



La-Net

MED 608

GASTROENTEROLOGY II



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Course code: MED 608

Course title: Gastroenterology II

Course credit load: 2 Units

Series 1: Peptic Ulcer Disease

- **Part 1.1: Pathophysiology and Risk Factors of Peptic Ulcer Disease**
 - Sub Part 1.1.1: Anatomy and physiology of gastric and duodenal mucosal defence
 - Sub Part 1.1.2: Helicobacter pylori and peptic ulcer disease
 - Sub Part 1.1.3: NSAID-induced and other causes of peptic ulcer disease
- **Part 1.2: Clinical Presentation of Peptic Ulcer Disease**
 - Sub Part 1.2.1: Symptoms and signs of gastric ulcer
 - Sub Part 1.2.2: Symptoms and signs of duodenal ulcer
 - Sub Part 1.2.3: Differentiating peptic ulcer disease from other causes of dyspepsia
- **Part 1.3: Investigation of Peptic Ulcer Disease**
 - Sub Part 1.3.1: Upper gastrointestinal endoscopy
 - Sub Part 1.3.2: Tests for Helicobacter pylori
 - Sub Part 1.3.3: Investigating for malignancy in peptic ulcer disease
- **Part 1.4: Management and Complications of Peptic Ulcer Disease**
 - Sub Part 1.4.1: Medical management of peptic ulcer disease
 - Sub Part 1.4.2: Helicobacter pylori eradication therapy
 - Sub Part 1.4.3: Complications of peptic ulcer disease

Introduction

Few conditions in gastroenterology are as common, or as instructive, as **peptic ulcer disease**, a genuinely everyday diagnosis that nonetheless demands careful pathophysiological understanding, structured clinical assessment, and thoughtful investigation before a confident diagnosis and appropriate treatment can be reached. This opening Series of **Gastroenterology II** works through this single condition in real depth, building a genuinely thorough foundation before the course moves on to other hepatobiliary and gastrointestinal topics.



The aim of this Series is to equip you with an understanding of the pathophysiology and risk factors of peptic ulcer disease, including the role of *Helicobacter pylori* and non-steroidal anti-inflammatory drugs, the clinical presentation of gastric and duodenal ulcer and how to differentiate this from other causes of dyspepsia, the investigation of peptic ulcer disease including endoscopy and testing for malignancy, and its medical management, eradication therapy, and complications.

This Series opens with **pathophysiology and risk factors**, moves through **clinical presentation** and **investigation**, and closes with **management and complications**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 1.1:** describe the pathophysiology of peptic ulcer disease
- **SLO 1.2:** describe the role of *Helicobacter pylori* and non-steroidal anti-inflammatory drugs in peptic ulcer disease
- **SLO 1.3:** describe the clinical presentation of gastric and duodenal ulcer
- **SLO 1.4:** discuss differentiating peptic ulcer disease from other causes of dyspepsia
- **SLO 1.5:** describe the investigation of peptic ulcer disease, including endoscopy and *Helicobacter pylori* testing
- **SLO 1.6:** discuss investigating for malignancy in peptic ulcer disease
- **SLO 1.7:** describe the medical management and *Helicobacter pylori* eradication therapy of peptic ulcer disease
- **SLO 1.8:** describe the complications of peptic ulcer disease

1.1 Pathophysiology and Risk Factors of Peptic Ulcer Disease

1.1.1 anatomy and physiology of gastric and duodenal mucosal defence

A protective barrier constantly balanced against acid exposure

The gastric and duodenal mucosa is normally protected from the genuinely corrosive effects of gastric acid and pepsin through a coordinated defence system, including a protective mucus and bicarbonate layer, adequate mucosal blood flow, and rapid epithelial cell turnover and repair, a genuinely finely balanced system that, under normal physiological circumstances, keeps aggressive and protective factors in equilibrium.



Peptic ulcer disease, a break in the mucosal lining extending through the muscularis mucosae, develops when this normal balance is disrupted, either through increased aggressive factors, excess acid or pepsin, or through weakened protective mechanisms, and the two most common causes of this disrupted balance, *Helicobacter pylori* infection and non-steroidal anti-inflammatory drug use, are each covered in the following two sub-Parts of this Part.

Fill in the blank: Peptic ulcer disease is a break in the mucosal lining extending through the _____ mucosae.

1.1.2 *Helicobacter pylori* and peptic ulcer disease

A genuinely common bacterial cause of a genuinely common disease

Helicobacter pylori, a spiral-shaped bacterium colonising the gastric mucosa, is the most common identifiable cause of peptic ulcer disease worldwide, producing chronic gastritis and, in a proportion of colonised individuals, progressing to genuine ulceration through a combination of direct mucosal damage and disruption of the normal protective mucus layer, alongside triggering a chronic local inflammatory response that itself contributes further to mucosal injury.

Notably, only a minority of individuals colonised with *Helicobacter pylori* actually develop clinically significant peptic ulcer disease, reflecting genuine variation in bacterial strain virulence, host genetic susceptibility, and environmental factors, and this variable disease penetrance, despite the bacterium's genuinely wide prevalence in many populations, remains an area of ongoing research interest within the broader field of gastroenterology.

Fill in the blank: Only a minority of individuals colonised with *Helicobacter pylori* actually develop clinically significant peptic ulcer _____.

1.1.3 NSAID-induced and other causes of peptic ulcer disease

A genuinely important, largely preventable alternative cause

Non-steroidal anti-inflammatory drugs, the second most common identifiable cause of peptic ulcer disease, produce mucosal injury both through direct local irritation and, genuinely more importantly, through systemic inhibition of prostaglandin synthesis, disrupting the normal protective mucus and bicarbonate secretion these prostaglandins would otherwise support, a mechanism explaining why even non-oral routes of NSAID administration can still produce peptic ulceration.

Other, genuinely less common causes of peptic ulcer disease include **Zollinger-Ellison syndrome**, a gastrin-secreting tumour producing severe, refractory acid hypersecretion, and, rarely, stress-related mucosal disease in critically unwell patients, and, while these causes are



considerably less common than *Helicobacter pylori* and NSAID use, actively considering them in a patient with atypical, severe, or treatment-refractory peptic ulcer disease genuinely matters clinically.

Fill in the blank: Zollinger-Ellison syndrome is a gastrin-secreting tumour producing severe, refractory acid _____.

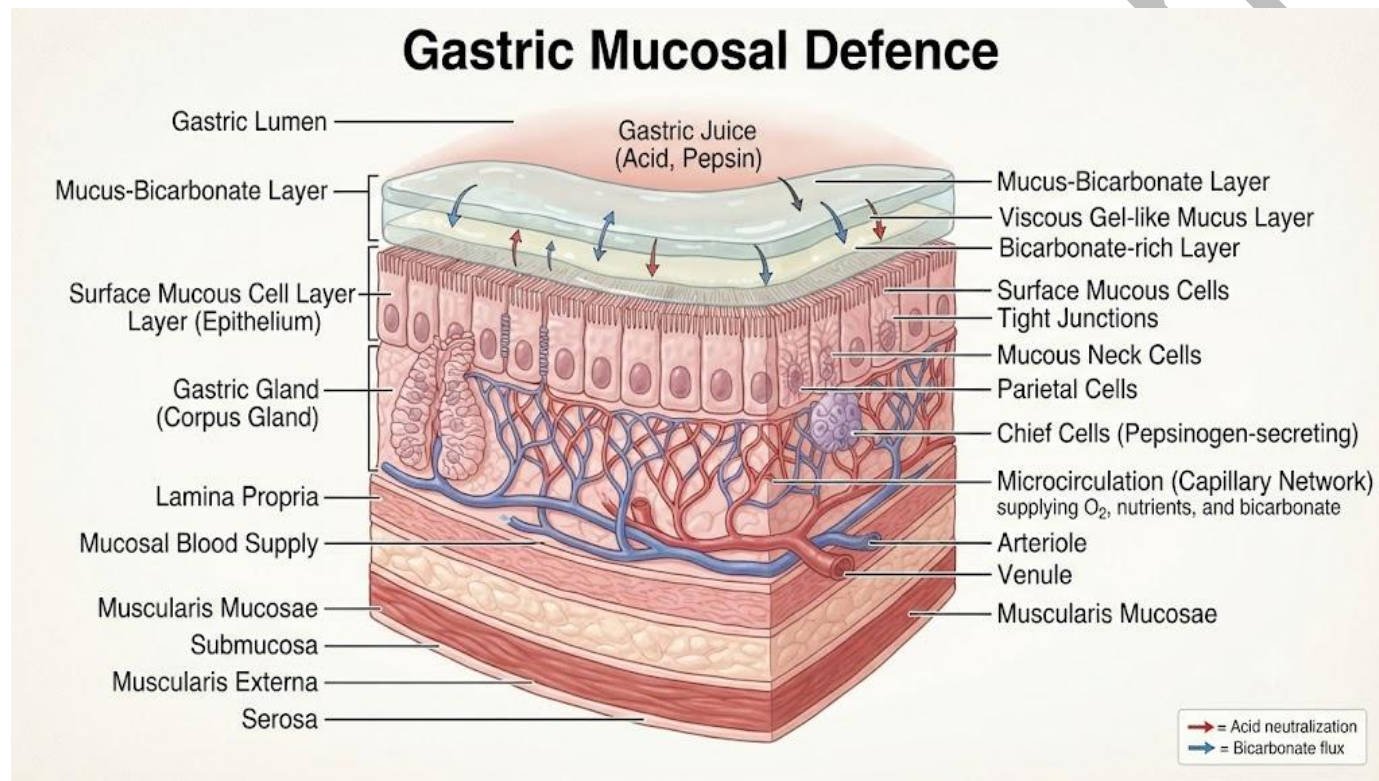


Figure 1.1: A labelled diagram of the gastric mucosal defence layer.

Pilot questions 1.1

1. Describe the normal mechanisms protecting the gastric and duodenal mucosa. (Linked to SLO 1.1)
2. Define peptic ulcer disease in anatomical terms. (Linked to SLO 1.1)
3. Describe how *Helicobacter pylori* contributes to peptic ulcer disease. (Linked to SLO 1.2)
4. Explain why only a minority of *Helicobacter pylori*-colonised individuals develop peptic ulcer disease. (Linked to SLO 1.2)
5. Describe how NSAIDs cause peptic ulcer disease. (Linked to SLO 1.2)



1.2 Clinical Presentation of Peptic Ulcer Disease

1.2.1 symptoms and signs of gastric ulcer

Pain that often worsens, rather than improves, with food

Gastric ulcer typically presents with epigastric pain that characteristically worsens with eating, reflecting the increased acid secretion food itself stimulates, and can be associated with weight loss given the discomfort eating produces and the patient's understandable tendency to reduce intake in response, a symptom pattern genuinely important to elicit carefully during history taking given its diagnostic value.

Physical examination in uncomplicated gastric ulcer is frequently unremarkable beyond mild epigastric tenderness, and the relative paucity of specific examination findings underlines why the clinical history, alongside the investigations covered later in this Series, carries the genuinely greater diagnostic weight in confidently identifying and characterising this specific condition.

Fill in the blank: Gastric ulcer pain characteristically worsens with eating, reflecting increased acid secretion food itself _____.

1.2.2 symptoms and signs of duodenal ulcer

Pain relieved, rather than worsened, by food

Duodenal ulcer, considerably more common overall than gastric ulcer, typically presents with epigastric pain that characteristically improves with eating and recurs a few hours later, often waking the patient at night, a genuinely distinctive pattern reflecting the buffering effect food has on acid reaching the duodenum, contrasting directly with the food-worsened pattern already discussed for gastric ulcer.

This specific symptomatic distinction between gastric and duodenal ulcer, though genuinely useful as a general clinical pattern, is not entirely reliable in every individual patient, meaning the distinction should inform, rather than definitively confirm, the underlying diagnosis, with endoscopic confirmation, covered in the following Part of this Series, remaining the genuinely definitive diagnostic step regardless of the specific symptomatic pattern present.

Fill in the blank: Duodenal ulcer pain characteristically improves with eating and recurs a few hours later, often waking the patient at _____.



1.2.3 differentiating peptic ulcer disease from other causes of dyspepsia

A genuinely broad differential requiring careful, structured thinking

Dyspepsia, upper abdominal discomfort or pain, has a genuinely wide differential diagnosis beyond peptic ulcer disease, including gastro-oesophageal reflux disease, functional dyspepsia without an identifiable structural cause, gallstone disease, and, genuinely importantly, gastric malignancy, a differential this Series returns to in the investigation Part that follows, given the specific importance of actively excluding malignancy in appropriate patients.

Alarm features, including unintentional weight loss, dysphagia, persistent vomiting, gastrointestinal bleeding, and an abdominal mass, warrant urgent further investigation regardless of the specific symptom pattern otherwise present, since these features raise genuine concern for a more serious underlying pathology, particularly malignancy, that must not be overlooked amid the genuinely much higher prevalence of benign peptic ulcer disease in the overall dyspeptic patient population.

Fill in the blank: Alarm features in dyspepsia include unintentional weight loss, dysphagia, and persistent _____.

Table 1.1: Comparing gastric and duodenal ulcer symptom patterns

Feature	Gastric ulcer	Duodenal ulcer
Relationship to eating	Pain worsens with food	Pain improves with food, recurs later
Typical timing	Soon after meals	A few hours after meals, often at night
Relative frequency	Less common	More common overall

Pilot questions 1.2

1. Describe the typical relationship between gastric ulcer pain and eating. (Linked to SLO 1.3)
2. Describe the typical relationship between duodenal ulcer pain and eating. (Linked to SLO 1.3)
3. Explain why the symptomatic distinction between gastric and duodenal ulcer is not entirely reliable. (Linked to SLO 1.4)
4. List conditions within the differential diagnosis of dyspepsia. (Linked to SLO 1.4)
5. List alarm features warranting urgent investigation of dyspepsia. (Linked to SLO 1.4)



1.3 Investigation of Peptic Ulcer Disease

1.3.1 upper gastrointestinal endoscopy

Direct visualisation, and the ability to biopsy where indicated

Upper gastrointestinal endoscopy, directly visualising the oesophagus, stomach, and duodenum using a flexible camera passed through the mouth, is the definitive investigation for suspected peptic ulcer disease, allowing direct identification of an ulcer, assessment of its specific characteristics, and, genuinely importantly, biopsy of the ulcer edge to exclude malignancy and biopsy of the surrounding mucosa to test for *Helicobacter pylori* infection.

Endoscopy is genuinely indicated for any patient with dyspepsia and alarm features already discussed in the previous Part, or for older patients presenting with new dyspepsia even without specific alarm features, given the genuinely increased background risk of malignancy with advancing age, while younger patients without alarm features can often be initially managed with a non-invasive *Helicobacter pylori* testing and treatment strategy without requiring immediate endoscopy.

Fill in the blank: Endoscopy allows biopsy of the ulcer edge to exclude malignancy and biopsy of surrounding mucosa to test for *Helicobacter* _____.

1.3.2 tests for *helicobacter pylori*

Invasive and non-invasive options, each with specific advantages

Non-invasive tests for *Helicobacter pylori* include the **urea breath test**, detecting bacterial urease activity through labelled carbon dioxide in exhaled breath, **stool antigen testing**, and serological antibody testing, though the latter cannot reliably distinguish current from previous infection given persisting antibodies long after successful eradication, limiting its usefulness for confirming genuine treatment success.

Invasive tests, performed on tissue obtained during endoscopy, include rapid urease testing, histological examination, and bacterial culture, and, since certain medications, particularly proton pump inhibitors and recent antibiotic use, can produce a genuinely false negative result on several of these tests, appropriate medication cessation before testing, where clinically feasible, meaningfully improves diagnostic accuracy.

Fill in the blank: Serological antibody testing cannot reliably distinguish current from previous *Helicobacter pylori* _____.



1.3.3 investigating for malignancy in peptic ulcer disease

A genuinely essential step, particularly for gastric ulcers

Gastric ulcers, unlike duodenal ulcers, carry a genuine, meaningful risk of representing an underlying malignancy rather than a purely benign process, meaning biopsy of a gastric ulcer at the time of diagnostic endoscopy is genuinely essential in every case, taken from multiple sites around the ulcer edge to maximise diagnostic sensitivity, given the genuinely serious consequences of a missed gastric malignancy.

Follow-up endoscopy, confirming complete healing of a diagnosed gastric ulcer after an appropriate course of treatment, is genuinely recommended given the small but real possibility that an initial biopsy, despite careful technique, missed a focus of malignancy within the ulcer, and a gastric ulcer failing to heal fully despite adequate treatment should prompt active, renewed suspicion for malignancy rather than simply extending the same treatment course indefinitely.

Fill in the blank: Follow-up endoscopy confirms complete _____ of a diagnosed gastric ulcer after an appropriate course of treatment.



Figure 1.2: A clinician performing upper gastrointestinal endoscopy.

Pilot questions 1.3



1. Describe the role of upper gastrointestinal endoscopy in peptic ulcer disease. (Linked to SLO 1.5)
2. List indications for endoscopy in a patient with dyspepsia. (Linked to SLO 1.5)
3. List non-invasive tests for *Helicobacter pylori*. (Linked to SLO 1.5)
4. Explain why gastric ulcers must always be biopsied. (Linked to SLO 1.6)
5. Explain why follow-up endoscopy is recommended for gastric ulcer. (Linked to SLO 1.6)

1.4 Management and Complications of Peptic Ulcer Disease

1.4.1 medical management of peptic ulcer disease

Reducing acid to allow the mucosa to heal

Proton pump inhibitors, powerfully suppressing gastric acid secretion, form the mainstay of medical management for peptic ulcer disease regardless of the specific underlying cause, allowing the mucosal defect to heal over a typically several-week treatment course, and, where NSAID use is identified as the underlying or contributing cause, discontinuing the offending medication where genuinely clinically feasible is an equally essential part of comprehensive management.

Where NSAID therapy genuinely cannot be discontinued given the patient's underlying condition requiring it, co-prescription of a proton pump inhibitor for ongoing gastroprotection, alongside using the lowest effective NSAID dose for the shortest genuinely necessary duration, meaningfully reduces the risk of further ulceration, a pragmatic, individualised approach balancing the patient's genuine ongoing need for the NSAID against the recognised gastrointestinal risk it carries.

Fill in the blank: Proton pump inhibitors form the mainstay of medical management by powerfully suppressing gastric acid _____.

