MADE SIMPLE SERIES

DDSEP 9 MADE SIMPLE

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Hepatology & Gastroenterology
Comprehensive Review Guide

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ESOPHAGEAL DISORDERS

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Structured for rapid review, clinical pattern recognition, and MCQ recall

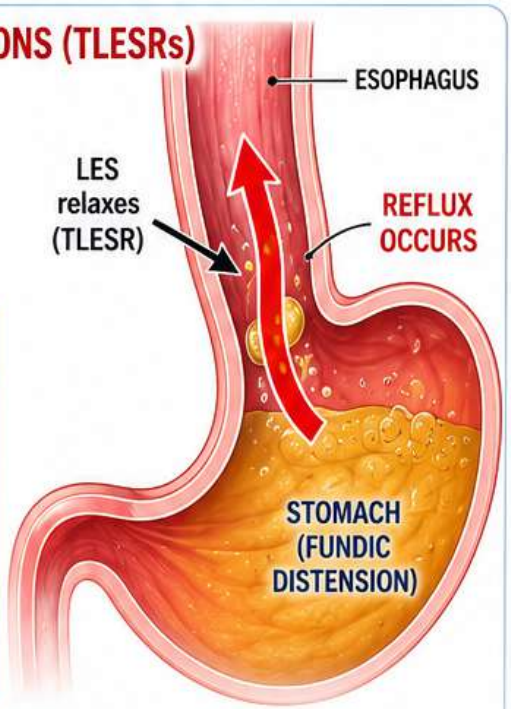
SECTION I – EXAM PEARL NOTES

1. GASTROESOPHAGEAL REFLUX: CORE PATHOPHYSIOLOGY

1.1 TRANSIENT LOWER ESOPHAGEAL SPHINCTER RELAXATIONS (TLESRs)

Transient lower esophageal sphincter relaxations (TLESRs) are physiologic, swallow-independent LES relaxations that allow venting of swallowed air from the stomach.

They are triggered mainly by proximal gastric or fundic distension and represent the **most frequent mechanism** of gastroesophageal reflux.



↑ FACTORS ASSOCIATED WITH INCREASED TLESRs

- Postprandial gastric distension
- Obesity
- Obstructive sleep apnea
- Hiatus hernia
- GERD in some patients

↓ FACTORS ASSOCIATED WITH REDUCED TLESRs

- Baclofen, a GABA-B receptor agonist
- Deep sleep
- Disorders with impaired LES relaxation, such as esophagogastric junction outflow obstruction

! Gastric acid production **does not** determine TLESR frequency.

DDSEP Q1–Q2 MEMORY: OSA opens; baclofen blocks.
Meal distends the fundus → TLESRs increase → reflux occurs.

1.2 POSTPRANDIAL REFLUX

Postprandial gastric distension increases the number of TLESRs.

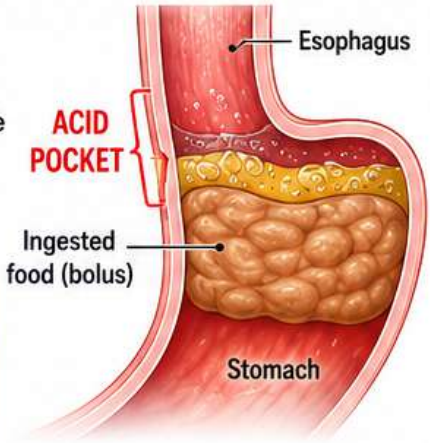
A pH study showing the following suggests a predominantly postprandial TLESR-driven reflux pattern:

- ✓ Reflux episodes clustered after meals
- ✓ Little or no nocturnal reflux
- ✓ Normal endoscopy
- ✓ No major hiatus hernia

Typical GERD is generally an **abnormal reflux problem** rather than a state of excessive gastric acid production.

1.3 THE ACID POCKET

After a meal, newly secreted gastric acid forms an **unbuffered layer** above the ingested food near the gastroesophageal junction. This is called the **acid pocket.**



DURING A TLESR

Acid pocket loads the reflux →

✓ THE ACID POCKET:

- Lies close to the gastroesophageal junction
- May extend into a hiatus hernia
- May extend into the distal esophagus
- Increases the volume and acidity of refluxate during a TLESR

✗ THE ACID POCKET DOES NOT:

- Increase TLESR frequency
- Increase gastric acid production
- Enlarge a hiatus hernia
- Delay gastric emptying

Critical distinction: gastric distension increases the **number of reflux events,** whereas the acid pocket increases the **acid content** of each event.

DDSEP Q3 MEMORY: The acid pocket **loads** the reflux; it **does not** open the sphincter.

1.4 ESOPHAGEAL PERCEPTION

Symptom severity is influenced by both reflux burden and central perception.

INCREASED PERCEPTION
occurs with

- Anxiety
- Psychological stress
- Sleep deprivation
- Hypervigilance
- Affective disorders

REDUCED PERCEPTION
occurs with

- Older age
- Barrett's esophagus
- Diabetes mellitus
- Sleep

Patients with anxiety may report marked symptoms despite modest reflux burden.

Older patients and patients with Barrett's esophagus may have advanced mucosal disease with few symptoms.

DDSEP Q4 MEMORY: Anxiety amplifies; age, Barrett's, diabetes and sleep blunt.

1.5 SALIVA AND ESOPHAGEAL ACID CLEARANCE

Esophageal clearance has two components:

1) VOLUME CLEARANCE

Peristalsis removes most of the refluxate.

2) CHEMICAL CLEARANCE

Salivary bicarbonate neutralizes residual acid.

Reduced saliva production, including after head and neck radiotherapy, prolongs mucosal acidification and increases the risk of erosive esophagitis.

Reduced salivary flow does not directly cause

- Increased gastric acid volume
- Ineffective motility
- Esophageal stricture
- Barrett's esophagus

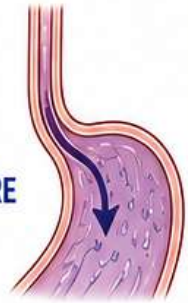
DDSEP Q7 MEMORY: Peristalsis clears the volume; saliva clears the acid.

2. DRUGS AND REFLUX

2.1 THEOPHYLLINE

Theophylline reduces **basal LES pressure** and can promote symptomatic reflux.

↓ LES PRESSURE



2.2 BACLOFEN

Baclofen is a GABA-B receptor agonist that **reduces TLESR frequency and reflux episodes** and may help selected patients with troublesome regurgitation.

MECHANISM:
- ↓ TLESR frequency
- ↓ Reflux episodes

2.3 PILL INJURY VERSUS REFLUX

Alendronate, doxycycline and NSAIDs can cause **direct mucosal injury** if retained in the esophagus.

They **do not** necessarily increase the tendency for gastroesophageal reflux.

2.4 BETA-ADRENERGIC DRUGS

- Beta-blockers **do not** significantly increase reflux.
- Beta-adrenergic agonists **may promote** reflux.

Beta-blockers → No significant effect

Beta-agonists → May promote reflux

DDSEP Q6 MEMORY: Theophylline **loosens** the LES; **pills may injure** without causing reflux.

3. RISK FACTORS FOR COMPLICATED GERD

Factors associated with **erosive esophagitis** or **Barrett's esophagus** include:

Male sex | Obesity | Increasing age | Hiatus hernia | Smoking | Chronic GERD

- Hiatus hernia increases esophageal acid exposure, particularly supine reflux burden.
- Older patients may have advanced reflux disease despite limited symptoms because perception declines with age.

HELICOBACTER PYLORI

↓ *H. pylori* infection, especially when associated with atrophic gastritis and reduced acid production, is associated with a **lower risk** of Barrett's esophagus in observational studies.

↑ Eradication **may increase reflux symptoms** in some patients but has not been shown to increase reflux complications.

✓ This association is **not** a reason to avoid indicated H. pylori eradication.

DDSEP Q8 MEMORY: **Male, obese, older and herniated** means higher complication risk.

4. CHEST PAIN AND GERD

Heavy or pressure-like retrosternal pain in a patient with **cardiovascular risk factors** must be considered **cardiac** until appropriately evaluated.

CARDIAC RED FLAGS IN THE GERD CLINIC

- Middle or older age
- Male sex
- Obesity
- Hypertension or other cardiovascular risks
- Heavy or pressure-like pain
- Atypical relationship to meals
- No typical regurgitation

Improvement with antacid or belching is **nonspecific** and does not exclude myocardial ischemia.

A normal endoscopy does not exclude cardiac disease, nonerosive reflux disease, esophageal hypersensitivity, or motility disorders.

After cardiac disease is excluded, further evaluation may include **prolonged reflux monitoring** and **high-resolution manometry**.

Barium esophagram has low yield in isolated chest pain without dysphagia.

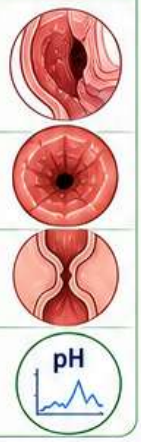
DDSEP Q5 MEMORY: **Chest pressure plus cardiac risk:** clear the heart first.

5. OBJECTIVE DIAGNOSIS OF GERD

5.1 FINDINGS THAT ESTABLISH GERD

CONCLUSIVE OBJECTIVE EVIDENCE may include:

- Barrett's esophagus with intestinal metaplasia in an appropriate visible segment
- Severe erosive esophagitis
- Peptic stricture
- Abnormal ambulatory acid exposure



SUPPORTIVE BUT NON-DIAGNOSTIC FINDINGS

These findings support GERD but do not establish the diagnosis.

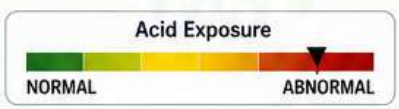
- Hiatus hernia
- Ineffective esophageal motility (IEM)
- Typical heartburn
- Bile in the gastric fundus

Bile may normally be present in the stomach and **does not** establish GERD.

DDSEP Q9 MEMORY: **Barrett's proves; hernia and IEM support; heartburn suggests.**

5.2 ABNORMAL ACID EXPOSURE PREDICTS PPI RESPONSE

Among patients with nonerosive reflux disease, abnormal acid exposure on ambulatory pH monitoring is the **strongest predictor** of symptomatic benefit from PPI therapy.



Less reliable predictors include:

- Papillary elongation
- Basal cell hyperplasia
- Reflux seen on barium swallow
- Regurgitation alone
- Absence of a hiatus hernia

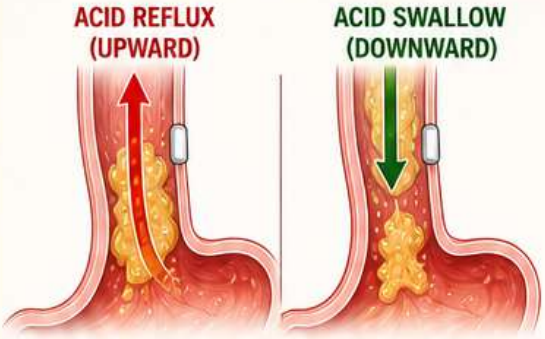
Regurgitation responds less reliably than heartburn because **acid suppression** changes refluxate acidity but **may not prevent reflux events**.

DDSEP Q10 MEMORY: **Abnormal pH predicts PPI response.**

6. AMBULATORY REFLUX MONITORING

6.1 WIRELESS pH MONITORING PITFALLS

A wireless pH capsule has a single recording site and cannot always distinguish acid moving upward during reflux from acidic food or beverages moving downward during swallowing.



One sensor cannot distinguish.

Acidic oral intake may therefore **falsely elevate acid exposure time.** Meal periods should be identified using an accurate diary and excluded or interpreted carefully.

Alkaline reflux is **not detected** by pH-only monitoring.

Antacids may reduce acid exposure.

Esophageal shortening **does not displace** a wall-attached wireless capsule.

Catheter-based sensors **may move** into the stomach during shortening.

DDSEP Q11 MEMORY: One pH sensor cannot distinguish an acid swallow from acid reflux.

6.2 PROVEN VERSUS UNPROVEN GERD

UNPROVEN GERD

No previous conclusive evidence such as severe erosive esophagitis, Barrett's esophagus, peptic stricture, or a previous abnormal reflux study.

Persistent symptoms with unproven GERD should be assessed with reflux monitoring **OFF PPI THERAPY.**

PROVEN GERD

Previous objective evidence of GERD exists (e.g., severe erosive esophagitis, Barrett's esophagus, peptic stricture, or abnormal reflux study).

Persistent symptoms despite optimized treatment should be assessed using pH-impedance monitoring **ON PPI THERAPY.**

DDSEP Q12 & Q14 MEMORY: Unproven GERD: test **OFF PPI.** Proven GERD with persistent symptoms: test **ON PPI.**

6.3 PERSISTENT REGURGITATION IN PROVEN GERD

In a patient with prior LA grade C esophagitis that healed on PPI but with continued regurgitation, **pH-impedance monitoring ON THERAPY** evaluates:

- Residual acid reflux
- Weakly acidic reflux
- Non-acid reflux
- Excessive reflux episodes
- Symptom–reflux association
- Rumination
- Supragastric belching

If reflux testing is **negative**, consider **rumination syndrome**, supragastric belching, gastroparesis, or functional symptoms.

6.4 EXTRAESOPHAGEAL SYMPTOMS

Cough, hoarseness and globus have many potential causes.

In patients without typical heartburn or regurgitation, an empirical PPI response has poor diagnostic accuracy.

When reflux remains unproven, prolonged ambulatory reflux monitoring should be performed **OFF THERAPY.**

Antireflux surgery **should not** be offered for isolated extraesophageal symptoms without convincing objective evidence.

DDSEP Q10 MEMORY: Abnormal pH predicts PPI response.

7. PPI THERAPY

7.1 TIMING

Conventional PPIs work best when taken **before meals**, generally 30–60 minutes before breakfast for once-daily therapy and before breakfast and the evening meal for twice-daily therapy.

Taking a PPI after meals reduces its effectiveness.

7.2 PREDICTORS OF PPI RESPONSE

PREDICTORS OF BETTER PPI RESPONSE

- Abnormal acid exposure
- Erosive esophagitis
- Correct administration before meals
- Typical heartburn

PREDICTORS OF SUBOPTIMAL RESPONSE

- Irritable bowel syndrome
- Disorders of gut–brain interaction
- Anxiety
- Depression
- Visceral hypersensitivity
- Hypervigilance
- Predominant regurgitation
- Cough, hoarseness or globus

DDSEP Q15 MEMORY: Objective reflux predicts response; functional overlap predicts persistence.

7.3 REBOUND ACID HYPERSECRETION

Abrupt withdrawal after long-term PPI use may cause transient severe heartburn from **rebound acid hypersecretion**.

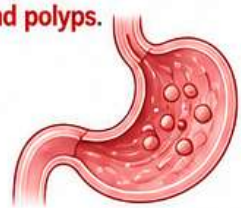
This does not necessarily justify indefinite PPI therapy.

- Gradual dose reduction
- On-demand PPI use
- Temporary H2-receptor antagonist
- Lifestyle measures

DDSEP Q31 MEMORY: Stopping suddenly may make acid bounce back—**step down** rather than automatically **stepping up**.

7.4 LONG-TERM PPI ASSOCIATIONS

The most consistent recognized association among common examination options is the development of **fundic gland polyps**.



- PPI-associated fundic gland polyps are generally small, benign, located in the fundus or body, and non-dysplastic in most sporadic cases.
- Associations with dementia, fracture, chronic kidney disease and iron deficiency are less consistent and may be affected by confounding.

DDSEP Q25 MEMORY: The clearest chronic PPI footprint is **fundic gland polyps**.

7.5 PPI DISCONTINUATION AFTER MALLORY-WEISS TEAR

A Mallory-Weiss tear caused by isolated forceful vomiting generally heals spontaneously.

- In an asymptomatic patient
- Stable hemoglobin
- No active bleeding
- No other indication for acid suppression

PPI therapy can be discontinued.

Repeat endoscopy is not routinely required.

DDSEP Q32 MEMORY: Vomiting stopped, tear healed, no indication—**stop the PPI**.

8. LIFESTYLE MANAGEMENT

Nocturnal reflux is promoted by large evening meals, late meals, lying flat soon after eating, and travel-related change in eating pattern.

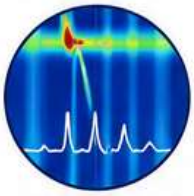
- Avoid eating within approximately three hours of bedtime
- Avoid heavy evening meals
- Elevate the head of the bed
- Sleep on an incline
- Consider left lateral sleeping position

Patients who are otherwise controlled on once-daily PPI do not automatically need continuous twice-daily therapy.

DDSEP Q27 MEMORY: Late meal plus lying flat equals **nocturnal reflux**.

9. ANTIREFLUX SURGERY

9.1 PREOPERATIVE EVALUATION



HIGH-RESOLUTION MANOMETRY

Before fundoplication, high-resolution manometry should be performed to:

- Exclude achalasia or another major motor disorder
- Assess peristaltic function and contraction reserve
- Predict postoperative dysphagia risk

DDSEP Q28 MEMORY:
Before wrapping the LES, prove that the esophagus can push.

9.2 DYSPHAGIA AFTER FUNDOPLICATION

EARLY DYSPHAGIA

- Common
- Usually related to edema
- Often resolves spontaneously

LATE OR PERSISTENT DYSPHAGIA

- Tight wrap
- Slipped wrap
- Herniated wrap
- Recurrent hiatus hernia
- Esophageal hypomotility
- Recurrent stricture
- Recurrent complicated reflux

Risk factors include preoperative dysphagia and absent contraction reserve.

DDSEP Q16 MEMORY: Fundoplication controls reflux but may obstruct swallowing.

9.3 EVALUATION OF NEW SYMPTOMS AFTER FUNDOPLICATION

New swallowing-associated chest discomfort or dysphagia after surgery should initially be assessed with **UPPER ENDOSCOPY.**

- Wrap position
- Recurrent hiatus hernia
- Slipped fundoplication
- Stricture
- Recurrent esophagitis

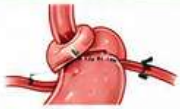
BARIUM ESOPHAGRAM complements endoscopy by defining anatomy.

MANOMETRY is performed when endoscopy does not explain symptoms.

DDSEP Q17 MEMORY:
Symptoms after fundoplication: inspect the wrap first.

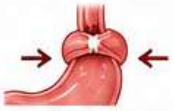
9.4 HERNIATED FUNDOPLICATION

A wrap found above the diaphragmatic hiatus represents transdiaphragmatic migration or a herniated fundoplication.



HERNIATED WRAP: wrap above the diaphragm

SLIPPED WRAP: wrap around proximal stomach instead of the GEJ



TIGHT WRAP: resistance to scope passage

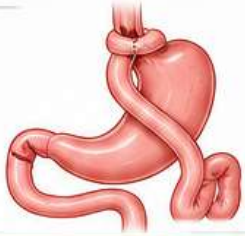
COMPLETE DISRUPTION: fundoplication folds absent

DDSEP Q47 MEMORY:
Wrap above the diaphragm equals herniated fundoplication.

9.5 SEVERE GERD IN MORBID OBESITY

For severe GERD with medically complicated obesity and a large hiatus hernia, **ROUX-EN-Y GASTRIC BYPASS WITH HERNIA REPAIR** is generally the most effective operative strategy among the listed options.

- Weight loss
- Improvement in diabetes and metabolic disease
- Reduced acid and bile exposure
- Lower recurrence risk than fundoplication alone in severe obesity



Sleeve gastrectomy may worsen or produce reflux.

DDSEP Q29 MEMORY: Morbid obesity plus severe GERD: bypass the reflux and the weight.

9.6 DELAYED GASTRIC EMPTYING AFTER REDO SURGERY

Vagal injury is a recognized complication of antireflux surgery, especially redo procedures.

- Impaired gastric accommodation
- ↓ Reduced antral contractility
- ↓ Impaired pyloric relaxation
- ↓ Delayed gastric emptying
- ↓ Nausea and vomiting

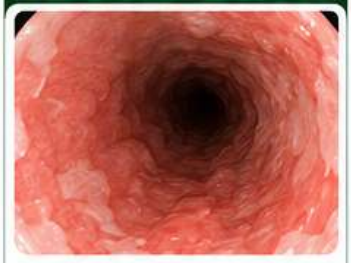
DDSEP Q30 MEMORY: Redo fundoplication plus gastroparesis: suspect vagal injury.

10. BARRETT'S ESOPHAGUS

10.1 DIAGNOSIS

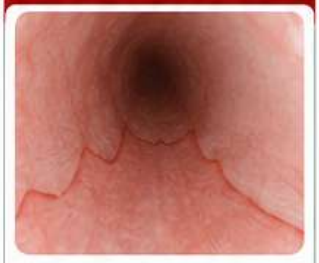
Barrett's esophagus is identified by **visible columnar or salmon-colored** mucosa in the distal esophagus with **histological intestinal metaplasia** in an appropriate segment.

BARRETT'S ESOPHAGUS



Visible columnar mucosa with intestinal metaplasia

NOT BARRETT'S



Irregular Z-line with normal biopsies is **NOT** Barrett's

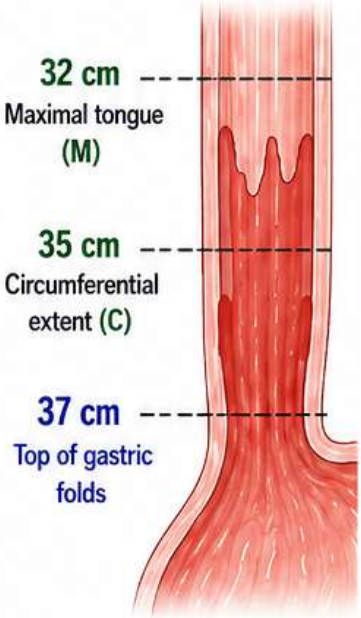
An irregular Z-line with normal biopsies is **not** Barrett's esophagus and should **not be ablated.**

DDSEP Q36 MEMORY:
C is continuous; M is maximum contiguous.

10.2 PRAGUE CLASSIFICATION

The Prague classification records C, the circumferential extent, and M, the maximal contiguous extent.

Measurements begin at the top of the gastric folds.



Example:
Gastric folds at 37 cm, circumferential mucosa to 35 cm and maximal tongue to 32 cm gives **C2M5.**

A separate proximal island is documented separately and is **not included in M.**

DDSEP Q36 MEMORY:
C is continuous; M is maximum contiguous.

10.3 LOW-GRADE DYSPLASIA

The diagnosis of low-grade dysplasia has substantial interobserver variability. The first step is **confirmation by an expert gastrointestinal pathologist.**

Active reflux inflammation may mimic low-grade or indefinite dysplasia.

If inflammation may have affected interpretation:

- Optimize acid suppression
- Repeat high-quality endoscopy and systematic biopsies
- Obtain expert pathology review

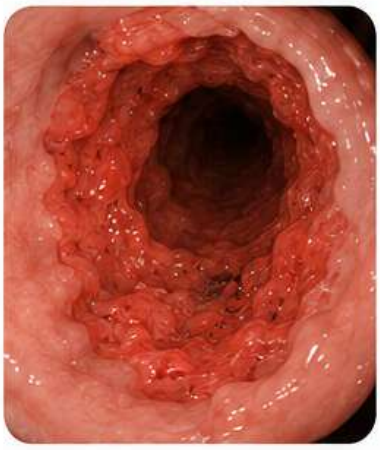
Confirmed low-grade dysplasia may be managed with **endoscopic eradication therapy** or **close surveillance** in selected patients.

DDSEP Q18 & Q37 MEMORY:
Heal inflammation, repeat and confirm. **Confirm before ablation.**

10.4 VISIBLE BARRETT'S LESIONS

Visible lesions require **endoscopic resection** for histological staging.

- Depression
- Ulceration
- Loss of normal mucosal pattern
- Irregular vascular pattern



Depressed ulcerated lesions have the **greatest risk** of submucosal invasion and lymph-node metastasis and are **least likely** to be cured by endoscopic therapy alone.

DDSEP Q38 MEMORY:
Depressed and ulcerated means **deeper and more dangerous.**

10.5 HIGH-GRADE DYSPLASIA

When a visible lesion containing **high-grade dysplasia** is completely removed by EMR, the remaining Barrett's segment should be **ablated** because of the risk of synchronous lesions, metachronous lesions and recurrent dysplasia.

1. Resect visible abnormalities.

2. Ablate the remaining flat Barrett's epithelium.

DDSEP Q39 MEMORY:
Resect what is visible; ablate what remains.

10.6 RFA FAILURE

Persistent Barrett's epithelium after several adequate RFA sessions may be treated with salvage **cryotherapy**.

Photodynamic therapy is now **rarely used** because of adverse effects.

Esophagectomy is not indicated for persistent metaplasia alone when dysplasia has been eradicated.

CRYOTHERAPY

PHOTODYNAMIC THERAPY

DDSEP Q40 MEMORY:
RFA failure: change the energy—consider cryotherapy.

10.7 SURVEILLANCE AFTER ERADICATION

Complete eradication of intestinal metaplasia does not eliminate recurrence risk. Patients treated for previous high-grade dysplasia require **continued surveillance**.

- **Every 3 months** during the first year.
- **Every 6 months** during the second year.
- **Annually** thereafter.

DDSEP Q41 MEMORY:
Eradicated does not mean discharged.

10.8 BARRETT'S SCREENING

Screening has the highest yield in patients with **multiple risk factors:**

- Male sex
- Age over 50
- White ethnicity
- Central obesity
- Smoking
- Chronic GERD
- Family history

Women generally have **lower risk** and are screened selectively.

Repeat screening is usually **unnecessary** after a high-quality negative examination.

DDSEP Q42 MEMORY:
Older, White, male, obese, smoker, chronic reflux.

10.9 PROGRESSION RISK

- Male sex
- Smoking
- Longer Barrett's segment
- Confirmed low-grade dysplasia

Multiple negative surveillance examinations **reduce future risk.**

Biopsies obtained during severe active esophagitis may **overdiagnose dysplasia.**

DDSEP Q43 MEMORY:
Male, smoker, longer segment, confirmed LGD: higher progression risk.

11. EOSINOPHILIC ESOPHAGITIS

11.1 CLINICAL CLUES


Young male patient


Intermittent solid-food dysphagia


Food sticking with bread or meat


Food impaction


Atopic background

Endoscopy may show rings, furrows, white exudates, edema, narrow-caliber esophagus, or strictures. The esophagus may also appear normal.


DDSEP Q19 MEMORY:
Young man plus meat-and-bread dysphagia: scope and biopsy for EoE.

11.2 DIAGNOSIS


Upper endoscopy with biopsies from multiple esophageal levels is required.


A count of at least 15 eosinophils per high-power field supports the diagnosis in the appropriate clinical setting.


A normal endoscopic appearance does not exclude EoE.

11.3 TREATMENT


PPI therapy


Swallowed topical corticosteroids


Dietary elimination


Dilation for fixed fibrostenotic disease


Cromolyn and montelukast do not consistently produce histological remission.


Dilation treats narrowing but not inflammation.

11.4 DILATION OF EOE STRICTURES

A dominant stricture causing recurrent impaction should be dilated when inflammation is controlled. When a standard endoscope cannot pass, begin with a small balloon, such as 8–10 mm.


Avoid immediate large-caliber bougie dilation


Avoid immediate 18–20 mm balloon dilation

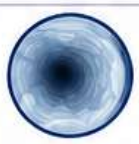

Avoid excessive single-session diameter increase


EoE mucosa is fragile, and overly aggressive dilation may cause deep mucosal rents, severe chest pain, or perforation.


DDSEP Q35 MEMORY:
Tight EoE stricture: start small and dilate gradually.

12. LYMPHOCYTIC ESOPHAGITIS


Histological features include peripapillary intraepithelial lymphocytosis, spongiosis, and few or no eosinophils or neutrophils.


Endoscopic findings may resemble EoE, including rings, narrow-caliber esophagus, strictures, and furrows.


PPI therapy


Swallowed topical steroids


Serial dilation of structural narrowing


DDSEP Q46 MEMORY:
Lymphocytes plus rings: steroid for inflammation, dilation for narrowing.

13. INFECTIOUS AND PILL ESOPHAGITIS

13.1 CANDIDA ESOPHAGITIS

- Diabetes mellitus
- HIV/AIDS
- Immunosuppressive therapy
- Corticosteroids
- Antibiotic exposure
- Esophageal stasis
- Achalasia
- Esophageal cancer

GERD and Barrett's esophagus are **not** direct risk factors.

DDSEP Q21 MEMORY:
Candida favors **diabetes, immune weakness, steroids, antibiotics** and **stasis**.

13.2 DOXYCYCLINE PILL ESOPHAGITIS

Doxycycline can lodge in the **mid** or **proximal esophagus**, often near the aortic arch, and produce **direct mucosal injury**. Clinical features include acute **retrosternal pain, odynophagia** and **dysphagia**.

- Take tablets with **adequate water**
- Remain upright for **at least 30 minutes**
- Avoid taking immediately **before sleep**

DDSEP Q22 MEMORY:
Doxycycline **sticks, burns** and **ulcerates**.

14. ESOPHAGEAL FOREIGN BODIES

An esophageal button battery requires **immediate endoscopic removal**. Serious injury can develop rapidly even when the patient is asymptomatic, can swallow secretions, or has no complete obstruction.

TIME CRITICAL
injury can occur in as little as **2 hours**.

Do not delay removal for CT, contrast swallow, glucagon, or routine fasting intervals.

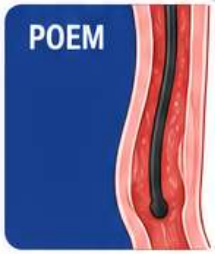
CT may be required when perforation, vascular injury, or fistula is suspected.

No CT delay	No contrast swallow	No glucagon	No fasting delay

DDSEP Q23 MEMORY:
Battery in the esophagus: **remove now.**

15. ACHALASIA AND ESOPHAGEAL MOTILITY DISORDERS

15.1 REFLUX AFTER ACHALASIA THERAPY



POEM has the **highest risk** of postoperative GERD among common achalasia treatments because it performs a myotomy without adding a fundoplication.

Heller myotomy is commonly combined with partial fundoplication, **reducing reflux risk.**

Patients after POEM may have **objective reflux** even without symptoms.

DDSEP Q24 MEMORY:
POEM opens the LES **without** adding a **reflux barrier.**

15.2 PSEUDOACHALASIA

A bird's-beak appearance **does not** automatically establish idiopathic achalasia.

- Older age
- Rapid symptom progression
- Short duration
- Marked weight loss
- Difficulty passing the endoscope through the GEJ

Upper endoscopy should be performed **before** definitive achalasia therapy.

After malignancy is excluded, **manometry confirms** and subtypes achalasia.

DDSEP Q45 MEMORY:
Older, rapid and thin: **look for cancer within.**

15.3 SCLERODERMA ESOPHAGUS

Systemic sclerosis causes smooth-muscle atrophy and fibrosis.

- Dilated fluid-filled esophagus
- Patulous LES
- Low LES pressure or low IRP
- Absent distal contractility
- **Severe reflux** due to weak barrier and poor clearance

This differs from achalasia, where **IRP is elevated.**

DDSEP Q48 MEMORY:
Scleroderma makes the esophagus **weak** and the sphincter **loose.**

15.4 RECURRENT ACHALASIA AFTER HELLER MYOTOMY

Recurrent dysphagia with a dilated tortuous esophagus, elevated IRP, absent contractility and resistance at the GEJ indicates recurrent or persistent outflow obstruction.

A standard 20 mm balloon is **not** equivalent to pneumatic dilation. Graded pneumatic dilation may be attempted **before** more invasive redo therapy.

- Redo Heller myotomy
- POEM
- Esophagectomy in selected end-stage disease
- Botox is generally reserved for patients **unfit** for definitive intervention.

DDSEP Q49 MEMORY:
Recurrent achalasia after myotomy: try **true pneumatic dilation before** major redo surgery.

Key Principle: Evaluate carefully, confirm the diagnosis, and choose the therapy that **relieves obstruction** while **minimizing complications**, especially **reflux.**

15.5 ESOPHAGOGASTRIC JUNCTION OUTFLOW OBSTRUCTION

- Elevated IRP
- Preserved peristalsis
- No criteria for achalasia
- Obstructive symptoms
- Supportive evidence of impaired EGJ emptying

SUPPORTIVE FINDINGS INCLUDE:

- Compartmentalized pressurization
- Barium tablet hold-up
- Abnormal timed barium esophagram
- Abnormal FLIP findings

CAUSES MAY INCLUDE:

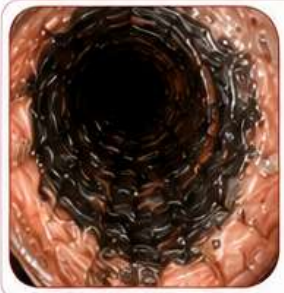
Early or evolving achalasia, hiatus hernia, stricture, previous fundoplication, opioids, infiltrative disease, malignancy, or measurement artifact.

An abnormal pH study may reflect esophageal stasis and fermentation rather than true primary GERD.

Fundoplication should not be performed in an unrecognized obstructive motor disorder because it may worsen dysphagia.

DDSEP Q50 MEMORY:
High IRP plus preserved peristalsis plus tablet hold-up equals clinically relevant EGJOO.

16. ACUTE ESOPHAGEAL NECROSIS



Acute esophageal necrosis, or **black esophagus**, presents with circumferential black ulcerated mucosa, often in a critically ill patient.

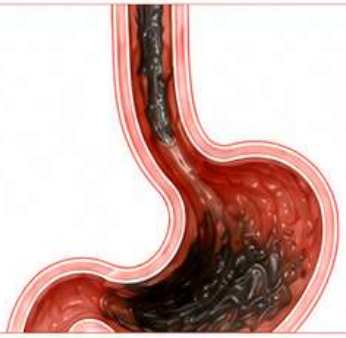
- Low-flow ischemia
- Impaired mucosal defenses
- Severe reflux exposure
- Systemic illness

COMMON ASSOCIATIONS

- Cardiogenic or septic shock
- Hypotension
- Diabetic ketoacidosis
- Malnutrition
- Vascular disease
- Multiorgan failure

MANAGEMENT

- Hemodynamic stabilization
- Treatment of the precipitating illness
- IV acid suppression
- Avoidance of unnecessary traumatic instrumentation
- Monitoring for perforation and stricture



DDSEP Q44 MEMORY:
Black esophagus follows a low-flow, low-defense state.

SECTION II — DDSEP SCENARIO-BASED EXAM TABLE

Integrated rapid-review table for DDSEP Esophageal Disorders Questions 1-50.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	Obesity or obstructive sleep apnea with increased reflux events	Increased TLESR frequency	OSA opens; baclofen blocks.
Q2	Postprandial heartburn with reflux episodes clustered after meals and no nocturnal reflux	Postprandial gastric distension	Meal distension triggers TLESRs.
Q3	Postprandial acidic layer close to the gastroesophageal junction	Acid pocket increases acidic reflux during a TLESR	Loads the reflux; does not open the sphincter.
Q4	Severe symptoms during anxiety, stress or hypervigilance	Increased esophageal perception	Anxiety amplifies; age, Barrett's and diabetes blunt.
Q5	Middle-aged obese man with heavy pressure-like retrosternal pain	Cardiac evaluation first	Clear the heart before investigating the esophagus.
Q6	Symptomatic reflux in a patient taking theophylline	Reduced basal LES pressure	Theophylline loosens the LES.
Q7	Xerostomia after head and neck radiotherapy	Increased risk of erosive esophagitis	Peristalsis clears volume; saliva clears acid.
Q8	H. pylori compared with obesity, male sex, age and hiatus hernia	Lowest risk of complicated GERD	H. pylori-associated atrophy may reduce acid burden.
Q9	Salmon-colored distal mucosa with intestinal metaplasia	Barrett's esophagus confirms GERD	Barrett's proves; hernia and IEM support.
Q10	Normal endoscopy but abnormal prolonged acid exposure	Best predictor of PPI response	Abnormal pH predicts PPI response.
Q11	Acid drops during, acidic meals on wireless on therapy and no previous GERD proof	Acidic swallows may falsely elevate acid exposure	A single sensor cannot distinguish swallowed acid from reflux.
Q12	Atypical reflux symptoms after cardiac disease is excluded	Noncardiac chest pain is most likely to respond to PPI	Unproven GERD: test off PPI.
Q13	Previous LA grade C esophagitis healed, but regurgitation persists on PPI	pH-impedance monitoring on PPI	Proven GERD plus persistent symptoms: test on PPI.
Q14	Heartburn with coexisting IBS or functional bowel disorder	Suboptimal PPI response	Functional overlap predicts persistence.
Q15	Dysphagia after fundoplication	Recognized postoperative complication	Early edema often resolves; late dysphagia needs evaluation.
Q17	Swallowing-associated pain years after fundoplication	Upper endoscopy	Inspect the wrap first.
Q18	Barrett's with apparent low-grade dysplasia before effective acid suppression	Start PPI and repeat biopsies	Inflammation can imitate dysplasia.
Q19	Young man with intermittent bread-and-meat dysphagia	Endoscopy with multiple esophageal biopsies	Think eosinophilic esophagitis.
Q20	Food impaction with at least 15 eosinophils/hpf and normal-looking mucosa	Initiate anti-inflammatory EoE therapy; DDSEP answer: BID PPI	EoE may be endoscopically normal.
Q21	Diabetes with esophageal Candida infection	Diabetes is a direct risk factor	Candida favors diabetes, immune weakness and stasis.
Q22	Odynophagia and proximal erosion while taking doxycycline	Pill esophagitis	Doxycycline sticks, burns and ulcerates.
Q23	Button battery retained in the thoracic esophagus	Immediate endoscopic removal	Remove now, even if asymptomatic.
Q24	Achalasia therapy associated with highest reflux risk	Peroral endoscopic myotomy (POEM)	Myotomy without fundoplication increases reflux.
Q25	Long-term PPI use	Fundic gland polyps	Most consistent PPI-associated structural finding.
Q26	Chronic cough without heartburn after pulmonary and ENT evaluation	Prolonged wireless pH monitoring off PPI	Isolated cough: test before treating reflux.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q27	Nocturnal reflux during travel after large late meals	Avoid late heavy meals and elevate the head of the bed	Late meal plus lying flat equals nocturnal reflux.
Q28	Objectively proven GERD before planned Nissen fundoplication	High-resolution manometry	Prove the esophagus can push before wrapping it.
Q29	BMI 42, diabetes, large hiatus hernia and severe GERD	Hernia repair with Roux-en-Y gastric bypass	Morbid obesity plus GERD: bypass.
Q30	Nausea and delayed gastric emptying after redo fundoplication	Vagal nerve injury	Redo surgery plus gastroparesis: think vagus.
Q31	Severe heartburn shortly after abrupt PPI withdrawal	Rebound acid hypersecretion	Step down with an H2 blocker or on-demand treatment.
Q32	Healed Mallory–Weiss tear after isolated vomiting	Discontinue PPI	No routine repeat endoscopy is required.
Q33	Hoarseness, normal endoscopy and no response to BID PPI	Wireless pH monitoring off therapy	Laryngeal erythema does not prove GERD.
Q34	Globus with sensation that food passes slowly	High-resolution manometry	Globus plus slow swallowing: test motility.
Q35	EoE stricture too narrow for scope passage with controlled inflammation	Graded 8–10 mm balloon dilation	Start small and dilate gradually.
Q36	Gastric folds at 37 cm, circumferential Barrett's to 35 cm, tongue to 32 cm	Prague C2M5	C is continuous; M is maximum contiguous.
Q37	Initial report of Barrett's low-grade dysplasia	Expert GI pathology confirmation	Confirm before ablation.
Q38	Ulcerated depressed Barrett's lesion with irregular mucosa and vessels	Highest risk of submucosal invasion	Depressed and ulcerated means deeper.
Q39	High-grade dysplasia nodule completely removed by EMR	RFA of the remaining Barrett's segment	Resect the lesion; ablate the field.
Q40	Persistent Barrett's after five RFA sessions	Salvage cryotherapy	RFA failure: switch energy.
Q41	Complete eradication after treatment of high-grade dysplasia	Continued intensive endoscopic surveillance	Eradicated does not mean discharged.
Q42	Older White obese male smoker on long-term acid suppression	Best candidate for Barrett's screening	Multiple adenocarcinoma risk factors.
Q43	Male with a long Barrett's segment and confirmed low-grade dysplasia	Highest progression risk	Male sex, smoking, length and LGD increase risk.
Q44	Critically ill hypotensive patient with circumferential black esophagus	Acute esophageal necrosis	Black esophagus follows low flow.
Q45	Elderly patient with rapidly progressive dysphagia, major weight loss and bird's-beak appearance	Upper endoscopy first	Exclude pseudoachalasia before manometry or myotomy.
Q46	Rings and narrow-caliber esophagus with peripapillary lymphocytes and spongiosis	Topical steroids plus serial dilation	Steroid for inflammation; dilation for narrowing.
Q47	Fundoplication wrap located above the diaphragmatic hiatus	Herniated fundoplication	Wrap above the diaphragm equals a herniated wrap.
Q48	Scleroderma with a dilated fluid-filled esophagus	Low IRP and absent contractility	Weak body, loose sphincter.
Q49	Recurrent dysphagia after Heller myotomy with high IRP and absent peristalsis	Graded pneumatic dilation	A 20 mm standard balloon is not true achalasia dilation.
Q50	Elevated IRP, preserved peristalsis, pressurization and tablet hold-up at the GEJ	EGJ outflow obstruction	High IRP plus tablet hold-up equals EGJOO.

DDSEP 9 - CHAPTER 2
ACID DISEASES OF THE STOMACH

DDSEP Exam-Oriented Notes and Scenario Framework

Questions 1-51

Structured for rapid review, clinical pattern recognition, and MCQ recall

SECTION I – EXAM PEARL NOTES

1. PROTON PUMP INHIBITORS: POTENCY, TIMING, AND SAFE USE

PPI POTENCY (DDSEP HIERARCHY)

HIGHEST POTENCY

1. Esomeprazole
2. Rabeprazole
3. Omeprazole
4. Lansoprazole
5. Dexlansoprazole

LOWEST POTENCY

6. Pantoprazole (LEAST POTENT)

Pantoprazole is the lowest-potency PPI in the DDSEP hierarchy.

TIMING OF PPI

30–60 MINUTES BEFORE A MEAL

ONCE-DAILY DOSING

- Before breakfast

TWICE-DAILY DOSING

- Before breakfast
- Before evening meal

PPI FAILURE: COMMON CAUSE

Wrong timing or poor adherence is the most common reason for apparent PPI failure.

PPI switching can be considered for incomplete symptom control.

LONG-TERM SAFE USE

- For chronic GERD or healed erosive disease, continue treatment only when indicated.
- Reduce to the lowest effective dose.
- Media-reported associations (dementia, cardiac disease, pneumonia, C. difficile) do not prove causality.

MEMORY

DDSEP Q1, Q14, Q22, Q44 Memory:
Pantoprazole is least potent; PPI failure usually means wrong timing; long-term PPI = lowest effective dose.

2. PEPTIC ULCER BLEEDING: RESUSCITATION, ENDOSCOPY, AND PREVENTION

RESUSCITATION COMES FIRST

- Give IV fluids for orthostasis or hypovolemia.
- Transfuse unstable patients or severe anemia to a safe threshold before therapeutic endoscopy.

RISK STRATIFICATION

- Glasgow-Blatchford Score (GBS) is the best pre-endoscopic score for deciding admission and need for early endoscopy.
- Rockall score is more useful for mortality risk after endoscopic data are available.

ENDOSCOPIC THERAPY FOR HIGH-RISK STIGMATA

- Combination therapy is preferred.
- Epinephrine alone is not definitive; add a mechanical or thermal modality.

FORREST CLASSIFICATION		REBLEEDING RISK
IA	Spurting bleeding	HIGHEST
IB	Oozing bleeding	
IIA	Visible vessel	HIGH
IIB	Adherent clot	MODERATE
IIC	Flat pigmented spot	LOW
III	Clean base ulcer	LOWEST

PPI THERAPY

- PPI before endoscopy reduces the need for endoscopic therapy.
- After successful hemostasis, oral twice-daily PPI is cost-effective compared with prolonged IV therapy.

ASPIRIN & CARDIOVASCULAR PREVENTION

- In secondary CV prevention, aspirin is usually resumed early after hemostasis with PPI protection.
- Enteric-coated or buffered aspirin does not meaningfully reduce recurrent bleeding.

MEMORY

DDSEP Q5, Q6, Q15, Q25, Q33, Q40, Q41 Memory:
Resuscitate first, risk-stratify with GBS, treat visible vessels with combination therapy, then protect with PPI.

3. NSAIDs, DRUGS, AND STRESS ULCERS

MECHANISM OF NSAID GASTRIC INJURY

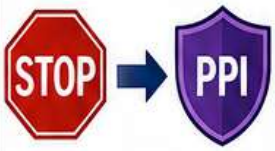
- NSAIDs inhibit COX → ↓ prostaglandins → ↓ mucosal defense → ulcers.
- Direct epithelial injury may occur, but prostaglandin depletion is the key exam mechanism.

RISK AMONG COMMON NSAIDS

Naproxen	Ibuprofen
(HIGHER ulcer-complication risk)	(LOWER risk)

- Enteric coating does not prevent NSAID ulceration because the mechanism is systemic.

MANAGEMENT OF NSAID-ASSOCIATED ULCERS



1. Stop or replace the NSAID (first and most important step).
2. Continue PPI therapy.
3. Other ulcerogenic drugs include:
   - Alendronate
   - Oral iron

STRESS-ULCER PROPHYLAXIS: WHO REALLY NEEDS IT?

MAJOR RISK DRIVERS

- Mechanical ventilation > 48 hours
- Coagulopathy
  - Platelets < 50,000
  - INR > 1.5

SECONDARY RISK FACTORS

Sepsis | High-dose steroids | Burns | Renal or liver failure

KEY POINT

- NSAIDs remove prostaglandin protection.
- Naproxen is high risk.
- Stress-ulcer risk = ventilator or coagulopathy.

MEMORY

DDSEP Q7, Q9, Q10, Q30, Q32, Q37, Q39 Memory:
NSAIDs remove prostaglandin protection; naproxen is high risk; stress-ulcer risk = ventilator or coagulopathy.

4. *H. PYLORI*: TESTING AND TREATMENT LOGIC

TESTING FOR *H. PYLORI*

BEST TEST FOR ACTIVE INFECTION

Urea Breath Test (Preferred when endoscopy is not otherwise needed)

NOT USEFUL FOR ACTIVE INFECTION

Serology (Antibody test) Antibodies may persist after eradication → poor for active infection.

INDICATIONS TO TEST

- Dyspepsia in appropriate patients
- Peptic ulcer disease
- MALT lymphoma
- Family history of gastric cancer

Family history increases the importance of detecting and eradicating H. pylori.

FIRST-LINE THERAPY WHEN MACROLIDE RESISTANCE RISK

BISMUTH QUADRUPLE THERAPY (PREFERRED)

PPI + Bismuth + Metronidazole + Tetracycline

When macrolide exposure or resistance risk is present, avoid clarithromycin regimens.

Older DDSEP options may list clarithromycin-containing concomitant therapy; modern practice favors bismuth quadruple therapy when resistance is likely.

AFTER TREATMENT FAILURE

1. Reassess adherence.
2. Check for prior antibiotic exposure (resistance risk).
3. Switch to an alternative regimen (e.g., bismuth quadruple if not used before, levofloxacin-based, or rifabutin-based as appropriate).

PENICILLIN ALLERGY LABEL

- Many patients labelled "penicillin-allergic" are not truly allergic.
- Allergy testing can be useful, because amoxicillin is highly effective and valuable in salvage regimens.

MEMORY

DDSEP Q19, Q20, Q23, Q29, Q46 Memory:
Active H. pylori = breath or stool test; resistance risk = bismuth quadruple; failed therapy + penicillin label = consider allergy testing.

5. AUTOIMMUNE GASTRITIS, PERNICIOUS ANEMIA, AND B12 ABSORPTION

AUTOIMMUNE METAPLASTIC ATROPHIC GASTRITIS

Suggested by the following DDSEP features:

- Autoimmune clustering (other autoimmune diseases)
- Body-predominant oxyntic (fundic) atrophy
- Pseudopyloric (intestinal) metaplasia
- Vitamin B_{12}-deficiency anemia

KEY TEST

Anti-parietal cell antibody
(the key DDSEP test)

PARIETAL CELL LOSS → ACHLORHYDRIA

PERNICIOUS ANEMIA (AUTOIMMUNE)	GASTRINOMA (ZES)
	
↑ Gastric pH **(HIGH)**	↓ Gastric pH **(LOW)**
↑ Gastrin **(HIGH)**	↑ Gastrin **(HIGH)**
Secretin stimulation **(NEGATIVE)**	Secretin stimulation **(POSITIVE)**

VS

KEY DISTINCTION

Pernicious anemia: high pH, high gastrin, negative secretin test
Gastrinoma: low pH, high gastrin, positive secretin test

VITAMIN B12 ABSORPTION PHYSIOLOGY

1. Food-bound B_{12} is released by gastric acid and pepsin in the stomach.
2. HC — B_{12} binds to haptocorrin (R-protein) and is protected as it passes through the stomach.
3. In the duodenum, pancreatic enzymes degrade haptocorrin and release free B_{12}.
4. IF — B_{12} then binds to intrinsic factor (IF) secreted by parietal cells.
5. The B_{12}–IF complex is absorbed in the terminal ileum via cubilin receptor.

