MADE SIMPLE SERIES

DDSEP 10 MADE SIMPLE

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Hepatology & Gastroenterology
Comprehensive Review Guide

DDSEP 10 Novel Questions Compared with DDSEP 09 - Improved Section I Notes

Chapters 1-15, excluding Chapter 11

Only genuinely **new** or **materially new** DDSEP 10 questions whose idea/answer was not already present in the DDSEP 09 merged chapter notes.

Structure used in each chapter

SECTION I

EXAM PEARL NOTES

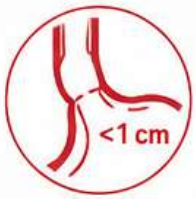
SECTION II

DDSEP SCENARIO-BASED EXAM TABLE

CHAPTER 1 — ESOPHAGEAL DISORDERS

SECTION I - EXAM PEARL NOTES

1. BARRETT'S, REFLUX CLASSIFICATION, AND ANTIRELFUX BARRIER LOGIC

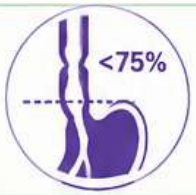


OVERDIAGNOSIS TRAP

An irregular Z-line or columnar mucosa less than 1 cm is **NOT** Barrett's esophagus and should **NOT** enter routine biopsy or surveillance pathways.

RISK & SURVEILLANCE

Nondysplastic Barrett's has low annual adenocarcinoma progression risk, **~0.1%** (DDSEP 10 stem). Long-segment nondysplastic Barrett's favors the shorter end of the **3–5 year** surveillance range.



LOS ANGELES GRADE C

Mucosal breaks continuous between mucosal folds but involving **less than 75%** of the esophageal circumference.

ANTIRELFUX BARRIER LOGIC

Persistent regurgitation with a large hiatal hernia is a **MECHANICAL** antireflux-barrier problem. PPIs reduce acidity but **do not** reliably stop reflux events. Weight reduction remains part of reflux control in overweight patients.

MEMORY PEARLS

<1 cm Z-line = **DO NOT LABEL**

Long Barrett = **SHORTER** surveillance

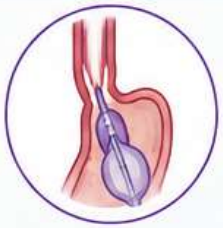
LA Grade C = Continuous breaks <75% circumference

Hernia regurgitation = **BARRIER FAILURE**, not acid failure

2. EOSINOPHILIC AND IMMUNE-MEDIATED ESOPHAGEAL DISEASE

EoE TREATMENT – EMPIRIC DIET

Six-food elimination for EoE removes **DAIRY, EGG, WHEAT, SOY, NUTS,** and **FISH/SHELLFISH.** DDSEP 10 emphasizes ***empiric elimination and reintroduction*** rather than skin-prick or serum IgE testing for food triggers.



DILATION IN EoE

When EoE inflammation is controlled but rings/strictures persist, dilation treats fixed fibrostenosis. The common adverse outcome is **POSTPROCEDURAL CHEST PAIN,** whereas perforation and significant bleeding are uncommon with graded technique.

STEROID SIDE EFFECT

Swallowed topical steroids can cause **Candida esophagitis**; persistent dysphagia on steroids should not automatically mean active EoE inflammation.

ESOPHAGEAL LICHEN PLANUS

A mimic to remember when dysphagia is accompanied by **ORAL PLAQUES** plus **ESOPHAGEAL PLAQUES, WEBS,** or **STRICTURES.**

MEMORY PEARLS

EoE diet = **EMPIRIC SIX-FOOD ELIMINATION,** not allergy testing.

Dilation treats fibrostenosis; **CHEST PAIN** is most common adverse effect.

Steroid can grow **CANDIDA**; persistent dysphagia ≠ always active EoE.

Think **LICHEN PLANUS** when oral plaques + esophageal plaques/ webs/strictures.

KEY MEMORY SUMMARY | EoE diet is **empiric**; steroid can grow **Candida**; dilation **hurts** more often than it **perforates.**

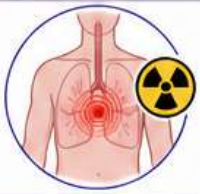
3. OROPHARYNGEAL, STRUCTURAL, AND INFECTIOUS DYSPHAGIA CLUES

OROPHARYNGEAL DYSPHAGIA

Coughing, choking, or aspiration during swallowing points to oropharyngeal dysphagia. Evaluate first with a **MODIFIED BARIUM SWALLOW** rather than routine esophageal testing.

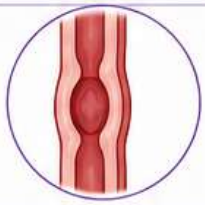
ZENKER'S DIVERTICULUM

Presents in older adults with **regurgitation of old undigested food**, cough, halitosis, and a pharyngoesophageal pouch.



RADIATION-INDUCED STRICTURE

Can appear **YEARS** after thoracic radiation and causes slowly progressive solid-food dysphagia.



SCHATZKI RING

Causes intermittent meat/bread dysphagia.
Treated by endoscopic disruption, commonly **BALLOON DILATION**.

INFECTIOUS ESOPHAGITIS (HSV)

In transplant or immunosuppressed patients, odynophagia with small ulcers and multinucleated giant cells supports viral esophagitis, especially HSV.

MEMORY PEARLS

Cough/aspiration = OROPHARYNGEAL → do **MODIFIED BARIUM SWALLOW** first.

Zenker's = old food back, cough + halitosis + pharyngoesophageal pouch.

Radiation stricture = late effect → slow progressive solid dysphagia.

Schatzki ring = intermittent meat/bread dysphagia → balloon dilation.

Transplant/immuno + odynophagia + ulcers + giant cells = HSV esophagitis.

4. FUNCTIONAL, MANOMETRIC, AND DRUG-RELATED ESOPHAGEAL DISORDERS

FUNCTIONAL DYSPHAGIA

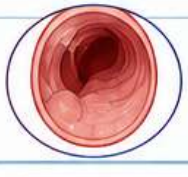
Chronic troublesome dysphagia after structural disease, mucosal disease, GERD, EoE, and major motor disorders have been **EXCLUDED**.

MANOMETRY PEARL (IEM NOT OVERDIAGNOSED)

A few weak swallows do not equal ineffective esophageal motility.
DDSEP 10 tests that **NORMAL IRP** with only **20% WEAK SWALLOWS** can still be **NORMAL** motor function.

OPIOID-INDUCED ESOPHAGEAL DYSFUNCTION

Chronic opioids can produce opioid-induced esophageal dysmotility, including distal esophageal spasm or EGJOO patterns.
Opioid cessation is the **FIRST** management step when feasible.



PROXIMAL INLET PATCH

Asymptomatic proximal inlet patch is an **INCIDENTAL** finding and needs **NO TREATMENT**.

MEMORY PEARLS

Normal scope + normal biopsies + normal reflux/motility = **FUNCTIONAL DYSPPHAGIA**.

Normal IRP + ~20% weak swallows can still be **NORMAL** motor function.

Opioid spasm/EGJOO pattern?
STOP OPIOID FIRST (if possible).

Asymptomatic inlet patch = **INCIDENTAL**, no treatment.

KEY MEMORY SUMMARY

Oropharyngeal clues → **Modified barium swallow** first.
Manometry pearls → **Normal IRP + few weak swallows** can be normal.
Opioid dysmotility → **Stop opioid first** when feasible.

SECTION II – DDSEP SCENARIO-BASED EXAM TABLE

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q3	Annual cancer progression risk in nondysplastic Barrett's	0.1%	Nondysplastic Barrett's has low annual adenocarcinoma progression risk.
Q4	Irregular Z-line / columnar mucosa <1 cm	Continue current regimen	Do not biopsy or surveil an irregular Z-line <1 cm as Barrett's.
Q5	Long-segment nondysplastic Barrett's surveillance interval	Repeat endoscopy in 3 years	Nondysplastic Barrett's surveillance is usually 3–5 years; long segment favors 3 years.
Q8	Six-food elimination diet in EoE	Dairy, eggs, fish/shellfish, nuts, soy, wheat	Classic six-food elimination = milk, egg, wheat, soy, nuts, seafood.
Q10	Common adverse outcome after EoE dilation	Postprocedural pain	Chest pain/pain is common after EoE dilation; perforation is rare.
Q11	Adverse event of swallowed topical steroid in EoE	Candida esophagitis	Swallowed budesonide/fluticasone can cause esophageal candidiasis.
Q12	Identifying patient-specific EoE food triggers	Empiric elimination diet	Skin prick/IgE testing correlates poorly with EoE triggers.
Q14	Dysphagia + oral plaques + esophageal plaques	Lichen planus	Esophageal lichen planus can cause plaques, strictures, and dysphagia.
Q15	Coughing while swallowing / oropharyngeal dysphagia	Modified barium swallow	Cough during meals points to oropharyngeal dysphagia.
Q19	Regurgitation of old food with barium pouch	Zenker's diverticulum	Elderly + undigested food regurgitation/halitosis = Zenker's.
Q20	Progressive solid dysphagia years after thoracic radiation	Radiation-induced stricture	Radiation strictures can present late and progress slowly.
Q21	Schatzki ring management	Balloon dilation	Intermittent meat/bread dysphagia + ring = disrupt/dilate ring.
Q22	Incidental proximal esophageal inlet patch	No further therapy	Asymptomatic inlet patch needs no treatment.

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q24	Post-transplant odynophagia, small ulcers, multinucleated giant cells	Viral esophagitis	Multinucleated giant cells suggest HSV esophagitis.
Q29	LA esophagitis classification: breaks involving 50% circumference	Los Angeles C esophagitis	LA C = mucosal breaks continuous between folds but <75% circumference.
Q36	Refractory GERD/regurgitation in overweight patient	Counsel on weight management	Weight/lifestyle optimization remains part of refractory GERD care.
Q37	Large hiatal hernia with regurgitation despite PPI	Disrupted antireflux barrier	PPI reduces acid, not mechanical reflux from a defective barrier.
Q43	HRM: normal IRP with only 20% weak swallows	Normal esophageal motor function	Minor weak swallows do not meet IEM criteria.
Q47	Dysphagia with normal EGD, biopsies, reflux testing, and manometry	Functional dysphagia	Functional dysphagia requires exclusion of structural, mucosal, reflux, and major motor disease.
Q48	Dysphagia/chest pain in chronic opioid user	Opioid cessation	Opioids can cause EGJOO/spastic esophageal dysmotility; stop if possible.

CHAPTER 2 — ACID DISEASES OF THE STOMACH

SECTION I - EXAM PEARL NOTES

1. GASTRIC INTESTINAL METAPLASIA, BIOPSY MAPPING, AND *H. PYLORI*

MANAGEMENT	DDSEP 10 shifts GIM management toward **risk stratification:** treat ***H. pylori***, confirm eradication, and individualize surveillance rather than surveilling every nondysplastic case automatically.
HIGHEST CANCER-RISK GIM PATTERN	**Incomplete metaplasia** with **extensive** involvement of both antrum and body.
BIOPSY MAPPING	When endoscopy is otherwise normal but gastric pathology is being assessed, **sample body, antrum, and incisura** in a Sydney-style biopsy strategy.
***H. PYLORI* TEST OF CURE**	**Stool antigen or urea breath testing** is used after therapy when the patient has been off confounding antibiotics/bismuth/PPI long enough.
MEMORY	**GIM risk = incomplete + extensive; H. pylori is treated and cure is documented.**

2. MODERN ACID SUPPRESSION: METABOLISM, VONOPRAZAN, H2 BLOCKERS, AND ALGINATES

RAPID PPI METABOLISM	Persistent symptoms despite correct PPI timing and twice-daily dosing can reflect **rapid PPI metabolism**, not simply nonadherence.
VONOPRAZAN	Vonoprazan is a potassium-competitive acid blocker that provides **potent acid suppression** and may be selected when stronger suppression is needed and antireflux surgery is unsuitable.
H2 BLOCKERS	Famotidine and other H2 blockers have **faster onset** than PPIs and are useful for intermittent/on-demand symptoms, but **tachyphylaxis** limits continuous daily use.
ALGINATE	Alginate is useful for persistent postprandial regurgitation because it **forms a raft** over the acid pocket; it is not merely another acid-suppressive drug.
MEMORY	**rapid metabolizer = PPI failure; vonoprazan = stronger suppression; alginate = acid-pocket raft.**

3. PPI SAFETY AND DRUG-SPECIFIC ADVERSE EFFECTS

STRONG INDICATION NO ROUTINE SURVEILLANCE	When the indication is strong, such as high-risk ulcer bleeding prevention, DDSEP 10 **does not recommend routine** laboratory or imaging surveillance for speculative PPI harms.
ENTERIC INFECTION RISK	Enteric infection is one of the more accepted PPI-associated risks; **avoid overstating weak associations** such as dementia or fracture as proven causality.
PPI-INDUCED HYPOMAGNESEMIA	PPI-induced hypomagnesemia can present with **cramps, weakness, tremor, or arrhythmia** clues; **omeprazole** in the stem is the trap drug.
CLOPIDOGREL PROTECTION	**Pantoprazole** is favored when GI protection is needed with clopidogrel because it has less CYP2C19 inhibition than omeprazole.
MEMORY	**strong indication beats PPI fear; | cramps on PPI = magnesium; clopidogrel protection = pantoprazole.**

4. ULCER PREVENTION, HYPERGASTRINEMIA, AND GASTRIC CANCER RISK

MÉNÉTRIER DISEASE	Ménétrier disease is linked to **TGF-alpha/EGFR signaling** and presents conceptually with giant folds, foveolar hyperplasia, protein loss, and increased gastric cancer concern.
HYPERGASTRINEMIA INTERPRETATION	Hypergastrinemia should be interpreted with **gastric pH or acid output**; **stop PPI** and repeat gastrin when safe before labeling Zollinger-Ellison syndrome.
SOMATOSTATIN PHYSIOLOGY	Somatostatin is the **physiologic brake** on gastrin and acid secretion.
ULCER PREVENTION AND BLEEDING RISK	Misoprostol prevents NSAID ulcer complications but is limited by **diarrhea and cramping**; **NSAID plus anticoagulation** is a major bleeding-risk combination.
AUTOIMMUNE ATROPHIC GASTRITIS (PERNICIOUS ANEMIA)	Autoimmune metaplastic atrophic gastritis/pernicious anemia increases risk of **noncardia gastric adenocarcinoma** and **type 1 gastric NET**.
MEMORY	**hypergastrinemia needs context; somatostatin stops gastrin; misoprostol helps but causes diarrhea; autoimmune gastritis → cancer + NET.**

5. PRACTICAL MANAGEMENT TRAPS

ASPIRIN PRIMARY PREVENTION	Primary-prevention aspirin should usually be **stopped** after significant erosive gastropathy or bleeding risk; secondary **prevention** decisions are different.
GASTROPARESIS DIAGNOSIS	Diabetic nausea, early satiety, and weight loss should **not be called gastroparesis** until obstruction, ulceration, or malignancy has been excluded by **upper endoscopy**.
MEMORY	**primary aspirin can stop; gastroparesis is diagnosed after obstruction is excluded.**

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q1	H. pylori-positive gastric intestinal metaplasia	Treat H. pylori and confirm eradication	In GIM, eradicate H. pylori and document cure.
Q2	Nondysplastic GIM surveillance decision	Shared decision based on patient concern/risk	GIM surveillance is individualized, not automatic.
Q3	Highest-risk GIM pattern	Incomplete GIM involving antrum and body	Extensive + incomplete GIM carries higher gastric cancer risk.
Q4	PPI nonresponse despite correct timing/BID dosing	Rapid PPI metabolism	Rapid CYP2C19 metabolism can explain PPI failure.
Q6	Severe reflux with absent contractility, surgery unsuitable	Switch lansoprazole to vonoprazan	Vonoprazan gives stronger acid suppression than standard PPIs.
Q7	Long-term PPI after high-risk ulcer bleed	No monitoring of PPI side effects	When PPI indication is strong, avoid unnecessary surveillaned for speculative harms.
Q8	Best-supported PPI adverse association	Enteric infection	Enteric infection is among the more accepted PPI-associated risks.
Q9	Ménétrier disease pathway	EGFR	Ménétrier disease involves TGF-alpha/EGFR signaling and giant folds.
Q14	Normal EGD in dyspepsia: biopsy protocol	Biopsy body, antrum, and incisura	Sydney-style sampling improves H. pylori/gastritis assessment.
Q15	Persistent regurgitation despite PPI, nonsurgical option	Alginate	Alginate targets the postprandial acid pocket/reflux raft.

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q18	Autoimmune gastritis/pernicious anemia cancer risk	Noncardia gastric adenocarcinoma	AMAG/pernicious anemia increases gastric adenocarcinoma and type 1 NET risk.
Q25	Recurrent postbulbar ulcers + diarrhea + elevated gastrin on PPI	Withdraw PPI and repeat gastric acid analysis	Interpret gastrin with gastric pH/acid output; PPI can falsely elevate gastrin.
Q26	Physiologic inhibitor of gastric acid	Somatostatin	Somatostatin suppresses gastrin and acid secretion.
Q27	Diarrhea after non-PPI ulcer protective regimen	Misoprostol	Misoprostol prevents NSAID ulcers but commonly causes diarrhea/cramping.
Q28	Acid suppressant with tachyphylaxis	Famotidine	H2 blockers lose effect with continuous use.
Q29	Muscle cramps/weakness after OTC acid suppression	Omeprazole	PPI-induced hypomagnesemia can cause cramps and weakness.
Q37	Elevated gastrin while on omeprazole	Stop omeprazole and recheck gastrin	Confirm hypergastrinemia off PPI before labeling ZES.
Q38	H. pylori test of cure	Stool antigen	Stool antigen confirms eradication when off PPI/antibiotics.
Q39	Greatest NSAID-related GI bleeding risk modifier	Need for anticoagulation	NSAID + anticoagulation markedly increases bleeding risk.
Q40	Aspirin used only for prophylaxis with erosive gastropathy	Stop aspirin	Primary-prevention aspirin should usually stop after significant GI injury.
Q42	Diabetic nausea/early satiety/weight loss before gastroparesis diagnosis	Upper endoscopy	Exclude obstruction/malignancy before diagnosing gastroparesis.
Q43	FDA-approved non-PPI prevention of NSAID ulcer complications	Misoprostol	Misoprostol prevents NSAID ulcers but is limited by tolerability.
Q45	Recent MI/stent on aspirin + clopidogrel needing GI protection	Pantoprazole	Pantoprazole is preferred when avoiding omeprazole-clopidogrel interaction.
Q50	Intermittent mild reflux needing quick on-demand therapy	Famotidine	H2 blockers act faster than PPIs for intermittent symptoms.