1.4.2 *helicobacter pylori* eradication therapy

A combined antimicrobial and acid-suppressing regimen

***Helicobacter pylori* eradication therapy**, typically combining a proton pump inhibitor with two antibiotics for a defined treatment course, is indicated for every patient with confirmed peptic ulcer disease and confirmed *Helicobacter pylori* infection, since successful eradication considerably reduces the risk of ulcer recurrence compared with acid suppression alone, addressing the underlying infectious cause rather than simply treating its downstream mucosal consequence.



Confirming successful eradication, typically through a urea breath test or stool antigen test performed at least several weeks after completing treatment and after an appropriate interval off proton pump inhibitor therapy to avoid a false negative result, is genuinely important, and treatment failure, whether from antibiotic resistance or inadequate treatment adherence, requires a modified second-line eradication regimen rather than simply repeating the identical initial treatment course.

Fill in the blank: Confirming successful *Helicobacter pylori* eradication requires an appropriate interval off proton pump inhibitor _____.

1.4.3 complications of peptic ulcer disease

When an ulcer progresses beyond simple mucosal breakdown

Gastrointestinal bleeding, from erosion of the ulcer into an underlying blood vessel, is the most common complication of peptic ulcer disease, presenting with haematemesis or melaena, and, given its potential severity, is discussed in considerably more depth in a later Series of this course dedicated specifically to gastrointestinal bleeding as a genuinely major topic in its own right.

Perforation, the ulcer eroding completely through the full thickness of the gastric or duodenal wall, presents with sudden, severe abdominal pain and signs of peritonitis, a genuine surgical emergency, while **gastric outlet obstruction**, scarring and oedema from chronic or recurrent ulceration obstructing gastric emptying, presents more insidiously with vomiting and early satiety, and both complications, though considerably less common than bleeding, carry genuine significant morbidity requiring prompt recognition and appropriate management.

Fill in the blank: Perforation presents with sudden, severe abdominal pain and signs of _____.

Table 1.2: Comparing the complications of peptic ulcer disease

Complication	Key presentation	Relative frequency
Gastrointestinal bleeding	Haematemesis or melaena	Most common
Perforation	Sudden severe pain, peritonitis	Less common, genuine emergency
Gastric outlet obstruction	Vomiting, early satiety	Least common, insidious onset

Pilot questions 1.4

1. Describe the role of proton pump inhibitors in peptic ulcer management. (Linked to SLO 1.7)



2. Describe the general composition of *Helicobacter pylori* eradication therapy. (Linked to SLO 1.7)
3. Explain why confirming successful eradication genuinely matters. (Linked to SLO 1.7)
4. List the complications of peptic ulcer disease. (Linked to SLO 1.8)
5. Distinguish perforation from gastric outlet obstruction by presentation. (Linked to SLO 1.8)

Series Summary

This opening Series worked through peptic ulcer disease in genuine depth, from underlying pathophysiology through to complications. Part 1.1 covered mucosal defence, *Helicobacter pylori*, and NSAID-related causes, while Part 1.2 turned to the clinical presentation of gastric and duodenal ulcer and differentiating peptic ulcer disease from other causes of dyspepsia. Part 1.3 covered endoscopy, *Helicobacter pylori* testing, and investigating for malignancy, and Part 1.4 closed with medical management, eradication therapy, and complications.

You should now be able to describe the pathophysiology, presentation, investigation, and management of peptic ulcer disease across all four Parts of this Series, meeting the outcomes set for this Series. Series 2 turns next to cholecystitis, pancreatitis, and diagnostic procedures in gastroenterology.

Glossary of Terms

- **Alarm features:** symptoms in dyspepsia warranting urgent investigation, including weight loss and dysphagia.
- **Duodenal ulcer:** a peptic ulcer of the duodenum, typically relieved by eating.
- **Dyspepsia:** upper abdominal discomfort or pain with a genuinely wide differential diagnosis.
- **Gastric outlet obstruction:** obstruction of gastric emptying from chronic or recurrent ulceration.
- **Gastric ulcer:** a peptic ulcer of the stomach, typically worsened by eating.
- ***Helicobacter pylori*:** a spiral-shaped bacterium and the most common identifiable cause of peptic ulcer disease.



- **Peptic ulcer disease:** a break in the gastric or duodenal mucosa extending through the muscularis mucosae.
- **Perforation:** an ulcer eroding through the full thickness of the gastric or duodenal wall.
- **Proton pump inhibitor:** medication powerfully suppressing gastric acid secretion.
- **Upper gastrointestinal endoscopy:** direct visualisation of the oesophagus, stomach, and duodenum.
- **Urea breath test:** a non-invasive test detecting *Helicobacter pylori* through bacterial urease activity.
- **Zollinger-Ellison syndrome:** a gastrin-secreting tumour producing severe, refractory acid hypersecretion.

Concept Check Questions [Series 1]

Objective questions

1. Peptic ulcer disease is a break in the mucosal lining extending through the _____. (Linked to SLO 1.1)
2. Gastric ulcer pain characteristically worsens with eating, reflecting increased acid secretion food itself _____. (Linked to SLO 1.3)
3. Endoscopy allows biopsy of the ulcer edge to exclude malignancy and biopsy of surrounding mucosa to test for *Helicobacter* _____. (Linked to SLO 1.5)
4. Confirming successful *Helicobacter pylori* eradication requires an appropriate interval off proton pump inhibitor _____. (Linked to SLO 1.7)
5. Perforation presents with sudden, severe abdominal pain and signs of _____. (Linked to SLO 1.8)

Short-answer questions

6. Explain why only a minority of *Helicobacter pylori*-colonised individuals develop peptic ulcer disease. (Linked to SLO 1.2)
7. List alarm features warranting urgent investigation of dyspepsia. (Linked to SLO 1.4)
8. List non-invasive tests for *Helicobacter pylori*. (Linked to SLO 1.5)
9. Explain why gastric ulcers must always be biopsied. (Linked to SLO 1.6)
10. List the complications of peptic ulcer disease. (Linked to SLO 1.8)

Long-answer questions

11. Discuss the pathophysiology and risk factors of peptic ulcer disease, including *Helicobacter pylori* and NSAIDs. (Linked to SLO 1.1, 1.2)



12. Describe the clinical presentation of gastric and duodenal ulcer and how peptic ulcer disease is differentiated from other causes of dyspepsia. (Linked to SLO 1.3, 1.4)
13. Discuss the investigation of peptic ulcer disease, including endoscopy, *Helicobacter pylori* testing, and investigating for malignancy. (Linked to SLO 1.5, 1.6)
14. Describe the medical management and eradication therapy of peptic ulcer disease. (Linked to SLO 1.7)
15. Discuss the complications of peptic ulcer disease. (Linked to SLO 1.8)

Answers to Concept Check Questions [Series 1]

Objective questions

1. Muscularis.
2. Stimulates.
3. *Pylori*.
4. Therapy.
5. Peritonitis.

Short-answer questions

6. Disease penetrance varies with bacterial strain virulence, host genetic susceptibility, and environmental factors.
7. Alarm features include weight loss, dysphagia, persistent vomiting, gastrointestinal bleeding, and an abdominal mass.
8. Non-invasive tests include urea breath testing, stool antigen testing, and serological antibody testing.
9. Gastric ulcers carry a genuine risk of representing underlying malignancy, unlike duodenal ulcers.
10. Complications include gastrointestinal bleeding, perforation, and gastric outlet obstruction.

Long-answer questions

11. **Basic response:** Peptic ulcer disease develops when protective mucosal mechanisms are overwhelmed by aggressive factors; *Helicobacter pylori* causes chronic gastritis and mucosal damage, while NSAIDs inhibit protective prostaglandin synthesis.



Value-added response: A value-added response notes that Zollinger-Ellison syndrome and stress-related mucosal disease are less common but important alternative causes.

12. **Basic response:** Gastric ulcer pain worsens with eating, while duodenal ulcer pain improves with eating and recurs later; differentiation from other dyspepsia causes relies on the broader differential and active screening for alarm features.

Value-added response: A value-added response notes that the symptomatic pattern is a useful general guide but not entirely reliable in every individual patient.

13. **Basic response:** Endoscopy directly visualises and allows biopsy of an ulcer; *Helicobacter pylori* testing includes invasive and non-invasive options; gastric ulcers require mandatory biopsy and follow-up endoscopy given malignancy risk.

Value-added response: A value-added response notes that proton pump inhibitors and recent antibiotics can produce false negative *Helicobacter pylori* test results.

14. **Basic response:** Proton pump inhibitors suppress acid to allow healing; eradication therapy combines a proton pump inhibitor with two antibiotics for confirmed *Helicobacter pylori* infection.

Value-added response: A value-added response notes that treatment failure requires a modified second-line regimen rather than repeating the same initial course.

15. **Basic response:** Gastrointestinal bleeding is the most common complication; perforation is a genuine surgical emergency; gastric outlet obstruction presents insidiously with vomiting and early satiety.

Value-added response: A value-added response notes that gastrointestinal bleeding is covered in considerably more depth in a later Series of this course.

Answers to Pilot Questions [Series 1]

Part 1.1

1. The mucosa is protected by a mucus and bicarbonate layer, adequate blood flow, and rapid epithelial repair.
2. Peptic ulcer disease is a break extending through the muscularis mucosae.



3. *Helicobacter pylori* produces chronic gastritis, direct mucosal damage, and disruption of the protective mucus layer.
4. Disease development depends on bacterial strain virulence, host susceptibility, and environmental factors.
5. NSAIDs inhibit prostaglandin synthesis, disrupting normal protective mucus and bicarbonate secretion.

Part 1.2

1. Gastric ulcer pain typically worsens soon after eating.
2. Duodenal ulcer pain typically improves with eating and recurs a few hours later, often at night.
3. The pattern is a useful general guide but not entirely reliable in every individual patient.
4. The differential includes gastro-oesophageal reflux disease, functional dyspepsia, gallstone disease, and gastric malignancy.
5. Alarm features include weight loss, dysphagia, persistent vomiting, bleeding, and an abdominal mass.

Part 1.3

1. Endoscopy directly visualises an ulcer and allows biopsy for malignancy and *Helicobacter pylori* testing.
2. Indications include alarm features and new dyspepsia in older patients.
3. Non-invasive tests include urea breath testing, stool antigen testing, and serological antibody testing.
4. Gastric ulcers carry a genuine risk of malignancy, unlike duodenal ulcers.
5. Follow-up confirms healing and excludes a missed focus of malignancy within the ulcer.

Part 1.4

1. Proton pump inhibitors suppress acid secretion, allowing the mucosal defect to heal.
2. Eradication therapy combines a proton pump inhibitor with two antibiotics for a defined course.
3. Confirming eradication ensures the underlying infectious cause has genuinely been addressed, reducing recurrence risk.
4. Complications include gastrointestinal bleeding, perforation, and gastric outlet obstruction.
5. Perforation presents with sudden severe pain and peritonitis, while obstruction presents insidiously with vomiting.



References and Attributions [Series 1]

- Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
- Feldman, M., Friedman, L. S., & Brandt, L. J. (2020). Sleisenger and Fordtran's Gastrointestinal and Liver Disease (11th ed.). Elsevier.
- National Institute for Health and Care Excellence (2019). Dyspepsia and Gastro-Oesophageal Reflux Disease (CG184). NICE.
- Malfertheiner, P., Megraud, F., O'Morain, C. A., et al. (2022). Management of Helicobacter Pylori Infection: The Maastricht VI/Florence Consensus Report. Gut.

Figures, Plates, Tables, and Boxes

- Figure 1.1: A labelled diagram of the gastric mucosal defence layer. AI-generated using the prompt: "Create a clean, accurate labelled anatomical diagram titled Gastric Mucosal Defence, showing a cross-section of the stomach wall with the mucus and bicarbonate layer, surface epithelium, and underlying mucosal blood supply clearly labelled. Educational anatomical diagram style, no photographic content."
- Table 1.1: Comparing gastric and duodenal ulcer symptom patterns. AI-generated using the prompt: "Create a clean reference table titled Comparing Gastric and Duodenal Ulcer Symptom Patterns, listing feature, gastric ulcer, and duodenal ulcer. Simple clinical reference table style with outside border."
- Figure 1.2: A clinician performing upper gastrointestinal endoscopy. AI-generated using the prompt: "Create a realistic clinical illustration titled Upper Gastrointestinal Endoscopy, showing a clinician guiding a flexible endoscope into a sedated patient's mouth while viewing the procedure on a monitor, endoscopy suite setting. Clean clinical illustration style, no text."
- Table 1.2: Comparing the complications of peptic ulcer disease. AI-generated using the prompt: "Create a clean reference table titled Comparing the Complications of Peptic Ulcer Disease, listing complication, key presentation, and relative frequency. Simple clinical reference table style with outside border."



Course code: MED 608

Course title: Gastroenterology II

Course credit load: 2 Units

Series 2: Cholecystitis, Pancreatitis, and Diagnostic Procedures in Gastroenterology

- **Part 2.1: Cholelithiasis and Cholecystitis**
 - Sub Part 2.1.1: Pathophysiology of gallstone formation
 - Sub Part 2.1.2: Clinical presentation of biliary colic and acute cholecystitis
 - Sub Part 2.1.3: Management of cholecystitis
- **Part 2.2: Acute and Chronic Pancreatitis**
 - Sub Part 2.2.1: Aetiology and pathophysiology of acute pancreatitis
 - Sub Part 2.2.2: Clinical presentation and severity assessment of acute pancreatitis
 - Sub Part 2.2.3: Chronic pancreatitis
- **Part 2.3: Laboratory and Imaging Diagnostic Procedures**
 - Sub Part 2.3.1: Laboratory tests in hepatobiliary and pancreatic disease
 - Sub Part 2.3.2: Abdominal ultrasound and cholecystogram
 - Sub Part 2.3.3: Cross-sectional imaging in hepatobiliary disease
- **Part 2.4: Endoscopic Retrograde Cholangiopancreatography**
 - Sub Part 2.4.1: Principles and indications for ERCP
 - Sub Part 2.4.2: The ERCP procedure
 - Sub Part 2.4.3: Complications of ERCP



Introduction

This Series turns to the gallbladder and pancreas, two closely related organs sharing an intimate anatomical and functional relationship with the biliary tree, before closing with the diagnostic procedures, laboratory, imaging, and endoscopic, that together confirm and characterise disease across this whole hepatobiliary and pancreatic system. **Cholecystitis and pancreatitis** are both genuinely common causes of acute abdominal presentation, and confidently distinguishing between them, and from each other's complications, is a core clinical skill this Series aims to build.

The aim of this Series is to equip you with an understanding of gallstone formation and cholecystitis and its management, the aetiology, presentation, and severity assessment of acute pancreatitis and the presentation of chronic pancreatitis, the laboratory and imaging investigations used in hepatobiliary and pancreatic disease, including ultrasound and cholecystogram, and the principles, indications, procedure, and complications of endoscopic retrograde cholangiopancreatography.

This Series opens with **cholelithiasis and cholecystitis**, moves through **acute and chronic pancreatitis** and **laboratory and imaging diagnostic procedures**, and closes with **endoscopic retrograde cholangiopancreatography**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 2.1:** describe the pathophysiology of gallstone formation and cholecystitis
- **SLO 2.2:** discuss the management of cholecystitis
- **SLO 2.3:** describe the aetiology, presentation, and severity assessment of acute pancreatitis
- **SLO 2.4:** describe chronic pancreatitis
- **SLO 2.5:** describe laboratory tests used in hepatobiliary and pancreatic disease
- **SLO 2.6:** describe imaging investigations including ultrasound, cholecystogram, and cross-sectional imaging
- **SLO 2.7:** describe the principles, indications, and procedure of endoscopic retrograde cholangiopancreatography
- **SLO 2.8:** discuss the complications of endoscopic retrograde cholangiopancreatography



2.1 Cholelithiasis and Cholecystitis

2.1.1 pathophysiology of gallstone formation

Bile chemistry falling out of solution

Gallstones form when the normal balance of cholesterol, bile salts, and phospholipids within bile is disrupted, most commonly through cholesterol supersaturation exceeding the bile's capacity to keep it dissolved in solution, producing **cholesterol gallstones**, the most common type overall, while **pigment gallstones**, formed from excess bilirubin, are less common and typically associated with conditions causing increased red cell breakdown or biliary infection.

Risk factors for cholesterol gallstone formation include obesity, female sex, pregnancy, rapid weight loss, and certain medications, and, while gallstones are genuinely common in the general population, the majority remain entirely asymptomatic throughout life, identified only incidentally, meaning the presence of gallstones alone does not automatically indicate a need for treatment in an otherwise asymptomatic individual.

Fill in the blank: Cholesterol gallstones form when cholesterol supersaturation exceeds bile's capacity to keep it dissolved in _____.

2.1.2 clinical presentation of biliary colic and acute cholecystitis

From transient obstruction to genuine inflammation

Biliary colic, right upper quadrant or epigastric pain resulting from transient obstruction of the cystic duct by a gallstone, typically presents with pain lasting minutes to a few hours, often triggered by a fatty meal, and resolving spontaneously as the obstructing stone passes or the gallbladder relaxes, without the associated fever or significant local inflammation characteristic of the more serious condition discussed next.

Acute cholecystitis, genuine inflammation of the gallbladder wall following persistent cystic duct obstruction, presents with more prolonged, severe right upper quadrant pain, fever, and **Murphy's sign**, pain and inspiratory arrest on palpation of the right upper quadrant during deep inspiration, a specific examination finding reflecting the inflamed gallbladder descending onto the examining hand, distinguishing this genuinely more significant condition from simple biliary colic.

Fill in the blank: Murphy's sign is pain and inspiratory arrest on palpation of the right upper quadrant during deep _____.



2.1.3 management of cholecystitis

Supportive treatment, then definitive surgical removal

Initial management of acute cholecystitis includes intravenous fluids, analgesia, and antibiotics given the genuine risk of secondary bacterial infection, with **cholecystectomy**, surgical removal of the gallbladder, remaining the definitive treatment, and early cholecystectomy, performed within days of presentation rather than delayed for weeks to allow inflammation to settle, is now generally preferred given evidence of improved outcomes and shorter overall hospital stay with this earlier approach.

Laparoscopic cholecystectomy, performed through several small incisions using a camera and specialised instruments, is the standard surgical approach given its reduced postoperative pain and faster recovery compared with open surgery, though conversion to open surgery remains occasionally necessary if the anatomy proves genuinely difficult or unclear during the laparoscopic procedure, a pragmatic safety consideration the surgical team must always keep available.

Fill in the blank: Early cholecystectomy is now generally preferred given evidence of improved outcomes and shorter overall hospital _____.



Figure 2.1: A clinician eliciting Murphy's sign by palpating the right upper quadrant during inspiration.