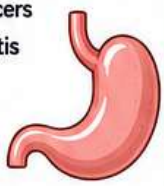
DDSEP MEMORY

DDSEP Q3, Q26, Q49 MEMORY:
Pernicious anemia = parietal cell loss → high pH, high gastrin, low IF; B12 finally binds IF in the duodenum.

6. HYPERGASTRINEMIA: GASTRINOMA, RETAINED ANTRUM, AND ATROPHIC CORPUS GASTRITIS

ZOLLINGER-ELLISON SYNDROME (ZES) CLINICAL CLUES

- Refractory or multiple ulcers
- Postbulbar (duodenal) ulcers
- Severe erosive esophagitis
- Diarrhea
- Weight loss
- MEN1 clues (hypercalcemia, pituitary or pancreatic NET, family history)



INITIAL EVALUATION

Measure **FASTING** serum gastrin and **INTERPRET** with gastric pH.

INTERPRETING HIGH GASTRIN

HIGH GASTRIN + LOW GASTRIC pH
(ACID PRESENT)

- Zollinger-Ellison syndrome (gastrinoma)
- Acid hypersecretion state

HIGH GASTRIN + HIGH GASTRIC pH
(LOW/ABSENT ACID)

- Pernicious anemia (autoimmune atrophic gastritis)
- Corpus-predominant atrophic H. pylori gastritis
- Reduced parietal cell mass

CLINICAL PEARLS

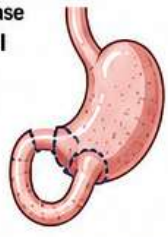
Weight gain after successfull ZES treatment reflects restoration of fat and nutrient absorption after acid hypersecretion is controlled.

Cimetidine (older H_2 blocker) may cause gynecomastia.

Always interpret gastrin in the context of gastric pH and clinical scenario.

RETAINED ANTRUM AFTER BILLROTH II (WITHOUT VAGOTOMY)

Recurrent ulcer disease years after Billroth II without vagotomy suggests a retained antrum as the ulcer driver.



DISTINGUISHING RETAINED ANTRUM FROM GASTRINOMA

Use SECRETIN STIMULATION TEST

Condition	Gastrin Response to Secretin
Retained antrum	INCREASES (positive)
Gastrinoma (ZES)	NO INCREASE (negative)

DDSEP TAKE-HOME MESSAGE

HIGH GASTRIN + LOW pH
= ACID HYPERSECRETION
(ZES / GASTRINOMA)

HIGH GASTRIN + HIGH pH
= ATROPHIC PARIETAL CELL LOSS
(PERNICIOUS ANEMIA / ATROPHIC GASTRITIS)

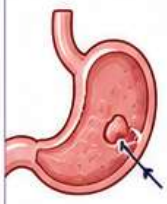
DDSEP MEMORY

DDSEP Q4, Q12, Q27, Q35, Q45, Q50 MEMORY:
High gastrin + low pH = acid hypersecretion / ZES; high gastrin + high pH = atrophic parietal cell loss.

7. GASTRIC POLYPS AND GASTRIC NEUROENDOCRINE TUMORS

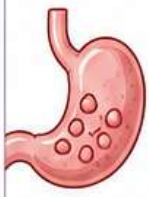
GASTRIC POLYPS

HYPERPLASTIC POLYPS



- Arise from inflammatory proliferation of mucous-producing foveolar (fundic surface) cells.
- Often in a background of chronic mucosal injury such as bile exposure, H. pylori, or atrophic gastritis.

FUNDIC GLAND POLYPS (FGPs)



- Common in PPI users.
- Usually benign.
- Dysplasia risk increases with:
  - ✓ Larger size (> 1 cm)
  - ✓ Antral gastritis
  - ✓ Familial adenomatous polyposis (FAP)

Memory: FGP dysplasia risk = size / FAP / antral gastritis

GASTRIC NEUROENDOCRINE TUMORS (CARCINOIDS)

Arise from enterochromaffin-like (ECL) cells.

TYPE	ASSOCIATION	FEATURES	MALIGNANT POTENTIAL
TYPE 1	Chronic atrophic gastritis → hypergastrinemia	• Multiple • Small (< 1–2 cm) • Glandular body predominant	LOW (<2%)
TYPE 2	Zollinger–Ellison syndrome (MEN1)	• Multiple • Small • Associated with gastrinoma	INTERMEDIATE (10–30%)
TYPE 3	Sporadic (No atrophic gastritis)	• Solitary • Larger (> 2 cm) • Usually in antrum	HIGH (>50%) Highest metastatic potential

MEMORY

DDSEP Q2, Q8, Q18 MEMORY:
Hyperplastic polyp = foveolar cells;
FGP dysplasia risk = size / FAP / antral gastritis;
Solitary large carcinoid = type 3.

KEY POINTS

- Hyperplastic polyps are reactive and inflammatory in origin.
- FGPs are strongly associated with proton pump inhibitor use.
- FAP patients are at higher risk of dysplasia in FGPs.
- A solitary large carcinoid tumor should raise concern for type 3.

8. FUNCTIONAL DYSPEPSIA AND DYSPEPSIA WORKUP

1. WHEN TO DO ENDOSCOPY

INDICATED AT AGE ≥ 60 OR WITH ALARM FEATURES

- Unintentional weight loss
- Progressive dysphagia
- Overt bleeding (melena/hematemesis)
- Persistent vomiting
- Fe Iron-deficiency anemia

Weight loss, even if partly explained by lifestyle or weather, should prompt upper endoscopy.

2. IF NO ALARM FEATURES AND AGE < 60 YEARS

Consider a 4–8 week proton pump inhibitor (PPI) trial.

If symptoms improve: continue and then step down to the lowest effective dose.

If symptoms persist despite adequate PPI trial → proceed to upper endoscopy.

3. AFTER NORMAL ENDOSCOPY AND PPI FAILURE

Likely diagnosis:
FUNCTIONAL DYSPEPSIA

- No structural disease on EGD.
- Symptoms persist despite adequate PPI therapy.
- Manage based on symptom pattern (epigastric pain syndrome or postprandial distress syndrome).

4. ROLE OF NEUROMODULATORS AND PROKINETICS

NEUROMODULATOR

Low-dose Amitriptyline (10–25 mg at bedtime) may help pain-predominant symptoms.

PROKINETIC / ANXIOLYTIC

Buspirone improves fundic accommodation and may help early satiety and postprandial distress syndrome.

Tailor therapy to dominant symptom and patient preference.

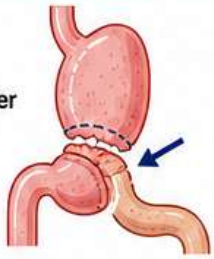
MEMORY

DDSEP Q13, Q36, Q43, Q48 MEMORY:
No alarm features and age < 60 = trial therapy; weight loss = scope;
normal EGD + PPI failure = functional dyspepsia / neuromodulator.

9. ROUX-EN-Y GASTRIC BYPASS AND MARGINAL ULCERS

WHAT IS A MARGINAL ULCER?

Occurs at the **gastrojejunal anastomosis** after Roux-en-Y gastric bypass.



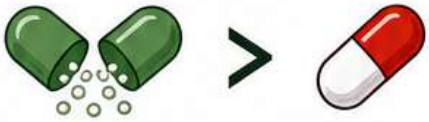
RISK FACTORS

- Ischemia
- Acid exposure
- Diabetes mellitus
- Smoking
- NSAID use
- Gastrogastric fistula
- Foreign material (sutures, staples)
- Long gastric pouch

MEDICAL MANAGEMENT

PPI DELIVERY AFTER BYPASS

OPENED PPI CAPSULE > **INTACT PPI CAPSULE**



Accelerates healing compared with intact capsules.

Slower healing after bypass.

SUCRALFATE

May be used clinically, but in the DDSEP scenario it did not outperform optimized PPI delivery.

OPTIMIZED PPI STRATEGY

- Use opened PPI capsule.
- Take 30–60 minutes before meals.
- Consider twice-daily dosing.
- Ensure adherence and avoid NSAIDs and smoking.

WHEN TO SEEK SURGICAL REVIEW

Persistent ulcer symptoms despite optimized PPI therapy require surgical review to assess:

- Anastomotic ischemia
- Gastrogastric fistula
- Anatomy or pouch size
- Foreign body
- Other structural causes

NUTRITIONAL NOTE

Vitamin B12 deficiency after bypass is suggested by elevated methylmalonic acid (MMA).

DDSEP MEMORY

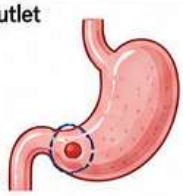
DDSEP Q16, Q17, Q31, Q38 MEMORY:
Marginal ulcer = anastomotic ischemia/acid/NSAID/smoking; after bypass, open the PPI capsule; refractory ulcer = surgical review.

10. GASTRIC OUTLET OBSTRUCTION AND GIANT GASTRIC ULCERS

CAUSE: LARGE PREPYLORIC OR PYLORIC ULCERS

May cause gastric outlet obstruction due to:

- Edema
- Deformity
- Scarring/Fibrosis



CLINICAL CLUES

- Nausea
- Vomiting (non-bilious, postprandial)
- Abdominal discomfort / fullness
- Dilated, fluid-filled stomach on exam or imaging

DIAGNOSIS AND EVALUATION

CT ABDOMEN — Helps confirm obstruction and evaluate for complications (distended stomach, wall thickening, masses, perforation).

UPPER ENDOSCOPY — Confirms the ulcer, assesses severity, and allows biopsy to rule out malignancy.

LABS — Assess dehydration, electrolyte imbalance, and nutritional status.

GIANT GASTRIC ULCER

Ulcers ≥ 2.5–3 cm are considered giant gastric ulcers.

REPEAT ENDOSCOPY AFTER PPI THERAPY

to document healing and exclude malignancy.

PHYSIOLOGY: EFFECT ON GASTRIN AND GASTRIC pH

GASTRIN LEVEL ↑

May be elevated due to retained antral stimulation.

GASTRIC pH ↓

Remains LOW if acid secretion is preserved.

MANAGEMENT PRINCIPLES

- Optimize PPI therapy.
- Treat underlying H. pylori if present.
- Correct fluids and electrolytes.
- Endoscopic follow-up for healing and to exclude malignancy.
- Surgery if obstruction persists despite medical therapy.

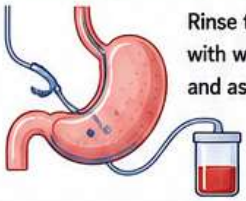
DDSEP MEMORY

DDSEP Q34, Q35, Q42 MEMORY:
Giant/prepyloric ulcer + vomiting = check obstruction; giant gastric ulcer = repeat EGD to prove healing.

11. TUMOR BLEEDING, NG LAVAGE, AND UPPER-GI BLEEDING TRAPS

NG LAVAGE: ROLE AND LIMITATIONS

WHAT IS NG LAVAGE?



Rinse the stomach with water via NG tube and aspirate.

POTENTIAL BENEFIT (in selected UGIB)

- Clears fundic blood and clots
- Improves endoscopic visualization
- May reduce need for repeat endoscopy

LIMITATIONS

- Uncomfortable for the patient
- Does **NOT** reduce mortality
- Not needed in all UGIB cases

BOTTOM LINE

Use selectively when large amounts of blood obscure endoscopic view.

TUMOR (MALIGNANT) BLEEDING

Diffuse oozing from a cardia or gastric mass is often difficult to control with standard focal therapy (clips, coagulation).

HEMOSTATIC POWDER (TC-325)

- Provides broad, non-contact surface hemostasis
- Useful for diffuse tumor bleeding
- Useful as a bridge to definitive therapy (oncologic / surgical / radiologic)

BEST PRACTICE

- Optimize resuscitation and correction of coagulopathy.
- Use powder for uncontrolled diffuse tumor ooze or as a temporizing measure.
- Plan definitive management early.

DON'T MISS THE BIG TRAP:

UPPER-GI BLEEDING CAN PRESENT WITH HEMATOCHEZIA

THINK UPPER GI WHEN:

- Melena is present
- High BUN (> 30 mg/dL)
- Hemodynamic instability (shock, tachycardia, hypotension)
- Massive blood loss

KEY MESSAGE

Massive hematochezia with shock may still be brisk UPPER-GI bleeding. Do NOT assume lower-GI source.

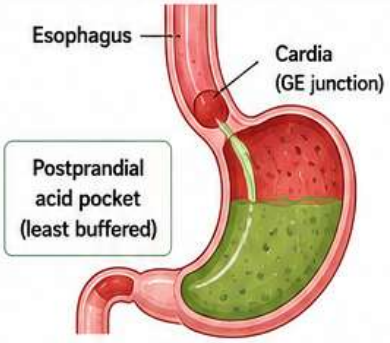
DDSEP MEMORY

DDSEP Q21, Q24, Q33, Q41 MEMORY:

Massive hematochezia can be upper GI; NG lavage helps visualization, not survival; tumor ooze = powder.

12. GERD OVERLAP, CARDIA ACID POCKET, AND SEX DIFFERENCES

THE CARDIA ACID POCKET (PROPENSITY TO REFLUX)

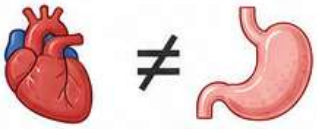


- After meals, acid collects in the proximal stomach (cardia) and is least buffered in this region.
- This explains why the area near the GE junction is most vulnerable to acidic refluxate.

CLINICAL IMPLICATION

Proximal reflux of acid-rich contents can cause symptoms and mucosal injury in the distal esophagus.

CHEST BURNING IN OLDER PATIENTS: RULE OUT HEART DISEASE FIRST



KEY POINT

Chest pain or burning in an older patient must be evaluated for **CARDIAC** causes before attributing symptoms to GERD.

EVALUATE CARDIAC RISK FACTORS

- Age > 40–50 years
- Diabetes mellitus
- Hypertension
- Dyslipidemia
- Smoking
- Family history of CAD
- Known CAD / vascular disease

Always obtain appropriate cardiac assessment (ECG, ± stress test, cardiac enzymes if indicated).

SEX DIFFERENCES IN GERD OUTCOMES

♂ VS ♀

Erosive Esophagitis	Men > Women
Barrett Esophagus	Men > Women
GERD Symptoms	Similar prevalence, but women report atypical symptoms more often
Proposed pH Thresholds	Women: higher acid exposure may be needed to cause symptoms (Not standard in DDSEP)

KEY TAKEAWAY

Men have higher rates of erosive esophagitis and Barrett esophagus than women. Sex-specific pH thresholds are proposed but not standard in DDSEP.

DDSEP MEMORY

DDSEP Q11, Q28, Q51 MEMORY:

Cardia holds the postmeal acid pocket; chest pain needs cardiac clearance; men have more erosive GERD/Barrett risk.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q25	UGIB triage for admission/early endoscopy	Glasgow-Blatchford score	Best pre-endoscopic low-risk exclusion tool.
Q26	Pernicious anemia physiology	High gastrin, high pH, negative secretin	Parietal-cell loss -> achlorhydria.
Q27	SE Asian patient with corpus-predominant H. pylori atrophy	High gastrin, high pH, decreased parietal mass	Atrophic corpus behaves like acid loss.
Q28	Older man with chest burning after heavy meals	Cardiac workup	Cardiac first before GERD attribution.
Q29	China immigrant with H. pylori; DDSEP regimen option	Clarithro + amox + metro + PPI	Modern update: bismuth quadruple if available.
Q30	Multiple antral ulcers/erosions with NSAID use	Stop NSAIDs + continue PPI	Remove the cause; test H. pylori as appropriate.
Q31	Post-bypass severe postprandial pain; nutritional clue	Elevated methylmalonic acid	Bypass predisposes to B12 deficiency.
Q32	Gnawing pain with chronic ibuprofen for migraine	Switch to non-NSAID agent	Enteric-coated NSAID is not protective.
Q33	Melena/orthostasis, Hb 9 on apixaban	Start IV fluids	Resuscitation begins before procedures.
Q34	Recent giant prepyloric ulcer + nausea/vomiting	CT scan	Confirm gastric outlet obstruction.
Q35	Pyloric deformity/GOO from large ulcer	High gastrin, low gastric pH	Antral stimulation with preserved acid output.
Q36	Age 59 dyspepsia, no alarm features	4-8 week PPI trial	Scope at >=60 or with alarm features.
Q37	ICU GI bleed; stress-ulcer risk clue	Platelets 21,000	Coagulopathy is an independent stress-ulcer risk.
Q38	Marginal ulcer after RYGB not responding to PPI	Surgical referral	Look for fistula, ischemia, pouch/anastomotic problem.
Q39	Ulcer/IDA with osteoporosis drug exposure	Alendronate	Bisphosphonate can injure upper GI mucosa.
Q40	Active ulcer bleed; pre-endoscopy therapy effect	PPI drip before endoscopy	Reduces need for endoscopic intervention.
Q41	Shock + melena + Hb 6.6	Transfuse 2 units PRBC	Resuscitate before endoscopy.
Q42	Giant 3 cm gastric ulcer, Forrest III	Repeat EGD in 12 weeks	Nonhealing gastric ulcer suggests malignancy.
Q43	Normal EGD + dyspepsia not responding to PPI	Start amitriptyline	Functional dyspepsia: consider neuromodulator.
Q44	Concern about long-term PPI harms	Taper to lowest effective dose	Associations are not proof of causation.
Q45	Diarrhea + severe esophagitis + multiple duodenal ulcers	Serum gastrin level	Classic ZES screen.
Q46	H. pylori failure + reported penicillin allergy	Refer for allergy testing	Many penicillin allergies are false; amoxicillin useful.
Q47	Transplant patient + dysphagia/ nausea/vomiting	Multiple shallow ulcers, possible viral	Think CMV/HSV and request special testing.
Q48	Dyspepsia worse with meals + 20 lb weight loss	Upper endoscopy	Weight loss is an alarm feature.
Q49	Essential step in B12 absorption	Cobalamin binds intrinsic factor in duodenum	IF is made in stomach but binds B12 in small bowel.
Q50	MEN1/ZES clue + older acid-suppression fact	Cimetidine may cause gynecomastia	H. pylori does not explain MEN1/ZES pattern.
Q51	Female GERD phenotype	Women have lower erosive esophagitis rate than men	Men have higher erosive GERD and Barrett risk.

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

Integrated rapid-review table for DDSEP 9 Chapter 2, Questions 1-51.

DDSEP Q#	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	PPI potency question	Pantoprazole	Lowest PPI potency in DDSEP hierarchy.
Q2	Bilious stomach + hyperemic pedunculated antral polyp	Gastric foveolar cells	Hyperplastic polyp = foveolar-cell proliferation.
Q3	Autoimmune background + B12 anemia + body oxyntic atrophy	Anti-parietal cell antibody	AMAG = autoimmune parietal-cell loss.
Q4	Bilroth II without vagotomy + recurrent ulcers years later	Secretin stimulation test	Think retained antrum vs gastrinoma.
Q5	CAD on aspirin + duodenal ulcer bleed after hemostasis	Continue low-dose aspirin + daily PPI	Secondary prevention aspirin resumes early with PPI protection.
Q6	After ulcer hemostasis, cost-effective recurrent-bleed prevention	Oral twice-daily PPI	Oral PPI after hemostasis is effective and cheaper than prolonged IV.
Q7	NSAID choice with highest ulcer-complication risk	Naproxen	Naproxen higher risk; ibuprofen lower risk.
Q8	Solitary 2.5 cm ulcerated gastric-body carcinoid	Type 3	Solitary sporadic gastric NET = more aggressive.
Q9	Trauma ICU stress-bleed prophylaxis risk	Mechanical ventilation	Independent risk if ventilation >48 h.
Q10	NSAID effect on gastric protection	Decreased prostaglandin synthesis	COX inhibition removes mucosal defense.
Q11	Postprandial stomach site least buffered by food	Cardia	Acid pocket sits near the cardia/GEJ.
Q12	ZES treated successfully then weight gain	Appropriate fat and nutrient absorption	Control acid → correct malabsorption.
Q13	Functional dyspepsia, early satiety, buspirone benefit	Fundic relaxation/accommodation	Buspirone improves accommodation.
Q14	GERD controlled on PPI, needs chronic therapy	Reduce to 20 mg	Use lowest effective PPI dose.
Q15	Bleeding ulcer with non-bleeding visible vessel	Epinephrine + hemostatic clips	Epinephrine alone is not definitive.
Q16	Post-RYGB abdominal pain/fatigue, marginal ulcer risk	Long gastric pouch	Long pouch increases acid/ischemic marginal-ulcer risk.
Q17	Accelerating marginal-ulcer healing after RYGB	Opened PPI capsules	Open capsule improves delivery after bypass.
Q18	Fundic gland polyp dysplasia risk	Antral gastritis	Also consider size and FAP.
Q19	Noninvasive active H. pylori test	Urea breath test	Serology does not prove active infection.
Q20	H. pylori + prior macrolide/ fluoroquinolone exposure	PPI + bismuth + metronidazole + tetracycline	Resistance risk → bismuth quadruple.
Q21	Large-volume suspected UGIB with poor visualization	NG lavage may reduce additional endoscopy	Helps visualization; not mortality.
Q22	Most important reason for PPI failure	Poor compliance / wrong administration	Take 30–60 min before meals.
Q23	Indication to test H. pylori	Family history of gastric cancer	H. pylori eradication reduces cancer pathway risk.
Q24	Bleeding malignant cardia/ gastric mass	Hemostatic powder TC-325	Diffuse tumor bleeding needs broad surface therapy.

DDSEP 9 - CHAPTER 3
PANCREATIC PHYSIOLOGY & DISEASE

DDSEP Exam-Oriented Notes and Scenario Framework

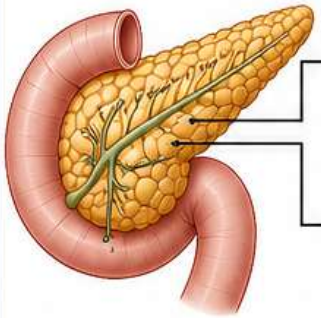
Questions 1-50

Structured for rapid review, clinical pattern recognition, and MCQ recall

SECTION I – EXAM PEARL NOTES

1 PANCREATIC SECRETION AND PHYSIOLOGY

1.1 DUCT CELLS, ACINAR CELLS & & HORMONAL CONTROL



Duct Cells (secrete bicarbonate)

Acinar Cells (secrete enzymes)

SECRETIN

Released from duodenal S-cells when duodenal pH < 4.5

ACTION

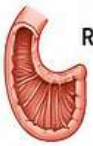
Stimulates **DUCT CELLS** via cyclic AMP

RESULT

Bicarbonate-rich fluid secretion (washes acid)

HCO_3^-

CHOLECYSTOKININ (CCK)



Released post-meal from duodenal I-cells

ACTION

Stimulates **ACINAR CELLS** (mainly via Ca^{2+} pathways)

RESULT

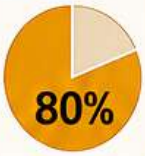
Enzyme-rich zymogen secretion (proteins, amylase, lipases, nucleases)

ENZYME CATEGORIES (PANCREATIC)

- **Proteases** (Trypsinogen, Chymotrypsinogen, Procarboxypeptidases)
- **Amylase** (Amylase)
- **Lipases** (Lipase, Phospholipase, Cholesterol esterase)
- **Nucleases** (RNase, DNase)

PHASES OF PANCREATIC SECRETION

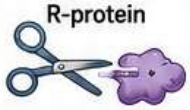
Meal-related INTESTINAL phase accounts for ~80% of total secretion.

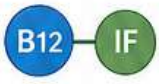


DDSEP Q2, Q4, Q5, Q9–Q12 MEMORY:
Acid in the duodenum calls **SECRETIN**; **SECRETIN** washes with bicarbonate, **CCK** squeezes enzymes.

1.2 PANCREAS AND VITAMIN B12

Dietary B12 bound to R-protein → Pancreatic proteases break down R-protein → B12 binds to Intrinsic Factor (IF) → Absorbed in Terminal Ileum





DDSEP Q43 MEMORY:
Pancreatic proteases free B12 from R-protein.

2 ACUTE PANCREATITIS: DIAGNOSIS, ETIOLOGY & INITIAL CARE

DIAGNOSIS

Diagnose clinically when typical epigastric pain is accompanied by elevated **LIPASE.**

CT is **NOT** required when diagnosis is clear, early in course, without complications.

ETIOLOGY

Transabdominal **ULTRASOUND** should be performed to look for **GALLSTONES.**

FLUID RESUSCITATION

Lactated Ringer's (LR) is the preferred initial resuscitation fluid.

MONITORING

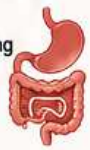
Monitor **Hematocrit, BUN** and **Creatinine** to adjust fluid therapy.

ANTIBIOTICS

Do **NOT** give prophylactic antibiotics for sterile necrosis.

NUTRITION

Enteral feeding is preferred over TPN.



If oral feeding fails in severe disease, use **TUBE FEEDING.**

DDSEP Q3, Q6, Q11, Q36, Q41, Q48 MEMORY:
Diagnose clinically, look for gallstones, resuscitate with **LR**, feed the gut.

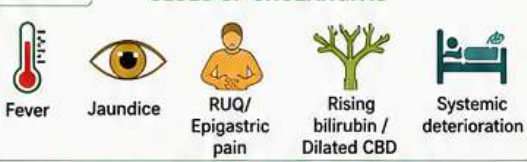
2.1 GALLSTONE PANCREATITIS AND ERCP

WHEN IS ERCP INDICATED URGENTLY?

When gallstone pancreatitis is accompanied by:

- Cholangitis
- Persistent biliary obstruction
- Rising bilirubin
- Dilated CBD on imaging
- Systemic deterioration

CLUES OF CHOLANGITIS



NO OBSTRUCTION / NO CHOLANGITIS
→ Supportive care + Cholecystectomy during the same admission when appropriate.

ERCP RISK MANAGEMENT

High-risk ERCP with repeated pancreatic duct cannulation

↓

Place prophylactic **PANCREATIC DUCT STENT**

DDSEP Q21, Q34, Q38–Q40, Q49 MEMORY:
Gallstone pancreatitis alone does not equal ERCP; **CHOLANGITIS** does.

3. PANCREATIC FLUID COLLECTIONS

Revised Atlanta terminology depends on TIME from pancreatitis onset and whether NECROSIS is present.

TIME FROM ONSET	PANCREATITIS TYPE	COLLECTION TYPE	DESCRIPTION / FEATURES
< 4 WEEKS	INTERSTITIAL PANCREATITIS	ACUTE PERIPANCREATIC FLUID COLLECTION	Fluid only, located in or around the pancreas, not encapsulated.
< 4 WEEKS	NECROTIZING PANCREATITIS	ACUTE NECROTIC COLLECTION	Contains both fluid and non-viable necrotic tissue, not encapsulated.
> 4 WEEKS	(ANY TYPE) FLUID ONLY	PSEUDOCYST	Encapsulated collection containing fluid only, no necrosis.
> 4 WEEKS	(ANY TYPE) WITH NECROSIS	WALLED-OFF NECROSIS (WON)	Encapsulated collection containing both fluid and necrotic tissue.

MANAGEMENT PRINCIPLES

Asymptomatic pseudocysts or collections without infection can be observed with repeat imaging.

DRAINAGE / INTERVENTION INDICATED FOR:

- Symptoms (pain, early satiety, vomiting, bloating)
- Infection
- Rapid enlargement
- Complications (bleeding, obstruction, rupture, etc.)

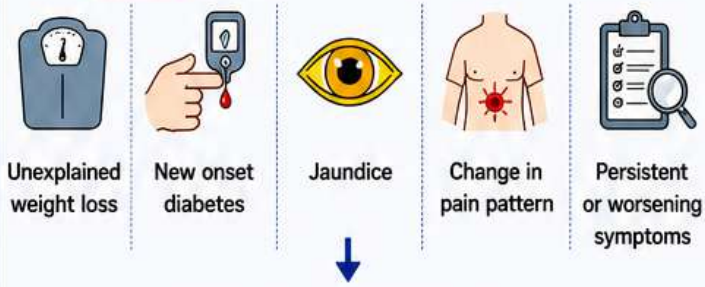
DDSEP Q22, Q23, Q26, Q35 MEMORY:
Four weeks and necrosis decide the name; symptoms decide drainage.

4. CHRONIC AND HEREDITARY PANCREATITIS

4.1 GENETICS AND CANCER RISK

GENE / DEFECT	ASSOCIATION	KEY POINTS
PRSS1 (Cationic trypsinogen mutation)	Hereditary pancreatitis	• Autosomal dominant with incomplete penetrance • **Highest risk** of pancreatic adenocarcinoma among listed disorders
SPINK1 (Serine protease inhibitor Kazal type 1)	Recurrent idiopathic pancreatitis / Tropical pancreatitis association	• Impaired trypsin inhibition → trypsin activation in pancreas • Lower cancer risk than PRSS1

ALARM SYMPTOMS IN CHRONIC PANCREATITIS



→ Requires evaluation for **PANCREATIC CANCER**

DDSEP Q1, Q37, Q44, Q50 MEMORY:
PRSS1 is cationic trypsinogen; hereditary pancreatitis is a **pancreatic cancer warning sign.**

4.2 CHRONIC PANCREATITIS: COMPLICATIONS & PAIN STRATEGY

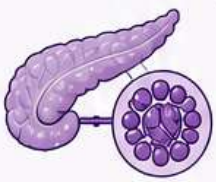
COMMON COMPLICATIONS

- Osteoporosis
- Exocrine insufficiency → steatorrhea, fat-soluble vitamin deficiency
- Vitamin B12 deficiency (impaired R-protein breakdown by pancreatic proteases)

PAIN STRATEGY BY PATTERN

LARGE-DUCT DISEASE
(Marked main pancreatic duct dilation)
→ Best long-term treatment: **DRAINAGE OPERATION**
Example: Lateral pancreaticojejunostomy (Puestow)

SMALL-DUCT DISEASE
(Hereditary, e.g., PRSS1) with severe pain
→ Consider **TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION (TPIAT)** in selected patients



KEY POINT

Tailor intervention to **DUCT PATTERN & PAIN SEVERITY.**

Goal: Pain relief, preserve function, improve quality of life.

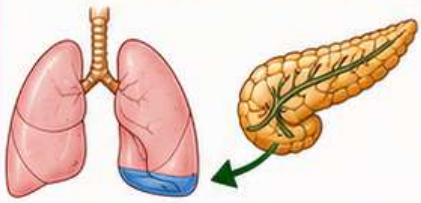
DDSEP Q8, Q13, Q15, Q33 MEMORY:
Large duct drains with **Puestow**; **PRSS1** small-duct pain may need **TPIAT.**

5. DUCT DISRUPTION AND VASCULAR COMPLICATIONS

Necrotizing pancreatitis may produce duct disruption, fistulas and vascular complications.

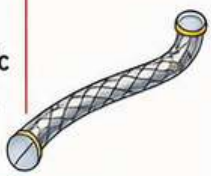
The scenario clue usually determines the organ involved.

PANCREATICOPLEURAL FISTULA

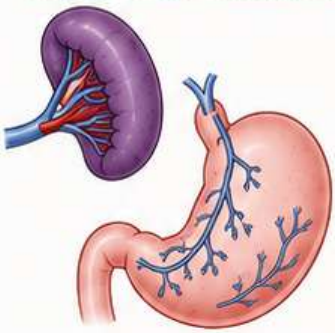


CLUE: Pleural effusion after necrotizing pancreatitis

MANAGEMENT:
ERCP with pancreatic duct stent to bridge disruption.



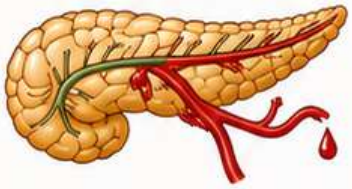
SPLENIC VEIN THROMBOSIS



CLUE: Isolated gastric variceal bleeding after pancreatitis

MANAGEMENT:
Definitive treatment is **SPLENECTOMY.**

HEMOSUCCUS PANCREATICUS



CLUE: Bleeding through the pancreatic duct with instability

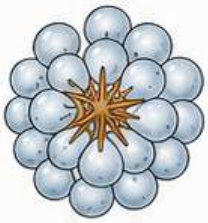
MANAGEMENT:
ANGIOGRAPHY and **EMBOLIZATION.**

DDSEP Q16, Q17, Q42 MEMORY:
Pleura needs duct stent; isolated gastric varices need spleen; duct bleeding needs embolization.

6. PANCREATIC CYSTS AND CYSTIC NEOPLASMS

Pancreatic cyst management is driven by symptoms, size, duct dilation, mural nodules, solid components and whether the cyst is clearly benign.

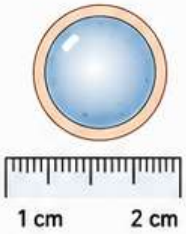
CLASSIC SEROUS CYSTADENOMA



- Grape-cluster (microcystic) lesion with central scar
- Benign

MANAGEMENT:
Observe if asymptomatic.

SMALL INCIDENTAL CYST (1–2 CM)



- No high-risk features
- Asymptomatic

MANAGEMENT:
MRI surveillance.

HIGH-RISK FEATURES

- Cyst > 3 cm
- Mural nodule
- Solid component
- Dilated pancreatic duct

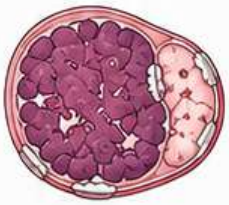
→ **MANAGEMENT:**
EUS–FNA

MAIN-DUCT IPMN OR SYMPTOMATIC IPMN (WITH PANCREATITIS)

- High risk for malignancy

→ **MANAGEMENT:**
Surgical resection if patient is fit.

SOLID PSEUDOPAPILLARY TUMOR (SPT)



- Young woman
- Solid-cystic lesion in tail
- Peripheral calcification

→ **MANAGEMENT:**
Surgical resection.

DDSEP Q7, Q14, Q20, Q27–Q30 MEMORY:
Serous cysts reassure; mucinous clues and main duct involvement escalate.

7. PANCREATIC TUMORS AND CANCER PATTERNS

7.1 PANCREATIC ADENOCARCINOMA

Cigarette smoking is the **best-established** environmental risk factor.

Chronic pancreatitis increases risk.

New alarm features should trigger pancreas-protocol CT.

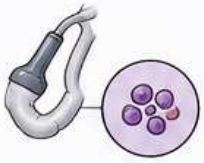
CLINICAL CLUE

Painless jaundice, weight loss and smoking

↓

Suggests pancreatic head cancer.

DIAGNOSIS



Evaluate with **EUS–FNA** when tissue diagnosis is needed.

RESECTABLE DISEASE (PANCREATIC HEAD MASS WITH OBSTRUCTIVE JAUNDICE)

Pancreaticoduodenectomy (Whipple operation) preferred.
Avoid routine preoperative **ERCP**.

LOCALLY ADVANCED UNRESECTABLE DISEASE

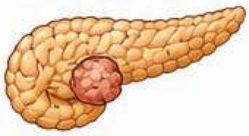
Neoadjuvant or **systemic therapy** is the standard approach.

DDSEP Q25, Q31–Q33, Q39 MEMORY:
Smoking risks it; **jaundice reveals it; resectable head mass goes** to Whipple.

7.2 UNUSUAL PANCREATIC TUMORS AND NEUROENDOCRINE SYNDROMES

INSULINOMA

(Most common functioning pancreatic endocrine tumor)

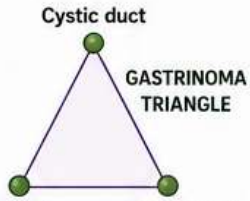


- Excess insulin → hypoglycemia

Symptoms: sweating, palpitations, tremor, confusion, hunger.

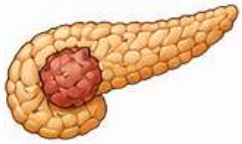
GASTRINOMA

(Zollinger–Ellison syndrome)

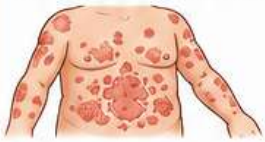


- Refractory peptic ulcers
- Esophagitis
- Diarrhea

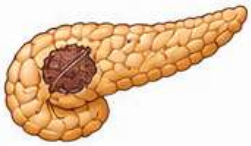
GLUCAGONOMA



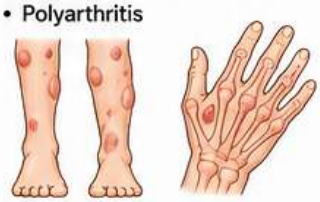
- Diabetes mellitus
- Diarrhea
- Weight loss
- Characteristic rash (necrolytic migratory erythema)



ACINAR CELL CARCINOMA



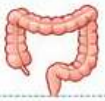
- Pancreatic mass
- Panniculitis (tender subcutaneous nodules)
- Polyarthritis



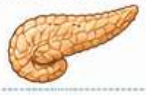
DDSEP Q18, Q19, Q24, Q47 MEMORY:
Insulinoma drops glucose; **gastrinoma** makes acid; **glucagonoma gives diabetes–rash–diarrhea; acinar carcinoma** gives **panniculitis.**

8. AUTOIMMUNE AND DRUG-INDUCED PANCREATITIS

8.1 AUTOIMMUNE PANCREATITIS – TYPE 2



Associated with inflammatory bowel disease, especially **ulcerative colitis.**



Can present with obstructive jaundice and a pancreatic head mass.

Responds well to glucocorticoids.

Important: Do not automatically resect in this context.

8.2 DRUG-INDUCED PANCREATITIS – AZATHIOPRINE

Azathioprine can cause idiosyncratic acute pancreatitis.

Usually occurs early after initiation (weeks to a few months).

Management:
Stop azathioprine immediately.

Do **NOT** rechallenge with azathioprine or 6-mercaptopurine.

DDSEP Q45–Q46 MEMORY:
UC plus pancreatic mass may be type 2 AIP; azathioprine pancreatitis means stop and do not rechallenge.

SECTION II – DDSEP SCENARIO-BASED EXAM TABLE

Integrated rapid-review table for DDSEP Pancreatic Physiology & Disease Questions 1-50.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	Recurrent pancreatitis since childhood with family history	PRSS1	Hereditary pancreatitis = cationic trypsinogen mutation.
Q2	Secretin-stimulated bicarbonate secretion	Duct cells	Secretin → duct cell → bicarbonate-rich fluid.
Q3	Acute pancreatitis etiology search	Transabdominal ultrasound	All AP patients need gallstone assessment.
Q4	CCK-stimulated zymogen secretion	Acinar cells	CCK squeezes enzymes from acinar cells.
Q5	Meal-related intestinal phase of pancreatic secretion	80 percent	Intestinal phase dominates pancreatic secretion.
Q6	Early acute pancreatitis needing fluid resuscitation	Lactated Ringer's	LR is preferred initial fluid.
Q7	Classic asymptomatic serous cystadenoma	Observation	Serous cystadenoma is benign; observe if asymptomatic.
Q8	Common chronic pancreatitis association	Osteoporosis	Bone disease is common in chronic pancreatitis.
Q9	Duodenal secretin release trigger	pH < 4.5	Duodenal acid calls secretin.
Q10	Not a pancreatic enzyme category	Pepsinogen	Pepsinogen is gastric, not pancreatic.
Q11	Post-ERCP pancreatitis next morning	LR 300 cc/hr IV	Treat post-ERCP pancreatitis like AP: aggressive LR.
Q12	Intracellular event after stimulation	Secretin increases cAMP	Secretin classically acts through cAMP.
Q13	Large-duct chronic pancreatitis, duct 11 mm	Puestow procedure	Large duct pain drains surgically.
Q14	Main duct IPMN / duct dilation with pancreatitis	Surgical resection	Main duct involvement has high malignant potential.
Q15	PRSS1 small-duct chronic pancreatitis with severe pain	TPIAT	TPIAT treats selected hereditary small-duct disease.
Q16	Pleural effusion after necrotizing pancreatitis	ERCP with pancreatic duct stent	Pancreaticopleural fistula: bridge the duct leak.
Q17	Massive UGIB from isolated gastric varices after pancreatitis	Splenectomy	Splenic vein thrombosis → isolated gastric varices → spleen.
Q18	Most common functioning pancreatic endocrine tumor	Insulinoma	Functioning NET most often presents with hypoglycemia.
Q19	Refractory ulcers + esophagitis + diarrhea	Gastrinoma	Acid plus diarrhea = ZES.
Q20	2.5 cm cyst with pancreatic duct dilation	EUS-FNA	Duct dilation is a worrisome cyst feature.
Q21	Acute pancreatitis with cholangitis	ERCP	Cholangitis with AP needs biliary decompression.
Q22	<4 weeks interstitial pancreatitis + fluid only	Acute fluid collection	Time <4 weeks, no necrosis → acute fluid collection.
Q23	>4 weeks after necrotizing pancreatitis + mature wall	Walled-off pancreatic necrosis	Late encapsulated necrosis = WON.
Q24	Pancreatic mass + panniculitis + subcutaneous nodules	Acinar cell carcinoma	Acinar carcinoma can cause panniculitis/polyarthritis.
Q25	Chronic pancreatitis + head mass + jaundice, resectable	Whipple procedure	Cancer until proven otherwise; resect if operable.
Q26	>4 weeks necrotizing pancreatitis + symptomatic collection	Walled-off pancreatic necrosis	Encapsulated necrosis after 4 weeks = WON.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q27	Young woman, tail cystic lesion with peripheral calcification	**Solid pseudopapillary tumor**	Young female + solid-cystic tail mass → resect.
Q28	Grape-cluster cyst with central scar	**Serous cystadenoma**	Central scar and microcysts reassure.
Q29	Cyst >3 cm with mural nodule / duct dilation	**EUS-FNA**	Worrisome cyst features need EUS-FNA.
Q30	Incidental 15 mm cyst without high-risk features	**MRI in one year**	Small low-risk cyst: MRI surveillance.
Q31	Locally advanced pancreatic cancer	**Neoadjuvant chemotherapy**	Unresectable/locally advanced disease starts systemic therapy.
Q32	Painless jaundice, weight loss, smoker	**EUS-FNA**	EUS confirms pancreatic mass and obtains tissue.
Q33	Chronic pancreatitis with new weight loss	**Pancreas-protocol CT**	Alarm change in CP: look for cancer.
Q34	Gallstone pancreatitis with CBD dilation and cholangitis	**Urgent ERCP**	Cholangitis overrides everything.
Q35	Asymptomatic pseudocyst after gallstone pancreatitis	**Expectant management with repeat imaging**	No symptoms/infection → observe.
Q36	Early AP with hemoconcentration and high BUN	**LR 20 mL/kg bolus -> 3 mL/kg/hr**	Hemoconcentration = aggressive goal-directed fluids.
Q37	Recurrent idiopathic pancreatitis after negative ERCP	**SPINK1**	SPINK1 links to recurrent pancreatitis.
Q38	Gallstone pancreatitis without obstruction/cholangitis	**ERCP not necessary**	Stones alone do not mandate ERCP.
Q39	Pancreatic adenocarcinoma risk factor	**Cigarette smoking**	Best-established environmental risk.
Q40	Young female, difficult cannulation, repeated PD access	**Pancreatic duct stent**	High-risk post-ERCP pancreatitis: stent the duct.
Q41	Severe AP improving but oral diet worsens symptoms	**Nasojejunal tube feeds**	Feed enterally rather than prolonged NPO.
Q42	Melena after necrotizing pancreatitis, duct bleeding	**IR angio-embolization**	Hemosuccus pancreaticus -> angiography/embolization.
Q43	Chronic pancreatitis with B12 deficiency	**Reduced R-protein breakdown**	Pancreatic proteases free B12 from R-protein.
Q44	Greatest pancreatic cancer risk condition	**PRSS1 mutation**	Hereditary pancreatitis carries very high cancer risk.
Q45	UC patient, pancreatic head mass, AIP pattern	**Glucocorticoids**	Type 2 AIP responds to steroids.
Q46	Crohn's on azathioprine with acute pancreatitis	**Azathioprine**	Stop azathioprine; do not rechallenge.
Q47	Diabetes + diarrhea + rash + pancreatic mass	**Glucagonoma**	Glucagonoma = diabetes, rash, diarrhea, weight loss.
Q48	Necrotizing AP without infection	**Monitor Hct/BUN/Cr and adjust fluids**	No prophylactic antibiotics; enteral feeding when needed.
Q49	Gallstone AP + fever/jaundice/ CBD stone probability	**Urgent ERCP**	Charcot triad with AP = urgent ERCP.
Q50	True statement about hereditary pancreatitis	**Mutation in cationic trypsinogen**	PRSS1 = cationic trypsinogen.

DDSEP 9 - CHAPTER 4
DISEASES OF THE BILIARY TRACT

DDSEP Exam-Oriented Notes and Scenario Framework

Questions 1-50

Structured for rapid review, clinical pattern recognition, and MCQ recall

SECTION I – EXAM PEARL NOTES

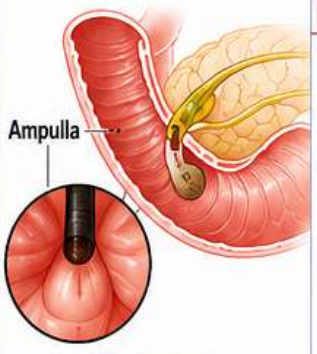
1 Ampullary Lesions: Adenoma Versus Neuroendocrine Tumor

AMPULLARY ADENOMA (SPORADIC)

- May progress to adenocarcinoma.
- If EUS shows small lesion WITHOUT MP, CBD, PD or nodal invasion.

↓

Endoscopic ampullectomy / endoscopic resection



Endoscopic resection

AMPULLARY NEUROENDOCRINE TUMOR (NET)

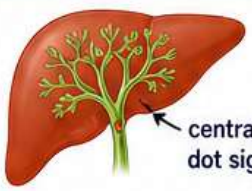
- Biologic behavior is malignant regardless of small size.
- Even small lesions may metastasize.

↓

Pancreaticoduodenectomy (Whipple procedure)

MEMORY (DDSEP Q1–Q2): Adenoma without invasion → endoscopic resection.
Ampullary NET → Whipple because size does not guarantee safety.

2 Choledochal Cysts, Choledochocele, Pancreaticobiliary Maljunction and Caroli Syndrome

Condition	Key Features	Imaging / Clue	Malignancy Risk	Management
Type I Choledochal cyst 	Fusiform extrahepatic bile duct dilation (commonest)		↑ Risk with age (Cholangiocarcinoma)	Cyst excision + Cholecystectomy + Hepaticoenterostomy (Roux-en-Y hepaticojejunostomy) when possible
Type III Choledochocele 	Cystic dilation of the intraduodenal CBD		Lower, but present	Endoscopic sphincterotomy / endoscopic unroofing or excision
Type IVB Choledochal cyst 	Multiple extrahepatic biliary dilations		↑ Risk	Cholecystectomy + Hepaticojejunostomy ± Hepatic resection if intrahepatic disease
Pancreaticobiliary Maljunction 	Long common channel → reflux of pancreatic juice into bile duct		↑ Risk of Gallbladder cancer	Cholecystectomy (mainstay) ± biliary reconstruction
Caroli Syndrome 	Multifocal intrahepatic duct dilation + congenital hepatic fibrosis		↑ Risk of Cholangiocarcinoma	Manage complications, recurrent cholangitis and surveillance for CCA

MEMORY (DDSEP Q3, Q20, Q25, Q30, Q36, Q40, Q49):
Cystic biliary anatomy is malignant until proven otherwise:
resect / drain surgically when extrahepatic dilation is present.

3 Acute Cholangitis, Choledocholithiasis and Acute Cholecystitis

A. ACUTE CHOLANGITIS (TOKYO GUIDELINES)

- Reynolds pentad: Fever, RUQ pain, Jaundice, Hypotension, Altered mental status
- Severe cholangitis or organ dysfunction

URGENT ERCP (within 24 h)

⚠ Do NOT delay for antiplatelet complexity when sepsis is present.

B. ACUTE CHOLECYSTITIS

- Symptomatic cholecystitis with GB wall thickening / pericholecystic fluid
- No strong CBD-stone predictors

↓

SURGICAL CHOLECYSTECTOMY (not diagnostic ERCP)

C. INITIAL APPROACH TO BILIARY PAIN

- RUQ pain ± jaundice
- Mixed hepatocellular / cholestatic LFTs

↓

START WITH RUQ ULTRASOUND

Next step depends on probability of choledocholithiasis

MRCP	EUS	ERCP
		

D. CHOLEDO.CHOLITHIASIS & GALLSTONE PANCREATITIS MANAGEMENT

1. CBD STONE VISUALIZED OR VERY STRONG PREDICTORS

ERCP WITH SPHINCTEROTOMY

↓

THEN CHOLECYSTECTOMY

2. GALLSTONE PANCREATITIS + CHOLANGITIS

↓

URGENT ERCP (Do not delay)

3. GALLSTONE PANCREATITIS WITHOUT OBSTRUCTION / CHOLANGITIS

↓

NO ROUTINE ERCP

4. AFTER ERCP CLEARANCE OF STONES

↓

EARLY CHOLECYSTECTOMY (preferably during same admission if fit)

- ✓ Reduces recurrent biliary events
- ✓ Prevents readmission

MEMORY (DDSEP Q4, Q6, Q11, Q16, Q39):
Cholangitis decompressed by ERCP; cholecystitis fixed by cholecystectomy; ultrasound is the first map.

