arises through visceral pain (dull, midline, from distension or spasm of a hollow viscus), parietal pain (sharp, well localised, from peritoneal irritation, worsened by movement), and referred pain (perceived at a distant site due to shared spinal nerve roots). History follows the SOCRATES framework, with attention to onset (sudden onset suggests perforation), radiation (to the back in pancreatitis; to the right shoulder in biliary disease), and associations including fever, change in bowel habit, and jaundice. Physical examination identifies guarding, involuntary rigidity (board-like in generalised peritonitis), and rebound tenderness. Specific signs include Murphy's sign (cholecystitis), Rovsing's sign (appendicitis), and the succussion splash (intestinal obstruction).

    Value-added response: Clinical assessment must integrate knowledge of regional diagnoses: sudden onset epigastric pain with peritonism requires an urgent erect chest X-ray to identify free gas confirming perforated peptic ulcer and mandating immediate surgical referral. In Nigerian practice, the differential must always include sickle cell painful crisis, typhoid fever, and amoebic liver abscess, which can mimic a surgical

emergency and must be excluded before referring for surgery, as unnecessary laparotomy in a patient with sickle cell disease carries a very high perioperative risk.

14. **Basic response:** The serum-ascites albumin gradient is calculated by subtracting the ascitic fluid albumin from the serum albumin. A SAAG of 11 grams per litre or above indicates portal hypertension, with liver cirrhosis from hepatitis B or C being the most common cause in Nigeria, followed by right heart failure and Budd-Chiari syndrome. A SAAG below 11 grams per litre indicates an exudative cause such as peritoneal carcinomatosis, tuberculous peritonitis, or nephrotic syndrome. Investigations on ascitic fluid include albumin for SAAG, total protein, cell count (neutrophils above 250 per microlitre indicates spontaneous bacterial peritonitis), cytology for malignant cells, Gram stain and bacterial culture, and adenosine deaminase with AFB culture for tuberculous peritonitis.

    Value-added response: The diagnostic paracentesis is also therapeutic when performed as a large-volume paracentesis in patients with tense ascites: removing 4 to 6 litres of fluid relieves dyspnoea and abdominal discomfort significantly. However, intravenous albumin at 6 to 8 grams per litre of fluid removed must be given simultaneously to prevent paracentesis-induced circulatory dysfunction, a complication that can precipitate hepatorenal syndrome and increase short-term mortality in cirrhotic patients.

15. **Basic response:** Clinically significant unintentional weight loss is the loss of more than 5 percent of usual body weight over 6 to 12 months without deliberate restriction. In Nigeria, the most important differential diagnoses include tuberculosis (presenting with night sweats, cough, and haemoptysis), HIV-related enteropathy and opportunistic gastrointestinal infections, malabsorption syndromes including tropical sprue and chronic pancreatitis, and gastrointestinal malignancy. Investigation includes full blood count, ESR, sputum for AFB and chest X-ray for tuberculosis, thyroid function tests, stool microscopy and culture, and upper and lower GI endoscopy with biopsy for malignancy and malabsorption.

    Value-added response: Dysphagia is a red flag symptom that demands urgent upper GI endoscopy, as oesophageal carcinoma in Nigeria is frequently diagnosed at an advanced stage when curative resection is no longer possible. A change in bowel habit in any

patient above 40 years must similarly prompt colonoscopy to exclude colorectal carcinoma. Early nutritional assessment and dietitian involvement are essential adjuncts, because correcting malnutrition improves the patient's tolerance of subsequent treatment, whether antituberculous chemotherapy, antiviral therapy, or surgery.

Answers to Pilot Questions [Series 1]

Part 1.1

1. Visceral pain arises from distension, spasm, or ischaemia of a hollow viscus and is dull, cramping, and poorly localised to the midline because visceral afferent fibres are bilateral. Parietal pain arises from irritation of the parietal peritoneum, is sharp and well localised, and is worsened by movement. Referred pain is perceived at a site distant from the diseased organ due to shared spinal nerve roots, as in right shoulder tip pain from diaphragmatic irritation.
2. Parietal pain is sharp and well localised, whereas visceral pain is dull and diffusely felt in the midline. Parietal pain is also worsened by movement and coughing, whereas visceral pain is not significantly affected by position.
3. Site describes where the pain is worst. Onset describes whether pain started suddenly or gradually. Radiation identifies whether pain moves to another site, such as to the back in pancreatitis. Associations include accompanying symptoms such as nausea, vomiting, fever, or change in bowel habit.
4. Murphy's sign is arrest of inspiration on deep palpation of the right hypochondrium, indicating acute cholecystitis. Rovsing's sign is pain in the right iliac fossa on pressure applied to the left iliac fossa, indicating peritoneal irritation from appendicitis.
5. Free gas under the right hemidiaphragm on an erect chest X-ray indicates bowel perforation, requiring immediate surgical referral. Dilated loops of bowel with air-fluid levels on a plain abdominal X-ray indicate intestinal obstruction, also requiring urgent surgical assessment.

Part 1.2

1. Haematemesis is the vomiting of blood from a source proximal to the ligament of Treitz; it may be bright red or coffee-ground in appearance. Melaena is the passage of black, tarry, foul-smelling stools from degradation of blood in the upper GI tract. Both indicate an upper GI source of bleeding.
2. The most common cause is peptic ulcer disease including gastric and duodenal ulcers. Oesophageal varices from portal hypertension in liver cirrhosis is the second most common and the most life-threatening cause. Mallory-Weiss tears and oesophagitis are also important but less frequent causes.
3. Haemorrhoids are the most common cause of lower GI bleeding in young adults in Nigeria. Anal fissures produce bright red blood coating the outside of the stool with pain on defaecation. Infectious colitis from bacterial dysentery or amoebic colitis is also a frequent cause in areas with limited sanitation.
4. A heart rate above 100 beats per minute indicates significant blood loss and the need for urgent resuscitation. A systolic blood pressure below 100 millimetres of mercury, especially with postural dizziness and cold peripheries, indicates haemodynamic compromise requiring immediate fluid resuscitation and blood transfusion.
5. Upper GI endoscopy is the investigation of choice. It allows adrenaline injection and haemoclip placement to achieve haemostasis in peptic ulcer bleeding. It also allows band ligation of oesophageal varices, the endoscopic treatment of choice for variceal haemorrhage.

Part 1.3

1. Jaundice is the yellow discolouration of the skin, sclerae, and mucous membranes from an elevated serum bilirubin. It becomes clinically apparent when serum bilirubin exceeds approximately 35 to 50 micromoles per litre.

2. In pre-hepatic jaundice, the elevated bilirubin is unconjugated because haemolysis overwhelms the liver's conjugating capacity; urine is normal because unconjugated bilirubin is not water-soluble, and stools are dark from excess urobilinogen. In post-hepatic jaundice, the elevated bilirubin is conjugated because bile duct obstruction prevents excretion; urine is dark because conjugated bilirubin spills into the urine, and stools are pale because bile cannot reach the bowel.
3. Viral hepatitis B is the most common cause of hepatocellular jaundice in Nigeria given the high prevalence of chronic hepatitis B. Alcoholic liver disease is an important cause in men with chronic alcohol use. Drug-induced liver injury from conventional medications and traditional herbal medicines widely used in Nigeria is also significant.
4. Courvoisier's law states that a palpable, non-tender gallbladder in the presence of jaundice suggests malignant obstruction rather than gallstones. It is clinically significant because it directs investigation towards pancreatic carcinoma or cholangiocarcinoma, which require surgical or palliative management rather than the endoscopic stone extraction used for gallstone disease.
5. Magnetic resonance cholangiopancreatography is the investigation of choice for suspected bile duct obstruction. It provides detailed non-invasive imaging of the biliary tree without the procedural risks of contrast injection into the bile ducts, making it safer than ERCP when the purpose is purely diagnostic.

Part 1.4

1. Clinically significant unintentional weight loss is the loss of more than 5 percent of the patient's usual body weight over 6 to 12 months without deliberate dietary restriction or increased physical activity.
2. Tuberculosis is the most important infective cause to exclude, presenting with night sweats, cough, haemoptysis, and weight loss. HIV-related enteropathy and opportunistic GI infections cause weight loss through malabsorption and increased metabolic demand. Gastrointestinal malignancy affecting the oesophagus, stomach, or colorectum presents

with weight loss alongside red flag symptoms such as dysphagia or a change in bowel habit.

3. The five Fs are Fat (obesity), Fluid (ascites from portal hypertension, malignancy, or infection), Flatus (gaseous distension from obstruction or ileus), Faeces (faecal loading from constipation or large bowel obstruction), and Foetus (pregnancy, which must be considered in all women of reproductive age).

4. The SAAG is calculated by subtracting the ascitic fluid albumin from the serum albumin. A value of 11 grams per litre or above indicates portal hypertension, with liver cirrhosis from hepatitis B or C being the most common cause in Nigeria. A value below 11 grams per litre indicates an exudative cause such as peritoneal carcinomatosis, tuberculous peritonitis, or nephrotic syndrome.

5. Cell count detects a neutrophil count above 250 cells per microlitre, indicating spontaneous bacterial peritonitis and requiring immediate intravenous antibiotics. Cytology identifies malignant cells, confirming peritoneal carcinomatosis from an underlying primary tumour. Adenosine deaminase with AFB culture is used to investigate tuberculous peritonitis, in which ADA is elevated above 30 to 40 units per litre.

References and Attributions [Series 1]

Textual References

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- Longmore, M., Wilkinson, I., Baldwin, A. and Wallin, E. (2014). Oxford Handbook of Clinical Medicine (9th ed.). Oxford: Oxford University Press.
- Kasper, D. L., Fauci, A. S., Hauser, S. L., Longo, D. L., Jameson, J. L. and Loscalzo, J. (2018). Harrison's Principles of Internal Medicine (20th ed.). New York: McGraw-Hill.
- British Society of Gastroenterology. (2015). UK Guidelines on the Management of Upper Gastrointestinal Haemorrhage. Gut, 64(7), 1 to 27.

Figures, Plates, Tables, and Boxes

- Figure 1.1: Regional differential diagnosis of abdominal pain. AI-generated using the prompt: "Create a clean labelled body outline diagram showing the abdominal regions and their key differential diagnoses. Label each region with the two to three most important conditions. Professional medical illustration style." Suggested OER search string: "abdominal pain regions differential diagnosis labelled body diagram OER."
- Table 1.1: Comparison of upper and lower gastrointestinal bleeding. AI-generated using the prompt: "Create a clear two-column comparison table titled Upper GI versus Lower GI Bleeding with rows for source, presentation, common causes in Nigeria, and investigation." Suggested OER search string: "upper lower gastrointestinal bleeding comparison table causes investigation OER."
- Table 1.2: Classification of jaundice by mechanism, causes, and clinical and investigative features. AI-generated using the prompt: "Create a clean three-column medical table titled Classification of Jaundice with columns for Type, Mechanism and Causes, and

Urine and Stool Changes and Key Investigation." Suggested OER search string: "jaundice classification pre-hepatic hepatic post-hepatic table OER."

- Table 1.3: Clinical assessment of ascites and investigations performed on ascitic fluid. AI-generated using the prompt: "Create a clear two-column medical table titled Approach to Ascites covering the five Fs, SAAG classification, and diagnostic paracentesis investigations." Suggested OER search string: "ascites SAAG classification diagnostic paracentesis investigations table OER."

Series 2: Nutritional and Metabolic Disorders

- **Part 2.1: Malnutrition and Nutritional Deficiencies**
    - Sub Part 2.1.1: Protein-energy malnutrition
    - Sub Part 2.1.2: Micronutrient deficiencies
    - Sub Part 2.1.3: Nutritional assessment and management
- **Part 2.2: Disorders of Carbohydrate and Lipid Metabolism**
    - Sub Part 2.2.1: Malabsorption syndromes
    - Sub Part 2.2.2: Disorders of lipid metabolism and the gut
    - Sub Part 2.2.3: Lactose intolerance and coeliac disease
- **Part 2.3: Metabolic Liver Disease**
    - Sub Part 2.3.1: Non-alcoholic fatty liver disease
    - Sub Part 2.3.2: Alcoholic liver disease
    - Sub Part 2.3.3: Wilson's disease and haemochromatosis
- **Part 2.4: Nutritional Support and Refeeding**
    - Sub Part 2.4.1: Enteral nutrition
    - Sub Part 2.4.2: Parenteral nutrition
    - Sub Part 2.4.3: Refeeding syndrome

Introduction

Nutritional and metabolic disorders represent a significant but often underappreciated dimension of gastroenterology. In Nigeria, protein-energy malnutrition and micronutrient deficiencies continue to affect large segments of the population, particularly children, pregnant women, and

patients with chronic illness. At the same time, the rising prevalence of obesity, metabolic syndrome, and non-alcoholic fatty liver disease reflects the nutrition transition occurring in urban populations. Understanding the pathophysiology, clinical features, and management of these conditions is essential for any clinician practising in Nigeria.

The aim of this Series is to equip you with the knowledge needed to recognise, investigate, and manage the major nutritional and metabolic gastrointestinal disorders. We shall commence by examining malnutrition and nutritional deficiencies, covering protein-energy malnutrition, micronutrient deficiencies, and nutritional assessment. Next, we will explore disorders of carbohydrate and lipid metabolism, including malabsorption syndromes, coeliac disease, and lactose intolerance. Moving on, we will consider metabolic liver disease, covering non-alcoholic fatty liver disease, alcoholic liver disease, and the inherited metabolic disorders Wilson's disease and haemochromatosis. Finally, we will conclude with nutritional support, covering enteral and parenteral nutrition and the clinically important refeeding syndrome.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 2.1:** describe the clinical features, assessment, and management of protein-energy malnutrition and common micronutrient deficiencies,
- **SLO 2.2:** describe the pathophysiology, clinical features, and investigation of malabsorption syndromes including coeliac disease and lactose intolerance,
- **SLO 2.3:** describe the pathophysiology, clinical features, and management of non-alcoholic fatty liver disease and alcoholic liver disease,
- **SLO 2.4:** describe the clinical features and investigation of Wilson's disease and haemochromatosis, and
- **SLO 2.5:** describe the principles of enteral and parenteral nutrition and explain the refeeding syndrome.

2.1 Malnutrition and Nutritional Deficiencies

2.1.1 Protein-energy malnutrition

Protein-energy malnutrition (PEM) results from an inadequate intake of protein and calories relative to metabolic requirements and is the most prevalent nutritional disorder in Nigeria. It is classified into three clinical syndromes. **Kwashiorkor** results predominantly from protein deficiency in the presence of adequate caloric intake: the child presents with **generalised oedema** (pitting oedema from hypoalbuminaemia), a **distended abdomen**, skin changes (**flaky paint dermatosis**), **hair discolouration** (flag sign: alternating bands of light and dark hair), and profound anorexia and misery.

Marasmus results from a combined deficiency of protein and calories, producing severe wasting: the child has a **wizened, old-man appearance** with visible ribs, absence of subcutaneous fat, muscle wasting, and a large head relative to body size. Unlike kwashiorkor, **oedema is absent** in marasmus. **Marasmic kwashiorkor** is the most severe form, combining features of both. In adults, PEM is assessed using body mass index (BMI below 18.5 kg/m²), mid-upper arm circumference, and serum albumin (hypoalbuminaemia below 35 g/L indicates severe depletion).

_____ presents with generalised oedema, distended abdomen, and skin changes from protein deficiency, while _____ presents with severe wasting and absence of oedema from combined protein and caloric deficiency.

2.1.2 Micronutrient deficiencies

Iron deficiency is the most common micronutrient deficiency globally and in Nigeria: it presents with **microcytic hypochromic anaemia** (low MCV, low MCH, low serum ferritin, elevated TIBC), angular stomatitis, koilonychia (spoon-shaped nails), glossitis, and dysphagia from post-cricoid web (Plummer-Vinson syndrome). **Vitamin A deficiency** causes night blindness (the earliest symptom), Bitot's spots (foamy white patches on the conjunctiva), xerophthalmia, and corneal ulceration leading to blindness; it is a leading preventable cause of childhood blindness in Nigeria.

Vitamin B12 deficiency causes **megaloblastic anaemia** (macrocytic anaemia with hypersegmented neutrophils), subacute combined degeneration of the spinal cord (dorsal column and corticospinal tract demyelination causing paraesthesiae, weakness, and ataxia), and glossitis. Causes include pernicious anaemia (autoimmune destruction of gastric parietal cells abolishing intrinsic factor production), strict veganism, gastrectomy, and ileal disease or resection.**Vitamin D deficiency** causes rickets in children (bowing of long bones, rachitic rosary, craniotabes) and osteomalacia in adults (bone pain, proximal myopathy, and pseudofractures on X-ray).

_____ deficiency causes microcytic hypochromic anaemia, koilonychia, and dysphagia from post-cricoid web. _____ deficiency causes night blindness and Bitot's spots and is a leading cause of childhood blindness in Nigeria.

2.1.3 Nutritional assessment and management

Nutritional assessment uses anthropometric measurements (weight, height, BMI, mid-upper arm circumference, skinfold thickness), biochemical markers (serum albumin, prealbumin, transferrin, full blood count, iron studies, vitamin levels), clinical examination (signs of specific deficiencies), and dietary history. The Malnutrition Universal Screening Tool (MUST) is widely used in clinical practice and classifies patients as low, medium, or high risk for malnutrition based on BMI, unintentional weight loss, and acute disease effect.

Management of PEM requires a stepwise approach: correct dehydration and electrolyte imbalances first (particularly hypokalaemia and hypophosphataemia), then commence cautious refeeding with gradually increasing caloric intake to avoid refeeding syndrome. Micronutrient deficiencies are corrected with specific supplements: iron (ferrous sulphate orally), vitamin A (high-dose supplementation in deficient children), vitamin B12 (intramuscular hydroxocobalamin in pernicious anaemia), and vitamin D (cholecalciferol). Community-level interventions including food supplementation programmes and dietary education are essential for prevention.

The _____ Universal Screening Tool classifies malnutrition risk using BMI, unintentional weight loss, and acute disease effect. Vitamin B12 deficiency from pernicious anaemia requires intramuscular _____ because the patient lacks intrinsic factor for oral absorption.

Kwashiorkor	Marasmus
Cause: Predominantly protein deficiency	**Cause**: Combined protein and caloric deficiency
Clinical features: • Generalised pitting oedema from hypoalbuminaemia • Distended abdomen (ascites, hepatomegaly) • Flaky paint dermatosis (skin lesions) • Hair discolouration with "flag sign" (alternating bands) • Anorexia and misery • Oedema present	**Clinical features**: • Severe wasting with wizened "old-man" appearance • Visible ribs and bony prominences • Absent subcutaneous fat • Muscle wasting • Large head relative to body • Oedema absent
Marasmic Kwashiorkor (combined features of both; most severe form)	**Marasmic Kwashiorkor** (combined features of both; most severe form)

Table 2.1: Clinical features distinguishing kwashiorkor from marasmus

Pilot questions 2.1

1. Distinguish between kwashiorkor and marasmus in terms of cause and clinical features. (Linked to SLO 2.1)
2. Describe the clinical features of iron deficiency. (Linked to SLO 2.1)
3. Describe the clinical features of vitamin A deficiency and state its significance in Nigeria. (Linked to SLO 2.1)
4. Describe the clinical features of vitamin B12 deficiency and state the neurological syndrome it causes. (Linked to SLO 2.1)
5. Describe the nutritional assessment tools used to identify malnutrition. (Linked to SLO 2.1)

2.2 Disorders of Carbohydrate and Lipid Metabolism

2.2.1 Malabsorption syndromes

Malabsorption is the failure of the small intestine to absorb nutrients, leading to steatorrhoea (pale, bulky, offensive, floating stools from fat malabsorption), weight loss, abdominal bloating and distension, and deficiencies of fat-soluble vitamins (A, D, E, and K) and other micronutrients. The small intestinal mucosa has three essential requirements for normal absorption: adequate mucosal surface area (villi and microvilli), normal mucosal epithelial cells, and adequate digestive enzymes and bile salts.

Causes of malabsorption are classified by mechanism. Mucosal causes include coeliac disease (gluten-sensitive enteropathy causing villous atrophy) and tropical sprue (a condition endemic in Nigeria and other tropical regions causing progressive villous atrophy and megaloblastic anaemia). Pancreatic causes include chronic pancreatitis (inadequate pancreatic enzyme secretion causing fat malabsorption) and cystic fibrosis. Luminal causes include bacterial overgrowth (excess bacteria in the small bowel deconjugating bile salts) and Giardia lamblia infection (a common parasitic cause of malabsorption in Nigeria).

Malabsorption presents with _____ (pale, bulky, floating stools from fat malabsorption), weight loss, and deficiencies of fat-soluble vitamins _____, D, E, and K.

2.2.2 Disorders of lipid metabolism and the gut

Short bowel syndrome results from surgical resection of large segments of the small intestine, leaving insufficient absorptive surface area. The consequences depend on the extent and site of resection: loss of the terminal ileum impairs vitamin B12 absorption (requiring intramuscular supplementation) and bile salt reabsorption (causing bile salt diarrhoea and fat malabsorption). Management includes a high-calorie, high-protein, low-fat diet, nutritional supplementation, and long-term parenteral nutrition in severe cases.

Abetalipoproteinaemia is a rare autosomal recessive disorder characterised by the inability to synthesise apolipoprotein B, resulting in failure of chylomicron and VLDL formation. Fat-soluble vitamins A, E, and K cannot be transported, producing fat malabsorption, acanthocytosis (spiky red cells on blood film), retinitis pigmentosa, and progressive ataxia and neuropathy.

Serum cholesterol and triglycerides are markedly reduced. Treatment requires high-dose fat-soluble vitamin supplementation.

Loss of the _____ ileum impairs vitamin B12 absorption and bile salt reabsorption. Abetalipoproteinaemia results from failure to synthesise _____ B, impairing chylomicron formation and fat-soluble vitamin transport.

2.2.3 Lactose intolerance and coeliac disease

Lactose intolerance results from deficiency of lactase, the brush border enzyme that hydrolyses lactose to glucose and galactose. Undigested lactose passes to the colon where it is fermented by bacteria, producing osmotic diarrhoea, abdominal bloating, flatulence, and cramping pain. Primary lactase deficiency is the most common form and is prevalent in Nigeria; symptoms begin in adolescence or adulthood. Diagnosis is by the hydrogen breath test (elevated breath hydrogen after lactose load) or empirical improvement on a lactose-free diet.

Coeliac disease (gluten-sensitive enteropathy) is caused by an abnormal immune response to **gluten** (the protein in wheat, rye, and barley) in genetically susceptible individuals (HLA-DQ2 and HLA-DQ8). The immune response causes **villous atrophy** and crypt hyperplasia in the small intestine, reducing absorptive surface area and causing malabsorption. Presentation includes diarrhoea, steatorrhoea, weight loss, abdominal bloating, iron and folate deficiency anaemia, and in children, failure to thrive. Diagnosis requires **positive anti-tissue transglutaminase IgA antibodies** and duodenal biopsy (showing villous atrophy). Treatment is a strict, lifelong gluten-free diet.

Lactose intolerance results from deficiency of _____, causing fermentation of undigested lactose in the colon. Coeliac disease causes _____ atrophy and crypt hyperplasia from an immune response to gluten.

Lactose Intolerance	Coeliac Disease
Mechanism: Lactase deficiency → undigested lactose fermented in colon	**Mechanism**: Immune response to gluten → villous atrophy and malabsorption
Clinical features: • Osmotic diarrhoea • Bloating • Flatulence • Cramping abdominal pain after dairy intake	**Clinical features**: • Diarrhoea, steatorrhoea • Weight loss • Iron and folate deficiency anaemia • Failure to thrive in children
Diagnosis: Hydrogen breath test or empirical lactose-free diet	**Diagnosis**: Positive anti-tissue transglutaminase IgA antibodies and duodenal biopsy (villous atrophy, crypt hyperplasia)
Treatment: Lactose-free diet or lactase enzyme supplements	**Treatment**: Strict lifelong gluten-free diet

Table 2.2: Comparison of lactose intolerance and coeliac disease

Pilot questions 2.2

1. Describe the clinical features of malabsorption and state two causes. (Linked to SLO 2.2)
2. Describe the consequences of terminal ileal resection. (Linked to SLO 2.2)
3. Describe the mechanism of lactose intolerance and its clinical features. (Linked to SLO 2.2)
4. Describe the pathophysiology and clinical features of coeliac disease. (Linked to SLO 2.2)
5. State the investigation of choice for coeliac disease and the treatment. (Linked to SLO 2.2)

2.3 Metabolic Liver Disease

2.3.1 Non-alcoholic fatty liver disease

Non-alcoholic fatty liver disease (NAFLD) is the most common chronic liver disease in high-income countries and is increasingly prevalent in Nigeria with the rise of obesity and metabolic syndrome. It encompasses a spectrum from **simple steatosis** (fat accumulation in hepatocytes without inflammation, which is benign and reversible) to **non-alcoholic steatohepatitis (NASH)** (steatosis with hepatocyte inflammation and ballooning, which can progress to fibrosis, cirrhosis, and hepatocellular carcinoma). Risk factors include obesity, type 2 diabetes, hypertension, dyslipidaemia, and insulin resistance.

Most patients with NAFLD are asymptomatic, presenting incidentally with elevated liver enzymes (ALT and AST) or hepatic steatosis on abdominal ultrasound. A minority present with right upper quadrant discomfort or fatigue. Liver function tests show a hepatocellular pattern with ALT typically greater than AST. Diagnosis is confirmed by ultrasound (bright echogenic liver) and liver biopsy (the gold standard, required to distinguish simple steatosis from NASH and to stage fibrosis). Management is primarily lifestyle modification: weight loss of 7 to 10 percent of body weight improves histology in NASH. There is no approved pharmacological therapy for NAFLD.

NAFLD ranges from simple _____ (benign and reversible) to NASH (with inflammation and ballooning), which can progress to fibrosis and cirrhosis. The most effective treatment for NAFLD is _____ modification with weight loss.

2.3.2 Alcoholic liver disease

Alcoholic liver disease (ALD) describes the spectrum of hepatic injury caused by excessive alcohol consumption and ranges from **alcoholic fatty liver (steatosis)** (reversible on abstinence), to **alcoholic hepatitis** (acute inflammatory injury causing jaundice, fever, right upper quadrant pain, and elevated liver enzymes with an AST:ALT ratio above 2:1), to **alcoholic cirrhosis** (irreversible scarring from repeated injury, presenting with portal hypertension, ascites, variceal bleeding, hepatic encephalopathy, and coagulopathy).