Pilot questions 2.1

- Describe the pathophysiology of cholesterol gallstone formation. (Linked to SLO 2.1)
- List risk factors for gallstone formation. (Linked to SLO 2.1)
- Distinguish biliary colic from acute cholecystitis. (Linked to SLO 2.1)
- Describe Murphy's sign. (Linked to SLO 2.1)
- Describe the management of acute cholecystitis. (Linked to SLO 2.2)

2.2 Acute and Chronic Pancreatitis

2.2.1 aetiology and pathophysiology of acute pancreatitis

Premature activation of digestive enzymes within the pancreas itself

Acute pancreatitis, acute inflammation of the pancreas, most commonly results from gallstones obstructing the pancreatic duct or excessive alcohol consumption, together accounting for the great majority of cases, with less common causes including certain medications, hypertriglyceridaemia, and, following endoscopic retrograde cholangiopancreatography, discussed in detail later in this Series given its recognised association with this specific complication.

The underlying pathophysiology involves premature intrapancreatic activation of digestive enzymes, normally activated only once safely within the duodenum, leading to autodigestion of pancreatic tissue and a resulting local and, in more severe cases, systemic inflammatory response, a genuinely destructive process directly underlying both the local pancreatic damage and the systemic complications severe acute pancreatitis can produce.

Fill in the blank: Acute pancreatitis involves premature intrapancreatic activation of digestive _____, normally activated only in the duodenum.

2.2.2 clinical presentation and severity assessment of acute pancreatitis

Severe epigastric pain, and a genuine spectrum of severity

Acute pancreatitis presents with severe, constant epigastric pain, often radiating through to the back, accompanied by nausea and vomiting, and diagnosis is confirmed by a significantly



elevated serum lipase or amylase, typically at least three times the upper limit of normal, alongside the characteristic clinical presentation, since a mildly elevated level alone is not specific enough to reliably confirm the diagnosis.

Severity assessment, using structured scoring systems combining clinical, laboratory, and, where indicated, imaging findings, distinguishes mild, self-limiting disease from severe pancreatitis genuinely at risk of significant complications including pancreatic necrosis and multi-organ failure, and this structured severity assessment, performed within the first day or two of admission, directly guides the intensity of monitoring and treatment required for each individual patient.

Fill in the blank: Structured severity assessment distinguishes mild, self-limiting disease from severe pancreatitis genuinely at risk of pancreatic _____.

2.2.3 chronic pancreatitis

Progressive, irreversible pancreatic damage over time

Chronic pancreatitis, progressive, irreversible inflammatory damage to the pancreas, most commonly resulting from longstanding alcohol excess, presents with chronic or recurrent abdominal pain alongside features of progressive **exocrine insufficiency**, malabsorption and steatorrhea from inadequate pancreatic enzyme production, and **endocrine insufficiency**, diabetes mellitus from progressive loss of the pancreas's insulin-producing islet cells.

Management addresses pain control, often genuinely challenging given the chronic, sometimes severe nature of the pain this condition produces, alongside pancreatic enzyme replacement therapy for exocrine insufficiency and standard diabetes management for endocrine insufficiency where present, and abstinence from alcohol, where this is the identified underlying cause, remains a genuinely essential component of comprehensive management to slow further disease progression.

Fill in the blank: Chronic pancreatitis produces progressive endocrine insufficiency, presenting as diabetes mellitus from loss of insulin-producing islet _____.

Table 2.1: Comparing acute and chronic pancreatitis

Feature	Acute pancreatitis	Chronic pancreatitis
Onset	Sudden	Progressive over years
Reversibility	Potentially reversible	Irreversible damage



Key complication	Necrosis, organ failure	Exocrine and endocrine insufficiency
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Pilot questions 2.2

1. List the two most common causes of acute pancreatitis. (Linked to SLO 2.3)
2. Describe the underlying pathophysiology of acute pancreatitis. (Linked to SLO 2.3)
3. Describe the diagnostic criteria for acute pancreatitis. (Linked to SLO 2.3)
4. Explain the purpose of structured severity assessment in acute pancreatitis. (Linked to SLO 2.3)
5. Describe chronic pancreatitis and its exocrine and endocrine consequences. (Linked to SLO 2.4)

2.3 Laboratory and Imaging Diagnostic Procedures

2.3.1 laboratory tests in hepatobiliary and pancreatic disease

Interpreting the biochemical pattern

Liver function tests, including bilirubin, alanine transaminase, aspartate transaminase, alkaline phosphatase, and gamma-glutamyl transferase, help distinguish a **hepatocellular pattern** of injury, predominantly elevated transaminases, from a **cholestatic pattern**, predominantly elevated alkaline phosphatase and gamma-glutamyl transferase, a genuinely important initial distinction guiding subsequent, more targeted investigation.

Serum amylase and lipase, already discussed in the context of acute pancreatitis diagnosis earlier in this Series, alongside specific tumour markers where malignancy is genuinely suspected, complete the core laboratory panel relevant to hepatobiliary and pancreatic disease, and interpreting these tests together, rather than any single result in isolation, allows a considerably more confident, nuanced clinical picture to be reached.

Fill in the blank: A cholestatic pattern shows predominantly elevated alkaline phosphatase and gamma-glutamyl _____.



2.3.2 abdominal ultrasound and cholecystogram

The first-line, non-invasive imaging investigations

Abdominal ultrasound remains the first-line imaging investigation for suspected gallstone disease, providing rapid, non-invasive, radiation-free visualisation of gallstones, gallbladder wall thickening, and biliary duct dilatation, findings that together support a diagnosis of cholecystitis or biliary obstruction, and its wide availability and low cost make it a genuinely practical initial investigation across most clinical settings.

Oral cholecystogram, an older imaging technique using an orally administered contrast agent excreted into bile to visualise the gallbladder on subsequent radiography, has been largely superseded by ultrasound given ultrasound's superior sensitivity, speed, and avoidance of both oral contrast and radiation exposure, though understanding this historical technique remains genuinely relevant to appreciating how hepatobiliary imaging has evolved over time.

Fill in the blank: Oral cholecystogram has been largely superseded by ultrasound given its superior sensitivity, speed, and avoidance of radiation _____.

2.3.3 cross-sectional imaging in hepatobiliary disease

Detailed anatomical characterisation where ultrasound alone is insufficient

Computed tomography, providing detailed cross-sectional anatomical information, is particularly valuable in acute pancreatitis for assessing pancreatic necrosis and local complications, and in staging suspected hepatobiliary or pancreatic malignancy, offering considerably more comprehensive anatomical detail than ultrasound alone, particularly for assessing deeper structures or planning surgical intervention.

Magnetic resonance cholangiopancreatography, a non-invasive magnetic resonance imaging technique specifically visualising the biliary and pancreatic ductal system, offers detailed ductal anatomy without the risks associated with the invasive endoscopic retrograde cholangiopancreatography procedure discussed in the following Part, making it a genuinely valuable diagnostic step before committing a patient to an invasive procedure where purely diagnostic information is required.

Fill in the blank: Magnetic resonance cholangiopancreatography visualises the biliary and pancreatic _____ system non-invasively.





Figure 2.2: A sonographer performing abdominal ultrasound over the right upper quadrant.

Pilot questions 2.3

1. Distinguish a hepatocellular from a cholestatic pattern of liver function test abnormality. (Linked to SLO 2.5)
2. Describe the role of abdominal ultrasound in suspected gallstone disease. (Linked to SLO 2.6)
3. Explain why oral cholecystogram has been largely superseded. (Linked to SLO 2.6)
4. Describe the role of computed tomography in acute pancreatitis. (Linked to SLO 2.6)
5. Describe magnetic resonance cholangiopancreatography and its advantage over ERCP for purely diagnostic purposes. (Linked to SLO 2.6)

2.4 Endoscopic Retrograde Cholangiopancreatography

2.4.1 principles and indications for ercp



Combining endoscopy with direct ductal access

Endoscopic retrograde cholangiopancreatography, commonly abbreviated as ERCP, combines upper gastrointestinal endoscopy with fluoroscopic imaging to directly access and visualise the biliary and pancreatic ducts, and, unlike the purely diagnostic magnetic resonance cholangiopancreatography already discussed in the previous Part, ERCP offers the genuinely important additional capability of therapeutic intervention during the same procedure.

Indications for ERCP include confirmed or strongly suspected **choledocholithiasis**, a bile duct stone requiring extraction, malignant or benign biliary strictures requiring stenting, and, in selected cases of pancreatitis related to pancreatic duct obstruction, therapeutic intervention on the pancreatic duct itself, with ERCP genuinely reserved for situations where therapeutic intervention is anticipated rather than performed for purely diagnostic purposes given the availability of the less invasive magnetic resonance alternative.

Fill in the blank: ERCP is genuinely reserved for situations where therapeutic _____ is anticipated rather than performed for purely diagnostic purposes.

2.4.2 the ercp procedure

Cannulating the duct and performing the indicated intervention

The ERCP procedure involves passing a side-viewing endoscope to the duodenum, identifying the major duodenal papilla, and cannulating the bile duct, or, where indicated, the pancreatic duct, before injecting contrast under fluoroscopic guidance to visualise the ductal anatomy and identify any stone, stricture, or other abnormality requiring the specific therapeutic intervention the procedure has been performed to address.

Common therapeutic interventions performed during ERCP include **sphincterotomy**, a small incision widening the opening of the bile duct to facilitate stone extraction, direct **stone extraction**, and **stent insertion** across a stricture to restore adequate biliary drainage, and the specific combination of interventions performed depends genuinely on the particular pathology identified during the procedure itself.

Fill in the blank: Sphincterotomy is a small incision widening the opening of the bile duct to facilitate stone _____.



2.4.3 complications of ercp

A genuinely more invasive procedure with its own specific risks

Post-ERCP pancreatitis, the most common significant complication, results from mechanical or chemical irritation of the pancreatic duct during the procedure, and, while most cases are mild and self-limiting, a proportion of patients develop genuinely severe pancreatitis, making careful patient selection and appropriate preventive measures, including specific medication administered around the time of the procedure in selected patients, genuinely important.

Other recognised complications include bleeding, particularly following sphincterotomy, **perforation** of the duodenum or bile duct, and **cholangitis**, infection of the biliary tree, and, given this genuine risk profile compared with the less invasive alternative imaging already discussed earlier in this Series, ERCP should genuinely be reserved for situations where the anticipated therapeutic benefit clearly outweighs these specific procedural risks, closing this Series on the same theme of individualised, risk-benefit clinical decision-making that has run throughout this whole programme.

Fill in the blank: Cholangitis is infection of the biliary _____, a recognised complication of ERCP.

Table 2.2: Comparing complications of ERCP

Complication	Mechanism	Relative frequency
Post-ERCP pancreatitis	Pancreatic duct irritation	Most common
Bleeding	Usually following sphincterotomy	Less common
Perforation	Duodenal or bile duct injury	Uncommon, but serious

Pilot questions 2.4

1. Distinguish ERCP from magnetic resonance cholangiopancreatography. (Linked to SLO 2.7)
2. List the indications for ERCP. (Linked to SLO 2.7)
3. Describe sphincterotomy and its purpose. (Linked to SLO 2.7)
4. Describe post-ERCP pancreatitis. (Linked to SLO 2.8)
5. List other recognised complications of ERCP beyond pancreatitis. (Linked to SLO 2.8)



Series Summary

This Series covered cholecystitis, pancreatitis, and the diagnostic procedures used across hepatobiliary and pancreatic disease. Part 2.1 covered gallstone formation and the presentation and management of cholecystitis, while Part 2.2 turned to acute and chronic pancreatitis. Part 2.3 covered laboratory and imaging investigations, and Part 2.4 closed with the principles, procedure, and complications of endoscopic retrograde cholangiopancreatography.

You should now be able to describe and manage cholecystitis and pancreatitis and describe the relevant diagnostic procedures covered across all four Parts of this Series, meeting the outcomes set for this Series. Series 3 turns next to chronic liver disease and inflammatory bowel disease.

Glossary of Terms

1. **Acute cholecystitis:** genuine inflammation of the gallbladder wall from persistent cystic duct obstruction.
2. **Biliary colic:** right upper quadrant pain from transient cystic duct obstruction by a gallstone.
3. **Cholangitis:** infection of the biliary tree.
4. **Cholecystectomy:** surgical removal of the gallbladder.
5. **Choledocholithiasis:** a stone within the common bile duct.
6. **Chronic pancreatitis:** progressive, irreversible inflammatory damage to the pancreas.
7. **Endoscopic retrograde cholangiopancreatography:** an endoscopic procedure combining imaging and therapeutic ductal access.
8. **Exocrine insufficiency:** inadequate pancreatic enzyme production causing malabsorption.
9. **Magnetic resonance cholangiopancreatography:** a non-invasive MRI technique visualising the biliary and pancreatic ducts.
10. **Murphy's sign:** pain and inspiratory arrest on right upper quadrant palpation, suggesting cholecystitis.
11. **Post-ERCP pancreatitis:** the most common significant complication of ERCP.
12. **Sphincterotomy:** a small incision widening the bile duct opening during ERCP.



Concept Check Questions [Series 2]

Objective questions

- Cholesterol gallstones form when cholesterol supersaturation exceeds bile's capacity to keep it dissolved in _____. (Linked to SLO 2.1)
- Acute pancreatitis involves premature intrapancreatic activation of digestive _____, normally activated only in the duodenum. (Linked to SLO 2.3)
- A cholestatic pattern shows predominantly elevated alkaline phosphatase and gamma-glutamyl _____. (Linked to SLO 2.5)
- Magnetic resonance cholangiopancreatography visualises the biliary and pancreatic _____ system non-invasively. (Linked to SLO 2.6)
- Sphincterotomy is a small incision widening the opening of the bile duct to facilitate stone _____. (Linked to SLO 2.7)

Short-answer questions

1. Distinguish biliary colic from acute cholecystitis. (Linked to SLO 2.1)
2. Describe the diagnostic criteria for acute pancreatitis. (Linked to SLO 2.3)
3. Describe chronic pancreatitis and its exocrine and endocrine consequences. (Linked to SLO 2.4)
4. Explain why oral cholecystogram has been largely superseded. (Linked to SLO 2.6)
5. Describe post-ERCP pancreatitis. (Linked to SLO 2.8)

Long-answer questions

6. Discuss the pathophysiology, presentation, and management of gallstone disease and cholecystitis. (Linked to SLO 2.1, 2.2)
7. Describe the aetiology, presentation, severity assessment, and chronic sequelae of pancreatitis. (Linked to SLO 2.3, 2.4)
8. Discuss the laboratory and imaging investigations used in hepatobiliary and pancreatic disease. (Linked to SLO 2.5, 2.6)
9. Describe the principles, indications, and procedure of ERCP. (Linked to SLO 2.7)
10. Discuss the complications of ERCP. (Linked to SLO 2.8)

Answers to Concept Check Questions [Series 2]

Objective questions

11. Solution.



12. Enzymes.
13. Transferase.
14. Ductal.
15. Extraction.

Short-answer questions

16. Biliary colic is transient obstruction without fever, while acute cholecystitis is genuine inflammation with fever and Murphy's sign.
17. Diagnosis requires characteristic pain alongside serum lipase or amylase at least three times the upper limit of normal.
18. Chronic pancreatitis causes progressive exocrine insufficiency, malabsorption, and endocrine insufficiency, diabetes.
19. Ultrasound offers superior sensitivity, speed, and avoids oral contrast and radiation exposure.
20. Post-ERCP pancreatitis results from mechanical or chemical irritation of the pancreatic duct during the procedure.

Long-answer questions

21. **Basic response:** Gallstones form from cholesterol supersaturation or excess bilirubin; biliary colic reflects transient obstruction, while cholecystitis reflects genuine inflammation with Murphy's sign; management combines supportive treatment with early cholecystectomy.

Value-added response: A value-added response notes that most gallstones remain asymptomatic and do not automatically require treatment.

22. **Basic response:** Acute pancreatitis most commonly results from gallstones or alcohol, presenting with severe epigastric pain and elevated lipase, assessed for severity using structured scoring; chronic pancreatitis causes irreversible damage with exocrine and endocrine insufficiency.

Value-added response: A value-added response notes that severity assessment directly guides the intensity of monitoring and treatment required.

23. **Basic response:** Liver function tests distinguish hepatocellular from cholestatic patterns; ultrasound is first-line imaging for gallstone disease; cross-sectional imaging and magnetic resonance cholangiopancreatography provide further detail.

Value-added response: A value-added response notes that oral cholecystogram has been largely superseded by ultrasound given its practical advantages.



24. **Basic response:** ERCP combines endoscopy and fluoroscopy to access the biliary and pancreatic ducts, indicated for confirmed stones or strictures requiring therapeutic intervention; the procedure involves cannulation, contrast injection, and interventions such as sphincterotomy or stenting.

Value-added response: A value-added response notes that ERCP is reserved for anticipated therapeutic intervention rather than purely diagnostic purposes.

25. **Basic response:** Complications include post-ERCP pancreatitis, the most common, alongside bleeding, perforation, and cholangitis.

Value-added response: A value-added response notes that ERCP should be reserved for situations where therapeutic benefit clearly outweighs these procedural risks.

Answers to Pilot Questions [Series 2]

Part 2.1

1. Cholesterol gallstones form when cholesterol supersaturation exceeds bile's capacity to keep it dissolved.
2. Risk factors include obesity, female sex, pregnancy, and rapid weight loss.
3. Biliary colic is transient without fever, while cholecystitis is persistent inflammation with fever.
4. Murphy's sign is pain and inspiratory arrest on right upper quadrant palpation during deep breathing.
5. Management includes fluids, analgesia, antibiotics, and early cholecystectomy.

Part 2.2

6. The two most common causes are gallstones and excessive alcohol consumption.
7. Premature intrapancreatic enzyme activation causes autodigestion of pancreatic tissue.
8. Diagnosis requires characteristic pain alongside lipase or amylase at least three times the upper limit of normal.
9. Severity assessment identifies patients at risk of complications, guiding monitoring and treatment intensity.
10. Chronic pancreatitis causes progressive exocrine insufficiency, malabsorption, and endocrine insufficiency, diabetes.



Part 2.3

11. Hepatocellular patterns show predominantly elevated transaminases, while cholestatic patterns show elevated alkaline phosphatase and gamma-glutamyl transferase.
12. Ultrasound provides rapid, non-invasive visualisation of gallstones and biliary duct changes.
13. Ultrasound offers superior sensitivity, speed, and avoids oral contrast and radiation exposure.
14. Computed tomography assesses pancreatic necrosis and local complications in acute pancreatitis.
15. Magnetic resonance cholangiopancreatography avoids the risks of invasive ERCP for purely diagnostic purposes.

