

UPPER GI BLEEDING

Hematemesis Coffee-ground vomitus Melena Rockall Score Endoscopic hemostasis TIPS

ACUTE UPPER GASTROINTESTINAL HEMORRHAGE

TOPIC 16

KMU MCQ PICKUP LINES

- Hematemesis + History of NSAIDs/ASPIRIN = Peptic ulcer disease (Most common cause worldwide)
- Hematemesis + Chronic liver disease signs = Esophageal varices (Most common cause in Pakistan/Asia)
- Massive hematemesis + Severe retching = Mallory-Weiss tear (Longitudinal mucosal tear at GE junction)
- Hematemesis + Weight loss + Dysphagia = Gastric carcinoma or Esophageal cancer
- Coffee-ground vomitus = Partially digested blood; indicates slower bleed than fresh red hematemesis

PRIMARY REFERENCE

Bailey & Love's Short Practice of Surgery - Acute Upper GI Bleeding, Emergency General Surgery

DEFINITION & SURGICAL IMPORTANCE

Definition: Bleeding from the esophagus, stomach, or duodenum proximal to the ligament of Treitz. **Massive UGIB:** Hemodynamic instability, hemoglobin drop $>2\text{ g/dL}$, or requiring >2 units PRBC transfusion within 24 hours. **Surgical Emergency:** 10-15% mortality; mortality increases with age, comorbidities, and delayed intervention. Unlike LGIB, melena indicates upper source in 95% cases.

ANATOMY & SURGICAL LANDMARKS

- Esophageal varices:** Form at gastroesophageal junction, 2-5cm above cardia (coronary vein tributaries)
- Duodenal ulcer bleeding:** Posterior duodenal bulb \rightarrow erosion of **gastrooduodenal artery**
- Gastric ulcer bleeding:** Lesser curvature \rightarrow **left gastric artery**
- Forrest Classification site:** Determines endoscopic intervention urgency

ETIOLOGY & RISK FACTORS

Cause	%	Key Feature
Peptic ulcer (DU > GU)	35-50%	NSAIDs, H. pylori, stress
Esophageal varices	10-30%	Portal hypertension (cirrhosis)
Mallory-Weiss tear	5-15%	Retaching, hiatal hernia
Erosive gastritis/duodenitis	5-10%	Alcohol, drugs, stress
Malignancy	2-5%	Gastric cancer, lymphoma
Dieulafoy's lesion	1-3%	Large tortuous submucosal artery
Aortoenteric fistula	Rare	Prior aortic graft, "herald bleed"

INVESTIGATIONS ALGORITHM

1 **ABG + Lactate:** Base deficit correlates with severity; lactate $>4\text{ mmol/L}$ indicates severe shock

2 **FBC:Hb** may be normal initially (hemodilution takes 4-6h); platelets $<50\text{ k}$ increases bleeding risk

3 **Coagulation profile:** INR >1.5 requires FFP; correct platelets $>50,000$

4 **Liver function:** Albumin, bilirubin for Child-Pugh score (predicts variceal bleed mortality)

5 **Urea disproportionately high:** Blood digestion in gut; BUN:Creatinine ratio $>30:1$ suggests UGIB

6 **Emergency upper GI endoscopy:** Within 24h for all, within 12h if high-risk (hemodynamic instability, continuous bleeding)

SURGICAL DECISION-MAKING

ED Triage: Two large-bore IV cannulas (14G), crystalloid bolus 1-2L, type & crossmatch 4-6 units PRBC

Unstable: ICU admission, arterial line, continuous monitoring

Transfusion trigger: Hb $<7\text{ g/dL}$ (8g/dL if cardiac disease);

DO NOT over-transfuse variceal bleed (maintain Hb 7-9 to prevent rebound portal pressure)

Emergency surgery indications: Failed endoscopic + pharmacologic therapy, continuous bleed >6 units PRBC, perforation, suspected malignancy

NAMED OPERATIONS (WITH INDICATIONS)

For Peptic Ulcer Bleeding:

- Oversewing (Graham patch for duodenal ulcer):** Duodenotomy, 3-0 silk sutures ligate gastrooduodenal artery, omental patch. Indication: Failed endoscopy, hemodynamically unstable, no expertise for angio
- Truncal vagotomy + pyloroplasty:** Rare now; only if extensive scarring
- Partial gastrectomy:** Large ulcer, suspected malignancy, rebleed after oversewing

For Variceal Bleed:

- Esophageal transection with stapler (Sugiura procedure):** Rare, salvage only
- Portosystemic shunts: TIPS (preferred), selective distal splenorenal shunt (Warren) if good liver function**

Aortoenteric fistula: Emergency laparotomy, aortic control, graft excision, extra-anatomic bypass

MANAGEMENT LADDER

Step 1 - Resuscitation: Airway protection (intubate if encephalopathy/aspiration risk), IV access x2, fluids (crystalloid initially, blood when available), correct coagulopathy

Step 2 - Pharmacologic: PPI IV bolus then infusion (80mg + 8mg/hr); Octreotide 50mcg bolus + 50mcg/hr (varices); Terlipressin 2mg IV q4h (varices, especially if cirrhosis); IV antibiotics (ceftriaxone) for variceal bleed

Step 3 - Endoscopic: Band ligation (varices), injection sclerotherapy, thermal coagulation, hemoclips, injection therapy (adrenaline 1:10,000)

Step 4 - Interventional Radiology: TIPS (Transjugular Intrahepatic Portosystemic Shunt) for refractory variceal bleed; Angiographic embolization for non-variceal bleed if surgery high-risk

Step 5 - Surgery: See below

POST-OP CARE & COMPLICATIONS

ICU monitoring: Hemoglobin q6h, continuous pulse oximetry, urine output $>0.5\text{ mL/kg/hr}$

Rebleeding: 10-20% after endoscopy; second endoscopy attempt, then angio/surgery

Stress ulcer prophylaxis: Continue PPI IV 72h, then oral

Complications: Aspiration pneumonia (most common), hepatic encephalopathy (varices), abdominal compartment syndrome (massive resuscitation), ischemic hepatitis (shock liver)

RARE BUT TESTED

Dieulafoy's lesion: Arterial malformation 2-5cm below GE junction, massive intermittent bleed, normal endoscopy between episodes \rightarrow endoscopic ultrasound or angiography for diagnosis. **Cameron lesions:** Linear erosions at diaphragm level in large hiatal hernia causing occult bleed. **Hemosuccus pancreaticus:** Bleeding from pancreatic duct (chronic pancreatitis, pseudoaneurysm).