The clinical history must quantify alcohol intake in units (one unit equals 8 grams of pure alcohol in the UK); harmful use is defined as above 14 units per week for women and above 21 units per week for men. The Maddrey Discriminant Function (MDF) score, calculated as 4.6 times the prothrombin time prolongation plus serum bilirubin, identifies severe alcoholic hepatitis (MDF above 32) in which prednisolone reduces short-term mortality. Pentoxifylline is an alternative when corticosteroids are contraindicated. Abstinence from alcohol is the cornerstone of management at all stages.

In alcoholic liver disease, an AST to ALT ratio above _____ to 1 suggests alcoholic hepatitis. A Maddrey Discriminant Function score above _____ identifies severe alcoholic hepatitis in which prednisolone reduces mortality.

2.3.3 Wilson's disease and haemochromatosis

Wilson's disease is an autosomal recessive disorder of copper metabolism caused by mutations in the ATP7B gene, resulting in copper accumulation in the liver, brain, kidneys, and eyes. Clinical features include hepatic disease (hepatitis, cirrhosis, fulminant hepatic failure), neuropsychiatric manifestations (dysarthria, tremor, psychiatric symptoms), and **Kayser-Fleischer rings** (golden-brown copper deposits in the corneal periphery, visible on slit-lamp examination, which are pathognomonic of Wilson's disease with neurological involvement). Diagnosis requires serum caeruloplasmin (low in 85 percent of cases), 24-hour urinary copper (elevated), liver biopsy, and slit-lamp examination. Treatment is with copper chelation using D-penicillamine or trientine.

Hereditary haemochromatosis is the most common inherited metabolic disorder in populations of northern European descent and is caused by mutations in the HFE gene (C282Y homozygosity in 85 percent), resulting in excessive iron absorption from the gut. Iron accumulates in the liver, pancreas, heart, joints, pituitary, and skin, causing **liver cirrhosis**, **diabetes mellitus (bronze diabetes)**, **cardiomyopathy**, **arthropathy**, and **skin bronzing**. Diagnosis is by elevated transferrin saturation (above 45 percent) and serum ferritin (above 300 micrograms per litre in men), confirmed by HFE gene testing and liver biopsy. Treatment is regular venesection (phlebotomy) to remove excess iron.

_____ rings (golden-brown corneal deposits visible on slit-lamp) are pathognomonic of Wilson's disease with neurological involvement. Hereditary haemochromatosis is caused by mutations in the _____ gene and is treated by regular venesection.

Wilson's Disease	Hereditary Haemochromatosis
Inheritance & Gene: Autosomal recessive; ATP7B gene mutation	**Inheritance & Gene**: Autosomal recessive; **HFE gene** (C282Y homozygosity most common)
Accumulated Substance: Copper	**Accumulated Substance**: Iron
Organs Affected: • Liver: hepatitis, cirrhosis • Brain: dysarthria, tremor, psychiatric symptoms • Eyes: Kayser–Fleischer rings	**Organs Affected**: • Liver: cirrhosis • Pancreas: diabetes mellitus • Heart: cardiomyopathy • Joints: arthropathy • Skin: bronzing
Diagnosis: • Low serum caeruloplasmin • Elevated 24-hour urinary copper • Slit-lamp exam for Kayser–Fleischer rings • Liver biopsy	**Diagnosis**: • Elevated transferrin saturation (>45%) • Elevated serum ferritin • HFE gene testing • Liver biopsy
Treatment: D-penicillamine or trientine (copper chelation)	**Treatment**: Regular venesection (phlebotomy) to remove excess iron

Table 2.3: Comparison of Wilson's disease and hereditary haemochromatosis

Pilot questions 2.3

1. Describe the spectrum of non-alcoholic fatty liver disease and state the risk factors. (Linked to SLO 2.3)
2. Describe the spectrum of alcoholic liver disease and the management of severe alcoholic hepatitis. (Linked to SLO 2.3)
3. Describe the clinical features of Wilson's disease and state the pathognomonic sign. (Linked to SLO 2.4)

4. Describe the organ manifestations of hereditary haemochromatosis. (Linked to SLO 2.4)
5. State the investigations used to diagnose Wilson's disease and haemochromatosis. (Linked to SLO 2.4)

2.4 Nutritional Support and Refeeding

2.4.1 Enteral nutrition

Enteral nutrition (EN) refers to the delivery of nutrients directly into the gastrointestinal tract via a tube, bypassing the need for oral intake. It is indicated when the patient has a functioning gut but is unable to meet nutritional requirements orally, including in cases of dysphagia (stroke, head and neck cancer, oesophageal disease), reduced consciousness, critical illness with inadequate oral intake, and inflammatory bowel disease with severe malnutrition. The principle of 'if the gut works, use it' applies: enteral nutrition preserves gut mucosal integrity, maintains the intestinal immune barrier, and reduces bacterial translocation across the gut wall.

Routes of enteral nutrition include a nasogastric tube (short-term: up to 4 to 6 weeks), a nasojejunal tube (for patients at high risk of aspiration or with gastroparesis), a percutaneous endoscopic gastrostomy (PEG: long-term access placed endoscopically for patients requiring feeding beyond 4 to 6 weeks, such as those with stroke or motor neurone disease), and a jejunostomy (for patients with gastric outlet obstruction or after oesophagogastric surgery). Complications of enteral feeding include aspiration pneumonia (the most serious), diarrhoea, tube blockage, and nasopharyngeal discomfort.

Enteral nutrition is preferred over parenteral nutrition when the _____ is functioning because it preserves gut mucosal integrity and reduces bacterial translocation. A _____ endoscopic gastrostomy is the preferred long-term enteral access route for patients requiring feeding beyond 4 to 6 weeks.

2.4.2 Parenteral nutrition

Parenteral nutrition (PN) is the intravenous delivery of all essential nutrients (glucose, amino acids, lipids, vitamins, minerals, and trace elements) when the gastrointestinal tract cannot be used. Indications include short bowel syndrome with insufficient absorptive capacity, prolonged paralytic ileus, high-output intestinal fistula, severe acute pancreatitis with intestinal failure, and post-operative ileus in major GI surgery. PN is delivered through a central venous catheter (subclavian or internal jugular vein) because of the hyperosmolarity of the solution.

Total parenteral nutrition (TPN) requires meticulous monitoring: daily fluid balance, electrolytes (sodium, potassium, magnesium, phosphate), liver function tests (PN causes cholestasis in long-term use), blood glucose (hyperglycaemia is common and requires insulin sliding scale), and regular assessment of catheter insertion site for infection. Complications of PN include catheter-related bloodstream infection (the most serious complication), metabolic complications (hyperglycaemia, hyperlipidaemia, electrolyte disturbances), and hepatic steatosis with prolonged use.

Parenteral nutrition is delivered through a _____ venous catheter because of the hyperosmolarity of the solution. The most serious complication of parenteral nutrition is catheter-related _____ infection.

2.4.3 Refeeding syndrome

Refeeding syndrome is a potentially life-threatening metabolic complication that occurs when nutrition is reintroduced too rapidly in severely malnourished patients after a period of starvation. During starvation, the body depletes intracellular stores of phosphate, potassium, and magnesium. When carbohydrates are reintroduced, insulin secretion increases dramatically and drives phosphate, potassium, and magnesium from the extracellular to the intracellular space, causing dangerous **hypophosphataemia**, hypokalaemia, and hypomagnesaemia. Hypophosphataemia impairs ATP production in all cells, causing cardiac arrhythmias, respiratory failure, haemolytic anaemia, seizures, and cardiac arrest.

Patients at risk of refeeding syndrome include those with BMI below 16 kg/m², unintentional weight loss greater than 15 percent in 3 to 6 months, little or no nutritional intake for more than 10 days, and low pre-feeding levels of phosphate, potassium, or magnesium. Prevention requires identifying at-risk patients, commencing nutritional support at no more than 10 kcal per kg per day, increasing slowly over 4 to 7 days, and replacing phosphate, potassium, magnesium, and B vitamins (particularly thiamine) before and throughout refeeding.

Refeeding syndrome is caused by rapid introduction of _____ (carbohydrates) after starvation, driving phosphate, potassium, and magnesium into cells and causing dangerous _____. Thiamine supplementation is given before refeeding to prevent Wernicke's encephalopathy.

Enteral Nutrition	Parenteral Nutrition
Definition: Delivery of nutrients into GI tract via tube	**Definition**: Intravenous delivery of all nutrients, bypassing GI tract
Indication: Functioning gut but unable to meet needs orally	**Indication**: Non-functioning gut (short bowel syndrome, prolonged ileus, high-output fistula, severe pancreatitis)
Routes: • Nasogastric tube (short-term) • PEG (long-term >4–6 weeks) • Nasojejunal (high aspiration risk) • Jejunostomy (post-oesophagogastric surgery)	**Route**: Central venous catheter (due to hyperosmolarity)
Advantages: Preserves gut mucosal integrity, reduces bacterial translocation, lower cost, lower infection risk	**Monitoring**: Daily electrolytes, liver function tests, blood glucose, catheter site
Complications: Aspiration pneumonia (most serious), diarrhoea, tube blockage	**Complications**: Catheter-related bloodstream infection (most serious),

Enteral Nutrition	Parenteral Nutrition
	hyperglycaemia, hepatic cholestasis (long-term use)

Table 2.4: Comparison of enteral and parenteral nutrition

Pilot questions 2.4

1. State the indications for enteral nutrition and list three routes of delivery. (Linked to SLO 2.5)
2. State the indications for parenteral nutrition and describe the route of administration. (Linked to SLO 2.5)
3. Describe the metabolic complications of parenteral nutrition. (Linked to SLO 2.5)
4. Explain the pathophysiology of refeeding syndrome. (Linked to SLO 2.5)
5. State four features that identify a patient at high risk of refeeding syndrome. (Linked to SLO 2.5)

Series Summary

This Series has examined the major nutritional and metabolic disorders relevant to gastroenterology practice in Nigeria. We began with malnutrition and nutritional deficiencies, distinguishing kwashiorkor (protein deficiency with oedema) from marasmus (combined deficiency with severe wasting), and describing the clinical features of iron, vitamin A, vitamin B12, and vitamin D deficiencies alongside the tools for nutritional assessment. Next, we explored disorders of carbohydrate and lipid metabolism, covering the mechanisms and causes of malabsorption including tropical sprue and Giardia infection, the consequences of short bowel syndrome and terminal ileal resection, and the features and management of lactose intolerance and coeliac disease. Moving on, we examined metabolic liver disease, covering the spectrum from simple NAFLD steatosis to NASH and cirrhosis, the stages of alcoholic liver disease and

management with prednisolone in severe hepatitis, and the inherited metabolic disorders Wilson's disease (copper accumulation with Kayser-Fleischer rings) and hereditary haemochromatosis (iron accumulation treated by venesection). Finally, we addressed nutritional support, distinguishing enteral from parenteral nutrition, and describing the pathophysiology, recognition, and prevention of refeeding syndrome.

By completing this Series, you should now be able to describe the assessment and management of protein-energy malnutrition and common micronutrient deficiencies, investigate malabsorption syndromes including coeliac disease and lactose intolerance, describe the pathophysiology and management of NAFLD and alcoholic liver disease, recognise the clinical features and investigations of Wilson's disease and haemochromatosis, and describe the principles of enteral and parenteral nutrition with the prevention of refeeding syndrome. These abilities align with the Series Learning Outcomes (SLOs 2.1 to 2.5) and equip you to manage nutritional and metabolic gastrointestinal conditions in the Nigerian clinical setting.

Glossary of Terms

- **Coeliac disease:** an immune-mediated small intestinal enteropathy triggered by gluten ingestion in genetically susceptible individuals, causing villous atrophy and malabsorption; treated with a strict lifelong gluten-free diet.
- **Enteral nutrition:** delivery of nutrients directly into the gastrointestinal tract via a tube, used when the gut is functioning but the patient cannot meet nutritional needs orally.
- **Haemochromatosis:** an inherited disorder of iron metabolism (HFE gene mutations) causing excessive iron absorption and accumulation in the liver, pancreas, heart, joints, and skin; treated by regular venesection.
- **Hypophosphataemia:** abnormally low serum phosphate; the key biochemical disturbance in refeeding syndrome, caused by rapid insulin-driven phosphate shift into cells when carbohydrates are reintroduced.

- **Kayser-Fleischer rings:** golden-brown copper deposits in the periphery of the cornea visible on slit-lamp examination; pathognomonic of Wilson's disease with neurological involvement.
- **Kwashiorkor:** a form of protein-energy malnutrition caused predominantly by protein deficiency; characterised by generalised pitting oedema, distended abdomen, and skin and hair changes.
- **Lactase deficiency:** deficiency of the brush border enzyme lactase, causing inability to hydrolyse lactose, resulting in osmotic diarrhoea, bloating, and flatulence after dairy consumption.
- **Marasmus:** a form of protein-energy malnutrition caused by combined protein and caloric deficiency; characterised by severe wasting, absent subcutaneous fat, and absence of oedema.
- **NAFLD:** non-alcoholic fatty liver disease; a spectrum from simple hepatic steatosis to non-alcoholic steatohepatitis (NASH), which can progress to fibrosis, cirrhosis, and hepatocellular carcinoma.
- **Parenteral nutrition:** intravenous delivery of all essential nutrients via a central venous catheter, used when the gastrointestinal tract cannot be used.
- **Refeeding syndrome:** a potentially life-threatening metabolic complication of rapid nutritional reintroduction in malnourished patients, characterised by dangerous hypophosphataemia, hypokalaemia, and hypomagnesaemia.
- **Wilson's disease:** an autosomal recessive disorder of copper metabolism (ATP7B gene) causing copper accumulation in the liver, brain, and eyes; diagnosed by low serum caeruloplasmin and elevated urinary copper; treated with D-penicillamine.

Concept Check Questions [Series 2]

Objective questions

1. _____ presents with generalised oedema and skin changes from predominantly protein deficiency, while marasmus presents with severe wasting from combined protein and caloric deficiency. (Linked to SLO 2.1)
2. Iron deficiency causes _____ hypochromic anaemia with low serum ferritin and elevated total iron-binding capacity. (Linked to SLO 2.1)
3. Coeliac disease causes _____ atrophy and crypt hyperplasia from an immune response to gluten in wheat, rye, and barley. (Linked to SLO 2.2)
4. _____ rings, visible on slit-lamp examination as golden-brown corneal deposits, are pathognomonic of Wilson's disease with neurological involvement. (Linked to SLO 2.4)
5. Refeeding syndrome is caused by rapid reintroduction of carbohydrates causing dangerous _____ as phosphate shifts into cells under insulin stimulation. (Linked to SLO 2.5)

Short-answer questions

6. Distinguish kwashiorkor from marasmus in terms of cause and two clinical features. (Linked to SLO 2.1)
7. Describe the clinical features of vitamin B12 deficiency. (Linked to SLO 2.1)
8. Describe the clinical features of coeliac disease and state the investigation of choice. (Linked to SLO 2.2)
9. Describe the spectrum of alcoholic liver disease. (Linked to SLO 2.3)
10. Explain why parenteral nutrition is delivered via a central venous catheter. (Linked to SLO 2.5)

Long-answer questions

11. Describe the pathophysiology, clinical features, diagnosis, and management of coeliac disease. (Linked to SLO 2.2)
12. Describe the spectrum of non-alcoholic fatty liver disease, including risk factors, diagnosis, and management. (Linked to SLO 2.3)
13. Compare Wilson's disease and hereditary haemochromatosis in terms of inheritance, organs affected, diagnosis, and treatment. (Linked to SLO 2.4)
14. Describe the indications for and differences between enteral and parenteral nutrition. (Linked to SLO 2.5)
15. Explain the pathophysiology of refeeding syndrome, identify patients at risk, and describe its prevention. (Linked to SLO 2.5)

Answers to Concept Check Questions [Series 2]

Objective questions

1. Kwashiorkor.
2. Microcytic.
3. Villous.
4. Kayser-Fleischer.
5. Hypophosphataemia.

Short-answer questions

6. Kwashiorkor results from predominantly protein deficiency and presents with generalised pitting oedema and flaky paint dermatosis. Marasmus results from combined protein and caloric deficiency and presents with severe wasting, visible ribs, and absent subcutaneous fat without oedema.

7. Vitamin B12 deficiency causes megaloblastic anaemia (macrocytic anaemia with hypersegmented neutrophils) and subacute combined degeneration of the spinal cord (dorsal column and corticospinal tract demyelination causing paraesthesiae, weakness, and ataxia). Glossitis and a beefy red tongue are also characteristic.
8. Coeliac disease presents with diarrhoea, steatorrhoea, weight loss, abdominal bloating, and iron and folate deficiency anaemia; in children, failure to thrive is a key feature. The investigation of choice is positive anti-tissue transglutaminase IgA antibodies confirmed by duodenal biopsy showing villous atrophy and crypt hyperplasia.
9. Alcoholic liver disease progresses from alcoholic fatty liver (steatosis, reversible on abstinence) to alcoholic hepatitis (acute inflammatory injury with jaundice, fever, and right upper quadrant pain) and finally to alcoholic cirrhosis (irreversible scarring with portal hypertension, ascites, variceal bleeding, and hepatic encephalopathy).
10. Parenteral nutrition solutions are hyperosmolar and would cause thrombophlebitis and sclerosis of peripheral veins. A central venous catheter (subclavian or internal jugular) delivers the solution into a high-flow central vein where rapid dilution prevents vessel damage.

Long-answer questions

11. **Basic response:** Coeliac disease is caused by an abnormal immune response to gluten (found in wheat, rye, and barley) in individuals carrying HLA-DQ2 or HLA-DQ8. The immune response destroys the villous architecture of the small intestine, causing villous atrophy and crypt hyperplasia that reduce absorptive surface area. Clinical features include diarrhoea, steatorrhoea, weight loss, abdominal bloating, iron and folate deficiency anaemia, and failure to thrive in children. Diagnosis requires positive anti-tissue transglutaminase IgA antibodies followed by duodenal biopsy confirming villous atrophy. Treatment is a strict, lifelong gluten-free diet.

    Value-added response: Complications of untreated coeliac disease include lymphoma of the small intestine (enteropathy-associated T-cell lymphoma), osteoporosis from vitamin

D and calcium malabsorption, and increased risk of other autoimmune diseases such as type 1 diabetes and autoimmune thyroiditis.

12. **Basic response:** NAFLD encompasses a spectrum from simple hepatic steatosis (fat accumulation in hepatocytes without inflammation, benign and reversible) to non-alcoholic steatohepatitis or NASH (steatosis with inflammation and hepatocyte ballooning, which can progress to fibrosis and cirrhosis). Risk factors include obesity, type 2 diabetes, hypertension, dyslipidaemia, and insulin resistance. Most patients are asymptomatic with incidental elevated liver enzymes or bright echogenic liver on ultrasound. Diagnosis is confirmed by ultrasound and liver biopsy (gold standard). Management is primarily lifestyle modification with 7 to 10 percent weight loss improving histology.

    Value-added response: NASH can progress to cirrhosis and hepatocellular carcinoma even in the absence of alcohol use, making it an increasingly important cause of liver transplantation globally. The rising prevalence of obesity and metabolic syndrome in Nigerian urban populations means that NAFLD will become an increasingly important clinical entity in Nigeria.

13. **Basic response:** Wilson's disease is autosomal recessive (ATP7B gene) and causes copper accumulation in the liver, brain, and eyes. Clinical features include hepatitis and cirrhosis, neuropsychiatric manifestations (dysarthria, tremor, psychiatric symptoms), and Kayser-Fleischer rings (pathognomonic). Diagnosis uses low serum caeruloplasmin, elevated 24-hour urinary copper, and slit-lamp examination. Treatment is copper chelation with D-penicillamine or trientine. Hereditary haemochromatosis is autosomal recessive (HFE gene, C282Y mutation) and causes iron accumulation in the liver (cirrhosis), pancreas (diabetes mellitus), heart (cardiomyopathy), joints (arthropathy), and skin (bronzing). Diagnosis uses elevated transferrin saturation above 45 percent and serum ferritin, confirmed by HFE gene testing. Treatment is regular venesection.

    Value-added response: Both conditions cause cirrhosis if untreated, but are entirely preventable and reversible in their early stages with appropriate treatment. Family screening is essential in both conditions because first-degree relatives carry a significant risk of the same mutation.

14. **Basic response:** Enteral nutrition is the delivery of nutrients via a tube into the functioning GI tract. It is indicated when the patient cannot meet nutritional needs orally despite having a functioning gut (dysphagia, reduced consciousness, critical illness). Routes include nasogastric tube for short-term use and PEG for long-term use above 4 to 6 weeks. Parenteral nutrition is intravenous delivery of all nutrients via a central venous catheter, indicated when the gut cannot be used (short bowel syndrome, prolonged ileus, high-output fistula, severe pancreatitis). Enteral nutrition is preferred when feasible because it is cheaper, safer, and preserves gut mucosal integrity.

    Value-added response: Enteral nutrition maintains the intestinal immune barrier and prevents bacterial translocation from the gut into the systemic circulation, which is the mechanism of sepsis in critically ill patients with prolonged bowel rest. Even small volumes of enteral feeding (trophic feeding) provide this benefit when full enteral nutrition is not yet achievable.

15. **Basic response:** During starvation, intracellular stores of phosphate, potassium, and magnesium are depleted while serum levels remain normal. When carbohydrates are reintroduced, insulin secretion surges and drives these electrolytes from the extracellular space into cells, causing dangerous hypophosphataemia, hypokalaemia, and hypomagnesaemia. Hypophosphataemia impairs ATP production, causing cardiac arrhythmias, respiratory failure, haemolytic anaemia, and seizures. Patients at risk include those with BMI below 16, weight loss greater than 15 percent in 3 to 6 months, no nutritional intake for more than 10 days, and pre-feeding electrolyte deficiencies. Prevention requires starting nutrition at 10 kcal per kg per day maximum, increasing slowly over 4 to 7 days, and replacing phosphate, potassium, magnesium, and thiamine before and throughout refeeding.

    Value-added response: Thiamine (vitamin B1) is given before refeeding in all at-risk patients because glucose metabolism requires thiamine as a cofactor; without it, Wernicke's encephalopathy (confusion, ophthalmoplegia, and ataxia) can develop acutely when carbohydrates are reintroduced.

Answers to Pilot Questions [Series 2]

Part 2.1

1. Kwashiorkor results from predominantly protein deficiency and presents with generalised pitting oedema, distended abdomen, flaky paint dermatosis, hair discolouration (flag sign), and anorexia. Marasmus results from combined protein and caloric deficiency and presents with severe wasting, a wizened old-man appearance, visible ribs, absent subcutaneous fat, and large head relative to body, with no oedema.
2. Iron deficiency presents with microcytic hypochromic anaemia (low MCV and MCH), fatigue, pallor, angular stomatitis, koilonychia (spoon-shaped nails), glossitis, and dysphagia from post-cricoid web (Plummer-Vinson syndrome). Laboratory findings show low serum ferritin and elevated total iron-binding capacity.
3. Vitamin A deficiency causes night blindness (the earliest feature), Bitot's spots (foamy white patches on the conjunctiva), xerophthalmia, and corneal ulceration progressing to blindness. It is clinically significant in Nigeria because it is a leading preventable cause of childhood blindness, preventable by routine high-dose vitamin A supplementation.
4. Vitamin B12 deficiency causes megaloblastic anaemia (macrocytic anaemia with hypersegmented neutrophils), subacute combined degeneration of the spinal cord (demyelination of dorsal columns and corticospinal tracts causing paraesthesiae, weakness, and ataxia), and glossitis. The neurological syndrome is subacute combined degeneration of the spinal cord.
5. Nutritional assessment uses the Malnutrition Universal Screening Tool (MUST), which classifies malnutrition risk based on BMI, unintentional weight loss, and the effect of acute disease on nutritional intake. Anthropometric measurements (weight, height, mid-upper arm circumference) and biochemical markers (serum albumin, prealbumin, iron studies, vitamin levels) complete the assessment.

Part 2.2

1. Malabsorption presents with steatorrhoea (pale, bulky, offensive, floating stools), weight loss, abdominal bloating and distension, and deficiencies of fat-soluble vitamins A, D, E, and K. Two causes are coeliac disease (gluten-sensitive enteropathy causing villous atrophy) and tropical sprue (endemic in Nigeria, causing progressive villous atrophy and megaloblastic anaemia).

2. Resection of the terminal ileum impairs vitamin B12 absorption (the terminal ileum is the site of intrinsic factor-vitamin B12 complex absorption), requiring lifelong intramuscular vitamin B12 supplementation. It also impairs bile salt reabsorption, causing bile salt diarrhoea and reduced fat absorption with fat-soluble vitamin deficiencies.

3. Lactose intolerance results from deficiency of lactase, the brush border enzyme that hydrolyses lactose into glucose and galactose. Undigested lactose reaches the colon where bacterial fermentation produces gas and osmotic diarrhoea. Clinical features include bloating, flatulence, cramping abdominal pain, and watery diarrhoea occurring within 30 minutes to 2 hours of dairy consumption.

4. Coeliac disease is caused by an abnormal immune response to gluten in HLA-DQ2 or HLA-DQ8 individuals, destroying villous architecture and causing villous atrophy and crypt hyperplasia that reduce absorptive surface area. Clinical features include diarrhoea, steatorrhoea, weight loss, iron and folate deficiency anaemia, and failure to thrive in children.

5. The investigation of choice is positive anti-tissue transglutaminase IgA antibodies (sensitivity above 95 percent), confirmed by duodenal biopsy on upper GI endoscopy showing villous atrophy and crypt hyperplasia. Treatment is a strict, lifelong gluten-free diet, which leads to mucosal healing and resolution of symptoms in the majority of patients.

Part 2.3

1. NAFLD encompasses simple steatosis (fat accumulation without inflammation, benign and reversible) and non-alcoholic steatohepatitis (NASH: steatosis with inflammation and ballooning, which can progress to fibrosis, cirrhosis, and hepatocellular carcinoma). Risk factors include obesity, type 2 diabetes, hypertension, dyslipidaemia, and insulin resistance.