Part 2.4

1. ERCP is invasive and therapeutic, while MRCP is non-invasive and purely diagnostic.
2. Indications include confirmed bile duct stones, biliary strictures, and pancreatic duct intervention.
3. Sphincterotomy widens the bile duct opening to facilitate stone extraction.
4. Post-ERCP pancreatitis results from mechanical or chemical irritation of the pancreatic duct.
5. Other complications include bleeding, perforation, and cholangitis.

References and Attributions [Series 2]

1. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
2. Feldman, M., Friedman, L. S., & Brandt, L. J. (2020). Sleisenger and Fordtran's Gastrointestinal and Liver Disease (11th ed.). Elsevier.
3. National Institute for Health and Care Excellence (2014). Gallstone Disease: Diagnosis and Management (CG188). NICE.
4. International Association of Pancreatology (2013). IAP/APA Evidence-Based Guidelines for the Management of Acute Pancreatitis. Pancreatology.



Figures, Plates, Tables, and Boxes

1. Figure 2.1: A clinician eliciting Murphy's sign by palpating the right upper quadrant during inspiration. AI-generated using the prompt: "Create a realistic clinical illustration titled Eliciting Murphy's Sign, showing a clinician pressing gently on a patient's right upper abdomen while the patient takes a deep breath, examination room setting. Clean clinical illustration style, no text."
2. Table 2.1: Comparing acute and chronic pancreatitis. AI-generated using the prompt: "Create a clean reference table titled Comparing Acute and Chronic Pancreatitis, listing feature, acute pancreatitis, and chronic pancreatitis. Simple clinical reference table style with outside border."
3. Figure 2.2: A sonographer performing abdominal ultrasound over the right upper quadrant. AI-generated using the prompt: "Create a realistic clinical illustration titled Abdominal Ultrasound Scanning, showing a sonographer moving an ultrasound probe over a patient's right upper abdomen while viewing the image on a nearby screen, examination room setting. Clean clinical illustration style, no text."
4. Table 2.2: Comparing complications of ERCP. AI-generated using the prompt: "Create a clean reference table titled Comparing Complications of ERCP, listing complication, mechanism, and relative frequency. Simple clinical reference table style with outside border."



Course code: MED 608

Course title: Gastroenterology II

Course credit load: 2 Units

Series 3: Chronic Liver Disease and Inflammatory Bowel Disease

- **Part 3.1: Chronic Hepatitis**
 - Sub Part 3.1.1: Causes and classification of chronic hepatitis
 - Sub Part 3.1.2: Clinical presentation of chronic active hepatitis
 - Sub Part 3.1.3: Investigation and management of chronic hepatitis
- **Part 3.2: Primary Biliary Cirrhosis**
 - Sub Part 3.2.1: Pathophysiology of primary biliary cirrhosis
 - Sub Part 3.2.2: Clinical presentation and diagnosis of primary biliary cirrhosis
 - Sub Part 3.2.3: Management of primary biliary cirrhosis
- **Part 3.3: Crohn's Disease**
 - Sub Part 3.3.1: Pathophysiology and distribution of Crohn's disease
 - Sub Part 3.3.2: Clinical presentation of Crohn's disease
 - Sub Part 3.3.3: Management of Crohn's disease
- **Part 3.4: Ulcerative Colitis**
 - Sub Part 3.4.1: Pathophysiology and distribution of ulcerative colitis
 - Sub Part 3.4.2: Clinical presentation of ulcerative colitis
 - Sub Part 3.4.3: Management and comparison with Crohn's disease

Introduction

This Series covers four genuinely distinct, chronic, sometimes lifelong gastrointestinal and hepatic conditions, **chronic hepatitis, primary biliary cirrhosis, Crohn's disease,** and



ulcerative colitis, each demanding careful, ongoing clinical management well beyond a single acute episode of care. Though genuinely different in the organ system primarily affected, these conditions share the common thread of chronic, often relapsing-remitting inflammation requiring sustained, structured long-term care.

The aim of this Series is to equip you with an understanding of the causes, presentation, investigation, and management of chronic hepatitis, the pathophysiology, presentation, and management of primary biliary cirrhosis, and the pathophysiology, distribution, presentation, and management of Crohn's disease and ulcerative colitis, alongside the ability to confidently compare these two forms of inflammatory bowel disease.

This Series opens with **chronic hepatitis**, moves through **primary biliary cirrhosis** and **Crohn's disease**, and closes with **ulcerative colitis**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 3.1:** describe the causes, classification, and clinical presentation of chronic hepatitis
- **SLO 3.2:** discuss the investigation and management of chronic hepatitis
- **SLO 3.3:** describe the pathophysiology and clinical presentation of primary biliary cirrhosis
- **SLO 3.4:** discuss the management of primary biliary cirrhosis
- **SLO 3.5:** describe the pathophysiology, distribution, and clinical presentation of Crohn's disease
- **SLO 3.6:** discuss the management of Crohn's disease
- **SLO 3.7:** describe the pathophysiology, distribution, and clinical presentation of ulcerative colitis
- **SLO 3.8:** discuss the management of ulcerative colitis and compare it with Crohn's disease

3.1 Chronic Hepatitis

3.1.1 causes and classification of chronic hepatitis



Persistent liver inflammation from several distinct causes

Chronic hepatitis, liver inflammation persisting beyond six months, results from several distinct underlying causes, including chronic viral infection, most commonly hepatitis B or hepatitis C, **autoimmune hepatitis**, immune-mediated attack against the liver's own tissue, and, less commonly, certain medications and metabolic conditions, each requiring a genuinely distinct diagnostic and treatment approach despite the shared common endpoint of chronic hepatic inflammation.

Historically termed **chronic active hepatitis** when accompanied by significant ongoing inflammatory activity and progressive fibrosis, this specific descriptive terminology has largely been superseded by classification according to the specific underlying cause and a formal histological assessment of grade, the degree of inflammatory activity, and stage, the degree of established fibrosis, providing a considerably more precise, clinically useful characterisation than the older terminology alone offered.

Fill in the blank: Chronic hepatitis is liver inflammation persisting beyond _____ months.

3.1.2 clinical presentation of chronic active hepatitis

Often silent for years before becoming clinically apparent

Chronic hepatitis is frequently entirely asymptomatic for years, identified incidentally through abnormal liver function tests performed for another reason, or through targeted screening in patients with recognised risk factors, and, when symptoms do develop, they are often genuinely non-specific, including fatigue and mild right upper quadrant discomfort, symptoms that can easily be attributed to another, unrelated cause without a genuinely high index of clinical suspicion.

As chronic hepatitis progresses towards established **cirrhosis**, more specific signs of chronic liver disease can develop, including jaundice, spider naevi, palmar erythema, and, in more advanced disease, ascites and other features of decompensated liver failure, discussed in considerably more depth in a later Series of this course dedicated specifically to hepatic failure, underlining why this Series's coverage of chronic hepatitis genuinely sits at the earlier end of a much longer disease continuum.

Fill in the blank: As chronic hepatitis progresses towards established cirrhosis, signs including jaundice, spider naevi, and palmar _____ can develop.

3.1.3 investigation and management of chronic hepatitis



Confirming the specific cause, then treating it directly

Investigation of suspected chronic hepatitis combines liver function testing with specific viral serology, autoantibody testing where autoimmune hepatitis is genuinely suspected, and, in selected cases, **liver biopsy**, providing the definitive histological grade and stage already discussed earlier in this Part, alongside imaging to assess for established cirrhosis and screen for hepatocellular carcinoma in patients with confirmed, longstanding chronic liver disease.

Management is genuinely cause-specific, with **antiviral therapy** available for chronic hepatitis B and C, offering genuine viral suppression or, for hepatitis C specifically, cure in the great majority of appropriately treated patients, and **immunosuppressive therapy**, typically corticosteroids and azathioprine, forming the mainstay of treatment for autoimmune hepatitis, illustrating how genuinely different the specific treatment approach is depending on the underlying cause identified through careful investigation.

Fill in the blank: Antiviral therapy for hepatitis C offers genuine cure in the great majority of appropriately treated _____.

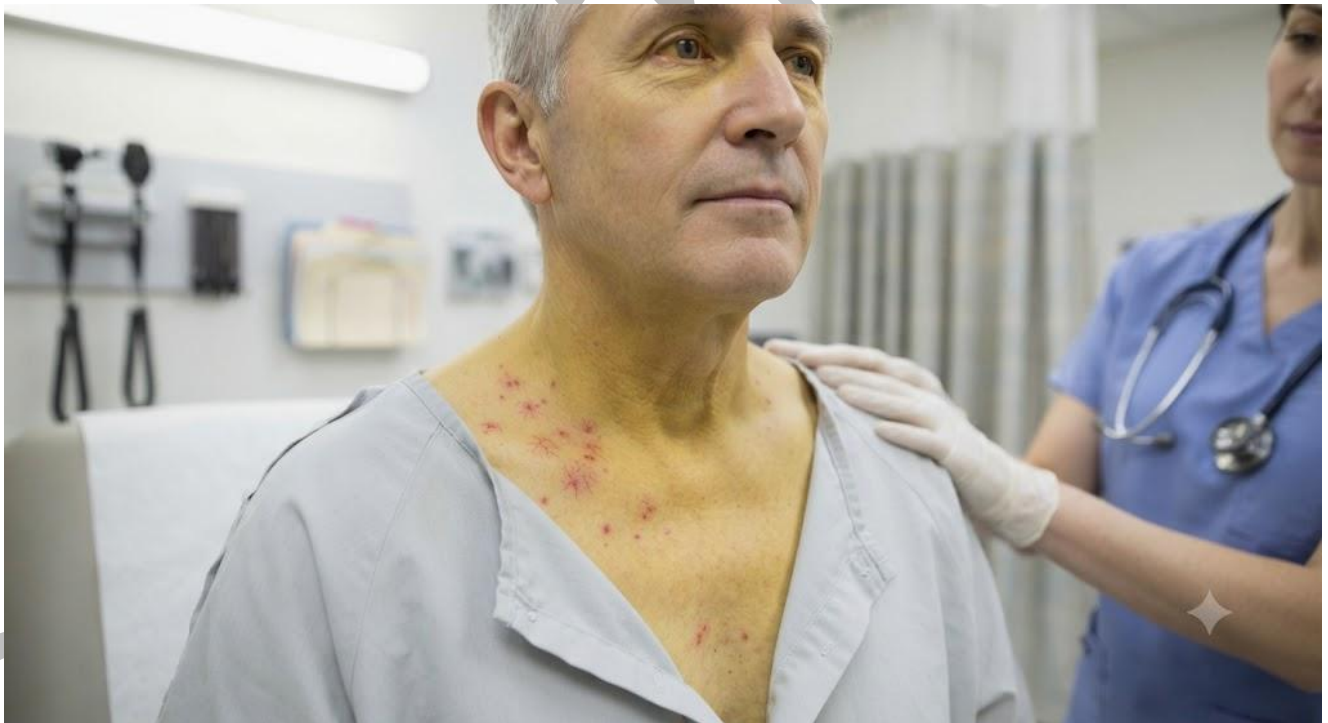


Figure 3.1: Jaundice and spider naevi as stigmata of chronic liver disease.

Pilot questions 3.1

- List the causes of chronic hepatitis. (Linked to SLO 3.1)
- Describe chronic hepatitis in terms of grade and stage. (Linked to SLO 3.1)



- Describe the typical early presentation of chronic hepatitis. (Linked to SLO 3.1)
- List signs of chronic liver disease that can develop as chronic hepatitis progresses. (Linked to SLO 3.1)
- Describe the treatment options for chronic viral and autoimmune hepatitis. (Linked to SLO 3.2)

3.2 Primary Biliary Cirrhosis

3.2.1 pathophysiology of primary biliary cirrhosis

An autoimmune attack on the small bile ducts

Primary biliary cirrhosis, also termed primary biliary cholangitis, an autoimmune condition predominantly affecting middle-aged women, results from immune-mediated destruction of the small intrahepatic bile ducts, leading to progressive **cholestasis**, impaired bile flow, and, over time, secondary hepatic fibrosis and, eventually, established cirrhosis if the condition remains untreated or inadequately controlled.

The precise trigger for this autoimmune process remains genuinely incompletely understood, though a combination of genetic susceptibility and environmental factors is thought to contribute, and the condition is frequently associated with other autoimmune diseases, reflecting a shared underlying predisposition to autoimmunity that this specific patient population appears to genuinely carry more commonly than the general population.

Fill in the blank: Primary biliary cirrhosis results in progressive cholestasis, impaired bile _____.

3.2.2 clinical presentation and diagnosis of primary biliary cirrhosis

Fatigue and itching, often preceding jaundice by years

Fatigue and pruritus, itching, are frequently the earliest presenting symptoms of primary biliary cirrhosis, often preceding the development of overt jaundice by a considerable period, and, since fatigue and itching are both genuinely non-specific symptoms with a wide range of possible causes, primary biliary cirrhosis should be actively considered in a middle-aged woman presenting with these symptoms alongside a cholestatic pattern of liver function test abnormality already discussed in an earlier Series of this course.



Diagnosis relies on the characteristic cholestatic biochemical pattern combined with **antimitochondrial antibody** testing, positive in the great majority of affected patients, and, where the diagnosis remains genuinely uncertain despite these findings, liver biopsy can provide additional supportive histological confirmation, though it is genuinely not required in every case given the high specificity of a positive antimitochondrial antibody result in the appropriate clinical context.

Fill in the blank: Diagnosis of primary biliary cirrhosis relies on antimitochondrial _____ testing alongside the cholestatic biochemical pattern.

3.2.3 management of primary biliary cirrhosis

Slowing progression and controlling troublesome symptoms

Ursodeoxycholic acid, a bile acid genuinely improving bile flow and reducing the toxic effect of retained bile acids on hepatocytes, forms the mainstay of disease-modifying treatment for primary biliary cirrhosis, meaningfully slowing disease progression in the majority of appropriately treated patients, with a second-line agent, **obeticholic acid**, available for patients with an inadequate response to ursodeoxycholic acid alone.

Symptomatic management of pruritus, genuinely challenging and sometimes significantly impacting quality of life, includes specific medication targeting the underlying mechanism of itch in cholestatic liver disease, and monitoring for disease progression towards established cirrhosis, alongside screening for the same complications of chronic liver disease already discussed in relation to chronic hepatitis earlier in this Series, forms an essential, ongoing part of comprehensive long-term care.

Fill in the blank: Ursodeoxycholic acid meaningfully slows disease _____ in the majority of appropriately treated patients.

Table 3.1: Comparing chronic hepatitis and primary biliary cirrhosis

Feature	Chronic hepatitis	Primary biliary cirrhosis
Primary target	Hepatocytes	Small intrahepatic bile ducts
Typical early symptom	Fatigue, often asymptomatic	Fatigue and pruritus
Key diagnostic test	Viral serology or autoantibodies	Antimitochondrial antibody



Pilot questions 3.2

1. Describe the pathophysiology of primary biliary cirrhosis. (Linked to SLO 3.3)
2. Describe the typical early symptoms of primary biliary cirrhosis. (Linked to SLO 3.3)
3. Describe the diagnostic approach to suspected primary biliary cirrhosis. (Linked to SLO 3.3)
4. Describe the role of ursodeoxycholic acid in management. (Linked to SLO 3.4)
5. Describe the challenge of managing pruritus in primary biliary cirrhosis. (Linked to SLO 3.4)

3.3 Crohn's Disease

3.3.1 pathophysiology and distribution of crohn's disease

Transmural inflammation that can affect any part of the gut

Crohn's disease, a chronic inflammatory bowel disease of genuinely uncertain, likely multifactorial cause involving genetic susceptibility and an abnormal immune response to gut microbiota, is characterised by **transmural inflammation**, extending through the full thickness of the bowel wall, and can affect any part of the gastrointestinal tract from mouth to anus, though the terminal ileum and proximal colon are most commonly involved.

A genuinely distinctive feature of Crohn's disease is its characteristic **skip lesions**, discontinuous patches of inflammation separated by segments of entirely normal, uninvolved bowel, a pattern directly contrasting with the continuous inflammation characteristic of ulcerative colitis discussed later in this Series, and this specific distribution pattern carries genuine diagnostic and, as discussed further in the following sub-Part, clinical significance.

Fill in the blank: Crohn's disease is characterised by transmural inflammation extending through the full thickness of the bowel _____.

3.3.2 clinical presentation of crohn's disease

A genuinely wide-ranging presentation reflecting variable disease location

Crohn's disease presents with chronic diarrhoea, abdominal pain, and weight loss, with the specific symptom pattern varying considerably depending on the location and extent of bowel involvement, and, given the transmural nature of the inflammation already discussed, Crohn's



disease carries a genuine risk of complications including **fistula** formation, an abnormal connection between the bowel and another structure, and **stricture**, narrowing of the bowel lumen from chronic inflammation and fibrosis.

Extraintestinal manifestations, affecting the joints, skin, and eyes, occur in a genuinely meaningful proportion of patients with Crohn's disease, reflecting the condition's underlying systemic immune dysregulation rather than a purely localised bowel process, and actively screening for these manifestations, alongside the intestinal symptoms themselves, forms a genuinely important part of comprehensive assessment for any patient with confirmed or suspected Crohn's disease.

Fill in the blank: A fistula is an abnormal connection between the bowel and another _____.

3.3.3 management of crohn's disease

Inducing then maintaining remission across a chronic, relapsing course

Induction of remission in an acute flare of Crohn's disease uses corticosteroids or, in selected cases, specific nutritional therapy, followed by **maintenance therapy**, typically immunomodulator medication such as azathioprine or, for more significant disease, biological therapy targeting specific inflammatory pathways, aiming to sustain remission and reduce the frequency of future disease flares over the genuinely long-term, chronic course this condition typically follows.

Surgical management, resection of a severely affected bowel segment, is reserved for complications including stricture causing obstruction, fistula, or disease genuinely refractory to medical therapy, though surgery in Crohn's disease is generally considered a treatment for complications rather than a cure, since the underlying disease process can genuinely recur at a new site even following apparently successful surgical resection of the originally affected segment.

Fill in the blank: Surgery in Crohn's disease treats complications rather than curing the disease, since it can genuinely recur at a new _____.