CHAPTER 3 — PANCREATIC PHYSIOLOGY AND DISEASE

SECTION I - EXAM PEARL NOTES

1. DEVELOPMENTAL, CFTR-RELATED, AND EXOCRINE PANCREATIC DISEASE

 **ANNULAR PANCREAS**	Annular pancreas reflects abnormal rotation/fusion of the ventral and dorsal pancreatic buds, producing a ring of pancreatic tissue around the second part of the duodenum; **Down syndrome** is a classic association.
 **CFTR-RELATED PANCREATIC DISEASE**	CFTR-related pancreatic disease is a **ductal bicarbonate problem:** thick acidic secretions injure the gland and reduce enzyme delivery and function.
 **FECAL ELASTASE**	Fecal elastase is a **practical screening test for EPI** after major pancreatic resection and in long-standing diabetes with steatorrhea, weight loss, or pancreatic atrophy.
 **CYSTIC FIBROSIS (CF)**	CF can present with EPI, recurrent pancreatitis in residual-function variants, and later dysglycemia; **early referral matters** before islet reserve is lost.
 **MEMORY**	**CFTR blocks bicarbonate; fecal elastase documents EPI.**

2. ACUTE PANCREATITIS, NECROSIS, AND POST-NECROSIS DUCT/VASCULAR COMPLICATIONS

 **CANCER-WARNING PRESENTATION**	First idiopathic pancreatitis in an older smoker with weight loss or new diabetes is a **cancer-warning presentation** and needs high-quality pancreatic imaging.
 **SEVERE vs MILD DISEASE**	Severe pancreatitis with persistent organ failure belongs in **ICU-level monitoring**; mild lipase elevation without pain/imaging criteria is **not pancreatitis**.
 **INFECTED NECROSIS MANAGEMENT**	Stable infected necrosis can be managed with **antibiotics, enteral nutrition**, and **delayed step-up drainage/ necrosectomy** rather than reflex immediate intervention.
 **LAMS MANAGEMENT**	After LAMS drainage/necrosectomy, **remove the metal stent** once the collection resolves to reduce delayed bleeding, buried stent, and other adverse events.
 **SPLENIC VEIN THROMBOSIS**	Isolated splenic vein thrombosis after pancreatitis without bleeding may be followed with **interval imaging** rather than **automatic anticoagulation**.
 **MEMORY**	**Idiopathic pancreatitis in older smoker = rule out cancer; severe = ICU; stable infected necrosis = step-up; remove LAMS; isolated SVT (no bleed) = observe with imaging.**

3. AUTOIMMUNE PANCREATITIS AND PANCREATIC MASS TRAPS

 **MASS EVALUATION**	A pancreatic mass with possible autoimmune pancreatitis still requires **tissue evaluation before steroids** when malignancy remains plausible; elevated IgG4 alone is not diagnostic.
 **TYPE 1 AIP**	Type 1 AIP is IgG4-related lymphoplasmacytic sclerosing pancreatitis, typically in **older men** and often with other IgG4-related organ disease.
 **TYPE 2 AIP AND IBD**	Type 2 AIP is associated with IBD; recurrent diarrhea in this context should prompt **ileocolonoscopy** with **biopsies**.
 **DISCONNECTED PANCREATIC DUCT SYNDROME**	Disconnected pancreatic duct syndrome after necrosis requires **multidisciplinary surgical/endoscopic planning** because viable upstream pancreas may continue to leak or inflame.
 **MEMORY**	**Do not steroid a mass until cancer is reasonably excluded.**

4. CYSTS, CHRONIC PANCREATITIS, GENETICS, AND TUMORS

 **PANCREATIC CYST EVALUATION**	**MRI/MRCP** is preferred to characterize an incidental pancreatic cyst and duct communication; surveillance is not useful when the patient is **not a surgical candidate**.
 **HIGH-RISK FEATURES**	Interval cyst **growth**, **mucinous** fluid profile, **low glucose**, **high CEA**, **KRAS mutation**, or **neuroendocrine cytology** should escalate evaluation.
 **MCN AND CYSTIC NET**	• MCN classically occurs in **women** in the pancreatic body/tail and **does not communicate** with the duct. • Cystic NET has **salt-and-pepper chromatin** and **neuroendocrine markers**.
 **CHRONIC PANCREATITIS TESTING & MANAGEMENT**	• **EUS** when CT is unrevealing. • Adequate **PERT dosing: 40,000–50,000** USP lipase units with meals. • **Genetic testing** in young idiopathic disease. • **Celiac plexus block** when pain is refractory without a ductal target.
 **GENETICS & FAMILIAL RISK**	Familial pancreatic cancer kindreds should undergo **genetic counseling and germline testing** before surveillance planning.
 **SUSPECTED PANCREATIC CANCER**	Suspected pancreatic cancer with biliary obstruction needs **tissue diagnosis and drainage** when neoadjuvant or palliative pathways are expected.
 **MEMORY**	**Do not steroid a mass until cancer is reasonably excluded.**

SECTION II – DDSEP SCENARIO-BASED EXAM TABLE

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q5	Down syndrome newborn with feeding issues and bilious emesis	Annular pancreas	Annular pancreas is linked to Down syndrome.
Q6	Child with chronic pancreatitis and sweat chloride positive	CFTR-related pancreatic disease	CFTR defect → thick secretions → duct injury and EPI.
Q7	Steatorrhea after distal pancreatectomy with weight loss	Fecal elastase measurement	Fecal elastase screens for EPI in resection and diabetes.
Q8	Long-standing CF patient now developing impaired glucose tolerance	CF-related diabetes	CF can lead to pancreatitis and dysglycemia.
Q9	Idiopathic pancreatitis in 62-year-old smoker with 10-kg weight loss	High-quality pancreatic imaging (EUS/MRI pancreas)	Cancer-warning presentation; image the pancreas.
Q10	Severe pancreatitis with organ failure and AKI	ICU monitoring and support	Persistent organ failure = severe disease → ICU.
Q11	Lipase 1.5 × ULN with no abdominal pain or imaging findings	Not pancreatitis	Diagnosis requires pain + enzyme criteria + imaging.
Q14	Walled-off pancreatic necrosis with infection but patient is stable	Antibiotics, enteral nutrition, delayed step-up approach	Step-up drainage/necrosectomy rather than immediate intervention.
Q15	LAMS placed for WON drainage; collection resolved	Remove LAMS	Remove LAMS to prevent delayed bleeding and buried stent.
Q18	Isolated splenic vein thrombosis after pancreatitis; no GI bleeding	Interval imaging; no anticoagulation	Isolated SVT without bleeding → observe with imaging.
Q19	Pancreatic mass with high IgG4 and jaundice	Tissue diagnosis before steroids	Do not steroid a mass until cancer is excluded.

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q20	Incidental 2.5-cm pancreatic cyst on CT in fit 58-year-old	MRI/MRCP	MRI/MRCP best characterizes cyst and duct communication.
Q21	Pancreatic cyst in non-surgical candidate	No surveillance	Surveillance is not useful if not a surgical candidate.
Q22	Pancreatic cyst with high CEA, low glucose, and KRAS mutation	High-risk mucinous cyst → Surgical evaluation	High-risk features mandate escalation.
Q23	Mucinous cyst in 45-year-old woman in the tail	Mucinous cystic neoplasm (MCN)	MCN: women, body/tail, no duct communication.
Q24	Cystic pancreatic lesion with salt-and-pepper chromatin and chromogranin positive	Cystic neuroendocrine tumor	Cystic NET: salt-and-pepper chromatin and neuroendocrine markers.
Q25	Chronic pancreatitis; CT nondiagnostic	Endoscopic ultrasound (EUS)	EUS detects changes when CT is unrevealing.
Q27	Pancreatic insufficiency due to chronic pancreatitis	PERT 40,000–50,000 USP dose with meals	Adequate PERT dosing improves steatorrhea and nutrition.
Q28	28-year-old with idiopathic CP	Genetic testing	Genetic testing in young idiopathic pancreatitis.
Q29	Chronic pancreatitis with severe pain and no ductal target	Celiac plexus block	Block when pain refractory and no ductal target.
Q42	Pancreatic head mass with obstructive jaundice	Tissue diagnosis + biliary drainage	Obstruction needs diagnosis and drainage for management pathway.
Q44	Strong family history of pancreatic cancer (≥2 first-degree relatives)	Genetic counseling and germline testing	Genetic testing before planning surveillance.
Q47	Pancreatic NET with liver metastases	Somatostatin analog therapy	Somatostatin analogs for functional and many nonfunctional NETs.
Q49	Pancreatic cancer planned for neoadjuvant therapy	Tissue confirmation and biliary drainage if needed	Neoadjuvant pathways require tissue and drainage when obstructed.
Q50	Walled-off necrosis with persistent symptoms after drainage	Endoscopic necrosectomy (step-up)	Step-up necrosectomy when symptoms persist.

CHAPTER 4 — DISEASES OF THE BILIARY TRACT

SECTION I - EXAM PEARL NOTES

1. RISK-STRATIFIED CBD STONES, CHOLANGITIS, AND ALTERED ANATOMY

INTERMEDIATE-RISK CHOLEDOCHOLITHIASIS (AND GALLSTONE PANCREATITIS)

- Intermediate-risk choledocholithiasis is confirmed with **MRCP or EUS** before ERCP.
- DDSEP 10 applies the same logic to **gallstone pancreatitis** with intermediate retained-stone probability.

ACUTE CHOLANGITIS AND DECOMPRESSION

- Acute cholangitis generally needs **urgent biliary decompression.**
- If the patient has prohibitive anesthesia risk or is cardiopulmonary unstable, decompression with **percutaneous transhepatic biliary drainage** is preferred.

ROUX-EN-Y GASTRIC BYPASS: ERCP OPTIONS

- Options: **balloon-enteroscopy-assisted ERCP,** lap-assisted ERCP, and EDGE.
- DDSEP 10 selected balloon-enteroscopy-assisted ERCP as the **safest** listed option despite lower success.

MEMORY

INTERMEDIATE STONE = IMAGE FIRST

UNSTABLE FOR ERCP = DRAIN PERCUTANEOUSLY

2. POSTOPERATIVE, TRANSPLANT, AND STENT PROBLEMS

POST-LIVER-TRANSPLANT ANASTOMOTIC STRICTURES AND BILE LEAKS

- Accessible post-liver-transplant anastomotic strictures and bile leaks are usually treated by **ERCP** with sphincterotomy and plastic stenting.

LATE POST-TRANSPLANT STRICTURES

- Late post-transplant anastomotic strictures are commonly **endoscopic.**
- Non-anastomotic multifocal strictures should raise **vascular ischemic** concern.

OCCLUDED PLASTIC BILIARY STENT IN PANCREATIC CANCER

- Occluded plastic biliary stent during neoadjuvant pancreatic cancer care favors exchange to a **self-expanding metal stent** for more durable drainage.

HEMOBILIA WITH WARFARIN COAGULOPATHY

- Stable hemobilia with severe warfarin coagulopathy should be evaluated with **CT angiography.**
- **Correct the coagulopathy** before definitive embolization decisions.

TYPE II PERIAMPULLARY PERFORATION

- Type II periampullary perforation after sphincterotomy/ balloon dilation can be sealed with a **covered metal biliary stent** in appropriate cases.

MEMORY

TRANSPLANT PROBLEMS = ERCP FIRST

OCCLUDED PLASTIC STENT = EXCHANGE TO METAL STENT

HEMOBILIA = CTA + CORRECT COAGULOPATHY

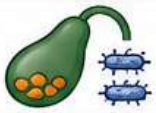
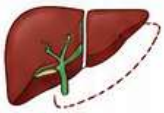
3. AMPULLARY, HILAR, AND INDETERMINATE BILIARY LESIONS

	**ATYPICAL OR ENLARGED AMPULLA**	• An atypical or enlarged ampulla should be sampled with **cold forceps biopsy** before ablation, ampullectomy, or surgery.
	**POST-AMPULECTOMY: PANCREATIC DUCT STENTING**	• After endoscopic ampullectomy, prophylactic **pancreatic duct stenting** reduces post-ampullectomy pancreatitis risk.
	**PERIHILAR CHOLANGIOCARCINOMA WORK-UP**	• Work-up should **avoid tumor seeding**. • Combine ductal brush cytology with **drainage** of adequate functional liver sectors.
	**INDETERMINATE CBD STRICTURE**	• After repeated negative brushings, escalate to **cholangioscopy** with targeted biopsies.
	**FAP WITH HIGH-GRADE AMPULLARY DISEASE**	• FAP with high-grade ampullary disease plus severe duodenal polyposis may require surgical referral for **pancreaticoduodenectomy-level planning**.

MEMORY

- ✅ AMPULLA = BIOPSY FIRST
- ✅ POST-AMPULECTOMY = PD STENT
- ✅ HILAR CCA = AVOID SEEDING + DRAIN ADEQUATE SECTORS
- ✅ INDETERMINATE STRICTURE = CHOLANGIOSCOPY
- ✅ FAP + HIGH-GRADE AMPULLARY DISEASE = SURGICAL REFERRAL

4. SPECIAL BILIARY SYNDROMES AND EXAM TRAPS

	**CHRONIC SALMONELLA TYPHI CARRIAGE**	• Salmonella typhi persists in gallbladder stones/biofilm and may require **cholecystectomy** for eradication.
	**PORTAL BILIOPATHY**	• Caused by cavernous portal collaterals compressing the bile duct. • **Portal decompression** (e.g., TIPS) may treat the **mechanism** better than repeated biliary stenting alone.
	**LOBE-LIMITED RECURRENT PYOGENIC CHOLANGITIS**	• Lobe-limited disease with atrophy or dominant stricture favors **hepatic resection**.
	**BILIARY ASCARIASIS**	• Ascaris can obstruct the bile duct or pancreatic duct. • **Stool microscopy for ova** supports the diagnosis.
	**PNEUMOBILIA AND EXAM TRAPS**	• Pneumobilia alone after prior instrumentation is not an ERCP indication when the true problem is **shock/ congestive hepatopathy**. • Nonurgent ERCP also requires **valid consent** after sedation has cleared.

MEMORY

- ➔ TYPHOID CARRIAGE → CHOLECYSTECTOMY
- ➔ PORTAL BILIOPATHY → TIPS > REPEATED STENTS
- ➔ LOBE-LIMITED RPCH → RESECTION
- ➔ ASCARIASIS → STOOL OVA
- ➔ PNEUMOBILIA NOT AN INDICATION; CONSENT AFTER SEDATION

SECTION II – DDSEP SCENARIO-BASED EXAM TABLE

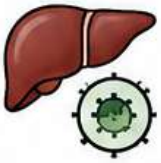
DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q1	Intermediate-risk choledocholithiasis	MRCP	Intermediate probability → confirm with MRCP/EUS before ERCP.
Q3	Perihilar CCA with sectoral obstruction	ERCP brush cytology + plastic stents into ≥2 sectors	Avoid seeding; drain adequate functional liver.
Q5	Living-donor LT anastomotic stricture + bile leak	Biliary sphincterotomy + plastic stent	Accessible post-LT leak/stricture is usually treated endoscopically.
Q6	Incidental atypical/enlarged ampulla	Cold forceps biopsies	Biopsy ampullary lesions before definitive therapy.
Q7	Intraductal papillary neoplasm of bile duct with carcinoma	Surgical bile duct resection/ liver dissection ± hepatectomy	IPNB is a biliary premalignant/malignant papillary lesion.
Q8	Post-cholecystectomy biliary-type pain, normal labs/imaging	Diagnostic upper endoscopy	Pain alone is not an ERCP indication; exclude nonbiliary causes.
Q9	Preventing pancreatitis after endoscopic ampullectomy	Pancreatic duct stent after resection	Ampullectomy has high PEP risk; prophylactic PD stent helps.
Q10	Stable hemobilia with severe warfarin coagulopathy	CT angiography + correct coagulopathy	Stable hemobilia: image and correct coagulopathy before embolization.
Q11	Chronic Salmonella typhi carrier with gallstones	Cholecystectomy	S. typhi persists in gallbladder biofilm/stones.
Q12	ERCP after Roux-en-Y gastric bypass	Balloon-enteroscopy-assisted ERCP	Safest approach among options but lower success than EDE/lap-assisted ERCP.
Q15	Biliary ascariasis diagnosis	Stool microscopy for ova	Ascaris can obstruct biliary/pancreatic ducts.
Q19	Portal biliopathy with biliary obstruction	TIPS	Portal decompression treats collateral compression.
Q25	Recurrent pyogenic cholangitis limited to atrophic left lobe	Left hepatectomy	Lobe-limited RPC with atrophy/dominant stricture favors resection.
Q27	UC with cholestatic liver enzymes and normal ultrasound	MRCP	MRCP is first-line noninvasive test for suspected PSC.

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q28	Anomalous pancreaticobiliary junction with common-channel obstruction	Pancreatic sphincterotomy + stent	APBJ can cause pancreatitis/obstruction at the common channel.
Q29	Gallstone pancreatitis, intermediate retained CBD stone risk	MRCP	Intermediate-risk CBD stone → MRCP/EUS, not direct ERCP.
Q30	Occluded plastic biliary stent during neoadjuvant pancreatic cancer care	ERCP with metal stent	Longer preoperative drainage favors metal stent.
Q32	FAP ampullary HGD/intramucosal cancer + severe duodenal polyposis	Surgical referral for possible Whipple	Advanced ampullary/duodenal FAP disease may need pancreaticoduodenectomy.
Q38	Late post-LT anastomotic biliary stricture	ERCP with stent placement	Anastomotic LT strictures are usually endoscopic.
Q43	Pneumobilia with shock/CHF and ischemic hepatitis pattern	Supportive cardiac care	Treat the shock/congestive hepatopathy; pneumobilia alone is not ERCP indication.
Q45	ERCP consent after recent anesthesia/somnolence	Wait until next day and consent patient	Nonurgent procedure requires valid consent.
Q47	Type II periampullary perforation after sphincterotomy/dilation	Covered metal biliary stent	Covered SEMS can seal periampullary perforation.
Q48	Indeterminate CBD stricture after negative brushings	Cholangioscopy with bile duct biopsies	Direct visualization with targeted biopsies increases yield.
Q50	Acute cholangitis in unstable severe cardiomyopathy patient	Percutaneous transhepatic biliary drain	If ERCP/anesthesia risk is prohibitive, PTBD can decompress.