2. Alcoholic liver disease progresses from alcoholic fatty liver (steatosis, reversible on abstinence) to alcoholic hepatitis (jaundice, fever, right upper quadrant pain, AST:ALT ratio above 2:1) to alcoholic cirrhosis (portal hypertension, ascites, variceal bleeding, encephalopathy). Severe alcoholic hepatitis (Maddrey Discriminant Function above 32) is treated with prednisolone to reduce short-term mortality; abstinence from alcohol is the cornerstone of management at all stages.

3. Wilson's disease presents with hepatic disease (hepatitis, cirrhosis, or fulminant hepatic failure), neuropsychiatric manifestations (dysarthria, tremor, psychiatric symptoms), and Kayser-Fleischer rings (golden-brown copper deposits in the corneal periphery on slit-lamp examination). Kayser-Fleischer rings are pathognomonic when neurological involvement is present.

4. Hereditary haemochromatosis causes iron accumulation in the liver (cirrhosis), pancreas (diabetes mellitus called bronze diabetes), heart (cardiomyopathy and arrhythmias), joints (arthropathy, particularly of the second and third metacarpophalangeal joints), pituitary (hypogonadism), and skin (bronze discolouration).

5. Wilson's disease is diagnosed by low serum caeruloplasmin (below 0.2 g/L in 85 percent of cases), elevated 24-hour urinary copper (above 100 micrograms per day), slit-lamp examination for Kayser-Fleischer rings, and liver biopsy with quantitative copper measurement. Haemochromatosis is diagnosed by elevated transferrin saturation (above 45 percent) and serum ferritin (above 300 micrograms per litre in men), confirmed by HFE gene testing (C282Y homozygosity in 85 percent).

Part 2.4

1. Enteral nutrition is indicated when the patient has a functioning gut but cannot meet nutritional needs orally: dysphagia (stroke, head and neck cancer), reduced consciousness, critical illness with inadequate oral intake, and severe malnutrition in inflammatory bowel disease. Routes include nasogastric tube (short-term use up to 4 to 6 weeks), nasojejunal tube (high aspiration risk), percutaneous endoscopic gastrostomy (long-term use beyond 4 to 6 weeks), and jejunostomy (after oesophagogastric surgery).
2. Parenteral nutrition is indicated when the gastrointestinal tract cannot be used: short bowel syndrome with insufficient absorptive capacity, prolonged paralytic ileus, high-output intestinal fistula, severe acute pancreatitis with intestinal failure, and major GI surgery with post-operative ileus. It is administered via a central venous catheter (subclavian or internal jugular vein) because the hyperosmolar solution would cause thrombophlebitis in peripheral veins.
3. Metabolic complications of parenteral nutrition include hyperglycaemia (from the high glucose content of PN solutions, requiring insulin sliding scale), hyperlipidaemia (from lipid emulsion), electrolyte disturbances (hypophosphataemia, hypokalaemia, hypomagnesaemia), and hepatic cholestasis with steatosis in long-term use (caused by continuous glucose infusion and lack of enteral stimulation of bile flow).
4. During starvation, intracellular stores of phosphate, potassium, and magnesium are depleted while serum levels remain near-normal. When carbohydrates are reintroduced, insulin surges and drives these electrolytes from the extracellular space into cells, causing severe hypophosphataemia, hypokalaemia, and hypomagnesaemia. Hypophosphataemia impairs ATP synthesis in all cells, leading to cardiac arrhythmias, respiratory muscle failure, haemolytic anaemia, and seizures.
5. Patients at high risk of refeeding syndrome include those with BMI below 16 kg/m^2, unintentional weight loss greater than 15 percent in 3 to 6 months, no or minimal nutritional intake for more than 10 days, and low pre-feeding levels of phosphate, potassium, or magnesium (any one of these identifies high risk).

References and Attributions [Series 2]

Textual References

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- National Institute for Health and Care Excellence. (2006). Nutrition Support for Adults: Oral Nutrition Support, Enteral Tube Feeding and Parenteral Nutrition. NICE Clinical Guideline 32. London: NICE.

Figures, Plates, Tables, and Boxes

- Table 2.1: Clinical features distinguishing kwashiorkor from marasmus. AI-generated using the prompt: "Create a clean two-column comparison table titled Clinical Features of Protein-Energy Malnutrition. Left column: Kwashiorkor. Right column: Marasmus. Include rows for cause, oedema, skin and hair changes, and nutritional status." Suggested OER search string: "kwashiorkor marasmus comparison table clinical features protein energy malnutrition OER."
- Table 2.2: Comparison of lactose intolerance and coeliac disease. AI-generated using the prompt: "Create a clear two-column comparison table titled Comparison of Lactose Intolerance and Coeliac Disease with rows for mechanism, clinical features, diagnosis, and treatment." Suggested OER search string: "lactose intolerance coeliac disease comparison table mechanism diagnosis treatment OER."
- Table 2.3: Comparison of Wilson's disease and hereditary haemochromatosis. AI-generated using the prompt: "Create a clear two-column comparison table titled Wilson's

Disease versus Hereditary Haemochromatosis with rows for inheritance and gene, accumulated substance, organs affected, diagnosis, and treatment." Suggested OER search string: "Wilson's disease haemochromatosis comparison table inheritance diagnosis treatment OER."

- Table 2.4: Comparison of enteral and parenteral nutrition. AI-generated using the prompt: "Create a clear two-column comparison table titled Enteral versus Parenteral Nutrition with rows for definition, indication, route, advantages, monitoring, and complications." Suggested OER search string: "enteral parenteral nutrition comparison table indication route complications OER."

Series 3: Upper and Lower GI Diseases

- **Part 3.1: Peptic Ulcer Disease and Gastro-Oesophageal Reflux**
    - Sub Part 3.1.1: Peptic ulcer disease
    - Sub Part 3.1.2: Gastro-oesophageal reflux disease
    - Sub Part 3.1.3: Complications and management
- **Part 3.2: Inflammatory Bowel Disease**
    - Sub Part 3.2.1: Crohn's disease
    - Sub Part 3.2.2: Ulcerative colitis
    - Sub Part 3.2.3: Complications and management of IBD
- **Part 3.3: Colorectal Disease**
    - Sub Part 3.3.1: Colorectal carcinoma
    - Sub Part 3.3.2: Diverticular disease
    - Sub Part 3.3.3: Anorectal disorders
- **Part 3.4: Intestinal Infections**
    - Sub Part 3.4.1: Acute infective gastroenteritis
    - Sub Part 3.4.2: Typhoid fever
    - Sub Part 3.4.3: Intestinal parasitic infections

Introduction

Upper and lower gastrointestinal diseases are among the most commonly encountered conditions in clinical practice in Nigeria. From peptic ulcer disease and gastro-oesophageal reflux disease, which are driven by Helicobacter pylori infection and dietary habits, to inflammatory bowel

disease, colorectal malignancy, and intestinal infections, the range of conditions is broad and the clinical consequences can be severe. An understanding of the pathophysiology, clinical features, and management of these diseases is essential for any clinician.

This Series covers the major upper and lower GI diseases systematically. We shall commence with peptic ulcer disease and gastro-oesophageal reflux disease, examining their pathogenesis, clinical features, complications, and management. Next, we will explore inflammatory bowel disease, distinguishing Crohn's disease from ulcerative colitis and describing their management. Moving on, we will consider colorectal disease, covering colorectal carcinoma, diverticular disease, and anorectal disorders. Finally, we will address intestinal infections, covering acute gastroenteritis, typhoid fever, and the major intestinal parasitic infections relevant to Nigeria.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 3.1:** describe the pathogenesis, clinical features, complications, and management of peptic ulcer disease and GORD,
- **SLO 3.2:** distinguish between Crohn's disease and ulcerative colitis and describe their management,
- **SLO 3.3:** describe the clinical features, investigation, and management of colorectal carcinoma and diverticular disease,
- **SLO 3.4:** describe the clinical features and management of common anorectal disorders, and
- **SLO 3.5:** describe the clinical features, diagnosis, and management of acute gastroenteritis, typhoid fever, and intestinal parasitic infections in Nigeria.

3.1 Peptic Ulcer Disease and Gastro-Oesophageal Reflux

3.1.1 Peptic ulcer disease

Peptic ulcer disease (PUD) is characterised by mucosal ulceration in the stomach or duodenum from an imbalance between damaging and protective factors. **Helicobacter pylori** infection (a Gram-negative spiral bacterium that colonises the gastric mucosa, causes chronic gastritis, and disrupts the mucus-bicarbonate barrier) is the causative factor in approximately 90 percent of duodenal ulcers and 70 percent of gastric ulcers in Nigeria. The second most important cause is use of **non-steroidal anti-inflammatory drugs (NSAIDs)**, which inhibit cyclo-oxygenase and reduce prostaglandin-mediated mucus and bicarbonate secretion.

Duodenal ulcers present with epigastric pain that is relieved by food and antacids and worsens 2 to 3 hours after meals (hunger pain). Gastric ulcers present with epigastric pain worsened by food. Both may present with nausea and anorexia. Diagnosis is by upper GI endoscopy (OGD), which allows direct visualisation, biopsy, and testing for H. pylori (rapid urease test on biopsy). H. pylori can also be detected non-invasively by the urea breath test and stool antigen test.

H. pylori causes approximately 90 percent of _____ ulcers and 70 percent of gastric ulcers. Duodenal ulcer pain is characteristically relieved by _____ and antacids.

3.1.2 Gastro-oesophageal reflux disease

Gastro-oesophageal reflux disease (GORD) results from reflux of gastric acid into the oesophagus due to incompetence of the **lower oesophageal sphincter (LOS)**. Predisposing factors include obesity, pregnancy, hiatus hernia, smoking, alcohol, fatty foods, and caffeine. The cardinal symptom is **heartburn** (retrosternal burning worse after meals and on lying down), often with **acid regurgitation** (sour or bitter taste in the mouth). Atypical symptoms include chronic cough, hoarseness, and asthma-like symptoms from laryngopharyngeal reflux.

Oesophagogastroduodenoscopy identifies oesophagitis (erythema, erosions, or ulceration), hiatus hernia, and Barrett's oesophagus. Barrett's oesophagus (columnar metaplasia of the lower oesophageal mucosa from chronic acid exposure) is a premalignant condition that requires endoscopic surveillance because of the risk of progression to oesophageal adenocarcinoma. 24-hour oesophageal pH monitoring is the gold standard investigation to quantify acid exposure.

GORD results from incompetence of the _____ oesophageal sphincter. _____ oesophagus is columnar metaplasia of the lower oesophagus and is a premalignant complication of chronic GORD.

3.1.3 Complications and management

Complications of PUD include: **haemorrhage** (the most common: presents as haematemesis or melaena, managed by endoscopic haemostasis), **perforation** (sudden severe epigastric pain with peritonitis and free gas under the diaphragm on erect chest X-ray: surgical emergency), and **gastric outlet obstruction** (from pyloric scarring: persistent vomiting of undigested food, succussion splash, weight loss: treated endoscopically or surgically). H. pylori eradication using **triple therapy** (proton pump inhibitor plus clarithromycin plus amoxicillin for 7 to 14 days) achieves ulcer healing in over 90 percent of cases.

Management of GORD: lifestyle modification (weight loss, elevating the head of the bed, avoiding fatty food, caffeine, alcohol, and smoking), proton pump inhibitors (PPIs: omeprazole 20 to 40 mg daily: the most effective treatment), and H2 receptor antagonists (ranitidine) for milder symptoms. Laparoscopic Nissen fundoplication is the surgical option for patients with refractory GORD or large hiatus hernia.

The most common complication of PUD is _____, while perforation presents with sudden severe pain and free gas under the diaphragm. H. pylori eradication uses _____ therapy with a PPI plus two antibiotics.

Peptic Ulcer Disease (PUD)	Gastro-oesophageal Reflux Disease (GORD)
Cause: • *H. pylori* (≈90% of duodenal ulcers) • NSAIDs	**Cause**: • Lower oesophageal sphincter incompetence • Predisposed by obesity, pregnancy, hiatus hernia, smoking, fatty food

Peptic Ulcer Disease (PUD)	Gastro-oesophageal Reflux Disease (GORD)
Clinical features: • Duodenal ulcer: epigastric pain relieved by food • Gastric ulcer: pain worsened by food	**Clinical features**: • Heartburn • Acid regurgitation • Atypical: cough, hoarseness
Investigations: • OGD with biopsy and rapid urease test • Urea breath test • Stool antigen test	**Investigations**: Primarily clinical; OGD if complications suspected
Complications: • Haemorrhage (most common) • Perforation • Gastric outlet obstruction	**Complications**: • Oesophagitis • Barrett's oesophagus • Oesophageal adenocarcinoma
Treatment: • *H. pylori* eradication (triple therapy: PPI + clarithromycin + amoxicillin for 7–14 days) • PPI for NSAID-related ulcers	**Treatment**: • Lifestyle modification • PPIs, H2 antagonists • Nissen fundoplication for refractory cases

Table 3.1: Comparison of peptic ulcer disease and gastro-oesophageal reflux disease

Pilot questions 3.1

1. State the two most common causes of peptic ulcer disease and describe how each damages the gastric mucosa. (Linked to SLO 3.1)
2. Distinguish the pain of duodenal ulcer from that of gastric ulcer. (Linked to SLO 3.1)
3. Describe the clinical features of GORD and state its premalignant complication. (Linked to SLO 3.1)
4. Describe the three major complications of peptic ulcer disease. (Linked to SLO 3.1)
5. Describe the treatment of H. pylori infection and the management of GORD. (Linked to SLO 3.1)

3.2 Inflammatory Bowel Disease

3.2.1 Crohn's disease

Crohn's disease is a chronic, relapsing, transmural inflammatory disease that can affect any part of the gastrointestinal tract from mouth to anus, most commonly the terminal ileum and proximal colon. The hallmark pathological feature is **discontinuous (skip) lesions** with areas of normal bowel between inflamed segments, **transmural inflammation** affecting all layers of the bowel wall, and **non-caseating granulomas** on biopsy. Clinical features include cramping abdominal pain (most often in the right iliac fossa from terminal ileal involvement), diarrhoea (which may not be bloody), weight loss, fever, and perianal disease (fistulae, abscesses, and skin tags).

Extra-intestinal manifestations are common and include erythema nodosum (tender red nodules on the shins), pyoderma gangrenosum (deep ulcers on the legs), uveitis, episcleritis, primary sclerosing cholangitis, and peripheral arthropathy. Investigations include full blood count (anaemia, leucocytosis), CRP and ESR (elevated in active disease), faecal calprotectin (elevated marker of intestinal inflammation), colonoscopy with biopsy (skip lesions, cobblestone mucosa), and CT or MRI enterography (small bowel involvement, fistulae, abscesses).

Crohn's disease causes _____ (skip) lesions with transmural inflammation and non-caseating granulomas, most commonly affecting the _____ ileum and proximal colon.

3.2.2 Ulcerative colitis

Ulcerative colitis (UC) is a chronic relapsing inflammatory disease confined to the **colonic mucosa and submucosa** (unlike Crohn's, which is transmural). It always begins at the rectum and extends proximally in a **continuous (unbroken) pattern**. The cardinal symptom is **bloody diarrhoea** with mucus, urgency, and tenesmus. Other features include cramping lower abdominal pain relieved by defaecation, weight loss, and anaemia.

Investigations show a similar pattern to Crohn's but colonoscopy reveals continuous mucosal inflammation from the rectum, with loss of haustra, granular friable mucosa, and pseudopolyps in chronic disease. Biopsy shows crypt abscesses and goblet cell depletion. UC carries a risk of colorectal carcinoma that increases with the duration and extent of disease, requiring

colonoscopic surveillance. Toxic megacolon (acute colonic dilatation above 6 cm on plain X-ray with systemic toxicity) is a life-threatening emergency.

UC begins at the _____ and extends proximally in a continuous pattern, causing bloody diarrhoea. Toxic megacolon is defined as acute colonic dilatation above _____ cm with systemic toxicity.

3.2.3 Complications and management of IBD

Both Crohn's disease and UC are managed using a step-up approach. Mild to moderate disease: aminosalicylates (mesalazine: first-line for UC; less effective in Crohn's), corticosteroids (prednisolone or budesonide for acute flares). Moderate to severe disease: azathioprine or 6-mercaptopurine (steroid-sparing immunomodulators), methotrexate (for Crohn's). Severe or refractory disease: biological agents including anti-TNF therapy (infliximab, adalimumab) and anti-integrin therapy (vedolizumab).

Surgical management: UC can be cured by colectomy with ileal pouch-anal anastomosis. Crohn's disease cannot be cured by surgery (recurrence after resection is common), so surgery is reserved for complications including stricture, fistula, abscess, or failure of medical therapy. Nutritional support (enteral or parenteral) is important in severe Crohn's disease with malnutrition.

UC can be cured by _____ with ileal pouch-anal anastomosis. Anti-TNF agents such as _____ and adalimumab are used for severe or refractory IBD.

Crohn's Disease	Ulcerative Colitis
Location: Any part of GI tract, most commonly terminal ileum and proximal colon	**Location**: Colon and rectum only
Pattern: Discontinuous "skip lesions"	**Pattern**: Continuous from rectum proximally

Crohn's Disease	Ulcerative Colitis
Depth of inflammation: Transmural	**Depth of inflammation**: Mucosal and submucosal only
Pathology: Non-caseating granulomas	**Pathology**: Crypt abscesses, goblet cell depletion, pseudopolyps
Symptoms: Right iliac fossa pain, diarrhoea (bloody or non-bloody), weight loss, perianal disease	**Symptoms**: Bloody diarrhoea with mucus, urgency, tenesmus, lower abdominal pain
Extra-intestinal manifestations: Erythema nodosum, pyoderma gangrenosum, uveitis, arthropathy, primary sclerosing cholangitis (PSC)	**Complications**: Toxic megacolon, increased colorectal carcinoma risk
Surgery: Not curative; reserved for complications	**Surgery**: Curative colectomy with ileal pouch-anal anastomosis

Table 3.2: Comparison of Crohn's disease and ulcerative colitis

Pilot questions 3.2

1. Describe the pathological features and clinical presentation of Crohn's disease. (Linked to SLO 3.2)
2. Describe the clinical features and colonoscopic findings in ulcerative colitis. (Linked to SLO 3.2)
3. Distinguish Crohn's disease from ulcerative colitis in terms of location, depth of inflammation, and pattern. (Linked to SLO 3.2)
4. Describe the step-up approach to the medical management of IBD. (Linked to SLO 3.2)
5. State the role of surgery in Crohn's disease and ulcerative colitis. (Linked to SLO 3.2)

3.3 Colorectal Disease

3.3.1 Colorectal carcinoma

Colorectal carcinoma (CRC) is the third most common cancer globally and is increasing in Nigeria with the adoption of Western dietary habits. The majority are **adenocarcinomas** arising from adenomatous polyps (the adenoma-carcinoma sequence: normal mucosa to adenoma to carcinoma). Risk factors include a high-fat, low-fibre diet, obesity, physical inactivity, smoking, alcohol, a family history of CRC or adenomatous polyps, inflammatory bowel disease, and hereditary syndromes (familial adenomatous polyposis, Lynch syndrome).

Clinical features depend on tumour location: right-sided (caecal and ascending colon) tumours present with iron deficiency anaemia (from occult bleeding), weight loss, and a right iliac fossa mass; left-sided (descending and sigmoid colon) tumours present with change in bowel habit (alternating constipation and diarrhoea), rectal bleeding, and colicky lower abdominal pain. Rectal tumours present with fresh rectal bleeding, tenesmus, and a change in bowel habit. Investigations include colonoscopy with biopsy (gold standard), CT colonography, carcinoembryonic antigen (CEA), and CT chest, abdomen, and pelvis for staging.

CRC arises from adenomatous polyps via the _____ sequence. Right-sided CRC presents with iron deficiency anaemia, while left-sided CRC presents with _____ in bowel habit and rectal bleeding.

3.3.2 Diverticular disease

Diverticular disease refers to the presence of diverticula (pouches formed by herniation of the colonic mucosa through weak points in the muscularis), which occur most commonly in the **sigmoid colon**. The condition is rare before age 40 and becomes increasingly common with age, related to a low-fibre diet that increases intraluminal pressure. Most diverticula are asymptomatic (diverticulosis).**Diverticulitis** (inflammation or infection of a diverticulum) presents with left iliac fossa pain, fever, nausea, and a change in bowel habit.

Complications of diverticulitis include abscess (pericolic or pelvic), perforation (causing peritonitis), fistula formation (colovesical fistula presents with pneumaturia and faecaluria), and

large bowel obstruction. Uncomplicated diverticulitis is managed conservatively with oral antibiotics (metronidazole and ciprofloxacin), a liquid diet, and analgesia. Complicated diverticulitis (abscess, perforation, or obstruction) requires hospital admission, intravenous antibiotics, and surgical intervention.

Diverticula occur most commonly in the _____ colon. A colovesical fistula from diverticulitis presents with _____ (air in the urine).

3.3.3 Anorectal disorders

Haemorrhoids (piles) are enlarged vascular cushions in the anal canal, classified as internal (above the dentate line: painless unless prolapsed or thrombosed) or external (below the dentate line: painful, especially when thrombosed). They present with **bright red rectal bleeding** (fresh blood on the toilet paper or dripping into the pan after defaecation), anal pruritus, and in prolapsed haemorrhoids, a feeling of incomplete evacuation.**Anal fissure** is a tear in the anoderm below the dentate line, causing severe anal pain during and after defaecation and bright red bleeding on the toilet paper. Most fissures are posterior and associated with hard stools.

Management of haemorrhoids: high-fibre diet and adequate fluid intake for all grades; rubber band ligation (the most effective outpatient treatment for grades I to III internal haemorrhoids); surgical haemorrhoidectomy for grade IV or recurrent symptomatic haemorrhoids. Anal fissure management: topical glyceryl trinitrate (GTN) or diltiazem to relax the internal anal sphincter and promote healing; botulinum toxin injection; lateral internal sphincterotomy for chronic refractory fissures.

Internal haemorrhoids are above the _____ line and are painless unless prolapsed. Anal fissures are managed with topical _____ trinitrate to relax the internal anal sphincter.

Colorectal Carcinoma & Diverticular Disease	Anorectal Disorders
Colorectal Carcinoma (CRC): • Pathogenesis: adenoma–carcinoma sequence • Right-sided: iron deficiency anaemia, abdominal mass •	**Haemorrhoids**: • Internal: above dentate line, painless unless prolapsed • External: below dentate line, painful if thrombosed •

Colorectal Carcinoma & Diverticular Disease	Anorectal Disorders
Left-sided: change in bowel habit, rectal bleeding • Investigations: colonoscopy with biopsy, CT for staging • Treatment: surgical resection; chemotherapy for metastatic disease	Features: bright red rectal bleeding, pruritus • Treatment: high-fibre diet, rubber band ligation (grades I–III), haemorrhoidectomy (grade IV)
Diverticular Disease: • Location: sigmoid colon • Diverticulosis: often asymptomatic • Diverticulitis: left iliac fossa pain, fever • Complications: abscess, perforation, colovesical fistula, obstruction • Treatment: antibiotics for uncomplicated cases; surgery for complicated disease	**Anal Fissure**: • Definition: tear in anoderm below dentate line • Features: severe pain during defaecation, posterior location most common • Treatment: topical GTN or diltiazem, botulinum toxin injection, lateral internal sphincterotomy for refractory cases

Table 3.3: Common colorectal and anorectal conditions

Pilot questions 3.3

1. Describe the adenoma-carcinoma sequence and state the risk factors for CRC. (Linked to SLO 3.3)
2. Distinguish the clinical presentation of right-sided from left-sided colorectal carcinoma. (Linked to SLO 3.3)
3. Describe the complications of diverticulitis. (Linked to SLO 3.3)
4. Describe the clinical features and classification of haemorrhoids. (Linked to SLO 3.4)
5. Describe the management of an anal fissure. (Linked to SLO 3.4)

3.4 Intestinal Infections

3.4.1 Acute infective gastroenteritis

Acute infective gastroenteritis is characterised by the sudden onset of diarrhoea (three or more loose stools per day), nausea, vomiting, and abdominal cramping from ingestion of contaminated food or water. **Viral gastroenteritis** (rotavirus in children, norovirus in adults: the most common causes globally) is self-limiting and managed with oral rehydration. **Bacterial gastroenteritis** includes Salmonella (undercooked poultry; non-typhoidal; watery or bloody diarrhoea), Shigella (dysentery: bloody diarrhoea with mucus and fever; requires antibiotics), Campylobacter (undercooked chicken; cramping abdominal pain and bloody diarrhoea; ciprofloxacin), Vibrio cholerae (profuse watery rice-water stools causing life-threatening dehydration; immediate oral or IV rehydration is the priority), and enterotoxigenic Escherichia coli (traveller's diarrhoea).

Management: oral rehydration solution (ORS) is the cornerstone of treatment for all diarrhoeal illnesses. Stool microscopy and culture identify the organism. Antibiotics are indicated for Shigella (ciprofloxacin), cholera (doxycycline reduces disease duration), severe Salmonella (ciprofloxacin in immunocompromised or severe illness), and Campylobacter (azithromycin or ciprofloxacin). Antidiarrhoeal agents (loperamide) are avoided in dysentery and bloody diarrhoea.

Oral _____ solution is the cornerstone of management of acute gastroenteritis. _____ (rice-water stools) requires immediate rehydration as cholera can cause life-threatening fluid loss.

3.4.2 Typhoid fever

Typhoid fever is caused by **Salmonella typhi** (and to a lesser extent S. paratyphi), transmitted via faecal-oral route through contaminated food and water. It is endemic in Nigeria and represents a major public health problem. The clinical course: **Week 1** (stepwise fever, headache, malaise, relative bradycardia (Faget's sign), rose spots on the trunk in 30 percent of patients), **Week 2** (fever plateaus; abdominal distension and constipation or diarrhoea; splenomegaly;

hepatomegaly), and **Week 3** (complications: intestinal perforation, haemorrhage, encephalopathy, myocarditis).