Skip Lesions in Crohn's Disease

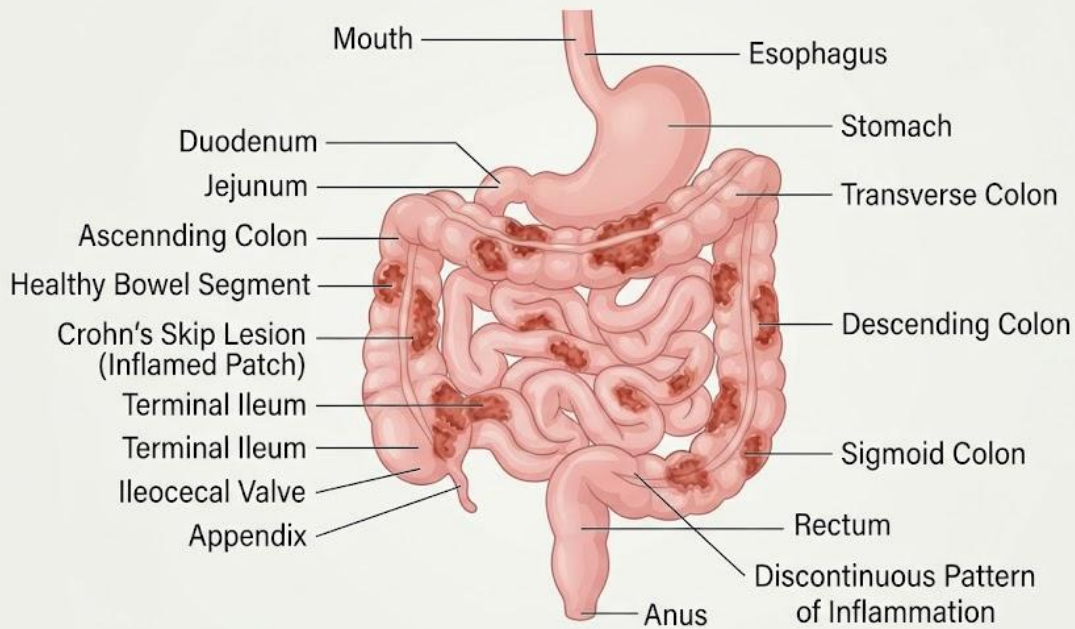


Figure 3.2: A labelled diagram showing the skip lesion distribution of Crohn's disease along the gastrointestinal tract.

Pilot questions 3.3

1. Describe the transmural nature of Crohn's disease inflammation. (Linked to SLO 3.5)
2. Describe skip lesions and their significance. (Linked to SLO 3.5)
3. Describe fistula and stricture as complications of Crohn's disease. (Linked to SLO 3.5)
4. List extraintestinal manifestations of Crohn's disease. (Linked to SLO 3.5)
5. Describe the general approach to inducing and maintaining remission in Crohn's disease. (Linked to SLO 3.6)

3.4 Ulcerative Colitis

3.4.1 pathophysiology and distribution of ulcerative colitis

Continuous, superficial inflammation confined to the colon

Ulcerative colitis, the second major form of inflammatory bowel disease, is characterised by **continuous inflammation** confined to the mucosal and submucosal layers of the bowel wall, in



genuine contrast to the transmural inflammation of Crohn's disease already discussed in the previous Part, and, unlike Crohn's disease, ulcerative colitis is genuinely confined to the colon, always involving the rectum and extending proximally to a variable extent.

This **continuous**, rather than skip-pattern, distribution, always beginning at the rectum and extending proximally without the discontinuous patches characteristic of Crohn's disease, is a genuinely important distinguishing feature, and the extent of colonic involvement, whether confined to the rectum alone or extending to involve the entire colon, carries direct relevance to both symptom severity and the specific treatment approach chosen.

Fill in the blank: Ulcerative colitis always involves the rectum, extending proximally in a continuous, rather than _____, pattern.

3.4.2 clinical presentation of ulcerative colitis

Bloody diarrhoea as the genuinely characteristic presenting symptom

Ulcerative colitis characteristically presents with **bloody diarrhoea**, alongside urgency and tenesmus, a sensation of incomplete rectal emptying, symptoms directly reflecting the rectal and colonic mucosal inflammation this condition produces, and the severity of symptoms genuinely correlates reasonably well with the extent and activity of colonic inflammation present, unlike the sometimes more variable symptom-disease correlation seen in Crohn's disease.

Acute severe ulcerative colitis, a genuine medical emergency presenting with frequent bloody stools, fever, tachycardia, and significant systemic upset, carries a genuine risk of **toxic megacolon**, dangerous colonic dilatation with risk of perforation, and requires urgent hospital admission and intensive medical treatment, with surgical colectomy considered where medical treatment fails to achieve adequate improvement within a defined, closely monitored timeframe.

Fill in the blank: Acute severe ulcerative colitis carries a genuine risk of toxic megacolon, dangerous colonic dilatation with risk of _____.

3.4.3 management and comparison with crohn's disease

Broadly similar principles, with genuinely important specific differences

Management of ulcerative colitis follows broadly similar principles to Crohn's disease, inducing then maintaining remission using aminosalicylates, corticosteroids, immunomodulators, and biological therapy as appropriate to disease severity and extent, though **colectomy**, surgical removal of the entire colon, is genuinely curative for ulcerative colitis given the disease's



confinement to this single organ, in direct contrast to the non-curative role of surgery already discussed for Crohn's disease.

Long-term colorectal cancer surveillance, given the genuinely increased risk of colorectal malignancy in longstanding, extensive ulcerative colitis, forms an essential part of ongoing care, and confidently comparing ulcerative colitis with Crohn's disease across distribution, inflammation depth, characteristic symptoms, and the curative potential of surgery, closing this Series on this comparative theme, provides a genuinely useful, consolidated framework for approaching inflammatory bowel disease as a whole.

Fill in the blank: Colectomy is genuinely curative for ulcerative colitis given the disease's confinement to a single _____.

Table 3.2: Comparing Crohn's disease and ulcerative colitis

Feature	Crohn's disease	Ulcerative colitis
Distribution	Anywhere mouth to anus, skip lesions	Colon only, continuous from rectum
Depth of inflammation	Transmural	Mucosal and submucosal
Curative surgery	No, disease can recur elsewhere	Yes, colectomy is curative

Pilot questions 3.4

1. Describe the distribution of ulcerative colitis. (Linked to SLO 3.7)
2. Describe the characteristic presenting symptom of ulcerative colitis. (Linked to SLO 3.7)
3. Describe acute severe ulcerative colitis and toxic megacolon. (Linked to SLO 3.7)
4. Explain why colectomy is curative for ulcerative colitis but not for Crohn's disease. (Linked to SLO 3.8)
5. Compare the distribution and depth of inflammation between Crohn's disease and ulcerative colitis. (Linked to SLO 3.8)



Series Summary

This Series covered chronic hepatitis, primary biliary cirrhosis, and the two major forms of inflammatory bowel disease. Part 3.1 covered the causes, presentation, investigation, and management of chronic hepatitis, while Part 3.2 turned to primary biliary cirrhosis. Part 3.3 covered Crohn's disease, and Part 3.4 closed with ulcerative colitis and a direct comparison between the two forms of inflammatory bowel disease.

You should now be able to describe and manage chronic hepatitis, primary biliary cirrhosis, Crohn's disease, and ulcerative colitis across all four Parts of this Series, meeting the outcomes set for this Series. Series 4 turns next to gastrointestinal bleeding and malignancies.

Glossary of Terms

1. **Antimitochondrial antibody:** an autoantibody positive in the great majority of primary biliary cirrhosis cases.
2. **Autoimmune hepatitis:** immune-mediated liver inflammation, a cause of chronic hepatitis.
3. **Cholestasis:** impaired bile flow, characteristic of primary biliary cirrhosis.
4. **Chronic hepatitis:** liver inflammation persisting beyond six months.
5. **Colectomy:** surgical removal of the colon, curative for ulcerative colitis.
6. **Crohn's disease:** a chronic inflammatory bowel disease with transmural inflammation and skip lesions.
7. **Extraintestinal manifestations:** features of inflammatory bowel disease affecting joints, skin, or eyes.
8. **Fistula:** an abnormal connection between the bowel and another structure.
9. **Primary biliary cirrhosis:** autoimmune destruction of the small intrahepatic bile ducts.
10. **Skip lesions:** discontinuous patches of inflammation separated by normal bowel in Crohn's disease.
11. **Toxic megacolon:** dangerous colonic dilatation with risk of perforation in severe colitis.
12. **Ulcerative colitis:** a chronic inflammatory bowel disease with continuous mucosal inflammation confined to the colon.



Concept Check Questions [Series 3]

Objective questions

- Chronic hepatitis is liver inflammation persisting beyond _____ months. (Linked to SLO 3.1)
- Diagnosis of primary biliary cirrhosis relies on antimitochondrial _____ testing alongside the cholestatic biochemical pattern. (Linked to SLO 3.3)
- Crohn's disease is characterised by transmural inflammation extending through the full thickness of the bowel _____. (Linked to SLO 3.5)
- Ulcerative colitis always involves the rectum, extending proximally in a continuous, rather than _____, pattern. (Linked to SLO 3.7)
- Colectomy is genuinely curative for ulcerative colitis given the disease's confinement to a single _____. (Linked to SLO 3.8)

Short-answer questions

1. List the causes of chronic hepatitis. (Linked to SLO 3.1)
2. Describe the typical early symptoms of primary biliary cirrhosis. (Linked to SLO 3.3)
3. Describe skip lesions and their significance. (Linked to SLO 3.5)
4. Describe acute severe ulcerative colitis and toxic megacolon. (Linked to SLO 3.7)
5. Compare the distribution and depth of inflammation between Crohn's disease and ulcerative colitis. (Linked to SLO 3.8)

Long-answer questions

6. Discuss the causes, presentation, investigation, and management of chronic hepatitis. (Linked to SLO 3.1, 3.2)
7. Describe the pathophysiology, presentation, and management of primary biliary cirrhosis. (Linked to SLO 3.3, 3.4)
8. Discuss the pathophysiology, distribution, presentation, and management of Crohn's disease. (Linked to SLO 3.5, 3.6)
9. Describe the pathophysiology, distribution, and presentation of ulcerative colitis. (Linked to SLO 3.7)
10. Discuss the management of ulcerative colitis and compare it with Crohn's disease. (Linked to SLO 3.8)



Answers to Concept Check Questions [Series 3]

Objective questions

11. Six.
12. Antibody.
13. Wall.
14. Skip.
15. Organ.

Short-answer questions

16. Causes include chronic viral hepatitis, autoimmune hepatitis, and less commonly medications and metabolic conditions.
17. Fatigue and pruritus are frequently the earliest presenting symptoms, often preceding jaundice.
18. Skip lesions are discontinuous patches of inflammation separated by normal bowel, distinguishing Crohn's from ulcerative colitis.
19. Acute severe ulcerative colitis is a medical emergency with frequent bloody stools and systemic upset, risking toxic megacolon.
20. Crohn's disease can affect anywhere with transmural, skip-pattern inflammation, while ulcerative colitis is confined to the colon with continuous, mucosal inflammation.

Long-answer questions

21. **Basic response:** Chronic hepatitis results from viral infection, autoimmune disease, or other causes; presentation is often asymptomatic initially; investigation combines liver function tests with cause-specific testing and biopsy; management is cause-specific, using antiviral or immunosuppressive therapy.

Value-added response: A value-added response notes that antiviral therapy offers genuine cure for hepatitis C in most appropriately treated patients.

22. **Basic response:** Primary biliary cirrhosis is autoimmune destruction of small bile ducts causing cholestasis; presentation includes fatigue and pruritus; management uses ursodeoxycholic acid, with obeticholic acid as second-line therapy.

Value-added response: A value-added response notes that the condition is frequently associated with other autoimmune diseases.

23. **Basic response:** Crohn's disease causes transmural inflammation with skip lesions anywhere from mouth to anus; presentation includes diarrhoea, pain, and complications



such as fistula and stricture; management induces then maintains remission with immunomodulators or biologics.

Value-added response: A value-added response notes that surgery treats complications rather than curing the disease, which can recur elsewhere.

24. **Basic response:** Ulcerative colitis causes continuous mucosal inflammation confined to the colon, always involving the rectum; presentation is characteristically bloody diarrhoea with urgency and tenesmus.

Value-added response: A value-added response notes that acute severe ulcerative colitis carries genuine risk of toxic megacolon.

25. **Basic response:** Management follows similar principles to Crohn's disease, but colectomy is genuinely curative for ulcerative colitis given its confinement to the colon, unlike Crohn's disease.

Value-added response: A value-added response notes that longstanding, extensive ulcerative colitis carries increased colorectal cancer risk requiring surveillance.

Answers to Pilot Questions [Series 3]

Part 3.1

1. Causes include chronic viral hepatitis, autoimmune hepatitis, medications, and metabolic conditions.
2. Grade describes inflammatory activity, while stage describes the degree of established fibrosis.
3. Early presentation is often asymptomatic, identified incidentally on liver function testing.
4. Signs include jaundice, spider naevi, palmar erythema, and, later, ascites.
5. Antiviral therapy treats viral hepatitis, while immunosuppressive therapy treats autoimmune hepatitis.

Part 3.2

6. Primary biliary cirrhosis results from autoimmune destruction of small intrahepatic bile ducts, causing cholestasis.
7. Fatigue and pruritus are the typical early symptoms, often preceding jaundice.



8. Diagnosis combines the cholestatic biochemical pattern with antimitochondrial antibody testing.
9. Ursodeoxycholic acid improves bile flow and meaningfully slows disease progression.
10. Pruritus management is genuinely challenging and can significantly impact quality of life.

Part 3.3

11. Crohn's disease involves inflammation extending through the full thickness of the bowel wall.
12. Skip lesions are discontinuous inflamed patches separated by normal bowel, distinguishing Crohn's from ulcerative colitis.
13. A fistula is an abnormal connection between bowel and another structure; a stricture is bowel lumen narrowing.
14. Manifestations include joint, skin, and eye involvement.
15. Induction uses corticosteroids or nutritional therapy, followed by immunomodulator or biological maintenance therapy.

Part 3.4

1. Ulcerative colitis is confined to the colon, always involving the rectum and extending proximally continuously.
2. The characteristic symptom is bloody diarrhoea, alongside urgency and tenesmus.
3. Acute severe ulcerative colitis is a medical emergency risking toxic megacolon, dangerous colonic dilatation.
4. Colectomy removes the entire diseased organ in ulcerative colitis, whereas Crohn's disease can recur at any gut location.
5. Crohn's disease has skip-pattern, transmural inflammation anywhere in the gut; ulcerative colitis has continuous, mucosal inflammation confined to the colon.

References and Attributions [Series 3]

1. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
2. Feldman, M., Friedman, L. S., & Brandt, L. J. (2020). Sleisenger and Fordtran's Gastrointestinal and Liver Disease (11th ed.). Elsevier.



3. European Association for the Study of the Liver (2017). Clinical Practice Guidelines: The Diagnosis and Management of Patients with Primary Biliary Cholangitis. Journal of Hepatology.
4. National Institute for Health and Care Excellence (2019). Crohn's Disease and Ulcerative Colitis: Management (NG130). NICE.

Figures, Plates, Tables, and Boxes

1. Figure 3.1: Jaundice and spider naevi as stigmata of chronic liver disease. AI-generated using the prompt: "Create a realistic clinical illustration titled Stigmata of Chronic Liver Disease, showing a patient's upper chest and neck with visible yellowish skin discolouration and small spider-like vascular marks, clinical examination setting, respectful and clinical in tone. Clean clinical illustration style, no text."
2. Table 3.1: Comparing chronic hepatitis and primary biliary cirrhosis. AI-generated using the prompt: "Create a clean reference table titled Comparing Chronic Hepatitis and Primary Biliary Cirrhosis, listing feature, chronic hepatitis, and primary biliary cirrhosis. Simple clinical reference table style with outside border."
3. Figure 3.2: A labelled diagram showing the skip lesion distribution of Crohn's disease along the gastrointestinal tract. AI-generated using the prompt: "Create a clean, accurate labelled anatomical diagram titled Skip Lesions in Crohn's Disease, showing an outline of the gastrointestinal tract from mouth to anus with several discontinuous, labelled patches of inflammation separated by segments of normal bowel. Educational anatomical diagram style, no photographic content."
4. Table 3.2: Comparing Crohn's disease and ulcerative colitis. AI-generated using the prompt: "Create a clean reference table titled Comparing Crohn's Disease and Ulcerative Colitis, listing feature, Crohn's disease, and ulcerative colitis. Simple clinical reference table style with outside border."



Course code: MED 608

Course title: Gastroenterology II

Course credit load: 2 Units

Series 4: Gastrointestinal Bleeding and Malignancies

- **Part 4.1: Upper Gastrointestinal Bleeding**
 - Sub Part 4.1.1: Causes of upper gastrointestinal bleeding
 - Sub Part 4.1.2: Clinical presentation and initial assessment
 - Sub Part 4.1.3: Management of upper gastrointestinal bleeding
- **Part 4.2: Lower Gastrointestinal Bleeding**
 - Sub Part 4.2.1: Causes of lower gastrointestinal bleeding
 - Sub Part 4.2.2: Clinical presentation and assessment
 - Sub Part 4.2.3: Management of lower gastrointestinal bleeding
- **Part 4.3: Oesophageal and Gastric Malignancy**
 - Sub Part 4.3.1: Oesophageal cancer
 - Sub Part 4.3.2: Gastric cancer
 - Sub Part 4.3.3: Investigation and staging of upper gastrointestinal malignancy
- **Part 4.4: Colorectal and Other Gastrointestinal Malignancies**
 - Sub Part 4.4.1: Colorectal cancer: risk factors and presentation
 - Sub Part 4.4.2: Colorectal cancer screening and staging
 - Sub Part 4.4.3: Management principles of colorectal and other gastrointestinal cancers



Introduction

This Series turns to two genuinely major topics in gastroenterology, **gastrointestinal bleeding**, spanning both upper and lower sources, and **gastrointestinal malignancy**, spanning the oesophagus, stomach, and colorectum. Both topics demand the same structured, prioritised approach already emphasised throughout this course, since prompt recognition, whether of an actively bleeding patient or a cancer presenting with subtle early symptoms, genuinely determines outcome.

The aim of this Series is to equip you with an understanding of the causes, presentation, and management of upper and lower gastrointestinal bleeding, and the presentation, investigation, staging, and management principles of oesophageal, gastric, and colorectal cancer, alongside the screening approach used for colorectal cancer specifically.