CHAPTER 5 — VIRAL HEPATITIS

SECTION I – EXAM PEARL NOTES

1. HCV THERAPY EXPANSION: PREGNANCY, PWID, AND TRANSPLANT TRANSMISSION

 **HCV IN PREGNANCY**	• HCV in pregnancy is **not treated solely** to prevent vertical transmission. • **Avoid invasive fetal scalp monitoring** and other avoidable fetal blood exposure when the mother is viremic.
 **PEOPLE WHO INJECT DRUGS (PWID)**	• Ongoing injection drug use is **not a contraindication** to DAA therapy. • After SVR, patients with ongoing exposure need **HCV RNA surveillance** because antibody remains positive.
 **HCV-VIREMIC DONOR ORGANS**	• HCV-viremic donor organs can be used with **immediate prophylactic or preemptive DAA** therapy after transplant. • Regimen choice depends on organ, timing, cirrhosis status, and drug–drug interactions.
 **FOLLOW-UP AFTER SVR**	• Noncirrhotic patients after SVR **do not need** routine liver surveillance. • Cirrhosis or ongoing exposure changes follow-up plans.

MEMORY

TREAT PWID

MONITOR RNA FOR REINFECTION

VIREMIC DONOR ORGAN = TREAT EARLY

2. DAA SELECTION, RESISTANCE, AND INTERACTIONS

 **STRONG ENZYME INDUCERS**	• Phenytoin and other strong enzyme inducers can make DAA therapy **ineffective or unsafe**. • Observation or changing interacting therapy may be safer than a compromised regimen.
 **CYCLOSPORINE INTERACTION**	• Cyclosporine can **increase glecaprevir exposure**. • Transplant drug interactions can determine the DAA regimen.
 **RIBAVIRIN TOXICITY**	• Ribavirin causes **hemolytic anemia**. • Remains a dose-limiting toxicity in selected decompensated or difficult-to-treat cases.
 **GENOTYPE 3 CIRRHOSIS RESISTANCE / RETREATMENT**	• Genotype 3 cirrhosis with resistance/retreatment concern may require **sofosbuvir/velpatasvir/voxilaprevir**. • Decompensated cirrhosis **avoids protease-inhibitor regimens**.
 **PEDIATRIC / ADOLESCENT HCV TREATMENT**	• DAA therapy is now available for children and adolescents. • **Ledipasvir/sofosbuvir** is an option in pediatric/adolescent HCV per DDSEP 10 scenario.

MEMORY PEARLS

CHECK DRUG INTERACTIONS

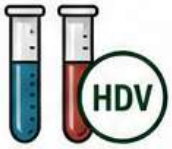
RIBAVIRIN = HEMOLYTIC ANEMIA

G3 CIRRHOSIS = SOF/VEL/VOX IF RETREATMENT

DECOMPENSATED CIRRHOSIS = AVOID PROTEASE INHIBITORS

DAAS ARE NOW APPROVED IN CHILDREN

3. HBV, HDV, AND REACTIVATION PREVENTION

 **HBV COINFECTION BEFORE HCV DAA**	• Active HBV coinfection should be **treated or protected before** starting HCV DAA therapy. • This prevents HBV flare/reactivation during or after DAA therapy.
 **HBV BREAKTHROUGH ON ENTECAVIR**	• HBV breakthrough on entecavir is often **nonadherence** before resistance. • Check adherence carefully before changing therapy.
 **SCREEN FOR HDV IN AT-RISK HBV PATIENTS**	• HBV patients with injection or sexual exposure risk should be **screened for HDV**. • HBV/HDV coinfection increases disease severity and strengthens the need for **HCC surveillance**.
 **FAMILY HISTORY OF HCC IN HBV**	• Family history of HCC can **drive HBV surveillance** even when cirrhosis alone may not mandate it. • Individualize screening based on overall risk.
 **VACCINATION & HEMODIALYSIS**	• Hemodialysis patients require **special HBV vaccine dosing/strategy** to achieve adequate response. • Susceptible HCV/HIV patients should receive **HBV vaccination**.

 **MEMORY PEARLS**

 PROTECT HBV BEFORE HCV DAA

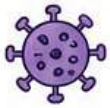
 CHECK ADHERENCE BEFORE SWITCH

 SCREEN HDV IN AT-RISK HBV

 FAMILY HISTORY INFORMS SURVEILLANCE

 SPECIAL VACCINE STRATEGY IN HD

4. HAV, HEV, CMV, EBV, COVID, AND EXTRAHEPATIC HCV

 **FALSE-POSITIVE HAV IgM**	• False-positive HAV IgM can mislead chronic liver test evaluation. • If the clinical course does not fit acute HAV, **investigate alternatives** (e.g., iron overload, autoimmune, etc.).
 **CHOLESTATIC HAV**	• Cholestatic HAV can cause prolonged pruritus. • Treat symptomatically with **cholestyramine**. • Improving HAV can be **monitored** even if liver tests lag behind clinical recovery.
 **CHRONIC HEV AFTER RENAL TRANSPLANT**	• Chronic HEV after renal transplant is first approached by **reducing immunosuppression** if feasible.
 **CMV AFTER LIVER TRANSPLANT**	• CMV mismatch (D+/R−) with diarrhea or hepatitis after LT should trigger **CMV viral-load testing**. • CMV can also cause **heterophile-negative mononucleosis-like hepatitis**.
 **EBV HEPATITIS**	• EBV hepatitis is usually self-limited. • **Observe** when the patient is stable and improving.
 **COVID-ASSOCIATED LIVER ENZYME ABNORMALITIES**	• Mild liver enzyme elevation in COVID is common and transient. • **Observe** if stable; search for alternative causes if severe or persistent.
 **EXTRAHEPATIC HCV: B-CELL LYMPHOMA**	• HCV-associated B-cell non-Hodgkin lymphoma can **improve after achieving SVR** with DAA therapy. • Treat HCV first; lymphoma may regress.

 **MEMORY PEARLS**

 QUESTECT POSITIVE HAV IgM

 CHOLESTYRAMINE FOR PRURITUS

 REDUCE IMMUNOSUPPRESSION IN CHRONIC HEV

 CHECK CMV VIRAL LOAD

 OBSERVE EBV & MILD COVID

 DAA CURE CAN IMPROVE HCV-RELATED LYMPHOMA

SECTION II – DDSEP SCENARIO-BASED EXAM TABLE

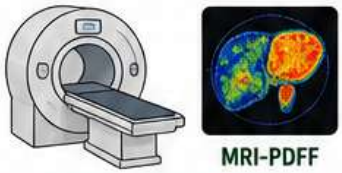
DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q1	Reducing HCV vertical transmission risk	Avoid fetal scalp monitoring	Avoid invasive fetal monitoring when HCV viremic.
Q6	HCV cirrhosis with phenytoin DAA interaction	Close observation	Strong enzyme inducers may make DAA therapy unsafe/ineffective.
Q7	HCV treatment with active HBV coinfection	Start tenofovir before DAA therapy	Prevent HBV flare/reactivation during HCV therapy.
Q8	Active injection drug use and HCV treatment	Initiate DAA therapy	Ongoing use is not a reason to withhold HCV treatment.
Q9	HCV-viremic donor heart transplant	Start DAA immediately after transplant	Use prophylactic/preemptive DAA strategy after viremic donor organ.
Q10	HCV-viremic donor liver into HCV-negative recipient	Glecaprevir/pibrentasvir for 12 weeks	Treat donor-derived HCV promptly.
Q11	Decompensated HCV cirrhosis option	Sofosbuvir/ledipasvir for 24 weeks	Decompensated disease needs specialist non-protease regimen.
Q12	Cyclosporine interaction	Increased glecaprevir level	Cyclosporine can raise glecaprevir exposure.
Q13	HCV-viremic kidney donor with severe post-transplant immune injury	Liver biopsy	Tissue may be needed to distinguish immune injury from viral/drug injury.
Q14	Ribavirin adverse effect	Hemolytic anemia	Ribavirin classically causes dose-limiting hemolysis.
Q15	HCV genotype 3 cirrhosis with resistance issue	Sofosbuvir/velpatasvir/voxilaprevir	VOX-containing triple therapy covers resistant retreatment scenarios.
Q16	HCV fibrosis progression factor	HIV coinfection	HIV accelerates HCV fibrosis progression.
Q17	Post-SVR with ongoing injection risk	Annual HCV RNA PCR	Reinfection risk requires RNA surveillance, not antibody.
Q18	HCV-associated B-cell lymphoma	Treat HCV with DAA therapy	Some HCV-associated lymphoproliferative disease improves after cure.

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q19	Noncirrhotic HCV after SVR	Follow-up as needed	No cirrhosis = no routine liver surveillance after SVR.
Q20	Pediatric/adolescent chronic HCV	Ledipasvir/sofosbuvir	Pediatric HCV has approved DAA options.
Q21	Post-LT fever/diarrhea in CMV mismatch	CMV viral load	CMV mismatch + diarrhea/hepatitis → check viral load.
Q23	EBV hepatitis	Close observation	EBV hepatitis is usually self-limited.
Q24	COVID-associated liver test abnormality	Close observation	Mild COVID liver enzyme elevation is usually monitored.
Q25	CMV mononucleosis-like hepatitis	CMV IgM	CMV can mimic heterophile-negative mono with hepatitis.
Q26	False-positive HAV IgM with chronic mild LFT elevation	Serum iron studies	Do not anchor on low-probability HAV IgM; investigate chronic liver disease.
Q29	Cholestatic/pruritic HAV course	Cholestyramine	Cholestatic HAV pruritus can be treated symptomatically.
Q30	HAV with clinical improvement but abnormal LFTs	Continue monitoring liver tests	HAV recovery may have prolonged biochemical lag.
Q31	HAV-triggered autoimmune hepatitis	Anti-smooth muscle antibody	Acute viral hepatitis can unmask autoimmune hepatitis.
Q33	HBV HCC surveillance driven by family history	Surveillance indicated due to family history	HBV HCC screening is not based only on cirrhosis.
Q34	Anti-HBc-positive patient starting adalimumab	Monitor ALT and HBV DNA every 3 months	Resolved HBV on biologic therapy needs risk-based monitoring/prophylaxis.
Q26	Chronic HEV after renal transplant	Reduce immunosuppression	First step in chronic HEV is reducing immunosuppression if feasible.
Q37	HBV vaccine dosing group	Hemodialysis patient	Dialysis patients need special HBV vaccination strategy/dosing.
Q38	Chronic HBV staging before treatment decision	Transient elastography	Fibrosis staging can drive HBV treatment decisions.
Q41	HBV/HDV coinfection surveillance	Liver ultrasound	HDV coinfection increases cirrhosis/HCC risk.
Q43	HCV/HIV patient nonimmune to HBV	HBV vaccination	Vaccinate susceptible chronic liver disease/HIV patients.
Q46	HBV viral breakthrough on entecavir	Medication noncompliance	Breakthrough is often adherence before resistance.
Q49	HBV with diabetes/possible steatosis assessment	Transient elastography with CAP	CAP helps assess steatosis with fibrosis estimate.
Q50	HBV patient with injection/sexual risk	HDV antibody	Screen HBV patients at risk for delta exposure.

CHAPTER 6 — METABOLIC, HEREDITARY, INFLAMMATORY, AND VASCULAR LIVER DISEASE

SECTION I – EXAM PEARL NOTES

1. MASLD/MASH QUANTITATIVE MANAGEMENT



MRI-PDFF: QUANTIFY LIVER FAT CHANGE

- **MRI-PDFF** is a **quantitative tool** for liver fat change, especially in trials or selected follow-up where change in steatosis is the outcome.

PRESCRIBE EXERCISE IN MINUTES

- **DDSEP** 10 explicitly tests exercise prescription:

150–300 MINUTES PER WEEK

of **moderate-intensity** activity for MASLD/MASH benefit.

BARIATRIC SURGERY VALUATION WHEN LIFESTYLE FAILS

- Eligible patients with obesity-driven NASH who fail lifestyle therapy should be referred for **bariatric surgery evaluation** rather than cycling ineffective lifestyle advice alone.

FOLLOW METABOLIC RISK & COMORBIDITIES

- Address **cardiometabolic risk factors**, diabetes, dyslipidemia, and hypertension as core management.

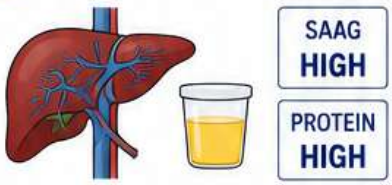
MEMORY

QUANTIFY FAT WITH MRI-PDFF

PRESCRIBE EXERCISE IN MINUTES

SURGERY REFERRAL WHEN LIFESTYLE FAILS

2. VASCULAR LIVER DISEASE AND ASCITES INTERPRETATION

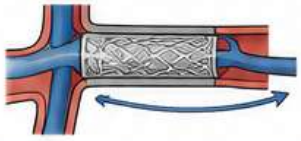


BUDD-CHIARI ASCITES: HIGH SAAG + HIGH PROTEIN

- Budd-Chiari ascites is **high SAAG** and **high protein** because hepatic venous outflow obstruction produces portal hypertension with **protein-rich sinusoidal leakage**.

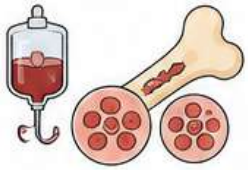
ACUTE BUDD-CHIARI: ANTICOAGULATE FIRST

- Start **anticoagulation** (unless contraindicated) as initial therapy.
- Treat underlying thrombophilia if identified.



ESCALATE TO DECOMPRESSION (TIPS) WHEN NEEDED

- Persistent ascites, pain, or liver dysfunction despite adequate anticoagulation → escalate to decompression such as **TIPS**.



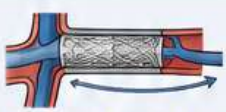
SINUSOIDAL OBSTRUCTION SYNDROME (SOS) AFTER HSCT

- Vascular-toxicity pattern with sinusoidal injury, hepatomegaly, jaundice, weight gain, and ascites.
- **Prevention** (conditioning regimen optimization, defibrotide in high-risk where indicated) and **early recognition** improve outcomes.

MEMORY

BUDD-CHIARI FLUID = HIGH SAAG + HIGH PROTEIN

ANTICOAGULATION FIRST



ANTICOAGULATION FAILURE = TIPS

CHAPTER 6 — METABOLIC, HEREDITARY, INFLAMMATORY, AND VASCULAR LIVER DISEASE

SECTION I – EXAM PEARL NOTES

3. ALCOHOL-ASSOCIATED HEPATITIS, BIOMARKERS, AND TRANSPLANT ASSESSMENT

SEVERE ALCOHOL-ASSOCIATED HEPATITIS SCORES & 28-DAY MORTALITY

- Severity scores (MDF, MELD, ABIC) estimate short-term mortality in severe alcohol-associated hepatitis.
- DDSEP 10 highlights **28-DAY MORTALITY** as the key time horizon for decision-making.

STOP STEROID IF NO RESPONSE

- Use Lille score (day 4–7) or nonresponse logic.
- **STOP** prednisolone when Lille ≥ 0.45 or no clinical improvement.
- Avoid infection risk and complications without survival benefit.

PHOSPHATIDYLETHANOL (PEth): OBJECTIVE ALCOHOL BIOMARKER

- PEth reflects **recent alcohol consumption** (within ~2–3 weeks).
- Objective, laboratory-verified, and harder to dismiss than self-report alone.

EARLY TRANSPLANT EVALUATION IN ALCOHOL HEPATITIS: BEYOND MEDICAL

- Early transplant evaluation should include psychosocial assessment.
- Absence of a **reliable spouse/partner or support system** is a major psychosocial weakness.
- Strong support increases post-transplant success and adherence.

MEMORY

SCORE FOR 28-DAY RISK

STOP STEROID IF NO RESPONSE

VERIFY ALCOHOL WITH PEth

ASSESS SUPPORT BEFORE TRANSPLANT

4. AUTOIMMUNE, DRUG-INDUCED, PEDIATRIC, AND CF LIVER DISEASE

NITROFURANTOIN: AUTOIMMUNE-LIKE DRUG-INDUCED HEPATITIS

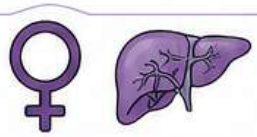
- Nitrofurantoin can cause autoimmune-like drug-induced hepatitis.
- Key **mimic** of idiopathic autoimmune hepatitis (AIH).
- Consider drug history in any suspected AIH.

TYPE 2 AUTOIMMUNE HEPATITIS IN CHILDREN

- Type 2 AIH in children is classically associated with **anti-LKM1** antibody.
- Usually presents at younger age with more acute onset.

IMMUNOSUPPRESSION IN AUTOIMMUNE HEPATITIS

- Azathioprine is first-line.
- **Mycophenolate mofetil** is the practical alternative when azathioprine cannot be used (intolerance, cytopenia, pregnancy).
- DDSEP 10 adds more pediatric/serologic discrimination to guide therapy.



CYSTIC FIBROSIS (CF)–RELATED CIRRHOSIS

- **Female sex** appears as a DDSEP 10 risk modifier for CF-related cirrhosis.
- Monitor liver disease progression closely in females with CF.

MEMORY

NITROFURANTOIN CAN IMITATE AIH

CHILD AIH TYPE 2 = ANTI-LKM1

MYCOPHENOLATE WHEN AZATHIOPRINE NOT USED

FEMALE SEX ↑ RISK IN CF-RELATED CIRRHOSIS

SECTION II – DDSEP SCENARIO-BASED EXAM TABLE

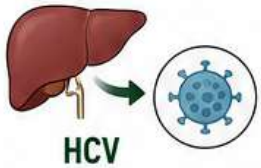
DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q5	Quantifying hepatic fat change in NASH trial/follow-up	**MRI-PDFF**	MRI-PDFF is useful for quantitative liver fat change.
Q6	Exercise prescription for NASH	**150–300 min moderate exercise weekly**	Exercise improves MASLD/MASH even without large weight loss.
Q7	NASH with obesity despite lifestyle therapy	**Refer for bariatric surgery evaluation**	Bariatric surgery can improve obesity-driven MASH in eligible patients.
Q24	Budd-Chiari ascites profile	**High SAAG, high protein**	Hepatic venous outflow obstruction gives portal hypertension with protein-rich ascites.
Q31	Acute Budd-Chiari not improving on anticoagulation	**TIPS**	Step-up Budd-Chiari therapy includes TIPS when anticoagulation fails.
Q35	Severe alcohol-associated hepatitis prognostic score purpose	**28-day mortality**	Maddrey/Lille/MELD-type tools estimate short-term mortality/response.
Q36	Alcohol-associated hepatitis not responding to prednisolone	**Stop prednisolone**	Stop steroids if nonresponse, usually by Lille assessment.
Q37	Transplant assessment in acute alcohol-associated hepatitis	**Lack of spouse/partner**	Psychosocial support is central in early transplant evaluation.
Q39	Objective alcohol-use biomarker	**Phosphatidylethanol**	PEth is a useful recent alcohol exposure biomarker.
Q40	Drug-induced autoimmune-like hepatitis	**Nitrofurantoin**	Nitrofurantoin can cause autoimmune-like DILI.
Q41	Type 2 autoimmune hepatitis in child	**Anti-LKM1 antibody**	Type 2 AIH is classically anti-LKM1-positive.
Q43	CF-related cirrhosis risk factor	**Female sex**	CF liver disease has specific demographic/genetic risk modifiers.