4 Post-Cholecystectomy Complications and Biliary Instrumentation

CLINICAL SCENARIO	KEY POINTS	BEST NEXT STEP	NOTES / EXPLANATION
1 Post-cholecystectomy bile leak with fever, RUQ pain and a formed collection 	• Formed collection (biloma / abscess) • Need source control and confirmation of bile	**PERCUTANEOUS DRAINAGE**  ERCP for duct decompression (if leak persists)	Percutaneous drainage gives source control and confirms bile. ERCP decompresses the duct but will NOT drain an established infected collection.
2 Early post-operative RUQ pain and fever with suspected duodenal perforation 	• Suspect duodenal perforation / injury • Need to localize leak before endoscopy	**WATER-SOLUBLE UPPER GI CONTRAST STUDY** 	Localize the leak with water-soluble contrast study before endoscopy.
3 Right postetior sectoral duct injury or stricture after cholecystectomy 	• Major duct injury or stricture (often sectoral) • Not suitable for endoscopic therapy	**SURGICAL REPAIR / RECONSTRUCTION** 	Right posterior sectoral duct injury or stricture is often surgical.
Simple cystic duct leak 	• Low-grade leak from cystic duct	**ERCP WITH STENTING OR SPHINCTEROTOMY** 	Most simple cystic duct leaks heal with decreased transpapillary pressure.
4 Hemobilia after liver biopsy or hepatobiliary instrumentation 	• Pain + jaundice + GI bleeding (Quincke triad)	**ANGIOGRAPHY WITH EMBOLIZATION** 	Definitive hemostasis is achieved by angiography with embolization.

MEMORY (DDSEP Q5, Q10, Q38, Q48):
Drain collections, image perforations, embolize hemobilia.

5 Functional Biliary Pain, Sphincter of Oddi and Functional Gallbladder Disorder

A. FUNCTIONAL BILIARY SPHINCTER DISORDER (POST-CHOLECYSTECTOMY)

Requires **ALL**:

- Biliary-type pain after cholecystectomy
- Objective support:
  - Abnormal liver tests OR
  - Bile duct dilation

MILAN PATTERN	
Type I	Pain + abnormal LFTs + duct dilation
Type II	Pain + either abnormal LFTs or duct dilation
Type III	Pain only (normal LFTs and no duct dilation)

Type II (selected cases) → ERCP with manometry

B. SPHINCTER OF ODDI MANOMETRY & MANAGEMENT

MANOMETRY RESULT

ABNORMAL

Biliary sphincterotomy may help

NORMAL

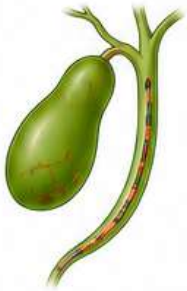
DO NOT cut the sphincter

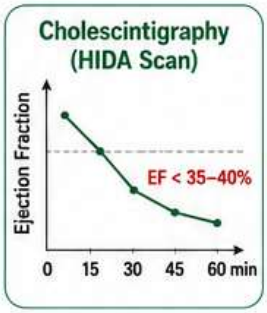
ERCP in this group carries HIGH risk of post-ERCP pancreatitis.

C. FUNCTIONAL GALLBLADDER DISORDER (GALLBLADDER PRESENT)

Requires **ALL**:

- Episodic severe biliary pain
- Normal liver tests and lipase
- No gallstones or structural disease
- Low gallbladder ejection fraction (on cholescintigraphy)





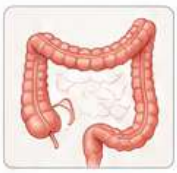
D. KEY POINTS

1. Biliary-type pain WITHOUT abnormal liver tests or duct dilation (Type III pattern) → DO NOT do ERCP for suspected sphincter dysfunction.
2. Manometry is the decision-maker in Type II: abnormal = treat, normal = do NOT cut.
3. Always balance benefit vs. high risk of post-ERCP pancreatitis.

MEMORY (DDSEP Q7–Q8, Q13, Q23):
Pain alone is not a license for ERCP; objective obstruction decides.

6 PSC, Dominant Strictures, Cholangiocarcinoma and Post-Transplant Biliary Disease

1. PSC & COLON SURVEILLANCE

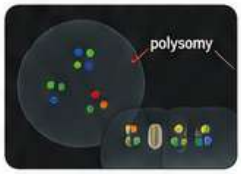


- PSC pattern on cholangiography → trigger colonoscopy with biopsies REGARDLESS of bowel symptoms.
- PSC-IBD → annual surveillance colonoscopy.
- PSC without IBD → periodic colonoscopy to detect occult colitis.

2. SUSPICIOUS HILAR MASS IN PSC

- Avoid percutaneous biopsy of the mass → tumor seeding may preclude transplant protocols.
- Sample nodes if needed.
- Use ERCP brushings / cytology / FISH and staging pathway.

3. DOMINANT STRICTURE & CHOLANGIOCARCINOMA RISK



- Biliary stricture with POLYSOMY on FISH is concerning even if CA 19-9 is low.
- Repeat ERCP with brushings / cytology in a short interval is a common surveillance strategy.

4. EARLY INTRAHEPATIC CHOLANGIOCARCINOMA (CCA)

Cirrhosis + Portal Hypertension

- Early iCCA in a cirrhotic patient with portal hypertension should be discussed at a TRANSPLANT CENTER because resection may be unsafe.

5. POST-LIVER TRANSPLANT BILIARY STRICTURES

- Fever / jaundice with biliary strictures after transplant → Doppler ultrasound of the hepatic artery.
- Hepatic artery thrombosis / stenosis causes NON-ANASTOMOTIC strictures.

6. KEY TAKE-HOME MESSAGES

- PSC means COLONOSCOPY with biopsies.
- Be vigilant for CCA.
- Use ERCP brushings, cytology, FISH and staging pathway.
- No careless needle through the hilum.
- Post-transplant strictures → always check hepatic artery first.

MEMORY (DDSEP Q9, Q18, Q26, Q27, Q29, Q31):
PSC means colonoscopy, CCA vigilance and no careless needle through the hilum.

7 Gallstones, Gallbladder Polyps and Gallbladder Cancer Risk

A. GALLSTONE RISK FACTORS

- Age > 40
- Female sex
- Pregnancy
- Obesity
- Dyslipidemia
- Diabetes mellitus
- Rapid weight loss
- Ileal disease / resection
- Ceftriaxone
- Fibrates
- Calcineurin inhibitors
- Octreotide

- Infliximab is NOT a gallstone risk drug

B. PIGMENT GALLSTONES

BLACK PIGMENT STONES

- Associated with:
  - Hemolysis
  - Sickle cell disease
  - Cirrhosis
  - Cystic fibrosis
  - Ileal disease / resection

BROWN PIGMENT STONES

- Infection / stasis stones
- Typical of recurrent pyogenic cholangitis

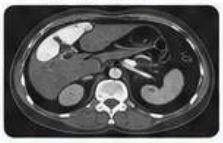
C. GALLBLADDER POLYPS

CHOLECYSTECTOMY INDICATED IF ANY OF THE FOLLOWING:

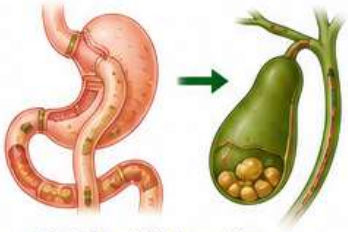
- Polyp ≥ 10 mm
- Symptoms (biliary colic, dyspepsia, etc.)
- Age > 60 years
- PSC (Primary Sclerosing Cholangitis)
- Gallstones present
- Solitary polyp
- Increase in size over time

D. PORCELAIN GALLBLADDER & CANCER RISK

- Porcelain gallbladder, especially selective mucosal calcification, increases risk of gallbladder cancer.
- Contrast-enhanced CT helps define risk and anatomy.



E. RAPID WEIGHT LOSS & GALLSTONES



- Rapid weight loss after Roux-en-Y gastric bypass promotes gallstone formation.
- Ursodeoxycholic acid (UDCA) may reduce gallstone formation.

F. TAKE-HOME POINTS

Cholesterol stones = metabolic (supersaturation)

Black pigment stones = hemolysis / cirrhosis

Brown pigment stones = infection / stasis

Polyp ≥ 10 mm or high-risk features → cholecystectomy

Porcelain gallbladder → increased cancer risk

MEMORY (DDSEP Q14–Q17, Q28, Q34–Q35, Q43, Q47, Q50):
Cholesterol stones are metabolic; black stones are hemolysis/cirrhosis; brown stones are infection/stasis.

8 PBC, IgG4 Disease and Inherited Cholestatic Syndromes

A. PRIMARY BILIARY CHOLANGITIS (PBC)

Diagnosis:

- Cholestatic liver tests (↑ ALP)
- Positive AMA (Anti-mitochondrial antibody)

Liver biopsy:

- Useful when AMA negative
- Or overlap / alternative disease is suspected

First-line therapy:

UDCA 13–15 mg/kg/day

↓

Inadequate Biochemical Response after UDCA
(usually ALP > 1.67 × ULN after 1 year)

↓

Second-line therapy discussion
(Fenofibrate was the answer in DDSEP scenario)

B. IgG4-RELATED DISEASE (SCLEROSING CHOLANGITIS)

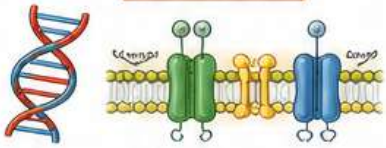
Mimics PSC but typically has:

- Pancreatic enlargement
- Biliary strictures
- Lymphadenopathy
- Other organ involvement (salivary glands, kidneys, etc.)
- Elevated serum IgG4

Check serum IgG4 level

C. PROGRESSIVE FAMILIAL INTRAHEPATIC CHOLESTASIS (PFIC)

PFIC Type III



Cause: ABCB4 (MDR3) mutation

↓

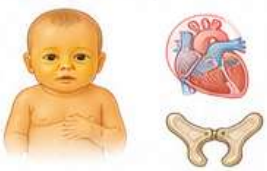
Impaired biliary phospholipid excretion → bile is toxic → cholestasis

D. ALAGILLE SYNDROME

Autosomal dominant (JAG1 mutation)

Features:

- Neonatal conjugated jaundice (ductopenia)
- Peripheral pulmonary stenosis
- Butterfly vertebrae
- Characteristic facies (triangular face, deep-set eyes)
- Posterior embryotoxon



E. KEY DIFFERENCES & PEARLS

Condition	Key Points
PBC	• Cholestasis + AMA positive • UDCA 13–15 mg/kg/day first line • ALP > 1.67 × ULN after 1 year → consider second line (Fenofibrate in DDSEP scenario)
IgG4-SC	• Mimics PSC • Pancreas + other organs + IgG4 ↑ • Steroid-responsive
PFIC III	• ABCB4 (MDR3) mutation • Defective phospholipid secretion → toxic bile
Alagille	• Ductopenia + cholestasis • Pulmonary stenosis + butterfly vertebrae

MEMORY (DDSEP Q21, Q32–Q33, Q37, Q41, Q44):
AMA–PBC–UDCA; IgG4 mimics PSC; ABCB4 is PFIC III; Alagille adds heart and vertebrae.

9 Infectious and Rare Biliary Syndromes

A. AIDS CHOLANGIOPATHY (in severe untreated HIV)

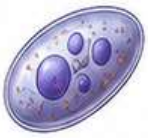
HIV

Occurs in advanced HIV (CD4 < 100 cells/μL)

Clinical & Imaging

- Papillary stenosis / sclerosing cholangitis
- Dilated intrahepatic and extrahepatic bile ducts

Etiology



- *Cryptosporidium parvum* is the commonest organism

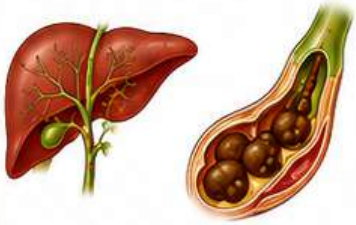
Key Clue

Unknown HIV status → always consider HIV testing

B. RECURRENT PYOGENIC CHOLANGITIS (ORIENTAL CHOLANGITIS)

Classic presentation

- Recurrent fever with rigors
- Jaundice
- Right upper quadrant pain
- Intrahepatic duct dilation
- Brown pigment stones



Etiology / Association

Clonorchis sinensis

- Lives in bile ducts
- Causes chronic inflammation and strictures
- Leads to pigment stones and recurrent cholangitis

Management Principles

Stone extraction | Stricture management | Treat infections

C. CHOLANGIOCARCINOMA RISK FACTORS

- Primary sclerosing cholangitis (PSC)
- Choledochal cysts
- Obesity
- Chronic liver disease / cirrhosis
- Thorotrast exposure
- Liver flukes:
  Clonorchis sinensis
  Opisthorchis viverrini

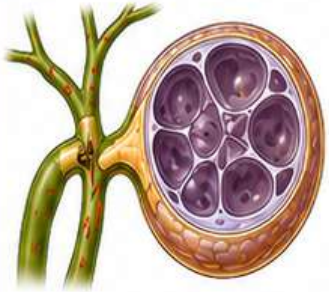
NOT a classic risk factor:

- Fasciola hepatica

Key Message

Chronic inflammation and biliary stasis increase risk of cholangiocarcinoma.

D. BILIARY CYSTADENOMA (RARE BILIARY CYSTIC TUMOR)

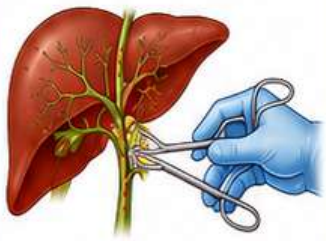


Key Features

- Slow-growing cystic tumor of the bile ducts
- Predominantly in women (middle age)
- May contain ovarian-like stroma
- Potential for malignant transformation (rare)

Management

Complete surgical removal is recommended.



MEMORY (DDSEP Q12, Q19, Q42, Q45–Q46):

HIV → Cryptosporidium cholangiopathy

Oriental Cholangitis → *Clonorchis* + brown stones

Cholangiocarcinoma risk = PSC, cysts, flukes, obesity, chronic liver disease

Biliary cystadenoma → rare but treat by complete excision

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

Integrated rapid-review table for DDSEP 9 Chapter 4: Diseases of the Biliary Tract Questions 1-50.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	Incidental 15 mm sporadic ampullary adenoma, no duct or muscle invasion	Endoscopic resection	Adenoma without invasion: remove endoscopically.
Q2	Small ampullary neuroendocrine tumor without obvious invasion	Pancreaticoduodenectomy	Ampullary NET can metastasize even when small.
Q3	Adult type I choledochal cyst	Surgical resection	Type I cyst: excise + cholecystectomy + hepaticoenterostomy.
Q4	Reynolds pentad / severe cholangitis	Urgent ERCP	Sepsis plus obstruction: decompress within 24 h.
Q5	Fever and RUQ collection shortly after cholecystectomy	Percutaneous drainage tube placement	Drain the infected bile collection first.
Q6	Acute cholecystitis without strong CBD-stone predictors	Surgical referral for cholecystectomy	GB wall + pericholecystic fluid -> surgery.
Q7	Post-cholecystectomy biliary pain with objective support for SOD	ERCP with manometry	Type II SOD: manometry before cutting.
Q8	Biliary pain but no abnormal LFTs or duct dilation	10-30 percent	Pain alone has poor ERCP yield; avoid sphincterotomy.
Q9	PSC-type cholangiogram	Colonoscopy with biopsies	PSC needs colonoscopy regardless of bowel symptoms.
Q10	Post-cholecystectomy fever/RUQ pain with suspected duodenal perforation	Upper GI series	Perforation: water-soluble contrast before endoscopy.
Q11	RUQ pain plus abnormal bilirubin/ ALP/transaminases	Right upper quadrant ultrasound	Ultrasound is the first biliary map.
Q12	Obstructive jaundice; ask non-risk factor for cholangiocarcinoma	Fasciola hepatica infection	CCA flukes are Clonorchis/Opisthorchis, not Fasciola.
Q13	Type II SOD scenario but normal manometry	Do not perform sphincterotomy	No manometric obstruction: do not cut.
Q14	Gallstones in Crohn short-gut patient; identify non-risk drug	Infliximab use	Infliximab is not a gallstone drug.
Q15	Sickle cell disease, post-cholecystectomy dilated CBD	20-30 percent	Hemolysis makes pigment stones; CBD stones recur.
Q16	Choledocholithiasis treated; risk without cholecystectomy	20-40 percent	Clear duct, then remove gallbladder early.
Q17	Symptomatic gallbladder polyp >=10 mm	Cholecystectomy	One centimeter polyp is the surgery threshold.
Q18	PSC with new hilar mass; what to avoid	Percutaneous biopsy of hilar mass	Needle through hilum may seed tumor and block transplant option.
Q19	Refugee with diarrhea, skin lesions and cholangiopathy clues	HIV testing	Think AIDS cholangiopathy.
Q20	Pancreaticobiliary maljunction	Gallbladder cancer	PBM particularly threatens the gallbladder.
Q21	Young patient, recurrent idiopathic pancreatitis and distal CBD stricture	IgG4 disease	IgG4 can mimic PSC and pancreatic cancer.
Q22	Pain after metal biliary stent for pancreatic cancer	Right upper quadrant ultrasound	Metal stent can precipitate cholangitis via cystic duct orifice.
Q23	Biliary pain, normal labs/lipase, low HIDA ejection fraction	Functional gallbladder disorder	Gallbladder present + biliary pain + low EF.
Q24	Gastric outlet obstruction with pneumobilia in elderly patient	Surgical consultation with therapeutic endoscopy consideration	Bouveret syndrome: endoscopy first, surgery often needed.
Q25	Choledochocele presentation	Pancreatitis	Type III cyst most commonly presents with pancreatitis.
Q26	PSC stricture with polysomy on FISH	Repeat ERCP with brushings/cytology in 3 months	FISH polysomy raises CCA concern despite low CA 19-9.

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

Integrated rapid-review table for DDSEP 9 Chapter 4: Diseases of the Biliary Tract Questions 1-50.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q27	Very early ICCA in cirrhosis with portal hypertension	Refer to liver transplantation center	Cirrhotic portal HTN makes resection risky.
Q28	12 mm gallbladder polyp with stones	Recommend laparoscopic cholecystectomy	Polyp >10 mm + stones/age risk: remove gallbladder.
Q29	PSC with ulcerative colitis	Colonoscopy with surveillance biopsies now	PSC-UC: annual surveillance starts now.
Q30	Caroli syndrome	Increased risk for cholangiocarcinoma	Caroli: central dot sign + CCA risk.
Q31	Post-transplant fever/jaundice with biliary complications	Doppler ultrasound of hepatic arteries	Hepatic artery problem causes non-anastomotic strictures.
Q32	PBC diagnosis/therapy statement	UDCA 13-15 mg/kg/day is recommended	AMA + ALP: treat with weight-based UDCA.
Q33	Young cholestasis/pruritus with PFIC III clue	ABCB4	ABCB4/MDR3 moves biliary phospholipid.
Q34	Pregnancy and gallstone mechanism/risk factor	Progesterone	Pregnancy promotes bile stasis and lithogenic bile.
Q35	Black pigment stone risk factor	Cirrhosis	Black stones: hemolysis, cirrhosis, CF, ileal disease.
Q36	Choledochocoele/PBM without duct dilation	Cholecystectomy	PBM: remove gallbladder cancer target.
Q37	Neonatal cholestasis, pulmonary stenosis, butterfly vertebrae	Alagille syndrome	Ductopenia + heart + vertebrae.
Q38	Post-cholecystectomy jaundice/fever with sectoral duct problem	Right posterior duct stricture	Sectoral duct injury often needs surgical expertise.
Q39	CBD stone seen/very strong predictor in symptomatic gallstones	ERCP with sphincterotomy followed by cholecystectomy	Stone in duct: ERCP first, gallbladder next.
Q40	Cystic structure on medial duodenal wall	Type III biliary cyst	Choledochocoele = intraduodenal CBD cyst.
Q41	PSC-like cholangiogram plus pancreatic enlargement/lymphadenopathy	Check serum IgG4 level	IgG4 disease is the steroid-responsive PSC mimic.
Q42	Large multiloculated biliary cystic mass in older woman	Biliary cystadenoma	Ovarian-like stroma; excise completely.
Q43	Porcelain gallbladder concern	CT abdomen/pelvis with IV contrast	Calcified gallbladder: stage anatomy and cancer concern.
Q44	PBC with inadequate UDCA response	Start fenofibrate 145 mg daily	Poor ALP response after UDCA needs add-on therapy.
Q45	Severe HIV cholangiopathy organism	Cryptosporidium parvum	CD4 <100 + papillary stenosis: Crypto.
Q46	Recurrent pyogenic cholangitis association	Clonorchis sinensis	Oriental cholangitis: parasites, infection, stones.
Q47	Recurrent pyogenic cholangitis stone type	Brown pigment stones	Brown = infected stagnant bile.
Q48	GI bleeding after liver biopsy with biliary bleeding	Interventional radiology for embolization	Hemobilia is treated by angiographic embolization.
Q49	Type IVB biliary cyst	Surgical referral for cholecystectomy and hepaticojejunostomy	Extrahepatic multifocal cysts need definitive surgical drainage.
Q50	Gallstone prevention after Roux-en-Y rapid weight loss	Ursodeoxycholic acid	Rapid weight loss stones: UDCA can help prevent.

DDSEP VIRAL HEPATITIS

VIRAL HEPATITIS

DDSEP Exam-Oriented Notes and Scenario Framework

Questions 1-50

Structured for rapid review, clinical pattern recognition, and MCQ recall

SECTION I – EXAM PEARL NOTES

1. HCV THERAPY: REGIMENS, RESISTANCE, RENAL DISEASE AND INTERACTIONS

FOUR KEY MODIFIERS BEFORE CHOOSING A REGIMEN

1. Genotype
2. Cirrhosis status
3. Prior DAA exposure
4. Drug interaction

REGIMEN PEARLS (DDSEP SCENARIOS)

Scenario	DDSEP Answer / Preferred Regimen	Duration
Prior NS5A failure after sofosbuvir/ledipasvir	Sofosbuvir / Velpatasvir / Voxilaprevir	12 weeks
Genotype 3 with compensated cirrhosis	Sofosbuvir / Velpatasvir	12 weeks
Severe renal impairment (eGFR <30 or on dialysis)	Elbasvir / Grazoprevir or Glecaprevir / Pibrentasvir	12 weeks

Note: Current guidance should be checked; sofosbuvir renal labeling has changed since older teaching.

KEY DRUG INTERACTIONS

- High-dose PPI ↓ exposure of ledipasvir/velpatasvir. Avoid if possible; if needed, use equivalent of omeprazole ≤20 mg.
- Amiodarone + sofosbuvir → risk of severe bradycardia. Avoid combination.
- Statins + glecaprevir/pibrentasvir → ↑ statin levels. Limit dose or choose alternative.

MEMORY (DDSEP Q1–Q3, Q8, Q27–Q28, Q31–Q32, Q36, Q49–Q50)

HCV regimen selection = Genotype + Cirrhosis + Prior DAA exposure + Renal function / Drug-interaction screen.

2. ACUTE HCV, HCV EXTRAHEPATIC CLUES AND SURVEILLANCE

ACUTE HCV: DIAGNOSIS

In suspected acute HCV, antibody may still be negative.

↓

HCV RNA is the diagnostic test during the window period.

WHEN TO SEND HCV RNA

Recent injection drug use or needle exposure + acute hepatitis + negative HCV antibody

↓

Send HCV RNA

EXTRAHEPATIC CLUE

Porphyria cutanea tarda is a classic dermatologic clue to HCV.

VACCINE

There is no HCV vaccine.

HCC SURVEILLANCE

Patients with HCV cirrhosis or stage 4 fibrosis require:

Ultrasound + AFP every 6 months

Even after viral cure.

MEMORY (DDSEP Q12, Q18, Q21, Q30, Q38)

Acute HCV is RNA-first; cured cirrhosis is still watched.

3. HBV SEROLOGY, PHASES AND TREATMENT THRESHOLDS

STEP 1: READ THE SEROLOGY PATTERN

- HBsAg
- HBeAg / Anti-HBe
- Anti-HBc (IgM / total)
- Anti-HBs

↓

Then interpret by ALT, HBV DNA, HBeAg status and cirrhosis status.

HBV PHASES & TREATMENT THRESHOLDS

Phase / Situation	Key Features	Treatment / Action
Immune tolerant phase	• HBeAg positive • High HBV DNA (≥10^6 IU/mL) • Normal ALT • Young, endemic area	Monitor No treatment
Immune active HBeAg positive	• HBeAg positive • High HBV DNA • Elevated ALT • Favorable features	Peginterferon in selected compensated patients
HBeAg-negative immune reactivation	• Anti-HBe positive • HBV DNA >2,000 IU/mL • Elevated ALT	Tenofovir or Entecavir
Compensated cirrhosis	• Any detectable HBV DNA (>2,000 IU/mL or even lower) • Compensated	Antiviral therapy indicated
Decompensated cirrhosis	• Decompensated (ascites, HE, variceal bleed, etc.)	Interferon contraindicated Use potent oral NAs + evaluate transplant

KEY POINTS

- Normal ALT + high DNA in HBeAg positive young patient = immune tolerant → watch.
- High ALT + high DNA = immune active → treat.
- HBeAg-negative + DNA >2,000 + high ALT = reactivation → treat.
- Any detectable DNA in compensated cirrhosis = treat.
- Decompensation forbids interferon.

COMMON TARGETS

- DNA suppression
- ALT normalization
- HBeAg seroconversion (where applicable)
- Prevent cirrhosis progression and HCC

MEMORY (DDSEP Q15–Q17, Q22, Q37, Q40–Q41, Q47)

Normal ALT watches; active HBV treats; decompensation forbids interferon.

4. HBV IN PREGNANCY AND VERTICAL TRANSMISSION

AT DELIVERY

Infant receives **BOTH** HBV vaccine + Hepatitis B Immune Globulin (HBIG)

Within 12 hours of birth

HIGH MATERNAL HBV DNA IN 3RD TRIMESTER

Start Tenofovir (TDF) at ~28–32 weeks to reduce vertical transmission risk

EARLY PREGNANCY INACTIVE-CARRIER PATTERN

Repeat HBV DNA before 3rd trimester to decide if antiviral prophylaxis is needed

KEY POINTS

- At delivery: baby gets vaccine + HBIG.
- High maternal HBV DNA (≥200,000 IU/mL): start TDF in 3rd trimester.
- Inactive carrier early in pregnancy: repeat HBV DNA before 3rd trimester.

MEMORY (DDSEP Q6, Q9, Q35)
Baby gets vaccine + HBIG; mother gets tenofovir only if viral load is high.

5. HBV REACTIVATION: THE IMMUNOSUPPRESSION TRAP

THREE RECURRING SETTINGS

- Rituximab / Chemotherapy
- Biologics / Steroids
- DAA treatment for HCV

SCREEN ALL PATIENTS BEFORE IMMUNOSUPPRESSION

- HBsAg
- Anti-HBc (total)
- Anti-HBs
- HBV DNA (when indicated)

Rituximab & high-dose chemotherapy = HIGH RISK

INTERPRETATION & MANAGEMENT

1) HBsAg POSITIVE

- Active chronic HBV.
- Start antiviral prophylaxis/treatment before immunosuppression.

2) HBsAg NEGATIVE, Anti-HBc POSITIVE

- Resolved infection (usually anti-HBs positive).
- cccDNA persists → can reactivate.
- If high-risk regimen → consider prophylaxis and monitor.
- Continue monitoring after immunosuppression ends.

3) DAA THERAPY FOR HCV

- Before starting HCV DAA therapy:
- Assess HBV status and HBV DNA (when indicated).

KEY REMINDERS

- Resolved infection = HBsAg (–), Anti-HBc (+), usually Anti-HBs (+).
- Strong immunosuppression can reactivate HBV.
- Monitor ALT, HBV DNA during and after immunosuppression.
- Start prophylaxis early; do not wait for reactivation.

MEMORY (DDSEP Q14, Q23–Q26, Q29, Q34, Q48)
Core antibody remembers; immunosuppression reawakens.

6. HEPATITIS D AND ACUTE LIVER FAILURE

HEPATITIS D (HDV)

- HDV requires HBV (HBsAg must be present).
- Severe hepatitis in an HBsAg-positive patient with suppressed HBV DNA → think delta superinfection.
- HDV superinfection = high risk of fulminant hepatitis.

CLINICAL CLUE

HBsAg (+)

HBV DNA low/undetectable but ALT/AST very high + jaundice → Suspect HDV

ACUTE LIVER FAILURE (ALF)

Any acute hepatitis with:

- ☐ Encephalopathy (any grade)
- ☐ INR ≥ 1.5
- ☐ ± Elevated bilirubin

ALF = MEDICAL EMERGENCY
Transfer immediately to a liver transplant center

KEY REMINDERS

- HBV DNA low but ALT huge → think delta.
- INR elevation + encephalopathy = ALF → transplant center.

MEMORY (DDSEP Q4–Q5)
HBV DNA low but ALT huge → think delta; INR + encephalopathy → transplant center.

7. HAV AND HEV: ACUTE VIRAL HEPATITIS PATTERNS

HEPATITIS A VIRUS (HAV)

TRANSMISSION
Fecal–oral transmission. Travel, food, shellfish, outbreaks.

DIAGNOSIS
Positive HAV IgM = acute infection.

CHRONIC INFECTION
No chronic infection.

SEVERE DISEASE RISK
Chronic liver disease, especially chronic HCV, increases risk of severe HAV. Vaccinate susceptible chronic liver disease patients.

POST-EXPOSURE PROPHYLAXIS
Time-sensitive (within 2 weeks). Immune globulin is used in selected older or higher-risk exposed contacts.

HEPATITIS E VIRUS (HEV)

CLINICAL PATTERN
Acute hepatitis similar to HAV.

PREGNANCY RISK
Pregnancy (especially 2nd–3rd trimester) → increased risk of severe disease and mortality.

TRANSMISSION
Fecal–oral transmission, usually via contaminated water.

GENOTYPES
Genotype 3 (zoonotic) is common in developed countries.

MANAGEMENT
Immunocompetent patients (e.g., genotype 3) → usually observed / supportive care.

KEY TAKEAWAYS

- HAV = food / travel / outbreak; IgM positive; no chronicity.
- Chronic liver disease ↑ risk of severe HAV → vaccinate susceptible patients.
- HAV post-exposure prophylaxis is time-sensitive; IG for selected high-risk contacts.
- HEV = acute hepatitis like HAV.
- Pregnancy increases severity risk in HEV.
- Genotype 3 HEV in immunocompetent patients → supportive care.

MEMORY (DDSEP Q7, Q11, Q13, Q19–Q20, Q43–Q45)
HAV is food / travel / outbreak; HEV threatens pregnancy.

8. OTHER VIRAL HEPATITIDES IN SPECIAL HOSTS

HSV HEPATITIS

Postpartum or immunocompromised patient with fever, leukopenia, very high aminotransferases and acute liver failure pattern.

CLINICAL CLUES
- Recent delivery (postpartum) or immunocompromised state
- Fever
- Leukopenia
- Very high AST/ALT (often >1000 IU/L)
- Acute liver failure pattern (coagulopathy ± encephalopathy)

ACTION
Start IV ACYCLOVIR immediately – do not wait for confirmation.

CMV HEPATITIS / GI DISEASE

Transplant patient with diarrhea, cytopenia and hepatitis.

CLINICAL CLUES
- Solid organ or hematopoietic stem cell transplant recipient
- Diarrhea / colitis
- Cytopenia (anemia, leukopenia, thrombocytopenia)
- Elevated aminotransferases (hepatitis pattern)

MANAGEMENT
IV GANCICLOVIR or VALGANCICLOVIR according to severity and organ involvement.

KEY POINTS

- HSV hepatitis is rare but a medical emergency.
- Postpartum ALF pattern = think HSV → start acyclovir now.
- CMV disease in transplant patients often presents with GI symptoms, cytopenia and hepatitis.
- Early diagnosis and treatment improve outcomes.

MEMORY (DDSEP Q42, Q46)
Postpartum ALF = acyclovir now; transplant diarrhea + hepatitis = CMV treatment.

9. ONE-GLANCE SEROLOGY REMINDERS

MARKERS	HBsAg	Anti-HBs	Anti-HBc (total)	IgM Anti-HBc	INTERPRETATION	KEY POINT
	Negative	Positive	Negative	Negative	**Vaccine immunity** Immune due to vaccination.	Surface antibody alone = vaccination.
	Negative	Positive	Positive	Negative	**Resolved natural HBV infection** Past infection, immune now.	Core antibody means the immune system has seen real HBV.
	Positive	Negative or Positive	Positive	± Positive	**Current HBV infection (acute or chronic)** Use IgM anti-HBc, ALT, and duration > 6 months = chronic.	HBsAg + anti-HBc = active HBV infection.
	—	—	—	**IgM Positive**	**Acute HAV infection** IgM = acute infection.	HAV total (IgG) alone = prior exposure or vaccination.
	—	—	—	—	**HCV Ab positive + RNA negative** Previous cleared/treated infection, not active HCV.	Antibody alone does not indicate active HCV.

KEY SEROLOGY PEARLS

- HBsAg negative + anti-HBs positive + anti-HBc negative → vaccine immunity.
- HBsAg negative + anti-HBs positive + anti-HBc positive → resolved natural HBV infection.
- HBsAg positive + anti-HBc positive → acute or chronic HBV.
- HAV IgM → acute HAV. HAV total alone → prior exposure or vaccination.
- HCV Ab positive + RNA negative → previous cleared/treated infection, not active HCV.

MEMORY (DDSEP Q10, Q23–Q25, Q34)

Surface antibody alone vaccinates; core antibody means the immune system has seen real HBV.

10. FAST EXAM TRAPS

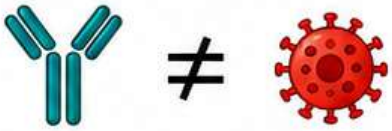
1 NO INTERFERON IN DECOMPENSATED HBV CIRRHOSIS

Interferon is **contraindicated** in decompensated cirrhosis.

2 DO NOT MISS HBV SCREENING BEFORE IMMUNOSUPPRESSION

Screen **all patients** before rituximab, chemotherapy, biologics or prolonged high-dose steroids.

3 DO NOT USE HCV ANTIBODY ALONE TO EXCLUDE ACUTE HCV



Acute HCV can be Ab negative. Use **HCV RNA** to diagnose acute infection.

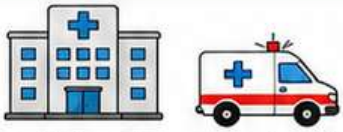
4 DO NOT STOP HCC SURVEILLANCE AFTER HCV CURE IF CIRRHOSIS

Cirrhosis remains → continue **HCC surveillance** (US ± AFP every 6 months).

5 DO NOT FORGET HDV IN SEVERE HEPATITIS WITH LOW HBV DNA

HBsAg positive + severe hepatitis + low HBV DNA → **think HDV (delta) infection.**

6 DO NOT DELAY TRANSPLANT REFERRAL IN ALF



Encephalopathy + coagulopathy (INR elevation) in acute hepatitis → **urgent transplant-center referral.**

MASTER MEMORY

HCV = RNA and interactions

HBV = serology and reactivation

HAV / HEV = acute

HSV / CMV = special hosts

SECTION II — DDSEP SCENARIO-BASED EXAM TABLE

Integrated rapid-review table for DDSEP Viral Hepatitis Questions 1-50.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	GT1a HCV; peg/RBV nonresponse + sofosbuvir/ledipasvir relapse	Sofosbuvir/velpatasvir/ voxilaprevir x12 wk	NS5A-experienced failure: add the third drug class.
Q2	Treatment-naive genotype 3 HCV, compensated cirrhosis, no RAS	Sofosbuvir/velpatasvir x12 wk	GT3 + compensated cirrhosis still needs full effective DAA course.
Q3	HCV with stage 5 CKD on hemodialysis	Elbasvir/grazoprevir x12 wk	Original DDSEP logic: avoid sofosbuvir in severe CKD; modern rules have evolved.
Q4	Known HBV on entecavir; severe hepatitis but HBV DNA suppressed	Superimposed hepatitis D	HDV flare often suppresses HBV DNA but worsens liver injury.
Q5	Acute hepatitis progresses to encephalopathy + INR 2.9	Emergent transfer to liver transplant center	Any acute hepatitis + INR + encephalopathy = ALF pathway.
Q6	HBsAg-positive mother presents at delivery	HBIG + HBV vaccine for infant	Vaccine alone is not immediate protection.
Q7	Raw shellfish; HAV question	Most Americans exposed by age five	DDSEP answer retained; core fact: HAV is fecal-oral and non-chronic.
Q8	GT1a HCV retreatment with compensated cirrhosis	12 weeks therapy	Cirrhosis prevents shortcut therapy.
Q9	34-week pregnancy; HBsAg+, HBeAg+, very high HBV DNA	Start tenofovir	High viral load late pregnancy: lower maternal DNA before delivery.
Q10	Mild ALT elevation; HBsAg negative, anti-HBc positive, anti-HBs positive	Biopsy would show steatosis/ballooning	Natural HBV immunity is not chronic HBV; look for MASLD/NASH.
Q11	Pregnancy + acute hepatitis; HAV/HBV/HCV negative	HEV IgM likely positive	HEV is dangerous in pregnancy, especially later trimesters.
Q12	Acute hepatitis picture but HCV antibody negative	HCV RNA	Antibody can be negative early, RNA wins the window period.
Q13	College traveler; acute hepatitis syndrome	HAV IgM	Travel + fever/GI prodrome + ALT >1000: check HAV IgM.
Q14	Rituximab/prednisone then fatal hepatitis	Prior HBV exposure	Screen HBV before B-cell depleting or major immunosuppression.
Q15	Young Korean woman; HBeAg+, HBV DNA high, ALT normal	Immune tolerant phase	High DNA alone in young vertical HBV is not always treatment.
Q16	Same immune-tolerant patient asks long-term plan	Monitor ALT and HBV DNA q6 months	Normal ALT: observe and watch for immune activation.
Q17	HBeAg+ HBV genotype A; high ALT, low DNA, compensated	Peginterferon alfa-2a	Best seroconversion chance when genotype A + high ALT + low DNA.
Q18	Which hepatotropic virus lacks vaccine?	Hepatitis C	HAV and HBV are vaccine-preventable; HCV is not.
Q19	HAV outbreak; who risks fulminant HAV?	Patients with chronic HCV	Chronic liver disease converts HAV from nuisance to danger.
Q20	Acute HAV; household exposure control	HAV immune globulin for unvaccinated contacts	Post-exposure protection is time-sensitive.
Q21	Porphyria cutanea tarda clue	Hepatitis C antibody	PCT is an HCV extrahepatic clue.
Q22	Decompensated HBV cirrhosis	Do not start peginterferon	Decompensation: oral nucleos(t)ide, not interferon.
Q23	Past HBV exposure; HBsAg negative before infliximab	No action required	Source answer retained; intensity of immunosuppression determines reactivation strategy.
Q24	HBsAg positive inactive carrier before infliximab	Entecavir prophylaxis/ treatment	HBsAg positive + biologic = prevent HBV reactivation.
Q25	Leukemia chemo; resolved HBV infection	HBV prophylaxis through chemo and 12-18 mo after	cccDNA sleeps; chemotherapy wakes it.
Q26	Before starting DAA therapy for HCV	HBV DNA	DAA therapy can unmask/reactivate HBV.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q26	Before starting DAA therapy for HCV	HBV DNA	DAA therapy can unmask/reactivate HBV.
Q27	HCV GT1b cirrhosis; high-dose PPI	Grazoprevir/elbasvir	Ledipasvir/velpatasvir absorption falls with high gastric pH.
Q28	Prior sofosbuvir/ledipasvir failure	Sofosbuvir/velpatasvir/voxilaprevir	NS5A failure: rescue with VOX-containing triple regimen.
Q29	HBsAg negative/anti-HBc positive; prednisone ≥20 mg >4 wk	Start tenofovir	Moderate/high-dose steroids can reactivate HBV.
Q29	HCV cirrhosis or stage 4 fibrosis after cure	Ultrasound + AFP every 6 months	SVR does not discharge cirrhotics from HCC surveillance.
Q31	Treatment-naive HCV genotype 2	Glecaprevir/pibrentasvir x8 wk	Simple noncirrhotic HCV: short pangenotypic regimen.
Q32	HCV genotype 3 with CKD stage IV	Glecaprevir/pibrentasvir	Protease/NS5A option fits renal impairment in DDSEP logic.
Q33	Decompensated HCV cirrhosis, MELD 28, variceal bleed/HRS concern	Liver transplant evaluation	Treating HCV is not enough when MELD is high.
Q34	Post-IVIG patient develops anti-HBc positivity	Reassurance	IVIG can passively transfer core antibody.
Q35	HBsAg+ pregnant woman at 28 weeks, likely inactive carrier	Measure HBV DNA before third trimester	Do not decide vertical prophylaxis from first-trimester DNA alone.
Q36	Recent decompensated HCV cirrhosis with ascites	Ledipasvir/sofosbuvir + ribavirin	DDSEP regimen retained; decompensated HCV needs specialist regimen.
Q37	HBeAg+ HBV on entecavir; seroconverts	Continue entecavir	After HBeAg seroconversion, give consolidation before stopping.
Q38	Recent IVDU; acute hepatitis; HCV Ab may be negative	HCV RNA	Acute HCV diagnosis is RNA-based.
Q39	Asian man >40 with chronic HBV	Liver ultrasound	HBV can cause HCC even without cirrhosis.
Q40	HBeAg negative immune-reactivation HBV	Tenofovir	ALT high + HBV DNA >2,000 → treat.
Q41	HBV patient wants highest HBsAg loss chance	Tenofovir + peginterferon	Source answer: modern practice rarely uses combination routinely.
Q42	Postpartum fever, leukopenia, ALF-like hepatitis	IV acyclovir	HSV hepatitis: treat before confirmation.
Q43	Acute HEV genotype 3 in immunocompetent older man	Observation	Immunocompetent HEV is usually supportive care.
Q44	Acute viral hepatitis; HAV IgM positive	Hepatitis A	HAV: acute, fecal-oral, not chronic.
Q45	Healthy 50-year-old exposed to household HAV	HAV immune globulin	Older exposed adult: IG within 2 weeks.
Q46	Kidney transplant; diarrhea + hepatitis + cytopenia	IV ganciclovir	CMV hepatitis/GI disease in transplant: ganciclovir/valganciclovir.
Q47	Compensated HBV cirrhosis with low-level viremia	Entecavir	Cirrhosis lowers the threshold to treat HBV.
Q48	Post-chemotherapy HBV reactivation with ALF	Transfer for transplant evaluation	Chemo + missed HBV prophylaxis can present as ALF.
Q49	HCV GT1a on amiodarone	Glecaprevir/pibrentasvir	Avoid sofosbuvir with amiodarone because of severe bradycardia risk.
Q50	HCV GT3; CAD patient on atorvastatin	Sofosbuvir/velpatasvir	Avoid G/P with statin interaction when safer alternative exists.

METABOLIC, HEREDITARY, INFLAMMATORY AND VASCULAR DISEASES OF THE LIVER

DDSEP Exam-Oriented Notes and Scenario Framework

Questions 1-50

Structured for rapid review, clinical pattern recognition, and MCQ recall

SECTION I – EXAM PEARL NOTES

1 MASLD/MASH: DIAGNOSIS, TREATMENT LIMITS AND MORTALITY

DDSEP uses **NAFLD/NASH** terminology. Modern wording is **MASLD/MASH**, but the exam logic is unchanged: **distinguish simple steatosis from steatohepatitis and fibrosis risk.**

HISTOLOGIC CRITERIA FOR MASH (NASH)

1. **Steatosis** (≥5% of hepatocytes)
2. **Lobular inflammation**
3. **Ballooned hepatocytes**

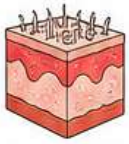
Mallory hyaline may occur but is **NOT** required.

Vitamin E (800 IU/day) can improve steatosis/inflammation in selected biopsy-proven NASH patients.

DDSEP EMPHASIS: It has **NOT** improved fibrosis.

Cardiovascular disease is the commonest long-term cause of death in NASH cirrhosis.

KEY POINT: Survival risk is often cardiovascular, not only hepatic.



Psoriasis is an associated inflammatory–metabolic risk factor for NAFLD/MASLD.

ASSOCIATED RISK: Immune-inflammatory burden promotes steatosis and fibrosis.

Microvesicular steatosis suggests special metabolic/toxic mechanisms; DDSEP answer: **Cholesteryl ester storage disease.**

EXAM CLUE: Think metabolic storage disease (CESD) in microvesicular steatosis.

MEMORY PEARL

DDSEP Q1–Q5:
MASH = FAT + LOBULAR INFLAMMATION + BALLOONING
Survival risk is often **CARDIOVASCULAR**, not only **HEPATIC.**

2 BENIGN HYPERBILIRUBINEMIA AND HEREDITARY CHOLESTASIS

CONDITION	PATTERN	LAB FEATURES	KEY CLINICAL POINT	DDSEP PEARL
Gilbert Syndrome	Isolated unconjugated hyperbilirubinemia	• Mild ↑ unconjugated bilirubin • Normal liver enzymes • Normal hematocrit	Young, healthy patient. Benign. **Reassurance only.**	Classic exam scenario: isolated indirect bilirubin with normal labs.
Dubin–Johnson Syndrome	Conjugated hyperbilirubinemia	• ↑ Conjugated bilirubin • Normal liver enzymes • Normal serum bile acids	Defect in canalicular transport (MRP2). **Benign course.**	Conjugated bilirubin with normal enzymes and normal bile acids.
Crigler–Najjar Type 1	Marked unconjugated hyperbilirubinemia	• Severe ↑ unconjugated bilirubin from birth • Normal liver enzymes	UGT1A1 complete failure. **Risk of kernicterus.**	Neonatal onset, very high indirect bilirubin, kernicterus risk.
PFIC Type 2	Cholestatic	• ↑ Conjugated bilirubin • Low/normal GGT	Associated with **INCREASED HCC RISK.**	This subtype carries the cancer-risk.
PFIC Type 3	Cholestatic	• ↑ Conjugated bilirubin • **ELEVATED GGT**	Defect in MDR3. Classically ↑ GGT.	PFIC subtype classically associated with ↑ GGT.

MEMORY PEARL

DDSEP Q6–Q9:
ISOLATED BILIRUBIN PATTERNS = TRANSPORTER / ENZYME QUESTIONS;
PFIC TYPE 2 = CANCER-RISK SUBTYPE.

3. HEREDITARY HEMOCHROMATOSIS

DIAGNOSIS

- C282Y homozygosity (HFE gene)
- ↑ Transferrin saturation (>45%)
- ↑ Ferritin

C282Y homozygosity with elevated transferrin saturation/ferritin supports hereditary hemochromatosis.

STAGING TRIGGER

Ferritin
>1000 ng/mL
is the exam trigger to stage fibrosis.

Stage fibrosis, often by liver biopsy in DDSEP scenario.

RESPONSE TO IRON DEPLETION

Improves:
- Cardiomyopathy
- Diabetes mellitus

Poor or minimal improvement:
- Arthropathy (joint disease)
- Hypogonadism
- Established cirrhosis

Phlebotomy helps heart/diabetes more than joints/gonads or advanced liver disease.

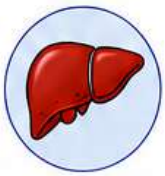
HEPCIDIN PHYSIOLOGY

- Most hemochromatosis forms → **LOW** hepcidin
- Ferroportin disease (type 4) → **NORMAL** hepcidin

Exception tested in exams: ferroportin disease has normal hepcidin.

MEMORY PEARL

DDSEP Q10–Q12:



FERRITIN >1000 = STAGE THE LIVER; PHLEBOTOMY HELPS HEART/DIABETES MORE THAN JOINTS/GONADS.