This Series opens with **upper gastrointestinal bleeding**, moves through **lower gastrointestinal bleeding** and **oesophageal and gastric malignancy**, and closes with **colorectal and other gastrointestinal malignancies**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 4.1:** describe the causes and clinical presentation of upper gastrointestinal bleeding
- **SLO 4.2:** discuss the initial assessment and management of upper gastrointestinal bleeding
- **SLO 4.3:** describe the causes, presentation, and management of lower gastrointestinal bleeding
- **SLO 4.4:** describe oesophageal and gastric cancer
- **SLO 4.5:** discuss the investigation and staging of upper gastrointestinal malignancy
- **SLO 4.6:** describe the risk factors and clinical presentation of colorectal cancer
- **SLO 4.7:** discuss colorectal cancer screening and staging
- **SLO 4.8:** describe the management principles of colorectal and other gastrointestinal cancers

4.1 Upper Gastrointestinal Bleeding

4.1.1 causes of upper gastrointestinal bleeding



Bleeding from the oesophagus, stomach, or duodenum

Upper gastrointestinal bleeding, originating proximal to the ligament of Treitz, most commonly results from peptic ulcer disease already covered in genuine depth in Series 1 of this course, alongside **oesophageal varices**, dilated submucosal veins in patients with portal hypertension, oesophagitis, and, less commonly, gastric malignancy, already introduced as an important consideration when assessing dyspepsia and gastric ulceration earlier in this course.

Variceal bleeding, though genuinely less common overall than peptic ulcer bleeding, carries considerably higher associated mortality given both the volume of blood loss it can produce and the underlying severe liver disease it typically reflects, making rapid identification of a genuine or suspected variceal source, discussed further in the management sub-Part of this Part, a genuinely important early consideration in any patient presenting with significant upper gastrointestinal bleeding.

Fill in the blank: Upper gastrointestinal bleeding originates proximal to the ligament of _____.

4.1.2 clinical presentation and initial assessment

Recognising the bleed, then assessing its severity

Upper gastrointestinal bleeding presents with **haematemesis**, vomiting blood or altered, coffee-ground material, and **melaena**, black, tarry, offensive-smelling stool from digested blood, and, in more severe or rapid bleeding, **haematochezia**, fresh red blood passed rectally, can occur despite an upper source given rapid transit through the bowel, a genuinely important point to remember when assessing apparent lower gastrointestinal bleeding.

Initial assessment prioritises haemodynamic status, following the same airway, breathing, circulation approach already emphasised throughout this course, alongside structured risk stratification using a validated scoring tool combining clinical and laboratory findings to estimate the likelihood of significant ongoing bleeding and the need for urgent endoscopic intervention, genuinely guiding both the urgency of endoscopy and the appropriate level of initial supportive care.

Fill in the blank: Haematochezia, fresh red blood passed rectally, can occur despite an upper source given rapid _____ through the bowel.



4.1.3 management of upper gastrointestinal bleeding

Resuscitation, endoscopic control, and, where relevant, treating the underlying cause

Management begins with resuscitation, intravenous fluid and, where indicated, blood product transfusion, alongside **proton pump inhibitor therapy**, already discussed in relation to peptic ulcer disease in Series 1, reducing the risk of rebleeding from a peptic ulcer source, and, where variceal bleeding is genuinely suspected, specific vasoactive medication and prophylactic antibiotics given the recognised association between variceal bleeding and infection in patients with underlying liver disease.

Urgent upper gastrointestinal endoscopy, already discussed in the context of peptic ulcer disease diagnosis in Series 1, allows direct identification of the bleeding source and, genuinely importantly, therapeutic haemostasis, including injection therapy, thermal coagulation, or mechanical clipping for a bleeding ulcer, or **band ligation** for bleeding oesophageal varices, and, where endoscopic treatment fails to achieve adequate control, radiological or surgical intervention offers a further treatment option.

Fill in the blank: Band ligation is used for endoscopic management of bleeding oesophageal _____.



Figure 4.1: A clinician performing endoscopic band ligation of a bleeding oesophageal varix.



Pilot questions 4.1

- List the causes of upper gastrointestinal bleeding. (Linked to SLO 4.1)
- Explain why variceal bleeding carries higher associated mortality than peptic ulcer bleeding. (Linked to SLO 4.1)
- Distinguish haematemesis, melaena, and haematochezia. (Linked to SLO 4.1)
- Describe the initial assessment priorities for upper gastrointestinal bleeding. (Linked to SLO 4.2)
- Describe endoscopic treatment options for a bleeding peptic ulcer and for bleeding varices. (Linked to SLO 4.2)

4.2 Lower Gastrointestinal Bleeding

4.2.1 causes of lower gastrointestinal bleeding

Bleeding distal to the ligament of Treitz

Lower gastrointestinal bleeding, originating distal to the ligament of Treitz already introduced in the previous Part, most commonly results from **diverticular disease**, colorectal malignancy already introduced in an earlier Series of this course and covered in more depth later in this Series, haemorrhoids, and inflammatory bowel disease, already covered in Series 3, with the relative likelihood of each cause varying considerably with patient age and specific clinical presentation.

Angiodysplasia, abnormal, fragile blood vessels within the bowel wall, is a further recognised cause, particularly in older patients, and, given this genuinely wide differential diagnosis, a structured, systematic diagnostic approach, rather than assuming the most common cause applies to every individual patient, is essential to reaching an accurate diagnosis and appropriately targeted treatment.

Fill in the blank: Angiodysplasia describes abnormal, fragile blood _____ within the bowel wall.



4.2.2 clinical presentation and assessment

From minor spotting to genuine haemodynamic compromise

Lower gastrointestinal bleeding typically presents with fresh or altered blood passed rectally, with the specific character of blood, bright red versus darker, altered blood, providing some clue to the likely source location, though this pattern is genuinely not entirely reliable, and, as already noted in the previous Part, a brisk upper gastrointestinal bleed can mimic lower gastrointestinal bleeding given rapid intestinal transit.

Assessment combines haemodynamic evaluation with a careful history, including the specific pattern and duration of bleeding, associated symptoms such as altered bowel habit or weight loss raising concern for malignancy, and relevant risk factors, and, given the genuine possibility that an apparent lower gastrointestinal bleed actually originates from an upper source, upper endoscopy should genuinely be considered to exclude this possibility, particularly in a haemodynamically unstable patient.

Fill in the blank: Upper endoscopy should genuinely be considered to exclude an upper source in an apparent lower gastrointestinal bleed, particularly if haemodynamically _____.

4.2.3 management of lower gastrointestinal bleeding

Localising the source, then achieving definitive control

Management begins with the same resuscitation principles already established for upper gastrointestinal bleeding, and **colonoscopy**, once adequate bowel preparation has been achieved where clinically feasible, remains the key investigation, allowing direct visualisation, identification of the specific bleeding source, and, where accessible, therapeutic intervention similar in principle to the endoscopic techniques already discussed for upper gastrointestinal bleeding.

Where colonoscopy cannot adequately identify or control the bleeding source, particularly in a patient with ongoing, significant haemorrhage, **CT angiography** or catheter angiography can localise active bleeding and, in selected cases, allow therapeutic embolisation, and surgical intervention, resection of the bleeding bowel segment, is reserved for patients with ongoing, life-threatening haemorrhage genuinely refractory to these less invasive treatment approaches.

Fill in the blank: CT angiography can localise active bleeding and, in selected cases, allow therapeutic _____.



Table 4.1: Comparing upper and lower gastrointestinal bleeding

Feature	Upper gastrointestinal bleeding	Lower gastrointestinal bleeding
Typical presentation	Haematemesis, melaena	Fresh or altered rectal bleeding
Common causes	Peptic ulcer, varices	Diverticular disease, malignancy
Key investigation	Upper endoscopy	Colonoscopy

Pilot questions 4.2

1. List the causes of lower gastrointestinal bleeding. (Linked to SLO 4.3)
2. Describe angiodysplasia. (Linked to SLO 4.3)
3. Explain why an upper gastrointestinal source should be considered in apparent lower gastrointestinal bleeding. (Linked to SLO 4.3)
4. Describe the key investigation for lower gastrointestinal bleeding. (Linked to SLO 4.3)
5. Describe alternative approaches when colonoscopy cannot identify or control the bleeding source. (Linked to SLO 4.3)

4.3 Oesophageal and Gastric Malignancy

4.3.1 oesophageal cancer

Two distinct histological types, two distinct risk factor profiles

Oesophageal cancer occurs as two genuinely distinct histological types, **squamous cell carcinoma**, associated with smoking and alcohol excess and more common in the proximal and mid oesophagus, and **adenocarcinoma**, associated with chronic gastro-oesophageal reflux disease and Barrett's oesophagus, a premalignant change in the oesophageal lining, and more commonly affecting the distal oesophagus, each with a genuinely distinct underlying risk factor profile despite sharing the same eventual anatomical location of origin.

Progressive dysphagia, difficulty swallowing initially affecting solids and progressing to liquids as the tumour enlarges and increasingly obstructs the oesophageal lumen, is the classic presenting symptom, and, given the oesophagus's genuine lack of a serosal layer, oesophageal



cancer can spread locally with genuine ease, contributing to the often advanced stage at which this specific malignancy is unfortunately frequently diagnosed.

Fill in the blank: Barrett's oesophagus is a premalignant change in the oesophageal lining associated with chronic gastro-oesophageal reflux _____.

4.3.2 gastric cancer

Often genuinely silent until relatively advanced

Gastric cancer, most commonly adenocarcinoma, is strongly associated with *Helicobacter pylori* infection, already discussed extensively in relation to peptic ulcer disease in Series 1, alongside dietary factors including high salt intake and smoked or preserved food, and its incidence, though genuinely declining in many populations over recent decades, remains a significant global cause of cancer mortality given its frequently late-stage presentation.

Presentation includes the alarm features already discussed in relation to dyspepsia in Series 1, weight loss, dysphagia, and persistent vomiting, alongside, in more advanced disease, a palpable epigastric mass or signs of metastatic spread, and this genuinely often subtle, non-specific early presentation, easily confused with the far more common benign dyspepsia and peptic ulcer disease already covered in this course, underlines why active screening for alarm features in every dyspeptic patient genuinely matters.

Fill in the blank: Gastric cancer is strongly associated with *Helicobacter pylori* infection, alongside dietary factors including high salt intake and smoked or preserved _____.

4.3.3 investigation and staging of upper gastrointestinal malignancy

Confirming the diagnosis, then mapping its extent

Upper gastrointestinal endoscopy, already established as the key investigation for suspected oesophageal or gastric cancer given its ability to directly visualise and biopsy any suspicious lesion, confirms the histological diagnosis, and, once malignancy is confirmed, staging investigations, including CT scanning to assess for metastatic spread and, where available, endoscopic ultrasound to assess the specific depth of local tumour invasion, determine the most appropriate treatment approach.

Accurate staging genuinely matters clinically, since it directly determines whether curative treatment, typically surgical resection with or without perioperative chemotherapy, remains a realistic option, or whether the disease has progressed to a stage where treatment intent genuinely shifts towards palliation, and this staged, structured approach to investigation and



treatment planning reflects the same individualised, evidence-based decision-making principle already emphasised throughout this whole course.

Fill in the blank: Endoscopic ultrasound assesses the specific depth of local tumour _____ as part of staging.



Figure 4.2: A clinician assessing swallowing function in a patient with dysphagia.

Pilot questions 4.3

1. Distinguish squamous cell carcinoma from adenocarcinoma of the oesophagus by risk factors and location. (Linked to SLO 4.4)
2. Describe the classic presenting symptom of oesophageal cancer. (Linked to SLO 4.4)
3. Describe the risk factors for gastric cancer. (Linked to SLO 4.4)
4. Describe the investigation used to confirm oesophageal or gastric cancer. (Linked to SLO 4.5)
5. Explain why accurate staging genuinely matters clinically. (Linked to SLO 4.5)



4.4 Colorectal and Other Gastrointestinal Malignancies

4.4.1 colorectal cancer: risk factors and presentation

The most common gastrointestinal malignancy overall

Colorectal cancer, arising predominantly from malignant transformation of a pre-existing adenomatous polyp over a genuinely prolonged period, has risk factors including advancing age, a diet low in fibre and high in processed meat, inflammatory bowel disease already discussed in Series 3, and specific inherited genetic conditions, including hereditary non-polyposis colorectal cancer and familial adenomatous polyposis, in a smaller proportion of affected patients.

Presentation varies with tumour location, with right-sided tumours more commonly presenting with iron deficiency anaemia and vague abdominal symptoms given the wider calibre of the proximal colon, and left-sided tumours more commonly presenting with altered bowel habit and rectal bleeding given the narrower calibre and closer proximity to the rectum, a genuinely useful anatomical pattern worth recognising alongside the alarm features already discussed for other gastrointestinal malignancies earlier in this Series.

Fill in the blank: Colorectal cancer arises predominantly from malignant transformation of a pre-existing adenomatous _____.

4.4.2 colorectal cancer screening and staging

Detecting and interrupting disease before it becomes symptomatic

Colorectal cancer screening, using faecal immunochemical testing to detect occult blood in stool, followed by colonoscopy for a positive result, aims to identify cancer at an earlier, more treatable stage, or, genuinely importantly, to identify and remove a premalignant adenomatous polyp before it has the opportunity to progress to genuine malignancy, a preventive rather than purely diagnostic function this specific screening approach uniquely offers.

Staging of confirmed colorectal cancer, using CT imaging to assess for metastatic spread alongside pathological assessment of the resected specimen following surgery, follows the same fundamental staging principle already discussed for oesophageal and gastric cancer earlier in this Series, directly informing prognosis and the specific combination of surgery, chemotherapy, and, for rectal cancer specifically, radiotherapy most appropriate for the individual patient.

Fill in the blank: Colorectal cancer screening can identify and remove a premalignant adenomatous polyp before it progresses to genuine _____.



4.4.3 management principles of colorectal and other gastrointestinal cancers

Surgery as the cornerstone, with adjuvant therapy where indicated

Surgical resection, removing the affected bowel segment along with its associated lymph node drainage, remains the cornerstone of curative treatment for localised colorectal cancer, with **adjuvant chemotherapy**, given following surgery, indicated for patients with higher-risk disease features to reduce the risk of recurrence, and neoadjuvant chemoradiotherapy, given before surgery specifically for rectal cancer, used to shrink the tumour and improve the chance of complete surgical resection in appropriately selected cases.

Management of metastatic colorectal cancer, and indeed the other gastrointestinal malignancies already discussed across this Series, genuinely requires individualised, multidisciplinary decision-making, weighing the specific extent of disease, the patient's overall fitness, and their personal treatment goals, and this Series closes on the same theme of coordinated, structured, individualised care that has run consistently throughout this whole programme of gastroenterological study.

Fill in the blank: Neoadjuvant chemoradiotherapy is given before surgery specifically for _____ cancer to improve resection chances.

Table 4.2: Colorectal cancer presentation by tumour location

Location	Typical presentation	Anatomical reason
Right-sided colon	Iron deficiency anaemia, vague symptoms	Wider calibre allows silent growth
Left-sided colon	Altered bowel habit, rectal bleeding	Narrower calibre obstructs earlier
Rectum	Rectal bleeding, tenesmus	Close proximity to anal canal

Pilot questions 4.4

1. List risk factors for colorectal cancer. (Linked to SLO 4.6)
2. Distinguish the typical presentation of right-sided from left-sided colorectal cancer. (Linked to SLO 4.6)
3. Describe the colorectal cancer screening approach. (Linked to SLO 4.7)
4. Describe the staging approach used for colorectal cancer. (Linked to SLO 4.7)
5. Describe the role of surgery and adjuvant therapy in colorectal cancer management. (Linked to SLO 4.8)



Series Summary

This Series covered gastrointestinal bleeding and malignancy. Part 4.1 covered upper gastrointestinal bleeding, while Part 4.2 turned to lower gastrointestinal bleeding. Part 4.3 covered oesophageal and gastric malignancy, and Part 4.4 closed with colorectal and other gastrointestinal malignancies.

You should now be able to describe and manage upper and lower gastrointestinal bleeding and describe the presentation, staging, and management of oesophageal, gastric, and colorectal cancer across all four Parts of this Series, meeting the outcomes set for this Series. Series 5 turns next to malabsorption syndromes and hepatic failure.

Glossary of Terms

1. **Adenomatous polyp:** a premalignant colorectal growth that can progress to cancer over time.
2. **Angiodysplasia:** abnormal, fragile blood vessels within the bowel wall, a cause of lower gastrointestinal bleeding.
3. **Band ligation:** an endoscopic technique treating bleeding oesophageal varices.
4. **Barrett's oesophagus:** a premalignant change in the oesophageal lining from chronic reflux.
5. **Colonoscopy:** direct visualisation of the colon, the key investigation for lower gastrointestinal bleeding.
6. **Colorectal cancer screening:** faecal testing followed by colonoscopy to detect cancer or premalignant polyps early.
7. **Dysphagia:** difficulty swallowing, the classic presenting symptom of oesophageal cancer.
8. **Haematemesis:** vomiting blood or altered, coffee-ground material.
9. **Haematochezia:** fresh red blood passed rectally.
10. **Melaena:** black, tarry, offensive-smelling stool from digested blood.
11. **Oesophageal varices:** dilated submucosal veins in patients with portal hypertension.
12. **Upper gastrointestinal bleeding:** bleeding originating proximal to the ligament of Treitz.



Concept Check Questions [Series 4]

Objective questions

- Upper gastrointestinal bleeding originates proximal to the ligament of _____. (Linked to SLO 4.1)
- Angiodysplasia describes abnormal, fragile blood _____ within the bowel wall. (Linked to SLO 4.3)
- Barrett's oesophagus is a premalignant change in the oesophageal lining associated with chronic gastro-oesophageal reflux _____. (Linked to SLO 4.4)
- Colorectal cancer arises predominantly from malignant transformation of a pre-existing adenomatous _____. (Linked to SLO 4.6)
- Neoadjuvant chemoradiotherapy is given before surgery specifically for _____ cancer to improve resection chances. (Linked to SLO 4.8)

Short-answer questions

1. Distinguish haematemesis, melaena, and haematochezia. (Linked to SLO 4.1)
2. Explain why an upper gastrointestinal source should be considered in apparent lower gastrointestinal bleeding. (Linked to SLO 4.3)
3. Describe the classic presenting symptom of oesophageal cancer. (Linked to SLO 4.4)
4. Distinguish the typical presentation of right-sided from left-sided colorectal cancer. (Linked to SLO 4.6)
5. Describe the colorectal cancer screening approach. (Linked to SLO 4.7)

Long-answer questions

6. Discuss the causes, presentation, and management of upper gastrointestinal bleeding. (Linked to SLO 4.1, 4.2)
7. Describe the causes, presentation, and management of lower gastrointestinal bleeding. (Linked to SLO 4.3)
8. Discuss oesophageal and gastric cancer, including their investigation and staging. (Linked to SLO 4.4, 4.5)
9. Describe the risk factors, presentation, screening, and staging of colorectal cancer. (Linked to SLO 4.6, 4.7)
10. Discuss the management principles of colorectal and other gastrointestinal cancers. (Linked to SLO 4.8)



Answers to Concept Check Questions [Series 4]

Objective questions

11. Treitz.
12. Vessels.
13. Disease.
14. Polyp.
15. Rectal.

Short-answer questions

16. Haematemesis is vomiting blood, melaena is black tarry stool, and haematochezia is fresh red rectal bleeding.
17. A brisk upper bleed can mimic lower bleeding given rapid intestinal transit, so this should be actively excluded.
18. Progressive dysphagia, initially affecting solids and progressing to liquids, is the classic presenting symptom.
19. Right-sided tumours present with anaemia and vague symptoms; left-sided tumours present with altered bowel habit and bleeding.
20. Screening uses faecal immunochemical testing followed by colonoscopy for a positive result.