KMU EXAM TRAPS

△ **Do NOT wait for Hb to drop before transfusing in massive bleed—transfuse based on hemodynamics**

△ **NPO status: Keep NPO until endoscopy completed (risk of aspiration)**

△ **Variceal bleed: Give prophylactic antibiotics (ceftriaxone) reduces mortality—this is standard, not optional**

△ **Balloon tamponade (Sengstaken-Blakemore tube): Bridge to definitive therapy only, not treatment—risk of esophageal necrosis/rupture**

VIVA RAPID-FIRE Q/A

Q: What is the most common cause of UGIB in Pakistan?

Esophageal varices

Q: What is the transfusion threshold in variceal bleed? Hb 7-9 g/dL (avoid over-transfusion)

Q: What medication reduces portal pressure and is first-line for variceal bleed? Octreotide or Terlipressin

Q: What is the definitive treatment for refractory variceal bleed? TIPS procedure

Q: What is the surgical procedure for bleeding duodenal ulcer?
Graham patch (duodenotomy + omental patch)

GOLDEN RULE / MNEMONIC

BLATCHFORD SCORE (Pre-endoscopic risk stratification): B - Blood urea, L - Low hemoglobin, A - Age, T - Tachycardia, C - low systolic BP, H - Heart failure, F - Fluid retention (ascites/liver disease), O - Ongoing melena/hematemesis, R - Recent hematemesis, D - Disposition (admission). Score 0 = Outpatient management possible.

ROCKALL SCORE: Age, Shock (pulse/BP), Comorbidity, Diagnosis, Endoscopic stigmata. Score >5 = High mortality (>40%).

Crafted with ❤ Noaman Khan Musakhel | Page 1 of 5

INVESTIGATIONS OF LIVER DISEASES

Hepatocellular Cholestatic Synthetic function Child-Pugh MELD Score Liver biopsy

DIAGNOSTIC WORKUP OF HEPATIC DYSFUNCTION

TOPIC 17

KMU MCQ PICKUP LINES

- ALT > AST + Jaundice = Viral hepatitis or Drug-induced (hepatocellular pattern)
- AST:ALT ratio >2:1 + High GGT = Alcoholic liver disease (specific)
- ALP >3x normal + GGT elevated = Cholestatic/obstructive pattern (do imaging first)
- Isolated bilirubin elevation + Normal LFTs = Gilbert syndrome (unconjugated) or Dubin-Johnson (conjugated)
- Low albumin + Prolonged PT = Synthetic failure (chronic liver disease/cirrhosis)

PRIMARY REFERENCE

Bailey & Love's Short Practice of Surgery - Liver Function Tests, Preoperative Assessment, Portal Hypertension

DEFINITION & SURGICAL IMPORTANCE

Definition: Systematic evaluation of hepatic synthetic, excretory, and metabolic functions to diagnose etiology, assess severity, and determine operative risk. **Surgical Importance:** Liver dysfunction dramatically increases perioperative mortality; Child-Pugh C = 40-80% mortality for major surgery. Essential for hepatobiliary surgery candidacy, variceal bleed management, and transplantation evaluation.

ANATOMY & SURGICAL LANDMARKS

Couinaud segmentation: 8 segments based on vascular supply. **Segment IV:** Quadrate lobe, between gallbladder fossa and ligamentum teres. **Caudate lobe (Segment I):** Dual blood supply, drains directly to IVC. **Glissonian pedicle:** Portal triad (hepatic artery, portal vein, bile duct) encased in fibrous sheath. **Pringle maneuver:** Occlusion of hepatoduodenal ligament for inflow control during hepatectomy (safe <30min warm ischemia).

ETIOLOGY & RISK FACTORS (PATTERN-BASED)

Pattern	Common Causes
Hepatocellular	Viral (HBV, HCV), Alcohol, Drugs (paracetamol), Autoimmune, NAFLD
Cholestatic	Stones, strictures, malignancy (cholangio, pancreatic head), PSC, PBC
Isolated hyperbilirubinemia	Gilbert, Crigler-Najjar, Dubin-Johnson,Rotor
Infiltrative	Tumors, abscesses, amyloidosis, granulomatous disease

CLASSIFICATION: SEVERITY SCORES

Child-Pugh Score (5 parameters, 3 classes):

A (5-6 points): 100% 1-year survival, low surgical risk
B (7-9 points): 80% 1-year survival, moderate risk
C (10-15 points): 45% 1-year survival, high surgical risk, contraindication for elective surgery

MELD Score (Model for End-Stage Liver Disease):

Based on Child-Pugh score, serum bilirubin, serum creatinine, and INR.

CLINICAL FEATURES + PATHOGNOMONIC SIGNS

- Jaundice:** Scleral icterus first, then skin; pruritis in cholestasis
- Ascites:** Shifting dullness, fluid thrill; SAAG >1.1g/dL = portal hypertension
- Caput medusae:** Dilated periumbilical veins (recanalized umbilical vein)
- Fetor hepaticus:** Musty breath from dimethyl sulfide (portosystemic shunting)
- Asterixis:** Flapping tremor (hepatic encephalopathy)
- Gynecomastia, testicular atrophy:** Estrogen excess (impaired metabolism)

DIFFERENTIALS (SURGICAL DISCRIMINATORS)

Obstructive vs Hepatocellular jaundice: Dark urine + pale stools + itching = obstructive (surgical). AST/ALT > ALP = hepatocellular (medical). **Imaging:** Ultrasound first—dilated ducts = obstruction (CBD stone vs stricture vs tumor).