4. PBC, PSC, IgG4 DISEASE AND SARCOID

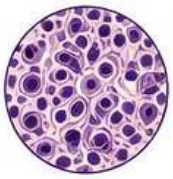
	PRIMARY BILIARY CHOLANGITIS (PBC)	PRIMARY SCLEROSING CHOLANGITIS (PSC)	IgG4-RELATED SCLEROSING CHOLANGITIS	HEPATIC SARCOIDOSIS
Typical Patient	• Middle-aged woman (40–60 years)	• Young to middle-aged man	• Middle-aged man (commoner than PSC)	• Adults (variable)
Key Laboratory	• Cholestatic ALP ↑↑ • AMA positive (>90%) • IgM elevated	• Cholestatic ALP ↑↑ • p-ANCA may be positive	• Cholestatic ALP ↑↑ • Serum IgG4 elevated	• Cholestatic pattern (ALP predominant) • ACE may be elevated
Key Associations	• Autoimmune diseases (esp. Sjögren, thyroid)	• Ulcerative colitis (classic association)	• Other IgG4-related disease: pancreatitis, retroperitoneal fibrosis, salivary/lacrimal gland enlargement	• Pulmonary sarcoidosis common
Histology / Biopsy Clue	• Florid duct lesion (destruction of small intrahepatic bile ducts)	• Periductal concentric "onion-skin" fibrosis	• Lymphoplasmacytic infiltrate rich in IgG4+ plasma cells and storiform fibrosis	• Noncaseating granulomas
Key Management Points	• UDCA improves outcomes (survival, transplant-free survival) • Does **NOT** improve: fatigue or osteoporosis	• No proven medical therapy to halt progression • Manage complications • **Screen gallbladder annually (↑ risk of cholangiocarcinoma)**	• Steroid-responsive • Treat systemic disease • Distinguish from PSC to avoid unnecessary transplant	• Treatment is **NOT** automatic • Treat only if symptomatic or with significant hepatic impairment
Key Complication / Monitoring	• Total bilirubin is the **STRONGEST survival predictor**	• Cholangiocarcinoma risk • Gallbladder carcinoma risk → Screen gallbladder annually	• Relapse when steroids tapered too quickly	• Portal hypertension (if advanced disease)

MEMORY PEARL

DDSEP Q13–Q19:
PBC = AMA/IgM/BILIRUBIN; PSC = UC/ONION-SKIN/GALLBLADDER RISK; IgG4 = STEROID-RESPONSIVE MIMIC.

5. LIVER TRANSPLANT COMPLICATIONS

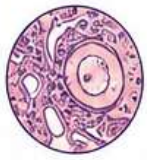
A. ACUTE CELLULAR REJECTION



- Immune attack on the graft.
- Histology (hallmark):
  Ductulitis
  (inflammation of bile ducts)
  Endotheliitis
  (endothelial inflammation)

Clinical clues: ↑ LFTs, fever, weight gain.
Treatment: High-dose steroids ± other immunosuppression.

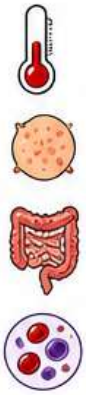
B. CHRONIC REJECTION



- Progressive injury → obliterative arteriopathy.
- Evolves toward ductopenia (loss of bile ducts) and cholestasis.
- Presents with persistent cholestasis, pruritus.
- May require retransplantation.

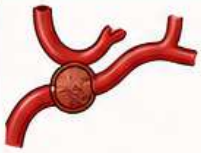
Key feature: Ductopenia and cholestasis.

C. GVHD AFTER LIVER TRANSPLANT (RARE BUT HIGH-YIELD)



- Occurs after intense immunosuppression.
- Classic triad + cytopenias:
  - ✓ Fever
  - ✓ Rash
  - ✓ Secretory diarrhea
  - ✓ Cytopenias (anemia, thrombocytopenia, leukopenia)
- Treat by reducing immunosuppression and supportive care.

D. EARLY HEPATIC ARTERY THROMBOSIS (HAT) AFTER TRANSPLANT



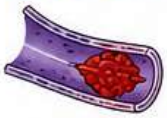
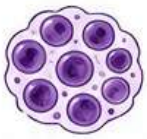
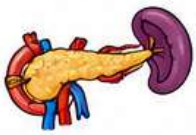
- Presents early after transplant with graft failure physiology: ↑ LFTs, painful graft, biliary complications.
- Doppler US: **ABSENCE OF HEPATIC ARTERY FLOW** is decisive.
- Requires **URGENT** evaluation for retransplantation.

Early HA thrombosis = **THREAT TO GRAFT SURVIVAL.**

MEMORY PEARL

DDSEP Q20–Q22:
REJECTION HITS DUCTS (DUCTULITIS) / ENDOTHELIUM (ENDOTHELIITIS);
GVHD ADDS RASH + DIARRHEA + CYTOPENIA;
EARLY HEPATIC ARTERY THROMBOSIS THREATENS GRAFT SURVIVAL.

6. PORTAL AND HEPATIC VASCULAR DISORDERS

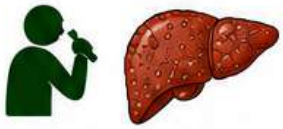
	A. SCHISTOSOMIASIS (PRESINUSOIDAL PORTAL HYPERTENSION) 	B. BUDD–CHIARI SYNDROME 	C. PORTAL VEIN THROMBOSIS (PVT) IN CIRRHOSIS 	D. NONCIRRHOTIC PVT 	E. SPLENIC VEIN THROMBOSIS 
Pathophysiology	Presinusoidal portal hypertension (periportal fibrosis).	Hepatic venous outflow obstruction (hepatic veins or IVC).	Thrombus in portal vein on a background of cirrhosis.	PVT without cirrhosis → think underlying prothrombotic state.	Thrombosis of splenic vein (usually from chronic pancreatitis).
Key Clinical Features	Splenomegaly, varices, hypersplenism. Usually **NO** ascites in early disease.	Abdominal pain, ascites, hepatomegaly, ± jaundice.	Often asymptomatic or abdominal pain; may present with worsening portal hypertension.	Symptoms of portal hypertension or mesenteric ischemia (acute cases).	Left-sided (sinistral) portal hypertension: isolated gastric varices.
Important Investigations	WHVP, FHVP, HVPG may all be **NORMAL** despite clinically significant portal hypertension.	Doppler US (first test): absent/abnormal hepatic vein flow, collateral circulation. **Caudate lobe hypertrophy** is a classic imaging clue.	1. Triphasic CT/MRI or Doppler US. 2. Exclude **HCC tumor thrombus.** 3. Evaluate extent of thrombosis.	Work up for myeloproliferative disorder: **JAK2 V617F** mutation, additional hematologic evaluation.	Contrast CT or Doppler US: thrombosed splenic vein with collateral flow (short gastric veins).
Management Principles	Endoscopic management of varices. Treat schistosomiasis if active infection.	Treat underlying cause (anticoagulation if thrombotic). Consider TIPS or liver transplant in refractory cases.	1. Exclude HCC tumor thrombus. 2. Manage high-risk varices. 3. **Anticoagulate** when appropriate.	**Anticoagulation** for acute PVT. Address underlying prothrombotic condition.	Usually managed conservatively. Treat underlying pancreatic disease.
Key Exam Point	**Presinusoidal disease may be "invisible" to HVPG.**	**Caudate lobe hypertrophy is a classic clue.**	**Rule out HCC tumor thrombus before anticoagulation.**	**Unprovoked PVT → think MPN (JAK2+) logic.**	**Causes isolated gastric varices.**

MEMORY PEARL

DDSEP Q23–Q25, Q41–Q43, Q46:
PRESINUSOIDAL DISEASE MAY HIDE FROM HVPG; UNPROVOKED PVT ASKS FOR MPN/JAK2 LOGIC;
SPLENIC VEIN THROMBOSIS → ISOLATED GASTRIC VARICES.

7. ALCOHOL-ASSOCIATED LIVER DISEASE AND ISCHEMIC INJURY

A. SEVERE ALCOHOLIC HEPATITIS

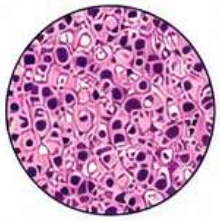


- Recent heavy alcohol use (within weeks–months)
- Jaundice
- **AST > ALT** (usually both <400 U/L)
- Systemic inflammation (fever, leukocytosis, ↑ CRP)

TREATMENT (WHEN INDICATED)

- Corticosteroids (e.g., prednisolone 40 mg/day for 28 days) when: Maddrey DF ≥32 or MELD ≥21
- **NO** contraindication (infection, GI bleeding, renal failure, uncontrolled diabetes, psychosis, etc.)

B. ALCOHOLIC HEPATITIS BIOPSY FEATURES

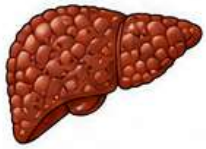


- **Ballooning** degeneration of hepatocytes
- **Mallory–Denk hyaline**
- **Neutrophilic lobulitis**
- **Pericellular** (chicken wire) fibrosis

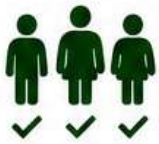
KEY POINT

Diagnosis is clinical; biopsy supports severity and excludes other causes.

C. ALCOHOL-RELATED CIRRHOSIS



ABSTINENCE FROM ALCOHOL GIVES THE GREATEST SURVIVAL BENEFIT AT ALL STAGES.



KEY POINT

Even advanced cirrhosis can show meaningful survival improvement with complete abstinence.

D. ISCHEMIC HEPATITIS (SHOCK LIVER)

- Occurs after cardiac failure, shock, respiratory failure, or major vascular events.
- AST/ALT often **>1000** U/L (may reach several thousands)
- LDH markedly elevated
- Histology: Centrilobular **(zone 3) necrosis**

TREATMENT

- Supportive care
- Restore perfusion and oxygenation
- Treat underlying cause

NOT a primary liver disease.

E. ISCHEMIC CHOLANGIOPATHY

- Due to ischemic injury of bile ducts
- Strongly associated with liver transplantation (hepatic arterial flow is essential for bile ducts)
- Presents with cholestasis after transplant

KEY POINT

Ischemic cholangiopathy = biliary strictures from hepatic arterial insufficiency.

MEMORY PEARL

DDSEP Q26–Q28, Q38–Q40:

ALCOHOLIC HEPATITIS = JAUNDICE + AST > ALT + MALLORY HYALINE;

ISCHEMIC HEPATITIS = SHOCK PHYSIOLOGY + ZONE 3 NECROSIS.

8. WILSON DISEASE: ACUTE AND CHRONIC EXAM PATTERNS

A. ACUTE WILSONIAN HEPATITIS

- Young patient (usually <30 years)
- Psychiatric / neurologic features (change in behavior, depression, tremor, dystonia, speech problems)
- Jaundice with hemolysis pattern
- **Low alkaline phosphatase (ALP) relative to bilirubin**
- Coombs-negative hemolytic anemia is common

KEY CLUE

Young psychiatric patient + jaundice + hemolysis + low ALP → Think Wilson disease until proven otherwise.

B. CHRONIC (HEPATIC OR NEUROPSYCHIATRIC) WILSON DISEASE

- Presents as unexplained liver disease or neuropsychiatric symptoms
- May have low ALP, ↑ AST/ALT
- Kayser–Fleischer ring (copper deposition in Descemet's membrane)
- 24-hour urinary copper >100 mcg supports diagnosis

DIAGNOSIS SUPPORT

- 24-hour urinary copper >100 mcg
- Low ceruloplasmin (<20 mg/dL)
- ↑ Hepatic copper on biopsy (>250 mcg/g dry weight)
- ATP7B mutation (if available)

C. MONITORING ON CHELATION THERAPY

- Chelation drugs: D-penicillamine or Trientine
- Monitor 24-hour urinary copper to assess treatment efficacy
- Target range (DDSEP): 200 – 500 mcg / 24 hours
- <200 mcg → may be under-treatment
- >500 mcg → may be over-treatment or poor compliance
- Also monitor: CBC, urinalysis, liver tests, and clinical status

KEY POINT

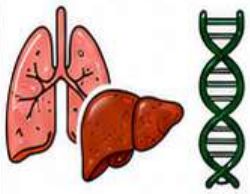
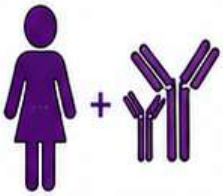
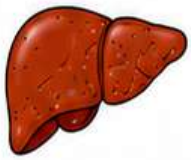
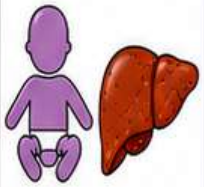
Maintain urinary copper 200–500 mcg/24 h to balance efficacy and toxicity.

MEMORY PEARL

DDSEP Q48–Q50:

LOW ALP + HEMOLYSIS + YOUNG PSYCHIATRIC PATIENT = WILSON DISEASE UNTIL PROVEN OTHERWISE.

9. GENETIC, AUTOIMMUNE AND PEDIATRIC METABOLIC LIVER CLUES

	A. ALPHA-1 ANTITRYPSIN (A1AT) DEFICIENCY	B. AUTOIMMUNE HEPATITIS (AIH)	C. CYSTIC FIBROSIS LIVER DISEASE (CFLD)	D. GAUCHER DISEASE (TYPE 1)	E. GLYCOGEN STORAGE DISEASE TYPE I (VON GIERKE)	F. TYROSINEMIA TYPE I
						
KEY CLINICAL CLUES	• COPD (emphysema), especially in young nonsmokers • Unexplained liver disease / cholestasis • Family history may be present	• Female predominance • Autoimmune background (thyroiditis, IBD, etc.) • ↑ Transaminases • ↑ Globulin / IgG • Anti-smooth muscle antibody (ASMA) +	• Known cystic fibrosis • Cholestatic pattern (↑ GGT, ALP) • Biliary disease common in CF	• Eastern European or Ashkenazi Jewish ancestry • Hepatosplenomegaly • Bone pain, fractures, avascular necrosis	• Fasting hypoglycemia • Seizures • Growth failure • Hepatomegaly	• French-Canadian ancestry • Infant: failure to thrive, vomiting, rickets, liver dysfunction • Hypoglycemia
PATHO-PHYSIOLOGY	• Misfolded A1AT retained in hepatocytes • PAS-positive, diastase-resistant globules → Liver injury	• Immune-mediated interface hepatitis	• Biliary plugging with inspissated secretions → cholestasis, focal biliary cirrhosis	• Glucocerebrosidase deficiency → accumulation of glucocerebroside in macrophages	• Glucose-6-phosphatase deficiency → impaired glycogen breakdown and gluconeogenesis	• Fumarylacetoacetate hydrolase deficiency → accumulation of toxic metabolites (succinylacetone)
DIAGNOSIS / TESTS	• Low serum A1AT level • Phenotyping (Pi*ZZ classic) • Liver biopsy: PAS-positive, diastase-resistant globules	• Elevated IgG • ASMA positive (Type 1 AIH) • Liver biopsy: interface hepatitis with plasma cell infiltrate	• CFTR mutation • Imaging: irregular bile ducts, lobular heterogeneity • May progress to portal hypertension	• Low glucocerebrosidase activity • Genetic testing (GBA mutations)	• Low glucose, lactic acid, uric acid, triglycerides • Hepatocyte glycogenosis on biopsy • Genetic testing	• Elevated succinylacetone in urine or blood (diagnostic) • Liver/kidney dysfunction
TREATMENT / KEY POINT	• No specific therapy for liver disease • A1AT augmentation for lung disease • Consider transplant if advanced liver disease	• Steroids (induction) • Azathioprine (AZA) is standard steroid-sparing therapy • If AZA pancreatitis → Mycophenolate mofetil (MMF)	• Ursodeoxycholic acid (UDCA) is the tested medication answer • Nutritional support • Manage complications	• Enzyme replacement therapy (imiglucerase, etc.) • Treat bone disease and complications	• Frequent high-complex carbohydrate feeds • Avoid fasting • Risk of hepatic adenomas and HCC	• NTBC (nitisinone) • Low tyrosine / phenylalanine diet • Early treatment prevents liver failure and HCC
	SIGNATURE PAIR: A1AT ↔ COPD	SIGNATURE PAIR: AIH ↔ ASMA	SIGNATURE PAIR: CFLD ↔ UDCA	SIGNATURE PAIR: GAUCHER ↔ GLUCOCEREBROSIDASE	SIGNATURE PAIR: GSD I ↔ FASTING HYPOGLYCEMIA + ADENOMA / HCC	SIGNATURE PAIR: TYROSINEMIA I ↔ SUCCINYLACETONE

MEMORY PEARL

DDSEP Q29–Q37, Q47:

A1AT = COPD + PAS GLOBULES;
AIH = ASMA + AZATHIOPRINE;
CFLD = CHOLESTATIC + UDCA;
GAUCHER = GLUCOCEREBROSIDASE;
GSD I = FASTING HYPOGLYCEMIA + ADENOMA/HCC;
TYROSINEMIA I = SUCCINYLACETONE.

SECTION II — DDSEP SCENARIO-BASED EXAM TABLE

Integrated rapid-review table for DDSEP Chapter 6 Questions 1-50.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	Echogenic liver; suspected MASH/NASH; biopsy diagnostic features	Ballooning + lobular inflammation + steatosis	NASH/MASH is histology: fat + inflammation + ballooning.
Q2	Biopsy proven NASH stage 1; treatment question	Vitamin E does not improve fibrosis	Vitamin E may improve activity, not fibrosis.
Q3	NASH cirrhosis; long-term mortality	Cardiovascular disease	In NASH, the heart usually kills before the liver.
Q4	Condition linked to NAFLD	Psoriasis	Psoriasis is a metabolic-inflammatory NAFLD clue.
Q5	Microvesicular steatosis question	Cholesteryl ester storage disease	Microvesicular fat: think mitochondrial/ toxic/metabolic storage.
Q6	Young healthy man; isolated indirect bilirubin; normal enzymes/CBC	No further evaluation or treatment	Gilbert = benign isolated unconjugated bilirubin.
Q7	Dubin–Johnson syndrome true statement	Normal liver enzymes and bile acids	Conjugated bilirubin transport defect, not progressive liver disease.
Q8	Crigler–Najjar true statement	Type 1 raises unconjugated bilirubin	UGT1A1 failure: unconjugated bilirubin from birth, kernicterus risk.
Q9	Hereditary cholestasis true statement	PFIC type 2 increases HCC risk	PFIC2 can get HCC early; PFIC3 has high GGT.
Q10	C282Y homozygote; high transferrin saturation/ferritin >1000	Proceed with liver biopsy	Ferritin >1000 in HH asks: how much fibrosis?
Q11	Hemochromatosis systemic complications	Cardiomyopathy and diabetes usually improve	Iron removal helps heart/diabetes more than joints/gonads/cirrhosis.
Q12	Hepcidin in hemochromatosis variants	Ferroportin disease has normal hepcidin	Most HH = low hepcidin; ferroportin is the exception.
Q13	Female; ALP high, AMA high, IgM high	Risk of varices without cirrhosis	PBC can cause presinusoidal portal HTN.
Q14	PBC prognosis question	Total bilirubin is best survival predictor	In PBC, bilirubin tells prognosis.
Q15	UDCA benefit in PBC	Reduces risk of variceal bleeding	UDCA improves disease course; it does not fix fatigue/osteoporosis.
Q16	UC patient with cholestatic liver tests; PSC histology	Periductal concentric fibrosis	PSC = onion-skin periductal fibrosis
Q17	New PSC cirrhosis + UC; surveillance choice	Annual gallbladder ultrasound	PSC watches gallbladder, cholangiocarcinoma and colon.
Q18	IgG4-related sclerosing cholangitis	Corticosteroids are first-line	PSC scars; IgG4 often melts with steroids.
Q19	Hepatic sarcoidosis pattern	High ALP with normal/ mild AST/ALT	Sarcoid liver is granulomatous cholestatic ALP disease.
Q20	8 months after liver transplant; elevated enzymes	Ductulitis and endotheliitis	Acute cellular rejection = ducts + endothelium.
Q21	Post-liver transplant fever, cytopenia, secretory diarrhea, rash	Graft-versus-host disease	GVHD after liver transplant: rash + diarrhea + cytopenias.
Q22	2 days post-transplant; no hepatic artery flow, severe injury	Relist Status 1A for retransplant	Early hepatic artery thrombosis can be graft-fatal.
Q23	Schistosomiasis portal hypertension; HVPG question	WHVP normal; FHVP normal; HVPG normal	Presinusoidal portal HTN can have normal HVPG.
Q24	Young woman on OCP; acute ascites/ RUQ pain/edema	Hepatic ultrasound with Doppler	Budd–Chiari is venous outflow: Doppler first.
Q25	Characteristic imaging feature of Budd–Chiari	Caudate lobe hypertrophy	Caudate drains separately into IVC, so it hypertrophies.
Q26	Heavy alcohol use; jaundice; severe alcoholic hepatitis	Corticosteroids	Severe AH without contraindication: steroids.
Q27	Alcoholic hepatitis biopsy	Ballooning degeneration and Mallory hyaline	Alcoholic hepatitis = ballooning + Mallory-Denk + neutrophils.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q28	Alcohol-related cirrhosis; survival intervention	Abstinence	Alcoholic liver disease survival begins with abstinence.
Q29	COPD + unexplained cirrhosis/ mild AST/ALT	Alpha-1 antitrypsin phenotyping	COPD plus liver disease: phenotype A1AT.
Q30	A1AT biopsy: PAS positive diastase-resistant globules	Misfolded A1AT accumulates in liver	A1AT liver injury is toxic gain-of-function in hepatocytes.
Q31	Autoimmune hepatitis with cirrhosis; maintenance drug	Azathioprine	AIH standard long-term steroid-sparing drug is azathioprine.
Q32	Young woman, autoimmune background, high globulin/hepatitis	Anti-smooth muscle antibody	AIH clues: female, autoimmune disease, high IgG, ASMA.
Q33	AIH on prednisone/azathioprine then pancreatitis	Mycophenolate mofetil	Azathioprine pancreatitis: stop and switch; do not rechallenge.
Q34	Cystic fibrosis child: hepatosplenomegaly, cholestatic labs	Ursodeoxycholic acid	CF liver disease is biliary/cholestatic; UDCA often used.
Q35	Eastern European child, fractures + hepatosplenomegaly	Glucocerebrosidase	Gaucher = glucocerebrosidase deficiency, bones + spleen + liver.
Q36	Infant fasting hypoglycemia/seizures; long-term risk	Hepatocellular carcinoma	GSD I causes adenomas that can transform.
Q37	Infant fasting hypoglycemia; biopsy finding	Glycogenosis of hepatocytes	Von Gierke liver is packed with glycogen.
Q38	Post-vascular surgery: AST/ALT >2000, modest ALP, INR normal	Supportive care only	Ischemic hepatitis: fix perfusion; no magic liver drug.
Q39	Post-CABG ischemic hepatitis; biopsy lesion	Zone 3 hepatocyte necrosis	Zone 3 is most hypoxic and dies first.
Q40	Risk factor for ischemic cholangiopathy	Liver transplantation	Bile ducts depend on hepatic artery; check artery after transplant strictures.
Q41	Cirrhosis with portal vein thrombosis and varices	Band varices, then anticoagulate	In cirrhosis PVT: exclude tumor thrombus and protect varices.
Q42	Noncirrhotic portal vein thrombosis	Myeloproliferative disorder	Unprovoked PVT = search JAK2/MPN.
Q43	Acute noncirrhotic PVT; 6 months anticoagulation	About 50% recanalization	Anticoagulation prevents extension and may recanalize.
Q44	HSCT day 15; weight gain, RUQ pain, bilirubin >2	Ursodeoxycholic acid may reduce risk	SOS/VOD prevention often tested with UDCA.
Q45	HSCT day 18; weight gain, RUQ pain, jaundice	Sinusoidal obstruction syndrome	Post-HSCT + painful hepatomegaly + fluid retention = SOS.
Q46	Hematemesis with isolated gastric varices; pancreatitis history	Splenic vein thrombosis	Chronic pancreatitis -> splenic vein thrombosis -> IGV1.
Q47	French-Canadian infant; hypoglycemia, jaundice, failure to thrive	Elevated succinylacetone	Tyrosinemia I: succinylacetone + HCC risk.
Q48	Young adult psychiatric disease; acute hepatitis, low ALP, hemolysis	Wilson disease	Low ALP plus hemolysis in acute hepatitis = Wilson.
Q49	Psychiatric disease + chronic liver disease; diagnostic test	24-hour urine copper 180 mcg	Urine copper >100 mcg/day supports Wilson.
Q50	Wilson therapy monitoring target	Urine copper 200–500 mcg/24 h	On chelation, urine copper stays elevated but controlled.

DDSEP 9 – CHAPTER 7

CIRRHOSIS AND LIVER TRANSPLANTATION

DDSEP Exam-Oriented Notes and Scenario Framework

Questions 1-50

Structured for rapid review, clinical pattern recognition, and MCQ recall

SECTION I – EXAM PEARL NOTES

1 CIRRHOSIS: COMPENSATED vs DECOMPENSATED AND FIRST WORK-UP

Cirrhosis can often be diagnosed clinically and radiologically; **liver biopsy is not mandatory when the picture is clear.** — DDSEP Q1

COMPENSATED CIRRHOSIS

No ascites, variceal bleed, encephalopathy or jaundice.

Survival is much better than after decompensation.

DDSEP Q2

DECOMPENSATED CIRRHOSIS

- Ascites
- Variceal bleeding
- Hepatic encephalopathy
- Jaundice

Survival significantly worse after first decompensating event.

DDSEP Q2

NEW CIRRHOSIS: FIRST WORK-UP

1. **ASSESS AETIOLOGY** — History, risk factors, serology, autoimmune markers, metabolic tests, imaging as indicated. — DDSEP Q3
2. **ASSESS STAGE / SYNTHETIC FUNCTION** — Child–Pugh, MELD/Na, albumin, bilirubin, INR, platelets. — DDSEP Q43
3. **SCREEN FOR PORTAL HYPERTENSION** — Upper GI endoscopy for varices. — DDSEP Q3
4. **HCC SURVEILLANCE** — Ultrasound liver ± AFP every 6 months. — DDSEP Q43

The commonest cause of portal hypertension overall is cirrhosis; remember non-cirrhotic causes when the stem is atypical. — DDSEP Q28

MEMORY: New cirrhosis asks four things: cause, stage, varices, HCC surveillance.

2 VARICES: PRIMARY, ACUTE AND SECONDARY PROPHYLAXIS

NSBB lower portal pressure through:
- β1 reduction in cardiac output
- β2-mediated reduction in splanchnic inflow

DDSEP Q4, Q44

PRIMARY PROPHYLAXIS

Who?
Patients with large varices (no prior bleeding).

What?
NSBB (e.g., propranolol, nadolol, carvedilol) OR Endoscopic Variceal Ligation (EVL).

When NSBB is unsafe?
In asthma, severe hypotension or intolerance → choose EVL.

DDSEP Q5

ACUTE VARICEAL BLEED

Immediate Management Package

1. **RESUSCITATE** — IV access, fluids, oxygen, restrictive transfusion (Hb 7–8 g/dL).
2. **ANTIBIOTICS** — IV antibiotics for all cirrhotic GI bleeds.
3. **VASOACTIVE THERAPY** — Start early: Terlipressin / Octreotide / Somatostatin.
4. **URGENT ENDOSCOPY** — Within 12 hours for diagnosis and endoscopic therapy.

DDSEP Q7, Q33

SECONDARY PROPHYLAXIS

Who?
Patients after a variceal bleed.

What?
EVL (serial sessions) **PLUS NSBB** (e.g., propranolol, nadolol, or carvedilol).

Goal:
Prevent rebleeding which carries high mortality.

DDSEP Q6

CARVEDILOL DOSE

For variceal prophylaxis, maximum dose classically capped at 12.5 mg/day in DDSEP-style questions.

DDSEP Q32

VERY LOW PLATELETS

Very severe thrombocytopenia (e.g., <20–30 ×10⁹/L) before urgent endoscopy may require platelet transfusion.

DDSEP Q35

TRANSFUSION STRATEGY

Restrictive transfusion strategy in cirrhotic GI bleed: Target Hb 7–8 g/dL (avoid over-transfusion).

DDSEP Q33

KEY MEMORY

Variceal bleed package = Resuscitate, Antibiotics, Vasoactive drug, Endoscopy; Prevent rebleed with EVL + NSBB.

CORE PRINCIPLE: Detect early, prevent first bleed, treat acute bleed promptly, prevent rebleed.

3 EARLY TIPS AND PORTAL HYPERTENSIVE BLEEDING

EARLY / PRE-EMPTIVE TIPS (Tested in High-Risk Variceal Bleeding)

Indications:

1. Child C (10–13 points), with variceal bleeding
2. Child B, with active bleeding after initial control

Goal: Reduce rebleeding and improve survival.

DDSEP Q8

REFRACTORY HEPATIC HYDROTHORAX

Do NOT insert chest tubes (Avoid – exam red flag).

- Consider TIPS
- Refer for liver transplant evaluation

DDSEP Q10

RECTAL BLEEDING AFTER NEGATIVE EGD

Bright red rectal bleeding with negative EGD

↓

Still requires **LOWER ENDOSCOPIC EVALUATION**

(e.g., colonoscopy / flexible sigmoidoscopy)

DDSEP Q26

MEMORY:

- Early TIPS saves high-risk bleeders: Child C (10–13) or Child B with active bleeding after control.
- Chest tube in hepatic hydrothorax is an exam **RED FLAG: AVOID IT.**

4 ASCITES, SBP, AKI AND HEPATIC ENCEPHALOPATHY

1. NEW ASCITES

Step 1: Diagnostic paracentesis

Step 2: If cirrhotic ascites

- Sodium restriction (<2 g/day)
- Start diuretics: Spironolactone ± Furosemide

DDSEP Q9, Q11

2. SAAG & ASCITIC PROTEIN (DIFFERENTIAL)

Cause	SAAG	Ascitic Protein
Cirrhotic (Portal HTN)	≥ 1.1 g/dL	Low (<2.5 g/dL)
Cardiac (CHF / Constrictive Pericarditis)	≥ 1.1 g/dL	High (≥2.5 g/dL)

Cardiac ascites = High SAAG + High protein

DDSEP Q12

3. SPONTANEOUS BACTERIAL PERITONITIS (SBP)

Definition:

Ascitic PMN count ≥ 250 /mm³

Management:

- IV antibiotics
- Add IV albumin in appropriate high-risk settings (reduces AKI and mortality)

DDSEP Q41

4. LARGE-VOLUME PARACENTESIS (LVP)

If ≥ 5 L ascites removed:

Give IV albumin (6–8 g per liter of ascites removed)

↓

Prevents paracentesis-induced circulatory dysfunction (PICD)

DDSEP Q40

5. AKI IN CIRRHOSIS: FIRST STEPS

1. Stop diuretics, lactulose (temporarily), NSAIDs and other nephrotoxins
2. Culture work-up: blood, urine and paracentesis (if ascites present)
3. Expand volume with IV albumin (1 g/kg/day up to 100 g/day for 2 days)
4. Assess response and consider Hepatorenal syndrome if no improvement
5. Early nephrology & hepatology input

DDSEP Q16, Q34

6. HEPATIC ENCEPHALOPATHY (HE)

COMMON TRIGGERS

- Infection (most common)
- GI bleeding
- Constipation
- Dehydration / overdiuresis
- Sedatives, benzodiazepines
- Narcotics, alcohol

MANAGEMENT

Treat / remove the precipitating factor

Lactulose: Titrate to 2–3 soft bowel motions per day

Recurrent / persistent HE: **Add Rifaximin 550 mg twice daily**

DDSEP Q14, Q15, Q27

MEMORY PEARLS

1 ADMITTED CIRRHOSIS + ASCITES = TAP FIRST

Always do diagnostic paracentesis before diuretics or antibiotics.

2 CONFUSION IN CIRRHOSIS = TREAT HE AND HUNT THE TRIGGER

Treat encephalopathy and actively search for the precipitating cause.

5. HCC AND LIVER LESIONS IN CIRRHOSIS

1. NEW LIVER LESION IN CIRRHOSIS

Do **NOT** reassure or use AFP alone.

↓

Do DYNAMIC MULTIPHASIC CT or MRI

(arterial, portal venous, delayed phases)

DDSEP Q36

2. HCC TREATMENT DEPENDS ON TUMOUR, LIVER FUNCTION & PORTAL HYPERTENSION

Scenario	Key Features	Preferred Treatment
Solitary tumour, accessible 	• Child A • No portal hypertension • Adequate future liver remnant	**RESECTION** 
Within Milan criteria (1 tumour ≤ 5 cm or up to 3 tumours ≤ 3 cm) without vascular invasion 	• Any Child class (usually B/C) • No macrovascular invasion • Acceptable performance status	**TRANSPLANT PATHWAY**  (± Bridging therapy)
Intermediate stage (multinodular, no vascular invasion)	• Good PS • Preserved function	Loco-regional therapies (TACE, TARE, etc.)
Advanced stage (vascular invasion or extrahepatic spread) 	• Any Child class • Macrovascular invasion or extrahepatic spread	**SYSTEMIC THERAPY**

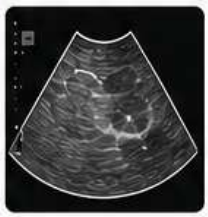
DDSEP Q18, Q37, Q38

3. MACROVASCULAR INVASION

is a **CONTRAINDICATION** to liver transplant.

DDSEP Q50

4. HEMANGIOMA IN THE DIFFERENTIAL



Hyperechoic lesion on ultrasound with progressive peripheral/ heterogeneous fill-in on contrast is **NOT** classic HCC.

DDSEP Q42

MEMORY PEARLS

- Cirrhosis + nodule = **DYNAMIC IMAGING** (CT/MRI).
- Single, resectable HCC + Child A + **NO PORTAL HYPERTENSION** = **RESECT.**
- Within **MILAN** criteria = **TRANSPLANT PATHWAY.**

6. PORTAL VEIN THROMBOSIS AND VASCULAR LOGIC

1. CIRRHOTIC PVT: APPROACH

1. Define extent of thrombosis (Doppler US ± CT/MRI)
2. Exclude HCC tumour thrombus (dynamic imaging, AFP)
3. Protect high-risk varices (NSBB or EVL as indicated)
4. Anticoagulate when appropriate (no absolute contraindication and reasonable bleeding risk)
5. Monitor response and reassess regularly

DDSEP Q39, Q41

2. NON-CIRRHOTIC UNPROVOKED PVT

Think underlying **MYELOPROLIFERATIVE DISORDER.**

Ask for **JAK2** mutation (or CALR/MPL if JAK2 negative), CBC, and consider bone marrow evaluation.

DDSEP Q42

3. ANTICOAGULATION OUTCOME IN ACUTE NON-CIRRHOTIC PVT

Six months of anticoagulation in acute non-cirrhotic PVT results in approximately **50% RECANALISATION.**

DDSEP Q43

4. CONGESTIVE HEPATOPATHY / CARDIAC LIVER DISEASE

Portal hypertension in cardiac disease behaves differently from cirrhosis.

Pressure measurements (HVPG/RVSP or wedge pressure) may be needed to confirm diagnosis and guide management.

DDSEP Q29, Q30

KEY DIFFERENCES: PVT WITH vs WITHOUT CIRRHOSIS

PVT WITHOUT CIRRHOSIS

- ✓ Consider myeloproliferative disorders (e.g., JAK2+).
- ✓ Long-term anticoagulation is standard.
- ✓ Higher recanalisation rate.

VS

PVT WITH CIRRHOSIS

- ✓ Exclude HCC tumour thrombus.
- ✓ Manage portal hypertension before anticoagulation.
- ✓ Weigh bleeding risk (individualised approach).

MEMORY PEARLS

- **PVT WITHOUT CIRRHOSIS = THINK MYELOPROLIFERATIVE DISEASE.**
- **PVT WITH CIRRHOSIS = EXCLUDE TUMOUR THROMBUS, PROTECT VARICES, THEN ANTICOAGULATE WHEN APPROPRIATE.**

7 TRANSPLANT REFERRAL, EXCEPTIONS AND CONTRAINDICATIONS

1. WHEN TO REFER

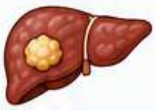
Refer for transplant evaluation at **FIRST DECOMPENSATION**, including:

- First variceal bleed
- Ascites
- Hepatic encephalopathy
- Jaundice
- Hepatorenal syndrome

Regardless of a deceptively **LOW MELD.**

DDSEP Q17

2. MELD EXCEPTION THEMES


Hepatocellular carcinoma (HCC)


Hepatopulmonary syndrome (HPS)


Portopulmonary hypertension (POPH)


Familial amyloid polyneuropathy (FAP)

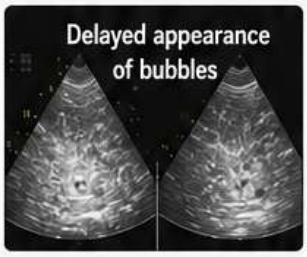

Polycystic liver disease


Primary hyperoxaluria (type 1)

DDSEP Q19, Q20

3. HEPATOPULMONARY SYNDROME (HPS)

- Hypoxaemia
- Delayed bubbles on contrast echo



O_2 $PaO_2 \leq 60$ mmHg is a classic exception threshold.

DDSEP Q20, Q49

4. PORTOPULMONARY HYPERTENSION (POPH)

Pulmonary hypertension in portal hypertension.

Severe disease can preclude transplant, especially very high mean pulmonary artery pressure.

MEAN PULMONARY ARTERY PRESSURE (mPAP)

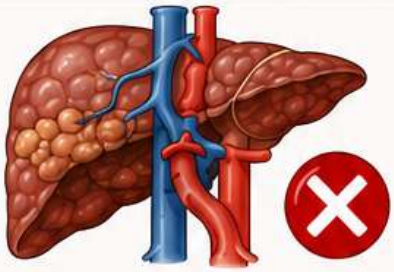
Very high mPAP

↓

Can preclude transplant.

DDSEP Q21, Q48

5. CONTRAINDICATION



Advanced HCC with **macroscopic vascular invasion** (portal vein / hepatic vein / IVC) is a **CONTRAINDICATION** to liver transplantation.

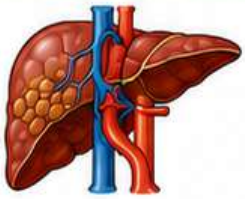
DDSEP Q50

MEMORY PEARLS

- **FIRST DECOMPENSATION** = TRANSPLANT DISCUSSION.
- **HPS** PRIORITISES.
- SEVERE **POPH** CAN PROHIBIT.

8 POST-TRANSPLANT LIVER TEST ABNORMALITIES AND LONG-TERM ISSUES

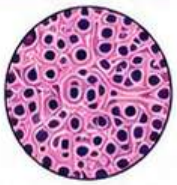
1. POST-TRANSPLANT ABNORMAL LFTs



- Abnormal liver tests require **vascular and biliary imaging.**
- Liver biopsy is needed when rejection vs recurrent disease remains uncertain.

DDSEP Q22–Q24

2. ACUTE CELLULAR REJECTION



Acute cellular rejection is a **histological diagnosis.**

HCV — Recurrent hepatitis C and low tacrolimus can **mimic or coexist** with rejection.

DDSEP Q22–Q24

3. POST-TRANSPLANT DE NOVO MALIGNANCIES

Skin cancers

PTLD (post-transplant lymphoproliferative disorder)

Prostate cancer is less characteristic as the DDSEP answer.

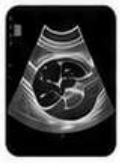
DDSEP Q25

4. PREGNANCY AFTER LIVER TRANSPLANT

Pregnancy after liver transplantation can be successful, but **hypertensive disorders are increased.**

DDSEP Q47

5. KEY POINT



Imaging first (Doppler/ MRCP).

If cause is still not clear, proceed to **liver biopsy.**

DDSEP Q22–Q24

MEMORY PEARL

POST-LT ABNORMAL LFTs: DOPPLER/MRCP FIRST, BIOPSY WHEN THE CAUSE IS STILL NOT CLEAR.

9 OBESITY AND METABOLIC LIVER DISEASE IN TRANSPLANT CONTEXT

1. OBESITY AND METABOLIC LIVER DISEASE

Severe obesity with metabolic syndrome and complications can be best managed by **BARIATRIC SURGERY ASSESSMENT** if liver disease is not decompensated.

DDSEP Q46

2. IN CIRRHOSIS: AVOID UNNECESSARY PPI

Avoid **unnecessary PPIs** in cirrhosis.

DDSEP Q40

3. AVOID INTERVENTIONS THAT WORSEN RENAL / ELECTROLYTE STATUS

Avoid interventions that worsen **renal / electrolyte status** unless clearly indicated.

DDSEP Q40

MEMORY PEARL

IN CIRRHOSIS, THE EXAM OFTEN REWARDS AVOIDING HARM:
NO CHEST TUBE FOR HYDROTHORAX,
NO OVER-TRANSFUSION, NO UNNECESSARY PPI,
NO MISSED DIAGNOSTIC TAP.

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

Rapid recall table: read the scenario clue, say the answer, then lock the pearl.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	Known cured HCV; new cirrhosis assessment	Abdominal ultrasound	Cirrhosis diagnosis/surveillance often begins with imaging; biopsy is not always needed.
Q2	Compensated cirrhosis, no varices or decompensation	12 years	Compensated cirrhosis has much longer survival than decompensated disease.
Q3	New cirrhosis; no prior bleed	Upper endoscopy	New cirrhosis = screen for varices unless clearly ruled out by accepted non-invasive criteria.
Q4	Mechanism of non-selective beta-blockers	Reduction in portal pressure	NSBB reduce portal inflow: beta-1 lowers CO, beta-2 causes splanchnic vasoconstriction.
Q5	Large varices; relative hypotension and asthma	Endoscopic band ligation	Primary prophylaxis: NSBB or EVL; choose EVL when NSBB contraindicated.
Q6	Survived acute esophageal variceal bleed	Band ligation plus nadolol	Secondary prophylaxis = EVL + NSBB.
Q7	Cirrhosis admitted with melena	Antibiotics	Acute GI bleed in cirrhosis: antibiotics + vasoactive drug + restrictive transfusion + early endoscopy.
Q8	Variceal bleed; high-risk Child class / active bleed logic	Early TIPS	Early/pre-emptive TIPS within 72 h for Child C 10-13 or Child B with active bleeding.
Q9	New ascites in alcoholic cirrhosis	Furosemide plus spironolactone	Ascites: sodium restriction + spironolactone/ furosemide; fluid restrict only if severe hyponatraemia.
Q10	Refractory hepatic hydrothorax	Liver transplant evaluation	Hydrothorax: avoid chest tube; consider TIPS and transplant evaluation.
Q11	Hospitalized cirrhotic with ascites	Diagnostic paracentesis	All admitted cirrhotics with ascites need diagnostic tap; SBP = PMN >=250/mm3.
Q12	Ascites with high SAAG and high protein context	Cardiac ascites	SAAG + protein separates cirrhosis from cardiac ascites.
Q13	Cirrhosis with GI bleed; SBP prophylaxis indication	Gastrointestinal bleed	Clear antibiotic prophylaxis triggers: acute GI bleed and prior SBP; selected high-risk ascites.
Q14	HE precipitant question; least likely option	Volume overload	HE precipitants: infection, bleeding, constipation, dehydration/overdiuresis, sedatives/narcotics.
Q15	Recurrent hepatic encephalopathy despite lactulose	Add rifaximin	Recurrent HE: lactulose titrated to 2-3 stools/day + rifaximin.
Q16	Cirrhosis with AKI after ascites/diuretics	Volume expand with albumin	AKI in cirrhosis: stop diuretics/nephrotoxins, rule out SBP, give albumin challenge.
Q17	First decompensation: variceal bleed	Liver transplant evaluation	First decompensation warrants transplant referral even if MELD is not dramatic.
Q18	HCC in HBV patient	Liver transplantation evaluation	HCC management asks: transplant candidacy, resection if preserved function/no portal HTN, bridge if listed.
Q19	MELD exception concepts; primary hyperoxaluria stem	Refractory ascites	MELD exceptions include HCC, HPS, POPH, amyloid, polycystic liver disease, hyperoxaluria - not routine ascites.
Q20	Hepatopulmonary syndrome MELD exception threshold	PaO2 <60 mmHg	HPS: hypoxaemia + intrapulmonary vascular dilatation; exception when severe.
Q21	Portopulmonary hypertension transplant risk	Mean pulmonary artery pressure >50 mmHg	Severe POPH can preclude transplant unless improved with therapy.
Q22	2 months post-transplant; mixed liver enzyme rise	Liver biopsy	Acute cellular rejection is a biopsy diagnosis after vascular/biliary imaging.
Q23	Post-transplant HCV patient with abnormal enzymes	Liver biopsy	Recurrent HCV vs rejection often cannot be separated safely without biopsy.
Q24	Low tacrolimus + recurrent HCV + prior rejection	Liver biopsy	Post-transplant abnormal LFTs: image first, then biopsy if vascular/biliary issues excluded.
Q25	Least likely de novo malignancy after LT	Prostate cancer	Post-transplant cancers: skin, lymphoma/PTLD, lung/oropharyngeal in smokers more typical.
Q26	Cirrhosis/ascites with BRBPR after negative EGD	Lower endoscopy	Bleeding source still needs localisation; do not assume varices after negative EGD.
Q27	Cirrhotic with recurrent confusion	Admit for infection/electrolyte work-up	HE is a diagnosis plus trigger search; do not jump to brain MRI first without neurologic clue.
Q28	Most common portal hypertension cause	Cirrhosis	Cirrhosis is the commonest cause; remember cardiac, Budd-Chiari, schisto, PVT as alternatives.
Q29	Pulsatile liver and cardiac disease; unclear portal HTN	Transjugular liver biopsy with pressure measurements	Congestive hepatopathy: consider pressure measurements when diagnosis/staging uncertain.

DDSEP 9 - Chapter 7 | Scenario-Based Exam Table

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q30	Heart failure patient with portal HTN concern	Upper endoscopy	Portal hypertension phenotype still requires variceal assessment when indicated.
Q31	Compensated cirrhosis; no varices on EGD	Repeat EGD in 2–3 years	No varices + compensated: repeat screening in a few years; repeat sooner after decompensation.
Q32	Maximum carvedilol dose for variceal prophylaxis	12.5 mg daily	Carvedilol beyond 12.5 mg/day adds hypotension risk more than portal benefit.
Q33	GOV2 fundal varix with bleeding context	IV antibiotics and vasoactive drugs	Gastric variceal bleed: stabilise with antibiotics/vasoactive drugs; glue/EUS/TIPS expertise follows.
Q34	Cirrhosis with Na 125 and creatinine 2.2	Admit for further management	Severe hyponatraemia/AKI in cirrhosis = hold diuretics, search SBP/infection/HRS, monitor.
Q35	Coffee-ground emesis; platelets 15 × 109/L before EGD	Platelet transfusion	Severe thrombocytopenia before urgent invasive endoscopy requires correction.
Q36	Cirrhosis; ultrasound 1.6 cm liver lesion	Triphasic CT liver	New lesion in cirrhosis → dynamic CT/MRI, not AFP alone.
Q37	Small HCC within transplant criteria	Ablative therapy plus transplant evaluation	Within Milan/T2: list and bridge with locoregional therapy.
Q38	Child A, no portal HTN, edge lesion 1.9 cm	Tumour resection	Single accessible HCC + preserved function + no portal HTN → resection can be best.
Q39	New portal vein thrombus in cirrhosis	MRI	Define extent and exclude tumour thrombus before anticoagulation decisions.
Q40	Large tense ascites requiring therapy	Albumin IV	Large-volume paracentesis needs albumin to prevent circulatory dysfunction.
Q41	SBP treatment	IV antibiotics plus IV albumin	SBP = cefotaxime/ceftriaxone type therapy plus albumin when indicated to prevent AKI.
Q42	Cirrhotic liver lesion with peripheral/nodular progressive fill-in, hyperechoic	Hemangioma	Not every enhancing lesion is HCC; hemangioma fills in progressively.
Q43	New compensated cirrhosis prognosis counselling	Upper endoscopy because varices may be present	Varices are common at diagnosis; compensated cirrhosis may regress if cause removed.
Q44	NSBB physiology in varices	Beta-1 and beta-2 effects	Beta-1 lowers cardiac output; beta-2 blockade increases splanchnic vasoconstriction.
Q45	PSC-UC cirrhosis; new ascites and pain	Diuretics plus vascular imaging	New ascites needs tap; abdominal pain in cirrhosis/IBD also raises PVT/Budd-Chiari concern.
Q46	BMI 52 with metabolic syndrome and knee pain	Bariatric surgery	Severe obesity with comorbidity: bariatric surgery gives biggest metabolic/liver benefit if compensated.
Q47	Pregnancy after liver transplantation	Increased hypertensive disorder risk	Fertility returns after LT; congential anomaly/live birth rates can be comparable, but HTN risk rises.
Q48	Transplant contraindication pulmonary complication	Portopulmonary hypertension	POPH can block transplant; HPS usually supports prioritisation when severe.
Q49	Suspected hepatopulmonary syndrome	Late bubbles after fourth beat on contrast echo	Intrapulmonary shunt gives delayed left-sided bubbles, unlike intracardiac shunt.
Q50	Contraindication to liver transplantation	HCC with macrovascular hepatic vein invasion	Macrovascular HCC invasion is outside transplant criteria; controlled HIV is not AIDS.

One-line chapter memory: *Cirrhosis exam stems test prevention of decompensation, control of varices/ascites/SBP/HE, early transplant thinking, and correct HCC/transplant triage.*

DDSEP 09 – CHAPTER 8

GASTROINTESTINAL AND LIVER DISEASE IN PREGNANCY

CONCISE ESOPHAGUS-STYLE EXAM NOTES AND SCENARIO FRAMEWORK

Use: 31 DDSEP questions condensed into pregnancy-specific GI and hepatology exam patterns.

Keep DDSEP answers for recall; verify time-sensitive medication/lactation recommendations against local references.