Long-answer questions

21. **Basic response:** Upper gastrointestinal bleeding most commonly results from peptic ulcer disease or varices, presenting with haematemesis and melaena; management combines resuscitation, proton pump inhibitors, and urgent endoscopy with therapeutic haemostasis.

Value-added response: A value-added response notes that variceal bleeding carries higher mortality given the severity of underlying liver disease it reflects.

22. **Basic response:** Lower gastrointestinal bleeding commonly results from diverticular disease, malignancy, or angiodysplasia, presenting with fresh or altered rectal bleeding; management uses colonoscopy, with angiography or surgery for refractory bleeding.

Value-added response: A value-added response notes that an apparent lower bleed should prompt consideration of an upper source given rapid transit.

23. **Basic response:** Oesophageal cancer includes squamous cell carcinoma and adenocarcinoma with distinct risk factors, presenting with progressive dysphagia; gastric



cancer is associated with *Helicobacter pylori*; both are investigated with endoscopy and staged with CT and endoscopic ultrasound.

Value-added response: A value-added response notes that accurate staging directly determines whether curative treatment remains a realistic option.

24. **Basic response:** Risk factors include age, diet, inflammatory bowel disease, and genetic conditions; presentation varies by tumour location; screening uses faecal testing and colonoscopy; staging uses CT and pathological assessment.

Value-added response: A value-added response notes that screening can remove premalignant polyps before they progress to genuine malignancy.

25. **Basic response:** Surgical resection is the cornerstone of curative treatment, with adjuvant or neoadjuvant chemotherapy or chemoradiotherapy used according to risk features and tumour location.

Value-added response: A value-added response notes that management of metastatic disease requires individualised, multidisciplinary decision-making.

Answers to Pilot Questions [Series 4]

Part 4.1

1. Causes include peptic ulcer disease, oesophageal varices, oesophagitis, and gastric malignancy.
2. Variceal bleeding reflects severe underlying liver disease and can produce large volume blood loss.
3. Haematemesis is vomiting blood, melaena is black tarry stool, and haematochezia is fresh red rectal blood.
4. Assessment prioritises haemodynamic status alongside structured risk stratification scoring.
5. Ulcers are treated with injection, thermal coagulation, or clipping; varices are treated with band ligation.



Part 4.2

6. Causes include diverticular disease, colorectal malignancy, haemorrhoids, and inflammatory bowel disease.
7. Angiodysplasia is abnormal, fragile blood vessels within the bowel wall.
8. A brisk upper bleed can mimic lower bleeding given rapid transit through the bowel.
9. Colonoscopy is the key investigation for lower gastrointestinal bleeding.
10. CT or catheter angiography can localise bleeding and allow therapeutic embolisation.

Part 4.3

11. Squamous cell carcinoma relates to smoking and alcohol, affecting the proximal oesophagus; adenocarcinoma relates to reflux, affecting the distal oesophagus.
12. Progressive dysphagia, initially for solids then liquids, is the classic presenting symptom.
13. Risk factors include *Helicobacter pylori* infection and a diet high in salt and preserved food.
14. Upper gastrointestinal endoscopy with biopsy confirms the diagnosis.
15. Staging determines whether curative treatment remains realistic or whether care shifts towards palliation.

Part 4.4

1. Risk factors include age, low-fibre diet, inflammatory bowel disease, and inherited genetic conditions.
2. Right-sided tumours present with anaemia and vague symptoms; left-sided tumours present with altered bowel habit and bleeding.
3. Screening uses faecal immunochemical testing followed by colonoscopy for a positive result.
4. Staging uses CT imaging and pathological assessment of the resected specimen.
5. Surgery is the cornerstone of curative treatment, with adjuvant or neoadjuvant therapy for higher-risk disease.



References and Attributions [Series 4]

1. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
2. Feldman, M., Friedman, L. S., & Brandt, L. J. (2020). Sleisenger and Fordtran's Gastrointestinal and Liver Disease (11th ed.). Elsevier.
3. National Institute for Health and Care Excellence (2016). Gastrointestinal Bleeding: Management (CG141). NICE.
4. National Institute for Health and Care Excellence (2011). Colorectal Cancer: Diagnosis and Management (CG131). NICE.

Figures, Plates, Tables, and Boxes

1. Figure 4.1: A clinician performing endoscopic band ligation of a bleeding oesophageal varix. AI-generated using the prompt: "Create a realistic clinical illustration titled Endoscopic Variceal Band Ligation, showing a clinician operating endoscopic equipment while viewing a monitor during a therapeutic upper gastrointestinal endoscopy procedure, endoscopy suite setting. Clean clinical illustration style, no text."
2. Table 4.1: Comparing upper and lower gastrointestinal bleeding. AI-generated using the prompt: "Create a clean reference table titled Comparing Upper and Lower Gastrointestinal Bleeding, listing feature, upper gastrointestinal bleeding, and lower gastrointestinal bleeding. Simple clinical reference table style with outside border."
3. Figure 4.2: A clinician assessing swallowing function in a patient with dysphagia. AI-generated using the prompt: "Create a realistic clinical illustration titled Assessing Dysphagia, showing a clinician observing a seated patient drinking a small amount of water while watching closely for signs of swallowing difficulty, consultation room setting. Clean clinical illustration style, no text."
4. Table 4.2: Colorectal cancer presentation by tumour location. AI-generated using the prompt: "Create a clean reference table titled Colorectal Cancer Presentation by Tumour Location, listing location, typical presentation, and anatomical reason. Simple clinical reference table style with outside border."



Course code: MED 608

Course title: Gastroenterology II

Course credit load: 2 Units

Series 5: Malabsorption Syndromes and Hepatic Failure

- **Part 5.1: Principles of Malabsorption**
 - Sub Part 5.1.1: Physiology of normal digestion and absorption
 - Sub Part 5.1.2: Classification and general presentation of malabsorption
 - Sub Part 5.1.3: Investigation of malabsorption
- **Part 5.2: Specific Malabsorption Syndromes**
 - Sub Part 5.2.1: Coeliac disease
 - Sub Part 5.2.2: Pancreatic exocrine insufficiency and other causes
 - Sub Part 5.2.3: Management of malabsorption syndromes
- **Part 5.3: Acute and Chronic Hepatic Failure**
 - Sub Part 5.3.1: Acute liver failure
 - Sub Part 5.3.2: Chronic liver failure and cirrhosis
 - Sub Part 5.3.3: Complications of cirrhosis: portal hypertension and varices
- **Part 5.4: Complications and Management of Hepatic Failure**
 - Sub Part 5.4.1: Hepatic encephalopathy
 - Sub Part 5.4.2: Ascites and spontaneous bacterial peritonitis
 - Sub Part 5.4.3: Management principles of hepatic failure



Introduction

This final Series closes the course with two genuinely major topics, **malabsorption syndromes**, where the gut fails to adequately absorb the nutrients it is presented with, and **hepatic failure**, the culmination of the chronic liver disease pathway this course has traced from chronic hepatitis through to its most severe, decompensated endpoint. Both topics demand a genuinely systematic, structured approach to reach an accurate diagnosis and provide appropriate, comprehensive management.

The aim of this Series is to equip you with an understanding of the physiology and classification of malabsorption, specific malabsorption syndromes including coeliac disease and pancreatic exocrine insufficiency, and their management, and acute and chronic hepatic failure, portal hypertension, and the specific complications of hepatic encephalopathy, ascites, and spontaneous bacterial peritonitis, alongside the general management principles of hepatic failure.

This Series opens with the **principles of malabsorption**, moves through **specific malabsorption syndromes** and **acute and chronic hepatic failure**, and closes with **complications and management of hepatic failure**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 5.1:** describe the physiology of digestion and absorption and classify malabsorption
- **SLO 5.2:** discuss the investigation of malabsorption
- **SLO 5.3:** describe coeliac disease and pancreatic exocrine insufficiency
- **SLO 5.4:** discuss the management of malabsorption syndromes
- **SLO 5.5:** describe acute and chronic hepatic failure
- **SLO 5.6:** describe portal hypertension and its complications
- **SLO 5.7:** describe hepatic encephalopathy, ascites, and spontaneous bacterial peritonitis
- **SLO 5.8:** discuss the management principles of hepatic failure

5.1 Principles of Malabsorption

5.1.1 physiology of normal digestion and absorption



A coordinated sequence spanning the entire small intestine

Normal digestion and absorption require the coordinated action of pancreatic enzymes breaking down carbohydrate, protein, and fat, bile salts emulsifying fat to allow effective enzymatic digestion, and an intact, healthy small intestinal mucosa with adequate surface area, provided by the characteristic villous architecture, to actually absorb the resulting digested nutrients into the bloodstream.

Different nutrients are preferentially absorbed at specific points along the small intestine, with iron and folate absorbed proximally in the duodenum and jejunum, and vitamin B12 absorbed specifically in the terminal ileum given its dependence on intrinsic factor binding, an anatomical distribution genuinely relevant to understanding why disease affecting a specific segment of small bowel produces a correspondingly specific, predictable pattern of nutrient deficiency.

Fill in the blank: Vitamin B12 is absorbed specifically in the terminal _____ given its dependence on intrinsic factor binding.

5.1.2 classification and general presentation of malabsorption

Failure at any stage of the digestive-absorptive sequence

Malabsorption can result from failure at any stage of the normal process already described, including inadequate digestion, from pancreatic exocrine insufficiency or bile salt deficiency, or inadequate absorption itself, from mucosal disease such as coeliac disease, discussed further in the following Part, or a reduced absorptive surface area following extensive small bowel resection, each producing a broadly similar clinical picture despite the genuinely distinct underlying mechanism.

General presentation includes chronic diarrhoea, often with **steatorrhoea**, pale, bulky, offensive-smelling stool from unabsorbed fat, weight loss despite adequate dietary intake, and specific nutritional deficiency features depending on which particular nutrients are predominantly affected, including anaemia from iron or vitamin B12 deficiency, and bone disease from vitamin D and calcium malabsorption.

Fill in the blank: Steatorrhoea is pale, bulky, offensive-smelling stool from unabsorbed _____.



5.1.3 investigation of malabsorption

Confirming malabsorption, then identifying its specific cause

Initial investigation includes basic blood tests screening for specific nutritional deficiencies, including iron studies, folate, vitamin B12, and vitamin D, alongside markers of inflammation that might suggest an underlying inflammatory bowel disease already discussed in an earlier Series of this course, and **faecal elastase**, a stool test assessing pancreatic exocrine function, helping distinguish pancreatic from small bowel mucosal causes of malabsorption.

Where the underlying cause remains genuinely unclear following this initial evaluation, further specific investigation, including small bowel endoscopy with duodenal biopsy for suspected coeliac disease, discussed in the following Part, or specific imaging of the pancreas, directs a genuinely targeted diagnostic pathway towards the specific underlying cause rather than treating malabsorption as a single, undifferentiated diagnosis.

Fill in the blank: Faecal elastase is a stool test assessing pancreatic exocrine _____.

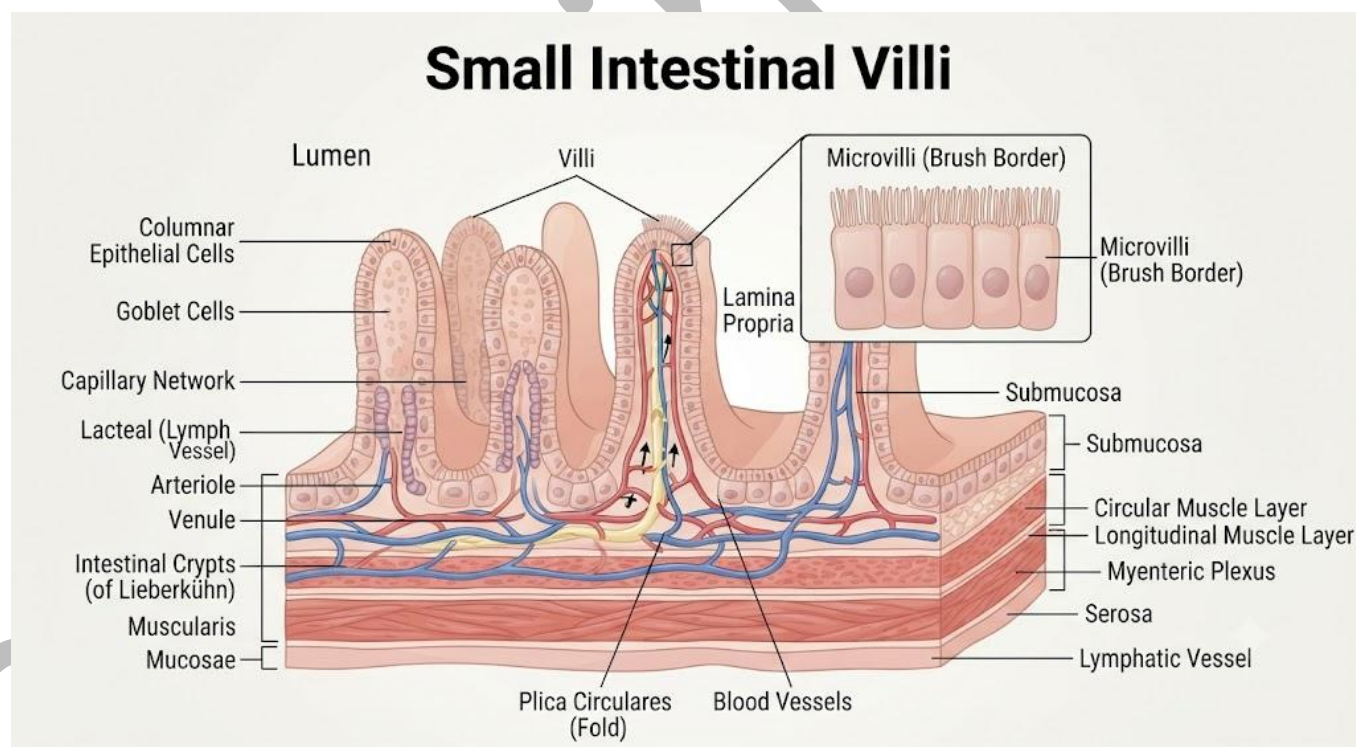


Figure 5.1: A labelled diagram of small intestinal villi and their absorptive surface.



Pilot questions 5.1

- Describe the components required for normal digestion and absorption. (Linked to SLO 5.1)
- Explain why vitamin B12 deficiency specifically suggests terminal ileal disease. (Linked to SLO 5.1)
- Describe the general presentation of malabsorption. (Linked to SLO 5.1)
- Describe steatorrhoea. (Linked to SLO 5.1)
- Describe the role of faecal elastase testing. (Linked to SLO 5.2)

5.2 Specific Malabsorption Syndromes

5.2.1 coeliac disease

An autoimmune reaction to dietary gluten

Coeliac disease, an autoimmune condition triggered by dietary gluten in genetically susceptible individuals, produces characteristic **villous atrophy** within the small intestine, flattening of the normal villous architecture already discussed earlier in this Series, directly reducing the absorptive surface area and producing the malabsorption pattern characteristic of this condition.

Presentation genuinely varies considerably, from classic malabsorption with diarrhoea and weight loss, to genuinely subtle, non-specific symptoms including fatigue and iron deficiency anaemia without prominent gastrointestinal symptoms, to entirely asymptomatic disease identified through screening in patients with a recognised association, such as type 1 diabetes or a first-degree relative with confirmed coeliac disease, and this genuinely wide clinical spectrum means coeliac disease should be actively considered well beyond the classic malabsorptive presentation alone.

Fill in the blank: Coeliac disease produces villous atrophy, flattening of the normal villous

_____.

5.2.2 pancreatic exocrine insufficiency and other causes

Inadequate digestion, rather than inadequate absorption

Pancreatic exocrine insufficiency, most commonly resulting from chronic pancreatitis already discussed in Series 2 of this course, or, less commonly, following pancreatic surgery, produces



malabsorption through inadequate digestive enzyme secretion rather than a primary mucosal absorptive defect, a genuinely important pathophysiological distinction from coeliac disease already discussed, since the small intestinal mucosa itself remains structurally normal in this specific condition.

Other, genuinely less common causes of malabsorption include **small intestinal bacterial overgrowth**, abnormal proliferation of bacteria within the small intestine interfering with normal digestion and absorption, and short bowel syndrome following extensive small bowel resection, and recognising this genuinely wide differential diagnosis, rather than assuming coeliac disease explains every case of malabsorption, is essential to reaching an accurate, appropriately targeted diagnosis.

Fill in the blank: Small intestinal bacterial overgrowth is abnormal proliferation of bacteria within the small intestine interfering with digestion and _____.

5.2.3 management of malabsorption syndromes

Addressing the underlying cause, then correcting deficiencies

Coeliac disease management centres on a genuinely strict, lifelong **gluten-free diet**, which, when adhered to diligently, allows villous architecture to gradually recover and resolves the associated malabsorption in the great majority of appropriately treated patients, while **pancreatic exocrine insufficiency** is managed with pancreatic enzyme replacement therapy taken with meals to support adequate digestion despite the underlying enzyme deficiency.

Regardless of the specific underlying cause, correcting identified nutritional deficiencies, whether iron, folate, vitamin B12, or fat-soluble vitamins, through appropriate supplementation forms an essential, complementary part of comprehensive management, and this Part's structured approach, addressing the underlying cause first before correcting downstream nutritional consequences, closes the malabsorption coverage of this Series before it turns to hepatic failure in the two closing Parts.

Fill in the blank: A gluten-free diet allows villous architecture to gradually _____ and resolves associated malabsorption in most patients.