INVESTIGATIONS ALGORITHM

Step 1 - Blood Tests (Pattern Recognition):

- Hepatocellular:** ALT, AST (released from damaged hepatocytes)
- Cholestatic:** ALP, GGT (biliary epithelium/induced by alcohol)
- Synthetic:** Albumin (half-life 20 days), PT/INR (vitamin K dependent)
- Excretory:** Bilirubin (conjugated vs unconjugated)

Step 2 - Viral Markers: HBsAg, Anti-HBc (IgM=acute), Anti-HCV

creatinine, bilirubin, INR. Used for transplant prioritization (higher score = higher priority, worse prognosis).

Step 3 - Autoimmune: ANA, SMA, anti-LKM (AIH), AMA (PBC)

Step 4 - Imaging:

- **USG Abdomen:** First-line, assess echotexture, masses, ducts, portal vein patency
- **CT/MRI:** Characterize masses, staging, vascular anatomy (hepatic veins, IVC)
- **MRCP:** Non-invasive biliary tree visualization
- **ERCP:** Diagnostic + therapeutic (stones, strictures, stenting)

Step 5 - Invasive:

- **Liver biopsy:** Gold standard for fibrosis staging, unexplained enzyme elevation; contraindicated if INR >1.5 or platelets $<50k$
- **FibroScan (Transient Elastography):** Non-invasive fibrosis assessment (stiffness correlates with fibrosis stage)

SURGICAL DECISION-MAKING

Preoperative assessment: Child-Pugh C = contraindication to elective surgery; MELD >15 = high risk. **Optimization:** Correct INR (vitamin K 10mg PO/IV 3 days, FFP if urgent), platelets $>50k$, treat ascites (spironolactone + furosemide), prevent SBP. **Operative risk:** Laparoscopic surgery preferred (less ascitic leak, faster recovery). Avoid halothane (hepatotoxic).

MANAGEMENT LADDER

Step 1 - Supportive: Nutrition (high protein except encephalopathy), avoid hepatotoxins (alcohol, NSAIDs)

Step 2 - Specific: Antivirals (tenofovir, entecavir for HBV; sofosbuvir/ledipasvir for HCV), steroids (autoimmune hepatitis, ursodeoxycholic acid (PBC))

Step 3 - Complications: Diuretics for ascites, lactulose/rifaximin for encephalopathy, band ligation/TIPS for varices

Step 4 - Surgery: Only if Child-Pugh A or optimized B; emergency surgery only if life-threatening

Step 5 - Transplant: MELD >15 or decompensated cirrhosis

RARE BUT TESTED

Wilson disease: Low ceruloplasmin, Kayser-Fleischer rings, hemolytic anemia. **Alpha-1 antitrypsin deficiency:** Panacinar emphysema + liver disease. **Hemochromatosis:** High ferritin, transferrin saturation $>45\%$, bronze diabetes, arthropathy. **Budd-Chiari syndrome:** Hepatic vein thrombosis, painful hepatomegaly, ascites; do Doppler USG, confirm with CT/MRI.

KMU EXAM TRAPS

- **ALP elevation alone does NOT mean liver disease—check GGT to confirm hepatic origin (bone also makes ALP)**
- **Prolonged PT in liver disease does NOT respond to vitamin K if hepatocellular (synthetic failure); only responds if obstructive (vitamin K malabsorption)**

NAMED OPERATIONS (WITH INDICATIONS)

Diagnostic Procedures:

- **Percutaneous liver biopsy (Menghini or Tru-cut):** Diffuse parenchymal disease staging; avoid if coagulopathy
- **Transjugular liver biopsy:** If coagulopathy present (safe route)
- **Laparoscopic liver biopsy:** Focal lesions, peritoneal assessment

Therapeutic (based on etiology):

- **ERCP with sphincterotomy:** CBD stones causing obstructive jaundice
- **Biliary stenting:** Malignant obstruction (palliative)
- **TIPS:** Refractory ascites, variceal bleed (see Topic 16)

POST-OP CARE & COMPLICATIONS

Post-biopsy: Supine position 4-6h, monitor vitals q15min x4, q30min x4, then hourly; observe for bleeding (pain, hypotension, tachycardia). **Post-operative liver patients:** Avoid hepatotoxic drugs, monitor glucose (risk of hypoglycemia), watch for hepatic encephalopathy (post-op stress precipitates), maintain electrolytes (hypokalemia worsens encephalopathy).

⚠ **Child-Pugh includes encephalopathy grade—don't forget to assess mental status**

⚠ **Low albumin is NOT acute—takes weeks to drop; normal albumin does not exclude acute liver failure**

Q: What is the first imaging in jaundiced patient? **Ultrasound (assess duct dilation)**

Q: AST:ALT ratio in alcoholic hepatitis? **>2:1 (both elevated but AST higher)**

GOLDEN RULE / MNEMONIC

LIVER FUNCTION MEMORY AID:

- S - Synthetic (Albumin, PT/INR, Clotting factors)
- E - Excretory (Bilirubin, Bile acids)
- C - Cellular integrity (ALT, AST, LDH)
- R - Regulatory (Glucose metabolism, ammonia)
- E - Enzymatic/Exocrine (ALP, GGT - biliary)
- T - Tumor markers (AFP - hepatocellular carcinoma screening)

Child-Pugh Components: ABC - Albumin, Bilirubin, Clotting (INR), EF - Encephalopathy, Fluid (ascites).