SECTION I – DDSEP EXAM PEARL NOTES

1. IBD, celiac disease, breastfeeding and infant vaccines
2. Biliary disease and therapeutic ERCP
3. Pregnancy-specific liver disease: ICP, preeclampsia/HELLP and AFLP
4. Viral hepatitis in pregnancy
5. Chronic liver disease, transplant and Wilson disease
6. Common GI symptoms and endoscopy in pregnancy
7. Rapid DDSEP pregnancy traps

1. IBD, CELIAC DISEASE, BREASTFEEDING AND INFANT VACCINES

1. **IBD fertility is near-normal when disease is quiescent; active disease worsens pregnancy outcomes. Aim for stable remission for at least 3 months, often 3-6 months, before conception.** (Q3, Q13)

2. **Do not stop effective safe maintenance therapy simply because of pregnancy.** Continue anti-TNF when needed to maintain remission; stop methotrexate before conception. (Q5)

3. **Sulfasalazine** can cause reversible male sperm dysfunction; switch/stop if infertility concern. (Q2)

4. **Assess disease activity objectively before conception** or flare decisions: fecal calprotectin is useful during pregnancy; colonoscopy/restaging may be needed before conception. (Q4, Q14)

5. **Severe UC flare in pregnancy** requires admission and escalation with IV steroids and rescue biologic therapy; urgent delivery is not the IBD treatment. (Q8)

6. **Perianal abscess in Crohn disease** still needs drainage/seton logic. DDSEP tests metronidazole avoidance during breastfeeding; use current lactation references for real-world nuance. (Q9)

7. **Infants exposed to significant maternal immunosuppression:** avoid live rotavirus vaccine; routine non-live vaccines continue. (Q10)

8. **Celiac disease:** continue gluten-free diet and correct nutritional deficits before pregnancy; never stop the diet for "baby nutrition." (Q7)

MEMORY IBD pregnancy principle = remission is safer than flare. Continue safe maintenance; stop methotrexate.

2. BILIARY DISEASE AND THERAPEUTIC ERCP

1. **Mild biliary colic** in late pregnancy with uncomplicated stones/sludge: dietary modification and conservative management. Avoid NSAIDs in the third trimester. (Q15)

2. **MRCP** is non-contrast and can be used when ultrasound is equivocal or complication is suspected, but do not over-investigate mild biliary colic. (Q15)

3. **Choledocholithiasis/obstructive CBD** stone, cholangitis or gallstone pancreatitis: therapeutic ERCP is appropriate in pregnancy with radiation-minimising precautions. (Q28)

MEMORY — **Mild stones = conservative; obstructed duct/cholangitis/pancreatitis = therapeutic ERCP.**

3. PREGNANCY-SPECIFIC LIVER DISEASE: ICP, PREECLAMPSIA/HELLP AND AFLP

1. **ICP:** pruritus, raised serum bile acids, often modest AST/ALT rise, normal ultrasound. Treat with ursodeoxycholic acid 10–15 or 13–15 mg/kg/day in DDSEP stems. (Q16, Q27)

2. **HCV infection** increases the risk of ICP; severe pruritus in HCV-positive pregnancy -> check AST/ALT and serum bile acids. (Q18)

3. **Preeclampsia-related liver injury:** after 20 weeks, hypertension/proteinuria plus organ dysfunction. Histology may show fibrin deposition, periportal hemorrhage, necrosis/infarction and microvesicular fat. (Q21)

4. **HELLP:** hemolysis, elevated liver enzymes, low platelets; classically 28–36 weeks but may occur postpartum. (Q26)

5. **AFLP:** RUQ pain, nausea/vomiting, jaundice, hypoglycaemia/coagulopathy clues; Swansea criteria can support diagnosis. Treatment is prompt delivery; infant needs monitoring for fatty-acid oxidation defects. (Q22)

MEMORY
ICP = itch + bile acids.
HELLP = hemolysis + enzymes + low platelets.
AFLP = sick mother + metabolic/coagulation failure -> deliver.

4. VIRAL HEPATITIS IN PREGNANCY

1. **HCV:** direct-acting antiviral treatment is not recommended during pregnancy/breastfeeding in DDSEP; do not start third-trimester DAA solely to prevent vertical transmission. (Q17)

2. **HBV** vertical transmission risk rises with high HBV DNA and HBeAg positivity. At HBV DNA >200,000 IU/mL in late pregnancy, start tenofovir. (Q20, Q30)

3. **Infant of HBV-infected mother:** give HBIG + HBV vaccine and confirm response later. Breastfeeding is not contraindicated when immunoprophylaxis has been given. (Q31)

4. **HAV** is a common acute viral hepatitis cause of jaundice in pregnancy; marked transaminases with systemic symptoms can fit acute HAV. (Q24)

5. **HEV** in pregnancy, especially genotypes 1/2, can be severe. Ribavirin is contraindicated; treatment is supportive. (Q29)

MEMORY — **HBV DNA >200,000 in late pregnancy -> tenofovir. Baby gets HBIG + vaccine; breastfeeding allowed after prophylaxis.**

5. CHRONIC LIVER DISEASE, TRANSPLANT AND WILSON DISEASE

1. **After liver transplantation,** pregnancy should be planned after stable graft function and stable immunosuppression; manage with transplant hepatology and high-risk maternal-fetal medicine. Risks include preeclampsia, C-section and preterm birth. (Q19)

2. **Autoimmune hepatitis:** continue the immunosuppressive regimen that achieved remission; monitor closely during pregnancy and postpartum. (Q23)

3. **Wilson disease:** continue chelation during pregnancy; DDSEP answer reduces penicillamine by 25–50% in the third trimester for wound-healing concerns. Continue propranolol if indicated and survey varices in the second trimester. (Q25)

MEMORY — **Pregnancy with chronic liver disease is safer with controlled disease than with treatment withdrawal.**

6. COMMON GI SYMPTOMS AND ENDOSCOPY IN PREGNANCY

1. **Nausea and vomiting of pregnancy:** early first trimester, peaks at 9–16 weeks. If conservative measures fail, use doxylamine + pyridoxine.
Avoid gastric-emptying nuclear tests in pregnancy. (Q1)

2. **Pregnancy GERD** without alarms: start with lifestyle/antacid; **calcium carbonate** is the classic safe answer. Endoscopy is reserved for bleeding or alarm symptoms. (Q11)

3. **Constipation:** progesterone, iron, and gravid uterus worsen transit. **Polyethylene glycol** is the preferred osmotic laxative answer. (Q12)

4. **Acute hematemesis** with Hb drop: proceed to **sedated EGD,** left lateral position, lowest effective sedation dose. **Pregnancy does not block** urgent endoscopy. (Q6)

MEMORY

Pregnancy does not cancel urgent endoscopy: bleeding or alarms justify EGD; routine/non-urgent radiation-based tests are avoided.

7. RAPID DDSEP PREGNANCY TRAPS

1. **Do not** choose delivery as the first treatment for severe UC flare; treat the colitis. (Q8)

2. **Do not** choose DAA therapy for HCV during pregnancy just because the viral load is high. (Q17)

3. **Do not** stop Wilson disease treatment during pregnancy. (Q25)

4. **Do not** stop the gluten-free diet in celiac pregnancy. (Q7)

5. **Do not** give routine live rotavirus vaccine after significant in utero biologic/immunosuppressive exposure. (Q10)

MEMORY

Avoid the traps.
Choose the evidence-based, pregnancy-safe answer.

SECTION II – DDSEP SCENARIO-BASED EXAM TABLE

One compact row per DDSEP question: scenario clue, retained best answer, and exam pearl.

DDSEP Q#	Scenario / Key clue	Best answer / mnenonic	Exam pearl / mnemonic
Q1	7 weeks pregnant; nausea/vomiting after conservative measures	Doxylamine + vitamin B6 (pyridoxine)	NVP peaks around 9–16 weeks; avoid gastric emptying studies/radiation logic in pregnancy.
Q2	Male UC patient planning conception on sulfasalazine	Stop/switch sulfasalazine	Sulfasalazine can cause reversible sperm dysfunction; do not blame quiescent UC itself.
Q3	Female UC patient wants pregnancy; disease just active	Fertility normal in quiescent disease	Aim for 3–6 months stable remission before conception; continue safe maintenance therapy.
Q4	Pregnant UC patient with mild symptom increase at 29 weeks	Fecal calprotectin	Non-invasive inflammation marker in pregnancy; CT is avoided and endoscopy is reserved when needed.
Q5	Crohn remission on adalimumab + methotrexate, preconception	Stop methotrexate; continue adalimumab	Do not stop all IBD drugs; methotrexate is teratogenic exception.
Q6	22 weeks pregnant with recurrent hematemesis and Hb drop	Sedated upper endoscopy, left lateral position	Urgent GI bleeding is a valid indication for EGD in pregnancy; use minimal sedation.
Q7	Celiac disease, planning pregnancy	Do NOT stop gluten-free diet	Pregnancy requires controlled celiac diseane; nutritional assessment, and continued gluten avoidance.
Q8	28 weeks pregnant, severe pancolitis flare	Hospital admission, IV steroids + infliximab	Severe UC in pregnancy is treated medically; urgent delivery is not the IBD treatment.
Q9	Breastfeeding patient with perianal Crohn abscess, antibotic choice question	Metronidazole not recommended in DDSEP stem	Pregnancy/lactation drug answers can be nuanced; DDSEP tests avoidance of metronidazole while breastfeeding.
Q10	Infant vaccine after maternal infliximab + azathioprine through pregnancy	Avoid rotavirus vaccine	Rotavirus is a live vaccine; in the first year routine series; non-live vaccines proceed.
Q11	14 weeks pregnant with uncomplicated heartburn, no alarm features	Calcium carbonate	Pregnancy GERD ladder: lifestyle → antacid/alginate → acid suppression if needed; EGD only for alarm/bleeding.
Q12	8 weeks pregnant; worsening constipation despite fiber/stool softeners	Polyethylene glycol	Osmotic laxative such as PEG are preferred; constipation worsens from progesterone/iron/gravid uterus.
Q13	UC in remission on mesalamine formulation with dibutyl phthalate; family planning	Switch to another agent before conception	Phthalate exposure/minimization suggested; switch to safer formulation/agent.
Q14	Crohn ileocolonic/perianal disease; feels well but planning pregnancy	Restage with colonoscopy	Objective remission matters before conception; symptoms alone underestimate disease.
Q15	33 weeks pregnant; mild biliary colic with stones/sludge, no complications	Dietary modification	Mild biliary colic in late pregnancy is conservative; MRCP/ERCP/ surgery only for complications.
Q16	34 weeks, pruritus, raised bile acids, normal ultrasound	UDCA 13–15 mg/kg/day	ICP diagnostic/Tx first line drugs; risk relates to fetal complications.
Q17	HCV-positive pregnant patient; routine antenatal care question	Do not start DAA in pregnancy for vertical transmission	HCV treatment is deferred during pregnancy/breastfeeding in DDSEP framework.
Q18	HCV pregnancy with severe pruritus/ pigmentary symptoms	Check AST/ALT and serum bile acids	HCV increases ICP risk; pruritus in pregnancy demands bile acids.
Q19	Liver transplant recipient counseling about future pregnancy	Multidisciplinary high-risk care	After LT: wait for stable graft + immunosuppression; higher preeclampsia, C-section, and preterm birth risks.
Q20	HBV pregnancy; risk factor for vertical transmission	HBeAg positivity	Transmission risk relates to high HBV DNA and HBeAg positivity.
Q21	32 weeks; hypertension/proteinuria+ phenotype with liver injury	Preeclampsia/HELLP-type liver pathology	Pregnancy hypertensive liver injury shows periportal hemorrhage/fibrin/necrosis and may overlap with HELLP.
Q22	AFLP by Swansea criteria; infant risk question	Monitor infant for LCHAD-type complications	AFLP: mother mitochondrial fatty acid oxidation defects; delivery is maternal treatment and baby needs monitoring.
Q23	Autoimmune hepatitis cirrhosis; 8 weeks pregnant	Continue current immunosuppression	AIH: keep stable meds; monitor flares; maintain remission and monitor especially postpartum.
Q24	21 weeks jaundice, RUQ pain, marked AST/ALT, positive acute hepatitis serologies	Acute hepatitis A	Acute viral hepatitis is a common jaundice cause in pregnancy; ICP usually begins with pruritus.
Q25	Wilson disease cirrhosis/varices, early pregnancy	Continue penicillamine with 25–50% 3rd trimester reduction; propranolol; 2nd trimester variceal survey	Do not stop Wilson therapy; reduce chelator late to help wound healing of delivery surgery.
Q25	37 weeks; RUQ pain, hypertension, microangiopathic hemolysis	HELLP syndrome	HELLP: hemolysis + elevated liver enzymes + low platelets; can occur late pregnancy or postpartum.
Q27	33 weeks; pruritus, high bile acids, cholestatic pregnancy picture	UDCA 10–15 mg/kg/day	ICP: UDCA is the classic treatment answer; bile acids are the key diagnostic lab.
Q28	33 weeks; gallstones plus dilated CBD/ obstruction concern	ERCP	Therapeutic ERCP is appropriate for choledocholithiasis/ cholangitis/pancreatitis in pregnancy; use radiation-minimizing techniques.
Q29	HEV in pregnancy; treatment statement	Ribavirin contraindicated	HEV (genotypes 1/2) can be severe in pregnancy; treatment is supportive and ribavirin is contraindicated.
Q30	32 weeks pregnant; chronic HBV with high viral load	Start tenofovir	HBV DNA >200,000 IU/mL in late pregnancy → antiviral prophylaxis; tenofovir preferred.
Q31	Postnatal HBV management exception	Breastfeeding is NOT contraindicated after immunoprophylaxis	Infant needs HBIG + vaccine and later serology; breastfeeding allowed when prophylaxis is given.

DDSEP 9 – CHAPTER 9

DIARRHEA AND CONSTIPATION

CONCISE ESOPHAGUS-STYLE EXAM PEARL NOTES AND SCENARIO FRAMEWORK

Chapter 9 | 51 DDSEP questions

Memory line: Read Section I for high-yield pattern recognition, then use Section II as a rapid DDSEP scenario table.
DDSEP answers are retained; keep updates concise and exam-focused.

SECTION I – DDSEP EXAM PEARL NOTES

1. IBS: Diagnose by Pattern, Then Screen Only Where Indicated
2. Bile Acid Diarrhea and SIBO
3. Celiac Disease and Celiac Mimics
4. Osmotic, Secretory, and Factitious Diarrhea
5. Microscopic Colitis: Normal-Looking Colon, Abnormal Biopsies
6. *C. difficile* Infection: Recurrence and Fulminant Disease
7. Constipation and Defecatory Disorders

1. IBS: DIAGNOSE BY PATTERN, THEN SCREEN ONLY WHERE INDICATED

IBS is abdominal pain related to defecation and associated with change in stool frequency or form; typical young patients without alarm features do not need routine colonoscopy.

EVALUATION

- IBS-D or IBS-M: check CBC/ inflammatory markers and celiac serology, especially in type 1 diabetes or other high-risk groups. (Q2, Q26)

- No alarm features in typical young patients → no routine colonoscopy.

IBS-C MANAGEMENT PATHWAY

1 LOW-RISK INITIAL OPTION
Soluble fiber/psyllium
Avoid insoluble bran if bloating/pain worsens.
(Q20, Q50)

↓

2 IF INSUFFICIENT RESPONSE
Polyethylene glycol (PEG)
Improves stool frequency but may not treat pain.

↓

3 PEG-UNRESPONSIVE OR MODERATE-TO-SEVERE IBS-C
Linaclotide is the DDSEP drug pattern.
(Q1, Q20)

IBS-D MANAGEMENT

FIRST STEP
Loperamide
Controls stool frequency.

↓

PERSISTENT PAIN DESPITE LOPERAMIDE
Use a neuromodulator; TCA fits because it reduces pain and slows transit. (Q3)

ELUXADOLINE CONTRAINDICATED
- After cholecystectomy
- History of pancreatitis
- Sphincter of Oddi disease
- Significant alcohol use
- Hepatic impairment (Q22)

MEMORY LINE

IBS-C: SOLUBLE FIBER → PEG → LINACLOTIDE. | IBS-D PAIN: LOPERAMIDE CONTROLS STOOL, TCA CONTROLS PAIN. | CHOLECYSTECTOMY + ELUXADOLINE = STOP.

2. BILE ACID DIARRHEA AND SIBO

BILE ACID DIARRHEA

Ileal disease or injury – Crohn disease, radiation ileitis, ileal resection – causes bile acid malabsorption and watery diarrhea.

↓

Bile acid binders such as colestipol/colestyramine fit the exam. (Q14)

SIBO RISK FACTORS

- Impaired motility or anatomy
- Systemic sclerosis
- Achlorhydria/ atrophic gastritis
- Blind loops

(Q15, Q27, Q30, Q33)

SIBO COMPLICATIONS

B12

Vitamin B12 deficiency because bacteria compete for B12.

Fat-soluble vitamin deficiency and iron deficiency may occur.
(Q15, Q27)

DIAGNOSIS & TREATMENT

DIAGNOSIS
Glucose hydrogen breath testing is a DDSEP diagnostic answer.
(Q30, Q33)

TREATMENT
Rifaximin is a treatment answer in systemic-sclerosis/ SIBO stems.
(Q30, Q33)

MEMORY LINE


Diseased ileum (ileal disease, resection, radiation)

→


Bile acids malabsorption – reach colon

→


Watery diarrhea

→


Bile acid binder (colestipol/ colestyramine)


SIBO steals B12.

3. CELIAC DISEASE AND CELIAC MIMICS

A. ON A GLUTEN-CONTAINING DIET

First-line test:
tTG-IgA + total IgA

If IgA deficient:
Use IgG-based testing such as DGP-IgG or tTG-IgG.

(Q6, Q8, Q26)

B. ALREADY GLUTEN-FREE

- Serology may become negative.

Options tested in DDSEP:

1. tTG-IgA + total IgA first
2. HLA-DQ2/DQ8 to exclude disease
3. Gluten challenge followed by duodenal biopsy when confirmation is required.

(Q6, Q25, Q36, Q39)

C. NONRESPONSIVE CELIAC DISEASE

The commonest cause is inadvertent gluten exposure.

First action:
Dietitian/Nutritionist reassessment,
not immunosuppression.

(Q43)

D. OLMESARTAN ENTEROPATHY

Mimics celiac histology with villous blunting and intraepithelial lymphocytes.

- Celiac serology: negative
- No response to gluten-free diet

(Q7)

E. TROPICAL SPRUE

Endemic travel + chronic diarrhea + macrocytosis/folate deficiency + villous blunting.

Treatment:
Tetracycline + folate.

(Q9)

F. WHIPPLE DISEASE

Chronic diarrhea, weight loss and arthralgia/arthritis.

First diagnostic step:
EGD with duodenal/small-bowel biopsies.

(Q10)

G. OTHER CONSIDERATIONS

- Consider other causes of villous atrophy if serology negative and no response to GFD.
- Always review medications, infections, and immune disorders.

MEMORY LINE

CELIAC TESTING DEPENDS ON GLUTEN EXPOSURE:
ON GLUTEN = tTG-IgA + TOTAL IgA; IgA DEFICIENT = IgG TESTS (DGP-IgG or tTG-IgG); OFF GLUTEN = HLA/GLUTEN CHALLENGE LOGIC.

4. OSMOTIC, SECRETORY, AND FACTITIOUS DIARRHEA

A. OSMOTIC DIARRHEA

Stool osmotic gap:
290 − 2 (Na + K)
(stool Na and stool K)

- Usually GAP > 100
- Improves with fasting
- Example: carbohydrate malabsorption

(Q11, Q19, Q21, Q34, Q40)

B. CARBOHYDRATE MALABSORPTION CLUES

- Lactose / fructose / sorbitol exposure
- Low stool pH (< 5.5)
- High osmotic gap (> 100)
- Symptoms after meals or sweetened products

(Q11, Q19, Q21, Q40)

C. SECRETORY DIARRHEA

- Persists despite fasting and occurs nocturnally
- Low osmotic gap (< 50)
- Causes include:
  - Carcinoid syndrome
  - VIPoma
  - Bile acid diarrhea
  - Infections

(Q34)

D. FACTITIOUS / LAXATIVE DIARRHEA

- Extensive negative evaluation
- Healthcare access or personality clues
- Abnormal stool studies:
  - High stool magnesium
  - High stool phosphate
  - Abnormal electrolytes

(Q37)

MEMORY LINE

GAP > 100 = OSMOTIC | GAP < 50 = SECRETORY | pH↓ LOW pH = CARBOHYDRATE | Mg HIGH STOOL Mg = LAXATIVE ABUSE

5. MICROSCOPIC COLITIS: NORMAL-LOOKING COLON, ABNORMAL BIOPSIES

A. CLINICAL PATTERN

- Older patient
- Chronic watery non-bloody diarrhea
- Often nocturnal
- Normal or near-normal colonoscopy

Diagnosis requires random biopsies.

(Q12, Q29, Q32, Q35, Q41, Q51)

B. BIOPSY APPROACH

Biopsy both **RIGHT AND LEFT COLON** when suspected.

(Q35)

C. ASSOCIATIONS / TRIGGERS

- Celiac disease
- Autoimmune disease
- Smoking
- SSRIs (e.g., sertraline)
- PPIs
- NSAIDs
- Statins

(Q24, Q32, Q35, Q51)

D. TREATMENT

First-line therapy in active microscopic colitis is **BUDESONIDE.**

Also stop possible offending drugs when feasible.

(Q12, Q29, Q41)

E. PROGNOSIS

Microscopic colitis **does not clearly increase colon cancer risk.**

(Q32)

MEMORY LINE

Watery diarrhea + Older woman + Normal colonoscopy = Biopsy for microscopic colitis + Treat with budesonide + Remove triggers

6. C. DIFFICILE INFECTION: RECURRENCE AND FULMINANT DISEASE

A. RECURRENT CDI AFTER VANCOMYCIN

Recurrent symptomatic CDI with positive toxin testing after prior vancomycin:

DDSEP answer is VANCOMYCIN, often as a taper/pulse depending on setting.

(Q28)

B. FULMINANT CDI

CLINICAL CLUES

- Fever
- Ileus / decreased stool output
- Abdominal distension
- Toxic features

TREATMENT

Oral vancomycin + Rectal vancomycin + IV metronidazole

(Q31)

C. PREVENTION / KEY POINTS

Avoid unnecessary antibiotics.

Routine dental antibiotic prophylaxis is not indicated without high-risk cardiac indications.

(Q42)

MEMORY LINE

CDI with ileus (poor drug delivery to colon) = Oral vancomycin + Rectal vancomycin + IV metronidazole = Best chance to control fulminant CDI

7. CONSTIPATION AND DEFECATORY DISORDERS

1. FIRST RULE: REVIEW MEDICATIONS

Review and **STOP** constipating medications when possible.

- Verapamil
- Tricyclic antidepressants
- Opioids
- Anticholinergic drugs

(Q16, Q23)

2. INITIAL EVALUATION

Includes careful **HISTORY** and **DIGITAL RECTAL EXAMINATION (DRE).**

A NORMAL DRE DOES NOT EXCLUDE DYSSYNERGIC DEFECATION.

(Q17)

3. WHEN TO SUSPECT DEFECATORY DISORDER

- Straining
- Incomplete evacuation
- Digital or vaginal pressure to evacuate
- High anal tone
- Paradoxical contraction
- Laxative failure

(Q17, Q47, Q48)

4. TESTING SEQUENCE & FIRST-LINE TREATMENT

1. **ANORECTAL MANOMETRY (ARM)**
2. **BALLOON EXPULSION TEST (BET)**

If dyssynergia confirmed: **BIOFEEDBACK** is **FIRST-LINE** treatment.

(Q18, Q44, Q47, Q48)

5. NEXT STEPS BASED ON RESULTS

ARM AND/OR BET DISCORDANT OR INCONCLUSIVE

→ **PROCEED TO DEFECOGRAPHY** (Q46)

ANORECTAL TESTING NORMAL BUT CONSTIPATION PERSISTS

→ **PERFORM COLON TRANSIT TESTING** (Q49)

6. OPIOID-INDUCED CONSTIPATION

Traditional laxatives and/or osmotic laxatives are **FIRST-LINE** before newer agents.

(Q45)

CONSTIPATION ALGORITHM (MEMORY LINE)

1. STOP CONSTIPATING DRUGS →
2. HISTORY + DRE →
3. ARM + BET →
4. BIOFEEDBACK IF DYSSYNERGIA CONFIRMED →
5. DEFECOGRAPHY IF DISCORDANT OR INCONCLUSIVE →
6. COLON TRANSIT TEST IF ANORECTAL TESTING NORMAL & SYMPTOMS PERSIST

KEY CLINICAL CLUES	Think defecatory disorder when there is: straining, incomplete evacuation, digital/vaginal pressure, high anal tone, paradoxical contraction, or laxative failure.	(Q17, Q47, Q48)
IMPORTANT POINT	A normal DRE does **NOT** exclude dyssynergic defecation.	(Q17)
TESTING SEQUENCE MATTERS	ARM + BET first → biofeedback if dyssynergia → defecography if discordant/ inconclusive → colon transit test if anorectal testing normal but symptoms persist.	(Q18, Q44, Q46, Q47, Q48, Q49)
TREATMENT PEARL	Biofeedback is first-line for dyssynergic defecation.	(Q44, Q47, Q48)
OPIOID CONSTIPATION	Use traditional laxatives/osmotic laxatives first before considering newer agents.	(Q45)

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

One compact row per DDSEP scenario: clue → best answer → exam pearl.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	IBS-C; PEG twice daily but persistent symptoms.	Stop PEG and start linaclotide	PEG improves stool frequency; linaclotide fits PEG-unresponsive/moderate IBS-C.
Q2	Type 1 diabetes + IBS-D/M-type symptoms.	tTG-IgA and total IgA	IBS-D evaluation includes celiac screen, especially high-risk autoimmune groups.
Q3	IBS-D: loperamide helps diarrhea but pain pesrists.	TCA	TCA treats visceral pain and slows transit; useful in IBS-D.
Q4	Diarrhea evaluation with alarm/abnormal features.	Colonoscopy	Do not label IBS if alarm clues or inflammatory concern exist.
Q5	New diarrhea after citalopram dose increase.	Stop citalopram	Temporal drug effect first; SSRIs also linked to microscopic colitis.
Q6	Possible celiac but already gluten-free.	tTG-IgA and total IgA	Start with serology(IgA); if negative off gluten, HLA or challenge may follow.
Q7	Villous blunting, negative celiac tests, olmesartan exposure.	Review medications	Olmesartan enteropathy mimics celiac but serology/GFD response are absent.
Q8	Celiac suspicion + IgA deficiency.	DGP-IgG	IgA deficient patients need IgG-based celiac serology.
Q9	Endemic travel + macrocytosis + villous blunting.	Tetracycline and folic acid	Tropical sprue: diagnosis supported by response to therapy.
Q10	Chronic diarrhea + weight loss + arthritis.	EGD with small-bowel biopsies	Whipple disease: diagnose from duodenal biopsies.
Q11	Lactose intolerance stool profile.	Stool osmotic gap 120	Osmotic diarrhea: gap usually >100.
Q12	Older patient; chronic watery nocturnal diarrhea.	Review medication list	Microscopic colitis: remove triggers; budesonide if active.
Q13	Microscopic colitis with persistent symptoms.	tTG-IgA and total IgA	Celiac disease is associated with microscopic colitis.
Q14	Prior pelvic radiation; chronic watery diarrhea.	Start colestipol	Radiation ileitis -> bile acid diarrhea -> bile acid binder.
Q15	Known SIBO counseling.	Vitamin B12 deficiency	Bacteria compete for B12; fat-soluble vitamin/iron deficiency may occur.
Q16	Elderly constipation on amitriptyline.	Change amitriptyline to sertraline	Stop constipating drugs before adding more therapy.
Q17	Chronic constipation; no alarm symptoms.	Digital rectal examination	DRE screens for defecatory disorder; normal DRE does not exclude it.
Q18	Dyssynergia treated with biofeedback, no response.	Repeat balloon expulsion	If still abnormal after biofeedback, assess structural disease with defecography.
Q19	Postprandial urgency; low stool pH; osmotic gap high.	Eliminate lactose	Carbohydrate malabsorption: acidic stool + osmotic diarrhea.
Q20	IBS-C; PEG works but patient wants alternative.	Linaclotide	Linaclotide is DDSEP alternative/step-up agent for IBS-C.
Q21	High soda/fructose intake diarrhea.	Stool osmotic gap >100	Fructose malabsorption gives osmotic diarrhea.
Q22	IBS-D patient post-cholecystectomy on eluxadoline.	Stop eluxadoline	No gallbladder = increased pancreatitis/ sphincter risk with eluxadoline.
Q23	New constipation after verapamil.	Stop verapamil	Medication review is first-line constipation management.
Q24	Medication predisposing to microscopic colitis.	Sertraline	SSRI association; also PPIs/NSAIDs/statins.
Q25	Suspected celiac but gluten-free; confirmation needed.	Gluten challenge + upper endoscopy	Biopsy confirmation requires gluten exposure.
Q26	Type 1 diabetes + loose stools/bloating.	tTG-IgA with total IgA	T1DM is a high-risk celiac group.
Q27	SIBO vitamin statement.	Bacteria compete for B12	SIBO steals B12; thiamine not typical.
Q28	First recurrence CDI after vancomycin; toxin positive.	Vancomycin	DDSEP: vancomycin/taper-pulse pattern for recurrence.
Q29	Biopsy-proven lymphocytic colitis.	Start oral budesonide	Budesonide is first-line active microscopic colitis therapy.
Q30	Atrophic gastritis + intermittent diarrhea.	Glucose hydrogen breath test	Achlorhydria predisposes to SIBO; breath test is exam answer.
Q31	CDI worsening with fever, ileus/distension.	Add rectal vancomycin enemas and IV metronidazole	Fulminant CDI: oral vanco plus rectal vanco and IV metronidazole.

DDSEP 9 | CHAPTER 8 | SCENARIO TABLE (CONTINUED)

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q32	Collagenous colitis statement.	Celiac disease is associated	Microscopic colitis: celiac association; no proven CRC excess.
Q33	Systemic sclerosis + bloating/loose stools.	Start rifaximin	Scleroderma dysmotility -> SIBO; rifaximin fits treatment stem.
Q34	Na 88, K 44; gap = 26.	Low osmotic gap = secretory diarrhea	Low osmotic gap = secretory diarrhea.
Q35	Diarrhea on PPI/statin, prior colonoscopy without biopsies.	Repeat colonoscopy with right/left biopsies	Microscopic colitis needs biopsies even if mucosa is normal.
Q36	Positive serology but normal/nondiagnostic biopsy on GFD.	Gluten challenge + repeat biopsy	Need gluten exposure to confirm/ exclude celiac disease.
Q37	Healthcare worker, extensive negative diarrhea workup.	Stool magnesium 107	High stool Mg suggests laxative/ factitious diarrhea.
Q38	Young IBS-like symptoms plus alarm clues/elevated CRP/family history.	Colonoscopy	Alarm features override routine IBS approach.
Q39	Already gluten-free; celiac evaluation.	Check HLA-DQ2/DQ8	Negative HLA-DQ2/DQ8 essentially excludes celiac disease.
Q40	High osmotic gap with normal basic workup.	Sorbitol ingestion	Sugar alcohols cause osmotic diarrhea.
Q41	Lymphocytic colitis on biopsy.	Start budesonide	Treat active microscopic colitis with budesonide.
Q42	Routine dental antibiotics after recent CDI, no cardiac indication.	Discontinue prophylactic antibiotics	Avoid unnecessary antibiotics; no role for routine prophylaxis here.
Q43	Persistent symptoms after celiac diagnosis on GFD.	Re-refer to nutritionist	Most nonresponsive celiac = hidden gluten ingestion.
Q44	Suspected dyssynergic defecation test result.	Expulsion of water-filled balloon in 3 minutes	Failed/ delayed in balloon expulsion supports dyssynergia.
Q45	Methadone-associated constipation.	Osmotic laxatives	Opioid-induced constipation: traditional laxatives first.
Q46	Discordant/inconclusive ARM and BET.	Defecography	Defecography clarifies structural/ evacuation disorders.
Q47	IBS-C symptoms but PEG failure and incomplete evacuation.	ARM + balloon expulsion	IBS-C can coexist with defecatory disorder.
Q48	DRE: high tone + paradoxical contraction.	ARM with balloon expulsion	Confirm dyssynergia before biofeedback; slow transit may coexist.
Q49	Normal anorectal manometry/BET; refractory constipation.	Colon transit testing	Normal outlet testing -> assess slow transit.
Q50	Young IBS-C without alarm features.	Start psyllium	Soluble fiber is first-line; avoid insoluble bran in IBS.
Q51	Older woman with watery diarrhea; collagenous colitis risk drug.	Sertraline	SSRI exposure is a microscopic colitis clue.

DDSEP 9 – CHAPTER 10

GASTROINTESTINAL MOTILITY

CONCISE EXAM-ORIENTED NOTES – ESOPHAGUS CHAPTER STYLE

Chapter 10 | 56 DDSEP questions

Chapter 10 | 56 DDSEP questions.
Use Section I for core exam patterns;
use Section II for rapid scenario recall.
DDSEP answers retained; wording is
condensed and clustered.

SECTION I – DDSEP EXAM-ORIENTED NOTES

1. RUMINATION, CYCLIC VOMITING AND FUNCTIONAL DYSPEPSIA

RUMINATION SYNDROME	Effortless post-prandial regurgitation within minutes, without nausea or retching; endoscopy is often normal or shows mild esophagitis. **Treat with diaphragmatic breathing.**
CYCLIC VOMITING	Discrete stereotyped vomiting attacks with normal periods between attacks. **Marijuana use should trigger cannabis hyperemesis counseling and cessation.**
FUNCTIONAL DYSPEPSIA	Upper abdominal symptoms without structural disease: • **Postprandial Distress Syndrome (PDS):** fullness/ early satiety. • **Epigastric Pain Syndrome (EPS):** bothersome epigastric pain/burning.
TREATMENT PEARLS	**Buspirone** before meals is useful for impaired accommodation/postprandial distress phenotype. Low-dose **TCA** is used for functional heartburn/visceral pain phenotypes.
MEMORY	**DDSEP Q1, Q21, Q28, Q51:** Effortless regurgitation -> breathing; episodic vomiting + cannabis -> stop cannabis; fullness -> PDS; epigastric pain -> EPS.

2. ESOPHAGEAL MOTILITY: MANOMETRY PATTERNS AND STEM CLUES

ACHALASIA	Impaired LES relaxation with aperistalsis; pathophysiology is loss of inhibitory myenteric neurons and reduced nitric oxide. **New achalasia in an elderly smoker with weight loss = pseudoachalasia until malignancy is excluded.**
TYPE III ACHALASIA / SPASTIC ACHALASIA	Responds best to POEM because the myotomy can be extended proximally.
BOTULINUM TOXIN IN ACHALASIA	Frail poor surgical candidates may receive LES botulinum toxin, but response wanes with repeat injections.
DIFFUSE ESOPHAGEAL SPASM (DES)	Corkscrew esophagus and premature contractions with short distal latency.
IEM ASSOCIATIONS	IEM is linked to GERD and scleroderma; EGJ outflow obstruction and hypercontractility may be opioid-associated.
MODERN EXAM VOCABULARY	DES requires >=20% premature swallows; EGJOO should be clinically relevant and supported by obstruction evidence, not manometry alone.
MEMORY	**DDSEP Q2, Q4, Q8-Q9, Q12, Q15, Q19-Q20, Q39, Q48-Q49:** Dysphagia for solids/liquids + abnormal IRP = achalasia/ EGJOO; elderly rapid onset = CT for cancer.

3. GERD, FUNCTIONAL HEARTBURN, REFLUX HYPERSENSITIVITY AND NCCP

BEFORE ANTIRELFUX SURGERY	• Prove GERD, perform EGD and high-resolution manometry. • If endoscopy is negative, pH testing off PPI is needed. • A **full PPI response** predicts better fundoplication outcome.
FUNCTIONAL HEARTBURN	• Normal endoscopy, normal/relevant motility excluded, and no reflux-symptom correlation. • Treat as **visceral hypersensitivity** with neuromodulators such as low-dose **nortriptyline**.
REFLUX HYPERSENSITIVITY (OVERLAPPING WITH GERD)	• Prior objective GERD plus persistent symptoms on PPI and **positive symptom association** on impedance-pH, despite healed mucosa.
NONCARDIAC CHEST PAIN (NCCP)	• Commonly reflects GERD or functional chest pain. • HRM is often normal after GERD and major motor disorders are excluded. • Night reflux events are fewer, but **more injurious** because clearance is reduced.
MEMORY	**DDSEP Q10, Q22, Q29–Q30, Q33–Q35, Q50:** Surgery needs proven GERD + manometry; positive PPI response predicts success; normal tests -> functional pain.

4. GASTROPARESIS, DUMPING SYNDROME AND GASTRIC MOTILITY

GASTROPARESIS	• Early satiety, postprandial fullness, nausea/vomiting and retained food/delayed scintigraphy after obstruction is excluded. • Early satiety/fullness **correlate better with emptying** than pain or bloating.
DIABETIC GASTROPARESIS	• Improve **glycemic control** first. • **Do not** perform scintigraphic solid-emptying testing when glucose is markedly high. • Chronic metoclopramide risks **tardive dyskinesia**, especially with long duration/cumulative dose.
SPECIAL SITUATIONS	• Parkinson disease or recurrent *C. difficile* makes chronic prokinetic/antibiotic choices unattractive. • Start with **dietary treatment**: small frequent meals, low fat, low fiber/low residue, liquid nutrition if needed.
DUMPING SYNDROME	• After gastric surgery: early post-meal cramps, diarrhea, palpitations, flushing and sweating after **carbohydrate-rich meals**. • Dietary measures first; **octreotide** for refractory/severe cases.
MEMORY	**DDSEP Q14, Q17, Q26–Q27, Q42–Q43, Q46:** Diabetic gastroparesis -> glucose control; metoclopramide -> dyskinesia; post-gastric carb symptoms -> dumping.

5. SMALL-BOWEL AND COLONIC MOTILITY PHYSIOLOGY

MIGRATING MOTOR COMPLEX (MMC)	• Migrating motor complex is the fasting/interdigestive cleansing pattern. • It cycles roughly every **90–120 minutes**. • It is partly **motilin**-regulated.
FED STATE: MIXING AND PROPULSION	• Fed state uses **segmentation** for mixing and **peristalsis** for propulsion. • Slow waves/basic electrical rhythm are generated by **interstitial cells of Cajal**.
CHRONIC INTESTINAL PSEUDO-OBSTRUCTION (CIPO)	• Recurrent bowel dilation without mechanical transition. • **Systemic sclerosis** suggests a myopathic process.
ACUTE COLONIC PSEUDO-OBSTRUCTION (OGILVIE SYNDROME)	• Hospitalized/postoperative patient with colonic dilation. • Stable patient and cecum not severely threatened → **conservative decompression**. • Bowel rest, stop triggers, NG/rectal decompression.
MEMORY	**DDSEP Q7, Q24, Q40, Q45, Q47:** Fasting gut sweeps by MMC; ICC makes slow waves; systemic sclerosis causes pseudo-obstruction.

6. CONSTIPATION: IBS-C, FUNCTIONAL CONSTIPATION, OIC AND SLOW TRANSIT

IBS-C vs FUNCTIONAL CONSTIPATION	• **IBS-C** requires abdominal pain related to defecation or stool change. • **Functional constipation** has straining, hard stools, incomplete evacuation, blockage sensation or manual maneuvers, with pain not predominant.
IBS-C THERAPIES	• Therapies in these stems include **linaclotide** or **lubiprostone** after simple measures. • Avoid **anticholinergic agents** that worsen constipation.
OPIOID-INDUCED CONSTIPATION (OIC)	• Traditional **laxatives** first. • If refractory, consider peripheral mu-opioid receptor antagonists such as **naloxegol**, **naldemedine** or **methylnaltrexone**.
SLOW-TRANSIT CONSTIPATION	• Severe slow-transit constipation may be treated surgically only after defecatory disorder and generalized dysmotility are excluded. • Delayed small-bowel transit predicts **poor colectomy outcome**.
MEMORY	**DDSEP Q6, Q13, Q18, Q25, Q52:** Pain-predominant constipation -> IBS-C; no pain predominant -> functional constipation; opioid constipation starts with laxatives.

7. DEFECATORY DISORDERS: DRE, BALLOON, ARM, DEFECOGRAPHY AND BIOFEEDBACK

DYSSYNERGIC DEFECATION	• Inadequate relaxation or paradoxical contraction during simulated defecation, with straining/incomplete evacuation and laxative failure. • First-line treatment is **pelvic-floor biofeedback**, **not** more laxatives.
BALLOON EXPULSION FAILURE	• Supports evacuation disorder and predicts biofeedback response. • If biofeedback fails, repeat balloon expulsion. • If still abnormal or ARM/balloon are discordant, proceed to **defecography**.
RAIR	• RAIR = transient internal anal sphincter relaxation after rectal distension. • **Absent RAIR** suggests Hirschsprung disease and needs rectal suction/deep biopsy. • **Normal RAIR** essentially excludes Hirschsprung in most stems.
RADIOPAQUE MARKERS	• Radiopaque markers clustered in rectosigmoid suggest outlet obstruction, • so **anorectal manometry** is the next physiologic test.
MEMORY	**DDSEP Q3, Q5, Q16, Q36–Q38, Q41, Q44, Q54–Q55:** Straining + incomplete evacuation -> DRE/ARM/balloon; dyssynergia -> biofeedback; absent RAIR -> biopsy.

8. ANAL SPHINCTER PHYSIOLOGY, INCONTINENCE AND ANAL FISSURE

ANAL SPHINCTER PHYSIOLOGY	• Internal anal sphincter is **smooth muscle** and provides most **resting tone**. • External anal sphincter is **striated** and provides **voluntary squeeze/urge control**.
INCONTINENCE PATTERNS	• Urge incontinence is linked to **reduced squeeze pressure/external sphincter dysfunction**. • Passive/nocturnal leakage, diabetic neuropathy and scleroderma clues point toward **internal sphincter weakness/resting pressure abnormality**.
ANAL FISSURE	• Classically has **posterior midline** tear, pain and hematochezia with **high resting sphincter tone**. • Therapy reduces sphincter tone with topical **smooth-muscle relaxants** or **sphincterotomy**.
MEMORY	**DDSEP Q11, Q23, Q37, Q44, Q53:** Resting pressure = internal sphincter; squeeze pressure = external sphincter; fissure = high resting tone.

9. DYSPHAGIA OUTSIDE CLASSIC ESOPHAGEAL PERISTALSIS

OROPHARYNGEAL / STRIATED-MUSCLE DYSPHAGIA	• Can occur with **polymyositis**. • Dysphagia **immediately after swallowing** while esophageal body manometry may be normal.
DYSPHAGIA LUSORIA	• Extrinsic vascular compression, classically from an **aberrant right subclavian artery**. • Symptoms may be **positional** and **solids-predominant**.
MEMORY	**DDSEP Q31–Q32:** Immediate swallow difficulty -> pharyngeal/striated muscle; dysphagia lusoria -> aberrant right subclavian artery.

DDSEP 9 - Chapter 10

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	Effortless post-prandial regurgitation without nausea/retching; EGD mild only	Diaphragmatic breathing	Rumination = behavioral regurgitation; breathing retrains abdominal wall/diaphragm.
Q2	Elderly smoker with new dysphagia, weight loss, achalasia-like manometry	Schedule chest CT	Pseudoachalasia: rapid onset/old age/ weight loss -> search malignancy.
Q3	Constipation with incomplete evacuation + ARM; supportive test for evacuation disorder	Failed expulsion of 50 mL balloon in two minutes	Failed balloon expulsion supports outlet dysfunction and biofeedback response.
Q4	Pre-fundoplication manometry: normal IRP, weak/failed swallows	Gastroesophageal reflux disease	IEM is commonly GERD-associated; high IRP would suggest EGJOO.
Q5	Constipation; ARM shows absent RAIR	Deep suction rectal biopsies	Absent RAIR -> Hirschsprung until biopsy proves otherwise.
Q6	Longstanding constipation plus abdominal pain relieved by BM; no alarms	Linaclotide	IBS-C after simple measures: secretagogue such as linaclotide.
Q7	Recurrent small-bowel dilation without mechanical obstruction	Systemic sclerosis	CIPO + scleroderma = myopathic pseudo-obstruction.
Q8	Corkscrew esophagus in dysphagia	Decreased distal latency in 40% of swallows	DES = premature contractions; corkscrew clue.
Q9	Type III achalasia, severe comorbidity/poor surgical candidate	Botulinum toxin injection into LES	Botox is palliative for frail achalasia patients.
Q10	GERD symptoms, reflux proven, manometry relevant before surgery	Lifestyle modification	Do not jump to fundoplication before full physiologic assessment/optimization.
Q11	Posterior anal fissure with pain/bleeding	Resting pressure of 110 mmHg	Fissure = high internal sphincter resting tone.
Q12	Achalasia pathophysiology	Decreased nitric oxide release	Loss of inhibitory myenteric neurons -> impaired LES relaxation.
Q13	Slow-transit constipation; negative predictor for colectomy	Increased small intestinal transit time	Generalized dysmotility predicts poor ileorectal anastomosis outcome.
Q14	Parkinson disease + gastroparesis + recurrent C. difficile	Low fiber, low residue diet	Avoid chronic erythromycin/metoclopramide risk; diet first.
Q15	NCCP/GERD-linked manometry abnormality	Failed and weak swallows pattern	IEM is the common GERD-associated manometry finding.
Q16	Normal defecation physiology	Bearing down relaxes internal anal sphincter	Defecation = push plus puborectalis/ IAS relaxation.
Q17	Post-gastric surgery early dumping refractory symptoms	Octreotide 50–100 mcg monthly	Dumping: diet first; octreotide for refractory severe symptoms.
Q18	IBS-C; anticholinergics would worsen constipation	Lubiprostone	Secretagogue for IBS-C/chronic idiopathic constipation.
Q19	Asymptomatic healthy control manometry	Large peristaltic break in 60%	Minor motility findings may occur in asymptomatic controls.
Q20	True statement about achalasia therapy	Botox reficacy decreases with repeats	Botox wanes; type II responds best overall; type III poorest except POEM.
Q21	Functional dyspepsia with fullness/early satiety, normal EGD	Buspirone 10 mg before meals	Buspirone improves accommodation/ PDS-type symptoms.
Q22	PPI-refractory heartburn, normal EGD, no surgical target	Nortriptyline 10 mg at bedtime	Functional heartburn -> neuromodulator, not fundoplication.
Q23	Urgency fecal incontinence	Decreased anal sphincter squeeze pressure	Squeeze = external anal sphincter/ voluntary continence.
Q24	Gastric/small bowel motility statement	Fasting MMC cycles every 90–120 minutes	MMC is the interdigestive housekeeping wave.
Q25	Chronic opioid use and constipation question stem	Eluxadoline mechanism option	OIC treatment is laxatives then peripheral mu antagonists; eluxadoline is IBS-D drug.
Q26	Poorly controlled diabetic with gastroparesis symptoms	Improved sugar control	Diabetic gastroparesis: optimize glucose before escalating drugs.
Q27	Refractory diabetic gastroparesis with severe vomiting	Improve symptoms and gastric emptying	Gastric electrical stimulation is reserved for refractory nausea/vomiting.
Q28	Stereotyped vomiting episodes, well between, marijuana use	Discontinue smoking marijuana	Cyclic vomiting/cannabinoid hyperemesis clue.
Q29	NCCP after negative cardiac, EGD, pH and PPI trial	Normal esophageal motility	Functional chest pain commonly has normal HRM.
Q30	Night GERD statement	Less reflux events during sleep	Night events: poorer clearance and greater injury risk.