Table 5.1: Comparing coeliac disease and pancreatic exocrine insufficiency

Feature	Coeliac disease	Pancreatic exocrine insufficiency
Primary defect	Mucosal villous atrophy	Inadequate enzyme secretion
Small bowel mucosa	Structurally abnormal	Structurally normal



Key management	Gluten-free diet	Pancreatic enzyme replacement
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Pilot questions 5.2

1. Describe the pathophysiology of coeliac disease. (Linked to SLO 5.3)
2. Describe the genuinely wide clinical spectrum of coeliac disease presentation. (Linked to SLO 5.3)
3. Describe pancreatic exocrine insufficiency and how it differs pathophysiologically from coeliac disease. (Linked to SLO 5.3)
4. Describe the management of coeliac disease. (Linked to SLO 5.4)
5. Describe the management of pancreatic exocrine insufficiency. (Linked to SLO 5.4)

5.3 Acute and Chronic Hepatic Failure

5.3.1 acute liver failure

Sudden, severe loss of liver function without prior chronic disease

Acute liver failure, sudden, severe loss of liver function in a patient without pre-existing chronic liver disease, most commonly results from paracetamol overdose, viral hepatitis already discussed in Series 3, or drug-induced liver injury, and presents with jaundice, coagulopathy, and, genuinely importantly, hepatic encephalopathy, altered mental status from liver failure discussed in more detail in the following Part of this Series.

Acute liver failure carries a genuinely high mortality without prompt, aggressive supportive management and, in appropriately selected patients, emergency liver transplantation, and identifying the specific underlying cause, particularly paracetamol overdose given the availability of a specific antidote, genuinely, directly influences both prognosis and the immediate treatment approach available.

Fill in the blank: Acute liver failure most commonly results from paracetamol overdose, viral hepatitis, or drug-induced liver _____.



5.3.2 chronic liver failure and cirrhosis

The end-stage endpoint of longstanding chronic liver disease

Cirrhosis, irreversible hepatic fibrosis and architectural distortion representing the end-stage consequence of longstanding chronic liver disease, whatever the underlying cause, whether chronic hepatitis, primary biliary cirrhosis, or another chronic liver condition already discussed across this course, can remain **compensated**, without overt clinical complications, or progress to **decompensated** cirrhosis, with the specific complications discussed later in this Series.

The transition from compensated to decompensated cirrhosis carries genuine prognostic significance, since decompensation, once it occurs, is associated with a considerably worse overall prognosis, and this specific transition, whether triggered by an intercurrent illness, further alcohol exposure, or simply the natural progression of the underlying disease, should always prompt active reassessment of the patient's overall management plan, including consideration of liver transplantation referral in appropriately selected patients.

Fill in the blank: Cirrhosis can remain compensated, without overt clinical complications, or progress to _____ cirrhosis.

5.3.3 complications of cirrhosis: portal hypertension and varices

Elevated pressure throughout the portal venous system

Portal hypertension, elevated pressure within the portal venous system, results directly from the increased resistance to blood flow that cirrhotic architectural distortion produces, and drives the development of **oesophageal varices**, already introduced in the previous Series in the context of upper gastrointestinal bleeding, alongside other complications discussed in the following Part of this Series, splenomegaly, and the formation of other portosystemic venous collaterals attempting to bypass the obstructed hepatic circulation.

Variceal screening, endoscopy performed at the time of cirrhosis diagnosis and periodically thereafter to identify oesophageal varices before they bleed, allows prophylactic treatment, typically with a non-selective beta-blocker or endoscopic band ligation, to genuinely reduce the risk of a first variceal bleeding episode, a preventive approach directly complementing the acute variceal bleeding management already discussed in the previous Series.

Fill in the blank: Variceal screening allows prophylactic treatment to genuinely reduce the risk of a first variceal bleeding _____.



Table 5.2: Comparing compensated and decompensated cirrhosis

Feature	Compensated cirrhosis	Decompensated cirrhosis
Clinical complications	Absent	Present, e.g. ascites, encephalopathy
Overall prognosis	Better	Considerably worse
Transplant referral	Not typically urgent	Should be actively considered

Pilot questions 5.3

1. List the common causes of acute liver failure. (Linked to SLO 5.5)
2. Describe the classic presentation of acute liver failure. (Linked to SLO 5.5)
3. Distinguish compensated from decompensated cirrhosis. (Linked to SLO 5.5)
4. Describe portal hypertension and its underlying cause. (Linked to SLO 5.6)
5. Describe the purpose and approach of variceal screening. (Linked to SLO 5.6)

5.4 Complications and Management of Hepatic Failure

5.4.1 hepatic encephalopathy

Toxins normally cleared by the liver reaching the brain

Hepatic encephalopathy, altered mental status resulting from the failing liver's inability to adequately clear neurotoxic substances, most notably ammonia, produced during normal protein metabolism, presents with a genuinely wide spectrum ranging from subtle cognitive change and altered sleep pattern to genuine confusion, drowsiness, and, in the most severe cases, coma, mirroring in principle, though through a genuinely distinct underlying mechanism, the coma already discussed in an earlier course of this programme.

Asterixis, a characteristic flapping tremor elicited by asking the patient to hold their arms outstretched with wrists extended, is a specific and genuinely valuable clinical sign supporting the diagnosis, and identifying and treating a **precipitating factor**, including infection, gastrointestinal bleeding, constipation, or electrolyte disturbance, is genuinely essential, since hepatic encephalopathy frequently reflects an acute decompensating trigger superimposed on chronic liver disease rather than a purely spontaneous deterioration.



Fill in the blank: Asterixis is a characteristic flapping tremor elicited by asking the patient to hold their arms outstretched with wrists _____.

5.4.2 ascites and spontaneous bacterial peritonitis

Fluid accumulation, and the genuine risk of its infection

Ascites, fluid accumulation within the peritoneal cavity, results from the combination of portal hypertension already discussed earlier in this Series and low serum albumin, itself reflecting reduced hepatic synthetic function, presenting with progressive abdominal distension and discomfort, and management combines dietary sodium restriction with diuretic therapy, and, for significant, symptomatic ascites, therapeutic paracentesis, draining the accumulated fluid directly.

Spontaneous bacterial peritonitis, infection of ascitic fluid without an identifiable primary intra-abdominal source, is a genuinely serious complication requiring active consideration in any patient with ascites presenting with fever, abdominal pain, or unexplained clinical deterioration, and diagnosis relies on ascitic fluid analysis, with prompt antibiotic treatment genuinely essential given the significant associated mortality if this specific infection is missed or treated late.

Fill in the blank: Spontaneous bacterial peritonitis is infection of ascitic fluid without an identifiable primary intra-abdominal _____.

5.4.3 management principles of hepatic failure

A genuinely comprehensive, multidisciplinary approach closing this course

General management principles for hepatic failure combine treating the underlying cause where genuinely identifiable and treatable, actively managing the specific complications already discussed throughout this Series, encephalopathy, ascites, and variceal bleeding risk, and comprehensive nutritional support, since malnutrition genuinely worsens outcomes in chronic liver disease and deserves proactive, rather than reactive, attention throughout the patient's care.

Liver transplantation, definitive treatment for appropriately selected patients with irreversible, decompensated liver failure, offers genuine cure for the underlying liver disease, though it demands careful, structured patient selection and lifelong post-transplant immunosuppression and follow-up, and this Series, and the entire course, closes on the same theme of coordinated, structured, individualised clinical decision-making that has genuinely run throughout this whole programme of gastroenterological study.



Fill in the blank: Liver transplantation demands careful patient selection and lifelong post-transplant _____ and follow-up.



Figure 5.2: A clinician examining a patient's abdomen for shifting dullness suggesting ascites.

Table 5.3: Comparing key complications of hepatic failure

Complication	Key clinical feature	Immediate management focus
Hepatic encephalopathy	Altered mental status, asterixis	Identify and treat precipitating factor
Ascites	Progressive abdominal distension	Sodium restriction, diuretics, paracentesis
Spontaneous bacterial peritonitis	Fever, abdominal pain, deterioration	Prompt antibiotic treatment



Pilot questions 5.4

1. Describe hepatic encephalopathy and its underlying mechanism. (Linked to SLO 5.7)
2. Describe asterixis and its clinical value. (Linked to SLO 5.7)
3. List precipitating factors for hepatic encephalopathy. (Linked to SLO 5.7)
4. Describe ascites and its underlying causes. (Linked to SLO 5.7)
5. Describe spontaneous bacterial peritonitis and its management. (Linked to SLO 5.8)

Series Summary

This final Series covered malabsorption syndromes and hepatic failure. Part 5.1 covered the physiology, classification, and investigation of malabsorption, while Part 5.2 turned to coeliac disease, pancreatic exocrine insufficiency, and their management. Part 5.3 covered acute and chronic hepatic failure and portal hypertension, and Part 5.4 closed with hepatic encephalopathy, ascites, spontaneous bacterial peritonitis, and the general management principles of hepatic failure.

You should now be able to describe and manage malabsorption syndromes and hepatic failure across all four Parts of this Series, meeting the outcomes set for this Series. Across the full course, Series 1 covered peptic ulcer disease, Series 2 covered cholecystitis, pancreatitis, and diagnostic procedures, Series 3 covered chronic liver disease and inflammatory bowel disease, Series 4 covered gastrointestinal bleeding and malignancies, and this Series has closed the course with malabsorption syndromes and hepatic failure, together forming a comprehensive foundation in Gastroenterology II.

Glossary of Terms

1. **Acute liver failure:** sudden, severe loss of liver function without pre-existing chronic liver disease.
2. **Ascites:** fluid accumulation within the peritoneal cavity from portal hypertension and low albumin.
3. **Asterixis:** a characteristic flapping tremor seen in hepatic encephalopathy.
4. **Cirrhosis:** irreversible hepatic fibrosis and architectural distortion, the end-stage of chronic liver disease.



5. **Coeliac disease:** an autoimmune reaction to gluten producing small intestinal villous atrophy.
6. **Hepatic encephalopathy:** altered mental status from the failing liver's inability to clear neurotoxins.
7. **Malabsorption:** failure of adequate nutrient digestion or absorption.
8. **Pancreatic exocrine insufficiency:** inadequate pancreatic enzyme secretion causing malabsorption.
9. **Portal hypertension:** elevated pressure within the portal venous system from cirrhotic resistance.
10. **Spontaneous bacterial peritonitis:** infection of ascitic fluid without an identifiable primary source.
11. **Steatorrhea:** pale, bulky, offensive-smelling stool from unabsorbed fat.
12. **Villous atrophy:** flattening of the normal small intestinal villous architecture.

Concept Check Questions [Series 5]

Objective questions

- Vitamin B12 is absorbed specifically in the terminal _____ given its dependence on intrinsic factor binding. (Linked to SLO 5.1)
- Coeliac disease produces villous atrophy, flattening of the normal villous _____. (Linked to SLO 5.3)
- Acute liver failure most commonly results from paracetamol overdose, viral hepatitis, or drug-induced liver _____. (Linked to SLO 5.5)
- Asterix is a characteristic flapping tremor elicited by asking the patient to hold their arms outstretched with wrists _____. (Linked to SLO 5.7)
- Spontaneous bacterial peritonitis is infection of ascitic fluid without an identifiable primary intra-abdominal _____. (Linked to SLO 5.7)

Short-answer questions

1. Describe steatorrhea. (Linked to SLO 5.1)
2. Describe the genuinely wide clinical spectrum of coeliac disease presentation. (Linked to SLO 5.3)
3. Distinguish compensated from decompensated cirrhosis. (Linked to SLO 5.5)
4. List precipitating factors for hepatic encephalopathy. (Linked to SLO 5.7)
5. Describe ascites and its underlying causes. (Linked to SLO 5.7)



Long-answer questions

6. Discuss the physiology, classification, and investigation of malabsorption. (Linked to SLO 5.1, 5.2)
7. Describe coeliac disease and pancreatic exocrine insufficiency and their management. (Linked to SLO 5.3, 5.4)
8. Discuss acute and chronic hepatic failure and portal hypertension. (Linked to SLO 5.5, 5.6)
9. Describe hepatic encephalopathy, ascites, and spontaneous bacterial peritonitis. (Linked to SLO 5.7)
10. Discuss the management principles of hepatic failure. (Linked to SLO 5.8)

Answers to Concept Check Questions [Series 5]

Objective questions

11. Ileum.
12. Architecture.
13. Injury.
14. Extended.
15. Source.

Short-answer questions

16. Steatorrhea is pale, bulky, offensive-smelling stool from unabsorbed fat.
17. Presentation ranges from classic malabsorption to subtle anaemia to entirely asymptomatic disease identified on screening.
18. Compensated cirrhosis has no overt complications, while decompensated cirrhosis presents with specific complications and worse prognosis.
19. Precipitating factors include infection, gastrointestinal bleeding, constipation, and electrolyte disturbance.
20. Ascites is peritoneal fluid accumulation from portal hypertension and low serum albumin.

Long-answer questions

21. **Basic response:** Normal absorption requires enzymes, bile salts, and healthy mucosa; malabsorption results from failed digestion or absorption; investigation combines nutritional screening with faecal elastase and targeted testing.



Value-added response: A value-added response notes that specific nutrients are absorbed at specific small bowel sites, producing predictable deficiency patterns.

22. **Basic response:** Coeliac disease is autoimmune gluten-triggered villous atrophy, managed with a gluten-free diet; pancreatic exocrine insufficiency is inadequate enzyme secretion, managed with enzyme replacement.

Value-added response: A value-added response notes that coeliac disease presentation genuinely varies from classic malabsorption to entirely asymptomatic disease.

23. **Basic response:** Acute liver failure is sudden severe dysfunction, commonly from paracetamol overdose; chronic failure culminates in cirrhosis, compensated or decompensated; portal hypertension drives varices and other complications.

Value-added response: A value-added response notes that variceal screening allows prophylactic treatment to reduce first bleeding risk.

24. **Basic response:** Hepatic encephalopathy is altered mental status from toxin accumulation, with asterixis a valuable sign; ascites is peritoneal fluid accumulation; spontaneous bacterial peritonitis is ascitic fluid infection requiring prompt antibiotics.

Value-added response: A value-added response notes that hepatic encephalopathy frequently reflects an acute precipitating trigger requiring active identification.

25. **Basic response:** Management treats the underlying cause, addresses specific complications, and provides nutritional support, with liver transplantation offering definitive cure for appropriately selected patients.

Value-added response: A value-added response notes that liver transplantation demands lifelong post-transplant immunosuppression and follow-up.

Answers to Pilot Questions [Series 5]

Part 5.1

1. Digestion and absorption require pancreatic enzymes, bile salts, and healthy intestinal mucosa with adequate surface area.



2. Vitamin B12 absorption specifically requires the terminal ileum given its dependence on intrinsic factor binding there.
3. Malabsorption presents with chronic diarrhoea, steatorrhoea, weight loss, and specific nutritional deficiency features.
4. Steatorrhoea is pale, bulky, offensive-smelling stool from unabsorbed fat.
5. Faecal elastase assesses pancreatic exocrine function, distinguishing pancreatic from mucosal causes.

Part 5.2

6. Coeliac disease is an autoimmune reaction to gluten producing villous atrophy and reduced absorptive surface area.
7. Presentation ranges from classic malabsorption to subtle anaemia to entirely asymptomatic disease on screening.
8. Pancreatic exocrine insufficiency causes inadequate enzyme secretion with structurally normal mucosa, unlike coeliac disease.
9. Coeliac disease is managed with a strict, lifelong gluten-free diet.
10. Pancreatic exocrine insufficiency is managed with pancreatic enzyme replacement therapy taken with meals.

Part 5.3

11. Common causes include paracetamol overdose, viral hepatitis, and drug-induced liver injury.
12. Presentation includes jaundice, coagulopathy, and hepatic encephalopathy.
13. Compensated cirrhosis has no overt complications, while decompensated cirrhosis presents with specific complications and worse prognosis.
14. Portal hypertension is elevated portal venous pressure from cirrhotic resistance to blood flow.
15. Variceal screening uses endoscopy to identify varices before bleeding, allowing prophylactic treatment.

Part 5.4

1. Hepatic encephalopathy is altered mental status from failure to clear neurotoxins such as ammonia.
2. Asterix is a flapping tremor, a specific and valuable clinical sign supporting the diagnosis.
3. Precipitating factors include infection, gastrointestinal bleeding, constipation, and electrolyte disturbance.
4. Ascites is peritoneal fluid accumulation from portal hypertension and low serum albumin.



5. Spontaneous bacterial peritonitis is ascitic fluid infection, managed with prompt antibiotic treatment.

References and Attributions [Series 5]

1. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
2. Feldman, M., Friedman, L. S., & Brandt, L. J. (2020). Sleisenger and Fordtran's Gastrointestinal and Liver Disease (11th ed.). Elsevier.
3. European Association for the Study of the Liver (2018). Clinical Practice Guidelines for the Management of Patients with Decompensated Cirrhosis. Journal of Hepatology.
4. National Institute for Health and Care Excellence (2015). Coeliac Disease: Recognition, Assessment and Management (NG20). NICE.

Figures, Plates, Tables, and Boxes

1. Figure 5.1: A labelled diagram of small intestinal villi and their absorptive surface. AI-generated using the prompt: "Create a clean, accurate labelled anatomical diagram titled Small Intestinal Villi, showing a cross-section of the small intestinal wall with finger-like villi, microvilli, and the underlying blood supply clearly labelled. Educational anatomical diagram style, no photographic content."
2. Table 5.1: Comparing coeliac disease and pancreatic exocrine insufficiency. AI-generated using the prompt: "Create a clean reference table titled Comparing Coeliac Disease and Pancreatic Exocrine Insufficiency, listing feature, coeliac disease, and pancreatic exocrine insufficiency. Simple clinical reference table style with outside border."
3. Table 5.2: Comparing compensated and decompensated cirrhosis. AI-generated using the prompt: "Create a clean reference table titled Comparing Compensated and Decompensated Cirrhosis, listing feature, compensated cirrhosis, and decompensated cirrhosis. Simple clinical reference table style with outside border."
4. Figure 5.2: A clinician examining a patient's abdomen for shifting dullness suggesting ascites. AI-generated using the prompt: "Create a realistic clinical illustration titled Abdominal Examination for Ascites, showing a clinician percussing and palpating a



patient's distended abdomen while the patient lies on an examination table, clinical examination room setting. Clean clinical illustration style, no text."

5. Table 5.3: Comparing key complications of hepatic failure. AI-generated using the prompt: "Create a clean reference table titled Comparing Key Complications of Hepatic Failure, listing complication, key clinical feature, and immediate management focus. Simple clinical reference table style with outside border."