Crafted with ❤ Noaman Khan Musakhel | Page 2 of 5

VIVA RAPID-FIRE Q/A

Q: What is the most specific marker of synthetic liver function?
PT/INR (albumin has long half-life)

Q: What is the safe platelet count for liver biopsy? **>50,000/ μ L**

Q: What MELD score indicates high surgical risk? **>15 (considered for transplant listing)**

ACUTE FULMINANT HEPATITIS & ACUTE LIVER FAILURE

Encephalopathy Coagulopathy Cerebral edema King's College Criteria NAC Emergency transplant

ACUTE LIVER FAILURE: A SURGICAL EMERGENCY

TOPIC 18

KMU MCQ PICKUP LINES

- Acute hepatitis + Encephalopathy + INR >1.5 + No prior liver disease = Acute Liver Failure (ALF)
- ALF within 8 weeks of jaundice onset = Fulminant; 8-26 weeks = Subfulminant (worse prognosis)
- Paracetamol overdose + ALF = Most common cause in UK/USA; give N-acetylcysteine (NAC) within 8-24h
- ALF + Shrunken liver on USG + Rising creatinine = Poor prognosis, list for transplant immediately
- ALF patient with fever + hypotension = Spontaneous bacterial peritonitis (SBP) or sepsis (common cause of death)

PRIMARY REFERENCE

Bailey & Love's Short Practice of Surgery - Acute Liver Failure, Hepatobiliary Emergencies, Transplantation

DEFINITION & SURGICAL IMPORTANCE

Definition: Acute liver failure (ALF) = Coagulopathy (INR ≥ 1.5) + Encephalopathy + Hepatic encephalopathy + No evidence of chronic liver disease + Illness < 26 weeks. **Fulminant:** Encephalopathy within 8 weeks of jaundice onset. **Surgical Importance:** Medical emergency with 80% mortality without transplant; cerebral edema is leading cause of death; requires ICU management and rapid transplant evaluation.

ANATOMY & PATHOPHYSIOLOGY

Massive hepatocellular necrosis: Loss of synthetic function (coagulopathy), metabolic function (hypoglycemia), detoxification (encephalopathy). **Cerebral edema mechanism:** Astrocyte swelling due to ammonia conversion to glutamine (osmotic effect), loss of autoregulation, cytokine storm. **Intracranial hypertension:** Leads to brainstem herniation (uncal/tonsillar).

ETOLOGY & RISK FACTORS

Cause	Features	Specific Treatment
Paracetamol	Most common in West; >4g/day or >10g single dose	NAC (best within 8h)
Viral Hepatitis	HBV (most common globally), HAV, HEV (pregnancy)	Antivirals for HBV
Drugs	Anti-TB (INH), antiepileptics, herbal meds	Stop offending drug
Wilson Disease	Young, hemolysis, low ceruloplasmin	Chelation, transplant
Budd-Chiari	Hypercoagulable, painful hepatomegaly	Anticoagulation, TIPS
Autoimmune	ANA positive, other autoimmune features	Steroids

CLINICAL FEATURES + PATHOGNOMONIC SIGNS

- Stage I (Mild confusion):** Sleep inversion, subtle personality changes
- Stage II (Lethargy):** Asterixis (flapping tremor) present, inappropriate behavior
- Stage III (Stupor):** Marked confusion, incoherent speech, hyperreflexia, clonus
- Stage IV (Coma):** Unresponsive, decerebrate posturing, loss of oculocephalic reflex

Critical Signs: Dilated/fixed pupils (herniation), hypertension + bradycardia (Cushing's triad), hyperventilation, hypothermia

DIFFERENTIALS (SURGICAL DISCRIMINATORS)

Acute-on-chronic liver failure (ACLF): Has underlying cirrhosis history, ascites, portal hypertension signs. **Sepsis-associated cholestasis:** Jaundice but no encephalopathy early. **Reye syndrome:** Children, aspirin use, fatty liver. **Malignant infiltration:** Lymphoma, breast cancer metastases (enlarged tender liver).

INVESTIGATIONS ALGORITHM

Immediate (ICU):

- INR: Essential for diagnosis and prognosis (>3.0 poor)
- Paracetamol level: If any possibility of overdose
- Viral markers: HBsAg, Anti-HBc IgM, Anti-HAV IgM, Anti-HEV

Pregnancy	Acute fatty liver, HELLP syndrome	Delivery
Indeterminate	15-20% cases	Supportive only

CLASSIFICATION: TIME-BASED

Hyperacute (<7 days): Best prognosis (paracetamol, HAV); cerebral edema common

Acute (7-28 days): Intermediate prognosis

Subacute (4-12 weeks): Worst prognosis; less cerebral edema but ascites, renal failure prominent; high transplant need

SURGICAL DECISION-MAKING

ICU Admission: All ALF patients require ICU (neuromonitoring, airway protection)

Intubation: Grade III-IV encephalopathy (prevent aspiration, control hyperventilation for ICP)

Emergency Transplant Criteria: King's College Criteria met, MELD > 30 , declining lactate

Contraindications to transplant: Irreversible brain damage (fixed dilated pupils > 1 h), uncontrolled sepsis, extrahepatic malignancy, severe cardiopulmonary disease

Living donor vs Cadaveric: If long wait expected, consider auxiliary transplant or hepatocyte transplantation (experimental)

MANAGEMENT LADDER (ICU PROTOCOL)

Step 1 - Airway/Breathing: Intubate if Grade III-IV; hyperventilate to PaCO₂ 25-30mmHg (cerebral vasoconstriction); avoid hypoxia

Step 2 - Circulation: Maintain MAP > 75 mmHg (cerebral perfusion pressure = MAP - ICP, target $> 50-60$); noradrenaline preferred; avoid overhydration

Step 3 - Specific therapy: NAC for all (benefit beyond paracetamol); Antivirals (tenofovir if HBV); Steroids (autoimmune); Delivery (pregnancy-related)

Step 4 - Cerebral edema: Mannitol 0.5-1g/kg (if responsive); Hypertonic saline (Na 145-155mmol/L); Hypothermia (32-34°C) if refractory; Barbiturate coma (last resort)