Diagnosis: blood culture (positive in 80 percent in the first week; the gold standard), bone marrow culture (the most sensitive: positive even after antibiotics), stool culture (week 2 onwards), and Widal test (rising antibody titres, but low specificity in endemic areas). Treatment: ceftriaxone (IV, the treatment of choice in Nigeria given rising ciprofloxacin resistance), azithromycin (oral alternative), or ciprofloxacin (where sensitive). Chloramphenicol, ampicillin, and cotrimoxazole are no longer first-line due to widespread resistance. Prevention: safe water, sanitation, and the Vi polysaccharide typhoid vaccine.

Typhoid fever is caused by Salmonella _____ and is transmitted via the faecal-oral route. _____ culture is the gold standard investigation in the first week of illness.

3.4.3 Intestinal parasitic infections

Entamoeba histolytica causes **amoebic dysentery** (bloody diarrhoea with mucus, tenesmus, and lower abdominal pain) and **amoebic liver abscess** (right upper quadrant pain, fever, and elevation of the right hemidiaphragm on CXR; aspirate yields anchovy sauce pus). Diagnosis is by stool microscopy (trophozoites or cysts), stool antigen test, serology (amoebic ELISA), and abdominal ultrasound (liver abscess). Treatment is metronidazole followed by diloxanide furoate (to eradicate luminal cysts).

Giardia lamblia causes giardiasis, the most common intestinal protozoan infection globally: characterised by chronic, foul-smelling, fatty diarrhoea (steatorrhoea), abdominal bloating, and flatulence without blood or mucus. Diagnosis is by stool microscopy (cysts or trophozoites) or stool antigen test; treatment is metronidazole or tinidazole. Ascaris lumbricoides (the most common intestinal worm globally and in Nigeria), hookworm (Ancylostoma duodenale and Necator americanus, causing iron deficiency anaemia from intestinal blood loss), and Strongyloides stercoralis are managed with albendazole or mebendazole.

Amoebic liver abscess contains _____ sauce pus on aspiration. Giardia causes fatty diarrhoea without blood from _____ (fat malabsorption); both are treated with metronidazole.

Bacterial Infections	Parasitic Infections
Cholera **(Vibrio cholerae) • Profuse rice-water stools • Life-threatening dehydration • Treatment: ORS + doxycycline**	Entamoeba histolytica • Amoebic dysentery: bloody diarrhoea • Amoebic liver abscess: RUQ pain, anchovy sauce pus • Diagnosis: stool microscopy, serology, ultrasound • Treatment: metronidazole → diloxanide furoate
Typhoid Fever **(Salmonella typhi) • Faecal-oral route • Stepwise fever, relative bradycardia, rose spots • Complications: perforation, haemorrhage (week 3) • Gold standard: blood culture • Treatment: ceftriaxone or azithromycin**	Giardia lamblia • Fatty, offensive diarrhoea (no blood) • Steatorrhoea, bloating • Diagnosis: stool microscopy or antigen test • Treatment: metronidazole
Shigella Dysentery **• Bloody diarrhoea with mucus and fever • Treatment: ciprofloxacin**	Helminths (Ascaris, hookworm, Strongyloides) • Common in Nigeria • Hookworm → iron deficiency anaemia • Treatment: albendazole or mebendazole

Table 3.4: Common intestinal infections in Nigeria

Pilot questions 3.4

1. State four bacterial causes of acute gastroenteritis and describe one distinguishing feature of each. (Linked to SLO 3.5)
2. Describe the clinical course of typhoid fever by week. (Linked to SLO 3.5)
3. State the gold standard investigation for typhoid fever and explain why the Widal test has limitations. (Linked to SLO 3.5)

4. Describe the clinical features and treatment of amoebic dysentery and amoebic liver abscess. (Linked to SLO 3.5)
5. Describe the clinical features and treatment of giardiasis. (Linked to SLO 3.5)

Series Summary

This Series covered the major upper and lower GI diseases. We began with peptic ulcer disease (caused by H. pylori and NSAIDs, presenting with epigastric pain, treated with triple therapy) and GORD (LOS incompetence causing heartburn and acid regurgitation, with Barrett's oesophagus as the premalignant complication, managed with PPIs). Next, we examined IBD, distinguishing Crohn's disease (transmural, skip lesions, non-caseating granulomas, perianal disease) from ulcerative colitis (mucosal, continuous from rectum, bloody diarrhoea, curable by colectomy), and described the step-up medical management. Moving on, we covered colorectal carcinoma (adenoma-carcinoma sequence; right-sided: anaemia; left-sided: change in bowel habit), diverticular disease (sigmoid colon, low-fibre diet, complications include abscess and colovesical fistula), and anorectal disorders (haemorrhoids: rubber band ligation; anal fissure: topical GTN). Finally, we addressed intestinal infections, covering the management of acute gastroenteritis, the clinical course and treatment of typhoid fever, and the features and treatment of amoebic dysentery, amoebic liver abscess, and giardiasis.

By completing this Series, you should now be able to describe the pathogenesis, complications, and management of PUD and GORD, distinguish Crohn's disease from ulcerative colitis, describe colorectal carcinoma and diverticular disease, manage common anorectal disorders, and diagnose and treat the major intestinal infections in Nigeria. These abilities align with SLOs 3.1 to 3.5 and equip you to manage upper and lower GI disease in clinical practice.

Glossary of Terms

- **Adenoma-carcinoma sequence:** the stepwise progression from normal colonic mucosa through adenomatous polyp to invasive adenocarcinoma, driven by accumulated genetic mutations.
- **Barrett's oesophagus:** columnar metaplasia of the lower oesophageal mucosa from chronic acid exposure; a premalignant condition requiring endoscopic surveillance for progression to oesophageal adenocarcinoma.

- **Diverticulitis:** inflammation or infection of a colonic diverticulum, presenting with left iliac fossa pain, fever, and altered bowel habit; complications include abscess, perforation, and colovesical fistula.
- **Faget's sign:** relative bradycardia (a lower heart rate than expected for the degree of fever) seen in typhoid fever and other specific infections.
- **Faecal calprotectin:** a stool biomarker of intestinal inflammation, elevated in IBD and used to distinguish inflammatory from functional bowel disorders.
- **Haemorrhoids:** enlarged vascular cushions in the anal canal, classified as internal (above the dentate line) or external (below the dentate line); present with bright red rectal bleeding and pruritus.
- **Helicobacter pylori:** a Gram-negative spiral bacterium that colonises the gastric mucosa, causes chronic gastritis, and is responsible for approximately 90 percent of duodenal ulcers; eradicated by triple therapy.
- **Lynch syndrome:** an autosomal dominant hereditary cancer syndrome caused by mutations in DNA mismatch repair genes, conferring a markedly elevated risk of colorectal and endometrial carcinoma.
- **Toxic megacolon:** acute dilatation of the colon above 6 cm with systemic toxicity, complicating severe ulcerative colitis or Crohn's colitis; a surgical emergency.
- **Triple therapy:** H. pylori eradication regimen comprising a proton pump inhibitor plus clarithromycin plus amoxicillin (or metronidazole) for 7 to 14 days; achieves eradication in over 90 percent of cases.
- **Urea breath test:** a non-invasive test for active H. pylori infection: the patient ingests labelled urea, which is hydrolysed by H. pylori urease to produce labelled carbon dioxide detected in expired breath.

Concept Check Questions [Series 3]

Objective questions

1. H. pylori causes approximately 90 percent of _____ ulcers and is the most important cause of peptic ulcer disease. (Linked to SLO 3.1)
2. _____ oesophagus is a premalignant complication of chronic GORD caused by columnar metaplasia of the lower oesophageal mucosa. (Linked to SLO 3.1)
3. Crohn's disease causes _____ (skip) lesions with transmural inflammation and non-caseating granulomas on biopsy. (Linked to SLO 3.2)
4. Typhoid fever is caused by Salmonella _____ and is transmitted via the faecal-oral route through contaminated food and water. (Linked to SLO 3.5)
5. Amoebic liver abscess contains _____ sauce pus on aspiration and is diagnosed by abdominal ultrasound and amoebic serology. (Linked to SLO 3.5)

Short-answer questions

6. Describe how H. pylori causes peptic ulcer disease. (Linked to SLO 3.1)
7. Distinguish Crohn's disease from ulcerative colitis in terms of location, depth of inflammation, and pattern of disease. (Linked to SLO 3.2)
8. Describe the clinical presentation of right-sided colorectal carcinoma. (Linked to SLO 3.3)
9. Describe the clinical features of typhoid fever in the first two weeks. (Linked to SLO 3.5)
10. State the treatment for H. pylori eradication and the first-line treatment for typhoid fever in Nigeria. (Linked to SLOs 3.1 and 3.5)

Long-answer questions

11. Describe the pathogenesis, clinical features, complications, and management of peptic ulcer disease. (Linked to SLO 3.1)
12. Compare Crohn's disease and ulcerative colitis, and describe the step-up approach to the management of IBD. (Linked to SLO 3.2)
13. Describe the investigation and management of colorectal carcinoma. (Linked to SLO 3.3)
14. Describe the diagnosis and management of typhoid fever in Nigeria, including the gold standard investigation and treatment. (Linked to SLO 3.5)
15. Describe the clinical features, diagnosis, and treatment of intestinal amoebiasis and giardiasis. (Linked to SLO 3.5)

Answers to Concept Check Questions [Series 3]

Objective questions

1. Duodenal.
2. Barrett's.
3. Discontinuous.
4. Typhi.
5. Anchovy.

Short-answer questions

6. H. pylori colonises the gastric mucosa and produces urease, which generates ammonia that damages the mucus-bicarbonate barrier. It also triggers chronic gastritis and

inflammatory cytokine release, which increases acid secretion while impairing mucosal defence, resulting in ulceration.

7. Crohn's disease can affect any part of the GI tract from mouth to anus, most commonly the terminal ileum and proximal colon, and causes transmural inflammation with discontinuous skip lesions. Ulcerative colitis is confined to the colon and rectum, always begins at the rectum and extends proximally in a continuous pattern, and causes mucosal and submucosal inflammation only.

8. Right-sided colorectal carcinoma typically presents insidiously with iron deficiency anaemia from chronic occult bleeding, weight loss, and occasionally a palpable right iliac fossa mass. Because the caecum and ascending colon have a wide lumen, obstruction is a late feature and the diagnosis is often delayed.

9. Week 1 presents with stepwise rising fever, headache, malaise, relative bradycardia (Faget's sign), and rose spots on the trunk in approximately 30 percent of patients. Week 2 sees fever plateau, with abdominal distension, constipation or diarrhoea, splenomegaly, and hepatomegaly.

10. H. pylori eradication uses triple therapy: a proton pump inhibitor plus clarithromycin plus amoxicillin for 7 to 14 days. First-line treatment for typhoid fever in Nigeria is intravenous ceftriaxone, due to widespread resistance to ciprofloxacin, ampicillin, and chloramphenicol.

Long-answer questions

11. **Basic response:** PUD results from an imbalance between mucosal damaging factors (H. pylori in 90 percent of duodenal ulcers, NSAIDs inhibiting prostaglandin synthesis) and protective factors (mucus, bicarbonate, mucosal blood flow). Duodenal ulcer presents with epigastric pain relieved by food; gastric ulcer pain is worsened by food. Complications include haemorrhage (the most common, managed by endoscopic haemostasis), perforation (surgical emergency with free gas on erect CXR), and gastric outlet obstruction (from pyloric scarring, presenting with vomiting of undigested food).

H. pylori is eradicated with triple therapy (PPI plus clarithromycin plus amoxicillin). NSAID-induced ulcers are managed by stopping the NSAID and prescribing a PPI.

Value-added response: In Nigeria, H. pylori prevalence exceeds 70 percent in symptomatic patients, and eradication dramatically reduces ulcer recurrence. A urea breath test or stool antigen test 4 to 6 weeks after completing treatment confirms successful eradication.

12. **Basic response:** Crohn's disease causes transmural skip lesions with non-caseating granulomas, most commonly in the terminal ileum, presenting with right iliac fossa pain, non-bloody diarrhoea, weight loss, and perianal disease. UC causes continuous mucosal inflammation from the rectum proximally, presenting with bloody diarrhoea, urgency, and tenesmus, with crypt abscesses on biopsy. Both are managed with a step-up approach: mild to moderate disease uses aminosalicylates (mesalazine, first-line for UC) and corticosteroids for flares; moderate to severe disease uses azathioprine or 6-mercaptopurine; severe or refractory disease uses anti-TNF agents (infliximab, adalimumab). UC can be cured by colectomy with ileal pouch-anal anastomosis; surgery for Crohn's is reserved for complications and is not curative.

Value-added response: Biological agents have transformed IBD management: anti-TNF therapy achieves mucosal healing and reduces steroid dependence in moderate to severe disease, and anti-integrin therapy (vedolizumab) selectively targets gut inflammation with fewer systemic side effects.

13. **Basic response:** CRC is investigated by colonoscopy with biopsy (the gold standard for diagnosis and histological confirmation), CT colonography (for patients unfit for colonoscopy), CEA (a tumour marker used for monitoring recurrence rather than diagnosis), and CT chest, abdomen, and pelvis for TNM staging (to identify local invasion, lymph node involvement, and distant metastases). Management: localised disease is treated by surgical resection (right hemicolectomy for right-sided, left hemicolectomy or anterior resection for left-sided, abdominoperineal resection for low rectal tumours). Adjuvant chemotherapy (FOLFOX: oxaliplatin plus 5-fluorouracil) is

used for stage III disease. Metastatic (stage IV) disease is treated with palliative chemotherapy.

Value-added response: CRC is largely preventable by colonoscopic removal of adenomatous polyps before malignant transformation. All patients above 40 years with unexplained iron deficiency anaemia, rectal bleeding, or change in bowel habit must be referred for urgent colonoscopy to exclude CRC.

14. **Basic response:** Typhoid fever is caused by Salmonella typhi, transmitted via contaminated food and water. Gold standard investigation is blood culture, positive in 80 percent in the first week. Bone marrow culture is the most sensitive (positive even after antibiotics). Widal test has poor specificity in endemic areas because background antibody titres are elevated from prior exposure or vaccination. First-line treatment in Nigeria is IV ceftriaxone (due to widespread resistance to ciprofloxacin, chloramphenicol, ampicillin, and cotrimoxazole); azithromycin is the oral alternative. Prevention: safe water, improved sanitation, and the Vi polysaccharide typhoid vaccine.

Value-added response: Intestinal perforation in week 3 is the most feared complication of typhoid fever and presents as acute abdomen with peritonitis and free gas on erect CXR; it requires urgent laparotomy, bowel repair or resection, and peritoneal lavage with broad-spectrum IV antibiotics covering Gram-negative organisms.

15. **Basic response:** Intestinal amoebiasis: caused by Entamoeba histolytica, presenting as amoebic dysentery (bloody diarrhoea with mucus, tenesmus, and lower abdominal pain) or amoebic liver abscess (right upper quadrant pain, fever, hepatomegaly, anchovy sauce pus on aspiration). Diagnosis by stool microscopy (trophozoites or cysts), stool antigen test, amoebic serology, and ultrasound. Treatment: metronidazole for invasive disease followed by diloxanide furoate to eradicate luminal cysts. Giardiasis: caused by Giardia lamblia, presenting as chronic offensive fatty diarrhoea (steatorrhoea), abdominal bloating, and flatulence without blood. Diagnosis by stool microscopy or stool antigen test. Treatment: metronidazole or tinidazole.

Value-added response: Both amoebiasis and giardiasis are waterborne infections preventable by access to safe drinking water and improved sanitation. In Nigeria, where these conditions are endemic, a high index of suspicion is necessary, and stool examination should always be requested in any patient with diarrhoea lasting more than two weeks.

Answers to Pilot Questions [Series 3]

Part 3.1

1. H. pylori produces urease, generating ammonia that damages the mucus-bicarbonate barrier and causes chronic gastritis, impairing mucosal defence and causing ulceration. NSAIDs inhibit cyclo-oxygenase (COX-1 and COX-2), reducing prostaglandin synthesis, which is required for mucus and bicarbonate secretion and mucosal blood flow.
2. Duodenal ulcer pain is epigastric, characteristically relieved by food and antacids, and worsens 2 to 3 hours after eating (hunger pain). Gastric ulcer pain is also epigastric but is typically worsened by food rather than relieved by it.
3. GORD presents with heartburn (retrosternal burning, worse after meals and on lying down) and acid regurgitation (sour or bitter taste in the mouth). Atypical symptoms include chronic cough, hoarseness, and asthma-like symptoms. The premalignant complication is Barrett's oesophagus (columnar metaplasia of the lower oesophageal mucosa), which carries a risk of progression to oesophageal adenocarcinoma.
4. Haemorrhage is the most common complication, presenting as haematemesis or melaena and managed by endoscopic haemostasis. Perforation presents with sudden severe epigastric pain, peritonitis, and free gas under the diaphragm on an erect chest X-ray and requires emergency surgery. Gastric outlet obstruction from pyloric scarring presents with persistent vomiting of undigested food, weight loss, and a succussion splash.
5. H. pylori is eradicated with triple therapy: a proton pump inhibitor plus clarithromycin plus amoxicillin for 7 to 14 days (or metronidazole in penicillin allergy). GORD is managed with lifestyle modification (weight loss, head-of-bed elevation, avoiding fatty

food, caffeine, and alcohol) and a PPI (omeprazole 20 to 40 mg daily). Laparoscopic Nissen fundoplication is reserved for refractory cases.

Part 3.2

1. Crohn's disease shows transmural inflammation with discontinuous skip lesions, non-caseating granulomas, and cobblestone mucosa. It most commonly affects the terminal ileum and proximal colon. Clinical features include right iliac fossa pain, non-bloody or bloody diarrhoea, weight loss, fever, and perianal disease (fistulae, abscesses, skin tags).
2. UC presents with bloody diarrhoea with mucus, urgency, and tenesmus. Colonoscopy shows continuous mucosal inflammation from the rectum proximally, with granular friable mucosa, loss of haustra, and pseudopolyps in chronic disease. Biopsy shows crypt abscesses and goblet cell depletion.
3. Crohn's can involve any part of the GI tract, causes transmural inflammation, and shows discontinuous skip lesions. UC is confined to the colon and rectum, causes mucosal and submucosal inflammation only, and always shows continuous disease from the rectum proximally.
4. Mild to moderate disease uses aminosalicylates (mesalazine, first-line for UC) and corticosteroids for acute flares. Moderate to severe disease adds azathioprine or 6-mercaptopurine as steroid-sparing agents. Severe or refractory disease uses biological agents including anti-TNF therapy (infliximab, adalimumab) or anti-integrin therapy (vedolizumab).
5. UC can be cured by total colectomy with ileal pouch-anal anastomosis, which removes all diseased colon and restores continence. Crohn's disease cannot be cured by surgery because of the risk of recurrence at the anastomosis; surgery is reserved for complications including stricture, fistula, abscess, or failed medical therapy.

Part 3.3

1. The adenoma-carcinoma sequence describes the progression from normal colonic mucosa to adenomatous polyp to invasive carcinoma through accumulated genetic mutations. Risk factors include a high-fat, low-fibre diet, obesity, physical inactivity, smoking, alcohol, family history of CRC or adenomas, inflammatory bowel disease, and hereditary syndromes including familial adenomatous polyposis and Lynch syndrome.
2. Right-sided CRC (caecal and ascending colon) presents with iron deficiency anaemia from chronic occult bleeding, weight loss, and a palpable right iliac fossa mass. Left-sided CRC (descending and sigmoid colon) presents with a change in bowel habit (alternating constipation and diarrhoea), rectal bleeding, and colicky lower abdominal pain. Obstruction is more common with left-sided tumours due to the narrower lumen.
3. Complications of diverticulitis include pericolic or pelvic abscess (fever, localised peritonism, a palpable mass), perforation (generalised peritonitis with free gas on erect CXR), colovesical fistula (presenting with pneumaturia and faecaluria), and large bowel obstruction. Complicated diverticulitis requires hospital admission with IV antibiotics and surgical intervention.
4. Haemorrhoids are classified as internal (above the dentate line; painless unless prolapsed or thrombosed; graded I to IV by degree of prolapse) or external (below the dentate line; painful, especially when thrombosed). Clinical features include bright red rectal bleeding (fresh blood on toilet paper or dripping into the pan), anal pruritus, and a sensation of incomplete evacuation in prolapsed haemorrhoids.
5. Acute anal fissure is managed conservatively with high-fibre diet, adequate hydration, and topical local anaesthetic cream. Chronic fissure is treated with topical glyceryl trinitrate (0.4 percent) or diltiazem to relax the internal anal sphincter and promote healing. Botulinum toxin injection into the internal anal sphincter is used for fissures resistant to topical therapy. Lateral internal sphincterotomy is the surgical treatment for refractory chronic fissures.

Part 3.4

1. Vibrio cholerae causes profuse watery rice-water stools with life-threatening dehydration (no fever). Shigella causes bloody diarrhoea with mucus, fever, and tenesmus (dysentery). Salmonella (non-typhoidal) causes watery or bloody diarrhoea from undercooked poultry with fever. Campylobacter causes cramping abdominal pain and bloody diarrhoea from undercooked chicken, often with a prodrome of fever.
2. Week 1: stepwise rising fever, headache, malaise, relative bradycardia (Faget's sign), and rose spots on the trunk in approximately 30 percent. Week 2: fever plateaus; abdominal distension, constipation or diarrhoea, splenomegaly, and hepatomegaly. Week 3: complications including intestinal perforation, haemorrhage, encephalopathy, and myocarditis.
3. Blood culture is the gold standard, positive in approximately 80 percent of cases in the first week of illness. The Widal test has poor specificity in endemic areas like Nigeria because baseline antibody titres are elevated from prior subclinical infection or vaccination, leading to many false-positive results that can mislead clinical management.
4. Amoebic dysentery presents with bloody diarrhoea with mucus, tenesmus, and lower abdominal cramping. Amoebic liver abscess presents with right upper quadrant pain, fever, and hepatomegaly; aspiration yields anchovy sauce pus. Both are diagnosed by stool microscopy, stool antigen test, amoebic ELISA serology, and ultrasound for liver abscess. Treatment is metronidazole for invasive disease, followed by diloxanide furoate to eliminate luminal cysts and prevent relapse.
5. Giardiasis presents as chronic offensive fatty diarrhoea (steatorrhoea), abdominal bloating, and flatulence without blood or mucus, distinguishing it from dysentery. Weight loss and malabsorption occur in prolonged infection. Diagnosis is by stool microscopy for cysts or trophozoites, or stool antigen test. Treatment is metronidazole 400 mg three times daily for 5 days or tinidazole 2 g as a single dose.

References and Attributions [Series 3]

Textual References

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- National Institute for Health and Care Excellence. (2019). Colorectal Cancer. NICE Guideline NG151. London: NICE.

Figures, Plates, Tables, and Boxes

- Table 3.1: Comparison of peptic ulcer disease and gastro-oesophageal reflux disease. AI-generated using the prompt: "Create a clean two-column comparison table titled Peptic Ulcer Disease versus GORD with rows for cause, features, investigations, complications, and treatment." Suggested OER search string: "peptic ulcer disease GORD comparison table cause features complications treatment OER."
- Table 3.2: Comparison of Crohn's disease and ulcerative colitis. AI-generated using the prompt: "Create a clear two-column comparison table titled Crohn's Disease versus Ulcerative Colitis with rows for location, pattern, depth of inflammation, pathology, symptoms, extra-intestinal manifestations, and surgical management." Suggested OER search string: "Crohn's disease ulcerative colitis comparison table features pathology management OER."
- Table 3.3: Common colorectal and anorectal conditions. AI-generated using the prompt: "Create a clear two-column table with CRC and diverticular disease in the left column and haemorrhoids and anal fissure in the right column, with rows for cause, features, and

treatment." Suggested OER search string: "colorectal carcinoma diverticular disease haemorrhoids anal fissure table features treatment OER."

- Table 3.4: Common intestinal infections in Nigeria. AI-generated using the prompt: "Create a clear two-column table with bacterial infections (cholera, typhoid, shigella) in the left column and parasitic infections (amoeba, Giardia, helminths) in the right column, with rows for cause, features, diagnosis, and treatment." Suggested OER search string: "intestinal infections Nigeria cholera typhoid amoeba giardia table features treatment OER."

Series 4: Hepatology and Liver Pathologies

- **Part 4.1: Viral Hepatitis**
    - **Sub Part 4.1.1: Hepatitis A and E**
    - **Sub Part 4.1.2: Hepatitis B**
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    - **Sub Part 4.2.1: Pathogenesis and aetiology of cirrhosis**
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- **Part 4.3: Hepatocellular Carcinoma**
    - **Sub Part 4.3.1: Epidemiology and risk factors**
    - **Sub Part 4.3.2: Clinical features and diagnosis**
    - **Sub Part 4.3.3: Management and prevention**
- **Part 4.4: Acute Liver Failure and Cholestatic Liver Disease**
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    - **Sub Part 4.4.3: Primary sclerosing cholangitis**

Introduction

Liver disease is a major cause of morbidity and mortality in Nigeria, driven predominantly by the high prevalence of chronic hepatitis B and C infections, which together affect an estimated 20 million Nigerians. The sequelae of chronic viral hepatitis, including liver cirrhosis, portal

hypertension, and hepatocellular carcinoma, represent the most common liver conditions encountered in Nigerian hospitals. Understanding their pathogenesis, clinical features, and management is therefore a clinical priority.

This Series examines the major liver pathologies systematically. We shall commence with viral hepatitis, covering the clinical features, serology, and management of hepatitis A, B, C, D, and E. Next, we will explore liver cirrhosis, covering its pathogenesis, clinical features, and the major complications including ascites, spontaneous bacterial peritonitis, variceal bleeding, and hepatic encephalopathy. Moving on, we will consider hepatocellular carcinoma, examining its epidemiology, diagnosis, and management in the Nigerian context. Finally, we will address acute liver failure and cholestatic liver diseases, covering primary biliary cholangitis and primary sclerosing cholangitis.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 4.1:** describe the clinical features, serological diagnosis, and management of viral hepatitis A, B, C, D, and E,
- **SLO 4.2:** describe the pathogenesis, clinical features, diagnosis, and complications of liver cirrhosis,
- **SLO 4.3:** describe the epidemiology, clinical features, diagnosis, and management of hepatocellular carcinoma in Nigeria, and
- **SLO 4.4:** describe the clinical features and management of acute liver failure, primary biliary cholangitis, and primary sclerosing cholangitis.