DDSEP 09 | Chapter 8 | Scenario Table

Scenario-based exam table

Q#	Scenario / Stem	Key Finding / Clue	Most Likely Answer / Take-Home
Q31	Dysphagia lusoria cause	Aberrant right subclavian artery	Vascular ring/compression dysphagia.
Q32	Dysphagia immediately after swallowing, normal esophageal manometry	Polymyositis	Pharyngeal/striated muscle disease can mimic esophageal dysphagia.
Q33	Predictor of good antireflux surgery outcome	Full response to PPI treatment	PPI responder is the best surgical responder.
Q34	NCCP with normal HRM initial treatment	PPI once daily	Empiric GERD treatment is first for many NCCP stems.
Q35	Post-fundoplication progressive dysphagia; pre-op EGD only	High resolution esophageal manometry	Missed achalasia before fundoplication is a classic trap.
Q36	Constipation; paradoxical external sphincter contraction on ARM	Biofeedback	Dyssynergic defecation is retrained, not simply laxative-treated.
Q37	RAIR definition	Transient internal sphincter relaxation to rectal distension	RAIR samples rectal content and excludes Hirschsprung if present.
Q38	Retained markers mainly rectosigmoid	Ano-rectal manometry	Rectosigmoid marker pile-up suggests outlet obstruction.
Q39	Chronic opioids with dysphagia; manometric association	Esophagogastric junction outflow obstruction	Opiates can induce EGJOO/spastic patterns.
Q40	True MMC statement	Partially regulated by motilin	Motilin drives interdigestive MMC cycles.
Q41	Lifelong constipation; suspected Hirschsprung on ARM	Lack of internal sphincter relaxation	Absent RAIR → Hirschsprung; confirm with rectal biopsy.
Q42	Accurate gastroparesis statement	Early satiety/fullness correlate better with emptying	Pain/bloating correlate poorly with gastric emptying delay.
Q43	Long-term metoclopramide with tardive dyskinesia concern	Drug holidays may decrease prevalence	Risk rises with duration/cumulative dose; may be reversible.
Q44	Scleroderma with fecal incontinence	Abnormally low anal resting pressure	Internal anal sphincter smooth muscle is affected by scleroderma.
Q45	Postoperative acute colonic pseudo-obstruction, cecum 11 cm, stable	Nasogastric tube attached to suction	Stable Ogilvie starts conservative decompression and correction.
Q46	Post-bypass symptoms 15 min after carbohydrate meal	Dumping syndrome	Early dumping = osmotic load + vasomotor symptoms.
Q47	Slow waves/basic electrical rhythm source	Interstitial cells of Cajal	ICC = GI pacemaker cells.
Q48	Type III achalasia/spastic features	Peroral endoscopic myotomy (POEM)	POEM allows longer tailored myotomy for spastic achalasia.
Q49	Scleroderma with GERD and dysphagia; HRM diagnosis	Ineffective esophageal motility	Scleroderma esophagus = weak/absent peristalsis + reflux risk.
Q50	Prior Grade C esophagitis, healed on PPI, persistent symptoms + positive SI	Reflux hypersensitivity overlapping with GERD	Objective GERD plus hypersensitive symptom correlation.
Q51	Bothersome epigastric pain/burning, negative work-up	Epigastric pain syndrome	FD subtype: EPS = pain/burning; PDS = fullness/satiety.
Q52	Longstanding hard stools, straining, manual maneuvers; pain not predominant	Functional constipation	Rome constipation symptoms without IBS pain predominance.
Q53	Diabetes neuropathy with passive/nocturnal incontinence clue	Weakness of internal anal sphincter	Passive leakage = low resting tone/internal sphincter.
Q54	Constipation without alarm features; physiologic evaluation needed	Anal rectal manometry	No alarm → no colonoscopy first; define outlet physiology.
Q55	ARM-proven dyssynergic defecation	Biofeedback	Target the pelvic floor pattern.
Q56	Recent bowel change with diarrhea/incontinence; test not indicated	CT scan	Colonoscopy and ARM may help; routine CT is not the first physiologic test.

DDSEP 09 – CHAPTER 11

GI INFECTIONS OF THE SMALL INTESTINE AND COLON

CONCISE EXAM-ORIENTED NOTES – ESOPHAGUS-STYLE EXAM NOTES

Chapter 11 | 52 DDSEP questions

Memory line: This chapter is organism-clue driven: exposure + host risk + stool pattern + complication = answer.

SECTION I – DDSEP EXAM-ORIENTED NOTES

1. Acute infectious diarrhea: first decision is severity, not organism
2. Clostridioides difficile: recognize recurrence and fulminant disease
3. Food poisoning and fish-linked syndromes
4. Bacterial clue organisms
5. Protozoa and tropical malabsorption
6. Helminths, flukes and tapeworms
- ★ High-Yield Organism Snapshot

1 ACUTE INFECTIOUS DIARRHEA: FIRST DECISION IS SEVERITY, NOT ORGANISM

CLINICAL SCENARIO	KEY CLUES	LIKELY CAUSE	MANAGEMENT
MILD Watery diarrhea in immunocompetent adult	• Afebrile • No severe dehydration • No blood in stool	Viral (eg, Norovirus) or non-invasive bacterial	**Hydration ± loperamide** **No empiric antibiotics**
MODERATE / SEVERE Traveler diarrhea (esp. Southeast Asia) or Pregnancy	• Fever, cramps, more severe • High-risk setting (travel, pregnancy)	Enterotoxigenic *E. coli* (ETEC) most common	**Azithromycin is the exam-safe answer**
BLOODY DIARRHEA without fever, severe cramps, later AKI/anemia/ thrombocytopenia	• Afebrile or low-grade fever • Severe abdominal cramps • May develop HUS (AKI, anemia, thrombocytopenia)	Shiga toxin-producing *E. coli* (STEC)	**Send Shiga toxin assay** **Avoid antibiotics** (↑ risk of HUS)
CHOLERA Profuse watery diarrhea	• Profuse "rice-water" diarrhea • Severe dehydration • Minimal/absent fever	*Vibrio cholerae*	**Aggressive fluid & electrolyte replacement** **Azithromycin shortens illness in severe cases**
CAMPYLOBACTER Bloody diarrhea after poultry / raw milk / water	• Fever, cramps, tenesmus • Self-limited in most • Later: Guillain-Barre or reactive arthritis	*Campylobacter jejuni*	**Azithromycin if severe** Later: Guillain-Barre or reactive arthritis

MEMORY LINE — Mild = fluids | Traveler / Severe = azithro | **Afebrile bloody + cramps = STEC: test toxin, no antibiotics**

2 CLOSTRIDIOIDES DIFFICILE: RECOGNIZE RECURRENCE AND FULMINANT DISEASE

CLINICAL SITUATION	KEY FEATURES / CLUES	MANAGEMENT	NOTES
FIRST RECURRENCE (after metronidazole) [in DDSEP]	• Symptoms return after initial response to metronidazole	**Oral vancomycin 125 mg QID for 10 days**	Recommended first-line for first recurrence in DDSEP.
MULTIPLE RECURRENCE (in DDSEP)	• ≥ 2 recurrences • Symptoms resolved between episodes	**Vancomycin course followed by rifaximin chaser**	Monitor if symptoms have resolved and stools are formed.
FULMINANT DISEASE / ILEUS / TOXIC MEGACOLON PATTERN	• Severe colitis, ileus, megacolon • Systemic toxicity	**High-dose oral/NG vancomycin** **+** **IV metronidazole** **+** **Rectal vancomycin** (when ileus is present)	Treat aggressively. Consider ICU care and surgical consult if deteriorating.

RISK OF FURTHER RECURRENCE

Rises sharply after the first recurrence.
Key Risk Factors: Older age | Nursing-home exposure | CKD | Steroids | Antibiotics | PPIs

MEMORY LINE

CDI RECURRENCE = VANCOMYCIN STRATEGY;
FULMINANT = ORAL + RECTAL VANCOMYCIN PLUS IV METRONIDAZOLE.

3 FOOD POISONING AND FISH-LINKED SYNDROMES

CONDITION	SOURCE / SETTING	KEY FEATURES	CAUSE / TOXIN	MANAGEMENT
Staphylococcus aureus (Preformed toxin)	Improperly handled food (meats, dairy, salads)	• Abrupt vomiting and cramps within hours (1–6 h) • Usually no fever or diarrhea	Preformed emetic toxin	Supportive care (fluid, antiemetics)
Scombroid ("Histamine poisoning")	Tuna, mackerel-type fish (poor refrigeration)	• Flushing, rash, headache, palpitations • Nausea, vomiting, diarrhea	Histamine from bacterial action (poor refrigeration)	Supportive care Antihistamines (if needed)
Ciguatera (reef fish poisoning)	Reef fish (tropical waters)	• GI illness (nausea, vomiting, diarrhea) • Temperature-related paresthesias (hot–cold reversal), neurologic symptoms	Ciguatoxin from dinoflagellates	Supportive care No specific antidote
Anisakis (Anisakiasis)	Raw / undercooked marine fish or squid	• Severe epigastric pain hours after ingestion • Nausea, vomiting	Anisakis worm infection	EGD diagnosis and worm extraction

MEMORY LINE

Vomiting-only outbreak = preformed toxin.

Flushing after fish = scombroid.

Hot-cold reversal after reef fish = ciguatera.

4 BACTERIAL CLUE ORGANISMS

ORGANISM	CLINICAL CLUES	SOURCE / RISK FACTORS	KEY FEATURES / COMPLICATIONS	TREATMENT
Yersinia enterocolitica	RLQ pain mimicking appendicitis + diarrhea	Pork (undercooked) Untreated water Iron overload, transfusion risk	• Terminal ileitis, mesenteric lymphadenitis • May mimic acute appendicitis	Supportive care (severe: TMP-SMX or fluoroquinolone)
Listeria monocytogenes	Immunocompromised or pregnant host	Unpasteurized dairy, soft cheeses, deli meats	Febrile gastroenteritis may progress to meningitis / encephalitis, sepsis	Ampicillin + Gentamicin (± TMP-SMX)
Vibrio vulnificus	Septic shock + bullous skin lesions	Raw seafood / shellfish Alcoholism, chronic liver disease	Rapidly progressive infection, septicemia, hemorrhagic bullae	Doxycycline + Ceftazidime (or 3rd gen ceph)
Norovirus (± Rotavirus)	Common food-borne outbreak with vomiting and non-bloody diarrhea (Rotavirus: severe in children)	Contaminated food, water, surfaces (Rotavirus: children, now vaccine-modified)	• Norovirus: short incubation (12–48 h), explosive outbreaks • Rotavirus: severe pediatric diarrhea, dehydration	Supportive care (ORS, hydration)

MEMORY LINE

Iron overload + RLQ pain = *Yersinia*.

Transplant / immunocompromised + fever / seizure = Listeria.

Alcohol + shellfish + shock = *V. vulnificus*.

5 PROTOZOA AND TROPICAL MALABSORPTION

ORGANISM / CONDITION	KEY CLUES	CLINICAL FEATURES	EXAM / DIAGNOSIS	TREATMENT (DDSEP FOCUS)
Entamoeba histolytica	• Subacute course • Right colon disease • Negative routine bacterial culture	• Bloody diarrhea • Abdominal cramps • Tenesmus • Possible liver abscess	• Stool O&P: trophozoites (erythrophagocytosis) • Antigen detection / PCR	**Invasive disease:** 1) Metronidazole 750 mg PO TID for 5–10 days 2) Then luminal agent: Paromomycin 25–35 mg/kg/day in 3 divided doses for 7 days
Cryptosporidium spp.	• Immunocompromised (esp. AIDS) • Watery diarrhea	• Profuse watery diarrhea • May cause: – AIDS cholangiopathy – Cholecystitis pattern	• Stool O&P: oocysts (acid-fast stain) • Antigen detection	Nitazoxanide 500 mg PO BID for 3 days
Cystoisospora belli	• HIV infection / travel • Eosinophilia	• Watery, malodorous diarrhea • Abdominal cramps • Weight loss	• Stool O&P: oocysts (acid-fast stain)	Trimethoprim–sulfamethoxazole (TMP–SMX) 160/800 mg PO BID for 7–10 days
Tropical sprue	• Prolonged travel / residence in endemic regions • Poor sanitation	• Chronic diarrhea • Weight loss, steatorrhea • Macrocytic anemia, folate deficiency	• Duodenal biopsy: villous atrophy • Folate low	Tetracycline 250 mg QID for 3–6 months + Folate supplementation

MEMORY LINE

AMEBA = Metronidazole then luminal agent.

CRYPTO = Immunocompromised watery diarrhea.

C. BELLI = HIV/Travel + Watery diarrhea + Eosinophilia.

TROPICAL SPRUE = Travel + Macrocytosis + Tetracycline / Folate.

6 HELMINTHS, FLUKES AND TAPEWORMS

PARASITE	KEY CLUES / EXPOSURE	CLINICAL FEATURES	EXAM / DIAGNOSIS	TREATMENT (DDSEP FOCUS)
Strongyloides stercoralis	• Endemic / tropical exposure • Years of waxing–waning GI, pulmonary, cutaneous symptoms • Eosinophilia	• Abdominal pain, diarrhea • Cough, wheeze • Risk of hyperinfection (life-threatening)	• Stool O&P: larvae (multiple samples) • Serology: supportive	Ivermectin 200 mcg/kg PO daily for 1–2 days (or until negative) Treat contacts if needed
⚠ **Steroids or TNF inhibitors can trigger hyperinfection with Gram-negative sepsis. Consider *Strongyloides* in any at-risk patient BEFORE immunosuppression.**				
Ascaris lumbricoides	• Travel / poor sanitation • Pig exposure	• Intestinal worms • Pulmonary symptoms (Löffler) • Biliary / pancreatic obstruction	• Stool O&P: ova • Imaging if obstruction	Albendazole 400 mg PO single dose
Hookworm (Ancylostoma, Necator)	• Barefoot exposure • Chronic symptoms • Eosinophilia, iron deficiency	• Abdominal pain, diarrhea • Iron-deficiency anemia • Pruritic rash at entry site	• Stool O&P: ova • CBC: microcytic anemia	Albendazole 400 mg PO single dose
Clonorchis / Opisthorchis	• Korea / China / SE Asia • Raw freshwater fish	• RUQ pain, biliary colic • Eosinophilia • Biliary tract disease (↑ risk cholangioma)	• Stool O&P: operculated ova • ↑ Eosinophils	Praziquantel 25 mg/kg PO TID for 1 day
Diphyllobothrium latum	• Raw freshwater fish / sushi • B12 deficiency risk	• Abdominal symptoms • Macrocytic anemia (B12 deficiency)	• Stool O&P: ova • ↓ Vitamin B12	Praziquantel 10 mg/kg PO single dose

MEMORY LINE

Steroids + Eosinophilia + Gram-negative sepsis = STRONGYLOIDES HYPERINFECTION until proven otherwise.

ASCARIS = Worms + Lungs + Biliary obstruction.

HOOKWORM = Iron-deficiency anemia + Eosinophilia.

CLONORCHIS / OPISTHORCHIS = Raw freshwater fish + Eosinophilia + Biliary disease.

DIPHYLLOBOTHRIUM = Raw fish + B12 deficiency + Macrocytosis.

HIGH-YIELD ORGANISM SNAPSHOT

ORGANISM / SYNDROME	EXAM TRIGGER	BEST DDSEP ACTION
Yersinia *Yersinia enterocolitica*	RLQ pain + diarrhea + sore throat; pork/iron overload	*Yersinia enterocolitica*
Listeria	Transplant/immunosuppression + febrile gastroenteritis + CNS disease	Ampicillin + gentamicin
STEC	Afebrile bloody diarrhea + severe cramps +/- HUS	Shiga toxin assay; avoid antibiotics
Campylobacter	Bloody diarrhea then ascending weakness/ reactive arthritis	Post-infectious GBS clue; azithro if severe
Vibrio vulnificus	Alcohol/liver disease + seafood + septic shock/bullae	Emergency recognition
Scombroid	Fish + flushing/rash/ palpitations within hours	Poor refrigeration -> histamine
Ciguatera	Reef fish + GI + hot-cold paresthesia	Supportive
Strongyloides	Eosinophilia + steroid/ TNF exposure + Gram-negative sepsis	Ivermectin
Amebiasis	Subacute bloody colitis; routine bacterial culture negative	Metronidazole then paromomycin
Cryptosporidium	Watery diarrhea + biliary tract disease in immunocompromised	Nitazoxanide
Tropical sprue	Endemic travel + weight loss + macrocytic anemia	Tetracycline + folate
Clonorchis	Korean/raw fish + RUQ pain + eosinophilia	Praziquantel

RAPID REVISION PEARLS

- RLQ pain is not always appendicitis: Yersinia penetrates submucosa and mesenteric nodes.
- Do not give antibiotics for suspected **STEC**; watch for HUS with CBC, creatinine and electrolytes.
- **Scombroid** is not allergy and not fish infection: it is histamine toxicity from poor refrigeration.
- Steroid exposure with eosinophilia should trigger *Strongyloides* thinking before immunosuppression is intensified.
- Invasive amebiasis needs two steps: tissue agent first, then luminal eradication.
- Biliary parasite clues: **Clonorchis** = East Asian/raw fish/RUQ/ eosinophilia; **Ascaris** can obstruct ducts.

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

Use this table for last-pass recall: identify the exposure/host clue, then lock the organism or management step.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	Hemochromatosis + RLQ pain/diarrhea/sore throat	Yersinia enterocolitica	Iron promotes virulence; pseudoappendicitis clue.
Q2	Korean man + RUQ pain + eosinophilia	Clonorchis sinensis	Chinese liver fluke; biliary disease.
Q3	Renal transplant + febrile gastroenteritis -> seizures	Listeria monocytogenes	Immunocompromised foodborne Listeria can invade CNS.
Q4	RA on prednisone/MTX + waxing GI symptoms + eosinophilia	Strongyloides stercoralis	Chronic infection persists for years; steroids dangerous.
Q5	Watery diarrhea + RUQ pain + thickened GB/CBD dilation	Cryptosporidium parvum	Think AIDS cholangiopathy pattern.
Q6	Raw salmon + acute epigastric pain + thickened stomach	Anisakis infection	EGD visualizes/extracts worm.
Q7	Travel + progressive postprandial pain/obstruction	Ascaris lumbricoides	Can obstruct biliary/pancreatic ducts.
Q8	Tuna + flushing/rash/palpitations within 1 hour	Proper fish refrigeration	Scombroid is histamine from poor storage.
Q9	Chinese man + iron-deficiency anemia	Ancylostoma duodenale	Hookworm causes chronic blood loss.
Q10	Reef fish + diarrhea/lightheadedness + temperature paresthesia	Ciguatera fish poisoning	Neurologic hot-cold reversal clue.
Q11	First CDI recurrence after metronidazole	Vancomycin 125 mg QID x10d	DDSEP recurrence regimen; high dose only fulminant.
Q12	Further CDI recurrence	Vancomycin then rifaximin chaser	Recurrent CDI needs different strategy.
Q13	After successful recurrent CDI treatment; formed stool	Observe for recurrence	Do not treat positive history alone.
Q14	Sushi + flushing/nausea/vomiting in atopic patient	Scombroid fish poisoning	Not oral allergy; fish histamine syndrome.
Q15	Elderly nursing-home patient + CDI + fever/abd pain	PO/rectal vancomycin + IV metronidazole	Fulminant CDI/ileus regimen.
Q16	Recurrent CDI fact	After first recurrence, 40-65% recur	Recurrence begets recurrence.
Q17	Bloody diarrhea -> AKI/anemia/thrombocytopenia	Shiga toxin E. coli	HUS after STEC.
Q18	STEC management statement	Avoid antibiotics	Supportive hydration; monitor CBC/renal function.
Q19	Puerto Rico travel + weight loss + steatorrhea/macrocytosis	Tropical sprue	Endemic travel + malabsorption.
Q20	Mexican farm worker + bloody colitis after steroids	Metronidazole then paromomycin	Invasive amebiasis needs luminal eradication.
Q21	Outbreak of abrupt vomiting/cramps	S. aureus preformed toxin	Short incubation vomiting = preformed toxin.
Q22	Kentucky RA + steroids/TNF + E. coli sepsis	Strongyloides infection	Hyperinfection carries enteric Gram-negative sepsis.
Q23	Confirmed Strongyloides hyperinfection	Ivermectin	Treatment of choice.
Q24	Moderate traveler diarrhea after Southeast Asia	Azithromycin	Best empiric TD agent in many exam stems.
Q25	Bloody diarrhea then ascending weakness	Campylobacter infection	GBS/reactive arthritis complication.
Q26	GBS after diarrhea fact	Up to 40% due to Campylobacter	Campylobacter is classic antecedent infection.
Q27	India/Puerto Rico endemic travel + chronic malabsorption	Tetracycline + folate 3-6 months	Tropical sprue treatment.

DDSEP 09 | CHAPTER 8 | SCENARIO TABLE (CONTINUED)

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q28	Mild acute watery diarrhea, healthy host	Hydration + loperamide PRN	No empiric antibiotics in mild disease.
Q29	Adult after child with vomiting/diarrhea/seizures	Rotavirus	Common severe pediatric diarrheal virus.
Q30	Group outbreak + vomiting/ non-bloody diarrhea	Norovirus	Most common foodborne gastroenteritis.
Q31	Pig farmer passes large roundworm	Albendazole 400 mg once	Ascaris treatment.
Q32	Rural Alabama + severe IDA + eosinophilia	Albendazole 400 mg once	Necator/hookworm.
Q33	Yersinia mechanism	Submucosal penetration + lymph nodes	RLQ mesenteric adenitis mimic.
Q34	HIV + watery malodorous diarrhea + eosinophilia	Cystoisospora	Protozoan exception causing eosinophilia.
Q35	Alcohol + seafood + shock/bloody diarrhea	Vibrio vulnificus	Shellfish-associated fatal sepsis.
Q36	Clonorchis treatment	Praziquantel	Eggs in stool/duodenal aspirate/bile.
Q37	Fish + flushing/rash/headache/GI	Scombroid fish poisoning	Rapid onset after contaminated fish.
Q38	Fish-related outbreak + GI/neurologic syndrome	Ciguatera fish poisoning	Reef fish toxin.
Q39	Afebrile bloody diarrhea + severe cramps	Shiga toxin EIA	STEC test; leukocytes may be rare.
Q40	Profuse watery diarrhea/cholera	Azithromycin	Fluids are mainstay; azithro shortens disease.
Q41	Chronic mild abdominal pain/diarrhea	Hookworm infection	IDA/eosinophilia clinches when provided.
Q42	Hookworm treatment	Albendazole	Single-dose exam answer.
Q43	Acute vomiting/cramps, no fever/diarrhea	Preformed toxin	S. aureus emetic toxin clue.
Q44	Severe/bloody Campylobacter diarrhea	Azithromycin	Treat only severe or high-risk cases.
Q45	Listeria CNS infection treatment	Ampicillin + gentamicin	Classic regimen for invasive Listeria.
Q46	Sushi/freshwater fish + fatigue/B12 issue	Diphyllobothrium latum	Fish tapeworm competes for B12.
Q47	Diphyllobothrium treatment	Praziquantel	Tapeworm treatment of choice.
Q48	Pregnant traveler diarrhea after Thailand	Azithromycin	Pregnancy-compatible TD answer.
Q49	Bloody diarrhea weeks + negative bacterial culture	Entamoeba histolytica	Right colon; stool antigen/serology.
Q50	Initial drug for invasive amebiasis	Metronidazole	Follow with luminal agent.
Q51	Strongyloidiasis treatment	Ivermectin	Albendazole alternative.
Q52	Cryptosporidium treatment	Nitazoxanide	Stool oocysts; biliary involvement clue.

DDSEP 09 – CHAPTER 12

GASTROINTESTINAL BLEEDING

CONCISE ESOPHAGUS-STYLE EXAM NOTES AND SCENARIO FRAMEWORK

Chapter 12 | 50 DDSEP questions

Memory line: Bleeding questions are algorithm questions: resuscitate first, risk-stratify, localize, then choose endoscopy/radiology/therapy.

SECTION I – DDSEP EXAM-ORIENTED NOTES

1. First move in GI bleeding: resuscitate before localization
2. Before EGD: PPI and prokinetics improve the field, not survival
3. Peptic ulcer bleeding: Forrest stigmata decide therapy
4. After ulcer control: remove the cause and balance antithrombotics
5. Variceal and vascular upper/lower lesions
6. Lower GI bleeding and radiology algorithms
7. Small-bowel bleeding: VCE localizes, enteroscopy treats, CTE finds masses

★ High-Yield Bleeding Snapshot

1. FIRST MOVE IN GI BLEEDING: RESUSCITATE BEFORE LOCALIZATION

Massive hematemesis or unstable bleeding:	Place large-bore IV access, give crystalloid/ blood support, protect airway when needed, then endoscopy.
Restrictive RBC transfusion is the DDSEP default:	Transfuse at hemoglobin **<7 g/dL** unless major active cardiovascular disease justifies a higher target.
Melena can occur with as little as about 50 cc of upper GI blood loss;	absence of melena does not exclude an upper source.
Nasogastric lavage is painful and rarely changes management;	a positive lavage has limited predictive value for high-risk lesions.
Glasgow-Blatchford score identifies very-low-risk upper GI bleed patients	who may undergo outpatient endoscopy.

MEMORY LINE: Hemodynamics first. *A stable low-risk bleed can wait; massive hematochezia can still be upper GI until proven otherwise.*

2. BEFORE EGD: PPI AND PROKINETICS IMPROVE THE FIELD, NOT SURVIVAL

Pre-endoscopy PPI	• Can reduce active bleeding/high-risk stigmata and decrease the need for endoscopic therapy. • Does not clearly reduce mortality or surgery.
PPI benefit in acute ulcer bleeding is mainly pH-mediated:	Higher gastric pH improves platelet aggregation and clot stability.
IV erythromycin or metoclopramide before urgent EGD	• Clears blood/clot, improves visualization and reduces repeat endoscopy. • It does not reliably reduce aspiration or mortality.
BUN elevation with normal creatinine	Supports upper GI bleeding due to digestion/ absorption of blood proteins.

MEMORY LINE: PPI downstages stigmata; prokinetic *clears the stomach.*

3. PEPTIC ULCER BLEEDING: FORREST STIGMATA DECIDE THERAPY

Peptic ulcer disease is the commonest cause of upper GI hemorrhage;	NSAIDs, *H. pylori*, older age and prior ulcer bleed are dominant exam risks.
Highest-risk lesion: active arterial spurting (Forrest Ia).	Non-bleeding visible vessel also requires endoscopic therapy.
Adherent clot is intermediate risk;	consider epinephrine, clot removal/irrigation, then definitive therapy if high-risk stigmata are uncovered.
Clean-based ulcer or flat pigmented spot:	no endoscopic therapy; oral PPI and outpatient/early discharge may be appropriate if otherwise low risk.
Injection alone is not definitive.	Mechanical clips or thermal therapy provide better hemostasis; recurrent ulcer bleeding after initial hemostasis usually gets repeat EGD before surgery.
Posterior duodenal wall and posterior lesser curvature gastric ulcers	are higher rebleeding-risk locations.

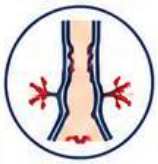
MEMORY LINE: Spurting/visible vessel = treat. Clean base = no therapy. Clot = decide, uncover, then treat if needed.

4. AFTER ULCER CONTROL: REMOVE THE CAUSE AND BALANCE ANTITHROMBOTICS

NSAID-related uncomplicated gastric ulcer:	stop NSAIDs and treat with PPI for about 8 weeks when no malignant features are present.
***H. pylori*-related bleeding ulcer:**	eradicate infection; long-term PPI may be discontinued if no other indication remains.
Bleeding-ulcer history strongly predicts future ulcer hemorrhage	even after healing; avoid recurrent NSAID exposure.
Aspirin decisions depend on cardiovascular indication:	high-risk secondary prevention usually favors early resumption after hemostasis; low-risk situations may permit short holding.
For significant bleeding, DDSEP support goals:	hemoglobin >7 g/dL and platelets at least 50,000/uL; do not delay hemostasis for mild INR elevation alone.

MEMORY LINE: Stop NSAIDs. Eradicate *H. pylori*. Resume aspirin early when cardiac risk outweighs rebleeding risk.

5. VARICEAL AND VASCULAR UPPER/LOWER LESIONS

 **Suspected esophageal variceal hemorrhage:**	• ICU-level resuscitation, airway protection when massive hematemesis. • Antibiotics, vasoactive therapy and urgent banding.
 **Bleeding gastric varix:**	• Cyanoacrylate injection is the classic DDSEP answer. • Epinephrine or clips are not definitive for a gastric varix.
 **GAVE (watermelon stomach):**	Chronic melena/iron-deficiency anemia in older women, systemic sclerosis or cirrhosis; treat with argon plasma coagulation (APC).
 **Radiation proctitis after prostate cancer:**	Painless rectal bleeding with telangiectasia; treat bleeding lesions with APC and avoid unnecessary biopsy.
 **Cameron lesion:**	Linear ulcer within hiatus hernia sac; recurrent/clinically significant bleeding may require hernia repair.
 **Dieulafoy lesion:**	Dilated aberrant submucosal artery, often proximal stomach, with brisk bleeding and little/no surrounding ulcer.

MEMORY LINE: Gastric varix = glue. GAVE/radiation = APC. Cameron = hernia. Dieulafoy = big artery, tiny defect.

6. LOWER GI BLEEDING AND RADIOLOGY ALGORITHMS

 **Hemodynamically stable painless hematochezia:**	Bowel purge until clear effluent, then colonoscopy; diverticular bleeding is the commonest severe painless hematochezia source.
 **Hematochezia with hemodynamic instability or high-risk anticoagulant/ NSAID context:**	Urgent EGD first to exclude brisk upper GI bleeding.
 **Ongoing massive lower GI bleeding after negative EGD and persistent instability:**	Emergent angiography for localization and embolization.
 **Angiography vs scintigraphy:**	• Angiography detects active bleeding at ~0.5–1 cc/min and can treat. • Scintigraphy is more sensitive but less therapeutic.
 **Aortoenteric fistula:**	Prior AAA repair + herald bleed, blood in distal duodenum or recurrent hematemesis/melena; urgent CT/CTA and surgical pathway.
 **Post-polypectomy bleeding, especially right colon/ large polyp/antithrombotics:**	Colonoscopic hemoclip across the vessel or ulcer.

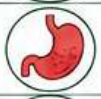
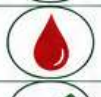
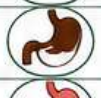
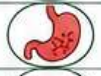
MEMORY LINE: Stable LGIB = prep + colonoscopy. Unstable hematochezia = rule out upper first; if lower and ongoing = angiography.

7. SMALL-BOWEL BLEEDING: VCE LOCALIZES, ENTEROSCOPY TREATS, CTE FINDS MASSES

 **After negative EGD and colonoscopy with recurrent melena/anemia:**	Video capsule endoscopy (VCE) is the usual next diagnostic step.
 **VCE yield is higher with:**	• Recent overt bleeding/melena • Severe anemia • Age >60 • Male sex • Longer bleeding history
 **LVAD, aortic stenosis and chronic anticoagulation point toward small-bowel angioectasias;**	Octreotide can reduce recurrent bleeding burden.
 **A mid-small-bowel lesion requires device-assisted enteroscopy,**	commonly antegrade double-balloon enteroscopy, when therapy is needed.
 **CT enterography is useful**	after negative VCE or when small-bowel mass/enhancing lesion is suspected, especially in an older smoker with occult IDA.

MEMORY LINE: Negative *EGD/colon + melena/IDA* = **capsule;** capsule lesion = deep enteroscopy; negative capsule + tumor concern = CTE.

HIGH-YIELD BLEEDING SNAPSHOT

Exam clue	Locked answer / action
 GBS very low risk	Outpatient endoscopy can be considered
 PPI before EGD	Less high-risk stigmata / less endoscopic therapy
 Forrest Ia or visible vessel	Endoscopic hemostasis required
 Clean-based ulcer	No endoscopic therapy
 Posterior duodenal wall ulcer	High rebleeding risk
 Gastric varix	Cyanoacrylate
 GAVE or radiation proctitis	APC
 Prior AAA repair + GI bleed	Aortoenteric fistula until excluded
 Stable painless hematochezia	Bowel prep then colonoscopy
 Negative EGD/colon + recurrent melena	VCE first, then directed enteroscopy

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

Use this table for last-pass recall: identify the bleed pattern, risk marker, or lesion, then lock the management step.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	Stable hematemesis before EGD; PPI infusion started	Decrease need for endoscopic therapy	Pre-EGD PPI downstages stigmata; not mortality/surgery benefit.
Q2	Minimum blood volume causing melena	50 cc	Small upper-GI blood loss can produce melena.
Q3	Coffee-ground emesis; dark brown stool; question on NG lavage	NG lavage unlikely to change management	NG lavage has poor yield and is poorly tolerated.
Q4	Non-cirrhotic UGIB; transfusion threshold	7 g/dL	Restrictive transfusion strategy is default.
Q5	Mild melena, stable labs, low-risk score	GBS supports outpatient endoscopy	Use GBS for disposition; AIMS65 predicts mortality.
Q6	Radiologic bleeding detection threshold	Angiography detects 0.5-1 cc/min	Angiography is diagnostic and therapeutic.
Q7	Hematemesis; prokinetic before EGD	Less repeat endoscopy	Prokinetic clears stomach/clot and improves views.
Q8	Older woman; intermittent melena + IDA	APC	GAVE/watermelon stomach = APC.
Q9	Cirrhosis; bleeding gastric cardia varix	Inject cyanoacrylate	Gastric varix = glue, not clips or epinephrine.
Q10	Most likely UGIB source in US	Peptic ulcer disease	PUD remains the commonest UGIB cause.
Q11	Adherent clot on gastric ulcer; no therapy	25 percent	Adherent clot has intermediate rebleeding risk.
Q12	Diverticular bleeding epidemiology	Clinically significant bleeding ~5%	Diverticulosis common; bleeding in minority.
Q13	Recurrent anemia/melena; negative EGD + colonoscopy	Recent melena increases VCE yield	Overt/recent bleeding improves capsule yield.
Q14	Mechanism of acute PPI benefit	Optimize platelet aggregation by raising pH	Higher pH stabilizes platelet plug/clot.
Q15	Aortic stenosis + recurrent small-bowel angioectasia bleed	Subcutaneous octreotide	Angioectasia bleeding; octreotide can reduce recurrence.
Q16	Peptic ulcer site with highest rebleeding risk	Posterior duodenal wall	Posterior duodenal ulcer overlies major vessels.
Q17	CAD on aspirin; ulcer clot removed to pigmented spot	Bid PPI and hold aspirin one week	DDSEP nuance: short hold when cardiac risk lower.
Q18	Linear ulcer in hiatal hernia sac	Refer for hiatus hernia repair	Cameron lesion persists due to mechanical trauma.
Q19	Ulcer bleeding recurrence after healing	10-20x recurrent hemorrhage risk	Prior ulcer bleed is a major risk marker.
Q20	Duodenal ulcer with visible vessel	Clips better than injection alone	Injection alone is not definitive hemostasis.
Q21	Older man; painless rectal bleeding; telangiectasia pattern	Prostate cancer	Radiation proctitis classically follows prostate RT.
Q22	Brisk UGIB with tiny mucosal defect	Dilated aberrant submucosal artery	Dieulafoy = large artery beneath small defect.
Q23	AAA repair + hematemesis/hematochezia; blood in D3	Stat CT abdomen	Think aortoenteric fistula; do not delay.
Q24	Variceal bleed initial bundle question	IV PPI drip	PPI is not the core variceal bundle; airway/antibiotics/vasoactive/EGD are.
Q25	Small NSAID-related gastric ulcer	Stop NSAIDs + PPI 8 weeks	Remove cause; no routine surveillance if low-risk benign ulcer.
Q26	Bleeding H. pylori ulcer after eradication	Discontinue PPI	After eradication, long-term PPI not needed without another indication.
Q27	Clean-based duodenal bulb ulcer	No endoscopic therapy	Forrest III = low rebleeding risk.
Q28	Massive hematemesis in ED	Two 18G IVs + crystalloid	Resuscitation precedes drugs, lavage, or endoscopy.
Q29	Pre-endoscopy PPI expected effect	Reduced high-risk stigmata needing therapy	Same principle as Q1: downstage lesion.
Q30	Ulcer without high-risk stigma	No endoscopic therapy	Treat based on Forrest classification.

DDSEP 09 | CHAPTER 8 | SCENARIO TABLE

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q31	Low-risk clean-based gastric ulcer; stable	Discharge with oral PPI	Low-risk post-EGD patients can be outpatient.
Q32	Most common severe UGIB source	Peptic ulcer disease	Massive hematochezia can still be upper source.
Q33	Highest-risk ulcer stigma	Active arterial spurting (Forrest Ia)	Forrest Ia has greatest rebleeding risk.
Q34	Recurrent bleeding after initial ulcer hemostasis	Repeat EGD for second hemostasis attempt	Repeat endoscopic therapy before surgery if feasible.
Q35	Large hematochezia + instability + warfarin/aspirin	Urgent upper endoscopy	Unstable hematochezia: exclude brisk UGIB first.
Q36	Stable painless hematochezia	Bowel prep then colonoscopy	Diverticular bleed workup starts with purge + colonoscopy.
Q37	Radiation proctitis bleeding	APC of bleeding lesions	Non-contact APC is standard endoscopic therapy.
Q38	Pain + hematochezia; watershed colonic thickening	Ischemic colitis	Splenic flexure/descending colon = watershed clue.
Q39	LVAD + anticoagulation + anemia after negative EGD/colon	Video capsule endoscopy	LVAD predisposes to small bowel angioectasia.
Q40	Older smoker, occult IDA after negative evaluation	CT enterography	CTE finds small-bowel masses/ enhancing lesions.
Q41	Massive LGIB unstable; EGD negative	Emergent angiography	Ongoing unstable lower bleed needs localization/embolization.
Q42	Prior AAA graft + herald bleed; stable	CT angiography	Stable suspected aortoenteric fistula = CTA.
Q43	Painless large-volume hematochezia with clots	Diverticular disease	Most common acute severe hematochezia source.
Q44	Advanced CAD on aspirin; ulcer bleed controlled	Resume aspirin now	High cardiac risk favors early aspirin resumption.
Q45	Post-polypectomy ulcer/vessel in right colon on warfarin	Mechanical hemoclip	Post-polypectomy bleeding = clip vessel/ulcer.
Q46	Ulcer stigma requiring therapy	Non-bleeding visible vessel	Visible vessel is high-risk.
Q47	Adherent clot in ulcer; ESRD/aspirin	Epinephrine then irrigate/snare clot removal	Treat clot if high-risk context and access allow.
Q48	Bleeding with platelets 80k, INR 2.1	RBC if Hb <7 g/dL	Keep platelets >=50k; restrictive RBC threshold.
Q49	Acute hematemesis; need better EGD view	IV prokinetic	Erythromycin/metoclopramide improve visualization.
Q50	Capsule shows mid-small-bowel AVM; anticoagulated	Antegrade double-balloon enteroscopy	Device-assisted enteroscopy reaches and treats mid-SB lesions.

DDSEP 09 – CHAPTER 13

INFLAMMATORY BOWEL DISEASE

CONCISE MRCP / DDSEP EXAM PEARLS IN CHAPTER 01 ESOPHAGUS STYLE

Chapter 13 | 50 DDSEP questions

Memory line: IBD questions are pattern recognition questions: disease activity, phenotype, complications, therapy safety, and long-term prevention.

SECTION I - EXAM PEARL NOTES

1. Mild UC, induction logic, and CAM traps
2. Moderate-severe IBD: escalation, induction, and drug class switching
3. Acute severe UC: do not miss infection, rescue, or thrombosis
4. Thiopurine safety: age, pancreatitis, TPMT, lymphoma
5. Biologic and small-molecule safety
6. Pregnancy, fertility, and infant vaccines
7. Postoperative Crohn's and pouch disorders
8. Extraintestinal disease, mimics, and metabolic clues
9. Skin, joints, and paradoxical reactions
10. Surveillance and prevention

1 MILD UC, INDUCTION LOGIC, AND CAM TRAPS

Goal: Induce remission in mild left-sided UC using proven therapies, not CAM alone.

KEY POINTS	DDSEP PEARLS
Mild Left-Sided UC: Combine Oral + Rectal 5-ASA Use both oral 5-ASA and rectal 5-ASA (enema or suppository). Topical treatment is not optional when distal disease is active.	• Topical therapy treats the mucosa where disease is active. • Oral + rectal 5-ASA is more effective than oral 5-ASA alone.
Cannabis (CAM) Can Soothe Symptoms, Not Mucosa Cannabis may improve symptoms or perceived quality of life, but DDSEP does not treat it as remission-inducing or remission-maintaining therapy.	• May help with symptoms (pain, sleep, appetite). • Not a substitute for evidence-based IBD therapy.
Sulfasalazine Intolerance or Adverse Effect Switch to mesalamine (5-ASA). Remember folate supplementation if sulfasalazine is used.	• Sulfasalazine can cause headache, nausea, rash, oligospermia. • Give folic acid 1 mg daily with sulfasalazine.
Indefinite Dysplasia During Active Inflammation Treat inflammation first (optimize medical therapy), then repeat surveillance colonoscopy after mucosal healing.	• Active inflammation can make dysplasia assessment unreliable. • Reassess after mucosa has healed.

MEMORY: **Mild left UC = oral + rectal 5-ASA.**
CAM can soothe symptoms, not mucosa.

2 MODERATE–SEVERE IBD: ESCALATION, INDUCTION, AND DRUG CLASS SWITCHING

Goal: Induce remission and maintain it with the right drug, at the right dose, in the right patient.

Steroid-Dependent Moderate–Severe UC

Needs a steroid-sparing induction and maintenance strategy.

Infliximab is a key DDSEP answer.

Thiopurines Are for Maintenance, Not Induction

Azathioprine/6-MP are maintenance agents, not reliable for induction.

Low TPMT activity pushes away from azathioprine/6-MP.

Adequate Infliximab Trough + Active Disease = Mechanistic Failure

If drug level is adequate and disease is still active, do not just increase dose.

Switch class (e.g., vedolizumab).

Low Drug Level Without Antibodies = PK Failure

If drug level is low and there are no/low antibodies, optimize the index drug:

increase dose or shorten interval.

MEMORY: **TDM RULE: LOW LEVEL → OPTIMIZE; ADEQUATE LEVEL + ACTIVE DISEASE → SWITCH MECHANISM.**

3 ACUTE SEVERE UC: DO NOT MISS INFECTION, RESCUE, OR THROMBOSIS

KEY POINTS	DDSEP PEARLS
 **Do Not Miss Infection** Hospitalized severe bloody diarrhea in UC: send stool testing for *C. difficile* and other pathogens before attributing everything to flare.	• C. difficile is common and can mimic or trigger a flare. • Always test before escalating immunosuppression.
 **IV Corticosteroids Are First-Line** IV steroids are the initial therapy. Lack of meaningful response by ~72 hours triggers rescue therapy.	• Reassess at ~72 hours using clinical criteria. • Rescue options: **infliximab** (preferred) or **cyclosporine.**
 **Cyclosporine Neurotoxicity Risk** Cyclosporine neurotoxicity and seizure risk is higher when serum cholesterol is very low. DDSEP flags **cholesterol 52 mg/dL.**	• Monitor neurologic status closely. • Very low cholesterol (<100 mg/dL) increases risk; **52 mg/dL** is a red flag.
 **Do Not Miss Thrombosis** Admitted IBD flare = pharmacologic VTE prophylaxis with LMWH unless contraindicated, even when hematochezia is present.	• IBD flare itself is a strong VTE risk. • Use LMWH unless active major bleeding, severe anemia with instability, or other contraindication.

MEMORY: ASUC = stool C. diff, IV steroids, reassess at 72 h, give LMWH.

4 THIOPURINE SAFETY: AGE, PANCREATITIS, TPMT, LYMPHOMA

KEY POINTS	DDSEP PEARLS
 **Older Patient in Long Remission** In an older patient in long-term remission on infliximab + 6-MP, stop 6-MP and maintain infliximab monotherapy. Thiopurine lymphoma risk rises with age.	• Age is a strong independent risk factor for **thiopurine-associated lymphoma.** • De-escalate thiopurine in older patients in sustained remission.
 **Hepatosplenic T-Cell Lymphoma (HSTCL)** Young male + thiopurine exposure, especially with anti-TNF combination, is the classic HSTCL risk frame.	• Typically affects males <35 years. • EBV-negative most commonly. • Counsel about symptoms: fever, night sweats, weight loss, abdominal pain.
 **Thiopurine-Induced Pancreatitis** 6-MP/azathioprine pancreatitis is idiosyncratic (not dose related) and usually requires permanent **discontinuation** rather than dose reduction.	• Rechallenge often leads to recurrence. • Switch to an alternative maintenance agent.
 **TPMT Testing Before Thiopurines** Check TPMT activity or genotype before starting thiopurines. Low activity predicts serious marrow toxicity.	• Low or absent TPMT → avoid or use reduced dose with extreme caution. • Monitor CBC and LFTs regularly after initiation.

MEMORY: Thiopurines: lymphoma in older patients, HSTCL in young males, pancreatitis = stop.

5 BIOLOGIC AND SMALL-MOLECULE SAFETY

KEY POINTS	DDSEP PEARLS
Anti-TNF Contraindicated in Demyelinating Disease Anti-TNF is contraindicated with personal demyelinating disease such as optic neuritis or multiple sclerosis.	• New or worsening demyelination can occur. • Choose a different mechanism.
Anti-TNF and Heart Failure Anti-TNF can worsen advanced heart failure. DDSEP flags class III/IV heart failure as a contraindication frame.	• Avoid anti-TNF in NYHA class III/IV heart failure. • Use alternative therapy.
Tofacitinib: Key Safety Points • Check lipids after 4–8 weeks. • Increased risk of herpes zoster. • Use inactivated recombinant zoster vaccine when appropriate (not live vaccine).	• Monitor CBC, LFTs, lipids per guidelines. • Counsel on infection risk (zoster, serious infections). • Use lowest effective dose.
Vedolizumab: Gut-Selective Safety Vedolizumab is a gut-selective anti-α4β7. Unlike natalizumab, it is NOT classically linked to PML in DDSEP reasoning.	• Favorable safety profile compared with systemic biologics. • Not associated with PML in DDSEP context.

MEMORY: **Anti-TNF:** no MS/optic neuritis, no advanced HF.
Tofacitinib: lipids + zoster.

6 PREGNANCY, FERTILITY, AND INFANT VACCINES

KEY POINTS	DDSEP PEARLS
Active IBD and Pregnancy Active IBD is more dangerous to pregnancy outcome than most maintenance therapy. Continue needed anti-TNF and azathioprine during pregnancy in DDSEP cases.	• Aim for remission before conception. • Continue infliximab and azathioprine if needed. • Monitor closely during pregnancy.
Methotrexate is Teratogenic • Stop methotrexate well before conception (at least 3 months). • Continue effective infliximab if it is maintaining remission.	• Use folic acid supplementation. • Avoid MTX in both men and women planning pregnancy.
IPAA and Fertility Considerations IPAA can reduce fertility. In dysplasia requiring surgery, consider colectomy with ileostomy and defer pouch creation until family completion.	• Counsel early about fertility impact. • Staged approach may be best in young patients.
Infant Vaccines After In-Utero Anti-TNF If anti-TNF was given after 20 weeks gestation, avoid live infant vaccines (e.g., rotavirus) during the first six months.	• Inactivated vaccines are safe. • Coordinate with pediatric team. • Live vaccines (rotavirus, BCG, oral polio) wait for 6 months.

MEMORY: **Pregnancy rule: remission first.**
Stop MTX; continue anti-TNF/azathioprine if needed;
infant live vaccines wait.

7 POSTOPERATIVE CROHN'S AND POUCH DISORDERS

KEY POINTS	DDSEP PEARLS
 **Watery Diarrhea After Ileal Resection Without Recurrent Crohn's = Bile Salt Diarrhea** • Occurs due to bile acid malabsorption. • Start cholestyramine. • Consider SIBO if no response.	• Bile salt diarrhea is common after ileal resection. • Cholestyramine 4 g 1–4 times daily before meals. • If non-response → evaluate for SIBO.
 **High-Risk Postoperative Crohn's: Early Prophylaxis + Endoscopic Monitoring** • High-risk features: smoking, penetrating disease, ≥2 prior resections, perianal disease. • Start early prophylaxis, often anti-TNF + immunomodulator. • Perform colonoscopy 6–12 months after resection.	• Early anti-TNF (e.g., infliximab) + immunomodulator reduces endoscopic recurrence. • Colonoscopy at 6–12 months to detect recurrence and guide therapy.
 **Post-IPAA: Increased Stool Frequency with Pouch Inflammation = Pouchitis** • Presents with increased stool frequency, urgency, cramps. • Usually antibiotic-responsive. • NSAIDs are a trigger → stop NSAIDs.	• Pouchitis = inflammation of the pouch. • First-line: antibiotics (e.g., ciprofloxacin or metronidazole). • Avoid NSAIDs (trigger).
 **Bleeding/Urgency from Retained Rectal Cuff = Cuffitis** • Occurs in retained rectal mucosa. • Presents with bleeding, urgency, tenesmus. • Treat with topical mesalamine suppositories.	• Cuffitis = inflammation of the rectal cuff. • Treat with topical 5-ASA suppositories. • Rule out dysplasia if persistent.

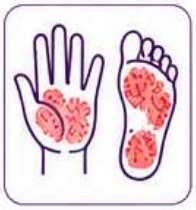
MEMORY: After ileal resection: bile salts.
After pouch: pouchitis = antibiotics; cuffitis = suppository.

8 EXTRAINTESTINAL DISEASE, MIMICS, AND METABOLIC CLUES

KEY POINTS	DDSEP PEARLS
 **Crohn's with Ileal Disease/Resection → Calcium Oxalate Stones** • Fat malabsorption → ↑ oxalate absorption. • Prevention: low oxalate, high calcium, low fat diet.	• Encourage adequate hydration. • Low oxalate (avoid spinach, nuts, chocolate, tea). • High calcium with meals binds oxalate. • Low fat diet (<25% of calories).
 **IBD with Persistent Symptoms Despite Therapy → Test for Celiac Disease** • Check total IgA and IgA-tTG. • Consider duodenal biopsy if serology positive or suspicion remains high.	• Celiac disease can coexist with IBD. • Total IgA first to rule out IgA deficiency. • IgA-tTG is the preferred initial test.
 **NSAID-Induced Enteropathy Can Mimic Small-Bowel Crohn's** • Can cause diaphragm-like strictures, ulcers, increased permeability. • First step = stop NSAIDs.	• NSAID enteropathy can mimic Crohn's disease. • Stopping NSAIDs may lead to improvement. • Consider capsule endoscopy if diagnosis unclear.
 **Older Woman with Watery Non-Bloody Diarrhea and Normal-Looking Colon → Microscopic Colitis** • Two types: collagenous and lymphocytic colitis. • Diagnosis requires colonic biopsy.	• Presents with chronic watery non-bloody diarrhea. • Colonoscopy is often normal. • Biopsy is required for diagnosis.
 **IBD Mimic Checklist** • Always think of other causes when IBD does not fit or does not respond.	• Celiac disease • C. difficile infection • NSAID enteropathy • Microscopic colitis • Bile salt diarrhea • SIBO

MEMORY: IBD mimic checklist: celiac, C. diff, NSAID enteropathy, microscopic colitis, bile salts, SIBO.