Step 5 - Coagulopathy: FFP only if bleeding or invasive procedure; vitamin K 10mg IV; Platelets $> 10-20$ k; Recombinant Factor VIIa if urgent surgery

Step 6 - Support: Lactulose (debatable benefit in ALF vs chronic); Rifaximin, PPI (stress ulcer prophylaxis), DVT prophylaxis (mechanical if coagulopathy)

Step 7 - Transplant: Contact transplant center immediately; listing based on severity

- Autoimmune screen:** ANA, SMA, immunoglobulins
- Ceruloplasmin:** If < 40 years (Wilson disease)
- USG Doppler:** Liver size (shrunken = necrosis), hepatic vein patency (Budd-Chiari), portal flow
- CT Head:** If concern for intracranial bleed or to rule out other causes of coma
- ICP monitoring:** Consider if Grade III-IV encephalopathy (controversial, risk of bleeding)

King's College Criteria (Transplant Indicators):

Paracetamol: pH < 7.3 OR all of (INR > 6.5 + Cr > 3.4 + Grade III-IV encephalopathy)

Non-paracetamol: INR > 6.5 OR any 3 of (Age < 10 or > 40 , Jaundice to encephalopathy > 7 days, INR > 3.5 , Bilirubin > 17.5 mg/dL, Unfavorable etiology)

NAMED OPERATIONS (WITH INDICATIONS)

Orthotopic Liver Transplantation (OLT): Standard of care for irreversible ALF meeting criteria. **Living Donor Liver Transplantation (LDLT):** If cadaveric organ unavailable; right lobe graft from living donor. **Auxiliary Transplantation:** Partial graft with native liver left in situ (potential for native liver regeneration, allows immunosuppression withdrawal later). **High-volume plasma exchange:** Bridge to transplant (removes toxins, improves hemodynamics). **Molecular Adsorbent Recirculating System (MARS):** Albumin dialysis (artificial liver support).

POST-OP CARE & COMPLICATIONS

Immediate post-transplant: Primary non-function (emergency re-transplant), hepatic artery thrombosis (Doppler daily x7 days), bleeding (coagulopathy takes time to correct)

Immunosuppression: Tacrolimus + Mycophenolate + Steroids (taper quickly to reduce infection risk)

Infections: Fungal (aspergillus), CMV, bacterial (line-related, pneumonia)

Neurological: Watch for persistent cerebral edema, seizures, central pontine myelinolysis (from rapid sodium shifts)

RARE BUT TESTED

Acute fatty liver of pregnancy (AFLP): Third trimester, microvesicular fatty infiltration, DIC, hypoglycemia; delivery is definitive treatment. **HELLP syndrome:** Hemolysis, Elevated Liver enzymes, Low Platelets; corticosteroids for fetal lung maturity then deliver. **Seronegative hepatitis:** 5-10% ALF with no identified cause; may have better transplant prognosis.

KMU EXAM TRAPS

- ALF patients do NOT typically have ascites (unlike chronic liver failure)—if ascites present, consider acute-on-chronic
- Lactulose is controversial in ALF (unlike chronic encephalopathy)—may cause bowel distension and aspiration; use cautiously
- Prophylactic antibiotics are recommended in ALF (reduce infection risk, improve transplant outcomes)
- NAC is beneficial even in non-paracetamol ALF (improves hemodynamics and transplant-free survival)

VIVA RAPID-FIRE Q/A

- What defines acute liver failure? INR >1.5 + Encephalopathy + No chronic liver disease + <26 weeks duration
- What is the most common cause of death in ALF? Cerebral edema/herniation
- What is the treatment for paracetamol overdose? N-acetylcysteine (NAC) - best within 8 hours
- King's College Criteria for paracetamol ALF? pH <7.3 after resuscitation OR INR >6.5 + Creatinine >3.4 + Grade III-IV encephalopathy
- What is the target cerebral perfusion pressure? >50-60 mmHg (MAP - ICP)

GOLDEN RULE / MNEMONIC

ALF MANAGEMENT - "HEPATIC ICU":

- H - Head (ICP management, CT if concern)
- E - Electrolytes & Glucose (frequent monitoring, prevent hypoglycemia)
- P - Pressure (MAP >75, CPP >50)
- A - Airway (intubate Grade III-IV)
- T - Transplant evaluation (King's Criteria)
- I - Infection control (prophylactic antibiotics)
- C - Coagulation (FFP only if bleeding)
- U - Urgent NAC (all patients)

ENCEPHALOPATHY GRADING: Confusion, Obnoxious behavior, Marked stupor, Absolutely unconscious (I, II, III, IV).

Crafted with ❤ Noaman Khan Musakhel | Page 3 of 5

HEPATIC ENCEPHALOPATHY

Ammonia Asterixis Lactulose Rifaximin West Haven Criteria Portosystemic shunt

NEUROPSYCHIATRIC SYNDROME OF LIVER FAILURE

TOPIC 19

KMU MCQ PICKUP LINES

- Cirrhosis + Altered mental status + Flapping tremor = Hepatic Encephalopathy (exclude other causes first)
- High ammonia level does NOT correlate with severity (can be normal in 10% with HE; levels used for monitoring response)
- HE + Normal ammonia = Check for other causes (hypoglycemia, sepsis, sedatives, Wernicke)
- Lactulose + Rifaximin combination = First-line for recurrent HE (reduces hospitalization)
- HE precipitated by: Infection (SBP most common), constipation, GI bleed, dehydration, sedatives, hypokalemia (all increase ammonia)

PRIMARY REFERENCE

Bailey & Love's Short Practice of Surgery - Portal Hypertension, Complications of Cirrhosis, Hepatic Encephalopathy

DEFINITION & SURGICAL IMPORTANCE

Definition: Brain dysfunction caused by liver insufficiency and/or portosystemic shunting; manifests as spectrum from subtle cognitive changes to coma. **Surgical Importance:** Marker of decompensated cirrhosis; increases surgical risk dramatically; may require preoperative optimization; recurrent HE may indicate need for TIPS revision or transplant evaluation; post-operative HE common in cirrhotics.