4.1 Viral Hepatitis

4.1.1 Hepatitis A and E

Hepatitis A virus (HAV) is a non-enveloped, single-stranded RNA picornavirus transmitted by the faecal-oral route through contaminated food and water. It causes **self-limiting acute hepatitis** with an incubation period of 2 to 6 weeks, presenting with fever, nausea, vomiting, right upper quadrant pain, jaundice, dark urine, and pale stools. It never causes chronic hepatitis. Diagnosis is by anti-HAV IgM (indicates acute infection). Treatment is supportive. Prevention is by improved sanitation and the inactivated HAV vaccine.

Hepatitis E virus (HEV) is also transmitted by the faecal-oral route (contaminated water) and causes self-limiting acute hepatitis clinically indistinguishable from HAV. HEV is notable for causing **fulminant hepatic failure in pregnant women** (mortality up to 20 to 25 percent in the third trimester), making it particularly important in Nigeria where endemic transmission occurs. Diagnosis is by anti-HEV IgM or HEV RNA by PCR.

HAV is diagnosed by anti-HAV _____ (acute infection marker). HEV is particularly dangerous in _____ women, in whom it can cause fulminant hepatic failure with mortality up to 25 percent.

4.1.2 Hepatitis B

Hepatitis B virus (HBV) is a partially double-stranded DNA hepadnavirus transmitted by blood, sexual contact, and vertical (mother-to-child) transmission. Nigeria is highly endemic with a surface antigen prevalence of approximately 10 to 15 percent. **Hepatitis B surface antigen (HBsAg)** is the marker of current HBV infection. **Anti-HBs antibody** indicates immunity (from vaccination or resolved infection). **HBe antigen (HBeAg)** and **HBV DNA** indicate active viral replication and high infectivity.

Acute HBV: self-limiting in 90 to 95 percent of immunocompetent adults. Chronic HBV (HBsAg positive for more than 6 months): develops in 5 to 10 percent of adults but up to 90 percent of infants infected perinatally. Chronic HBV progresses to cirrhosis in 15 to 25 percent and to hepatocellular carcinoma in 25 percent of cirrhotic patients. Treatment of chronic HBV: tenofovir or entecavir (nucleotide analogues that suppress viral replication; recommended when HBV DNA is above 2000 IU/mL with liver disease). Prevention: hepatitis B vaccination (3-dose schedule: the most important public health intervention in Nigeria).

HBsAg indicates _____ HBV infection. Chronic HBV develops in up to 90 percent of _____ infected perinatally, making birth-dose vaccination critically important.

4.1.3 Hepatitis C and D

Hepatitis C virus (HCV) is a single-stranded RNA flavivirus transmitted primarily by blood (unsafe injections, blood transfusions, intravenous drug use). Unlike HBV, there is no vaccine. Acute HCV is often asymptomatic; **chronic HCV** develops in approximately 80 percent of infected individuals and progresses to cirrhosis in 20 percent over 20 to 30 years. Diagnosis: anti-HCV antibody (screening) followed by HCV RNA PCR (confirms active infection and guides treatment). Treatment: **direct-acting antiviral (DAA) therapy** (sofosbuvir-based regimens: achieve cure in above 95 percent with 8 to 12 weeks of treatment).

Hepatitis D virus (HDV) is an incomplete RNA virus that can only infect individuals who are already infected with HBV (it requires HBsAg to form a complete viral particle). Co-infection (HBV and HDV simultaneously) usually causes self-limiting hepatitis. **Superinfection** (HDV in a patient with existing chronic HBV) causes a severe accelerated progression to cirrhosis. HDV is diagnosed by anti-HDV antibody or HDV RNA. Treatment of HBV-HDV co-infection targets HBV with tenofovir.

Chronic HCV develops in approximately _____ percent of infected individuals. DAA therapy achieves cure in above 95 percent with _____ to 12 weeks of treatment. HDV can only infect individuals with existing HBV infection.

Virus	Transmission and Key Features	Prevention
HAV	Faecal-oral; self-limiting; anti-HAV IgM; no chronicity	Sanitation and inactivated vaccine
HBV	Blood, sexual, vertical; HBsAg marker of infection; chronic in 5–10% of adults and 90% of neonates; treat with tenofovir or entecavir	3-dose vaccine; birth-dose prevents perinatal transmission; most important in Nigeria

Virus	Transmission and Key Features	Prevention
HCV	Blood; chronic in 80%; no vaccine; treat with DAA sofosbuvir-based regimen for 8–12 weeks with >95% cure	No vaccine; blood safety and harm reduction
HDV	Blood; requires HBV co-infection; superinfection accelerates cirrhosis	HBV vaccination prevents HDV
HEV	Faecal-oral; self-limiting; fulminant hepatic failure in pregnant women (up to 25% mortality)	Safe water and sanitation

Table 4.1: Viral hepatitis: transmission, key features, and prevention

Pilot questions 4.1

1. Describe the clinical features and diagnosis of hepatitis A and state why hepatitis E is particularly dangerous in Nigeria. (Linked to SLO 4.1)
2. Explain the serological markers of hepatitis B and state what each indicates. (Linked to SLO 4.1)
3. State the natural history of chronic hepatitis B and describe the treatment. (Linked to SLO 4.1)
4. Describe the natural history of hepatitis C and state the current treatment. (Linked to SLO 4.1)
5. Explain the relationship between HDV and HBV and state the clinical consequence of HDV superinfection. (Linked to SLO 4.1)

4.2 Liver Cirrhosis

4.2.1 Pathogenesis and aetiology of cirrhosis

Liver cirrhosis is the end stage of chronic liver disease, characterised by diffuse hepatic fibrosis with formation of regenerative nodules that distort the normal liver architecture. It results from sustained hepatocyte injury and repair, during which activated **hepatic stellate cells** (Ito cells) are transformed into myofibroblasts that deposit collagen, replacing functional hepatic parenchyma with fibrous scar tissue. The most common causes in Nigeria are **chronic hepatitis B** (the leading cause), **chronic hepatitis C**, and **alcoholic liver disease**. Other causes include NAFLD, autoimmune hepatitis, primary biliary cholangitis, and Wilson's disease.

Cirrhosis is classified as compensated (the liver maintains adequate synthetic function; patients are often asymptomatic or have only mild symptoms) or decompensated (liver synthetic function fails; patients develop the major complications of ascites, variceal bleeding, hepatic encephalopathy, or spontaneous bacterial peritonitis). The transition from compensated to decompensated cirrhosis markedly worsens prognosis: median survival in decompensated cirrhosis is approximately 2 years without liver transplantation.

Liver cirrhosis results from activation of hepatic _____ cells (Ito cells) that deposit collagen. The most common cause of cirrhosis in Nigeria is chronic hepatitis _____.

4.2.2 Clinical features and diagnosis

Compensated cirrhosis may be asymptomatic or present with fatigue, anorexia, and weight loss. Decompensated cirrhosis presents with jaundice, ascites (the most common presenting complication), peripheral oedema, and features of hepatic encephalopathy. Physical examination reveals the stigmata of chronic liver disease: spider naevi (more than 5 suggests cirrhosis), palmar erythema, leuconychia, finger clubbing, gynaecomastia, caput medusae, hepatosplenomegaly (or small shrunken liver in end-stage cirrhosis), and asterixis (liver flap) in hepatic encephalopathy.

Investigations: LFTs (elevated bilirubin; low albumin reflecting impaired synthetic function; elevated ALT and AST in active hepatitis; elevated alkaline phosphatase and GGT). Coagulation

screen (prolonged PT and INR reflecting impaired clotting factor synthesis: the most sensitive marker of hepatic synthetic function). FBC (thrombocytopaenia from portal hypertension and splenic sequestration; anaemia). Abdominal ultrasound (shrunken nodular liver, splenomegaly, ascites, portal vein diameter). The MELD score (Model for End-stage Liver Disease, using bilirubin, INR, and creatinine) quantifies prognosis and prioritises patients for liver transplantation.

The most common presenting complication of decompensated cirrhosis is _____. Prolonged prothrombin time reflects impaired _____ factor synthesis and is the most sensitive marker of hepatic synthetic function.

4.2.3 Complications of cirrhosis

Portal hypertension (elevated portal venous pressure above 12 mmHg) results from increased resistance to portal blood flow through the fibrosed liver, causing: **oesophageal and gastric varices** (which can rupture causing life-threatening haemorrhage, managed by band ligation and terlipressin), **ascites** (managed with salt restriction, spironolactone, and furosemide; spontaneous bacterial peritonitis requires IV cefotaxime), and **hepatic encephalopathy** (impaired consciousness, asterixis, and elevated serum ammonia from impaired hepatic ammonia detoxification; managed with lactulose and rifaximin, which reduce ammonia production).

Hepatorenal syndrome (HRS) is acute renal failure in cirrhotic patients with ascites, caused by splanchnic vasodilatation and severe renal vasoconstriction without intrinsic renal disease. It carries a very high mortality. HRS type 1 (rapidly progressive: creatinine doubles within 2 weeks) is the most severe form; treatment includes albumin infusion, terlipressin, and liver transplantation. Hepatopulmonary syndrome (intrapulmonary vascular dilatation causing hypoxaemia) and portopulmonary hypertension are other serious pulmonary complications.

Portal hypertension causes oesophageal varices, ascites, and hepatic encephalopathy. Hepatic encephalopathy is treated with _____ (reduces ammonia by acidifying the colonic

environment) and rifaximin. Hepatorenal syndrome is acute _____ failure in cirrhotic patients without intrinsic renal disease.

Complication	Features and Management
Portal Hypertension	Portal venous pressure >12 mmHg due to fibrosed liver; leads to varices, ascites, splenomegaly
Oesophageal Varices	Variceal haemorrhage (haematemesis); management: terlipressin to reduce portal pressure, band ligation on OGD within 24h, cefotaxime prophylaxis, TIPSS for refractory bleeding
Ascites	Salt restriction; spironolactone 100–400 mg + furosemide 40–160 mg; large-volume paracentesis with albumin for tense ascites
Spontaneous Bacterial Peritonitis (SBP)	Neutrophils >250 in ascitic fluid; treat with IV cefotaxime; secondary prophylaxis with oral norfloxacin
Hepatic Encephalopathy	Impaired consciousness, asterixis, elevated ammonia; identify and treat precipitant; lactulose and rifaximin
Hepatorenal Syndrome	Acute renal failure without intrinsic renal disease; terlipressin + albumin infusion; liver transplantation is the only curative option

Table 4.2: Complications of liver cirrhosis and their management

Pilot questions 4.2

1. Describe the pathogenesis of liver cirrhosis and state the most common causes in Nigeria. (Linked to SLO 4.2)
2. Describe the stigmata of chronic liver disease found on examination. (Linked to SLO 4.2)

3. Describe the investigations used to assess the severity of liver cirrhosis. (Linked to SLO 4.2)
4. Describe the management of oesophageal variceal haemorrhage. (Linked to SLO 4.2)
5. Describe the clinical features, diagnosis, and management of hepatic encephalopathy. (Linked to SLO 4.2)

4.3 Hepatocellular Carcinoma

4.3.1 Epidemiology and risk factors

Hepatocellular carcinoma (HCC) is the most common primary liver tumour and the fourth most common cause of cancer death globally. Nigeria has one of the highest incidence rates in the world, driven by the dual burden of endemic hepatitis B and C infection and aflatoxin B1 exposure (**aflatoxin B1**, a mycotoxin produced by Aspergillus flavus contaminating improperly stored maize, groundnuts, and cassava, is a potent hepatic carcinogen that synergises with HBV to increase HCC risk by up to 60-fold). Other risk factors include liver cirrhosis from any cause, alcohol use, diabetes mellitus, obesity, and male sex.

HCC almost always develops on a background of liver cirrhosis (in approximately 80 to 90 percent of cases). Cirrhotic patients in Nigeria should undergo 6-monthly surveillance with abdominal ultrasound and serum alpha-fetoprotein (AFP), as early-stage HCC detected during surveillance is potentially curable. An AFP above 400 ng/mL in a cirrhotic patient with a liver lesion is strongly suggestive of HCC.

HCC in Nigeria is driven by chronic HBV infection and exposure to _____ B1 from improperly stored staple foods. Cirrhotic patients should undergo 6-monthly surveillance with abdominal _____ and serum AFP.

4.3.2 Clinical features and diagnosis

Clinical features of HCC include right upper quadrant pain, weight loss, anorexia, fever, a palpable liver mass, and worsening ascites or jaundice in a patient with known cirrhosis. A hepatic bruit may be heard on auscultation. Rapidly deteriorating liver function in a known cirrhotic patient should always raise suspicion for HCC.

Diagnosis in cirrhotic patients follows a non-invasive pathway: a liver lesion above 1 cm on ultrasound surveillance is characterised by **dynamic CT or MRI with contrast** (the hallmark of HCC is **arterial enhancement followed by portal venous washout**: the tumour enhances brightly in the arterial phase then appears hypodense relative to background liver in the portal venous phase). This pattern is diagnostic of HCC without biopsy in lesions above 1 cm in cirrhotic patients. Serum AFP is elevated in approximately 70 percent of HCC cases. Liver biopsy is reserved for atypical lesions.

The radiological hallmark of HCC on dynamic CT or MRI is arterial _____ followed by portal venous _____. AFP above 400 ng/mL in a cirrhotic patient with a liver lesion is strongly suggestive of HCC.

4.3.3 Management and prevention

Management of HCC is guided by the Barcelona Clinic Liver Cancer (BCLC) staging system, which integrates tumour stage, liver function (Child-Pugh class), and performance status. Very early and early-stage HCC (single tumour below 2 to 3 cm; preserved liver function): surgical resection or radiofrequency ablation (potentially curative). Intermediate-stage HCC (multinodular, preserved liver function): transarterial chemoembolisation (TACE). Advanced-stage HCC (vascular invasion or extrahepatic spread): sorafenib (a multikinase inhibitor; first-line systemic therapy). Terminal-stage: palliative care. Liver transplantation is curative for patients meeting Milan criteria (single tumour below 5 cm or up to 3 tumours below 3 cm, no vascular invasion).

Prevention: hepatitis B vaccination (the most effective HCC prevention strategy in Nigeria, as it prevents the primary risk factor), HBV treatment with tenofovir or entecavir (reduces HCC risk in chronic HBV), reduction of aflatoxin exposure through improved grain storage, drying, and processing, and surveillance of cirrhotic patients.

BCLC early-stage HCC is treated by surgical _____ or radiofrequency ablation. The most effective HCC prevention strategy in Nigeria is hepatitis B _____.

Diagnosis	BCLC Staging and Management
Risk Factors	HBV (most important in Nigeria), aflatoxin B1, HCV, cirrhosis (any cause), alcohol, diabetes, obesity
Surveillance	6-monthly abdominal ultrasound + serum AFP in all cirrhotic patients
Clinical Features	Right upper quadrant pain, weight loss, liver mass, worsening ascites/jaundice in cirrhotic, hepatic bruit
Diagnosis	Dynamic CT/MRI with contrast: arterial enhancement then portal venous washout diagnostic for lesions >1 cm (no biopsy needed); AFP >400 ng/mL in cirrhotic supports diagnosis
Very Early / Early Stage	Single tumour <2–3 cm, preserved liver function → surgical resection or radiofrequency ablation (potentially curative)
Intermediate Stage	Multinodular, preserved liver function → TACE (transarterial chemoembolization)
Advanced Stage	Vascular invasion or extrahepatic spread → sorafenib systemic therapy
Terminal Stage	Best supportive care
Liver Transplantation	Milan criteria: single tumour <5 cm or up to 3 tumours <3 cm, no vascular invasion → curative option

Table 4.3: HCC diagnosis and BCLC staging with management

Pilot questions 4.3

1. Describe the epidemiology and risk factors for HCC in Nigeria. (Linked to SLO 4.3)
2. Describe the clinical features of HCC in a cirrhotic patient. (Linked to SLO 4.3)

3. Describe the radiological diagnosis of HCC and state the hallmark imaging finding. (Linked to SLO 4.3)
4. Describe the BCLC staging system and the treatment for each stage. (Linked to SLO 4.3)
5. Describe the prevention strategies for HCC in Nigeria. (Linked to SLO 4.3)

4.4 Acute Liver Failure and Cholestatic Liver Disease

4.4.1 Acute liver failure

Acute liver failure (ALF) is defined as severe acute hepatocellular dysfunction causing coagulopathy (INR above 1.5) and encephalopathy in a patient without pre-existing liver disease within 26 weeks of the onset of illness. Causes in Nigeria include **viral hepatitis** (hepatitis B, hepatitis E in pregnancy, hepatitis A in older patients), **drug-induced liver injury** (paracetamol overdose in high-income countries; traditional herbal medicines and antituberculous drugs in Nigeria), and **Wilson's disease** (in young patients presenting with acute hepatitis and haemolysis).

Clinical features: rapidly progressive jaundice, coagulopathy (spontaneous bleeding, bruising), hepatic encephalopathy (confusion, asterixis, seizures, coma), hypoglycaemia (from impaired gluconeogenesis), and multi-organ failure. Management is in an intensive care unit: correct hypoglycaemia (IV dextrose), treat encephalopathy (lactulose, avoid sedatives), manage coagulopathy (vitamin K; FFP only for active bleeding, not prophylactically), and N-acetylcysteine (IV: for all ALF regardless of cause, as it improves transplant-free survival). Liver transplantation is the definitive treatment for patients who fail to recover.

Acute liver failure is defined as coagulopathy (INR above 1.5) and _____ in a patient without pre-existing liver disease within 26 weeks of onset. In Nigeria, the most important herbal medicine and _____ drug-induced causes must always be considered.

4.4.2 Primary biliary cholangitis

Primary biliary cholangitis (PBC) is an autoimmune chronic cholestatic liver disease characterised by progressive destruction of the intrahepatic bile ducts, predominantly affecting middle-aged women (female to male ratio 9:1). The hallmark serological marker is **anti-mitochondrial antibody (AMA)** (positive in above 95 percent of cases; highly specific). Clinical features include progressive pruritus (often preceding jaundice by years), fatigue, jaundice with pale stools and dark urine, xanthomata and xanthelasma (from hypercholesterolaemia due to cholestasis), and osteoporosis from fat-soluble vitamin D malabsorption.

Liver function tests show a cholestatic pattern (markedly elevated alkaline phosphatase and GGT, with mildly elevated transaminases). Elevated serum IgM is characteristic. Liver biopsy shows non-suppurative destructive cholangitis (florid duct lesion). Treatment: ursodeoxycholic acid (UDCA: slows progression and improves liver function tests; the first-line treatment). Obeticholic acid is added in patients with inadequate response to UDCA. End-stage PBC requires liver transplantation.

PBC is characterised by a positive _____ antibody (AMA) in above 95 percent of cases. First-line treatment for PBC is _____ acid (UDCA), which slows disease progression.

4.4.3 Primary sclerosing cholangitis

Primary sclerosing cholangitis (PSC) is a chronic cholestatic liver disease characterised by progressive inflammation and fibrosis of both **intra- and extrahepatic bile ducts**, causing multifocal biliary strictures and dilatations producing a **beaded appearance on cholangiography** (MRCP: the investigation of choice). PSC predominantly affects young men (male to female ratio 2:1) and is **strongly associated with inflammatory bowel disease**, particularly ulcerative colitis (present in 70 to 80 percent of PSC patients). The risk of cholangiocarcinoma is markedly elevated.

Clinical features: pruritus, progressive jaundice, right upper quadrant pain, and recurrent cholangitis (fever, rigors, and jaundice from bile duct infection above a stricture). LFTs show a cholestatic pattern. There is no medical treatment proven to alter the natural history of PSC; ursodeoxycholic acid is no longer recommended. Endoscopic dilatation of dominant strictures

relieves symptoms. Liver transplantation is the only curative option. Annual surveillance for cholangiocarcinoma (CA19-9 and MRCP) and colorectal carcinoma (annual colonoscopy) is required.

PSC produces a _____ appearance of the bile ducts on MRCP from multifocal strictures and dilatations. PSC is associated with _____ bowel disease, particularly ulcerative colitis, in 70 to 80 percent of cases.

Primary Biliary Cholangitis (PBC)	Primary Sclerosing Cholangitis (PSC)
Predominant sex: Middle-aged women (female:male 9:1)	**Predominant sex**: Young men (male:female 2:1)
Pathology: Autoimmune destruction of intrahepatic bile ducts	**Pathology**: Inflammatory fibrosis of intra- and extrahepatic bile ducts → multifocal strictures & dilatations
Serological marker: Anti-mitochondrial antibody (AMA) positive in >95%; elevated serum IgM	**Association**: Inflammatory bowel disease (IBD) in 70–80%, especially ulcerative colitis
Clinical features: Pruritus (often precedes jaundice), fatigue, xanthomata, osteoporosis	**Clinical features**: Pruritus, progressive jaundice, recurrent cholangitis
Investigation: Cholestatic LFTs, AMA, liver biopsy (non-suppurative destructive cholangitis)	**Investigation**: MRCP (beaded bile ducts), cholestatic LFTs, CA19-9 for cholangiocarcinoma surveillance
Treatment: UDCA first-line; obeticholic acid if inadequate response; liver transplantation for end-stage	**Treatment**: No effective medical therapy; endoscopic stricture dilatation; liver transplantation

Primary Biliary Cholangitis (PBC)	Primary Sclerosing Cholangitis (PSC)
—	**Complications**: Cholangiocarcinoma; colorectal carcinoma in IBD

Table 4.4: Comparison of primary biliary cholangitis and primary sclerosing cholangitis

Pilot questions 4.4

1. Define acute liver failure and state the three most common causes in Nigeria. (Linked to SLO 4.4)
2. Describe the management of acute liver failure in the intensive care unit. (Linked to SLO 4.4)
3. Describe the clinical features and diagnosis of primary biliary cholangitis. (Linked to SLO 4.4)
4. Describe the clinical features, investigation, and complications of primary sclerosing cholangitis. (Linked to SLO 4.4)
5. Distinguish primary biliary cholangitis from primary sclerosing cholangitis in terms of sex predilection, serological markers, and associations. (Linked to SLO 4.4)

Series Summary

This Series examined the major liver pathologies relevant to Nigeria. We began with viral hepatitis: HAV and HEV (faecal-oral, self-limiting, HEV causes fulminant hepatic failure in pregnancy), HBV (highly endemic in Nigeria; HBsAg is the marker of infection; chronic in 90 percent of perinatally infected infants; treat with tenofovir or entecavir; 3-dose vaccine is the most important prevention), HCV (chronic in 80 percent; no vaccine; treat with DAA sofosbuvir-based regimens achieving above 95 percent cure), and HDV (requires HBV co-infection; superinfection accelerates cirrhosis). Next, we explored liver cirrhosis, covering the pathogenesis (hepatic stellate cell activation and collagen deposition from HBV as the leading cause in Nigeria), stigmata of chronic liver disease, complications (portal hypertension causing varices managed with band ligation and terlipressin; ascites managed with spironolactone; hepatic encephalopathy treated with lactulose; hepatorenal syndrome), and the MELD score. Moving on, we examined HCC (driven by HBV and aflatoxin B1 in Nigeria; arterial enhancement and portal venous washout on dynamic CT is diagnostic; BCLC staging guides management from resection for early stage to sorafenib for advanced stage; HBV vaccination is the most important prevention). Finally, we addressed acute liver failure (INR above 1.5 and encephalopathy; N-acetylcysteine for all causes), PBC (AMA positive; middle-aged women; UDCA first-line), and PSC (beaded bile ducts on MRCP; young men; associated with IBD; risk of cholangiocarcinoma).

By completing this Series, you should now be able to describe the serological diagnosis and management of viral hepatitis, outline the complications of liver cirrhosis and their management, describe the diagnosis and management of HCC in Nigeria, and describe the clinical features of acute liver failure, PBC, and PSC. These abilities align with SLOs 4.1 to 4.4 and equip you to manage liver diseases in the Nigerian clinical setting.

Glossary of Terms

- **Aflatoxin B1:** a mycotoxin produced by Aspergillus flavus contaminating improperly stored staple foods; a potent hepatic carcinogen that synergises with HBV to increase HCC risk by up to 60-fold.

- **Alpha-fetoprotein (AFP):** a serum tumour marker elevated in approximately 70 percent of HCC cases; used in combination with abdominal ultrasound for 6-monthly HCC surveillance in cirrhotic patients.
- **Anti-mitochondrial antibody (AMA):** a highly specific serological marker for primary biliary cholangitis, positive in above 95 percent of cases; targets the E2 subunit of pyruvate dehydrogenase complex.
- **Barcelona Clinic Liver Cancer (BCLC) staging:** the most widely used staging system for HCC, integrating tumour burden, liver function (Child-Pugh class), and performance status to guide treatment decisions.
- **Hepatic encephalopathy:** a neuropsychiatric complication of liver failure or portosystemic shunting, caused by impaired hepatic ammonia detoxification; manifests as confusion, asterixis, and coma; treated with lactulose and rifaximin.
- **Hepatorenal syndrome (HRS):** acute renal failure in cirrhotic patients with ascites, caused by splanchnic vasodilatation and renal vasoconstriction without intrinsic renal disease; treated with terlipressin and albumin infusion.
- **MELD score:** Model for End-stage Liver Disease; calculated from serum bilirubin, INR, and creatinine; quantifies 90-day mortality risk in cirrhosis and is used to prioritise patients for liver transplantation.
- **N-acetylcysteine:** an antidote for paracetamol overdose that replenishes glutathione and reduces hepatocyte damage; also improves transplant-free survival in all causes of acute liver failure when given intravenously.
- **Primary biliary cholangitis (PBC):** an autoimmune chronic cholestatic liver disease causing progressive destruction of intrahepatic bile ducts, predominantly in middle-aged women; characterised by positive AMA and elevated serum IgM; treated with UDCA.
- **Primary sclerosing cholangitis (PSC):** a chronic cholestatic liver disease causing progressive inflammatory fibrosis of intra- and extrahepatic bile ducts; associated with IBD in 70 to 80 percent; produces a beaded appearance on MRCP; carries a high risk of cholangiocarcinoma.

- **Sorafenib:** a multikinase inhibitor used as first-line systemic therapy for advanced-stage HCC with vascular invasion or extrahepatic spread.
- **Terlipressin:** a vasopressin analogue that constricts splanchnic blood vessels, reducing portal venous pressure; used in the management of acute variceal haemorrhage and hepatorenal syndrome.