9 SKIN, JOINTS, AND PARADOXICAL REACTIONS

KEY POINTS	DDSEP PEARLS
 **Erythema Nodosum** The most common IBD skin manifestation. **Usually parallels luminal disease activity.**	• Tender, erythematous nodules on the shins. • Treat underlying IBD activity. • Resolves as IBD improves.
 **Anti-TNF–Induced Psoriasis** Commonly involves **palms, soles, and scalp.** Mild cases may be managed dermatologically while continuing anti-TNF.	• Appears after starting anti-TNF (weeks to months). • Lesions: erythematous, scaly plaques. • Topical therapy ± dermatology referral for mild cases. • Continue anti-TNF if skin disease is mild and IBD control is good.
 **Severe Anti-TNF Psoriasis** Severe, extensive, or distressing psoriasis may require stopping anti-TNF and switching class. Example in Crohn's disease: ustekinumab (IL-12/23 inhibitor).	• Consider switch if psoriasis is severe/refractory or significantly impairs quality of life. • Choice of next agent guided by IBD phenotype and prior exposures.
 **Assess Disease Objectively** Inflammatory markers and objective disease assessment remain essential. **Symptoms alone are insufficient.**	• Use CRP, fecal calprotectin, endoscopy, and imaging as needed. • Treat to target: clinical, biomarker, and endoscopic remission.

MEMORY: EN follows IBD activity; anti-TNF psoriasis favors palms/soles/scalp.

10 SURVEILLANCE AND PREVENTION

KEY POINTS	DDSEP PEARLS
 **PSC With Colitis** Colonoscopy at diagnosis and annually thereafter. **PSC is the strongest CRC risk modifier in IBD.**	• Start colonoscopy at time of PSC diagnosis (if colitis present) and repeat annually. • Highest risk for colorectal cancer and dysplasia.
 **Non-PSC Extensive Colitis** First surveillance is usually **8–10 years** after diagnosis. Subsequent interval depends on individual risk.	• Consider risk factors: active inflammation, family history of CRC, strictures, prior dysplasia, primary sclerosing cholangitis. • Tailor interval: typically every 1–3 years.
 **Invisible/Flat Dysplasia** Should be confirmed by **expert pathology** and re-evaluated with high-quality **chromoendoscopy.**	• Flat lesions are common and easy to miss. • Expert GI pathologist confirmation is essential. • Repeat high-definition chromoendoscopy for complete assessment.
 **Prevention in IBD Care** Comprehensive preventive care reduces infections and complications.	• Vaccinations: pneumococcal, annual influenza. • HPV vaccination and cervical screening. • Zoster prevention (recombinant zoster vaccine) when appropriate. • Assess and manage osteoporosis, especially with steroid exposure.

MEMORY: PSC-colitis = annual colonoscopy from diagnosis. Flat dysplasia = expert pathology + chromoendoscopy.

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	Mild left UC asks about CAM/cannabis	Cannabis may improve symptoms but not remission	Symptom relief is not mucosal healing.
Q2	Post-ileal resection watery non-bloody diarrhea; no recurrence	Start daily cholestyramine	Ileal loss → bile salt diarrhea.
Q3	Age 73, long remission on infliximab + 6-MP	Stop 6-MP; continue infliximab	Older age magnifies thiopurine lymphoma risk.
Q4	Extensive UC + PSC early after UC diagnosis	Colonoscopy now and annually	PSC overrides the usual 8-10 year clock.
Q5	Pregnant Crohn's, controlled on biologic + thiopurine	Continue adalimumab and azathioprine	Active disease is the main pregnancy risk.
Q6	On adalimumab, asks vaccine/ travel prevention	Avoid yellow fever vaccine	Live vaccine + immunosuppression = contraindicated.
Q7	Active small-bowel CD despite therapeutic 6-TGN	Stop azathioprine; start infliximab	Therapeutic metabolite + active disease = thiopurine failure.
Q8	Indefinite dysplasia during active extensive UC flare	Oral + rectal 5-ASA; repeat scope in 3 months	Inflammation can mimic dysplasia.
Q9	Hospitalized severe UC flare	Test stool for C. difficile toxins	Exclude superinfection in every severe flare.
Q10	UC controlled on sulfasalazine; skin nodules/EN	Switch sulfasalazine to mesalamine	EN often tracks IBD activity; sulfa can confuse skin picture.
Q11	6-MP started; pancreatitis symptoms in 3 weeks	Stop 6-MP; later biologic	Thiopurine pancreatitis is idiosyncratic.
Q12	Crohn's ileal resection + calcium oxalate stone	Low oxalate, high calcium, low fat	Fat malabsorption frees oxalate absorption.
Q13	Tofacitinib for refractory UC	Check lipids after 4–8 weeks	JAK inhibitor = lipid and zoster vigilance.
Q14	IPAA patient with pouchitis after NSAIDs	Stop NSAIDs	NSAIDs can trigger pouchitis/enteritis.
Q15	IBD with anemia/persistent symptoms; celiac question	Total IgA + IgA-tTG	Celiac can coexist with IBD.
Q16	High-risk Crohn's after resection	Adalimumab + azathioprine; scope 6–12 months	High-risk postop CD needs early prophylaxis.
Q17	UC dysplasia but wants future fertility	Colectomy + ileostomy; defer IPAA	IPAA, not colectomy alone, reduces fertility.
Q18	Anti-TNF contraindication scenario	Optic neuritis history	Anti-TNF can worsen demyelination.
Q19	Perianal CD active on infliximab; low-level failure frame	Shorten infliximab interval to q6w	Pharmacokinetic failure → optimize index drug.
Q20	RA on NSAIDs with small-bowel strictures	Stop all NSAIDs	NSAID enteropathy can mimic Crohn's.
Q21	UC active despite adequate infliximab trough	Switch to vedolizumab	Mechanistic failure → change class.
Q22	Anti-TNF psoriasis clue	Palms, soles, scalp	Paradoxical psoriasis has typical distribution.
Q23	Cyclosporine rescue planned; cholesterol 52	Low cholesterol predicts neurotoxicity/seizures	Cyclosporine + low cholesterol is dangerous.
Q24	Target not approved for IBD therapy	IL-17	IL-17 blockade is not an IBD target in this frame.
Q25	Moderate UC failed oral + rectal mesalamine; low TPMT	Start vedolizumab induction	Thiopurines are unsafe/slow for induction.
Q25	Counseling on azathioprine complications	All of the above	Think marrow suppression, infection, lymphoma, pancreatitis.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q26	Counseling on azathioprine complications	All of the above	Think marrow suppression, infection, lymphoma, pancreatitis.
Q27	UC on tofacitinib: inactivated vaccine	Shingrix	Recombinant zoster vaccine is inactivated.
Q28	UC relapses on infliximab monotherapy	Check infliximab level/antibody	Do TDM before changing biologic.
Q29	Post-ileocecal resection Crohn's surveillance	Colonoscopy at 6 months	POCER logic: endoscopic recurrence guides therapy.
Q30	IBD osteoporosis screening risk factor	>3 months steroid use	Steroid exposure is the key DDSEP trigger.
Q31	Mild left-sided UC induction/maintenance	Oral + topical mesalamine	Distal disease needs rectal 5-ASA.
Q32	Wants pregnancy; on infliximab + methotrexate	Stop MTX; continue infliximab	MTX is teratogenic; anti-TNF can continue.
Q33	Drug approved for both CD and UC	Vedolizumab	Gut-selective anti-integrin works in both.
Q34	Steroid-dependent paracolonic UC	Infliximab	Induces and maintains remission in UC.
Q35	Tofacitinib complications	All of the above	JAK: zoster, lipids, infection/VTE vigilance.
Q26	Crohn's preventive measure before biologic escalation	Pneumococcal vaccination	Use inactivated vaccines; avoid live during biologics.
Q37	IBD remission then new watery diarrhea	C. difficile PCR/toxin testing	IBD itself increases CDI risk.
Q38	Older woman, watery diarrhea, NSAIDs, normal stool culture	Microscopic colitis	Normal colonoscopy still needs biopsies.
Q39	Post-IPAA urgency + blood from cuff	Mesalamine suppositories	Cuffitis is topical 5-ASA territory.
Q40	New moderate-severe UC; best combo evidence	Infliximab + azathioprine	UC SUCCESS style combination therapy.
Q41	Hepatosplenic T-cell lymphoma risk	Male gender	Young male + thiopurine + anti-TNF.
Q42	Severe anti-TNF psoriasis in controlled Crohn's	Stop infliximab; start ustekinumab	Severe paradoxical psoriasis -> switch class.
Q43	Vedolizumab adverse events	No important common AE difference	Gut-selective; PML concern belongs to natalizumab.
Q44	Hospitalized UC flare improving on steroids	LMWH prophylaxis	IBD flare is prothrombotic despite bleeding.
Q45	Obese UC flare; anti-TNF clearance factor	Increased BMI	High BMI/inflammation/low albumin increase clearance.
Q46	Infant exposed to adalimumab after week 20	Avoid/postpone rotavirus vaccine	No live vaccines for first 6 months.
Q47	New PSC with colitis	Annual colonoscopy from UC diagnosis	PSC-colitis = yearly surveillance immediately.
Q48	Crohn's with class III/IV heart failure	Infliximab contraindicated	Anti-TNF may worsen advanced HF.
Q49	Invisible dysplasia on surveillance UC colonoscopy	Repeat colonoscopy with chromoendoscopy	Flat dysplasia needs high-quality reassessment.
Q50	Colectomy risk factors in UC	All of the above	Assess disease risk, not only current severity.
Q51	Female with colonic Crohn's long-term risk	Cervical dysplasia	Pap/HPV prevention matters in IBD.
Q52	Crohn's patient asks about contraception/menses	Oral contraception allowed; discuss VTE risk	IBD raises VTE risk; symptoms may fluctuate with menses.

DDSEP 09 – CHAPTER 14

GASTROINTESTINAL CANCERS

CONCISE MRCP / DDSEP EXAM PEARLS IN CHAPTER 01 ESOPHAGUS STYLE

Chapter 14 | 50 DDSEP questions

Memory line: Read Section I as the clinical note map, then use Section II as a rapid scenario-to-answer recall table. The wording preserves DDSEP exam logic and deliberately avoids long textbook expansion.

SECTION I - EXAM PEARL NOTES

1. Esophageal cancer: risk, staging and endoscopic therapy
2. Gastric cancer, *H. pylori* and gastric lymphoma
3. Small intestinal cancers and neuroendocrine clues
4. Pancreatic cancer and pancreatic cystic neoplasms
5. Hepatobiliary malignancy patterns
6. Colorectal cancer screening and post-cancer surveillance
7. Lynch syndrome and hereditary diffuse gastric cancer
8. Polyposis syndromes: visual and endoscopic traps
9. Colon polyp surveillance, malignant polyps and gallbladder polyps
10. Gastric NET, GIST and pancreatic paraneoplastic clues

1. Esophageal cancer: risk, staging and endoscopic therapy

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Caustic alkali ingestion is a major squamous cancer risk.	Surveillance upper endoscopy with **chromoendoscopy**.
Barrett's with carcinoma in *situ*/intramucosal visible nodularity.	**EMR** of visible lesion + **RFA** of remaining Barrett's segment when no deep invasion or nodes.
Nodal staging in esophageal cancer.	**EUS-FNA** is superior to CT for nodal staging. CT/PET assess distant disease.
Epidemiology in the US.	Esophageal cancer is strongly male-predominant; in women, small intestinal cancer is even more common than esophageal cancer.

MEMORY PEARLS

- Caustic history → **SCC** surveillance.
- Barrett CIS → **EMR + RFA.**
- Esophageal nodes → **EUS-FNA.**

2. Gastric cancer, *H. pylori* and gastric lymphoma

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
H. pylori is the dominant risk factor for non-cardia intestinal-type gastric adenocarcinoma.	**Eradication** reduces intestinal-type gastric cancer risk.
Gastric cancer screening in the US general population.	General-population screening is **not recommended.** Targeted screening belongs to high-risk populations.
Gastric MALT lymphoma.	B-cell, often *H. pylori*-associated, and may regress after *H. pylori* eradication.
CDH1 pathogenic variant / hereditary diffuse gastric cancer.	Prophylactic **total gastrectomy** is the DDSEP answer.

MEMORY PEARLS

- ***H. pylori:*** ulcer + intestinal gastric cancer + MALT.
- Screen high-risk only, not the general population.
- **CDH1 =** total gastrectomy.

3. Small intestinal cancers and neuroendocrine clues

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Small intestinal cancer is rare; most common histology in the US.	Adenocarcinoma is the most common, usually **duodenal**.
Risk factors for small bowel adenocarcinoma.	Lynch syndrome, Crohn's disease, celiac disease and FAP. **CVID** is the DDSEP exception.
Terminal ileal yellow submucosal nodule with diarrhea suggests carcinoid.	Systemic carcinoid syndrome requires **hepatic metastasis** bypassing first-pass metabolism.
Diphyllobothrium and celiac lymphoma belong elsewhere.	Enteropathy-associated T-cell lymphoma is linked to **celiac disease**.

MEMORY PEARLS

- Ileal carcinoid + diarrhea = **liver metastasis.**
- Small bowel adenocarcinoma: **duodenum.**
- Risk factors: Crohn/celiac/FAP/Lynch.

HIGH-YIELD TAKEAWAY

Caustic history → SCC surveillance. Barrett CIS → EMR + RFA. Esophageal nodes → EUS-FNA.
***H. pylori*:** ulcer + intestinal gastric cancer + MALT. CDH1 = total gastrectomy.
Ileal carcinoid + diarrhea = liver metastasis. Small bowel adenocarcinoma: duodenum, Crohn/celiac/FAP/Lynch.

4. Pancreatic cancer and pancreatic cystic neoplasms

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Pancreatic adenocarcinoma risk factors.	Age, male sex, smoking, obesity, African American race, chronic pancreatitis. Female sex **is not** the risk answer.
Why pancreatic cancer has poor prognosis.	**Late presentation** is the main driver: most patients are **unresectable** at diagnosis; only a minority are Whipple candidates.
High-risk kindreds screening strategy.	Annual **EUS or MRCP** starting at **age 50** or 10 years before the youngest affected relative. Age 40 when the relative was diagnosed at 50.
Mucinous cystic neoplasm (MCN) management.	MCN in body/tail with ovarian stroma phenotype is **resection** territory. Resected non-invasive MCN needs **no surveillance**.

MEMORY PEARLS

- Pancreas: late unresectable disease.
- Familial screen = EUS/MRCP.
- MCN tail = distal pancreatectomy.

5. Hepatobiliary malignancy patterns

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Cholangiocarcinoma (CCA) risk factors.	PSC, Caroli disease, liver flukes, choledochal cysts, Thorotrast. Cholelithiasis alone is **not** the classic risk answer.
Hepatocellular carcinoma (HCC) epidemiology.	Tracks with **HBV** globally. Aflatoxin B1 and HBV act synergistically. HBV can cause HCC **without cirrhosis**.
When not to screen for HCC.	**Do not screen** when life expectancy or candidacy for curative treatment/ transplant is absent, e.g. severe non-transplantable comorbidity.
Vinyl chloride exposure with liver lesions.	Splenic nodules, ascites, abnormal LFTs → **hepatic angiosarcoma**, not simple hemangioma.

MEMORY PEARLS

- HBV + aflatoxin = HCC synergy.
- PSC/Caroli/ flukes = CCA.
- Vinyl chloride = hepatic angiosarcoma.

6. Colorectal cancer screening and post-cancer surveillance

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Family history: first-degree relative with CRC < 60.	Colonoscopy at **age 40 or 10 years** before diagnosis of the relative, then **every 5 years**.
After curative CRC resection.	Colonoscopy at **1 year**, then **3 years** later if normal.
Obstructing CRC prevents complete colonoscopy.	Complete colonoscopy **3–6 months** after surgery to detect synchronous lesions.
FIT vs FIT–fecal DNA screening.	FIT–fecal DNA has **higher sensitivity** but **lower specificity** than FIT alone. FIT is **annual**; FIT–DNA is repeated at **longer interval**.

MEMORY PEARLS

- FDR CRC <60 = 40 or 10 years earlier, q5y.
- CRC resection = 1 year, then 3 years.

HIGH-YIELD TAKEAWAY

- Pancreas: late unresectable disease. Familial screen = EUS/MRCP. MCN tail = distal pancreatectomy.
- Hepatobiliary: HBV + aflatoxin = HCC synergy. PSC/Caroli/flukes = CCA. Vinyl chloride = hepatic angiosarcoma.
- Colorectal: FDR CRC <60 = 40 or 10 years earlier, q5y. CRC resection = 1 year, then 3 years.

7. Lynch syndrome and hereditary diffuse gastric cancer

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Amsterdam-type family history.	Refer for genetic counselling/ testing; ideally test an **affected relative first.**
Lynch syndrome surveillance.	• Colonoscopy every **1–2 years.** • Gynecologic surveillance (endometrial/ovarian awareness). • EGD with antral biopsies in selected guidance frames. • Annual urinalysis.
Tumor IHC pattern in suspected Lynch.	Loss of **MSH2/MSH6** with intact MLH1/PMS2 supports Lynch pattern → **short-interval** colonoscopic surveillance.
CDH1 hereditary diffuse gastric cancer.	High lifetime risk of diffuse gastric cancer → prophylactic **total gastrectomy** is recommended.

MEMORY PEARLS

Lynch = **MMR** defect.

Colonoscopy every **1–2 years** + gynecologic awareness + EGD (antral bx) + annual urinalysis.

CDH1 = **stomach out.**

8. Polyposis syndromes: visual and endoscopic traps

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
CHRPE / "bear tracks" retinal pigment epithelium with iron deficiency anemia.	Suggests **FAP** until proven otherwise; perform upper endoscopy and colonoscopy.
MAP (MutYH-associated polyposis).	Recessive *MutYH* polyposis → add upper endoscopy with **side-viewing duodenoscopy** (duodenal adenoma/ cancer risk).
Peutz–Jeghers syndrome.	Mucocutaneous pigmentation, hamartomatous polyps, pancreatic cancer risk. **MRI/MRCP** and **breast MRI** surveillance appear in DDSEP frames.
Cowden syndrome (PTEN hamartoma tumor syndrome).	Esophageal glycogenic acanthosis/ hamartomas; high risks: **breast, thyroid, endometrial, renal, colon.**
Juvenile polyposis syndrome.	Hamartomatous GI polyps; increased GI cancer risk. Association with **SMAD4 and HHT.**
Serrated polyposis syndrome.	Requires colonoscopy surveillance every **6–12 months.**

MEMORY PEARLS

FAP = CHRPE / bear tracks + fundic gland polyps.

MAP = MutYH + duodenoscope.

PJS = **STK11** mutation + pancreas & breast risk.

Cowden = **PTEN** mutation.

HIGH-YIELD TAKEAWAY

- Lynch = MMR defect → colonoscopy q1–2y + gynecologic awareness + EGD (antral bx) + urinalysis.
- CDH1 (HDGC) = prophylactic total gastrectomy.
- FAP = CHRPE/fundic glands → upper + lower endoscopy.
- MAP = duodenal adenoma risk → side-viewing duodenoscopy.
- PJS = STK11 → pancreas & breast surveillance.
- Cowden = PTEN → multiple hamartomas & cancers.
- Serrated polyposis = colonoscopy every 6–12 months.

9. Colon polyp surveillance, malignant polyps and gallbladder polyps

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Three-year colonoscopy interval: advanced adenoma or advanced serrated features.	**3-year colonoscopy** if **ANY** of the following: • Adenoma ≥10 mm • Villous histology • High-grade dysplasia • 3–10 adenomas • Sessile serrated polyp (SSP) ≥10 mm • SSP with dysplasia • Traditional serrated adenoma (TSA)
Malignant polyp with unfavorable histology.	**Surgical resection** is required. Endoscopic surveillance alone is not appropriate.
Gallbladder polyp malignancy risk factors.	**Risk increases with ANY** of the following: • Size >1 cm • Age >50 years • Solitary lesion • Presence of gallstones • Symptoms (e.g., pain, dyspepsia) **Cholecystectomy** is the DDSEP answer when high-risk features are present.
Primary sclerosing cholangitis (PSC) with colitis.	**Colon cancer risk amplifier:** • Colonoscopy NOW at PSC diagnosis. • Then annually.

MEMORY PEARLS

- Advanced colon polyp = **3 years.**
- Malignant polyp with bad histology = **surgery.**
- Gallbladder polyp >1 cm (or high-risk features) = **cholecystectomy.**
- PSC + colitis = colonoscopy now then **annually.**

HIGH-YIELD TAKEAWAY

- Advanced colon polyp (≥10 mm, villous, HGD, 3–10 adenomas, SSP ≥10 mm, SSP dysplasia, TSA) = 3-year colonoscopy.
- Malignant polyp with unfavorable histology = surgical resection.
- Gallbladder polyp >1 cm or high-risk features = cholecystectomy.
- PSC with colitis = colonoscopy now + annually.

10. Gastric NET, GIST and pancreatic paraneoplastic clues

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Autoimmune atrophic gastritis / pernicious anemia with gastric polyp.	**Suggests Type 1 gastric neuroendocrine tumor (NET).** Histology: solid nests of monomorphic endocrine cells.
Gastrointestinal stromal tumor (GIST).	**Origin: muscularis propria (fourth EUS layer).** Metastatic or recurrent disease: treated with **imatinib.**
New glucose intolerance/diabetes, weight loss, and migratory thrombophlebitis (Trousseau syndrome).	**Think pancreatic cancer.** Order CT abdomen/pelvis with pancreatic protocol.
FAMMM syndrome (CDKN2A mutation).	Links melanoma / atypical moles to ↑ risk of **pancreatic cancer.**

MEMORY PEARLS

- AIG + B12/iron deficiency + gastric polyp = **gastric NET (Type 1).**
- GIST 4th layer = **imatinib** if metastatic.
- Trousseau syndrome = **pancreas CT.**
- FAMMM/CDKN2A = melanoma + **pancreatic cancer risk.**

HIGH-YIELD TAKEAWAY

- AIG/pernicious anemia + gastric polyp = Type 1 gastric NET.
- GIST arises from 4th EUS layer (muscularis propria) → metastatic = imatinib.
- Trousseau/migratory thrombophlebitis + new diabetes/weight loss → pancreatic cancer → CT A/P.
- FAMMM (CDKN2A) → melanoma/atypical moles ↑ pancreatic cancer risk.

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	Remote lye ingestion; new reflux symptoms	Upper endoscopy with chromoendoscopy	Caustic alkali history = high esophageal SCC risk.
Q2	US esophageal cancer epidemiology in women	Small intestinal cancer is more common than esophageal cancer in women	Esophageal cancer is strongly male-predominant.
Q3	Barrett long segment + carcinoma in situ; no nodes/metastasis	EMR of nodularity + RFA of Barrett segment	CIS/intramucosal Barrett cancer is endoscopic therapy territory.
Q4	Highest US gastric cancer risk ethnic groups	Asians, Latinos, African Americans	Higher-risk groups often reflect H. pylori/ background incidence.
Q5	Not a gastric cancer risk factor	Selenium exposure	H. pylori, CDH1, Lynch, PJS, Ménétrier are risks.
Q6	Antral ulcers + H. pylori stain positive	Therapy reduces intestinal-type gastric cancer risk	Eradicate H. pylori: ulcer healing + carcinogen removal.
Q7	General US population gastric cancer screening	Not recommended	Screening is targeted, not general US population.
Q8	Gastric lymphoma statement	Often regresses with H. pylori eradication	Gastric MALT is the exam association.
Q9	Small intestinal cancer in US	Adenocarcinoma is most common	Most small bowel adenocarcinomas are duodenal.
Q10	Terminal ileal yellow submucosal nodule + diarrhea	Metastasis to liver	Carcinoid syndrome needs systemic serotonin escape.
Q11	Painless jaundice/weight loss pancreatic cancer risk exception	Female sex	Pancreatic adenocarcinoma risk favors male sex.
Q12	Condition not increasing pancreatic cancer risk	Blue rubber bleb nevus syndrome	BRBNS = venous malformations, not pancreas cancer risk.
Q13	Why pancreatic adenocarcinoma prognosis is poor	Most present unresectable	Late presentation drives mortality.
Q14	Familial pancreatic cancer; FDR diagnosed at 50	Annual EUS or MRCP from age 40	Start 50 or 10 years before youngest case.
Q15	Asymptomatic 3 cm tail pancreatic mucinous cystic neoplasm	Distal pancreatectomy referral	MCN in body/tail is resection, not CEA surveillance.
Q16	Cholangiocarcinoma risk factor	Caroli disease	PSC, Caroli, flukes and Thorotrast are classic.
Q17	Global HCC epidemiology	Chronic HBV prevalence correlates with HCC	HBV drives global HCC burden.
Q18	HCC pathogenesis statement	Aflatoxin B1 and HBV synergize	Synergy is a classic HCC exam fact.
Q19	Vinyl chloride exposure + atypical liver/spleen lesions	Hepatic angiosarcoma from vinyl chloride	Hemangiosarcoma should not cause ascites/LFTs/splenic nodules.
Q20	Who does not merit HCC screening	Severe COPD + decompensated cirrhosis, not transplant candidate	Screen only when detection can alter outcome.
Q21	After CRC resection; 1-year colonoscopy normal	Repeat in 3 years	CRC surveillance: 1 year then 3 years.
Q22	Highest CRC incidence/mortality in US	Alaskan Natives	DDSEP epidemiology fact.
Q23	Young adopted man, microcytic anemia + CHRPE	Upper endoscopy and colonoscopy	CHRPE/beat tracks → evaluate for FAP.
Q24	Mother CRC at 55	Start age 40, then every 5 years	FDR <60: age 40 or 10 years earlier, q5y.
Q25	Family history meets Lynch criteria	Genetic counseling	Test affected relative when possible.
Q26	Likely Lynch; non-colonoscopy screening	Annual transvaginal ultrasound	Gynecologic screening is central in Lynch frames.
Q27	PJS woman; cancer surveillance	Annual breast MRI	PJS increases breast/pancreas/GI/gynecologic cancers.
Q28	Esophageal glycogenic acanthosis + GI hamartomas	Cowden syndrome	PTEN syndrome: hamartomas + thyroid/ breast/endometrial/renal risks.
Q29	Pathogenic CDH1, no symptoms	Refer for total gastrectomy	HDGC is prophylactic gastrectomy territory.
Q30	MAP with cecal mass/adenoma burden	Upper endoscopy with side-viewing duodenoscopy	MAP requires duodenal/ampullary surveillance.
Q31	FDR CRC; patient age 38	Colonoscopy at age 40	FDR CRC screening starts age 40 in this frame.

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q31	FDR CRC; patient age 38	Colonoscopy at age 40	FDR CRC screening starts age 40 in this frame.
Q32	First screening colonoscopy; advanced serrated/adenoma finding	Repeat colonoscopy in 3 years	Advanced adenoma/SSP ≥10 mm/TSA ≥ 3 years.
Q33	Malignant polyp with unfavorable features	Refer for surgical resection	Cancer in polyp: mucosal < 1 and histology = surgery.
Q34	Gastric aden polyp high-risk features/symptoms	Refer for cholecystectomy	1 cm or symptomatic/older/no gallbladder = surgery.
Q35	CRC with MSI/dMMR; meets Lynch family history	Colonoscopy in 1 year	Lynch surveillance is every 1–2 years.
Q36	Obstructing sigmoid cancer presents; full colonoscopy	Complete colonoscopy now after resection window	Need 3–6 months completion colonoscopy for synchronous lesions.
Q37	PJS with lip/buccal freckling and juvenile hamartomas	MRI/MRCP	PJS has extra-GI pancreatic cancer risk.
Q38	Young patient with family history polyposis/screening rule	Perform colonoscopy	Think FAP; evaluate colon.
Q39	Mary juvenile colonic polyps + gastric hamartomatous polyps	Juvenile polyposis syndrome	SMAD4/coffee ring gastric polyps and HHT.
Q40	A(δ)/pernicious anemia + gastric polyp	Add retest of immunostaining endocrine cells	Type 1 gastric NET due to hypergastrinemia.
Q41	Meets WHO serrated polyposis criteria	Colonoscopy q 1–2 months	Serrated polyposis needs short-interval clearance/surveillance.
Q42	FAP/MAP pancreatic cancer gene	CDKN2A	Atypical moles/hamartomas + pancreas = CDKN2A.
Q43	Trousseau syndrome + weight loss + glucose 110	CT abdomen/pelvis	Migratory thrombophlebitis can announce pancreatic cancer.
Q44	UC + new PSC diagnosis	Colonoscopy now	PSC-colitis surveillance begins at PSC diagnosis.
Q45	FUT-DNA vs FIT	Higher sensitivity, lower specificity	More sensitive screening creates more false positives.
Q46	18 mm right-sided sessile serrated lesion: biopsy	Cold snare polypectomy	Serrated pathway = DNA/methylation.
Q47	Gastric adenocarcinoma from H. pylori layers	Metastasis/recurrence if disease treated with irradible	GIST arises from muscularis propria.
Q48	Indication for H. pylori testing	After endoscopic resection for early gastric cancer	Also test/treat in gastric MALT lymphoma.
Q49	Esophageal cancer staging fact	EUS/FNA more sensitive for node staging than CT	EUS for T/N, CT/PET for M.
Q50	Not linked to small bowel adenocarcinoma	Common variable immune deficiency	Lynch, Crohn, celiac and FAP are risks.
Q51	Resected non-invasive pancreatic MCN	No further surveillance	Non-invasive MCN after resection rarely recurs.
Q52	Not an increased cholangiocarcinoma risk	Cholelithiasis	PSC/Caroli/flukes/Lynch are stronger risk frames.

DDSEP 09 - CHAPTER 15

NUTRITION, OBESITY AND EATING DISORDERS

CONCISE MRCP / DDSEP EXAM PEARLS IN CHAPTER 01 ESOPHAGUS STYLE

Chapter 15 | 50 DDSEP questions

Memory line: Read Section I as the clinical note map, then use Section II as a rapid scenario-to-answer recall table. DDSEP logic is preserved; outdated drug facts are flagged briefly.

SECTION I - EXAM PEARL NOTES

1. Short bowel syndrome, high-output stoma and intestinal failure
2. Enteral nutrition, pancreatitis and ICU feeding
3. PEG and vascular access complications
4. Cirrhosis, fistulae, chyle and protein-losing states
5. Micronutrient deficiencies and malnutrition screens
6. Bariatric surgery: deficiencies, complications and procedure effects
7. Refeeding syndrome, eating disorders and SMA syndrome
8. Obesity physiology and first-line management
9. Obesity drugs and current safety trap
10. Diet-specific traps, allergy and acid-base/electrolyte calculations

1. Short bowel syndrome, high-output stoma and intestinal failure

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Adult short bowel length <100 cm is highly predictive of permanent intestinal failure.	**<100 cm** of functional small bowel in adults → **high risk** of permanent intestinal failure → consider long-term parenteral nutrition (PN) or intestinal transplant discussion.
High-output ostomy after Crohn resections with AKI and 4–5 L/day output.	**Short bowel syndrome** until proven otherwise. SIBO can coexist, but **poor rifaximin response** supports SBS as the primary driver.
PN-dependent SBS despite optimized antimotility and acid suppression.	Escalate to **teduglutide** (GLP-2 analogue) therapy.
Ileal Crohn disease or ileal resection with diarrhea.	Consider **bile salt diarrhea.** Cholestyramine is effective. Bile salt malabsorption is classically more prominent after **>100 cm** of ileal resection.

MEMORY PEARLS

SBS: **<100 cm = permanent failure risk.**

High-output ileostomy + AKI = **SBS** until proven otherwise.

PN-dependent SBS → **GLP-2 / teduglutide.**

HIGH-YIELD TAKEAWAY

- SBS in adults: <100 cm of small bowel = high risk of permanent intestinal failure.
- High-output ileostomy (4–5 L/day) with AKI after Crohn resection = SBS until proven otherwise.
- PN-dependent SBS after medical optimization → GLP-2 analogue (teduglutide).
- Bile salt diarrhea after ileal disease/resection → cholestyramine; prominent after >100 cm ileal loss.

2. Enteral nutrition, pancreatitis and ICU feeding

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Acute pancreatitis nutrition strategy.	**Enteral nutrition** is preferred over PN. Prepyloric (nasojejunal/nasogastric) feeding is acceptable and should begin after adequate resuscitation when oral intake is not possible.
Severe pancreatitis with feed intolerance.	Switch to an **elemental formula** before abandoning the enteral route. **TPN only** when enteral feeding is impossible or insufficient.
ICU patient with diarrhea on tube feeds.	**Do not** reflexively stop feeds. Evaluate causes, especially **C. difficile.** Add **soluble/fermentable fiber** supplementation when appropriate.
Overfeeding mechanically ventilated patients.	Excess calories ↑ **CO_2 production** and ventilatory burden → **reduce feeding rate** and avoid overfeeding.

MEMORY PEARLS

Pancreatitis = **enteral first,** not **PN.**

Feed intolerance → **elemental formula** first.

Tube-feed diarrhea → check **C. diff,** do **not** automatically stop feeds.

Overfeeding → ↑ **CO_2 production.**

HIGH-YIELD TAKEAWAY

- Acute pancreatitis: enteral nutrition (preferably postpyloric) after resuscitation; PN only if enteral not possible.
- Feed intolerance in severe pancreatitis → try elemental formula before TPN.
- ICU tube-feed diarrhea → evaluate (especially C. difficile); add soluble/fermentable fiber; do NOT stop feeds automatically.
- Overfeeding mechanically ventilated patients increases CO_2 and ventilatory burden → reduce calories.

3. PEG and vascular access complications

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Pneumoperitoneum within hours after PEG.	Common and **may be observed** if the patient is clinically stable with normal lactate and no signs of peritonitis.
PEG-site hypergranulation tissue without infection or bleeding.	Treat locally with **silver nitrate**, repeated until tissue is level with surrounding skin.
Peripheral PN causing erythema, pain, phlebitis and poor vein visualization.	Suggests **vessel thrombosis**. Peripheral lines should be rotated every **48–72 hours**.
Longer-term PN access in a thrombosis-prone cancer patient.	**Tunneled silicone catheter** has lower infection and thrombosis rates than many alternatives in the DDSEP frame.

MEMORY PEARLS

PEG air + stable patient = **observe**.

PEG granulation tissue = **silver nitrate**.

PN line pain/redness = **thrombosis**.

HIGH-YIELD TAKEAWAY

- PEG pneumoperitoneum within hours → **observe** if stable, normal lactate, no peritonitis.
- PEG-site hypergranulation (no infection/bleeding) → **silver nitrate** until level with skin.
- Peripheral PN with pain/redness & poor vein visualization → suspect **thrombosis**; rotate lines every **48–72 h**.
- For long-term PN in thrombosis-prone cancer patient → **tunneled silicone catheter** preferred.

4. Cirrhosis, fistulae, chyle and protein-losing states

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Stable decompensated cirrhosis with BMI <30.	Target **35–40 kcal/kg/day** and **1.2–1.5 g/kg/day protein**. Avoid outdated protein restriction unless exceptional intolerance.
Late-evening high-protein snack in cirrhosis.	Improves muscle mass by **shortening the fasting/catabolic window**.
High-output enterocutaneous or colocutaneous fistula.	Target **30–35 kcal/kg/day** and protein about **1.5 g/kg/day**.
Chylous ascites/effusion after surgery.	High protein, low fat diet enriched with **medium-chain triglycerides (MCT)**. Diagnose with triglyceride-rich chylous fluid.
Fontan-associated diarrhea, ascites, greasy stool.	Can reflect **protein-losing enteropathy**. Manage with **high-protein/low-fat** nutrition + cardiac/diuretic-directed therapy.

MEMORY PEARLS

Cirrhosis: **feed protein, night snack.**

Fistula: **30–35 kcal + 1.5 g protein.**

Chyle = **MCT.**

HIGH-YIELD TAKEAWAY

- Cirrhosis (stable, BMI <30): 35–40 kcal/kg/day + 1.2–1.5 g/kg/day protein; avoid routine protein restriction.
- Late-evening high-protein snack → improves muscle mass by reducing catabolic fasting.
- High-output fistula → 30–35 kcal/kg/day + ~1.5 g/kg/day protein.
- Chylous ascites/effusion → high protein, low fat, MCT; confirm with triglyceride-rich fluid.
- Fontan-associated diarrhea/ascites/greasy stool → think protein-losing enteropathy; high-protein/low-fat + cardiac care.

5. Micronutrient deficiencies and malnutrition screens

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Strict gluten-free diet in celiac disease.	**Thiamine-poor** diet risk. Nutrient-dense enriched grains, legumes and protein foods are more important than reflex supplement-first practice.
Thiamine (vitamin B1) physiology.	Stored only in small amounts in liver. Becomes deficient **early** in malnutrition or malabsorption. Think **weakness and confusion BEFORE** carbohydrate refeeding.
Vitamin D deficiency risk factors.	Nursing home, dark skin, CKD, poor sun exposure, immobility and fat malabsorption.
Vitamin B12 deficiency diagnosis and risk.	Confirmed by increased **methylmalonic acid (MMA) and homocysteine.** Vegetarians often need B12 supplementation.
Zinc deficiency and post-bypass risk.	May present with **diarrhea,** impaired **wound healing** and **night blindness.** Post–Roux-en-Y patients can develop zinc or other micronutrient deficiencies.

MEMORY PEARLS

Thiamine first: becomes deficient early → think weakness/confusion before carbs.

Vitamin D risk: dark skin, indoor, CKD, immobility, low sun, fat malabsorption.

B12 deficiency = ↑ methylmalonic acid (MMA) + ↑ homocysteine.

Zinc deficiency = diarrhea, poor wound healing, night vision problems.

6. Bariatric surgery: deficiencies, complications and procedure effects

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Roux-en-Y after rapid weight loss with postprandial intermittent abdominal pain.	Think **internal hernia.** Urgent surgical consultation is the exam-safe response.
Essential fatty-acid deficiency after bariatric surgery.	Causes dermatitis / poor intake. Diagnostic **triene:tetraene ratio >0.2.**
Biliopancreatic diversion with duodenal switch (BPD-DS).	Produces the **greatest** excess-weight loss and metabolic improvement among listed procedures, but carries the **highest malabsorptive risk.**
After gastric surgery / resection.	Reduced acid and **intrinsic-factor** exposure → contributes to vitamin **B12** deficiency.

MEMORY PEARLS

RYGB pain after meals = **internal hernia** → call **surgery.**

EFAD (essential fatty-acid deficiency) rash = **triene:tetraene ratio >0.2.**

Duodenal switch (BPD-DS) = **most** weight loss & metabolic benefit.

Post-gastric surgery = ↓ acid & intrinsic factor → **B12** deficiency.

7. Refeeding syndrome, eating disorders and SMA syndrome

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Severe malnutrition / eating disorder (refeeding).	During refeeding, monitor **phosphorus** closely, along with potassium and magnesium.
Bulimia nervosa.	**Normal BMI** with binge eating/purging and **hematemesis** from repeated vomiting is a classic clue.
Anorexia nervosa.	**Low BMI**, electrolyte depletion, delayed gastric emptying / bezoar, early satiety and abdominal distension. Treatment: **nutritional rehabilitation.**
Superior mesenteric artery (SMA) syndrome.	Rapid weight loss and vomiting with obstructive symptoms can cause SMA syndrome. After decompression/ electrolytes, **nasojejunal feeding** provides nutritional therapy past obstruction.

MEMORY PEARLS

Refeeding syndrome = monitor **phosphate.**

Normal BMI + purging hematemesis = **bulimia nervosa.**

Low BMI + bezoar/electrolyte loss + SMA syndrome = **anorexia complications.**

8. Obesity physiology and first-line management

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Ghrelin physiology.	Ghrelin is secreted by gastric fundic oxyntic cells during fasting. After weight loss, **ghrelin rises** while satiety signals (PYY, CCK, leptin and insulin) fall.
Leptin physiology.	Leptin is secreted by adipocytes in proportion to fat mass and falls with fasting. Obesity often involves **leptin resistance** rather than leptin absence.
Clinically meaningful weight loss.	Clinically meaningful obesity benefit begins with **5–10% weight loss.** Lifestyle intervention, food diary and activity log remain first-line before pharmacotherapy.
Exercise target (DDSEP).	Vigorous aerobic exercise **150 min/week** plus muscle-strengthening on **2 days/week** for weight-loss planning.

MEMORY PEARLS

Fasting or weight loss → **ghrelin up.**

Fat mass ↑ → **leptin up.**

Weight-loss target = **5–10%.**

9. Obesity drugs and current safety trap

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
When to consider anti-obesity pharmacotherapy.	Consider for **BMI >30**, or **BMI >27 with comorbidities**, after comprehensive lifestyle intervention fails.
Phentermine/ topiramate safety trap.	**Contraindicated in pregnancy** because of **teratogenic risk**. Pregnancy testing and effective contraception/avoidance are central.
Lorcaserin (Belviq) in DDSEP.	DDSEP listed lorcaserin as a historical best choice in patient with alcohol-related pancreatitis/ seizure history. Lorcaserin/Belviq was **withdrawn from the US market** because **cancer risk** outweighed benefit.
Binge-eating disorder (BED) pharmacotherapy.	**Lisdexamfetamine** is the DDSEP answer. BED = recurrent loss-of-control binge episodes with distress, not compensatory purging.

MEMORY PEARLS

- Pregnancy blocks **phentermine/ topiramate.**
- Lorcaserin = historical best choice in DDSEP but **withdrawn (↑ cancer risk).**
- Binge-eating disorder → **lisdexamfetamine.**

10. Diet-specific traps, allergy and acid-base/electrolyte calculations

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Alpha-gal syndrome.	Delayed (3–6 h) abdominal symptoms, oral tingling, nausea after mammalian meat/fat in patient from the Southeast/Central US. **Lone Star tick** exposure is the clue.
Eosinophilic esophagitis (EoE) dietary therapy.	**Elemental diet** is most effective. Six-food elimination diet is more practical (milk, wheat, egg, soy, peanut/tree nuts, seafood). Rare triggers include nuts and seafood compared with milk/wheat/ egg/soy.
Hyperemesis gravidarum acid–base effect.	Persistent vomiting → loss of HCl → chloride and acid loss → **hypochloremic metabolic alkalosis.**
Corrected sodium in hyperglycemia.	Corrected Na = Measured Na + 1.6 × (glucose − 100) / 100 (Glucose in mg/dL) Example: Na 127, glucose 470 → Add 1.6 × 3.7 ≈ 5.9 → Corrected Na ≈ **133 mEq/L.**
Best validated malnutrition screen (DDSEP list).	Patient-reported **unintentional weight loss.**

MEMORY PEARLS

- Alpha-gal = Lone Star tick + delayed meat reaction.
- EoE most effective diet = elemental.
- Vomiting → loss of Cl^- & H^+ → hypochloremic metabolic alkalosis.
- Corrected Na = Na + 1.6/100 (glucose above 100 mg/dL).
- Best malnutrition screen = patient-reported unintentional weight loss.

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	Adult SBS; length predicting permanent intestinal failure	**100 cm**	<100 cm small bowel = permanent intestinal failure risk.
Q2	Acute pancreatitis unable to eat after resuscitation	**Enteral feeding prepylorically**	Enteral nutrition beats PN; prepyloric is acceptable.
Q3	Strict gluten-free diet in celiac disease	**Thiamine**	Gluten-free diet may be thiamine-poor.
Q4	Stable decompensated cirrhosis, BMI 28	**35-40 kcal/kg/day + 1.2-1.5 g/kg/day protein**	Do not protein-restrict stable cirrhosis.
Q5	Cirrhosis nutritional timing strategy	**Evening snack before bedtime**	Night snack shortens catabolic fasting.
Q6	12 h post-PEG pneumoperitoneum, stable labs/no lactate	**Monitor clinical status**	PEG air is common if no peritonitis.
Q7	PEG hypergranulation, no infection/bleeding	**Apply silver nitrate**	Granulation tissue = local cautery.
Q8	Peripheral PN IV site pain, erythema, vein not visualized	**Vessel thrombosis**	Peripheral PN lines can thrombose/phlebitis.
Q9	Malnutrition/weakness; first deficiency	**Thiamine**	Thiamine stores are small; give before carbohydrate.
Q10	Nursing-home dark-skinned man, CKD, immobile, spasms	**Vitamin D deficiency**	Low sun + CKD + age = vitamin D risk.
Q11	Stable ICU patient on standard enteral formula	**Fermentable fiber**	Routine useful additive; immunonutrition not routine.
Q12	Hyperemesis gravidarum	**Hypochloremic metabolic alkalosis**	Vomiting loses HCl.
Q13	Post-proctocolectomy/IPAA rehabilitation plan	**Probiotics + ORS + high starch/low simple sugars**	Rehabilitate pouch/intestinal function.
Q14	Micronutrient excess teratogenic in pregnancy	**Vitamin A**	High-dose vitamin A/isotretinoin is teratogenic.
Q15	Na 127, glucose 470	**133 mEq/L**	Corrected Na adds ~1.6 per 100 glucose above 100.
Q16	Ileal Crohn with persistent loose stools	**Cholestyramine**	Bile acid diarrhea responds to sequestration.
Q17	When to consider nutrition support if oral intake inadequate	**Seven to 14 days**	Enteral or parenteral support after sustained inadequate intake.
Q18	Post-hepatectomy chylous ascites	**Medium-chain triglycerides**	Chyle = high TG; use high-protein low-fat MCT diet.
Q19	BMI 17, severe electrolyte abnormalities/ refeeding risk	**Phosphorous**	Refeeding kills via phosphate shift.
Q20	Fontan patient, diarrhea/greasy stool, ascites	**Protein-losing enteropathy**	Fontan + edema/diarrhea = protein loss.
Q21	Hematemesis, normal BMI, purging behavior clue	**Bulimia nervosa**	Normal BMI does not exclude severe purging.
Q22	Fasting fundic hormone stimulating hunger	**Ghrelin**	Fundus/oxyntic cells secrete ghrelin in fasting.
Q23	Weight loss magnitude reducing obesity complications	**Five to 10 percent**	Clinically meaningful target.
Q24	Tennessee, heavy mammalian meat, tingling/nausea	**Lone star tick**	Alpha-gal allergy clue.
Q25	Validated malnutrition-risk screen	**Unintentional weight loss**	Most validated screening indicator among choices.
Q26	Post-colorectal surgery chylous fluid	**Low-fat diet + MCT oil**	Conservative chyle therapy: high protein, low fat, MCT.
Q27	Ventilated COPD patient overfed enteral formula	**Decrease feeding rate**	Overfeeding increases CO_2 load.
Q28	Rapid weight loss/vomiting, SMA syndrome	**Nasojejunal feeding tube**	Feed beyond obstruction after decompression.
Q29	Crohn resections, 4-5 L ostomy output, AKI	**Short bowel syndrome**	High-output stoma after resections = SBS.
Q30	UC flare with severe malnutrition and sodium losses	**Increased volume, 75% normal saline, low dextrose**	PN formula must replace sodium losses; avoid high dextrose.
Q31	PN catheter replacement in cancer + DVT risk	**Tunneled silicone catheter**	Lower infection/thrombosis rate in DDSEP frame.