PATHOPHYSIOLOGY & ANATOMY

Portosystemic shunting: Bypasses hepatic detoxification; blood from gut enters systemic circulation directly. **Ammonia hypothesis:** Gut bacteria metabolize protein → ammonia → crosses BBB → astrocyte swelling (converted to glutamine osmotically). **Other neurotoxins:** Mercaptans, short-chain fatty acids, manganese (deposition in basal ganglia). **GABA-ergic tone:** Increased inhibitory neurotransmission. **Neuroinflammation:** Systemic inflammation exacerbates encephalopathy.

ETIOLOGY & RISK FACTORS (PRECIPITANTS)

Precipitant	Mechanism	Management
Infection (SBP)	Cytokine release, increased ammonia	Antibiotics, diagnostic tap
GI Bleed	Protein load, hypovolemia	Band ligation, lactulose
Constipation	Increased ammonia absorption	Lactulose, enemas
Hypokalemia	Intracellular acidosis increases ammonia production	KCl replacement
Dehydration	Azotemia, hypovolemia	Fluid resuscitation
Sedatives/Opioids	Direct CNS depression + decreased metabolism	Avoid/reduce dose
TIPS procedure	Increased shunting	Reduce protein, medications

CLINICAL FEATURES + PATHOGNOMONIC SIGNS

Neurological: Asterixis (flapping tremor at wrist extension, also seen in CO2 retention/uremia), hyperreflexia, clonus, rigid extremities (Grade III-IV), decerebrate posturing (Grade IV)

Cognitive: Constructional apraxia (cannot draw 5-pointed star), number connection test abnormal

Behavioral: Day-night sleep reversal, personality changes, inappropriate behavior

Focal deficits: Rare; if present, consider other causes (intracranial bleed, stroke)

Physical: Fetur hepaticus, signs of chronic liver disease

DIFFERENTIALS (SURGICAL DISCRIMINATORS)

Must rule out in cirrhotic with altered mental status:

Hypoglycemia (liver failure causes impaired gluconeogenesis), Wernicke encephalopathy (thiamine deficiency), subdural hematoma (coagulopathy), meningitis/encephalitis (immunocompromised), hypoxia, uremia, drug overdose (benzodiazepines, opioids), seizures/post-ictal.

INVESTIGATIONS ALGORITHM

Blood: Ammonia (arterial more accurate than venous; levels >100 µg/dL suggest HE but not diagnostic), Glucose (rule out hypoglycemia), Electrolytes (K+, Na+), CBC (infection), Blood culture

Ascitic fluid analysis: Cell count, culture (rule out SBP—most common precipitant)

Chest X-ray: Pneumonia

High protein intake	Substrate for ammonia production	Moderate restriction
---------------------	----------------------------------	----------------------

CLASSIFICATION: WEST HAVEN CRITERIA

Minimal/Covert HE: No clinical evidence; abnormal psychometric testing (important for driving ability)

Grade I: Trivial lack of awareness, euphoria/anxiety, shortened attention span

Grade II: Lethargy, disorientation, inappropriate behavior, asterixis present

Grade III: Somnolent but rousable, confusion, marked confusion, rigid extremities

Grade IV: Coma (unresponsive to verbal/noxious stimuli)

SURGICAL DECISION-MAKING

Preoperative: Grade I-II HE can proceed with urgent surgery after optimization; Grade III-IV delay elective surgery; emergency surgery requires airway protection and ammonia reduction

Post-operative prophylaxis: All cirrhotics having surgery should receive lactulose pre- and post-op to prevent HE (surgery is a precipitant)

TIPS and HE: New/worsening HE in 15-45% post-TIPS; usually manageable medically; severe recurrent HE may require TIPS reduction/occlusion (embolization) or transplant

MANAGEMENT LADDER

Step 1 - Identify & treat precipitant: Infection (antibiotics), Bleeding (control + lactulose), Constipation (lactulose), Drugs (stop sedatives)

Step 2 - Reduce ammonia production: Lactulose (non-absorbable disaccharide; dose to achieve 2-3 soft stools/day, titrate from 30ml BD); **Lactulose enemas** if NPO (300ml in 700ml water PR)

Step 3 - Modify gut flora: Rifaximin 550mg BD (non-absorbable antibiotic; reduces ammonia-producing bacteria; add to lactulose if recurrent HE or inadequate response)

Step 4 - Nutrition: Do NOT restrict protein long-term (worsens nutrition); give 1.2-1.5g/kg/day vegetable/dairy protein preferred; small frequent meals + late-night snack

Step 5 - Refractory HE: Branched-chain amino acids (controversial), Probiotics, **Zinc supplementation** (if deficient), Evaluate for large spontaneous shunts (embolization if found)

Step 6 - Transplant: Recurrent HE despite compliance = indication for liver transplant evaluation

CT Head: Indicated if focal deficits, trauma, severe headache, or to rule out intracranial bleed (especially if coagulopathy)
EEG: Triphasic waves (not diagnostic but supportive)
Psychometric testing: Trail-making test, digit symbol test (for minimal HE)

⚠ Protein restriction is OUTDATED—maintain nutrition, only brief restriction if severe flare

⚠ Haloperidol is safe for agitation in HE (unlike benzodiazepines which worsen encephalopathy)

Q: First-line treatment for HE? **Lactulose** (titrate to 2-3 soft stools/day)

Q: When do you add Rifaximin? **Recurrent HE or inadequate response to lactulose alone**

Q: What surgical procedure can precipitate HE? **TIPS (transjugular intrahepatic portosystemic shunt)**

GOLDEN RULE / MNEMONIC

HE PRECIPITANTS - "HEPATICS":

- H - Hemorrhage (GI bleed)
- E - Electrolyte imbalance (hypokalemia)
- P - Protein excess
- A - Alkalosis (hypokalemic)
- T - Tranquilizers (sedatives, opioids)
- I - Infection (SBP, pneumonia)
- C - Constipation
- S - Surgery, dehydration

TREATMENT - "Lacto-Rifa-Zinc": Lactulose (first), Rifaximin (add-on), Zinc (if deficient), Nutrition support.