Concept Check Questions [Series 4]

Objective questions

1. _____ surface antigen (HBsAg) is the serological marker of current hepatitis B infection and indicates the patient is potentially infectious. (Linked to SLO 4.1)
2. Liver cirrhosis results from activated hepatic _____ cells (Ito cells) that deposit collagen, replacing functional liver parenchyma with fibrous scar tissue. (Linked to SLO 4.2)
3. The radiological hallmark of HCC on dynamic CT is arterial _____ followed by portal venous washout. (Linked to SLO 4.3)
4. Acute liver failure is defined as coagulopathy (INR above 1.5) and hepatic _____ without pre-existing liver disease within 26 weeks. (Linked to SLO 4.4)
5. Primary biliary cholangitis is characterised by a positive anti-_____ antibody (AMA) in above 95 percent of cases and is treated with ursodeoxycholic acid. (Linked to SLO 4.4)

Short-answer questions

6. Explain the serological markers of hepatitis B and state what HBsAg, anti-HBs, and HBeAg each indicate. (Linked to SLO 4.1)
7. Describe the stigmata of chronic liver disease found on physical examination. (Linked to SLO 4.2)
8. Describe the epidemiology and risk factors for HCC in Nigeria. (Linked to SLO 4.3)
9. Describe the clinical features of hepatic encephalopathy and state its management. (Linked to SLO 4.2)
10. Distinguish primary biliary cholangitis from primary sclerosing cholangitis in terms of sex predilection, serological markers, and associations. (Linked to SLO 4.4)

Long-answer questions

11. Describe the natural history, serological diagnosis, and management of chronic hepatitis B, including prevention. (Linked to SLO 4.1)
12. Describe the complications of liver cirrhosis and their management. (Linked to SLO 4.2)
13. Describe the diagnosis, staging, and management of hepatocellular carcinoma in Nigeria. (Linked to SLO 4.3)
14. Describe the clinical features, diagnosis, and management of acute liver failure. (Linked to SLO 4.4)
15. Compare primary biliary cholangitis and primary sclerosing cholangitis in terms of pathology, clinical features, investigation, and treatment. (Linked to SLO 4.4)

Answers to Concept Check Questions [Series 4]

Objective questions

1. Hepatitis B.
2. Stellate.
3. Enhancement.
4. Encephalopathy.
5. Mitochondrial.

Short-answer questions

6. HBsAg (hepatitis B surface antigen) indicates current HBV infection and potential infectivity to others. Anti-HBs antibody indicates immunity, either from vaccination or from resolved natural infection. HBeAg (hepatitis B e antigen) indicates active viral

replication and high infectivity, and its presence suggests the patient requires antiviral treatment.

7. Spider naevi (more than 5 suggests cirrhosis), palmar erythema, leuconychia (white nails from hypoalbuminaemia), finger clubbing, gynaecomastia, loss of body hair, caput medusae (dilated periumbilical veins from portal hypertension), hepatosplenomegaly or a small shrunken liver in end-stage cirrhosis, and asterixis (liver flap) in hepatic encephalopathy.

8. HCC has one of the highest incidence rates in the world in Nigeria, driven by the dual burden of endemic chronic hepatitis B (the most important risk factor) and aflatoxin B1 exposure from improperly stored maize, groundnuts, and cassava. Other risk factors include chronic hepatitis C, liver cirrhosis from any cause, alcohol use, diabetes mellitus, and obesity.

9. Hepatic encephalopathy presents as a spectrum from mild cognitive impairment and personality change through confusion and asterixis (liver flap on outstretched hands) to coma. The precipitating cause must be identified and treated (GI bleeding, infection, constipation, electrolyte disturbance, sedative drugs). Lactulose (which acidifies the colonic environment, trapping ammonia as ammonium and reducing its absorption) and rifaximin (a poorly absorbed antibiotic that reduces ammonia-producing colonic bacteria) are the treatments.

10. PBC predominantly affects middle-aged women (female to male ratio 9:1) and is characterised by a positive anti-mitochondrial antibody (AMA) in above 95 percent of cases. PSC predominantly affects young men (male to female ratio 2:1), has no specific autoantibody, and is strongly associated with inflammatory bowel disease (particularly ulcerative colitis) in 70 to 80 percent of cases.

Long-answer questions

11. **Basic response:** HBV is transmitted by blood, sexual contact, and vertical (mother-to-child) transmission. Acute HBV resolves in 90 to 95 percent of immunocompetent adults but becomes chronic (HBsAg positive for more than 6 months) in 5 to 10 percent of

adults and up to 90 percent of perinatally infected infants. Chronic HBV progresses to cirrhosis in 15 to 25 percent and carries a risk of HCC. Serology: HBsAg (current infection), HBeAg and HBV DNA (active replication and high infectivity), anti-HBs (immunity). Treatment: tenofovir or entecavir (nucleotide analogues that suppress viral replication, indicated when HBV DNA is above 2000 IU/mL with liver disease; do not cure but prevent progression). Prevention: 3-dose hepatitis B vaccine, with a birth dose within 24 hours to prevent perinatal transmission.

Value-added response: In Nigeria, universal infant vaccination with a birth dose of hepatitis B vaccine is the most important public health intervention for HCC prevention, as the majority of chronic HBV in adults is acquired perinatally. Vaccination of healthcare workers is also essential given occupational exposure risk.

12. **Basic response:** Portal hypertension (portal venous pressure above 12 mmHg) is the primary driver of most complications. Oesophageal variceal haemorrhage: haematemesis and melaena; managed with terlipressin (to reduce portal pressure), IV cefotaxime (SBP prophylaxis), and OGD with band ligation within 24 hours. Ascites: managed with salt restriction, spironolactone (100 to 400 mg), and furosemide (40 to 160 mg); large-volume paracentesis with albumin for tense ascites. Spontaneous bacterial peritonitis (SBP): neutrophils above 250 per microlitre in ascitic fluid; IV cefotaxime; secondary prophylaxis with oral norfloxacin. Hepatic encephalopathy: identify and treat precipitants; lactulose and rifaximin. Hepatorenal syndrome: albumin infusion and terlipressin; liver transplantation.

Value-added response: Early referral for liver transplantation assessment is critical in decompensated cirrhosis, as it is the only definitive treatment. The MELD score (bilirubin, INR, creatinine) quantifies 90-day mortality and prioritises patients on the transplant waiting list.

13. **Basic response:** HCC is almost always diagnosed on a background of liver cirrhosis. Surveillance: 6-monthly abdominal ultrasound and serum AFP in all cirrhotic patients. Diagnosis: a liver lesion above 1 cm on ultrasound is characterised by dynamic CT or MRI with contrast; the hallmark of HCC is arterial enhancement followed by portal

venous washout, which is diagnostic without biopsy. AFP above 400 ng/mL in a cirrhotic patient with a liver lesion is strongly supportive. BCLC staging guides treatment: very early or early stage (single tumour below 2 to 3 cm, preserved liver function): surgical resection or radiofrequency ablation (potentially curative); intermediate stage (multinodular, preserved function): TACE; advanced stage (vascular invasion or extrahepatic spread): sorafenib; terminal stage: palliative care. Milan criteria (single tumour below 5 cm or up to 3 tumours below 3 cm, no vascular invasion): liver transplantation.

Value-added response: In Nigeria, the majority of HCC is diagnosed at an advanced stage because of limited ultrasound surveillance infrastructure. Strengthening surveillance programmes in cirrhotic patients and reducing aflatoxin exposure through food safety interventions are the most important strategies for improving HCC outcomes.

14. **Basic response:** ALF is defined as severe coagulopathy (INR above 1.5) and hepatic encephalopathy in a patient without pre-existing liver disease within 26 weeks of onset. Causes in Nigeria include viral hepatitis (HBV, HEV in pregnancy), drug-induced liver injury from traditional herbal medicines and antituberculous drugs, and Wilson's disease in young patients. Management in ICU: correct hypoglycaemia with IV dextrose; treat encephalopathy with lactulose (avoid sedatives); manage coagulopathy with vitamin K (FFP only for active bleeding); N-acetylcysteine IV for all causes (improves transplant-free survival); manage cerebral oedema (head elevation, mannitol for raised intracranial pressure); treat renal failure and infection. Liver transplantation is the definitive treatment for patients who fail to recover.

Value-added response: N-acetylcysteine improves transplant-free survival in all causes of ALF, not only paracetamol overdose, and should be given intravenously as early as possible in all ALF patients. In Nigeria, traditional herbal medicine-induced ALF is particularly important to recognise, as patients may not volunteer this history; all ALF patients should be specifically asked about use of traditional herbal remedies.

15. **Basic response:** PBC: autoimmune destruction of intrahepatic bile ducts predominantly in middle-aged women (female to male ratio 9:1); AMA positive in above 95 percent (highly specific); elevated serum IgM; cholestatic LFTs (elevated alkaline phosphatase and GGT); clinical features of pruritus preceding jaundice, xanthomata, osteoporosis; liver biopsy shows non-suppurative destructive cholangitis; treatment is UDCA (first-line), obeticholic acid for non-responders, liver transplantation for end-stage. PSC: progressive inflammatory fibrosis of intra- and extrahepatic bile ducts predominantly in young men (male to female ratio 2:1); strongly associated with IBD (ulcerative colitis in 70 to 80 percent); no specific autoantibody; cholestatic LFTs; MRCP shows beaded appearance (multifocal strictures and dilatations); complications include cholangiocarcinoma (requires annual CA19-9 and MRCP surveillance) and colorectal carcinoma (annual colonoscopy); no effective medical therapy; endoscopic dilatation for dominant strictures; liver transplantation.

Value-added response: Both PBC and PSC progress to cirrhosis and require liver transplantation in end-stage disease, but PSC has an additional risk of cholangiocarcinoma that makes surveillance essential and makes any dominant stricture highly suspicious for malignancy, warranting brush cytology or biopsy during ERCP.

Answers to Pilot Questions [Series 4]

Part 4.1

1. HAV is transmitted by the faecal-oral route and causes self-limiting acute hepatitis with fever, nausea, right upper quadrant pain, jaundice, dark urine, and pale stools. It is diagnosed by anti-HAV IgM. HEV is also faecal-oral and causes a clinically similar illness but is particularly dangerous in Nigeria because it causes fulminant hepatic failure in pregnant women, with mortality up to 20 to 25 percent in the third trimester.
2. HBsAg (hepatitis B surface antigen) indicates current HBV infection. Anti-HBs antibody indicates immunity from vaccination or resolved infection. HBeAg and elevated HBV

DNA indicate active viral replication and high infectivity. Anti-HBc IgM indicates acute HBV infection.

3. Acute HBV resolves in 90 to 95 percent of immunocompetent adults but becomes chronic in 5 to 10 percent of adults and up to 90 percent of perinatally infected infants. Chronic HBV progresses to cirrhosis in 15 to 25 percent and HCC in 25 percent of cirrhotic patients. Treatment is tenofovir or entecavir when HBV DNA is above 2000 IU/mL with evidence of liver disease.
4. Acute HCV is often asymptomatic; chronic HCV develops in approximately 80 percent and progresses to cirrhosis in 20 percent over 20 to 30 years. Treatment is direct-acting antiviral (DAA) therapy with sofosbuvir-based regimens (8 to 12 weeks), which achieves cure in above 95 percent of patients with no significant side effects.
5. HDV is an incomplete RNA virus that requires HBsAg to form a complete viral particle, so it can only infect individuals with existing HBV infection. Superinfection (HDV in a patient with chronic HBV) causes severe accelerated progression to cirrhosis and has a worse prognosis than HBV alone. Prevention of HDV is achieved by hepatitis B vaccination.

Part 4.2

1. Cirrhosis results from sustained hepatocyte injury and repair, during which activated hepatic stellate cells (Ito cells) are transformed into myofibroblasts that deposit collagen, replacing functional parenchyma with fibrous scar tissue and regenerative nodules. The most common causes in Nigeria are chronic hepatitis B (the leading cause), chronic hepatitis C, and alcoholic liver disease.
2. Spider naevi (more than 5 on the upper body), palmar erythema, leuconychia, finger clubbing, gynaecomastia, loss of body hair, caput medusae (dilated periumbilical veins from portal hypertension), hepatosplenomegaly or a small shrunken liver in end-stage cirrhosis, and asterixis (bilateral coarse tremor of outstretched hands: the liver flap) in hepatic encephalopathy.

3. LFTs (elevated bilirubin, low albumin reflecting impaired synthesis, elevated transaminases in active hepatitis, elevated alkaline phosphatase and GGT). Coagulation screen (prolonged PT and INR: the most sensitive marker of synthetic function). FBC (thrombocytopaenia from splenic sequestration; anaemia). Abdominal ultrasound (shrunken nodular liver, splenomegaly, ascites, portal vein diameter). MELD score (bilirubin, INR, creatinine: quantifies 90-day mortality and guides liver transplantation listing).
4. Immediate management: terlipressin IV to reduce portal pressure; IV cefotaxime (broad-spectrum antibiotic for SBP prophylaxis; reduces rebleeding and mortality); blood transfusion to maintain haemoglobin above 70 to 80 g/L. Endoscopy: OGD within 24 hours for band ligation of varices (the most effective endoscopic therapy). Patients who rebleed after endoscopic therapy should be considered for TIPSS (transjugular intrahepatic portosystemic shunt, which decompresses portal pressure).
5. Hepatic encephalopathy presents as confusion, drowsiness, personality change, and asterixis (flapping tremor); coma occurs in severe cases. Precipitants include GI bleeding, infection, constipation, electrolyte disturbance (hypokalaemia, hyponatraemia), dehydration, and sedative drugs. Management: identify and treat the precipitant; lactulose (reduces ammonia absorption); rifaximin (reduces ammonia-producing bacteria); avoid sedatives and opiates.

Part 4.3

1. Nigeria has one of the highest HCC incidence rates globally, driven by the high prevalence of chronic HBV infection (the most important risk factor) and aflatoxin B1 exposure from improperly stored maize, groundnuts, and cassava. These two factors synergise to increase HCC risk by up to 60-fold. Other risk factors include chronic HCV, cirrhosis from any cause, alcoholic liver disease, diabetes mellitus, obesity, and male sex.
2. Right upper quadrant pain or discomfort, weight loss, anorexia, fever, a palpable liver mass or worsening hepatomegaly, and progressive jaundice or worsening ascites in a

known cirrhotic patient. A hepatic bruit may be audible on auscultation. Any rapid deterioration in a cirrhotic patient should raise suspicion for HCC development.

3. A liver lesion above 1 cm detected on surveillance ultrasound is characterised by dynamic CT or MRI with contrast. The hallmark of HCC is arterial phase enhancement (the tumour enhances brightly because it is supplied by new hepatic arterial vessels) followed by portal venous phase washout (the tumour appears hypodense relative to the surrounding liver). This pattern is diagnostic of HCC without biopsy in cirrhotic patients. AFP above 400 ng/mL in a cirrhotic patient with a liver lesion provides additional support.

4. Very early or early stage (single tumour below 2 to 3 cm, preserved liver function, performance status 0): surgical resection (preferred if adequate liver reserve) or radiofrequency ablation (for tumours below 3 cm). Intermediate stage (multinodular, preserved liver function, performance status 0 to 2): TACE (transarterial chemoembolisation). Advanced stage (vascular invasion or extrahepatic spread, performance status 0 to 2): sorafenib (first-line systemic therapy). Terminal stage (severe liver dysfunction or performance status 3 to 4): best supportive care. Liver transplantation: patients meeting Milan criteria have excellent 5-year survival above 70 percent.

5. Hepatitis B vaccination (3-dose schedule with a birth dose within 24 hours: prevents perinatal HBV transmission and is the single most effective HCC prevention strategy in Nigeria). Treatment of chronic HBV with tenofovir or entecavir (reduces HCC risk). Reduction of aflatoxin B1 exposure through improved grain storage, drying, and processing, and diversification away from susceptible staple foods. 6-monthly HCC surveillance (ultrasound and AFP) in all cirrhotic patients.

Part 4.4

1. ALF is defined as coagulopathy (INR above 1.5) and hepatic encephalopathy in a patient without pre-existing liver disease within 26 weeks of illness onset. The three most

common causes in Nigeria are viral hepatitis (HBV and HEV in pregnancy), traditional herbal medicine-induced liver injury (from a wide range of hepatotoxic botanical preparations), and antituberculous drug-induced liver injury (isoniazid, rifampicin, and pyrazinamide are all hepatotoxic).

2. ICU management: correct hypoglycaemia with IV 10 to 20 percent dextrose (frequent monitoring as gluconeogenesis is severely impaired); treat encephalopathy with lactulose and avoid all sedatives and opiates; manage coagulopathy with vitamin K (FFP only for active bleeding or before invasive procedures, not prophylactically as INR is used to monitor progress); N-acetylcysteine IV for all causes (improves transplant-free survival); manage cerebral oedema with head-of-bed elevation at 30 degrees and IV mannitol for raised intracranial pressure; treat sepsis aggressively; monitor and support renal function. Refer early for liver transplantation assessment.

3. PBC predominantly affects middle-aged women and presents with pruritus (often years before jaundice), fatigue, progressive jaundice with pale stools and dark urine, xanthomata and xanthelasma from hypercholesterolaemia, and osteoporosis from fat-soluble vitamin D malabsorption. The hallmark serological marker is a positive anti-mitochondrial antibody (AMA) in above 95 percent of cases; elevated serum IgM is also characteristic. LFTs show a cholestatic pattern. Liver biopsy shows non-suppurative destructive cholangitis.

4. PSC presents with progressive pruritus, jaundice, right upper quadrant pain, and episodes of recurrent bacterial cholangitis (fever, rigors, and jaundice from infection above a biliary stricture). Investigation: MRCP (investigation of choice) shows a beaded appearance of intra- and extrahepatic bile ducts from multifocal strictures and dilatations; cholestatic LFTs; CA19-9 for cholangiocarcinoma surveillance. Complications include cholangiocarcinoma (markedly elevated risk; annual CA19-9 and MRCP surveillance required) and colorectal carcinoma (annual colonoscopy given IBD association).

5. PBC predominantly affects middle-aged women (female to male ratio 9:1) and is characterised by a positive AMA in above 95 percent and elevated serum IgM; it has no specific IBD association. PSC predominantly affects young men (male to female ratio 2:1), has no specific autoantibody (pANCA is weakly positive in some), and is strongly

associated with inflammatory bowel disease (ulcerative colitis in 70 to 80 percent). Both cause a cholestatic pattern on LFTs and can progress to cirrhosis.

References and Attributions [Series 4]

Textual References

- World Health Organization. (2017). Global Hepatitis Report 2017. Geneva: WHO.
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- European Association for the Study of the Liver. (2018). EASL Clinical Practice Guidelines: Management of Hepatocellular Carcinoma. Journal of Hepatology, 69(1), 182 to 236.
- Kasper, D. L., Fauci, A. S., Hauser, S. L., Longo, D. L., Jameson, J. L. and Loscalzo, J. (2018). Harrison's Principles of Internal Medicine (20th ed.). New York: McGraw-Hill.

Figures, Plates, Tables, and Boxes

- Table 4.1: Viral hepatitis: transmission, key features, and prevention. AI-generated using the prompt: "Create a clean three-column table titled Viral Hepatitis Key Features with columns for Virus, Transmission and Key Features, and Prevention. Five rows: HAV, HBV, HCV, HDV, HEV." Suggested OER search string: "viral hepatitis comparison table HAV HBV HCV HDV HEV features prevention OER."
- Table 4.2: Complications of liver cirrhosis and their management. AI-generated using the prompt: "Create a clear two-column table titled Complications of Liver Cirrhosis with a list of complications in the left column and features and management in the right column." Suggested OER search string: "liver cirrhosis complications portal hypertension ascites hepatic encephalopathy management table OER."
- Table 4.3: HCC diagnosis and BCLC staging with management. AI-generated using the prompt: "Create a clear two-column table: left column covering HCC risk factors, surveillance, clinical features, and diagnosis; right column covering BCLC staging and

corresponding management." Suggested OER search string: "hepatocellular carcinoma HCC BCLC staging management diagnosis table OER."

- Table 4.4: Comparison of primary biliary cholangitis and primary sclerosing cholangitis. AI-generated using the prompt: "Create a clear two-column comparison table titled Primary Biliary Cholangitis versus Primary Sclerosing Cholangitis with rows for predominant sex, pathology, serological markers, clinical features, investigation, complications, and treatment." Suggested OER search string: "primary biliary cholangitis primary sclerosing cholangitis comparison table features investigation treatment OER."

Series 5: Complications and Preventive Strategies

- **Part 5.1: Gastrointestinal Emergencies**
    - Sub Part 5.1.1: Acute abdomen and surgical emergencies
    - Sub Part 5.1.2: Acute pancreatitis
    - Sub Part 5.1.3: Management of gastrointestinal emergencies
- **Part 5.2: Complications of GI and Liver Disease**
    - Sub Part 5.2.1: Complications of peptic ulcer disease and IBD
    - Sub Part 5.2.2: Complications of liver disease
    - Sub Part 5.2.3: GI malignancy complications
- **Part 5.3: Cancer Screening and Prevention**
    - Sub Part 5.3.1: Colorectal cancer screening
    - Sub Part 5.3.2: HCC surveillance and prevention
    - Sub Part 5.3.3: Prevention of GI infections
- **Part 5.4: Patient Education and Health Promotion**
    - Sub Part 5.4.1: Dietary modification and lifestyle
    - Sub Part 5.4.2: Vaccination and chemoprophylaxis
    - Sub Part 5.4.3: Health promotion in the Nigerian context

Introduction

The final Series of this course brings together the major complications of gastrointestinal and liver disease and the evidence-based strategies for their prevention. Recognising a gastrointestinal emergency, managing the complications of peptic ulcer disease, cirrhosis, and malignancy, and understanding when and how to screen for colorectal cancer and hepatocellular carcinoma are skills that directly save lives in clinical practice. In Nigeria, the burden of preventable GI disease remains high, making health promotion and vaccination strategies particularly important.

We shall commence this Series by examining the major gastrointestinal emergencies, including the acute abdomen and acute pancreatitis. Next, we will explore the complications of established GI and liver disease including PUD, IBD, and cirrhosis. Moving on, we will consider cancer screening and prevention strategies for colorectal cancer and HCC. Finally, we will conclude with patient education, dietary modification, vaccination, and health promotion relevant to the Nigerian context.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 5.1:** describe the clinical features and management of the acute abdomen and acute pancreatitis,
- **SLO 5.2:** describe the major complications of peptic ulcer disease, inflammatory bowel disease, and liver cirrhosis,
- **SLO 5.3:** describe evidence-based screening and prevention strategies for colorectal cancer and hepatocellular carcinoma,
- **SLO 5.4:** describe the prevention of major GI infections relevant to Nigeria, and

- **SLO 5.5:** describe dietary and lifestyle modification, vaccination, and health promotion strategies for GI disease prevention.

5.1 Gastrointestinal Emergencies

5.1.1 Acute abdomen and surgical emergencies

The acute abdomen is a clinical syndrome of sudden onset severe abdominal pain requiring urgent assessment. The most important surgical emergencies are **acute appendicitis** (periumbilical pain migrating to the right iliac fossa, anorexia, low-grade fever, raised white cell count and CRP; managed by appendicectomy), **bowel obstruction** (colicky pain, vomiting, distension, absolute constipation; dilated bowel loops with air-fluid levels on plain X-ray; managed by nasogastric decompression and surgery), and **bowel perforation** (sudden severe pain, peritonitis, free gas under diaphragm on erect chest X-ray; requires emergency laparotomy).

Mesenteric ischaemia is caused by occlusion of the superior mesenteric artery (from embolism or thrombosis in patients with atrial fibrillation or atherosclerosis), presenting with severe central abdominal pain out of proportion to physical signs, bloody diarrhoea, and rapid progression to shock. It requires urgent CT angiography and emergency vascular surgery or endovascular intervention. Mortality exceeds 70 percent without early intervention.

Acute appendicitis presents with pain migrating from the _____ region to the right iliac fossa. Bowel perforation shows _____ gas under the diaphragm on an erect chest X-ray.

5.1.2 Acute pancreatitis

Acute pancreatitis is inflammation of the pancreas caused by premature activation of pancreatic enzymes within the gland. The two most common causes are **gallstones** (accounting for approximately 45 percent of cases: a gallstone impacted at the ampulla of Vater obstructs pancreatic duct drainage) and **alcohol** (approximately 35 percent; chronic alcohol use increases

zymogen activation within acinar cells). Other causes include hypertriglyceridaemia, hypercalcaemia, drugs (azathioprine, thiazide diuretics), ERCP, and trauma.

Clinical features: sudden severe epigastric pain radiating to the back, nausea, vomiting, fever, and abdominal tenderness. Serum amylase or lipase above three times the upper limit of normal confirms the diagnosis. Severity is assessed by the modified Glasgow (Imrie) score or CT severity index. Severe pancreatitis (Glasgow score above 3) complications include pancreatic necrosis, pseudocyst, and systemic complications (ARDS, acute renal failure, sepsis). Management: aggressive IV fluid resuscitation, analgesia, nil by mouth initially, and early enteral nutrition. Infected pancreatic necrosis requires IV antibiotics (meropenem) and CT-guided drainage or necrosectomy.

The two most common causes of acute pancreatitis are _____ (45 percent) and alcohol (35 percent). Diagnosis is confirmed by serum amylase or _____ above three times the upper limit of normal.

5.1.3 Management of gastrointestinal emergencies

All GI emergencies are managed following the ABCDE approach. Resuscitation with intravenous fluids is the immediate priority: two large-bore cannulae, blood for FBC, urea and electrolytes, liver function tests, coagulation screen, group and crossmatch, blood cultures, and serum lactate (elevated in ischaemia and sepsis). Nasogastric tube decompression is used in bowel obstruction and ileus. Urinary catheter insertion allows fluid balance monitoring. Early surgical review is essential for all patients with peritonitis, perforation, or obstruction.

Analgesia must not be withheld from patients with acute abdominal pain: adequate analgesia does not mask signs and improves clinical assessment. Intravenous morphine or pethidine is appropriate for severe pain. Early antibiotics (piperacillin-tazobactam or cefuroxime plus metronidazole) are given for suspected intra-abdominal sepsis. CT of the abdomen and pelvis with contrast is the investigation of choice for most acute abdominal emergencies and guides the decision for surgical intervention.