KEY MEMORY: Quantify fat → Exercise in minutes → Bariatric if eligible → Budd-Chiari fluid = High SAAG + High protein → Anticoagulation failure = TIPS → Score for 28-day risk → Stop steroid if no response → Verify alcohol with PEth.

CHAPTER 7 — CIRRHOSIS AND LIVER TRANSPLANTATION

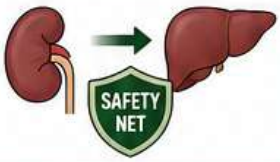
SECTION I — EXAM PEARL NOTES

1. TRANSPLANT TIMING, DONOR ACCESS, AND SAFETY-NET POLICY



HIGH-MELD HCV CANDIDATES MAY DEFER HCV TREATMENT

- If pretransplant cure **reduces access** to HCV-viremic **donors** or creates **MELD purgatory** without meaningful recovery, treatment can be deferred.
- **Prioritize transplant access** over pre-LT cure when appropriate.

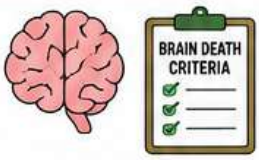


LIVER-AFTER-KIDNEY SAFETY-NET POLICY

- Provides **priority on the kidney transplant list** if kidney function worsens or fails after liver transplant.
- Protects LT recipients from irreversible kidney failure.

OLDER TRANSPLANT CANDIDATES

- Age alone is **not** a contraindication.
- Consider frailty, comorbidity, functional status, and inferior overall outcomes.



BRAIN DEATH DETERMINATION

- Requires **proper clinical context** (known cause, normothermia, no sedatives, metabolic derangements corrected).
- **Absent brainstem reflexes** are mandatory.
- **Fixed, fully dilated pupils** is a classic criterion clue.

MEMORY

CURE HCV BEFORE LT ONLY IF IT HELPS.

SAFETY-NET PROTECTS POST-LT KIDNEY FAILURE.

AGE ≠ CONTRAINDICATION; CHECK FRAILTY & COMORBIDITY.

BRAIN DEATH = PROPER CONTEXT + ABSENT BRAINSTEM REFLEXES.

2. CIRRHOSIS SYMPTOMS, ANALGESIA, AND PORTAL HYPERTENSIVE BLEEDING

COMMON CIRRHOSIS SYMPTOMS

- Muscle cramps and sleep disturbance are common in cirrhosis.
- Short-course **melatonin** (e.g., 3 mg at bedtime) may improve sleep quality in selected patients.

AVOID OPIOIDS WHEN POSSIBLE

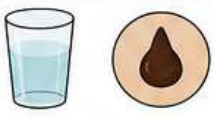
- Opioids precipitate **hepatic encephalopathy**, constipation, falls, and hospitalization.
- Use non-opioid analgesia and adjust dose for liver function.

PORTAL HYPERTENSIVE GASTROPATHY WITH IRON-DEFICIENCY ANEMIA

- Treat by lowering portal pressure with **NSBB** (e.g., carvedilol or propranolol) + **iron replacement**.
- Not an endoscopic problem unless other lesions are present.

PRIMARY PROPHYLAXIS WITH CARVEDILOL

- Start low: **6.25 mg at bedtime** (nightly).
- Titrate every 1–2 weeks to target **HR 55–60/min** or max tolerated dose (up to 12.5 mg/day if needed).
- Monitor blood pressure, renal function, and symptoms.



LACTULOSE TITRATION GOAL

- Overtreatment (4–5 watery stools/day) → dehydration, electrolyte disturbance, and can worsen encephalopathy risk.
- Target **2–3 soft stools/day**. Adjust dose to response.

MEMORY

MELATONIN MAY IMPROVE SLEEP IN CIRRHOSIS.

AVOID OPIOIDS – RISK OF HE, CONSTIPATION, FALLS.

PHG ANEMIA: NSBB + IRON REPLACEMENT.

START LOW (6.25 mg NIGHTLY) THEN TITRATE.

LACTULOSE: 2–3 SOFT STOOLS/DAY.

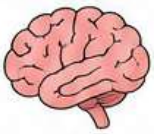
CHAPTER 7 — CIRRHOSIS AND LIVER TRANSPLANTATION

SECTION I — EXAM PEARL NOTES

3. ASCITES, HE, GAVE, HCC, AND RECOMPENSATION

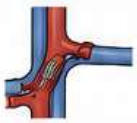

ASCITES MANAGEMENT

- Mild new ascites → start lower-dose spironolactone (e.g., 50–100 mg/day) when clinically appropriate.
- Ascites severity, **renal function**, and **electrolytes** guide escalation.



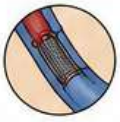

HE (HEPATIC ENCEPHALOPATHY) INITIAL APPROACH

- Minimal HE without overt episodes should **NOT** automatically receive HE medication.
- Educate, monitor, and treat precipitating factors.

REFRACTORY HE EVALUATION

- Refractory HE despite standard therapy → obtain imaging (CT/MRI) to look for **large spontaneous portosystemic shunts.**

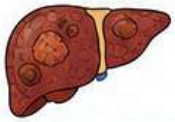



POST-TIPS REFRACTORY HE MANAGEMENT

- If medical therapy fails → consider **TIPS reduction** or **embolization.**
- Balance risk of recurrent portal hypertension.


GAVE VS PHG IN CIRRHOSIS

- Portal hypertensive gastropathy (PHG) and gastric antral vascular ectasia (GAVE) are **NOT** the same disease.
- Refractory GAVE can rarely become a transplant-level issue.



HCC MANAGEMENT IN ADVANCED DISEASE

- HCC outside treatment/transplant candidacy → move to **best supportive care.**
- Transarterial radioembolization (**TARE**) is a key locoregional option in selected candidates.

RECOMPENSATION POSSIBLE

- With etiologic control and optimal management, some patients can improve and **recompensate.** Continue surveillance and risk factor control.

MEMORY


START LOW FOR ASCITES


MINIMAL HE ≠ AUTOMATIC MEDS

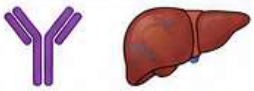

REFRACTORY HE → LOOK FOR SHUNTS


POST-TIPS HE → REDUCE OR EMBOLIZE


PHG ≠ GAVE


ADVANCED HCC → SUPPORTIVE CARE

4. POST-TRANSPLANT COMPLICATIONS AND RECURRENT DISEASE

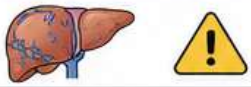


RECURRENT AUTOIMMUNE HEPATITIS AFTER LT

- Often requires **sustained/higher immunosuppression.**
- Confirm recurrence with liver **biopsy** when liver tests rise.

HBV RECURRENCE PREVENTION AFTER LT WITH DETECTABLE HBV DNA

- Requires **high-intensity prophylaxis.**
- Use **HBIG + potent nucleos(t)ide analogue** (e.g., entecavir or tenofovir).



IMMEDIATE SEVERE GRAFT FAILURE AFTER LT

- Suggests **primary graft nonfunction (PNF).**
- Early recognition → urgent retransplant evaluation.

EARLY CHOLANGITIS AFTER LT

- Common pathogen: **Enterococcus species.**
- Consider in fever, jaundice, or cholestasis soon after LT.

TACROLIMUS TOXICITIES

- **Neurotoxicity**: tremor, confusion, seizures.
- **Nephrotoxicity** and **hyperkalemia** are common.
- Check **trough level** if confusion or renal decline → adjust dose when appropriate.

RECURRENT PBC AFTER LT

- Histologic clue: **florid bile duct lesion.**

MEMORY


AIH RECURS → BIOPSY + MORE IS IMMUNOSUPPRESSION


HBV DNA+ → HBIG + POTENT NUCLEOS(T)IDE


IMMEDIATE FAILURE → THINK PNF


EARLY CHOLANGITIS → ENTEROCOCCUS


TACRO → NEURO, NEPHRO, ↑ K+


PBC RECURS → FLORID BILE DUCT LESION

SECTION II – DDSEP SCENARIO-BASED EXAM TABLE

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q3	Recurrent autoimmune hepatitis after liver transplant	**Higher baseline immunosuppression or dose reduction**	Recurrent AIH may require **more intensive immunosuppression.**
Q4	HCV therapy timing in high-MELD transplant candidate	**Defer HCV treatment if it may limit transplant access**	Treating before LT may **reduce access to HCV-viremic organs** or lead to improve outcomes.
Q6	Muscle cramps in cirrhosis	**Likely liver-related; supplements may help**	Cramps are **common in cirrhosis** and can be managed symptomatically.
Q8	Brain death criteria	**Fixed and fully dilated pupils**	Brain death requires absent brainstem reflexes in the **proper clinical context.**
Q11	Analgesia in cirrhosis	**Avoid opioids due to adverse events**	Opioids precipitate **HE, falls, constipation,** and **hospitalization.**
Q13	Dietary LT candidate	**Older recipients have inferior overall outcomes**	Chronologic age is not absolute, but **outcomes matter.**
Q14	Liver-after-kidney safety net concept	**Priority kidney listing if kidney function worsens after LT**	Safety-net policy protects patients with **post-transplant kidney failure.**
Q16	Portal hypertensive gastropathy with iron-deficiency anemia	**NSBB + iron**	PHG chronic bleeding is treated by **lowering portal pressure** and **replacing iron.**
Q19	Refractory GAVE in cirrhosis	**Liver transplantation**	Severe refractory GAVE may **improve only after LT** in selected cirrhotic patients.
Q20	Sleep disturbance in cirrhosis	**Short duration melatonin**	Melatonin may improve **sleep quality** in cirrhosis.
Q21	Bleeding gastric varix at active site	**Band ligation of bleeding area**	**When active bleeding point** is accessible, local endoscopic control may be used.
Q22	Recompensated alcohol-related liver disease	**No clear current LT indication**	Recompensation can **remove immediate transplant indication.**
Q23	Advanced HCC not transplant/treatment candidate	**Supportive care only**	Avoid futile locoregional/antineoplastic therapy in advanced HCC **outside criteria.**
Q25	Starting carvedilol for primary prophylaxis	**6.25 mg nightly then increase**	Start low and **titrate carvedilol** while monitoring.

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q26	New mild ascites	**Spironolactone 50 mg daily**	Mild ascites can start with **lower-dose aldosterone blocker.**
Q29	Lactulose overtreatment	4–5 liquid BM/day	Target **2–3 soft stools/day;** excess causes dehydration and worsens HE risk.
Q31	HCC locoregional therapy choice	**Transarterial radioembolization**	**TARE** is a key locoregional option in selected HCC.
Q33	HBV prophylaxis after LT with detectable HBV DNA	**High-intensity prophylaxis (HBIG + nucleos(t)ide analogue)**	**High-intensity HBV recurrence prophylaxis** plus potent NA therapy.
Q35	Minimal HE / no overt HE	**No HE medication needed**	Do not treat minimal HE **unless clear HE is documented.**
Q36	Refractory HE despite therapy	**Image for spontaneous portosystemic shunts**	Large **spontaneous shunts** can drive refractory HE.
Q37	Post-TIPS refractory HE	**TIPS embolization/reduction**	Severe refractory post-TIPS HE may require **shunt reduction/occlusion.**
Q38	HE classification in acute liver failure	**Type A**	Type A HE = **acute liver failure.**
Q41	Recurrent PBC after LT	**Florid bile duct lesion**	Recurrent PBC has characteristic **bile duct injury.**
Q42	Immediate post-LT graft failure	**Primary graft nonfunction**	Early severe graft failure without technical cause suggests **primary nonfunction.**
Q44	Early post-LT cholangitis organism	**Enterococcus**	**Enterococcus** is important in early post-transplant biliary infection.
Q45	Confusion in transplant patient on tacrolimus	**Check tacrolimus level**	**Tacrolimus neurotoxicity** can mimic **HE.**
Q47	Tacrolimus nephrotoxicity/hyperkalemia	**Decrease tacrolimus dose**	**Calcineurin toxicity** causes renal dysfunction and **hyperkalemia.**
Q48	Late post-LT abnormal LFTs with AIH history	**Liver biopsy**	Recurrent **AIH vs rejection often requires histology.**

CHAPTER 8 — GI AND LIVER DISEASE IN PREGNANCY

SECTION I — EXAM PEARL NOTES

1. ENDOSCOPY, LACTATION, BIOLOGICS, AND IBD PREGNANCY

	**BREASTFEEDING AFTER PROPOFOL ENDOSCOPY**	• Breastfeeding can generally **resume** after propofol endoscopy once the mother **has recovered.** • Routine pump-and-discard is **NOT required.**
	**BIOLOGICS AND BREASTFEEDING**	• Vedolizumab and many biologics are **compatible with breastfeeding.** • Continuing effective therapy supports maternal health and successful lactation.
	**CERTOLIZUMAB PLACENTAL TRANSFER**	• Certolizumab has the **least** placental transfer among anti-TNF agents. • It lacks an Fc region → minimal active transport across placenta.
	**CROHN OBSTRUCTION REQUIRING SURGERY**	• **Do NOT delay** needed surgery for obstruction. • When feasible, the **second trimester** is the preferred time for necessary surgery.
	**ACTIVE PERIANAL CROHN DISEASE**	• Active perianal Crohn disease affects delivery route planning. • It is a major reason to discuss **cesarean delivery.**

2. PREGNANCY DIAGNOSTICS: DIARRHEA, APPENDICITIS, LIVER TESTS, AND RADIATION

	**NEW DIARRHEA IN PREGNANCY**	• Still needs **infectious evaluation.** • **C. difficile toxin testing** is appropriate when risk factors and clinical presentation fit.
	**SUSPECTED APPENDICITIS IN PREGNANCY**	• **MRI** is the preferred imaging modality. • **CT** is acceptable when maternal life-threatening complications require decisive imaging (e.g., suspected hepatic hematoma).
	**LIVER TESTS IN PREGNANCY**	• ALT should remain **NORMAL** in pregnancy. • Even mild elevation (e.g., ALT 52 U/L) is **ABNORMAL** and should not be dismissed as physiologic.
	**HYPEREMESIS-RELATED LFT ABNORMALITIES**	• LFT abnormalities should **improve** when vomiting improves. • **Persistent** abnormal tests require further evaluation.
	**RADIATION EXPOSURE (CT) IN PREGNANCY**	• Late-pregnancy CT radiation risk is **LOWER** than early organogenesis risk. • Maternal stabilization and accurate diagnosis take **priority** in emergencies.

MEMORY – SECTION 1

- ☑ Resume breastfeeding after propofol once recovered.
- ☑ Vedolizumab & most biologics compatible with breastfeeding.
- ☑ Certolizumab = least placental transfer.
- ☑ Don't delay Crohn obstruction surgery; prefer **second trimester.**
- ☑ Active perianal Crohn → consider cesarean.

MEMORY – SECTION 2

- ☑ New diarrhea → evaluate infections, test **C. difficile.**
- ☑ MRI preferred for appendicitis; **CT** if life-threatening.
- ☑ ALT should be normal; mild elevation is abnormal.
- ☑ Hyperemesis LFTs improve with vomiting control.
- ☑ Late pregnancy CT risk lower; **stabilize & diagnose first.**

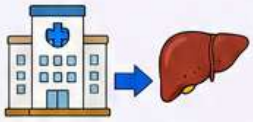
CHAPTER 8 — GI AND LIVER DISEASE IN PREGNANCY

SECTION I — EXAM PEARL NOTES

3. PREGNANCY-SPECIFIC LIVER EMERGENCIES AND VASCULAR LESIONS

HELLP SYNDROME MANAGEMENT
(Hematoma/Rupture Risk)

- Treated definitively by **DELIVERY** after maternal stabilization.
- Hepatic pain or concern for hematoma/rupture may require **urgent imaging** (US or MRI).

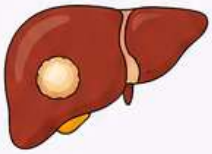


ACUTE LIVER FAILURE IN PREGNANCY

- Transfer to a **LIVER TRANSPLANT CENTER** regardless of presumed etiology.
- Do not delay transfer.

HYPEREMESIS GRAVIDARUM MANAGEMENT

- Give **THIAMINE** before carbohydrate or dextrose to prevent Wernicke encephalopathy.
- Correct electrolytes and provide supportive care.



HEPATIC ADENOMA IN PREGNANCY

- Large adenoma → **EMBOLIZE** or **RESECT** before pregnancy when possible.
- Adenomatosis during pregnancy → **SERIAL ULTRASOUND** through pregnancy and postpartum.
- Monitor for growth or hemorrhage.

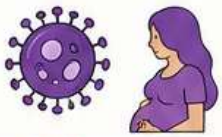
CIRRHOSIS IN PREGNANCY

- Perform **VARICEAL SCREENING**, often early in the **SECOND TRIMESTER**.
- **PROPRANOLOL** has more pregnancy experience than **CARVEDILOL** for variceal prophpylaxis.
- Avoid hypotension; monitor fetal growth.

4. CHRONIC LIVER DISEASE, VIRAL HEPATITIS, AND TRANSPLANT MEDICATIONS

HBV INFECTION IN PREGNANCY

- **TDF** (tenofovir disoproxil fumarate) is the **PREFERRED** drug for vertical-transmission prophylaxis.
- Switch from **TAF** during pregnancy when evidence of safety is the key concern.



HCV INFECTION IN PREGNANCY

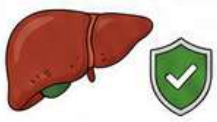
- Mode of delivery does **NOT** reduce vertical transmission.
- Avoid **ribavirin** exposure.
- Defer conception for ≥ **6 MONTHS** after ribavirin.

PBC AND PREGNANCY PRURITUS (ICP OVERLAP)

- Check **TOTAL BILE ACIDS**.
- **UDCA** is considered **SAFE** in pregnancy.
- Obeticholic acid lacks pregnancy safety evidence → **AVOID**.

HEV INFECTION IN PREGNANCY

- **HEV** can be **SEVERE** in pregnancy.
- Mother-to-child transmission can occur.
- Mild bile acid elevation alone does **NOT** explain severe hepatitis.



LIVER TRANSPLANT IN PREGNANCY

- Stable pregnancy → continue **COMPATIBLE IMMUNOSUPPRESSION**.
- Tacrolimus trough monitoring **EVERY 2–4 WEEKS** (pregnancy alters pharmacokinetics).
- Multidisciplinary care with maternal–fetal medicine and transplant team.