DDSEP SCENARIO TABLE (CONTINUED)

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q31	PN catheter replacement in cancer + DVT risk	**Tunneled silicone catheter**	Lower infection/thrombosis rates in DDSEP frame.
Q32	Severe pancreatitis feed intolerance on standard formula	**Switch to elemental formula**	Try elemental enteral feed before TPN.
Q33	Long cholestyramine use + fracture	**Obtain fat-soluble vitamin levels**	Bile acid binders impair fat-soluble vitamins.
Q34	Macrocytosis, low B12, paresthesias	**High MMA and homocysteine**	Confirms B12 deficiency.
Q35	PN-dependent SBS despite loperamide/PPI	**Start GLP analogue**	Teduglutide improves absorption and reduces PN need.
Q36	High-output colocutaneous fistula nutrition	**30-35 kcal/kg/day + protein 1.5 g/kg/day**	Fistula increases energy/protein needs.
Q37	Post-RYGB rash, poor intake, triene:tetraene clue	**Essential fatty acids**	EFAD dermatitis after inadequate fat intake.
Q38	Low BMI, electrolytes low, bezoar/delayed emptying	**Anorexia nervosa**	Nutritional rehabilitation is primary treatment.
Q39	ICU tube-feed diarrhea after esophagectomy	**Check stool for C. difficile**	Do not reflexively stop enteral feeds.
Q40	Hunger after weight loss	**Ghrelin**	Weight loss raises ghrelin and lowers satiety hormones.
Q41	Phentermine/topiramate contraindication	**Pregnancy**	Teratogenic risk is the trap.
Q42	Post-RYGB deficiency syndrome	**Zinc deficiency**	Bariatric patients need micronutrient surveillance.
Q43	EoE diet with highest efficacy	**Elemental diet**	Most effective but least practical.
Q44	Obesity with diabetes risk, no lifestyle trial	**Moderate-fat low-calorie diet + food/activity logs**	Lifestyle first before drugs if not attempted.
Q45	Binge-eating disorder therapy	**Lisdexamfetamine**	Binge episodes + distress, no purging requirement.
Q46	Vegetarian diet supplementation need	**Vitamin B12**	Animal products supply B12.
Q47	Alcoholism/pancreatic insufficiency + diarrhea/night blindness	**Zinc**	Zinc deficiency: diarrhea, wounds, night blindness/URIs.
Q48	BMI 50, diabetes; most weight loss procedure	**Biliopancreatic diversion with duodenal switch**	Greatest excess-weight loss, high malabsorption.
Q49	Leptin mechanism	**Secreted in proportion to fat mass**	Fasting lowers leptin; obesity has resistance.
Q50	Post-RYGB intermittent postprandial abdominal pain	**Internal hernia; immediate surgical consult**	Intermittent imaging-negative pain can be internal hernia.
Q51	Historical DDSEP obesity drug in alcohol/pancreatitis/seizure case	**Lorcaserin (Belviq) - historical; withdrawn**	DDSEP answer is outdated: lorcaserin withdrawn for cancer-risk signal.
Q52	Exercise program for weight loss	**Vigorous exercise 150 min/week + 2 days strengthening**	Aerobic plus resistance training.

DDSEP 09 - CHAPTER 16

ISSUES IN PEDIATRIC GASTROENTEROLOGY

CONCISE MRCP / DDSEP EXAM PEARLS IN CHAPTER 01 ESOPHAGUS STYLE

Chapter 16 | 50 DDSEP questions

Memory line: Read Section I as the pediatric clinical-note map, then use Section II as a rapid scenario-to-answer recall table. DDSEP answers are preserved, including the duplicated Q2 numbering in the source as Q2A/Q2B for clarity.

SECTION I - EXAM PEARL NOTES

1. Pediatric diarrhea: celiac risk, Giardia, lactose and SIBO
2. Pediatric IBD: Crohn phenotype, acute severe colitis and PSC link
3. Infant vomiting, reflux, food allergy and obstruction
4. Pediatric bleeding and urgent surgical/endoscopic traps
5. Functional and motility-type pediatric disorders
6. Constipation, continence and anorectal clues
7. Pediatric hepatology: cholestasis, biliary atresia, PALF and autoimmune disease
8. Pediatric metabolic and genetic liver/pancreas disorders
9. Cystic fibrosis, EoE, *H. pylori* and *C. difficile*
10. Pediatric nutrition, FAP surveillance and fat-soluble vitamins

1. Pediatric diarrhea: celiac risk, Giardia, lactose and SIBO

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Celiac disease risk associations in children.	Associated with: **type 1 diabetes, Turner syndrome, Williams syndrome, selective IgA deficiency, Sjogren disease,** and **autoimmune thyroiditis.**
Abrupt watery non-bloody diarrhea, flatulence and anorexia after freshwater exposure.	**Giardia lamblia** infection. Treat with weight-adjusted **metronidazole** in the DDSEP frame.
Dairy-linked gas, bloating, abdominal discomfort and diarrhea.	**Lactose intolerance.** Diagnostic test: **lactose hydrogen breath test.**
Post-ileocecal resection Crohn patient with bloating/diarrhea, normal markers/stool/ radiology and inactive disease.	Small intestinal bacterial overgrowth **(SIBO)** is likely. Consider breath testing and treat accordingly.

MEMORY PEARLS

Watery lake diarrhea = **Giardia.**

Dairy-linked bloating = **lactose breath test.**

Post-resection bloating with inactive Crohn = **SIBO.**

2. Pediatric IBD: Crohn phenotype, acute severe colitis and PSC link

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Moderate–severe stricturing ileocolonic Crohn with perianal fistula/phlegmon.	Warrants **early biologic therapy.** Mesalamine has no meaningful benefit in Crohn disease for this phenotype.
Acute severe ulcerative colitis (ASUC) in adolescents.	Follow adult logic: exclude infection, start **IV corticosteroids.** If poor response (3–5 days), consider **flexible sigmoidoscopy** to assess CMV and severity.
Primary sclerosing cholangitis (PSC) in a child/adolescent.	Strongly associated with IBD. **Ileocolonoscopy** is required even with mild diarrhea or minimal bowel symptoms.
After ileal Crohn resection, megaloblastic anemia.	Consider **vitamin B12 insufficiency,** especially with >20 cm ileal loss.

MEMORY PEARLS

Complex Crohn + fistula/ phlegmon = **biologic early.**

ASUC + steroid non-response = **flex sig / CMV.**

PSC in child = **scope for IBD,** even with mild symptoms.

3. Infant vomiting, reflux, food allergy and obstruction

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
 Six-week-old with non-bilious vomiting and palpable olive/mass.	**Infantile hypertrophic pyloric stenosis (IHPS).** Classic "olive" in RUQ/epigastrium.
 Healthy six-week-old with hematochezia and no systemic illness.	**Cow milk/soy protein intolerance.** Eliminate cow and soy proteins from breastfeeding mother's diet.
 Young infant with delayed vomiting, diarrhea and dehydration after food exposure.	**Food Protein–Induced Enterocolitis Syndrome (FPIES).** Non-IgE mediated food allergy. Common triggers: cow milk, soy, rice, oats, fish.
 Three-month-old thriving infant with recurrent spitting and guaiac-negative stool.	**Physiologic gastroesophageal reflux (GER).** Benign if thriving and no red flags (poor weight gain, bilious vomiting, blood in stool, apnea, etc.).
 Bilious emesis in a neonate or two-week-old.	**Malrotation with midgut volvulus until proven otherwise.** Surgical emergency→urgent evaluation and imaging.

MEMORY PEARLS


Non-bilious vomiting + palpable olive = **pyloric stenosis.**


Benign infant blood = **cow milk/soy protein intolerance.**


Thriving infant + spitting + guaiac negative = **physiologic GER.**


Bilious emesis in neonate = **malrotation/volvulus.**

4. Pediatric bleeding and urgent surgical/endoscopic traps

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
 Painless massive rectal bleeding at age two (toddler).	**Meckel diverticulum.** Technetium-99m pertechnetate scan ("Meckel scan") detects ectopic gastric mucosa.
 Child age 6–36 months with episodic abdominal pain, legs drawn up, and RUQ mass.	**Intussusception.** Stable patients → treat with air contrast enema (radiologic reduction).
 Fever, leukocytosis/CRP and periumbilical pain migrating to RLQ.	**Acute appendicitis.** Most common surgical emergency in children.
 Ingestion of esophageal button battery.	**Emergent endoscopic removal.** High risk of caustic injury, fistula and perforation.
 Ingestion of multiple or rare-earth magnets with abdominal pain or distension.	**Urgent surgical evaluation.** Risk of pressure necrosis, perforation, fistula—not managed endoscopically.
 Caustic ingestion (alkali > acid) in toddlers or young children.	**Risk of later esophageal stricture.** Symptomatic or severe ingestion → endoscopy within 24 hours.

MEMORY PEARLS


Painless toddler rectal bleeding = **Meckel diverticulum.**


Episodic pain, leg drawing, RUQ mass = **intussusception.**


Periumbilical → RLQ pain + fever = **acute appendicitis.**


Button battery = **emergent endoscopic removal.**


Magnets + pain/distension = **urgent surgery.**


Caustic ingestion = endoscopy within **24 hours** if symptomatic/severe.

5. Functional and motility-type pediatric disorders

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Functional chronic abdominal pain with anxiety/depression and no alarms.	**After appropriate workup and exclusion of red flags, treat with neuromodulators.** Obtain **EKG** before starting amitriptyline or nortriptyline.
Functional dyspepsia.	**Chronic epigastric burning/pain not related to defecation.** Bowel pattern is normal.
Cyclic vomiting syndrome (CVS).	**Recurrent stereotyped vomiting** episodes with symptom-free intervals. DDSEP option was **all listed** treatments.
Rumination syndrome.	**Painless regurgitation without retching.** Often in developmental disorders or adolescents. pH/impedance can be **normal during sleep.**
Diabetic gastroparesis in adolescents.	**Long-term treatment: improved glycemic control.** Prokinetics may be used as adjuncts.

MEMORY PEARLS

- Functional pain → exclude alarms, **EKG before TCA.**
- Functional dyspepsia = epigastric pain **not linked to defecation.**
- Cyclic vomiting syndrome = recurrent episodes with symptom-free intervals.
- Rumination syndrome = regurgitation **not during sleep.**
- Diabetic gastroparesis = improved **glycemic control.**

6. Constipation, continence and anorectal clues

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
Functional constipation with fecal incontinence and large rectal stool burden.	**Bowel cleanout is required first.** Then start maintenance therapy (laxatives, behavioral, dietary).
Hirschsprung disease.	**Abdominal distension**, poor feeding. Cause: failed neural crest migration. Diagnosis: **absent ganglion cells** on rectal biopsy.
Fecal incontinence with no rectal stool burden and absent or difficult anal wink.	**Concern for tethered cord** (spinal cord abnormality). **Lumbar spine MRI** is the next diagnostic step.
Childhood rectal prolapse.	**Most commonly due to constipation.** Other causes: diarrhea, cystic fibrosis, polyps.
Constipation workup key principle.	**Most children have functional constipation.** Treat **constipation** aggressively **before** extensive workup.

MEMORY PEARLS

- Stool-filled rectum = **cleanout first.**
- Hirschsprung = **no ganglion cells** on rectal biopsy.
- Empty rectum + absent anal wink = **spine MRI.**
- Rectal prolapse = usually **constipation**; others: diarrhea, CF, polyps.
- Most pediatric constipation = functional. **Treat first.**

7. Pediatric hepatology: cholestasis, biliary atresia, PALF and autoimmune disease

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
 **Alagille syndrome:** cholestatic jaundice, triangular facies, heart murmur and butterfly vertebrae.	**Alagille syndrome.** Due to JAG1 mutation. Cholestasis, cardiac defects, posterior embryotoxon common.
 **Biliary atresia:** direct hyperbilirubinemia with pale stool and high GGT.	**Biliary atresia.** Diagnosis confirmed by intraoperative cholangiogram. Early Kasai portoenterostomy (best < 60 days) improves bile flow and native liver survival.
 Post-Kasai infant with fever/irritability and cholestatic LFT rise.	**Ascending cholangitis.** Most common post-Kasai complication. Treat promptly with antibiotics and supportive care.
 Pediatric acute liver failure (PALF).	**ICU care with close monitoring. Check mental status frequently and monitor ammonia levels.** Definition: vitamin K–unresponsive **coagulopathy** (INR ≥ 1.5) with evidence of liver disease ± encephalopathy in a patient without pre-existing chronic liver disease.
 Autoimmune hepatitis in adolescents.	**Autoimmune hepatitis.** Elevated ANA, ASMA and IgG. Liver biopsy: interface hepatitis with predominant lymphocytes and plasma cells. Treat with corticosteroids ± azathioprine.

MEMORY PEARLS


Pale stool/ direct bilirubin = **biliary atresia.**


Post-Kasai fever/irritability = **ascending cholangitis.**


PALF = monitor **mental status and ammonia.**


Autoimmune hepatitis = **ANA/ ASMA/IgG high + interface hepatitis.**

8. Pediatric metabolic and genetic liver/pancreas disorders

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
 Wilson disease.	**Wilson disease (ATP7B defect).** Chronic aminotransferase elevation + low ceruloplasmin. Confirm with 24-h urine copper ↑, hepatic copper ↑ or Kayser–Fleischer rings.
 Galactosemia (newborn).	**Galactosemia.** Neonatal hypoglycemia, hypotonia/seizures, unconjugated jaundice, positive reducing substances in urine and *E. coli* sepsis. Management: switch to soy-based formula (lactose/galactose free).
 Pediatric NAFLD/NASH in obese Hispanic male.	**Nonalcoholic steatohepatitis (NASH).** Macrosteatosis with ballooning degeneration of hepatocytes ± lobular inflammation (fibrosis can progress).
 Recurrent childhood pancreatitis with family history (± pancreatic cancer).	**Can be due to multiple genetic/anatomic causes.** DDSEP answer: all listed defects (e.g., PRSS1, SPINK1, CFTR, CTRC, CASR, and anatomic variants) → evaluate accordingly.

MEMORY PEARLS


Low ceruloplasmin = ATP7B defect (Wilson disease).


Reducing substances + *E. coli* sepsis = galactosemia → soy formula.


Obese Hispanic + ↑ ALT = NASH (macrosteatosis with ballooning).


Recurrent pancreatitis + family history = evaluate multiple genetic/anatomic causes.

9. Cystic fibrosis, EoE, H. pylori and C. difficile

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
 Cystic fibrosis (CF) GI presentation classically includes meconium ileus.	**Meconium ileus.** Caused by thick, viscid intestinal secretions in neonates with CF.
 EoE in a child with atopy/feeding intolerance.	**Diagnose with upper endoscopy and esophageal biopsies.** Threshold in DDSEP: >15 eosinophils per high-power field (eos/hpf) while on PPI therapy.
 Pediatric H. pylori evaluation.	**Diagnose by endoscopy with biopsy, immunohistochemistry or culture when evaluation is indicated.** H. pylori is a common cause of pediatric gastritis.
 C. difficile testing in infants.	**Do NOT test infants under one year for C. difficile.** Asymptomatic/non-pathogenic colonization is common in this age group.

MEMORY PEARLS

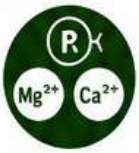

CF = meconium ileus.


EoE = endoscopy/ biopsy.


H. pylori pediatric diagnosis = biopsy-based.


C. diff <1 yr = do not test.

10. Pediatric nutrition, FAP surveillance and fat-soluble vitamins

Key Point / Clinical Clue	DDSEP Answer / Exam Pearl
 Child with APC/FAP mutation risk.	**Hepatoblastoma surveillance.** Abdominal ultrasound and AFP every 6 months until age 5.
 Refeeding syndrome in anorexia/malnutrition.	**Lab abnormalities:** Hypophosphatemia, **hypokalemia**, hypomagnesemia, hypocalcemia. DDSEP answer emphasized: **hypokalemia.**
 Persistent jaundice at 3 weeks of age.	**Obtain fractionated (direct and total) bilirubin.** Do this even if stool is yellow and physiologic jaundice is suspected.
 Cholestatic disorders (e.g., biliary atresia after Kasai).	**Predispose to fat-soluble vitamin deficiency (A, D, E, K).** Vitamin A deficiency can cause ocular (xerophthalmia) and skin changes.
 Exclusively breastfed toddler with rickets pattern and ↑ ALP.	**Vitamin D deficiency most likely.** Check: calcium, phosphorus, PTH and 25(OH) vitamin D.

MEMORY PEARLS


FAP child → AFP/US every 6 months until 5.

Refeeding → think electrolytes; DDSEP answer: **hypokalemia.**


Persistent jaundice >3 weeks → fractionate bilirubin.

Cholestasis → fat-soluble vitamins A, D, E, K.

Rickets pattern + ↑ ALP → **vitamin D deficiency.**

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	Celiac disease risk association	**Type 1 diabetes**	Also remember Turner, Williams, selective IgA deficiency, Sjogren and thyroiditis.
Q2A	Watery non-bloody diarrhea, flatulence, freshwater exposure	**Giardiasis**	Lake/camp exposure + flatulence = Giardia; treat weight-adjusted metronidazole in DDSEP.
Q2B	Teen with stricturing ileocolonic and perianal fistulizing Crohn + phlegmon	**Biologic therapy**	Complex Crohn phenotype needs early biologic therapy; mesalamine is ineffective.
Q3	Adolescent acute severe UC not responding to IV steroids	**Flexible sigmoidoscopy to rule CMV**	ASUC: exclude infection, steroids, then assess CMV/severity if no response.
Q4	Six-week infant, non-bilious vomiting, palpable abdominal mass	**Pyloric stenosis**	2-8 weeks + non-bilious projectile vomiting + olive.
Q5	Healthy six-week infant with hematochezia	**Maternal cow milk/soy elimination and reassurance**	Milk/soy protein intolerance; infant otherwise well.
Q6	Pediatric H. pylori testing when evaluation is indicated	**Upper endoscopy with biopsy/IHC or culture**	Pediatric diagnosis is biopsy-based, not casual serology.
Q7	C. difficile statement in infants	**Do not test infants <1 year**	High colonization makes testing misleading.
Q8	Functional chronic abdominal pain with anxiety/depression	**EKG then consider TCA**	Check QT before amitriptyline/nortriptyline.
Q9	Common pediatric gastritis cause	**Helicobacter pylori infection**	Also think NSAIDs/steroids/immunosuppression, but H. pylori is classic.
Q10	HSP with severe abdominal pain despite NSAIDs	**Corticosteroids**	Palpable purpura + arthralgia + abdominal pain.
Q11	Two-year-old with abrupt painless massive rectal bleeding	**Meckel diverticulum**	Distal ileum ectopic gastric mucosa; Tc-99m scan.
Q12	Child of parent with FAP/APC mutation	**Abdominal ultrasound + AFP q6mo until age 5**	Screen hepatoblastoma early; endoscopic surveillance later.
Q13	Refeeding syndrome electrolyte abnormality in anorexia	**Hypokalemia**	Also monitor phosphate and magnesium closely.
Q14	Adult transitioning after type C TEF repair	**GERD and future Barrett risk**	EA/TEF survivors need reflux/stricture/ Barrett awareness.
Q15	Dairy-linked bloating/diarrhea in teenager	**Lactose breath hydrogen testing**	Lactase non-persistence is common worldwide.
Q16	Post-ileocecal Crohn resection, bloating/diarrhea, inactive disease	**Small bowel bacterial overgrowth**	Normal markers/studies make Crohn recurrence less likely.
Q17	Extreme premature infant, formula, distention, hematochezia	**Necrotizing enterocolitis**	Prematurity + feeding intolerance + bloody stool.
Q18	Initial acetaminophen toxicity management	**Electrolyte, acetaminophen/salicylate levels, blood/urine tox**	Also follow liver enzymes and coagulation.
Q19	Infant cholestasis + triangular facies + murmur + butterfly vertebrae	**Alagille syndrome**	Syndromic cholestasis clue.
Q20	Biliary atresia after Kasai with fever and cholestatic labs	**Cholangitis**	Most common post-hepatoportoenterostomy complication.
Q21	Teen AIH histology	**Portal lymphoplasmacytic infiltrate crossing limiting plate**	Interface hepatitis + ANA/ASMA/high IgG.
Q22	Adolescent PSC labs with mild diarrhea	**Ileocolonoscopy for PSC-IBD association**	PSC frequently coexists with IBD even if symptoms are mild.
Q23	Pediatric acute liver failure management consideration	**Monitor mental status and ammonia**	PALF needs ICU-level monitoring for encephalopathy.

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q24	Common GI manifestation of cystic fibrosis	**Meconium ileus**	Thick secretions obstruct terminal ileum.
Q25	Ileal Crohn resection + megaloblastic anemia	**Vitamin B12 insufficiency**	Terminal ileum absorbs B12.
Q26	Functional constipation with fecal incontinence and stool-filled rectum	**Bowel cleanout**	Disimpact first, then maintenance regimen.
Q27	Infant abdominal distension/poor appetite; expected biopsy	**Absent ganglion cells on rectal biopsy**	Hirschsprung = aganglionosis.
Q28	Atopic child with chronic feeding intolerance	**Upper intestinal endoscopy**	EoE diagnosis requires esophageal biopsies.
Q29	Infant vomiting/diarrhea/dehydration after food exposure	**FPIES**	Non-IgE food allergy: cow milk/soy/rice/oats/fish.
Q30	Two-week-old with recurrent bilious vomiting	**Intestinal malrotation**	Volvulus risk: urgent evaluation.
Q31	Recurrent stereotyped vomiting over one year	**All of the above**	Cyclic vomiting syndrome treatment options can include listed therapies.
Q32	Fecal incontinence, no stool burden, difficult anal wink	**Lumbar spine MRI**	Tethered cord clue.
Q33	Toddler swallowed esophageal button battery	**Emergent endoscopic removal**	Button battery in esophagus is a true emergency.
Q34	Toddler caustic ingestion, long-term complication	**Esophageal stricture**	Assess symptomatic/severe ingestion early.
Q35	Multiple magnets with abdominal pain/distension	**Urgent surgical evaluation**	Bowel-wall entrapment → ischemia, perforation, fistula.
Q36	Thriving 3-month-old with recurrent spitting, guaiac negative	**Physiologic gastroesophageal reflux**	Common infant GER unless red flags.
Q37	Teen chronic epigastric burning, normal stooling	**Functional dyspepsia**	Epigastric pain/burning not linked to defecation.
Q38	Type 1 diabetes, abdominal discomfort/ weight loss, A1c 12.4	**Improved glycemic control**	Diabetic gastroparesis: fix glycemia long term.
Q39	18-month-old episodic severe pain, legs drawn up, RUQ mass	**Air contrast enema**	Intussusception stable treatment: pneumatic/hydrostatic enema.
Q40	Fever, leukocytosis/CRP, acute periumbilical pain	**Acute appendicitis**	Classic pediatric surgical abdomen.
Q41	Cerebral palsy teen with painless regurgitation	**Normal pH/impedance during sleep**	Rumination is not active during sleep and lacks retching.
Q42	Isolated rectal prolapse with dilated stool-filled rectum	**Chronic constipation**	Most common pediatric prolapse driver.
Q43	Low ceruloplasmin, chronic aminotransferase elevation	**ATP7B**	Wilson disease gene.
Q44	Neonate hypoglycemia, reducing substances, E. coli sepsis	**Change to soy-based formula**	Galactosemia: remove lactose/galactose.
Q45	20-day jaundice, pale stool, direct hyperbilirubinemia, high GGT	**Intraoperative cholangiogram**	Biliary atresia confirmation; early Kasai matters.
Q46	Obese Hispanic child, ALT elevation, echogenic liver	**Ballooning degeneration with macrosteatosis**	Pediatric NASH histology.
Q47	Three-week persistent jaundice, yellow stool	**Fractionated bilirubin**	Always check direct bilirubin in persistent neonatal jaundice.
Q48	Recurrent childhood pancreatitis + family history	**All of the above**	Consider hereditary genes plus anatomic/CF/galactone/autoimmune causes.
Q49	Biliary atresia post-Kasai with fat-soluble vitamin issue	**Vitamin A**	Cholestasis causes A/D/E/K deficiency.
Q50	Exclusively breastfed toddler, rickets signs, high ALP	**Vitamin D**	Open fontanelle, rachitic rosary and bowed legs.

DDSEP 09 - CHAPTER 17

SMALL BOWEL DISEASE

CONCISE MRCP / DDSEP EXAM PEARLS IN CHAPTER 01 ESOPHAGUS STYLE

Chapter 17 | 50 DDSEP questions

Memory line: Read Section I as the small-bowel clinical-note map, then use Section II as rapid scenario-to-answer recall. DDSEP answer logic is preserved, with concise exam traps and memory lines.

SECTION I - EXAM PEARL NOTES

1. Celiac disease: who to test, how to test, and when serology misleads
2. Villous atrophy is not always celiac
3. SIBO: risk factors, testing and the folate trap
4. Protein-losing enteropathy, lymphangiectasia and amyloidosis
5. Short bowel syndrome, GLP-2 and transplant indications
6. Acute mesenteric ischemia and vascular small-bowel mimics
7. Immune/allergic small bowel disease and Behcet
8. Small bowel bleeding and tumors
9. Tropical sprue and travel-related malabsorption

1. CELIAC DISEASE:
WHO TO TEST, HOW TO TEST, AND WHEN SEROLOGY MISLEADS

KEY POINT	DETAILS / CLINICAL CLUES	DDSEP ANSWER / EXAM PEARL
1 Who to test and how to test	• Test with **tTG-IgA** in patients on a gluten-containing diet. • Test if: unexplained diarrhea/bloating, iron deficiency, first-degree relative, or unexplained aminotransferase elevation.	**tTG-IgA** is the first-line test while the patient is eating gluten.
2 IgA deficiency	• If IgA deficiency is present, use **IgG**-based testing: – **IgG tTG** and/or deamidated gliadin peptide **(DGP)** antibodies.	**Do NOT** rely on **IgA tTG.** Use **IgG** tests instead.
3 Role of HLA-DQ2/DQ8	• HLA-DQ2/DQ8 is best used to **rule out** celiac disease. • Especially when the patient has already been on a gluten-free diet for >2 months before testing.	Negative HLA-DQ2/DQ8 virtually rules out celiac disease.
4 Persistent symptoms or elevated tTG after diagnosis	• Most commonly due to **inadvertent gluten exposure.** • First step: detailed gluten-free diet review with an expert dietician.	Non-response = gluten exposure until proven otherwise.
5 Long-term considerations	• Well-controlled celiac disease still carries hyposplenism/infection risk. • DDSEP answer includes: **pneumococcal vaccination.**	Think infection risk → **Pneumococcal vaccination.**

MEMORY PEARLS

Celiac testing = tTG-IgA while eating gluten.

IgA deficient = IgG tests.

Non-response = gluten exposure until proven otherwise.

Well-controlled celiac disease → infection risk → pneumococcal vaccination.

2. VILLOUS ATROPHY IS NOT ALWAYS CELIAC

CONDITION	KEY CLINICAL / PATHOLOGIC CLUES	DDSEP ANSWER / TREATMENT LOGIC
Giardiasis	• Can cause duodenal villous blunting. • Watery diarrhea, bloating, travel/ exposure to contaminated water.	• Treat with metronidazole or tinidazole. • Villous atrophy is a **pattern**, not a diagnosis.
Whipple disease	• Middle-aged man. • Diarrhea, weight loss, arthralgias/neurologic clues. • PAS-positive macrophages in duodenal lamina propria.	• Treat: **ceftriaxone IV** followed by **TMP-SMX.** • Confirm with PCR or PAS stain on biopsy.
Autoimmune enteropathy (AIE)	• Severe malabsorptive/secretory diarrhea. • Villous blunting, absent goblet/Paneth cells, crypt apoptosis. • Anti-enterocyte antibodies present.	• **Steroids** are the DDSEP treatment logic.
CVID enteropathy	• Mimics celiac disease. • Recurrent infections. • Villous atrophy with absent plasma cells in lamina propria.	• **Budesonide or prednisone** can treat GI disease. • **IVIG** does not reliably improve enteropathy.
Drug-induced enteropathy	• Olmesartan and NSAIDs can cause enteropathy/villous injury. • Symptoms: diarrhea, weight loss.	• **Stop the offending drug.** • Do **NOT** start gluten-free diet or biologics.

MEMORY PEARLS

Villous blunting differential:

- ✓ Giardia
- ✓ Whipple disease
- ✓ Autoimmune enteropathy
- ✓ CVID enteropathy
- ✓ Drugs (olmesartan, NSAIDs)

Don't label all villous atrophy as celiac.

3. SIBO: RISK FACTORS, TESTING AND THE FOLATE TRAP

KEY POINT	DETAILS / CLINICAL CLUES	DDSEP ANSWER / EXAM PEARL
1 Risk factors	• **Altered anatomy:** Roux-en-Y, strictures, blind loops, diverticula. • **Dysmotility:** diabetes, scleroderma. • Atrophic gastritis. • Celiac disease. • Immunodeficiency. • Prior radiation.	SIBO occurs when normal defenses are overcome by **anatomic** or **motility** abnormalities.
2 Testing	• Lactulose or glucose hydrogen breath testing is the DDSEP diagnostic pathway. • Accuracy is variable. • Results affected by: antibiotics, laxatives, probiotics, promotility drugs, and acid suppression.	No single test is perfect. Interpret in clinical context and after **withholding interfering agents** when possible.
3 Clinical & lab clues	• Causes bloating, flatulence, and diarrhea. • Labs may show vitamin B_{12} deficiency with normal or high **folate**. • **Folate deficiency** is the exam-negative answer.	**B_{12} low, folate normal/high** (Do NOT choose folate deficiency).
4 Recurrent SIBO management	• Recurrent SIBO from a fixed anatomic lesion (e.g., radiation-induced ileal stricture). • Manage by correcting or resecting the abnormal anatomy when feasible.	Treat the cause: **Fix or remove the anatomic problem** when possible.

MEMORY PEARLS

SIBO = bloating + diarrhea + risk anatomy/motility.

B_{12} low, folate not low. (Folate deficiency is incorrect.)

Recurrent anatomy-driven SIBO = fix anatomy.

4. PROTEIN-LOSING ENTEROPATHY, LYMPHANGIECTASIA AND AMYLOIDOSIS

KEY POINT	DETAILS / CLINICAL CLUES	DDSEP ANSWER / EXAM PEARL
1 Protein-losing enteropathy (PLE)	• Presents with diarrhea, edema/ascites, hypoalbuminemia. • Increased stool alpha-1-antitrypsin clearance (> 56 mL/24 hr) confirms protein loss through the gut.	Think PLE when: Low albumin + edema/ascites + positive stool **alpha-1-antitrypsin.**
2 Small bowel amyloidosis	• Causes: diarrhea, GI bleeding, weight loss, or protein loss. • Diagnose with Congo red stain on duodenal or rectal biopsy. • Shows **apple-green** birefringence under polarized light. • **Therapy:** mainly supportive care + control underlying disease.	**Congo red** stain with **apple-green birefringence** is diagnostic. Treat the underlying condition + supportive management.
3 Intestinal lymphangiectasia	**Primary (congenital)** • Associated with Turner syndrome. • Creamy yellow-white villi due to dilated lymphatics (lymphangiectasia). **Secondary (acquired)** • Due to impaired lymphatic drainage from malignancy, surgery, radiation, or other causes.	Look for **creamy, yellow-white villi** with dilated lymphatics on endoscopy or biopsy.
4 Management of intestinal lymphangiectasia	• High-protein, low-fat diet. • Medium-chain triglyceride (MCT) supplementation. • Replace fat-soluble vitamins (A, D, E, K) and monitor nutritional status.	Core treatment: **High-protein, low-fat diet + MCT** supplementation.

MEMORY PEARLS

PLE = low albumin + edema/ascites + positive stool alpha-1-antitrypsin.

Lymphangiectasia = creamy white villi + low-fat diet with MCT.

Amyloidosis = Congo red stain with apple-green birefringence.

5. SHORT BOWEL SYNDROME, GLP-2 AND TRANSPLANT INDICATIONS

KEY POINT	DETAILS / CLINICAL CLUES	DDSEP ANSWER / EXAM PEARL
1 Short bowel syndrome (SBS) 	• After repeated small bowel resections. • Causes high-output diarrhea/ostomy, dehydration, malabsorption. • Patients often become dependent on parenteral nutrition (PN).	SBS leads to intestinal failure when the remaining bowel cannot maintain adequate nutrition and hydration.
2 Teduglutide (GLP-2 analogue) 	• Teduglutide is a GLP-2 analogue. • Indicated for short bowel syndrome with intestinal failure. • Used when diet, oral rehydration, antisecretory therapy, and antidiarrheal therapy are insufficient.	Reduces PN requirements and increases intestinal absorption and fluid uptake.
3 Small bowel transplant: when to consider 	**Consider transplant for intestinal failure with TPN failure due to any of:** • Impending or overt liver failure • Major central venous thrombosis • Recurrent line sepsis • Line fungemia • Septic shock / ARDS from line infection • Frequent, severe dehydration despite optimal therapy	Transplant is considered when complications or failure of PN make continued PN unsafe or unsustainable.
4 Post-transplant complications 	• Infections: e.g., CMV, bacterial, fungal. • Post-transplant lymphoproliferative disorder (PTLD). • Rejection and other immunosuppression related complications.	Monitor closely for infections (CMV) and PTLD after transplant.

MEMORY PEARL

SBS not controlled by conservative therapy = TEDUGLUTIDE (GLP-2 analogue).

Transplant trigger = TPN failure:

- ✓ Liver failure
- ✓ Central venous thrombosis
- ✓ Recurrent line sepsis
- ✓ Line fungemia
- ✓ Septic shock/ARDS from line infection
- ✓ Frequent severe dehydration

6. ACUTE MESENTERIC ISCHEMIA AND VASCULAR SMALL-BOWEL MIMICS

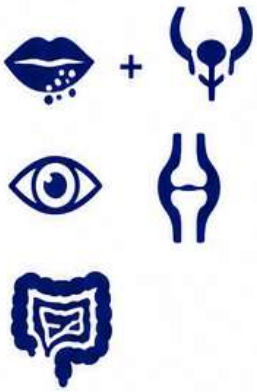
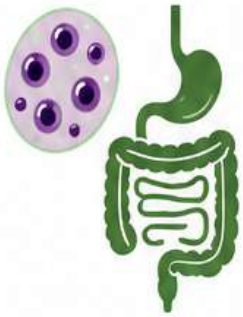
KEY POINT	DETAILS / CLINICAL CLUES	DDSEP ANSWER / EXAM PEARL
1 Acute mesenteric ischemia (AMI)  **TIME CRITICAL EMERGENCY**	• Intestinal blood flow is abruptly reduced. • Presents with severe abdominal pain out of proportion to exam. • After 24 hours, the chance of salvaging healthy bowel falls markedly (DDSEP logic). • Early diagnosis and revascularization are crucial.	Think AMI in older patients with risk factors (AF, atherosclerosis, low-flow states, embolus, thrombosis). **Delays > 24 hours greatly decrease bowel salvage and increase mortality.**
2 Severe pain + high lactate + thrombophilia / subtherapeutic anticoagulation 	• Severe abdominal pain with high lactate suggests ischemia. • Thrombophilia or subtherapeutic anticoagulation raises suspicion for mesenteric venous thrombosis. • Requires urgent CTA and operative evaluation / revascularization.	Do NOT observe. Urgent imaging and surgical consult are required.
3 Polyarteritis nodosa (PAN) 	• Necrotizing vasculitis of medium and small arteries. • Associated with hepatitis B infection. • GI involvement: recurrent abdominal pain, ischemia, bleeding, perforation.	Think PAN in HBV+ patient with systemic symptoms and abdominal ischemic complications.
4 Henoch–Schonlein purpura (IgA vasculitis) 	• Leukocytoclastic vasculitis with IgA deposition. • Can mimic Crohn ileitis. • Severe colicky abdominal pain, GI bleeding or intussusception. • Severe symptoms may require prednisone.	Consider HSP in children or young adults with palpable purpura, joint pain, renal findings and abdominal pain.

MEMORY PEARLS

Acute mesenteric ischemia = PAIN OUT OF PROPORTION + URGENT CTA / SURGERY.

HBV + medium-vessel abdominal disease = POLYARTERITIS NODOSA (PAN).

7. IMMUNE / ALLERGIC SMALL BOWEL DISEASE AND BEHÇET

KEY POINT	DETAILS / CLINICAL CLUES	DDSEP ANSWER / EXAM PEARL
1 Behçet disease 	• Young male. • Recurrent **oral** and **genital ulcers.** • Extraintestinal: uveitis, arthritis, skin lesions. • GI involvement: **ileocecal ulcers,** bleeding, abdominal pain, diarrhea. • **Path:** neutrophilic vasculitis of small/medium vessels.	**Treatment in DDSEP:** **Azathioprine** is the treatment answer. • Add corticosteroids for acute/severe flares. • Anti–TNF (e.g. infliximab, adalimumab) for selected severe or refractory disease.
2 Eosinophilic gastroenteritis 	• Can involve any part: **esophagus, stomach, small bowel,** and **colon.** • Symptoms vary with layer involved: – Dysphagia (mucosal) – Abdominal pain, diarrhea, malabsorption (mucosal) – Obstruction (submucosal) – Ascites, protein loss (serosal) • **Biopsy:** eosinophilic infiltration in GI tract.	**First-line treatment in DDSEP:** **Corticosteroids.** • Clinical response is usually good. • Diet elimination and other agents are **second-line/adjuncts.**
3 Systemic mastocytosis 	• Clonal mast cell disorder involving bone marrow and other organs including GI tract. • **GI features:** – Episodic abdominal pain, diarrhea – Flushing, urticaria, anaphylactoid symptoms – Peptic ulcers • **Triggers:** alcohol, NSAIDs, physical stress, foods, drugs. • **Path:** mast cell infiltration of GI tract.	**Diagnosis supported by mast cell markers:** • KIT (CD117) positive • Serum tryptase elevated • Aberrant CD25 expression **Classic stem (DDSEP):** Bone marrow biopsy with tryptase staining showing mast cell infiltration + aberrant CD25 expression.

MEMORY PEARLS

Behçet = oral + genital ulcers + ileocecal disease.

Treatment answer = **AZATHIOPRINE.**

Steroids or anti-TNF for severe disease.

Eosinophils in GI tract = eosinophilic gastroenteritis.

↓

First-line = STEROIDS.

Mast cells = KIT (CD117) / tryptase / CD25.

↓

Bone marrow tryptase stain with aberrant CD25 confirms systemic mastocytosis.

8. SMALL BOWEL BLEEDING AND TUMORS

KEY POINT	DETAILS / CLINICAL CLUES	DDSEP ANSWER / EXAM PEARL
1 When to think small bowel 	• Obscure GI bleeding or iron deficiency after negative EGD and colonoscopy. • Consider small-bowel source. • Especially in recurrent melena, overt bleeding, or unexplained iron deficiency anemia.	**Next step = small-bowel evaluation.** • CT enterography (preferred when stable; good for mural lesions, tumors, Crohn). • Capsule endoscopy (preferred for mucosal lesions; avoid if obstruction/stricture suspected). • Deep (device-assisted) enteroscopy for diagnosis/therapy if needed.
2 GIST (Gastrointestinal Stromal Tumor) 	• Submucosal / mural mass anywhere in the small bowel. • Presents with iron deficiency, melena, or bleeding. • May also cause abdominal pain, early satiety, or obstruction. • Diagnosis: IHC positive for KIT (CD117) ± DOG1.	**Mural mass + KIT (CD117) = GIST.** **Pathology:** spindle or epithelioid cells; KIT (CD117) positive.
3 Management of GIST 	• Localized jejunal/ileal GIST causing bleeding. • Recurrent, metastatic, or unresectable disease.	• Localized disease = **surgical** resection with clear margins. • Recurrent / metastatic disease = tyrosine kinase inhibitor (**imatinib**). • Risk stratify (size, mitotic rate, location) for recurrence.
4 Small-bowel mimics that can bleed or masquerade as Crohn / celiac 	• Whipple disease • Amyloidosis • Behçet disease • Vasculitis (e.g., PAN, HSP) • Mast cell disease (systemic mastocytosis) • Others: lymphoma, NSAID enteropathy, radiation enteritis.	These conditions can cause bleeding, mucosal ulceration, or malabsorption and may mimic Crohn disease or celiac disease.

MEMORY PEARLS

Negative EGD/colon + iron deficiency or melena = THINK SMALL BOWEL.

Mural mass + KIT (CD117) = GIST. ↓ Localized = resection.

Recurrent/metastatic = imatinib.

Think of mimics: Whipple, amyloid, Behçet, vasculitis, mast cell disease.

9. TROPICAL SPRUE AND TRAVEL-RELATED MALABSORPTION

KEY POINT	DETAILS / CLINICAL CLUES	DDSEP ANSWER / EXAM PEARL
1 Tropical sprue: who and when to suspect 	• Chronic malabsorptive diarrhea (steatorrhea, weight loss, bloating, weakness). • Occurs after travel or long-term residence in endemic regions (tropical/subtropical developing areas). • Symptoms may begin months to years after exposure. • Usually without fever or severe pain.	Think tropical sprue in chronic malabsorption after endemic travel, even if years later.
2 Laboratory clues 	• Folate deficiency is typical. • Vitamin B12 may also be low. • Other findings: anemia (macrocytic), hypoalbuminemia, low carotene, hypocalcemia, prolonged PT.	Folate low ± B12 low is classic.
3 DDSEP treatment (answer) 	• Treat with: – Folic acid – Vitamin B12 – Tetracycline (antibiotic)	**DDSEP treatment = FOLIC ACID + B12 + TETRACYCLINE.**
4 SIBO can mimic tropical sprue 	• SIBO can cause similar symptoms and malabsorption. • More likely when risk factors are present: long-standing diabetes, radiation, strictures, altered anatomy. • Symptoms often respond transiently to antibiotics.	If symptoms improve temporarily with antibiotics → consider SIBO as a mimic and evaluate accordingly.

MEMORY PEARLS

Tropical sprue = chronic malabsorption after travel/residence in endemic areas (may appear years later).

Folate deficiency (± B12 deficiency) → treat with FOLIC ACID + B12 + TETRACYCLINE.

SIBO can mimic: think risk factors (diabetes, radiation, strictures) and response to antibiotics.

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q1	Appropriate celiac tTG-IgA testing candidate	**Adult with elevated AST/ALT on unrestricted diet**	Celiac can present as unexplained transaminase elevation; test while eating gluten.
Q2	Disease that can cause duodenal villous blunting	**Giardiasis infection**	Villous blunting is not specific for celiac disease.
Q3	Diarrhea/weight loss + PAS-positive macrophages	**Ceftriaxone followed by TMP-SMX (Bactrim)**	Whipple disease = Tropheryma whipplei; induction then prolonged oral therapy.
Q4	Eight weeks diarrhea + positive anti-enterocyte antibody	**Oral steroid therapy**	Autoimmune enteropathy responds to steroids.
Q5	Diarrhea/weight loss/melena + restrictive cardiomyopathy	**Small bowel biopsy Congo red positive with birefringence**	Amyloid = apple-green birefringence under polarized light.
Q6	Dialysis patient, granular/polypoid small-bowel amyloid	**Supportive care and antidiarrheal therapy**	Small bowel amyloidosis management is mainly supportive plus underlying disease control.
Q7	True statement about acute mesenteric ischemia	**After 24 h, salvage of healthy bowel is <50%**	Time-critical ischemia; do not wait for late lactate changes.
Q8	IgA deficiency during celiac testing	**tTG plus deamidated gliadin peptide IgG strategy**	IgA deficiency requires IgG-based serology.
Q9	Chronic HBV + recurrent abdominal pain/vascular lesions	**Polyarteritis nodosa**	PAN affects medium/small arteries and is associated with HBV.
Q10	Diarrhea, edema, positive stool alpha-1 antitrypsin	**Amyloidosis involving the small intestine**	Protein-losing enteropathy can be amyloid-related.
Q11	Best SIBO breath-test candidate	**Roux-en-Y patient with bloating/loose stools not confounded by meds**	Breath tests are affected by antibiotics/laxatives/ probiotics/promotility drugs.
Q12	Celiac on GFD, new bloating/diarrhea after antibiotics	**Lactulose breath test**	Antibiotics and symptoms suggest SIBO.
Q13	Farmer with diarrhea, fatigue and arthralgias	**Tropheryma whipplei**	Whipple disease has GI plus joint/systemic features.
Q14	Intestinal failure/TPN with severe dehydration admissions	**Candidate for small intestinal transplant**	Frequent severe dehydration is a transplant trigger in intestinal failure.
Q15	EoE patient with new bloating, atrophic gastritis/diverticulosis	**Atrophic gastritis**	Atrophic gastritis predisposes to SIBO through hypochlorhydria.
Q16	Radiation history + ileal stricture + recurrent SIBO	**Refer to surgeon for resection of stricture**	Fix anatomic driver when feasible.
Q17	Turner syndrome + floating diarrhea + alpha-1 antitrypsin + lymphatics	**Low-fat diet with medium-chain triglycerides**	Primary intestinal lymphangiectasia/ Waldmann disease.
Q18	Dark stools + abdominal pain + oral/genital ulcers	**Behcet disease**	Ileocecal ulcerating vasculitis can bleed and mimic Crohn.
Q19	Ulcers triggered by alcohol/NSAIDs + systemic symptoms	**Mast cell disorder**	Look for KIT/CD117 and tryptase.
Q20	Scleroderma with SIBO; abnormality NOT associated	**Folate deficiency**	SIBO tends to cause B12 deficiency with normal/high folate.
Q21	Celiac on strict GFD; tTG rising, bloating/loose stools	**Inadvertent gluten exposure**	Most common cause of persistent activity.
Q22	Short gut after mesenteric ischemia, high output despite conservative therapy	**GLP-2 agonist**	Teduglutide reduces parenteral support needs.
Q23	HIV controlled, IDA, normal EGD/colonoscopy	**Gastrointestinal stromal tumor (GIST)**	Small bowel GIST can present as occult bleeding.
Q24	Protein C deficiency, severe abdominal pain, lactate 4.6	**Exploratory laparoscopy**	Suspected acute mesenteric ischemia needs urgent operative evaluation.
Q25	Short gut patient asks about transplant complications	**CMV infection and PTLD**	Small bowel transplant complications include infection and lymphoproliferative disease.

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

DDSEP Q	Scenario / Key Clue	Best Answer	Exam Pearl / Mnemonic
Q26	Celiac non-responder after 2–8 months; high tTG	**Careful GFD review with registered dietician**	First step is adherence/inadvertent gluten audit.
Q27	Recurrent pyrexia/rash + villous atrophy + absent plasma cells	**Budesonide**	CVID enteropathy can mimic celiac; steroids help GI disease.
Q28	Controlled celiac on strict GFD; additional management	**Pneumococcal vaccination**	Celiac-associated hyposplenism/ infection prevention clue.
Q29	Indication for celiac testing	**Unexplained elevated aminotransferases**	Gluten-dependent hypertransaminasemia can normalize on GFD.
Q30	Patient on GFD >2 months before any celiac testing	**HLA-DQ2/DQ8 genotyping**	Negative HLA effectively rules out celiac before gluten challenge.
Q31	Villous blunting, absent goblet/Paneth cells, crypt apoptosis	**Anti–enterocyte antibodies**	Autoimmune enteropathy histology + antibody clue.
Q32	Chronic diarrhea, negative workup, on olmesartan	**Stop olmesartan**	Olmesartan enteropathy mimics celiac.
Q33	Villous injury after hip fracture; NSAID exposure likely	**Stop NSAIDs**	Drug enteropathy; do not start GFD without support.
Q34	Chronic malabsorption year after Dominican Republic travel	**Folate + B12 + tetracycline**	Tropical sprue may be delayed/latent.
Q35	Diabetes/radiation, bloating, high B12/folate pattern	**Lactulose hydrogen breath testing**	Risk-factor SIBO; breath test is DDSEP next step.
Q36	Post-radiation PLE with low albumin/edema/ascites	**Creamy yellow-white jejunal villi**	Secondary intestinal lymphangiectasia endoscopic clue.
Q37	Diabetes + Haiti travel + recurrent greasy diarrhea after antibiotics	**Perform lactulose hydrogen breath testing**	High B12 and recurrence after antibiotics favor SIBO over tropical sprue.
Q38	TPN-dependent SBS; transplant discussion	**One episode of line-related fungemia**	Line fungemia alone meets transplant consideration criteria.
Q39	Crohn + ankylosing spondylitis, ↑ PL, hypergammaglobulinemia	**Rectal biopsy with Congo red staining**	AA amyloid from chronic inflammation.
Q40	Elderly recurrent post-polypectomy bleeding	**Rectal biopsy at relook colonoscopy with Congo red**	Senile amyloid can involve GI tract and bleeding.
Q41	Ovarian cancer surgery, edema, creamy duodenal mucosa	**Low-fat diet with medium-chain triglycerides**	Secondary lymphangiectasia treatment.
Q42	Nausea/vomiting/edema, nephrotic-range urine protein	**Bone marrow biopsy with tryptase staining and aberrant CD25**	Systemic mastocytosis diagnostic workup.
Q43	Whipple-like PAS-macrophages stain in exclusion mimics	**Acid-fast bacillus staining**	AFB helps distinguish Whipple from mycobacterial infection.
Q44	Whipple disease with cancer hx CNS relapse	**CSF T. whipplei PCR**	CNS involvement changes management and relapse risk.
Q45	Eosinophilic esophagitis + GI eosinophilic disease	**Corticosteroids**	Eosinophilic GI gut-predominant therapy.
Q46	Ileal Crohn stricture despite azathioprine; prior anti-TNF mechanistic failure	**Switch to ustekinumab**	IL-12/23 agent after different-class failure logic.
Q47	Turkish man, oral/genital ulcers, uveitis/arthritis, diarrhea	**Azathioprine**	Behcet GI disease can respond to immunosuppression.
Q48	Purpura/arthralgia + RLQ pain/diarrhea	**Prednisone**	IgA vasculitis with severe abdominal pain.
Q49	TPN-dependent SBS despite maximal conservative therapy	**Teduglutide**	GLP-2 analogue for SBS with intestinal failure.
Q50	IDA/melena, negative EGD/colon, mid-jejunal mural mass	**Surgical resection of jejunal mass**	Localized small-bowel GIST causing bleeding → surgery.