All GI emergencies begin with _____ resuscitation, two large-bore cannulae, and blood investigations. CT of the abdomen and pelvis with _____ is the investigation of choice for most GI emergencies.

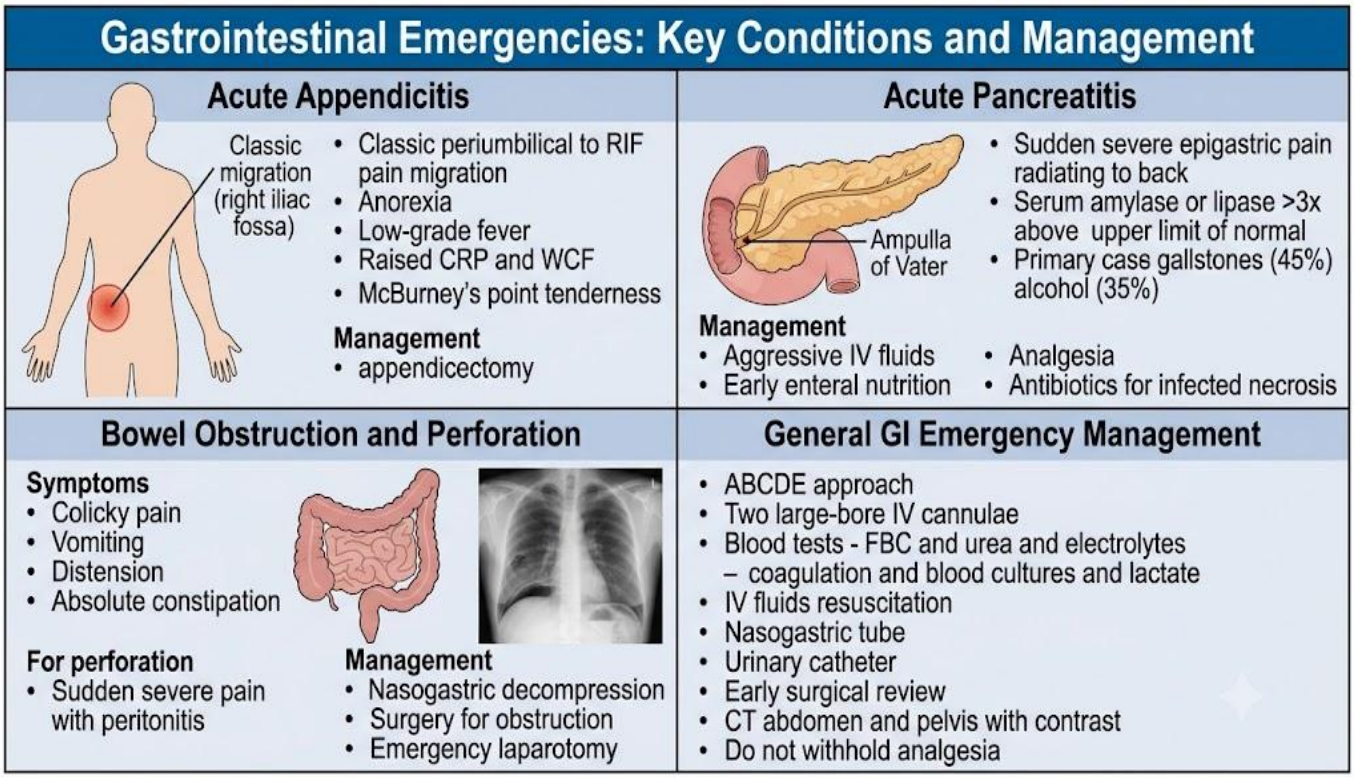

Figure 5.1: Gastrointestinal emergencies: key conditions and management

Figure 5.1: Gastrointestinal emergencies: key conditions and management

Pilot questions 5.1

1. Describe the clinical features and management of acute appendicitis. (Linked to SLO 5.1)
2. State the two most common causes of acute pancreatitis and describe how each causes pancreatic inflammation. (Linked to SLO 5.1)
3. Describe the clinical features and diagnosis of acute pancreatitis. (Linked to SLO 5.1)
4. Describe the immediate management of a patient with a GI emergency. (Linked to SLO 5.1)

5. State the investigation of choice for most GI emergencies and explain why. (Linked to SLO 5.1)

5.2 Complications of GI and Liver Disease

5.2.1 Complications of peptic ulcer disease and IBD

PUD complications include haemorrhage (the most common: haematemesis or melaena, managed by endoscopic haemostasis and IV PPI), perforation (sudden peritonitis and free gas on erect CXR: emergency laparotomy), and gastric outlet obstruction (pyloric scarring causing persistent vomiting of undigested food, succussion splash: endoscopic balloon dilatation or surgery). **IBD complications** include toxic megacolon in UC (colonic dilatation above 6 cm with systemic toxicity: IV steroids, IV antibiotics, and emergency colectomy if no improvement in 24 to 72 hours), stricturing and fistulating disease in Crohn's (requiring surgery), perianal disease, and malabsorption.

Both PUD and IBD carry an increased risk of GI malignancy with long-standing disease. UC patients have a markedly elevated risk of colorectal carcinoma proportional to the duration and extent of disease, requiring colonoscopic surveillance every 1 to 2 years after 8 to 10 years of pan-colitis. Crohn's disease patients have an elevated risk of small bowel and colonic adenocarcinoma. PUD patients with chronic H. pylori infection have an increased risk of gastric carcinoma and gastric MALT lymphoma.

Toxic megacolon in UC is defined as colonic dilatation above _____ cm with systemic toxicity and requires IV steroids and IV antibiotics. UC patients require colonoscopic surveillance every 1 to 2 years after _____ years of pan-colitis.

5.2.2 Complications of liver disease

The major complications of liver cirrhosis were addressed in Series 4. Key complications to emphasise in the context of prevention and management include spontaneous bacterial peritonitis (SBP: daily norfloxacin or ciprofloxacin as secondary prophylaxis reduces recurrence from 70

percent to below 20 percent), hepatic encephalopathy (identify and treat precipitants: GI bleeding, infection, constipation, dehydration, sedative drugs; long-term rifaximin reduces recurrence), and hepatorenal syndrome (terlipressin and albumin infusion as the medical bridge to liver transplantation).

Acute-on-chronic liver failure (ACLF) is a distinct syndrome of acute decompensation of cirrhosis with organ failure, precipitated by bacterial infection (most commonly), GI bleeding, active hepatitis, or drugs. It carries a 28-day mortality of 30 to 90 percent depending on the number of organ failures. Management is intensive supportive care in an ICU, treatment of the precipitating event, and urgent listing for liver transplantation. The European Association for the Study of the Liver (EASL) ACLF criteria use organ failure scores to define severity.

SBP secondary prophylaxis with daily _____ reduces recurrence from 70 percent to below 20 percent. ACLF is precipitated most commonly by bacterial _____ and carries a 28-day mortality of 30 to 90 percent.

5.2.3 GI malignancy complications

Complications of colorectal carcinoma include bowel obstruction (most common complication: presenting as large bowel obstruction requiring emergency decompressing colostomy or self-expanding metallic stent as bridge to elective surgery), perforation (peritonitis requiring emergency laparotomy), fistula formation, and iron deficiency anaemia from chronic bleeding. Liver metastases are present in approximately 20 percent of patients at diagnosis (synchronous metastases), causing hepatomegaly and jaundice.

Complications of oesophageal carcinoma include dysphagia (the cardinal symptom; managed by self-expanding metallic stent for palliation), tracheo-oesophageal fistula (aspiration pneumonia from food entering the airway), and malnutrition from obstruction. **Complications of gastric carcinoma** include gastric outlet obstruction (persistent vomiting), malnutrition, and peritoneal carcinomatosis (causing ascites with low SAAG). All GI malignancies require a multidisciplinary team approach combining surgery, oncology, gastroenterology, radiology, and palliative care.

The most common complication of colorectal carcinoma is large bowel _____. Oesophageal carcinoma is palliated with a self-expanding metallic _____ to relieve dysphagia.

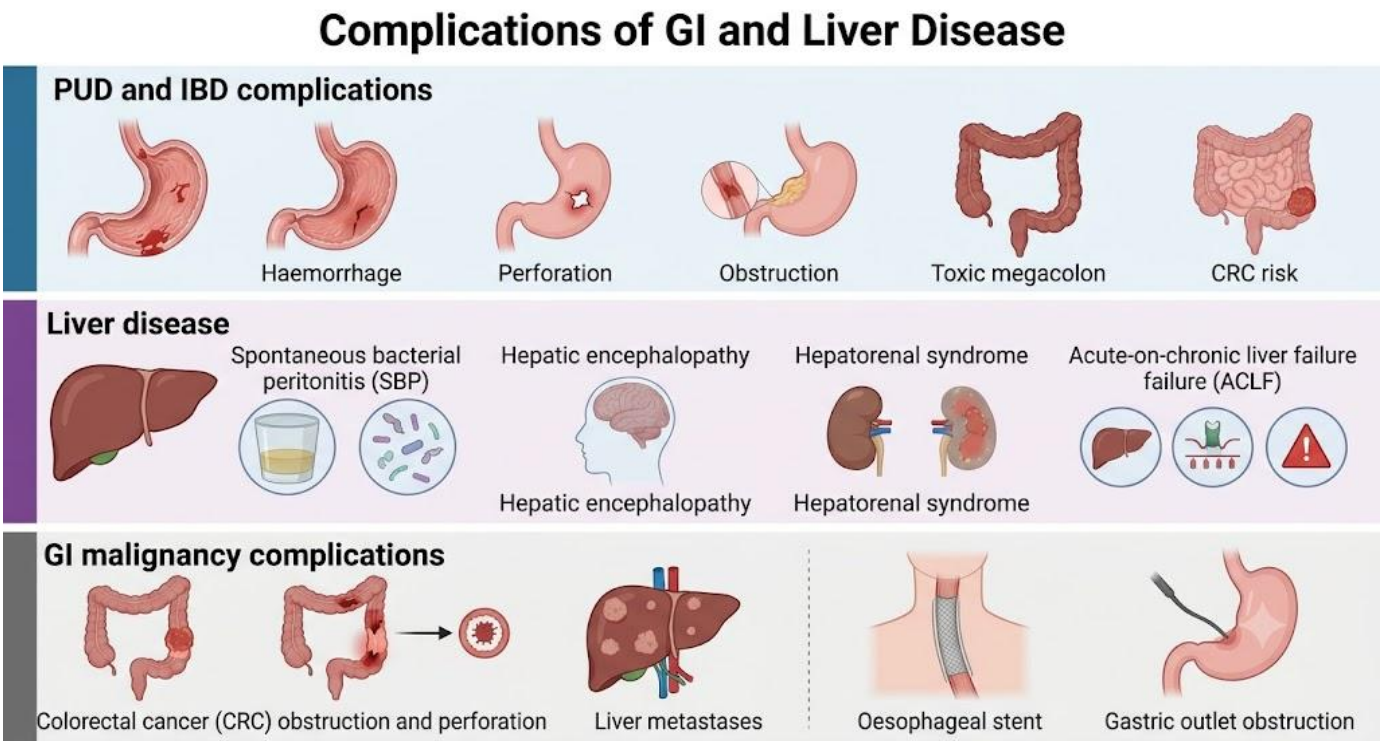

Figure 5.2: Complications of GI and liver disease

Pilot questions 5.2

1. Describe the three major complications of peptic ulcer disease. (Linked to SLO 5.2)
2. Describe toxic megacolon and state its management. (Linked to SLO 5.2)
3. Describe the secondary prophylaxis of spontaneous bacterial peritonitis. (Linked to SLO 5.2)
4. Describe the complications of colorectal carcinoma. (Linked to SLO 5.2)
5. Describe acute-on-chronic liver failure and state the most common precipitating cause. (Linked to SLO 5.2)

5.3 Cancer Screening and Prevention

5.3.1 Colorectal cancer screening

Colorectal cancer (CRC) screening aims to detect early-stage cancers and pre-malignant polyps before symptoms develop, when treatment is curative. In high-income countries, population-based screening with faecal immunochemical testing (FIT: detects occult blood in the stool; a non-invasive annual or biennial test) or colonoscopy (the gold standard for both detection and polyp removal) is recommended for average-risk individuals from age 50 years. In Nigeria, population-based screening is not yet established, but targeted screening should be offered to high-risk individuals: those above 40 years with rectal bleeding, change in bowel habit, iron deficiency anaemia, or a first-degree relative with CRC.

Colonoscopic surveillance is indicated for: UC patients after 8 to 10 years of pan-colitis (every 1 to 2 years), patients with personal or family history of adenomatous polyps (every 3 to 5 years depending on polyp number and size), Lynch syndrome carriers (every 1 to 2 years from age 25), and familial adenomatous polyposis (annual from age 13 to 15: prophylactic colectomy once polyps develop). Post-polypectomy surveillance intervals depend on the number, size, and histology of removed polyps.

Colorectal cancer screening in average-risk populations uses the faecal _____ test or colonoscopy. UC patients require colonoscopic surveillance every 1 to 2 years after _____ years of pan-colitis.

5.3.2 HCC surveillance and prevention

HCC surveillance is recommended for all patients with liver cirrhosis regardless of aetiology, and for non-cirrhotic chronic HBV patients with active viral replication or a family history of HCC. The standard protocol is 6-monthly abdominal ultrasound and serum alpha-fetoprotein (AFP). Surveillance detects HCC at an early and potentially curable stage; studies show that surveillance doubles 5-year survival compared with symptomatic detection.

HCC prevention: hepatitis B vaccination (the most effective strategy in Nigeria; prevents the primary risk factor), treatment of chronic HBV with tenofovir or entecavir (reduces HCC risk by

approximately 40 to 50 percent), treatment of chronic HCV with DAA therapy (reduces HCC risk in patients who achieve sustained virological response), reduction of aflatoxin B1 exposure through improved grain storage, drying, and processing, weight loss and management of metabolic risk factors (reduces NAFLD-related HCC risk), and smoking cessation and alcohol abstinence.

HCC surveillance uses 6-monthly abdominal _____ and serum AFP. HBV treatment with tenofovir or entecavir reduces HCC risk by approximately _____ to 50 percent.

5.3.3 Prevention of GI infections

Prevention of waterborne GI infections (cholera, typhoid, hepatitis A and E, amoebic dysentery, giardiasis) requires access to safe drinking water (chlorination and filtration), adequate sanitation (improved latrines and sewage disposal), food hygiene (cooking food thoroughly, avoiding raw shellfish, safe food handling), and hand hygiene (soap and water washing before eating and after using the toilet). These public health measures have the greatest impact at the population level.

Vaccination prevents several important GI infections: hepatitis B vaccine (3-dose schedule; birth dose for prevention of perinatal transmission), inactivated hepatitis A vaccine (2 doses; recommended for travellers to endemic areas, food handlers, and healthcare workers), oral typhoid vaccine (Ty21a: 3 doses) or injectable Vi polysaccharide typhoid vaccine (single dose; recommended for travellers and endemic communities), and oral cholera vaccine (Dukoral: 2 doses; recommended for travellers to endemic areas). Cotrimoxazole prophylaxis is given to HIV-positive patients with CD4 below 200 to prevent opportunistic GI infections.

Safe _____ water, sanitation, food hygiene, and hand washing are the most effective population-level measures to prevent waterborne GI infections. Cotrimoxazole prophylaxis is given to HIV-positive patients with CD4 below _____ to prevent opportunistic GI infections.

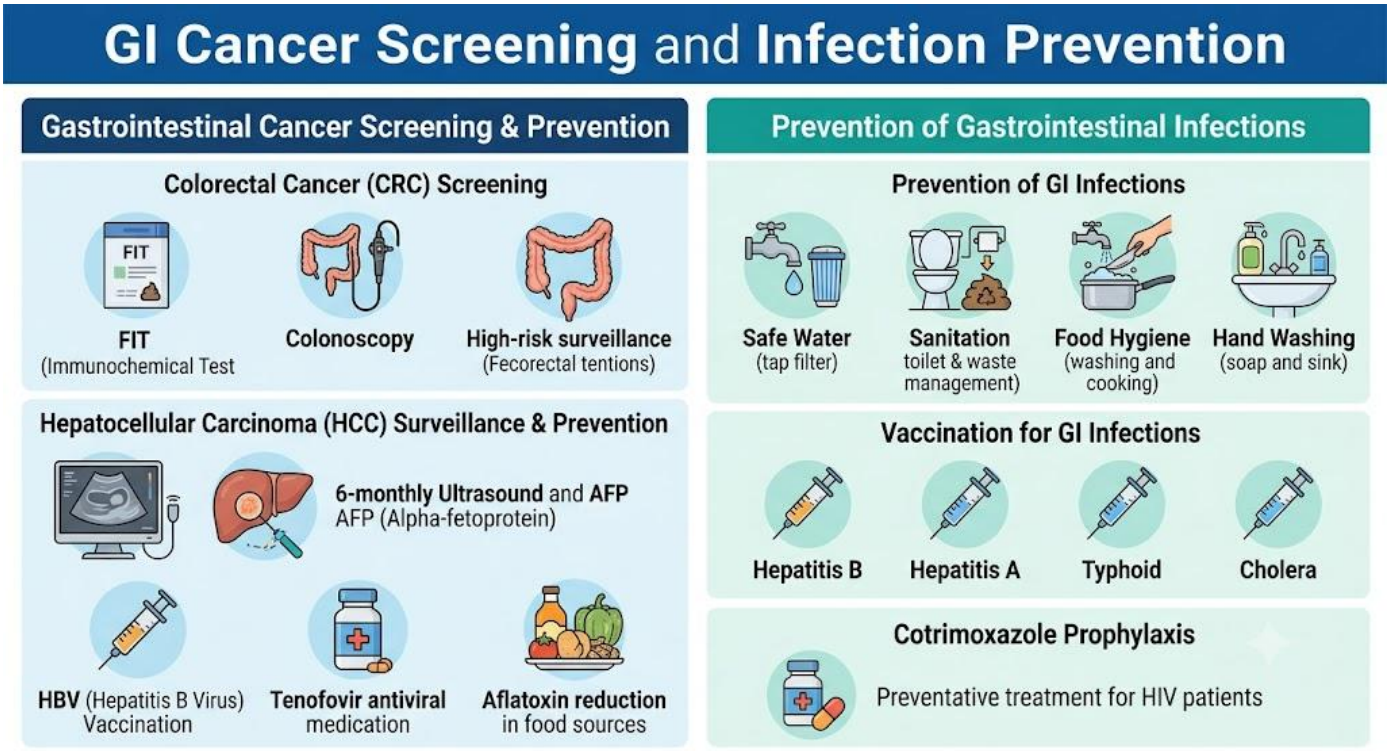

Figure 5.3: GI cancer screening and infection prevention strategies

Pilot questions 5.3

1. Describe the recommended colorectal cancer screening strategies for average-risk and high-risk populations. (Linked to SLO 5.3)
2. Describe the HCC surveillance protocol and state who should undergo surveillance. (Linked to SLO 5.3)
3. Describe the prevention strategies for HCC in Nigeria. (Linked to SLO 5.3)
4. Describe the public health measures for prevention of waterborne GI infections in Nigeria. (Linked to SLO 5.4)
5. State four GI infections preventable by vaccination and name the relevant vaccine for each. (Linked to SLO 5.4)

5.4 Patient Education and Health Promotion

5.4.1 Dietary modification and lifestyle

Dietary modification is the cornerstone of prevention for the majority of chronic GI diseases. A high-fibre diet (above 25 to 30 grams of fibre per day from vegetables, fruits, legumes, and whole grains) reduces the risk of colorectal cancer, diverticular disease, constipation, and haemorrhoids by reducing colonic transit time and intraluminal pressure. Adequate hydration (at least 2 litres of water per day) supports normal bowel function. Reduction of red and processed meat consumption (below 500 grams of cooked red meat per week) reduces colorectal cancer risk. Avoidance of alcohol reduces the risk of alcoholic liver disease, cirrhosis, and upper GI cancers.

Weight management (achieving and maintaining a healthy BMI of 18.5 to 24.9) reduces the risk of NAFLD, GORD, colorectal cancer, and gallstone disease. Physical activity (at least 150 minutes of moderate-intensity exercise per week) independently reduces colorectal cancer risk by approximately 20 to 25 percent. Smoking cessation reduces the risk of oesophageal carcinoma, gastric carcinoma, colorectal cancer, GORD, PUD, and Crohn's disease flares. These lifestyle measures are applicable and achievable in the Nigerian context with appropriate patient education.

A high-_____ diet reduces the risk of colorectal cancer, diverticular disease, and haemorrhoids. Physical activity reduces colorectal cancer risk by approximately _____ to 25 percent.

5.4.2 Vaccination and chemoprophylaxis

The hepatitis B vaccine is the single most important GI disease prevention measure in Nigeria. The 3-dose schedule (given at 0, 1, and 6 months) achieves protective anti-HBs titres in above 95 percent of recipients. A birth dose given within 24 hours of delivery, followed by the standard EPI schedule, prevents perinatal hepatitis B transmission and is recommended by WHO. Healthcare workers must be vaccinated and titres checked post-vaccination. Hepatitis A vaccine

is a 2-dose inactivated vaccine providing long-term immunity and recommended for food handlers, travellers to endemic areas, and patients with chronic liver disease.

Chemoprophylaxis in GI disease: low-dose aspirin (75 to 100 mg daily) reduces colorectal cancer risk by approximately 25 to 50 percent with long-term use and is recommended as secondary chemoprevention for patients with Lynch syndrome or a history of adenomatous polyps, balanced against the risk of GI bleeding. Proton pump inhibitors are given as primary prophylaxis to all patients on long-term NSAIDs or anticoagulants to reduce peptic ulceration risk. Cotrimoxazole prophylaxis in HIV-positive patients with CD4 below 200 prevents Pneumocystis pneumonia and GI opportunistic infections.

The hepatitis B birth dose given within _____ hours of delivery prevents perinatal HBV transmission. Low-dose aspirin reduces colorectal cancer risk by approximately 25 to _____ percent with long-term use.

5.4.3 Health promotion in the Nigerian context

Effective health promotion in Nigeria must address the specific epidemiological context: the high burden of chronic HBV, typhoid, amoebic dysentery, and intestinal parasitic infections driven by limited access to safe water, adequate sanitation, and healthcare infrastructure. Community health education campaigns should promote hand hygiene, safe food handling, exclusive breastfeeding (which reduces infant diarrhoeal disease mortality), oral rehydration therapy for diarrhoeal disease, and early health-seeking behaviour for GI symptoms including prolonged diarrhoea, rectal bleeding, jaundice, and unintentional weight loss.

Task-shifting to community health workers and traditional birth attendants can extend the reach of GI health promotion, particularly for HBV birth-dose vaccination, oral rehydration therapy distribution, and early referral of patients with chronic GI symptoms. **Aflatoxin control** through government-regulated grain storage standards, rapid testing of staple foods at markets, and community education on safe storage practices can significantly reduce HCC incidence in Nigeria. Integration of GI health promotion into existing HIV, tuberculosis, maternal and child health, and non-communicable disease programmes maximises efficiency and reach.

_____ health workers can extend GI health promotion by delivering HBV birth-dose vaccination and oral rehydration therapy. Aflatoxin control reduces the risk of _____ carcinoma (HCC) in Nigeria.

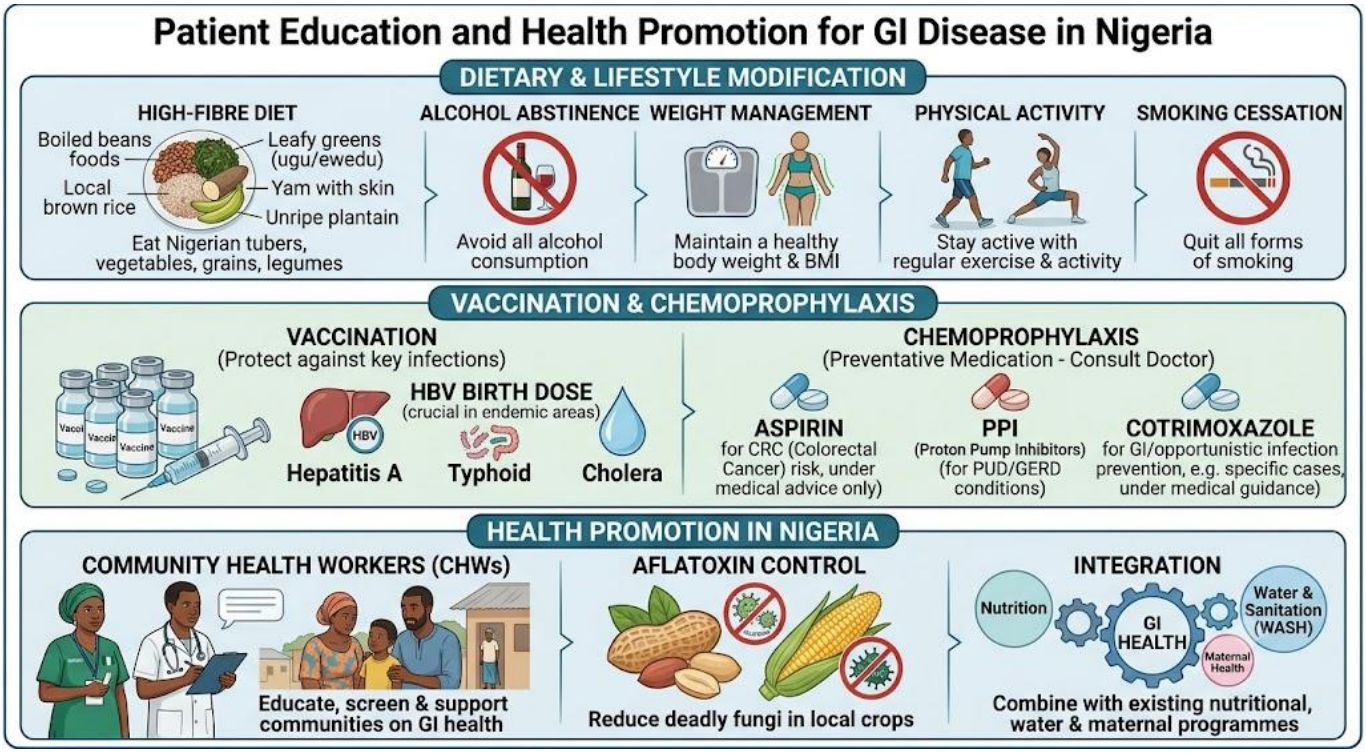

Figure 5.4: Patient education and health promotion for GI disease prevention in Nigeria

Pilot questions 5.4

1. Describe four dietary and lifestyle modifications that reduce the risk of colorectal cancer. (Linked to SLO 5.5)
2. Describe the hepatitis B vaccination schedule and explain the importance of the birth dose. (Linked to SLO 5.5)
3. Explain the role of low-dose aspirin in colorectal cancer prevention. (Linked to SLO 5.5)
4. Describe the key health promotion messages for GI disease prevention in Nigeria. (Linked to SLO 5.5)
5. Describe how task-shifting and aflatoxin control can reduce the GI disease burden in Nigeria. (Linked to SLO 5.5)

Series Summary

This Series brought together the major complications of GI and liver disease and the evidence-based strategies for prevention. We began with GI emergencies: acute appendicitis (periumbilical to RIF pain migration; appendicectomy), bowel obstruction and perforation (free gas on erect CXR; emergency laparotomy), and acute pancreatitis (gallstones 45 percent and alcohol 35 percent; aggressive IV fluids; amylase or lipase above three times normal). We then examined complications of established disease: PUD (haemorrhage, perforation, gastric outlet obstruction), IBD (toxic megacolon in UC; fistulating and stricturing Crohn's; CRC risk requiring surveillance after 8 to 10 years), and liver disease (SBP secondary prophylaxis with daily norfloxacin; hepatic encephalopathy treated with lactulose and rifaximin; HRS with terlipressin and albumin; ACLF with 30 to 90 percent mortality). Moving on, we covered cancer screening: CRC (FIT test or colonoscopy for average risk from age 50; surveillance colonoscopy for high-risk groups) and HCC (6-monthly ultrasound and AFP for all cirrhotic patients; HBV vaccination the most effective prevention). Finally, we addressed health promotion: high-fibre diet, weight management, physical activity, and smoking cessation for GI disease prevention; HBV birth-dose vaccination (the single most important GI prevention measure in Nigeria); low-dose aspirin for CRC chemoprevention; aflatoxin control and community health worker task-shifting in Nigeria.