MEMORY PEARLS – SECTION 3

- HELLP → stabilize then DELIVER; image if pain/hematoma.
- ALF in pregnancy → transfer to transplant center.
- Thiamine BEFORE carbs in hyperemesis.
- Large adenoma → embolize/resect before pregnancy.
- Cirrhosis → early 2nd trimester variceal screening; propranolol preferred.

MEMORY PEARLS – SECTION 4

- HBV → TDF is preferred for vertical transmission prophylaxis.
- HCV → delivery route does not matter; avoid ribavirin, delay conception 6 months.
- PBC pruritus → check bile acids; UDCA safe; OCA not safe.
- HEV → can be severe; transmission possible.
- Post-LT pregnancy → continue meds; tacrolimus trough every 2–4 weeks.

SECTION II – DDSEP SCENARIO-BASED EXAM TABLE

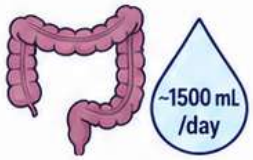
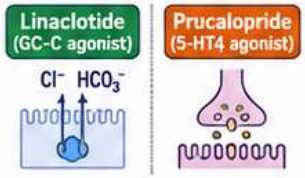
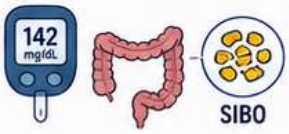
DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q3	Breastfeeding after propofol endoscopy	**Resume breastfeeding after recovery**	Propofol recovery does not usually require pump-and-discard.
Q11	Vedolizumab during breastfeeding	**Infuse vedolizumab and encourage breastfeeding**	Many biologics are compatible with breastfeeding.
Q16	Anti-TNF with least placental transfer	**Certolizumab**	Certolizumab has no minimal Fc-mediated placental transfer.
Q17	Crohn obstruction: requiring surgery in pregnancy	**Surgery in second trimester**	Necessary surgery should not be delayed; second trimester is preferred when possible.
Q18	Delivery route issue in Crohn disease	**Active perianal disease**	Active perianal Crohn favors cesarean delivery discussion.
Q21	New diarrhea in pregnancy	**Stool for C. difficile toxin**	Pregnancy does not exclude infectious diarrhea work-up.
Q22	Suspected appendicitis in pregnancy	**Abdominopelvic MRI**	MRI avoids ionizing radiation and evaluates appendix.
Q23	Liver enzymes in pregnancy: abnormal value	**ALT 52 U/L**	ALT should remain normal in pregnancy; mild elevation is abnormal.
Q25	Child IBD risk when one parent has Crohn disease	**8%**	Offspring risk is increased but not Mendelian.
Q26	HBV therapy in pregnancy: 1AF issue	**Switch to TDF**	TDF has the strongest pregnancy safety/vertical transmission evidence.
Q29	Acute liver failure in pregnancy	**Transfer to liver transplant center**	ALF in pregnancy needs transplant center care.
Q30	Suspected HELLP hepatic hematoma/complication	**CT abdomen with contrast**	Maternal life-threatening complications justify CT.
Q31	HELLP syndrome management	**Delivery**	Definitive HELLP treatment is delivery after stabilization.
Q32	Hyperemesis gravidarum	**Thiamine**	Give thiamine before carbohydrate/dextrose to prevent Wernicke.
Q33	Persistent abnormal LFTs after hyperemesis improvement	**Further testing**	Hyperemesis-related LFTs should improve with vomiting control.

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q34	Large hepatic adenoma before pregnancy	Embolization or resection before pregnancy	Large adenoma risk growth/rupture in pregnancy.
Q35	Hepatic adenomatosis during pregnancy	Ultrasound every per trimester and 3 months postpartum	Monitor adenomas throughout pregnancy and postpartum.
Q36	Cirrhosis pregnancy: variceal screening	Endoscopy early in second trimester	Screen for varices when portal hypertension risk exists.
Q37	Variceal prophylaxis in pregnancy	Propranolol instead of carvedilol	Propranolol has more pregnancy experience than carvedilol.
Q38	AHI and pregnancy flare risk	1 year remission before conception lowers postpartum flare risk	Stable remission before conception reduces flare risk.
Q39	Mycophenolate before conception	Stop ≥6 weeks before trying to conceive	Mycophenolate is teratogenic.
Q40	CT radiation counseling late pregnancy	Maternal risk after 25 weeks	Fetal radiation sensitivity is greatest early gestation.
Q41	HCV delivery counseling	Mode of delivery does not affect transmission risk	Do not perform C-section solely to prevent HCV transmission.
Q42	Ribavirin teratogenic safety	Defer conception ≥6 months after ribavirin	Ribavirin is teratogenic and requires contraception interval.
Q43	PBC pregnancy pruritus / ICP association	Serum total bile acids	Pruritus in pregnancy still requires rule out ICP.
Q44	PBC therapy in pregnancy	UDCA safe, obeticholic acid lacks safety evidence	Continue UDCA, avoid unsupported newer agents.
Q45	HEV in pregnancy	Mother-to-child transmission can occur	HEV can be severe and may transmit vertically.
Q47	Severe hepatitis not explained by ICP	Serum bile acids 18 micromol/L	Mild bile acid elevation does not explain severe hepatitis.
Q48	Pregnancy after liver transplant	No immunosuppression change needed	Stable LT pregnancy often continues compatible immunosuppression.
Q49	Tacrolimus in pregnancy	Monitor trough every 2–4 weeks	Pregnancy changes tacrolimus pharmacokinetics.

CHAPTER 9 — DIARRHEA AND CONSTIPATION

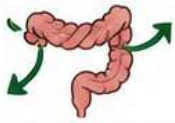
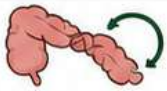
SECTION I — EXAM PEARL NOTES

1. FLUID HANDLING, NEUROLOGIC BOWEL DISEASE, AND DRUG MECHANISMS

	**FLUID ENTERS THE CECUM DAILY**	• About **1500 mL** of fluid enters the cecum daily. • Diarrhea develops when absorptive reserve is **exceeded** or secretion/motility **overwhelms** colonic salvage.
	**NEUROLOGIC DISEASE AND CONSTIPATION**	• Parkinson disease and multiple sclerosis can cause constipation through **autonomic, enteric,** or **neurogenic** bowel dysfunction.
	**DRUG MECHANISMS**	• Linaclotide activates **guanylate cyclase-C (GC-C)** on intestinal epithelium → ↑ chloride/bicarbonate secretion and ↓ visceral pain signaling. • Prucalopride is a selective **5-HT4** receptor agonist that enhances colonic motility.
	**DIABETIC DIARRHEA – MULTIPLE MECHANISMS**	• Diabetic diarrhea can reflect **autonomic/enteric neuropathy.** • The same patients may also have **SIBO** or **EPI.** Pattern recognition is key.

MEMORY PEARL – SECTION 1 | **GC-C** SECRETES (Cl^-/HCO_3^-) | **5-HT4** PROPELS (motility) | NEUROLOGIC DISEASE CAN CONSTITUTE OR DYSREGULATE

2. VOLVULUS AND CONSTIPATION SURGERY DECISION-MAKING

	**SIGMOID VOLVULUS (STABLE)**	• Initially decompress with **flexible sigmoidoscopy.** • After decompression → plan **definitive prevention** (elective sigmoid resection).
	**CECAL VOLVULUS**	• Usually surgical. • Treatment in DDSEP 10 scenario: **right hemicolectomy.**
	**COLECTOMY FOR CONSTIPATION**	• Requires **proven slow-transit** constipation. • Normal transit on wireless motility capsule argues **against colectomy.**
	**OUTCOMES AFTER COLECTOMY**	• Even properly selected slow-transit constipation patients can have persistent constipation after colectomy. • Rate of persistent symptoms ≈ **20%** in DDSEP 10 stem.

MEMORY PEARL – SECTION 2

 SIGMOID TWISTS CAN BE SCOPED

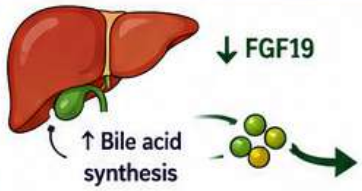
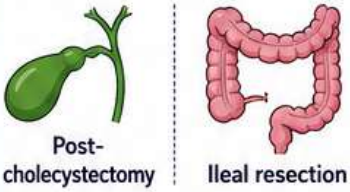
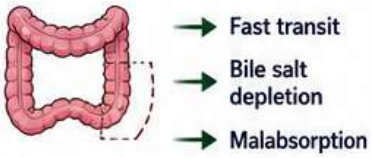
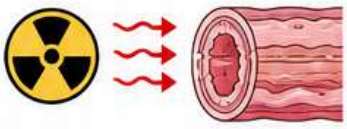
 CECAL TWISTS ARE USUALLY CUT

COLECTOMY NEEDS PROVEN SLOW TRANSIT

CHAPTER 9 — DIARRHEA AND CONSTIPATION

SECTION I — EXAM PEARL NOTES

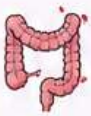
3. BILE ACID, ILEAL, SHORT-BOWEL, AND RADIATION DIARRHEA

	**BA REDUCTION AND BILE ACID DIARRHEA**	• Reduced **FGF19** → ↑ **hepatic bile acid synthesis.** • Excess bile acids reaching colon can cause **bile acid diarrhea** in an **IBS-D–like** phenotype.
	**POST-CHOLECYSTECTOMY AND ILEAL RESECTION DIARRHEA**	• Post-cholecystectomy: **continuous bile acid delivery** into colon → secretory diarrhea. • Ileal resection: **bile acid malabsorption.** • If extensive resection → **reduced bile salt pool** → fat malabsorption and diarrhea.
	**SHORT-BOWEL DIARRHEA**	• Not only fast transit. • Bile salt depletion → **steatorrhea.** • Risk of **fat-soluble vitamin deficiency** (A, D, E, K).
	**CHRONIC RADIATION ENTERITIS**	• Caused by **free radical injury → fibrosis → ischemia → obliterative arteritis.** • Explains delayed onset and progressive symptoms (months to years after radiation).
	**STOOL OSMOTIC GAP TRAP**	• Urine contamination can **falsely lower** the stool osmotic gap. • Alters measured stool electrolytes (↓ $[Na^+]$, ↓ $[K^+]$) → falsely suggests secretory diarrhea.

MEMORY PEARLS — SECTION 3


Low FGF19 → high bile acids → BA diarrhea

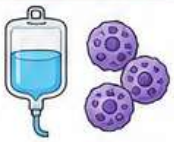
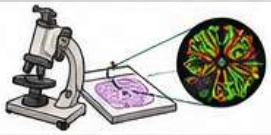

Post-CCY = continuous BA
Ileal resection = BA loss ± fat malabsorption


Short bowel → BA depletion + vitamin risk


Radiation enteritis = free radical → fibrosis → ischemia → arteritis


Urine contamination ↓ osmotic gap (falsely low)

4. SYSTEMIC AND MEDICATION-RELATED DIARRHEA TRAPS

	**POST-HSCT DIARRHEA**	• Post-hematopoietic stem cell transplant diarrhea should raise concern for **ACUTE LOWER GI GRAFT-VERSUS-HOST DISEASE (GVHD).** • Do not attribute to infection or drugs alone.
	**CHRONIC PANCREATITIS WITH DIARRHEA / STEATORRHEA**	• Consider exocrine pancreatic insufficiency **(EPI).** • Evaluate with **FECAL ELASTASE (≤200 µg/g suggests EPI).**
	**MEDICATION-RELATED DIARRHEA TRAPS**	• **Colchicine** and **GLP-1 receptor agonists** can cause diarrhea. • Stopping the offending drug or **drug holiday** may be the best diagnostic and therapeutic step.
	**GI AMYLOIDOSIS DIAGNOSIS**	• Confirm with **CONGO RED STAINING.** • **Apple-green birefringence** under polarized light is diagnostic.

MEMORY PEARLS — SECTION 4



HSCT diarrhea = think GVHD (low GI).


Chronic pancreatitis + diarrhea? Check elastase.


Colchicine & GLP-1 agonists can be culprit.


Congo red stains → green glow.

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q2	Normal colonic fluid physiology	~1500 mL enters cecum daily	The colon absorbs most fluid delivered from small bowel.
Q9	Parkinson disease GI manifestation	Constipation	Parkinson disease commonly causes constipation from autonomic/enteric dysfunction.
Q15	Linaclotide mechanism	Guanylate cyclase-C activation	Linaclotide increases intestinal secretion and reduces visceral pain signaling.
Q16	Sigmoid volvulus decompression	Flexible sigmoidoscopy	Stable sigmoid volvulus -> endoscopic detorsion/decompression.
Q18	Prucalopride mechanism	5-HT4 agonism	Prucalopride is a prokinetic serotonin 5-HT4 agonist.
Q21	Neurologic disease causing constipation	Multiple sclerosis	Neurogenic bowel dysfunction may cause constipation.
Q22	Refractory constipation before colectomy	Normal transit on wireless motility capsule argues against colectomy	Confirm true slow-transit constipation before colectomy.
Q23	Colectomy outcome in slow-transit constipation	~20% persistent constipation	Surgery helps selected patients but is not universally curative.
Q25	Cecal volvulus	Right hemicolectomy	Cecal volvulus usually needs operative management.
Q31	Falsely low stool osmotic gap	Urine contamination	Urine contamination alters stool electrolyte interpretation.
Q34	Bile acid diarrhea mechanism in IBS-D phenotype	Reduced FGF19 production	Low FGF19 increases hepatic bile acid synthesis.
Q36	Post-HSCT diarrhea	Acute lower GI GVHD	HSCT patient with diarrhea: consider lower GI graft-versus-host disease.
Q37	Chronic pancreatitis/EPI diarrhea	Fecal pancreatic elastase	Fecal elastase screens for EPI.
Q38	Diabetic diarrhea mechanism	Autonomic/enteric nervous system dysregulation	Diabetic autonomic neuropathy can cause diarrhea.
Q41	Chronic radiation enteritis mechanism	Free radical injury, fibrosis, ischemia, obliterative arteritis	Radiation enteritis is ischemic-fibrotic and progressive.
Q42	Colchicine-induced diarrhea	Stop colchicine and monitor	Medication-induced diarrhea improves after stopping the culprit.

DDSEP 10 NOVEL QUESTIONS vs DDSEP 09

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q43	GI amyloidosis diagnosis	Congo red apple-green birefringence	Amyloid deposits show apple-green birefringence.
Q44	Short bowel diarrhea mechanism	Reduced bile salt pool causing fat malabsorption	Extensive ileal loss reduces bile acid pool and causes steatorrhea.
Q47	Post-cholecystectomy diarrhea	Continuous bile acid entry into colon	Cholecystectomy can cause bile acid diarrhea.
Q49	GLP-1 agonist diarrhea	Drug holiday off exenatide	GLP-1 agonists can cause dose-related GI adverse effects.

CHAPTER 10 — NEUROGASTROENTEROLOGY AND GI MOTILITY

SECTION I — EXAM PEARL NOTES

1 GASTROPARESIS TESTING, CONFOUNDERS, AND TREATMENT

DIAGNOSTIC TESTING

- Four-hour solid-phase gastric scintigraphy is the **standard test** for gastroparesis.
- Wireless motility capsule can identify **multiregional dysmotility** but is not the classic gold standard.

- Opioids (e.g., hydromorphone)
- Anticholinergics (e.g., dicyclomine)

CONFOUNDERS – WITHHOLD BEFORE TESTING

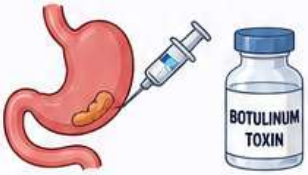
- Withhold medications that delay gastric emptying before testing.
- Especially opioids (e.g., hydromorphone) and anticholinergics (e.g., dicyclomine) which can worsen gastroparesis.

TREATMENT – METOCLOPRAMIDE

- Can help selected patients when delayed gastric emptying contributes to reflux/regurgitation.
- Use is limited by **neurologic adverse effects** (e.g., extrapyramidal symptoms, tardive dyskinesia).

POSTVIRAL GASTROPARESIS PROGNOSIS

- Postviral gastroparesis often **improves gradually over time.**
- This prognostic clue can prevent premature invasive therapy.



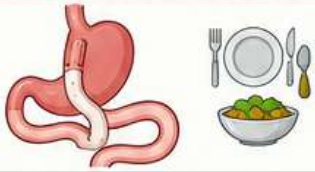
PYLORIC BOTULINUM TOXIN

- Pyloric botulinum toxin can provide short-term symptom relief in some patients.
- Effects are **temporary** and it should **not** be presented as durable disease-modifying therapy.

MEMORY PEARL – SECTION 1

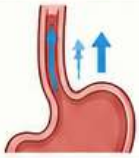
- 4h — 4-hour solid scintigraphy = standard test
- STOP — Stop opioids & anticholinergics before testing
- Metoclopramide helps but limited by neurotoxicity
- Postviral GP improves gradually
- Pyloric Botox = temporary, not curative

2 DUMPING, ACHALASIA CANCER RISK, AND INFECTIOUS MOTILITY DISEASE



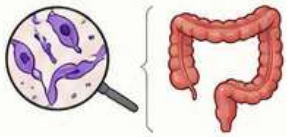
POST-RYGB DUMPING-TYPE SYMPTOMS

- Managed first with dietary measures.
- Multiple small meals.
- Lower simple carbohydrate load.
- Adequate protein, fiber, and structured meals.



ACHALASIA AND ESOPHAGEAL CANCER RISK

- Achalasia treatment (myotomy, POEM, dilation) improves emptying and symptoms.
- It does **NOT eliminate** the **increased risk** of esophageal cancer.
- Continue surveillance as indicated.



CHAGAS DISEASE (*Trypanosoma cruzi*)

- *Trypanosoma cruzi* **destroys enteric neurons** (myenteric plexus).
- Can cause achalasia-like megaesophagus and megacolon.
- Important cause of infectious motility disease.