Crafted with ❤ Noaman Khan Musakhel | Page 4 of 5

NAMED OPERATIONS/PROCEDURES

Large spontaneous portosystemic shunt embolization: If identified on imaging (CT/MRI) and refractory HE, interventional radiology can occlude shunt. **TIPS revision:** Reduction (placement of constricting stent) or occlusion if severe post-TIPS HE unresponsive to medical therapy. **Liver Transplantation:** Definitive treatment for recurrent/refractory HE in decompensated cirrhosis.

POST-OP CARE & COMPLICATIONS

Post-operative monitoring: HE common after any surgery in cirrhotics due to stress, medications, fasting; monitor ammonia levels and mental status

Medications to avoid: Benzodiazepines (enhance GABA), opioids (accumulate), tramadol, sedating antihistamines; if absolutely necessary, use short-acting agents in reduced doses with naloxone available

Complications of treatment: Dehydration from over-lactulosing (hypernatremia), aspiration pneumonia (from encephalopathy)

RARE BUT TESTED

Hepatocerebral degeneration: Chronic irreversible brain damage from recurrent HE; dementia, parkinsonism, ataxia (manganese deposition in basal ganglia). **Portosystemic myopathy:** Spastic paraparesis from chronic hepatic encephalopathy. **Minimal HE:** Subclinical but impairs driving ability and work performance; diagnosed only by psychometric testing.

KMU EXAM TRAPS

⚠ Ammonia level is NOT diagnostic of HE—clinical diagnosis; level used for monitoring trend, not absolute value

⚠ Neomycin is NO LONGER recommended (ototoxicity, nephrotoxicity)—use Rifaximin instead

VIVA RAPID-FIRE Q/A

Q: What is the most common precipitant of hepatic encephalopathy? **Infection (SBP is most common specific infection)**

Q: What is asterixis? **Flapping tremor due to intermittent loss of muscle tone (metabolic encephalopathy)**

APPROACH TO BLEEDING PER RECTUM

Hematochezia BRBPR Colonoscopy Meckel's Angiodysplasia Flexible sigmoidoscopy

LOWER GASTROINTESTINAL HEMORRHAGE

TOPIC 20

KMU MCQ PICKUP LINES

- Hematochezia + Hemodynamic instability = Rule out massive UGIB first (NG tube aspirate, EGD)
- BRBPR + Painful defecation + Blood on toilet paper = Anal fissure (posterior midline most common)
- Painless BRBPR + Straining + Bright red blood coating stool = Internal hemorrhoids (grade I-IV)
- Bloody diarrhea + Fever + Abdominal pain = Infectious colitis or Inflammatory bowel disease (UC > Crohn's for bleeding)
- Occult bleeding in elderly + Aortic stenosis = Angiodysplasia (Heyde syndrome - vWF destruction)
- Massive LGIB in patient <30 years = Meckel's diverticulum (ectopic gastric mucosa, scan with technetium-99m pertechnetate)

PRIMARY REFERENCE

Bailey & Love's Short Practice of Surgery - Lower Gastrointestinal Bleeding, Colorectal Emergencies

DEFINITION & SURGICAL IMPORTANCE

Definition: Bleeding from gastrointestinal tract distal to ligament of Treitz; manifests as hematochezia (bright red blood per rectum), maroon stools, or melena (if slow transit or proximal source). **Surgical Importance:** 20-30% require intervention; mortality 2-4% but higher in elderly; requires systematic approach to localize bleeding source; massive LGIB can masquerade as UGIB; urgent colonoscopy within 24h reduces rebleeding and mortality.

ANATOMY & SURGICAL LANDMARKS

Upper GI source bleeding briskly: Can present as hematochezia (melena requires 50-100ml blood + acid conversion). **Hemorrhoidal plexus:** Internal (superior hemorrhoidal plexus, above dentate line, painless) vs External (inferior plexus, below dentate line, painful). **Meckel's diverticulum:** 60cm from ileocecal valve (rule of 2's: 2% population, 2ft from valve, 2 inches long, 2% symptomatic, 2 types of mucosa). **Angiodysplasia:** Cecum and ascending colon (high wall tension, submucosal vein distortion). **Diverticula:** 90% right-sided colon (thin vasa recta penetrate muscularis).

ETOLOGY & RISK FACTORS (BY AGE)

Age Group	Common Causes
Young adults	Anal fissure, Hemorrhoids, IBD, Meckel's diverticulum, Polyps
Middle age	Diverticulosis, IBD, Colorectal cancer, Angiodysplasia
Elderly (>60)	Diverticulosis (40% of massive LGIB), Angiodysplasia, Cancer, Ischemic colitis

Risk factors: Anticoagulation, NSAIDs, Aortic stenosis (Heyde syndrome), Chronic kidney disease (uremic bleeding), Radiation proctitis.

CLASSIFICATION: SEVERITY

Minor: Self-limited, hemodynamically stable, no transfusion needed

Major: Hemodynamic instability, transfusion >2 units PRBC,

CLINICAL FEATURES + PATHOGNOMONIC SIGNS

Character of blood: Bright red (distal colon/rectum), Maroon (proximal colon/small bowel), Melena (slow LGIB or UGIB) **Associated symptoms:** Pain (fissure, proctitis), Change in bowel habits (cancer), Weight loss (malignancy), Fever (ischemia, infection), Abdominal pain (ischemia, IBD) **Digital rectal exam:** Essential—assess hemorrhoids, fissure (posterior midline), rectal mass, stool color **Anoscopy:** Visualizes internal hemorrhoids, fissures, anal canal tumors

DIFFERENTIALS (SURGICAL DISCRIMINATORS)

Must rule out UGIB: NG aspirate (blood = UGIB; bile + no blood = doesn't exclude UGIB completely); EGD if any doubt with hemodynamic instability.