By completing this Series, you should now be able to describe the management of GI emergencies and the complications of major GI and liver diseases, describe CRC and HCC screening and prevention strategies, describe the prevention of major GI infections, and outline dietary, lifestyle, vaccination, and health promotion strategies relevant to Nigeria. These abilities align with SLOs 5.1 to 5.5 and complete your preparation in Gastroenterology I.

Glossary of Terms

- **Acute-on-chronic liver failure (ACLF):** a syndrome of acute decompensation of cirrhosis with organ failure, most commonly precipitated by bacterial infection; carries a 28-day mortality of 30 to 90 percent depending on the number of organ failures.

- **Aflatoxin B1:** a mycotoxin produced by Aspergillus flavus on improperly stored staple foods; a potent hepatic carcinogen synergising with HBV to markedly increase HCC risk in Nigeria.
- **Acute pancreatitis:** acute inflammation of the pancreas caused by premature activation of pancreatic enzymes, most commonly from gallstones or alcohol; diagnosed by serum amylase or lipase above three times the upper limit of normal.
- **Faecal immunochemical test (FIT):** a non-invasive colorectal cancer screening test that detects haemoglobin in the stool from occult GI bleeding; used as an annual or biennial screening test for average-risk individuals from age 50.
- **Glasgow (Imrie) score:** a severity scoring system for acute pancreatitis assessed at 48 hours, using PaO2, age, white cell count, serum calcium, urea, LDH, albumin, and blood glucose; a score above 3 indicates severe pancreatitis.
- **Mesenteric ischaemia:** acute or chronic ischaemia of the intestine from occlusion or reduced flow in the superior mesenteric artery; presents with severe central abdominal pain out of proportion to signs; mortality exceeds 70 percent without early intervention.
- **Spontaneous bacterial peritonitis (SBP):** infection of ascitic fluid without a perforated viscus; diagnosed by a neutrophil count above 250 cells per microlitre in ascitic fluid; treated with IV cefotaxime; secondary prophylaxis with daily norfloxacin reduces recurrence.
- **Task-shifting:** the delegation of clinical tasks from specialist healthcare workers to community health workers, traditional birth attendants, or trained lay persons; extends the reach of health promotion and vaccination in resource-limited settings.
- **Toxic megacolon:** acute dilatation of the colon above 6 cm with systemic toxicity; a life-threatening complication of severe UC or Crohn's colitis; managed with IV steroids, IV antibiotics, and emergency colectomy if no improvement.
- **Vi polysaccharide typhoid vaccine:** a single-dose injectable vaccine against Salmonella typhi; provides protection for 2 to 3 years; recommended for travellers to endemic areas and communities with poor water sanitation in Nigeria.

Concept Check Questions [Series 5]

Objective questions

1. Acute pancreatitis is diagnosed by serum amylase or _____ above three times the upper limit of normal, in the context of severe epigastric pain. (Linked to SLO 5.1)
2. Toxic megacolon in ulcerative colitis is defined as colonic dilatation above _____ cm with systemic toxicity and requires IV steroids, IV antibiotics, and emergency colectomy. (Linked to SLO 5.2)
3. HCC surveillance uses 6-monthly abdominal ultrasound and serum _____ in all cirrhotic patients. (Linked to SLO 5.3)
4. The hepatitis B _____ dose given within 24 hours of delivery is the most important measure to prevent perinatal HBV transmission in Nigeria. (Linked to SLO 5.5)
5. _____ control, through improved grain storage and drying of staple foods, reduces the risk of hepatocellular carcinoma in Nigeria. (Linked to SLO 5.5)

Short-answer questions

6. Describe the two most common causes of acute pancreatitis and explain how each causes pancreatic inflammation. (Linked to SLO 5.1)
7. Describe the secondary prophylaxis of spontaneous bacterial peritonitis and state why it is important. (Linked to SLO 5.2)
8. State four GI infections preventable by vaccination and name the vaccine for each. (Linked to SLO 5.4)
9. Describe four lifestyle modifications that reduce the risk of colorectal cancer. (Linked to SLO 5.5)

10. Explain the role of community health workers in reducing the GI disease burden in Nigeria. (Linked to SLO 5.5)

Long-answer questions

11. Describe the clinical features, diagnosis, and management of acute pancreatitis, including its complications. (Linked to SLO 5.1)
12. Describe the major complications of liver cirrhosis and the evidence-based management of each. (Linked to SLO 5.2)
13. Describe the colorectal cancer screening strategies for average-risk and high-risk populations, including colonoscopic surveillance indications. (Linked to SLO 5.3)
14. Describe the HCC prevention and surveillance strategies applicable to Nigeria. (Linked to SLOs 5.3 and 5.4)
15. Describe the dietary, lifestyle, vaccination, and health promotion strategies for reducing the burden of GI disease in Nigeria. (Linked to SLO 5.5)

Answers to Concept Check Questions [Series 5]

Objective questions

1. Lipase.
2. 6.
3. AFP (alpha-fetoprotein).
4. Birth.
5. Aflatoxin.

Short-answer questions

6. Gallstones (45 percent) cause pancreatitis by impacting at the ampulla of Vater, obstructing pancreatic duct drainage and causing premature activation of pancreatic enzymes within the gland. Alcohol (35 percent) causes pancreatitis by directly stimulating premature zymogen activation within acinar cells and increasing pancreatic duct permeability, leading to autodigestion of the pancreas.

7. Secondary prophylaxis of SBP uses daily oral norfloxacin 400 mg (or ciprofloxacin 500 mg), which reduces the recurrence rate from approximately 70 percent to below 20 percent per year. It is important because SBP recurrence is associated with a very high 1-year mortality in cirrhotic patients, and long-term prophylaxis significantly improves survival.

8. Hepatitis B (3-dose schedule with birth dose: prevents perinatal HBV transmission). Hepatitis A (2-dose inactivated vaccine: prevents HAV infection). Typhoid fever (Vi polysaccharide vaccine single dose: prevents Salmonella typhi infection). Cholera (Dukoral oral vaccine 2 doses: prevents Vibrio cholerae infection).

9. High-fibre diet above 25 to 30 grams per day reduces colonic transit time and intraluminal carcinogen exposure. Regular physical activity (at least 150 minutes per week) reduces CRC risk by 20 to 25 percent. Alcohol abstinence reduces the risk of colorectal adenoma formation and carcinoma. Avoidance of red and processed meat (below 500 grams cooked red meat per week) reduces exposure to N-nitroso compounds that damage colonic DNA.

10. Community health workers extend GI health promotion by delivering HBV birth-dose vaccination within 24 hours of delivery to prevent perinatal transmission, distributing oral rehydration therapy for acute diarrhoeal disease, promoting hand hygiene and safe food handling in communities, and identifying and referring patients with chronic GI symptoms such as rectal bleeding, jaundice, and unintentional weight loss for clinical assessment.

Long-answer questions

11. **Basic response:** Acute pancreatitis presents with sudden severe epigastric pain radiating to the back, nausea, vomiting, fever, and abdominal tenderness; serum amylase or lipase above three times the upper limit of normal confirms the diagnosis. Causes: gallstones (45 percent: ampullary impaction obstructing pancreatic duct drainage) and alcohol (35 percent). Severity: Glasgow score above 3 at 48 hours indicates severe pancreatitis. Management: aggressive IV fluid resuscitation (the most important intervention), analgesia, nil by mouth then early enteral nutrition via nasogastric tube (superior to parenteral nutrition), and antibiotics (meropenem) only for confirmed infected pancreatic necrosis. Complications: pancreatic necrosis (CT-guided drainage or necrosectomy for infected necrosis), pseudocyst (drainage if symptomatic), systemic complications (ARDS, AKI, sepsis: managed in ICU).

Value-added response: Early and aggressive IV fluid resuscitation with Hartmann's solution (not normal saline) in the first 24 to 48 hours is the most important intervention in acute pancreatitis and significantly reduces the risk of pancreatic necrosis by maintaining pancreatic microcirculation. In Nigeria, cholecystectomy should be performed during the same hospital admission for gallstone pancreatitis to prevent recurrence.

12. **Basic response:** Portal hypertension causes oesophageal variceal haemorrhage: managed by terlipressin (reduces portal pressure), IV cefotaxime (SBP prophylaxis), and OGD band ligation within 24 hours; TIPSS for rebleeding. Ascites: managed with salt restriction, spironolactone (100 to 400 mg), and furosemide (40 to 160 mg); large-volume paracentesis with albumin for tense ascites; TIPSS for refractory ascites. Spontaneous bacterial peritonitis: IV cefotaxime for treatment; daily oral norfloxacin for secondary prophylaxis (reduces recurrence from 70 percent to below 20 percent). Hepatic encephalopathy: identify and treat precipitants; lactulose and rifaximin; avoid sedatives. Hepatorenal syndrome: terlipressin and albumin infusion; liver transplantation.

Value-added response: All patients with decompensated cirrhosis and any complication should be assessed for liver transplantation eligibility, as this is the only curative intervention. Early

referral to a hepatology centre capable of performing transplantation is the most important prognostic intervention in decompensated cirrhosis.

13. **Basic response:** Average-risk screening (age 50, no personal or family history of CRC or polyps): faecal immunochemical test (FIT) annually or biennially (detects occult blood; positive FIT requires colonoscopy) or colonoscopy every 10 years. High-risk surveillance: UC pan-colitis patients (colonoscopy every 1 to 2 years after 8 to 10 years of disease), patients with personal history of adenomatous polyps (3 to 5 years depending on polyp number and size), Lynch syndrome carriers (every 1 to 2 years from age 25), and familial adenomatous polyposis (annual from age 13 to 15; prophylactic colectomy once polyps develop). In Nigeria: no population-based screening; targeted colonoscopy for patients above 40 with rectal bleeding, change in bowel habit, iron deficiency anaemia, or family history of CRC.

Value-added response: Colonoscopic polypectomy is one of the most cost-effective cancer prevention interventions in medicine: removing an adenomatous polyp before malignant transformation prevents a CRC that would otherwise require surgery, chemotherapy, and long-term oncological follow-up. Investment in colonoscopy infrastructure in Nigerian tertiary hospitals is a priority for reducing future CRC burden.

14. **Basic response:** HCC surveillance: all cirrhotic patients regardless of aetiology should undergo 6-monthly abdominal ultrasound and serum AFP; non-cirrhotic chronic HBV patients with active viral replication or family history of HCC also require surveillance; surveillance doubles 5-year survival by detecting HCC at an early and potentially curable stage. Prevention: hepatitis B vaccination with 3-dose schedule and birth dose within 24 hours of delivery (the single most effective HCC prevention strategy in Nigeria, preventing the primary risk factor); treatment of chronic HBV with tenofovir or entecavir (reduces HCC risk by 40 to 50 percent); DAA therapy for chronic HCV (reduces HCC risk in patients achieving sustained virological response); reduction of aflatoxin B1 exposure through improved grain storage, drying, and processing standards; weight loss and management of metabolic risk factors (reduces NAFLD-related HCC risk); alcohol abstinence and smoking cessation.

Value-added response: In Nigeria, the combination of universal HBV vaccination with a birth dose and treatment of all HBsAg-positive individuals who meet treatment criteria with tenofovir has the potential to dramatically reduce HCC incidence over the next 20 to 30 years; the challenge is achieving sufficient vaccination coverage and linking HBsAg-positive patients to antiviral treatment.

15. **Basic response:** Dietary modification: high-fibre diet (above 25 to 30 grams per day: reduces CRC, diverticular disease, constipation), reduce red and processed meat (below 500 grams cooked per week: reduces CRC risk), avoid alcohol (reduces alcoholic liver disease, cirrhosis, oesophageal and gastric cancers), and maintain a healthy BMI (reduces NAFLD, GORD, gallstones, and CRC). Lifestyle: physical activity (at least 150 minutes per week: reduces CRC risk 20 to 25 percent), smoking cessation (reduces oesophageal and gastric carcinoma, PUD, and Crohn's disease flares). Vaccination: hepatitis B (birth dose within 24 hours: most important in Nigeria), hepatitis A (food handlers and travellers), typhoid (Vi polysaccharide), cholera (Dukoral). Chemoprophylaxis: low-dose aspirin (75 to 100 mg: reduces CRC risk 25 to 50 percent in Lynch syndrome and adenoma patients), PPI for NSAID users, cotrimoxazole for HIV patients with CD4 below 200. Health promotion: community health worker deployment for HBV birth-dose vaccination and ORT distribution; aflatoxin control through grain safety standards; integration into HIV, TB, maternal and child health, and NCD programmes for maximum reach.

Value-added response: In Nigeria, the most impactful single investment in GI disease prevention is achieving universal HBV birth-dose vaccination coverage, as this prevents perinatal HBV transmission, which accounts for the majority of chronic HBV, and will reduce the future burden of cirrhosis and HCC over the coming decades.

Answers to Pilot Questions [Series 5]

Part 5.1

1. Acute appendicitis presents with periumbilical pain that migrates to the right iliac fossa over 6 to 12 hours, anorexia, low-grade fever, nausea, and vomiting. Examination reveals McBurney's point tenderness, guarding, and Rovsing's sign. Investigations show raised white cell count and CRP; CT abdomen confirms the diagnosis. Management is appendicectomy (laparoscopic preferred).
2. Gallstones (45 percent): a stone impacted at the ampulla of Vater obstructs pancreatic duct drainage, causing back-pressure and premature activation of pancreatic enzymes within the gland, leading to autodigestion. Alcohol (35 percent): chronic alcohol use directly stimulates premature zymogen activation within acinar cells and increases pancreatic duct permeability, allowing activated enzymes to cause parenchymal destruction.
3. Sudden severe epigastric pain radiating to the back, nausea, vomiting, fever, and abdominal tenderness with periumbilical guarding. Diagnosis: serum amylase or lipase above three times the upper limit of normal. Severity assessed by modified Glasgow score at 48 hours (score above 3: severe disease) or CT severity index.
4. ABCDE approach: two large-bore IV cannulae, blood for FBC, urea and electrolytes, liver function tests, coagulation screen, group and crossmatch, blood cultures, and serum lactate. Aggressive IV fluid resuscitation. Nasogastric tube for bowel obstruction or ileus. Urinary catheter for fluid balance monitoring. Analgesia (IV morphine: do not withhold). Early surgical review for peritonitis, perforation, or obstruction. IV antibiotics (piperacillin-tazobactam) for intra-abdominal sepsis.
5. CT of the abdomen and pelvis with intravenous contrast is the investigation of choice because it accurately identifies the cause of the acute abdomen (appendicitis, bowel obstruction, perforation, mesenteric ischaemia, pancreatitis), delineates the extent of disease, guides the decision for surgical intervention, and is available within minutes in most tertiary hospitals.

Part 5.2

1. Haemorrhage (most common): haematemesis or melaena from a bleeding ulcer; managed by IV PPI infusion, endoscopic haemostasis (adrenaline injection and haemoclips), and blood transfusion. Perforation: sudden severe epigastric pain with peritonitis and free gas under the diaphragm on erect CXR; requires emergency laparotomy, closure of the perforation, and peritoneal lavage. Gastric outlet obstruction: persistent vomiting of undigested food, succussion splash, and weight loss from pyloric scarring; managed by endoscopic balloon dilatation or surgery.

2. Toxic megacolon is acute colonic dilatation above 6 cm on plain abdominal X-ray with systemic toxicity (fever above 38 degrees C, tachycardia, leucocytosis, anaemia, hypotension). Management: IV corticosteroids (methylprednisolone 60 mg daily), IV broad-spectrum antibiotics (piperacillin-tazobactam), nasogastric decompression, and nil by mouth. Emergency subtotal colectomy is performed if there is no improvement within 24 to 72 hours, perforation, or haemorrhage.

3. Secondary prophylaxis with daily oral norfloxacin 400 mg (or ciprofloxacin 500 mg) reduces SBP recurrence from approximately 70 percent to below 20 percent per year. It should be continued indefinitely in all patients who survive an episode of SBP because the 1-year mortality without prophylaxis exceeds 50 percent from recurrent episodes, and prophylaxis significantly improves both recurrence rates and survival.

4. Bowel obstruction (most common): large bowel obstruction presenting with abdominal distension, constipation, and colicky pain; managed by self-expanding metallic stent as a bridge to elective surgery or emergency decompressing colostomy. Perforation: generalised peritonitis requiring emergency laparotomy and bowel resection with or without primary anastomosis. Chronic occult bleeding causing iron deficiency anaemia. Liver metastases: present in 20 percent at diagnosis; hepatomegaly, jaundice, and elevated ALP.

5. ACLF is acute decompensation of liver cirrhosis with organ failure, most commonly precipitated by bacterial infection (spontaneous bacterial peritonitis, urinary tract infection, pneumonia), which accounts for approximately 33 percent of cases. Other

precipitants include GI bleeding, active hepatitis, and drugs or hepatotoxins. It carries a 28-day mortality of 30 to 90 percent depending on the number of organ failures, and management requires ICU admission, treatment of the precipitating event, and urgent liver transplantation listing.

Part 5.3

1. Average-risk individuals (age 50 years, no personal or family history of CRC): FIT test annually or biennially (positive test requires colonoscopy) or colonoscopy every 10 years. High-risk individuals: UC pan-colitis patients (colonoscopy every 1 to 2 years after 8 to 10 years of disease), patients with adenomatous polyps (surveillance colonoscopy 3 to 5 years depending on polyp burden), Lynch syndrome carriers (every 1 to 2 years from age 25 years), and familial adenomatous polyposis patients (annual colonoscopy from age 13 to 15; prophylactic colectomy once polyps develop). In Nigeria, targeted colonoscopy is recommended for patients above 40 with rectal bleeding, change in bowel habit, or iron deficiency anaemia.
2. 6-monthly abdominal ultrasound and serum AFP in all cirrhotic patients regardless of aetiology and in non-cirrhotic chronic HBV patients with active viral replication or a family history of HCC. Surveillance detects HCC at an early and potentially curable stage and doubles 5-year survival compared with symptomatic detection.
3. Hepatitis B vaccination with birth dose (within 24 hours of delivery: the most effective HCC prevention strategy in Nigeria, preventing perinatal HBV transmission). Treatment of chronic HBV with tenofovir or entecavir (reduces HCC risk by 40 to 50 percent). DAA therapy for chronic HCV (reduces HCC risk in patients achieving sustained virological response). Reduction of aflatoxin B1 exposure through improved grain storage standards, rapid food safety testing, and community education. Weight loss and management of metabolic risk factors (reduces NAFLD-related HCC risk). Alcohol abstinence and smoking cessation.

4. Safe drinking water (chlorination and filtration of community water supplies). Adequate sanitation (construction and use of improved latrines and proper sewage disposal). Food hygiene (thorough cooking of food, avoiding raw shellfish, safe food handling and storage). Hand hygiene (soap and water washing before eating and after toilet use). These measures prevent cholera, typhoid, HAV, HEV, amoebic dysentery, and giardiasis, which are all transmitted by the faecal-oral route.
5. Hepatitis B: 3-dose schedule (0, 1, 6 months) plus birth dose (Vi polysaccharide vaccine). Hepatitis A: 2-dose inactivated vaccine (recommended for food handlers, travellers, and patients with chronic liver disease). Typhoid: Vi polysaccharide vaccine (single dose; protects for 2 to 3 years). Cholera: Dukoral oral vaccine (2 doses; recommended for travellers to endemic areas and for outbreak control).

Part 5.4

1. High-fibre diet (above 25 to 30 grams per day from vegetables, fruits, and whole grains reduces colonic transit time and carcinogen exposure, reducing CRC risk). Avoidance of red and processed meat (below 500 grams cooked red meat per week reduces exposure to N-nitroso compounds that damage colonic DNA). Physical activity (at least 150 minutes of moderate-intensity exercise per week reduces CRC risk by 20 to 25 percent independently of BMI). Alcohol abstinence (alcohol is a recognised group 1 carcinogen for colorectal cancer and reduces rectal mucosal protection).
2. The standard schedule is 3 doses given at 0, 1, and 6 months, achieving protective anti-HBs titres in above 95 percent of recipients. The birth dose given within 24 hours of delivery, combined with the standard EPI schedule, is essential because the majority of chronic HBV in Nigeria is acquired through perinatal transmission from HBsAg-positive mothers; a birth dose given within 24 hours reduces perinatal transmission by approximately 95 percent.
3. Low-dose aspirin (75 to 100 mg daily) inhibits cyclo-oxygenase-2 (COX-2), which is overexpressed in colorectal adenomas and carcinomas; COX-2 inhibition reduces

prostaglandin E2, which promotes tumour growth and immune evasion. Long-term aspirin use (more than 5 years) reduces CRC risk by approximately 25 to 50 percent. It is recommended as secondary chemoprevention for patients with Lynch syndrome or a history of adenomatous polyps, balanced against the risk of GI bleeding and the need for PPI co-prescription.

4. Hand hygiene and safe food handling to prevent waterborne and foodborne GI infections. Recognition of danger signs in diarrhoeal illness (blood in stool, unable to drink, high fever, signs of dehydration) requiring prompt medical attention. Use of oral rehydration solution for diarrhoeal disease before and instead of unsafe home remedies. Early health-seeking for alarm symptoms including prolonged diarrhoea, rectal bleeding, jaundice, and unintentional weight loss. Hepatitis B birth-dose vaccination and completion of the EPI schedule.

5. Task-shifting to community health workers extends HBV birth-dose vaccination to home deliveries within 24 hours, distributes ORT packets to households with diarrhoeal illness, promotes hand hygiene and safe food storage, and identifies and refers patients with chronic GI symptoms for clinical assessment, thereby extending the reach of GI health promotion beyond health facilities. Aflatoxin control through government-regulated grain storage standards, rapid aflatoxin testing of staple foods at markets, community education on safe grain drying and storage practices, and promotion of aflatoxin-resistant crop varieties reduces aflatoxin B1 exposure and lowers HCC incidence in Nigeria.

References and Attributions [Series 5]

Textual References

- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.
- World Health Organization. (2017). Global Hepatitis Report 2017. Geneva: WHO.
- Bray, F., Ferlay, J., Soerjomataram, I., Siegel, R. L., Torre, L. A. and Jemal, A. (2018). Global Cancer Statistics 2018: GLOBOCAN Estimates of Incidence and Mortality Worldwide for 36 Cancers in 185 Countries. CA: A Cancer Journal for Clinicians, 68(6), 394 to 424.
- National Institute for Health and Care Excellence. (2019). Colorectal Cancer. NICE Guideline NG151. London: NICE.

Figures, Plates, Tables, and Boxes

- Figure 5.1: Gastrointestinal emergencies: key conditions and management. AI-generated using the prompt: "Create a professional medical infographic titled Gastrointestinal Emergencies with four quadrants covering acute appendicitis, acute pancreatitis, bowel obstruction and perforation, and general GI emergency management. Clean clinical infographic style." Suggested OER search string: "gastrointestinal emergency appendicitis pancreatitis bowel obstruction perforation management infographic OER."
- Figure 5.2: Complications of GI and liver disease. AI-generated using the prompt: "Create a professional medical infographic titled Complications of GI and Liver Disease with three vertical sections: PUD and IBD complications, liver disease complications, and GI malignancy complications. Clean clinical infographic style." Suggested OER search string: "GI liver disease complications peptic ulcer IBD cirrhosis colorectal cancer infographic OER."

- Figure 5.3: GI cancer screening and infection prevention strategies. AI-generated using the prompt: "Create a professional medical infographic titled GI Cancer Screening and Infection Prevention with two columns: left for CRC and HCC screening and prevention; right for GI infection prevention strategies. Clean professional infographic style." Suggested OER search string: "colorectal cancer screening HCC surveillance GI infection prevention vaccination infographic OER."
- Figure 5.4: Patient education and health promotion for GI disease prevention in Nigeria. AI-generated using the prompt: "Create a professional medical infographic titled Patient Education and Health Promotion for GI Disease in Nigeria with three sections: dietary and lifestyle modification, vaccination and chemoprophylaxis, and health promotion in Nigeria. Clean professional infographic style." Suggested OER search string: "GI disease prevention Nigeria diet lifestyle vaccination health promotion community infographic OER."