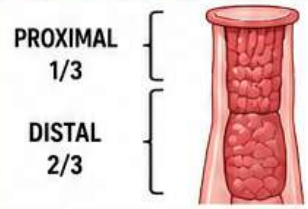
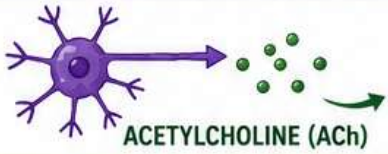
MEMORY PEARL – SECTION 2

- Treat achalasia but do NOT erase cancer risk
- Chagas disease kills enteric neurons
- Dumping: small meals, low simple carbs

CHAPTER 10 — NEUROGASTROENTEROLOGY AND GI MOTILITY

SECTION I — EXAM PEARL NOTES

3 HIGH-YIELD MOTILITY PHYSIOLOGY

	**ESOPHAGEAL MUSCLE COMPOSITION**	• The proximal third of the esophagus is **striated (skeletal)** muscle. • The distal two-thirds is **smooth** muscle.
	**ENTERIC NEUROTRANSMITTER**	• Acetylcholine (ACh) is a major **excitatory** enteric neurotransmitter.
	**ILEAL FAT AND ILEAL BRAKE**	• Fat in the ileum activates the ileal brake. • It **delays** gastric emptying and proximal small bowel transit.
	**AVERAGE TRANSIT ESTIMATES (DDSEP 10)**	• Mouth-to-cecum transit ≈ **6 hours.** • Colonic transit ≈ **36 hours.**

MEMORY PEARL – SECTION 3

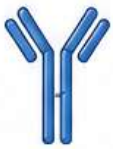
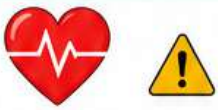
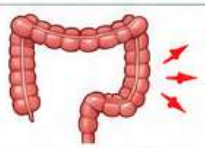
PROXIMAL ESOPHAGUS IS SKELETAL

ACh EXCITES

ILEAL FAT SAYS SLOW DOWN

MOUTH → CECUM ~6h
COLON ~36h

4 PSEUDO-OBSTRUCTION, IBS THERAPEUTICS, AND NEOSTIGMINE SAFETY

 Anti-Hu Antibody	**PARANEOPLASTIC INTESTINAL PSEUDO-OBSTRUCTION**	• Anti-Hu antibody suggests paraneoplastic intestinal pseudo-obstruction. • Due to **enteric neuropathy**, often associated with **malignancy** (e.g., small cell lung carcinoma).
	**TEGASEROD SAFETY**	• Tegaserod is restricted or contraindicated in patients with coronary artery disease (**CAD**) or **cardiovascular risk**.
	**IBS DIET THERAPEUTICS**	• Low-FODMAP elimination diet has the **strongest evidence** for IBS symptom reduction. • More effective than many other common dietary approaches.
	**ACUTE COLONIC PSEUDO-OBSTRUCTION TREATMENT**	• After conservative failure, **IV neostigmine** can be used. • Use only if **NO** perforation/ischemia and no **contraindication**.
	**NEOSTIGMINE SAFETY**	• Atropine should be available during neostigmine administration. • **Bradycardia** is the key immediate adverse event.

MEMORY PEARL – SECTION 4

Anti-Hu → think paraneoplastic pseudo-obstruction

Tegaserod: avoid in CAD / CV risk

Low-FODMAP = strongest IBS diet evidence

Neostigmine for ACPO when safe (no perforation/ ischemia)

Atropine at bedside – bradycardia risk

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q8	Post-Roux-en-Y dumping-type symptoms	**Multiple small meals daily**	Dumping improves with small, frequent, lower-carbohydrate meals.
Q10	Achalasia and cancer risk after treatment	**Risk increased and not eliminated by treatment**	Achalasia treatment improves emptying but does not normalize cancer risk.
Q14	Gastroparesis contributing to reflux symptoms	**Metoclopramide 5 mg QID**	Prokinetic therapy can help selected gastroparesis-driven reflux/regurgitation.
Q15	Gold-standard gastroparesis test	**Solid-phase gastric scintigraphy**	Four-hour solid meal scintigraphy is the standard test.
Q16	Wireless motility capsule in diabetic gastroparesis	**Globally delayed gastric emptying time**	WMC can identify multiregional dysmotility.
Q18	Drug worsening gastroparesis	**Dicyclomine**	Anticholinergics slow gastric emptying.
Q20	Postviral gastroparesis prognosis	**Symptoms likely resolve with time**	Postinfectious gastroparesis may gradually improve.
Q23	Esophageal striated muscle distribution	**Proximal third**	Proximal esophagus is striated muscle; distal is smooth muscle.
Q24	Pyloric botulinum in gastroparesis	**Temporary effects**	Botox benefit is short-lived and not durable.
Q25	Medication to withhold before gastric emptying study	**Hydromorphone**	Opioids delay gastric emptying and confound testing.
Q28	Paraneoplastic intestinal pseudo-obstruction	**Anti-Hu antibody**	Anti-Hu is linked to paraneoplastic enteric neuropathy.
Q29	Chagas achalasia/megacolon pattern	***Trypanosoma cruzi***	Chagas damages enteric neurons causing megaesophagus/megacolon.
Q30	IBS-C drug contraindicated in CAD	**Tegaserod**	Tegaserod has cardiovascular risk restrictions.
Q32	Chronic diarrhea work-up to exclude endocrine cause	**Cortisol level**	Adrenal insufficiency can mimic chronic diarrhea/weight loss.
Q37	IBS diet with strongest evidence	**Low-FODMAP elimination**	Low-FODMAP diet has the best diet evidence for IBS symptoms.
Q38	Major excitatory enteric neurotransmitter	**Acetylcholine**	ACh is a key excitatory enteric motor neurotransmitter.
Q39	Ileal brake	**Ileal fat delays gastric emptying**	Nutrients in ileum slow proximal GI transit.
Q40	Average transit times	**6 h mouth-to-cecum; 36 h colon**	Know broad normal transit estimates.
Q49	Acute colonic pseudo-obstruction treatment	**IV neostigmine**	Neostigmine treats ACPO after conservative failure/no contraindication.

DDSEP 10 NOVEL QUESTIONS vs DDSEP 09

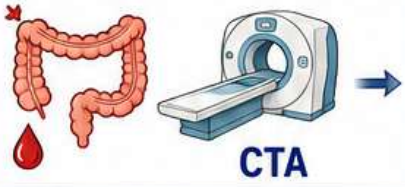
DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
	treatment		conservative failure/no contraindication.
Q50	Neostigmine adverse-effect rescue	**Atropine**	Monitor for bradycardia; keep atropine available.

CHAPTER 12 — GI BLEEDING

SECTION I — EXAM PEARL NOTES

1 LOCALIZATION AND ESCALATION IN MODERN BLEEDING ALGORITHMS

UNSTABLE HEMATOCHEZIA (BRISK BLEEDING)



- Brisk hematochezia with ongoing instability is rapidly evaluated with **CT ANGIOGRAPHY (CTA)**.
- CTA can localize active bleeding and direct **angiography** or **surgery**.

RISK SCORES GUIDE DISPOSITION

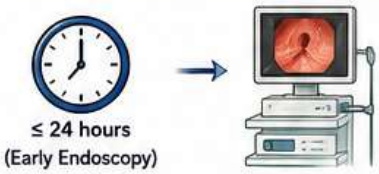
- Glasgow–Blatchford
- Rockall
- AIMS65
- Others

- Very-low-risk upper GI bleeding can be discharged with **outpatient endoscopy**.
- Risk scores are **DISPOSITION TOOLS**, not just academic calculations.

ESCALATION AFTER FAILED STANDARD HEMOSTASIS

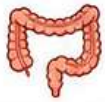
- Failed standard hemostasis (injection, thermal, clips) can be treated with an **OVER-THE-SCOPE CLIP (OTSC)**.
- Use OTSC as an escalation tool **before surgery** in selected cases.

EARLY ENDOSCOPY – IMPORTANT BUT NOT ALL-OUTCOME IMPROVING



- Early endoscopy helps diagnosis and therapy.
- It does **NOT** improve every outcome, including length of hospitalization in the DDSEP 10 trap.

MEMORY


Unstable hematochezia = CTA


Risk scores = disposition, not just numbers


Failed standard therapy = consider OTSC


Early endoscopy helps, but not all outcomes.

2 LESION-SPECIFIC ENDOSCOPIC THERAPY

DIEULAFOY LESION

- Dieulafoy lesions require **definitive mechanical or thermal** therapy.
- Epinephrine alone is **temporary**.
- **Tattoo** helps future localization.

DIVERTICULAR BLEEDING

- Treat by **clipping** the culprit diverticulum/neck.
- Epinephrine injection may be used first to slow bleeding and improve visualization. (**Temporary** measure)

RADIATION PROCTOPATHY

- Requires lesion-specific therapy.
- Argon plasma coagulation (**APC**) is the mainstay endoscopic treatment.

PROXIMAL SMALL-BOWEL ANGIOECTASIAS (JEJUNAL)

- Proximal small-bowel angioectasias require lesion-specific therapy.
- **Push enteroscopy** is useful for proximal jejunal angioectasias.

POLYPECTOMY IN GI BLEEDING CONTEXT

- **Cold snare** polypectomy is preferred for small polyps.
- Especially important when **anticoagulation** makes **thermal injury** and **delayed bleeding** undesirable.

MEMORY


Inject to see (Temporary)

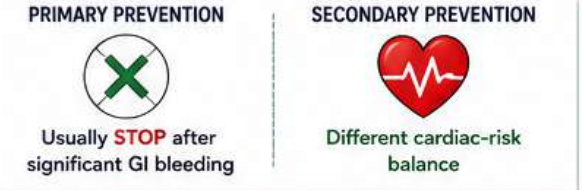

Clip / Cauterize to cure


Tattoo to localize


Cold snare small polyps

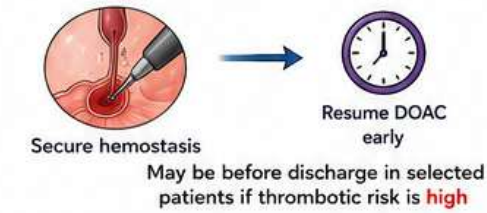
3 ANTITHROMBOTIC AND MEDICATION DECISIONS

ASPIRIN DECISIONS



- Primary-prevention aspirin is usually **stopped** after significant GI bleeding.
- Secondary prevention requires a **different cardiac-risk balance.**

DOAC RESUMPTION AFTER ULCER HEMOSTASIS



- After secure ulcer hemostasis, DOAC resumption may be **early** when **thrombotic risk is high.**
- May be before discharge in selected patients.

NSAID CHOICE

- Celecoxib is a **lower-GI-risk NSAID** option than nonselective NSAIDs.
- Use when NSAID therapy cannot be avoided.

TRANSFUSION GOAL

- Cardiovascular disease can justify a transfusion goal around hemoglobin **8 g/dL.**
- Default restrictive threshold is 7 g/dL.

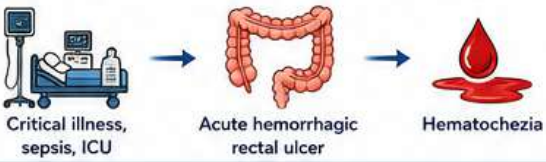
H. PYLORI–ASSOCIATED ULCER BLEEDING

- *H. pylori*–associated ulcer bleeding should remain protected with **PPI.**
- Continue PPI until **eradication is confirmed.**

MEMORY PEARL

4 SPECIAL BLEEDING PATTERNS AND RISK MODIFIERS

ACUTE HEMORRHAGIC RECTAL ULCER (AHRU)

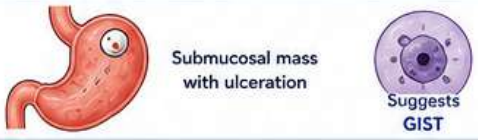


- Critical illness, sepsis, and **ICU status** can cause acute hemorrhagic rectal ulcer.
- Presents with painless hematochezia.

SYSTEMIC SCLEROSIS AND VASCULAR ECTASIA

- Thickened tight fingers point toward **systemic sclerosis.**
- Vascular ectasia/angioectasia is a common bleeding source.

GASTRIC SUBMUCOSAL MASS WITH ULCERATION



- A bleeding submucosal gastric mass with ulceration suggests **GIST.**
- Consider EUS and biopsy as appropriate.

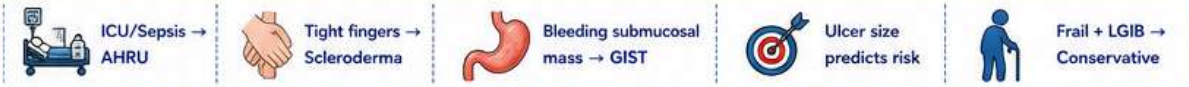
ULCER SIZE AND REBLEEDING RISK

- Ulcer size is an important rebleeding predictor.
- Clean-based ulcer is **low risk** but not zero risk in DDSEP 10 estimates.

FRAIL PATIENTS WITH LOWER GI BLEEDING

- In frail patients with lower GI bleeding, age and comorbid status may justify **conservative management.**
- Individualize; avoid over-testing.

MEMORY PEARL



SECTION II – DDSEP SCENARIO-BASED EXAM TABLE

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q5	Brisk hematochezia with instability/ongoing bleeding	**CT angiography**	CTA rapidly localizes active bleeding and guides angiography/surgery.
Q7	Dieulafoy lesion therapy	**Epinephrine + bipolar cautery + tattoo**	Injection alone is not definitive; tattoo helps future localization.
Q9	Diverticular bleeding therapy	**Epinephrine + clips along either side**	Clip the bleeding diverticulum/neck; injection alone is temporary.
Q11	Aspirin for primary prophylaxis after GI bleeding	**Avoid NSAIDs including aspirin 81 mg**	Primary-prevention aspirin is usually stopped after significant GI bleed.
Q14	Angioectasia clue	**Thickened tight skin of fingers**	Scleroderma/systemic vascular disease can associate with vascular ectasias.
Q15	Intermittent small-bowel bleeding in young patient	**Provocative angiography**	Intermittent obscure bleeding may need provocation in selected cases.
Q17	Clean-based ulcer rebleeding estimate	**10%**	Clean-base ulcers are low-risk but not zero-risk.
Q21	ICU/sepsis patient with hematochezia	**Acute hemorrhagic rectal ulcer**	Critically ill patients can bleed from acute rectal ulcers.
Q23	H. pylori ulcer bleed after discharge	**Continue PPI until eradication confirmed**	Maintain protection until cure is documented.
Q24	DOAC resumption after ulcer bleed	**Resume apixaban before discharge in 3 days**	Restart anticoagulation early when thrombotic risk is significant and hemostasis achieved.
Q25	Suspected Heyde/small-bowel bleeding	**Video capsule endoscopy**	Aortic stenosis ± bleeding suggests small-bowel angioectasias.
Q26	Conservative LGIB management decision	**Age and comorbid status**	Frai/ly/comorbidity may favor conservative strategy.
Q27	NSAID choice in high GI-risk patient	**Celecoxib**	COX-2 selective NSAID is lower GI risk than nonselective NSAIDs.
Q29	Ulcer feature predicting rebleeding	**Ulcer size**	Large ulcers have higher rebleeding risk.
Q30	Transfusion threshold with CAD	**1 unit PRBC; goal Hb 8 g/dL**	Cardiac disease may justify a higher threshold than 7 g/dL.
Q33	What early endoscopy does not improve	**Length of hospitalization**	Early EGD improves diagnosis/therapy, not every outcome.
Q40	Adherent clot management in ulcer	**No endoscopic intervention; high-dose PPI**	Management of adherent clot is nuanced; high-dose PPI may be selected.
Q41	Failed standard ulcer hemostasis	**Over-the-scope clip**	OTSC is useful for refractory peptic ulcer bleeding.

DDSEP 10 NOVEL QUESTIONS vs DSSEP 09

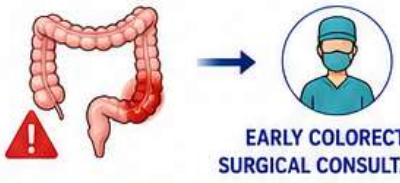
DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q45	Suspected proximal small-bowel angioectasia	**Push enteroscopy**	Push enteroscopy can treat proximal jejunal lesions.
Q47	Small polyp removal on anticoagulation	**Cold snare polypectomy**	Cold snare reduces delayed bleeding risk for small polyps.
Q49	Chronic outlet-type hematochezia, stable young patient	**Outpatient colonoscopy**	Stable chronic bleeding can be evaluated outpatient.
Q50	Bleeding submucosal gastric mass	**GIST**	GIST may present as submucosal mass with ulceration/bleeding.

CHAPTER 13 — INFLAMMATORY BOWEL DISEASE

SECTION I — EXAM PEARL NOTES

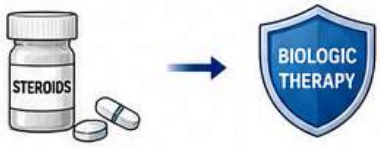
1 SEVERE, STEROID-DEPENDENT, AND POSTOPERATIVE IBD

ACUTE SEVERE ULCERATIVE COLITIS (ASUC) REFRACTORY



- ASUC that fails IV steroids and rescue biologic therapy needs **early colorectal surgical consultation.**
- Delayed surgery increases risk (toxic megacolon, perforation, colectomy morbidity).

STEROID-DEPENDENT MODERATE-SEVERE CROHN DISEASE



- Steroid dependence = need for steroids to maintain remission or relapse on taper.
- Requires a **STEROID-SPARING BIOLOGIC STRATEGY** rather than repeated steroid courses.

SEVERE CROHN FLARE AFTER MISSED BIOLOGIC INFUSIONS

- Loss of control after missed biologic doses can lead to severe flare.
- May need **inpatient IV corticosteroids** to regain control before long-term optimization.

POSTOPERATIVE CROHN RECURRENCE (MILD) ON INFLIXIMAB

- Mild endoscopic recurrence (Rutgeerts i1–i2) without clinical/radiologic activity.
- **Do NOT escalate** if patient is **stable** on infliximab and has no symptoms.

MEMORY PEARL

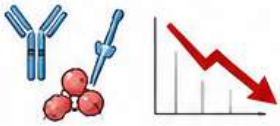
ASUC refractory = **CALL SURGERY**

Steroid dependence = **BIOLOGIC, NOT MORE STEROIDS**

Missed biologic → severe flare = IV steroids first, then optimize

2 THERAPEUTIC DRUG MONITORING, IMMUNOGENICITY, AND THIOPURINES

ANTI-INFLIXIMAB ANTIBODIES AND LOSS OF RESPONSE



- High anti-infliximab antibodies with loss of response indicate **IMMUNOGENIC FAILURE.**
- **SWITCH THERAPY** rather than simply increasing infliximab dose.

AFTER ANTI-INFLIXIMAB ANTIBODIES

Consider staying in class with a fully human anti-TNF (e.g., golimumab)

Switch out of class when failure pattern supports it

- Fully human anti-TNF (e.g., golimumab) may be considered if staying in class.
- Switch out of class (e.g., vedolizumab, ustekinumab) if failure pattern suggests immunogenicity or mechanistic failure.

THIOPURINE METABOLITES (6-TGN AND 6-MMP)

METABOLITE PROFILE	INTERPRETATION	ACTION
6-TGN LOW (< 235 pmol/8×10⁸ RBC)	Underdosing / non-adherence	Increase dose (if tolerated)
6-TGN THERAPEUTIC (235–450 pmol/8×10⁸ RBC)	Therapeutic range	Continue current dose
6-TGN LOW + 6-MMP HIGH (> 5700 pmol/8×10⁸ RBC)	Shunting / toxicity risk	Reduce dose ± allopurinol

- 6-TGN reflects efficacy (therapeutic 235–450 pmol/8×10⁸ RBC).
- High 6-MMP with low 6-TGN = shunting to 6-MMP → hepatotoxicity risk.
- Use metabolite-guided optimization when available.