Anal causes: Fissure (painful), Hemorrhoids (painless bleeding), Proctitis (tenesmus, mucus)

Colonic: Cancer (change habits, weight loss), Diverticula (painless massive bleed), Angiodysplasia (chronic occult), Ischemia (pain out of proportion, "painful bleeding")

INVESTIGATIONS ALGORITHM

Step 1 - Stabilize: Two large-bore IVs, crystalloid resuscitation, type & crossmatch 2-4 units PRBC

Step 2 - Rule out UGIB: NG tube aspirate; if blood or coffee-groundes → EGD first

Step 3 - Localization:

ongoing bleeding

Massive: Active ongoing bleed with shock, requires >4-6 units/24h

Occult: Positive FOBT, iron deficiency anemia, no visible bleeding

Anoscopy/Proctoscopy: If suspected anal source (hemorrhoids, fissure)

Colonoscopy: Diagnostic and therapeutic (within 24h for major bleed); identify source, perform hemostasis (clips, thermal, injection)

CT Angiography: If massive ongoing bleed (>0.5ml/min); identifies extravasation and anatomy for embolization

Tagged RBC scan: If intermittent bleeding (detects >0.1ml/min); less precise localization

Meckel's scan: Technetium-99m pertechnetate (uptake by gastric mucra)

Angiography: Diagnostic and therapeutic (embolization) if CT positive or massive ongoing bleed

Step 4 - If all negative: Capsule endoscopy (small bowel), Push enteroscopy, Intraoperative enteroscopy

SURGICAL DECISION-MAKING

ED Triage: Hemodynamic assessment first; unstable = ICU admission; massive transfusion protocol if needed

80-85% stop spontaneously: Supportive care, bowel prep for colonoscopy

15-20% require intervention: Endoscopic hemostasis first-line; angiographic embolization if colonoscopy fails or poor visualization; surgery for failure of above or peritonitis

Emergency surgery indications: Hemodynamic instability despite >4-6 units PRBC, recurrent bleed after endoscopic control, suspected ischemia/infarction, perforation

MANAGEMENT LADDER

Step 1 - Resuscitation: IV access, fluids, blood products (maintain Hb >7-8); correct coagulopathy; stop anticoagulants

Step 2 - Localization: NG aspirate, Anoscopy, Early colonoscopy (within 24h; bowel prep with polyethylene glycol 4-6L)

Step 3 - Endoscopic hemostasis: Clips (mechanical), Thermal coagulation (bipolar), Injection (epinephrine, sclerosants)

Step 4 - Angiography: Embolization with coils or microspheres (risk of bowel ischemia if proximal vessels embolized)

Step 5 - Surgery: Segmental colectomy if site identified; Subtotal colectomy with ileorectal anastomosis if site unidentified (10-20% mortality)

POST-OP CARE & COMPLICATIONS

Rebleeding: 10-25% after endoscopic therapy; second colonoscopy attempt

Post-colectomy: Anastomotic leak (especially if emergency surgery with unprepped bowel), diarrhea (short bowel if subtotal)

Hemorrhoidectomy complications: Urinary retention (most common), bleeding (day 7-10 when eschar falls), anal stricture, incontinence (if sphincter damage)

Angiographic complications: Bowel ischemia (if too proximal embolization), contrast nephropathy

Anticoagulation: Restart when hemostasis secure, usually 48-72h post-procedure

RARE BUT TESTED

Radiation proctitis: Pelvic radiation history; bleeding from telangiectasias; treated with formalin instillation or argon plasma coagulation.

Dieulafoy lesion of colon: Large submucosal artery, massive intermittent bleed, normal intervening mucosa; treated with endoscopic hemostasis or hemoclips.

Portal hypertensive colopathy: Dilated mucosal vessels in IBD-like pattern in portal hypertension; treat portal hypertension.

KMU EXAM TRAPS

BRBPR does NOT exclude UGIB—if unstable, rule out UGIB first with NG aspirate

VIVA RAPID-FIRE Q/A

Q: Most common cause of massive LGIB in elderly?

Diverticulosis

- ⚠ Diverticular bleeding is PAINLESS (unlike diverticulitis); pain + bleeding = ischemic colitis
- ⚠ Never band external hemorrhoids (painful somatic innervation); only band internal
- ⚠ Prep for colonoscopy in acute LGIB: Can use if stable (PEG solution); unprepped colonoscopy has low yield
- ⚠ Heyde syndrome: Aortic stenosis + Angiodysplasia + Acquired vWD (shear destruction); improves after aortic valve replacement

Q: Most common site of diverticular bleeding? **Right colon (ascending)**

Q: Diagnostic test for Meckel's diverticulum? **Technetium-99m pertechnetate scan (Meckel's scan)**

Q: What is the first step in suspected LGIB with hemodynamic instability? **Nasogastric aspirate to rule out UGIB**

Q: What is Heyde syndrome? **Aortic stenosis + Angiodysplasia + Bleeding due to acquired vWD**

GOLDEN RULE / MNEMONIC

CAUSES OF LGIB - "C A R T I N G":

- C - Cancer/Carcinoma (colorectal)
- A - Angiodysplasia (vascular ectasia)
- R - Radiation proctitis
- T - Trauma/Fissures
- I - IBD (Ulcerative colitis, Crohn's)
- N - Neoplasia (polyps)
- G - Gastroenteritis/Diverticulosis

MANAGEMENT PRIORITY - "S S S": Stabilize, Source localization, Stop bleeding (endoscopic → angiographic → surgical).