THIOPURINE SAFETY MONITORING

- CBC with differential is **CORE SAFETY MONITORING** for thiopurines.
- Myelosuppression (leukopenia, neutropenia) is the feared routine toxicity.

METHOTREXATE COUNSELING

- Methotrexate is **TERATOGENIC.**
- Provide explicit **CONTRACEPTION COUNSELING** for all patients of childbearing potential.

MEMORY PEARL

High ANA to infliximab = switch (don't just increase)

6-TGN in range = stay the course

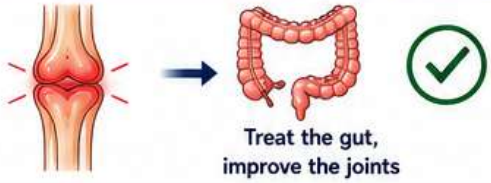
6-MMP high = shunting/toxicity

CBC diff = monitor myelosuppression

Methotrexate = teratogenic

3 EIMS, PERIANAL CROHN, AND TREATMENT SELECTION

IBD-ASSOCIATED ARTHRITIS (PERIPHERAL)



- Some peripheral arthritis parallels bowel inflammation.
- Treating underlying IBD often **improves joint symptoms.**

PYODERMA GANGRENOSUM (PG) AND SEVERE SKIN LESIONS

- PG requires urgent, multidisciplinary care (GI, dermatology, surgery, wound care).
- Trauma or debridement can worsen PG through **pathergy.**

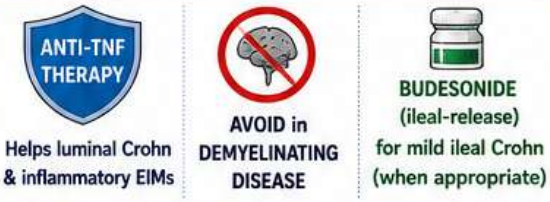
PERIANAL ABSCESS

- Abscess must be drained with EUA.
- **Do not** start or escalate biologic therapy over undrained sepsis.

PERIANAL FISTULA

- Perianal fistula is a **poor prognostic** feature.
- Antibiotics (e.g., ciprofloxacin) can reduce drainage short-term.
- Antibiotics alone are not definitive — requires combined medical ± surgical therapy.

TREATMENT SELECTION IN CROHN AND EIMS



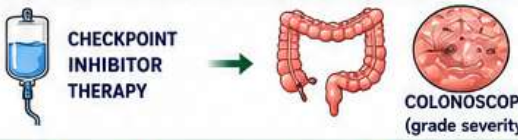
- Anti-TNF therapy helps luminal disease and inflammatory EIMs.
- **Avoid** anti-TNF in demyelinating disease.
- Mild ileal Crohn: **ileal-release budesonide** is an appropriate option.

MEMORY PEARL

- Drain abscess first before biologic.
- Fistula = poor prognosis.
- Anti-TNF helps EIMs, avoid in MS.
- PG: avoid trauma – pathergy worsens.

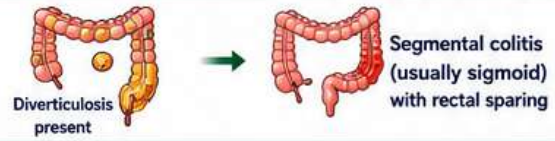
4 IBD MIMICS, SAFETY-DRIVEN CHOICES, AND OBJECTIVE INFLAMMATION

CHECKPOINT-INHIBITOR COLITIS



- Suspect by medication history.
- Colonoscopy helps grade inflammation.
- Guides steroid vs biologic decisions.

SCAD (SEGMENTAL COLITIS ASSOCIATED WITH DIVERTICULOSIS)



- Mimics IBD, but is limited to diverticular segment.
- Rectum is typically normal.
- Treat with mesalamine ± antibiotics.

SAFETY-DRIVEN CHOICE: VEDOLIZUMAB

- Gut-selective mechanism with **favorable systemic safety profile.**
- Consider when lymphoma or serious infection risk is high.

FECAL CALPROTECTIN (OBJECTIVE INFLAMMATION)

- Low: suggests IBS or functional overlap.
- High: supports inflammatory flare.
- Useful to guide treatment escalation.

NSAIDS AND DISEASE ACTIVITY

- Avoid or minimize NSAIDs (e.g., ibuprofen) in Crohn disease.
- Use alternative analgesics.

NEWLY DIAGNOSED ILEOCOLONIC CROHN

- MRE maps small bowel and extraintestinal disease.
- Helps phenotype, extent, and treatment plan.

MEMORY PEARL

- Always check drug history (ICI can cause colitis).
- SCAD = segmental colitis with diverticulosis.
- Vedolizumab = gut-selective, safer systemically.
- Calprotectin: low = IBS, high = IBD flare.
- New ileocolonic Crohn: map with MRE.

SECTION II - DDSEP SCENARIO-BASED EXAM TABLE

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q8	Severe UC not responding to steroids + infliximab	Consult colorectal surgeon	**Early surgical involvement** is essential in refractory acute severe UC.
Q10	Incidentally found mild colitis	Mesalamine 3600 mg daily	**Mild UC** colitis can be induced with oral mesalamine.
Q13	Lab monitoring in UC on sulfasalazine	Check for medication effects and FBC	Monitor **drug toxicity** and cholestatic liver disease risk.
Q16	Methotrexate reproductive counseling	Use 2 forms of birth control	MTX is **teratogenic**; contraception counseling is mandatory.
Q18	IBD-related inflammatory arthritis	Treat IBD	Some **peripheral arthritis parallels** luminal activity.
Q19	Suspected pyoderma gangrenosum/severe skin lesion	ED evaluation	PG can **worsen with trauma** and requires urgent multidisciplimary care.
Q20	Crohn disease with inflammatory EIM	Start infliximab	**Anti-TNF therapy** can treat luminal disease and inflammatory EIMs.
Q23	Diarrhea after checkpoint inhibitor exposure	Review medications	Always identify **immune-checkpoint inhibitor colitis** risk.
Q24	Suspected checkpoint-inhibitor colitis	Colonoscopy	Endoscopic assessment helps **grade and guide therapy**.
Q25	Segmected colitis with diverticulosis	SCAD	SCAD mimics IBD but is **localized to diverticular segment**.
Q27	Steroid-dependent moderate-severe Crohn disease	Start TNF inhibitor now	Steroid dependence requires **steroid-sparing biologic therapy**.
Q28	Ileal Crohn with MS/glaucoma context	Oral ileal-release budesonide	**Avoid anti-TNF** in demyelinating disease; budesonide fits mild ileal disease.
Q30	Perianal fistula without abscess	Ciprofloxacin	Antibiotics may **reduce fistula drainage** short-term.
Q33	Mild postoperative recurrence on infliximab	Continue infliximab at current dose	**Do not over-escalate** mild postoperative findings if controlled.
Q35	Low-risk Crohn remission on azathioprine	Continue azathioprine monotherapy	**Stable low-risk remission** may continue current maintenance.
Q38	Lower immunogenicity concern after anti-infliximab antibodies	Golimumab	**Fully human anti-TNF** may be less immunogenic than chimeric infliximab.
Q39	Loss of response with high infliximab antibodies	Stop infliximab and change to ustekinumab	High antibodies suggest immunogenic **failure**; switch therapy.

DDSEP 10 NOVEL QUESTIONS vs DDSEP 09

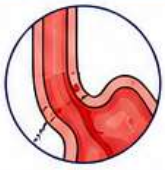
DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q40	Thiopurine routine safety monitoring	CBC with differential	**Myelosuppression** is a key thiopurine toxicity.
Q41	Thiopurine optimization	Check 6-TGN and 6-MMP	**Metabolites distinguish** underdosing, shunting, and toxicity risk.
Q42	Crohn with lymphoma-family history concern	Vedolizumab	**Gut-selective therapy** may be favored when systemic safety concerns dominate.
Q44	Symptoms may be IBS vs inflammatory flare	Fecal calprotectin	**Objective inflammation marker** helps separate IBS overlap from flare.
Q45	Medication associated with Crohn flare	Ibuprofen	**NSAIDs** can worsen IBD activity.
Q47	Severe Crohn flare after missed biologic infusions	IV prednisone	Severe flare often needs **inpatient corticosteroid induction**.
Q48	Perianal abscess in Crohn disease	Examination under anesthesia	Perianal sepsis requires **drainage/EUA**, not biologic escalation alone.
Q49	Poor prognostic Crohn feature	Perianal fistula	Perianal disease signals **aggressive Crohn phenotype**.
Q50	Newly diagnosed ileocolonic Crohn extent assessment	MR enterography	**MRE** maps small-bowel extent and complications.

CHAPTER 14 — GASTROINTESTINAL CANCERS

SECTION I — EXAM PEARL NOTES

1 ESOPHAGEAL AND GASTRIC CANCER RISK REFINEMENT

ULTRA-SHORT BARRETT'S / IRREGULAR Z-LINE



< 1 cm Barrett's (or irregular Z-line) = **VERY LOW CANCER RISK**

Should **NOT** be labeled as Barrett's esophagus. Should **NOT** enter Barrett's surveillance.

ESCC PREDISPOSITION SYNDROMES (NOT BARRETT ADENOCA RISK FACTORS)

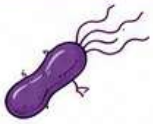
Tylosis

Plummer-Vinson syndrome

Fanconi anemia

These increase risk for ESCC, not for Barrett adenocarcinoma.

H. PYLORI CagA AND GASTRIC CANCER RISK



CagA = virulence marker associated with higher gastric cancer risk.

GIM management starts with **TESTING FOR** and **ERADICATING** ***H. pylori.***

LYNCH SYNDROME WITH GASTRIC CANCER FAMILY HISTORY

LS patients with family history of gastric cancer should have attention to ***H. pylori*** as a **MODIFIABLE RISK FACTOR.**

Eradication of ***H. pylori*** can reduce gastric cancer risk.

HEREDITARY DIFFUSE GASTRIC CANCER

Not only **CDH1** – **CTNNA1** is another associated gene.

Screen and counsel high-risk families appropriately.

MEMORY PEARL

Ultra-short Barrett's = NOT Barrett's.

ESCC risks: tylosis, PV syndrome, Fanconi anemia.

H. pylori (CagA) = gastric cancer risk.

Lynch + gastric cancer family hx = check & eradicate ***H. pylori.***

HDGC genes: **CDH1** and **CTNNA1.**

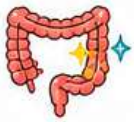
2 CRC SCREENING, COLONOSCOPY QUALITY, AND POLYP RESECTION

AVERAGE-RISK CRC SCREENING

Start at **AGE 45** for average-risk individuals.

This is the updated starting age in DDSEP 10.

BOWEL PREPARATION QUALITY MATTERS



Good bowel prep
→ better visualization
→ higher adenoma detection
→ more reliable surveillance.

Poor prep reduces adenoma detection and can lead to shorter, unnecessary follow-up.

POLYP PATHOLOGY AND SURVEILLANCE

Diminutive (≤5 mm) HYPERPLASTIC POLYP
→ Return to 10-year average-risk interval.

> 200 ADENOMAS
→ Genetic testing
→ Colectomy-level planning.

Polyp number guides risk stratification and genetics referral.

POLYP RESECTION PREFERENCE

For a 5-mm colon polyp: **COLD SNARE POLYPECTOMY** is preferred.

- Complete resection
- Safe
- Avoids cautery injury and delayed bleeding risk

ADENOMA → CANCER AND STOOL TESTING

Only a minority (10–20%) of adenomas progress to cancer.

Positive FIT-DNA = 3–5% chance of cancer.

Positive FIT-DNA **STILL** requires colonoscopy.

MEMORY PEARL

Screen average-risk people starting at age 45.

Good prep = quality colonoscopy.

Diminutive hyperplastic polyp → 10-year interval.

>200 adenomas → genetics & colectomy planning.

5-mm polyp → cold snare polypectomy.

Adenoma → cancer (10–20%); positive FIT-DNA → scope.

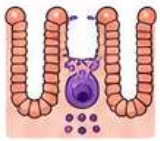
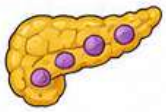
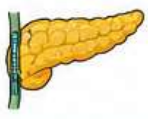
CHAPTER 14 — GASTROINTESTINAL CANCERS

SECTION I — EXAM PEARL NOTES

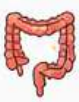
3 COLORECTAL LESION RISK AND EPIDEMIOLOGY

NONLIFTING OR DEPRESSED COLON LESIONS		• Suggest submucosal invasion risk. • Biopsy the **CENTER**. • Refer for **SURGICAL** management.	**PEARL:** Do NOT attempt routine EMR. Risk of incomplete resection and perforation.
DIET-ASSOCIATED CRC RISK FACTOR		High intake of **RED** and **PROCESSED MEAT** is associated with increased CRC risk.	**PEARL:** Diet is a modifiable CRC risk factor.
EPIDEMIOLOGY: HIGH CRC BURDEN POPULATION		**ALASKAN NATIVE** people have a high CRC burden.	**PEARL:** High-risk population appears in DDSEP 10 epidemiology answers.
CORE MEMORY PEARL		**BAD PREP** hides adenomas; **NONLIFTING / DEPRESSED** lesion may be cancer.	**MEMORY:** Quality + lesion features = cancer prevention.

4 SMALL BOWEL, BILIARY, PANCREATIC, AND NEUROENDOCRINE TUMORS

ENTEROPATHY-ASSOCIATED T-CELL LYMPHOMA		Expresses T-cell markers: **CD3** and **CD7**.	**PEARL:** T-cell phenotype supports diagnosis.
ILEAL NEUROENDOCRINE TUMORS (NETs)		• Arise from **enterochromaffin** cells in the crypts of Lieberkuhn. • Often **METASTATIC** at presentation.	**PEARL:** High rate of nodal and liver metastases.
CHOLANGIOCARCINOMA RISK FACTORS		• **Asbestos** exposure is a recognized risk factor. • Others: PSC, choledochal cysts, hepatolithiasis, liver fluke, viral hepatitis.	**PEARL:** Asbestos appears as a risk factor in DDSEP 10.
PANCREATIC CANCER SUSCEPTIBILITY		**ATM** is a pancreatic cancer susceptibility gene.	**PEARL:** Remember **ATM**.
INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM (IPMN) CLUE		Mucin **EXTRUDING** from the ampulla is the most specific clue for IPMN.	**PEARL:** Mucin at ampulla = think IPMN.
PANCREATIC NEUROENDOCRINE TUMORS (pNETs)		• Selected **METASTATIC** pNETs may benefit from surgical **resection / debulking**. • Consider in well-differentiated, low-grade disease.	**PEARL:** Surgery can improve outcomes in selected metastatic pNETs.
METASTATIC PANCREATIC ADENOCARCINOMA WITH OBSTRUCTION		Palliate with **ENDOSCOPIC** stenting (biliary or duodenal) for obstruction.	**PEARL:** Relieve obstruction; not curative.

CORE MEMORY PEARLS


Bad prep hides adenomas.


Nonlifting / depressed lesion may be cancer.


Red / processed meat ↑ CRC risk.


Alaskan Native CRC burden is high.


ATM = pancreatic cancer gene.


Mucin at ampulla = IPMN clue.

SECTION II – DDSEP SCENARIO-BASED EXAM TABLE

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q1	Most common GI cancers in US	CRC, pancreatic, liver, gastric	Know epidemiologic ranking for board-style questions.
Q4	Ultra-short Barrett's / irregular Z-line	Low risk; no surveillance	Ultra-short segments should not enter Barrett's surveillance.
Q5	ESCC predisposition syndromes	Tylosis, Plummer-Vinson, Fanconi anemia	ESCC syndromes differ from Barrett adenocarcinoma risks.
Q14	Colonoscopy quality issue	Bowel preparation	Poor prep reduces adenoma detection and exam quality.
Q15	Surveillance after diminutive hyperplastic polyp	10 years	Small (<5 mm) hyperplastic polyps generally return to average-risk interval.
Q16	Best resection for 5-mm colon polyp	Cold snare polypectomy	Cold snare is preferred for diminutive/small polyps.
Q18	Adenoma-carcinoma progression	Only minority of adenomas progress to cancer	Most adenomas never become CRC.
Q19	Positive FIT-DNA probability of CRC	5%	Positive stool DNA often means advanced neoplasia, but CRC probability is modest.
Q20	Average-risk CRC screening age	Start age 45	Current screening begins at 45 for average-risk adults.
Q21	>200 adenomas	Genetic testing and colectomy referral	Heavy polyposis needs genetics and surgical planning.
Q23	Nonlifting/depressed colon lesion	Biopsy center and refer for surgery	Nonlifting/depressed morphology suggests invasive cancer risk.
Q25	Diet-associated CRC risk	High red/processed meat	Processed/red meat is a CRC risk factor.
Q26a	CRC risk by racial/ethnic group	Alaskan Native people have higher CRC risk	Alaskan Native populations have high CRC burden.
Q26b	*H. pylori* virulence factor	CagA	CagA-positive strains carry higher gastric cancer risk.
Q30	Lynch syndrome + gastric cancer family history	Check/treat *H. pylori* if positive	Lynch gastric risk is modified by *H. pylori* and family background.
Q32	Limited complete gastric intestinal metaplasia	Test and eradicate *H. pylori* if present	GIM management begins with *H. pylori* eradication.
Q33	Hereditary diffuse gastric cancer variant beyond CDH1	CTNNA1 variant	CTNNA1 is another HDGC-associated gene.
Q35	Celiac-associated small-bowel lymphoma markers	CD3 and CD7	EATL is a T-cell lymphoma.

DDSEP 10 NOVEL QUESTIONS vs DDSEP 09

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q36	Ileal NET cell of origin	Enterochromaffin cells in crypts of Lieberkühn	Midgut NETs arise from enterochromaffin cells.
Q37	Small-bowel NET behavior	Often metastatic at presentation	Small-bowel NETs may be silent until metastatic.
Q40	Cholangiocarcinoma risk factor	Asbestos exposure	Occupational/environmental risks may appear in CCA questions.
Q41	FAP ampullary adenoma with advanced duodenal polyposis	Surgical referral	Advanced duodenal/ampullary FAP may need Whipple-level planning.
Q42	Pancreatic cancer susceptibility gene	ATM	ATM is a pancreatic cancer predisposition gene.
Q43	Metastatic pancreatic cancer with biliary obstruction	ERCP with stent placement	Palliate malignant biliary obstruction endoscopically.
Q47	Most specific IPMN feature	Mucin extruding from ampulla	Fish-mouth ampulla is classic for IPMN.
Q50	Metastatic pancreatic neuroendocrine tumor	Surgical resection/debulking	Selected metastatic pNET can benefit from cytoreduction.

CHAPTER 15 — NUTRITION, OBESITY, AND EATING DISORDERS

SECTION I — EXAM PEARL NOTES

1 OBESITY, ENDOSCOPIC BARIATRIC THERAPY, AND BARIATRIC PHYSIOLOGY

INTRAGASTRIC BALLOON THERAPY

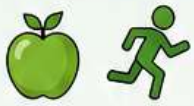
POTENTIAL ADVERSE EFFECTS

Reflux · Ulcer symptoms · Nausea · Pain

MANAGEMENT

- PPI therapy
- Supportive care as needed

AFTER REMOVAL



Structured lifestyle therapy is essential to maintain weight loss.

CANDIDATE SELECTION FOR ENDOSCOPIC BARIATRIC THERAPY

CLASS I OBESITY (BMI 30–34.9) WITH COMPLICATIONS

May qualify for selected endoscopic bariatric options (e.g., intragastric balloon).

ASIAN PATIENTS METABOLIC RISK AT LOWER BMI THRESHOLDS

- Overweight: BMI ≥ 23 kg/m²
- Obesity: BMI ≥ 25 kg/m²

Lower BMI can still be associated with high metabolic risk.

PEARL

Asian populations develop metabolic risk at lower BMI levels.

PHARMACOTHERAPY OPTIONS

LIRAGLUTIDE

- GLP-1 receptor agonist
- Promotes weight loss
- Improves glycemic control
- Cardiometabolic benefit

ORLISTAT

- Avoid in intestinal lymphangiectasia
- Avoid in fat-malabsorptive states
- Causes fat malabsorption and GI side effects

PEARL

Avoid orlistat in lymphangiectasia or fat-malabsorptive conditions.

BARIATRIC SURGERY AND PHYSIOLOGY

INCREASES GLP-1 RESPONSE

Most pronounced after BYPASS-TYPE procedures (e.g., Roux-en-Y gastric bypass).

Pre-surgery Lower GLP-1 → Post-surgery Higher GLP-1

PEARL

Bypass procedures produce the greatest boost in GLP-1.

WEIGHT REGAIN AFTER BARIATRIC PROCEDURES

Sleeve gastrectomy and other bariatric procedures can have **LATE WEIGHT REGAIN.**

- Causes: behavioral, hormonal, anatomical
- Requires: re-evaluation, nutritional counseling, physical activity, and sometimes endoscopic or surgical revision.

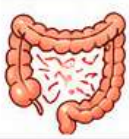
PEARL

Late weight regain is possible — long-term follow-up is essential.

MEMORY PEARLS

BALLOON NEEDS PPI + LIFESTYLE

ASIAN BMI THRESHOLD IS LOWER



BYPASS BOOSTS GLP-1

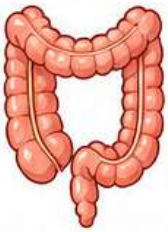
CHAPTER 15 — NUTRITION, OBESITY, AND EATING DISORDERS

SECTION I — EXAM PEARL NOTES

2 MALABSORPTION, RESECTION, AND MICRONUTRIENT PATTERNS

FAT-SOLUBLE VITAMIN DEFICIENCY

After long ileal resection or bile salt depletion



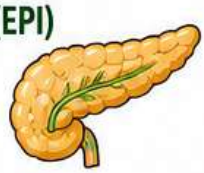
Impaired micelle formation and absorption leads to deficiency of fat-soluble vitamins.

A Vitamin A | D Vitamin D | E Vitamin E

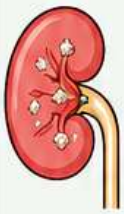
PEARL

Long ileal loss + bile salt depletion → ↓ absorption of vitamins A, D, and E.

CHRONIC PANCREATITIS / EXOCRINE PANCREATIC INSUFFICIENCY (EPI)



- Causes enteric hyperoxaluria → oxalate nephrolithiasis.
- Fat maldigestion → ↑ risk of deficiency of vitamins A, D, E, K (especially in alcoholic pancreatitis).



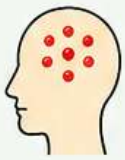
PEARL

Think "stones + stool" in EPI: fat malabsorption → oxalate stones + fat-soluble vitamin deficiency.

MICRONUTRIENT DEFICIENCY CLUES

Zn ZINC | Se SELENIUM | B_3 NIACIN (Niacin)

- **Zinc deficiency** → dysgeusia (altered taste).
- **Selenium deficiency** → can occur with long-term parenteral nutrition.
- **Niacin (B3) deficiency** → pellagra-like triad: diarrhea, flushing, neurocognitive changes.



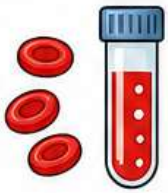
PEARL

Zn → taste
Se → PN patients
Niacin → 3 D's + dementia (pellagra).

3 D's = Diarrhea, Dermatitis (flushing), Dementia

ROUX-EN-Y GASTRIC BYPASS (RYGB) PATIENTS

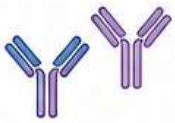
RYGB patients often need more **iron** than what standard multivitamins provide due to decreased absorption.



PEARL

Check iron studies regularly and supplement adequately in RYGB patients.

POSITIVE CELIAC SEROLOGY WITH NORMAL BIOPSIES

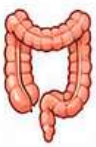


- **Do NOT** dismiss immediately.
- Review for: gluten intake, correct HLA, serology panel (tTG-IgA, EMA, DGP), and biopsy sampling adequacy.

PEARL

Confirm with repeat serology and review before excluding celiac disease.

MEMORY PEARLS



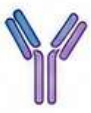
Ileal loss or bile salt depletion → ↓ fat-soluble vitamins (A, D, E, K).

EPI → oxalate stones + fat-soluble vitamin deficiency.

Zn Se B_3

Zn: taste
Se: PN patients
Niacin: 3 D's + dementia.

RYGB → needs more **iron** than standard multivitamins.



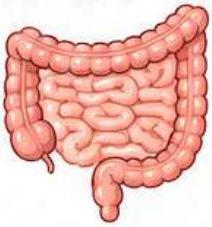
Positive celiac serology + normal biopsy → confirm, don't dismiss.

CHAPTER 15 — NUTRITION, OBESITY, AND EATING DISORDERS

SECTION I — EXAM PEARL NOTES

3 FEEDING ACCESS, SHORT BOWEL, PROTEIN LOSS, AND FISTULAS

1 SHORT BOWEL SYNDROME: FLUID & ELECTROLYTE MANAGEMENT



AVOID hypotonic free-water excess (can worsen sodium and water losses).

USE oral rehydration solution (ORS): isotonic, high-sodium, glucose-coupled solution to enhance absorption.

ORS = Isotonic + High sodium + Glucose (helps Na^+ and water absorption)

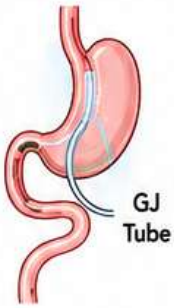
PEARL

Use ORS, not plain water.

Isotonic + high sodium + glucose = better absorption.

2 GASTROPARESIS WITH MALNUTRITION

- Severe refractory gastroparesis with malnutrition → consider **gastrojejunal (GJ) feeding.**
- Liquid metoclopramide 15–30 min before feeds can improve feeding intolerance in selected patients.

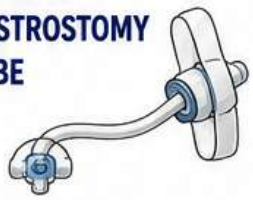


PEARL

GJ feeds bypass the stomach.

Liquid metoclopramide before feeds can help.

3 CLOGGED GASTROSTOMY TUBE

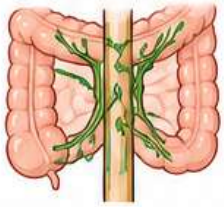


- Persistent clogged G-tube despite flushing and enzymatic attempts → may require **endoscopic tube replacement.**

PEARL

Do not delay—endoscopic replacement is often necessary.

4 PROTEIN-LOSING ENTEROPATHY (PLE)



DIAGNOSIS

- Test: fecal **alpha-1 antitrypsin** clearance (↑).

PRIMARY INTESTINAL LYMPHANGIECTASIA

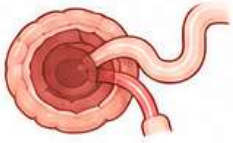
- Use medium-chain triglycerides (MCT) diet because MCTs bypass intestinal lymphatics.

PEARL

α1-antitrypsin clearance ↑ = PLE.

MCT diet reduces lymph flow and protein loss.

5 COLOCUTANEOUS FISTULA (LOW-OUTPUT, DISTAL)



- Distal low-output colocutaneous fistula can often be managed with:
  - ✓ High-protein oral supplements
  - ✓ Careful local skin care and monitoring

✗ **TPN is NOT automatic.**

PEARL

Support with oral nuttriion and local care first.

Reserve TPN for those who truly need it.

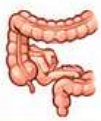
MEMORY PEARLS

Short bowel → ORS, not free water.

Severe GP → GJ feeds + liquid metoclopramide.

Clogged G-tube → endoscopic replacement.

PLE → α1-AT clearance ↑. MCT diet.



Low-output distal fistula → high-protein oral supplements, not automatic TPN.

CHAPTER 15 — NUTRITION, OBESITY, AND EATING DISORDERS

SECTION I — EXAM PEARL NOTES

4 FOOD REACTIONS, EoE DIET, EATING DISORDERS, AND NEUROLOGIC NUTRITION EMERGENCIES

GLUTEN-FREE DIET IN IBS (NOT CELIAC)

- Many IBS patients improve on a gluten-free diet due to reduction of **WHEAT FRUCTANS** (a FODMAP), not gluten.
- Explains why celiac serology and biopsies may be negative.

PEARL

IBS improvement on GFD ≠ celiac disease.

Think fructans, not gluten.

EoE DIET THERAPY

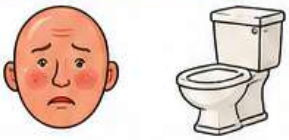
TWO-FOOD ELIMINATION DIET (FIRST-LINE STEP-UP)

Step-up alternative to immediate **SIX-FOOD ELIMINATION DIET.**

PEARL

Start with dairy + wheat elimination; escalate only if needed.

FOOD REACTIONS



- Food-triggered flushing + diarrhea/vomiting → think **MAST CELL ACTIVATION DISORDER.**
- Oral allergy syndrome (OAS): pollen-food cross-reactive IgE syndrome.

PEARL

Flushing + GI symptoms with foods → mast cell activation.

OAS = pollen-food cross-reaction.

EATING DISORDERS & NUTRITION EMERGENCIES

- Purging (vomiting) → dental enamel erosion & can cause **MALLORY-WEISS TEARS.**
- Suspected **WERNICKE ENCEPHALOPATHY** (alcohol use): give **HIGH-DOSE IV THIAMINE BEFORE** any carbohydrate.

PEARL

Give thiamine first, then carbohydrates.

Dose: 200–500 mg IV thiamine TID.

ACUTE PANCREATITIS (MILD)

- ✓ Resume **LOW-FAT SOLID DIET** immediately as tolerated.
- ✗ No need for prolonged bowel rest in mild acute pancreatitis.

PEARL

Early oral feeding **(LOW-FAT SOLID DIET)** is safe in mild AP.

MEMORY PEARLS

IBS better on GFD → fructans, not gluten.

EoE diet starts with **DAIRY + WHEAT.**

Flushing + GI symptoms → mast cell activation.

Purging → enamel erosion + Mallory-Weiss tears.

Wernicke? Thiamine FIRST, then carbs.

Mild AP → low-fat diet as tolerated.

5 RARE PHYSIOLOGY TRAPS

FAT & PYLORIC PRESSURES

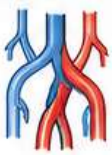
Fat intake ↑ **TONIC** and **PHASIC PYLORIC PRESSURES.**

→ **Delayed** gastric emptying and postprandial fullness, bloating, and nausea in selected motility stems.

PEARL

Fat slows gastric emptying via ↑ pyloric pressures.

VASCULAR COMPRESSION SYNDROME



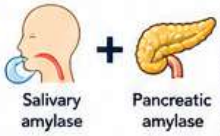
In severe weight loss or eating-disorder context, suspect vascular compression (e.g., SMA syndrome, Nutcracker syndrome).

→ **MR ANGIOGRAPHY OF ABDOMEN** may be required for diagnosis.

PEARL

Think vascular compression in severe weight loss. Use MR angiography.

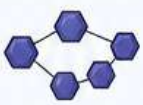
CARBOHYDRATE DIGESTION



Carbohydrate (starch) digestion depends on **SALIVARY** and **PANCREATIC AMYLASE.**

Loss of **BOTH** → impaired starch digestion and malabsorption of carbohydrates.

PEARL



Both salivary and pancreatic amylase are required for starch digestion.

MEMORY PEARLS

Fat ↑ tonic & phasic pyloric pressures → slower gastric emptying.

Severe weight loss → think vascular compression → MR angiography.

Carb digestion needs BOTH salivary + pancreatic amylase. Loss of both → starch malabsorption.

SECTION II – DDSEP SCENARIO-BASED EXAM TABLE

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q1	Intragastric balloon adverse symptoms	PPI	Balloon-related reflux/ulcer symptoms are treated with acid suppression.
Q2	Obesity drug option with constipation context	Liraglutide	GLP-1 agonists can be used for obesity with metabolic benefit.
Q3	Weight maintenance after intragastric balloon	Moderate/high-intensity lifestyle modification	Endoscopic obesity therapy requires structured lifestyle maintenance.
Q5	Class I obesity with complications, endoscopic bariatric option	Intragastric balloon	Balloons are an endoscopic option for selected lower-BMI obesity.
Q6	Complication after overseas weight-loss procedure/foreign body	Endoscopy with foreign body removal	Recognize nonstandard bariatric foreign-body complications.
Q7	Obesity drug contraindicated in intestinal lymphangiectasia	Orlistat	Avoid fat-malabsorption drugs in lymphatic/fat-malabsorptive disease.
Q10	Long ileal resection deficiency pattern	Low vitamins A, E, D	Ileal loss and bile salt depletion cause fat-soluble vitamin deficiency.
Q14	Bariatric hormonal response	Increased GLP-1	RYGB/metabolic surgery increases incretin response.
Q15	Chronic pancreatitis/EPI complication	Oxalate nephrolithiasis	Fat malabsorption increases enteric oxalate absorption.
Q16	Clogged gastrostomy tube	Endoscopy for tube replacement	Persistent tube obstruction may require endoscopic replacement.
Q18	Lower BMI threshold metabolic risk	Non-Hispanic Asian	Asian patients develop metabolic risk at lower BMI.

DDSEP 10 NOVEL QUESTIONS vs DDSEP 09

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q20	Oral rehydration in short bowel	Isotonic, high sodium:glucose ratio	SBS patients need high-sodium glucose-coupled ORS, not free water.
Q21	Malnutrition classification	Mild malnutrition, mild-moderate nutritional risk	Nutrition exams test formal malnutrition/risk classification.
Q22	Late issue after sleeve gastrectomy	Weight regain	Weight regain is a common long-term bariatric issue.
Q24	Carbohydrate digestion impairment	Reduced salivary and pancreatic amylase	Starch digestion begins with amylase.
Q25	IgE/skin-prick positive food syndrome	Oral allergy syndrome	Pollen-food cross-reactivity causes oral allergy syndrome.
Q26	Positive celiac serology with normal biopsies	Check EMA or deamidated gliadin peptide antibody	Discordant celiac testing needs confirmatory serology/review.

SECTION II – DDSEP SCENARIO-BASED EXAM TABLE

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q28	Trace element deficiency on long-term TPN	**Selenium**	Long-term PN can cause selenium deficiency.
Q29	Gluten-free diet helping IBS by FODMAP reduction	**Reduced fructans**	Wheat avoidance may help IBS through fructan reduction, not gluten itself.
Q30	Dysgeusia deficiency	**Zinc**	Zinc deficiency causes dysgeusia, poor wound healing, dermatitis/diarrhea.
Q31	Food-triggered flushing + diarrhea/vomiting	**Mast cell activation disorder**	Episodic flushing/GI symptoms after foods suggests mast-cell mediator disease.
Q35	Alcoholic pancreatitis nutritional deficiency	**Vitamin A**	Alcoholism and malabsorption predispose to fat-soluble vitamin deficiency.
Q36	Carcinoid-like diarrhea/flushing with pellagra features	**Niacin**	Tryptophan diversion can cause niacin deficiency/pellagra.
Q38	Early feeding in acute pancreatitis	**Low-fat solid diet immediately as tolerated**	Mild AP can resume oral low-fat solid diet as tolerated.
Q39	Severe refractory gastroparesis with malnutrition	**Percutaneous gastrojejunal tube**	Use jejunal feeding when oral intake fails.
Q40	Iron deficiency after Roux-en-Y	**Insufficient iron in multivitamin**	Standard multivitamins may not provide enough iron after RYGB.
Q41	Two-food elimination diet in EoE	**Dairy and wheat**	Step-up EoE diet often begins with milk and wheat elimination.
Q42	Dental enamel erosion/eating disorder complication	**Mallory-Weiss tears**	Purging causes enamel erosion and vomiting-related tears.
Q43	Protein-losing enteropathy test	**Fecal alpha-1 antitrypsin**	Stool A1AT clearance supports protein-losing enteropathy.
Q44	Primary intestinal lymphangiectasia nutrition	**Medium-chain fatty acids**	MCTs bypass intestinal lymphatics.
Q45	Fat and pyloric pressure after fundoplication	**Fat increases tonic/phasic pyloric pressures**	Fat slows gastric emptying through pyloric/duodenal feedback.
Q46	Tube feeding intolerance/prokinetic	**Liquid metoclopramide 10 mg before feeds**	Prokinetic can improve feeding tolerance in selected patients.
Q47	Suspected vascular compression syndrome/eating-disorder context	**MR angiogram abdomen**	SMA/vascular compression may require vascular imaging.
Q49	Wernicke risk in alcohol use	**IV thiamine 500 mg three times daily**	Suspected Wernicke requires high-dose IV thiamine.

DDSEP 10 NOVEL QUESTIONS vs DDSEP 09

DDSEP 10 Q	Scenario / New tested idea	Best answer	Exam pearl / mnemonic
Q50	Distal colocutaneous fistula nutrition	**High-protein oral supplements with meals**	Distal low-output fistula may be supported enterally/orally when feasible.