LIVER FUNCTION TEST

10 Questions with Explanation

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Liver Function Tests (LFTs) - Summary

Liver function tests assess liver health by measuring enzymes, proteins, and bilirubin in the blood. The most important tests include:

1. **Transaminases (AST & ALT):**
   - **AST (Aspartate Aminotransferase)** and **ALT (Alanine Aminotransferase)** are intracellular enzymes released during hepatocyte injury.
   - ALT is more liver-specific, while AST can be elevated in cardiac, muscle, or other organ damage.
   - **AST > ALT:** Suggests alcoholic liver disease. (1000 >) of ischemic (> 1000) (budd - chiari syndrome)
   - **ALT > AST:** Common in viral hepatitis and other liver injuries.
   - **Massive elevation (>1000 IU/L):** Viral hepatitis, acetaminophen toxicity, ischemic hepatitis.
2. **Alkaline Phosphatase (ALP) & Gamma-Glutamyl Transferase (GGT):**
   - **ALP:** Elevated in bile duct damage or obstruction (e.g., cholestasis).
   - **GGT:** Supports hepatic origin when ALP is elevated; also increased in alcohol consumption.
   - **High ALP + Normal GGT:** Consider non-hepatic causes (e.g., bone disease).
3. **Bilirubin:**
   - **Indirect (Unconjugated):** Increased in hemolysis or impaired conjugation (Gilbert's syndrome).
   - **Direct (Conjugated):** Increased in cholestasis or hepatocellular damage.
   - **Isolated elevation:** Gilbert's syndrome, Crigler-Najjar, Dubin-Johnson, Rotor syndrome.
4. **Albumin:**
   - Produced by the liver; low levels indicate chronic liver disease or malnutrition.
5. **Prothrombin Time (PT) and INR:**
   - Assess liver's ability to synthesize clotting factors (II, V, VII, X).
   - **Prolonged PT:** Severe liver dysfunction or vitamin K deficiency.

2. Patterns of LFT Abnormalities:

Pattern	Main Tests	Example Conditions
Hepatocellular	ALT, AST	Viral hepatitis, autoimmune hepatitis, ischemic hepatitis
Cholestatic	ALP, GGT	Bile duct obstruction, PBC, PSC
Isolated Bilirubin	Bilirubin (direct/indirect)	Gilbert's syndrome, Crigler-Najjar

Feature	PBC (Primary Biliary Cholangitis)	PSC (Primary Sclerosing Cholangitis)
Gender	Female	Male
Age	40–60 years	20–40 years
Association	Autoimmune diseases (e.g., Sjögren's)	Inflammatory Bowel Disease (esp. UC)
Antibodies	**AMA** (Anti-mitochondrial Ab)	p-ANCA (often positive)
Symptoms (early)	Fatigue, **pruritus**, RUQ discomfort	Often asymptomatic, fatigue, **pruritus**

Question:

A 55-year-old chronic alcoholic man has:

- **AST:** 160 U/L
- **ALT:** 70 U/L

What does his **AST:ALT ratio** most likely indicate?
a. Viral hepatitis
b. Alcoholic hepatitis
c. Ischemic hepatitis
d. Nonalcoholic fatty liver disease
Answer: b. Alcoholic hepatitis

Explanation:

Understanding the AST:ALT Ratio:

- In **alcoholic hepatitis**, the **AST:ALT ratio** is typically **>2** (AST is approximately 2-3 times higher than ALT).
- This occurs because:
  - **Chronic alcohol use** causes a deficiency of **vitamin B6**, which is required for ALT synthesis.
  - Alcohol damages **mitochondria**, leading to the release of **mitochondrial AST**.
- **AST** is predominantly found in mitochondria, while **ALT** is more cytosolic.
- Thus, in alcohol-related liver injury, AST tends to be **higher** than ALT.

Why the Answer is B (Alcoholic Hepatitis):

- The patient has a history of **chronic alcohol use**.
- **AST:ALT ratio = 160/70 ≈ 2.3** (greater than 2), which strongly suggests **alcoholic hepatitis**.

Why Other Options Are Incorrect:

- **a. Viral hepatitis:**
  - Typically shows **ALT > AST** (ratio usually <1).
- **c. Ischemic hepatitis:**
  - Shows **very high AST and ALT** (>1000 U/L), usually with no specific ratio pattern.
- **d. Nonalcoholic fatty liver disease (NAFLD):**
  - Usually shows **ALT > AST** or **AST:ALT ratio <1**.

Smart Note for Exam:

- **AST:ALT ratio > 2 → Think "alcoholic hepatitis."**
- **ALT > AST → Think "viral hepatitis" or "NAFLD."**
- **If both are extremely high (>1000 U/L) → Think "ischemic hepatitis" or "acute toxic injury."**
- **Mnemonic: "A**lcoholic **S**pikes **T**ransaminases" (AST high).

Question:

A 60-year-old woman's labs show:

- **ALP:** 450 U/L (↑)
- **GGT:** 380 U/L (↑)
- **AST, ALT:** mildly ↑

What is the most likely cause?
a. Primary biliary cholangitis
b. Acute viral hepatitis
c. Rhabdomyolysis
d. Gilbert's syndrome
Answer: a. Primary biliary cholangitis (PBC)

Explanation:

Liver Enzyme Patterns:

1. **Cholestatic Pattern (Intrahepatic/Extrahepatic):**
   - Markedly elevated **ALP and GGT**.
   - Mild elevation of **AST and ALT**.
   - Indicates **cholestasis**, which can be intrahepatic (like PBC) or extrahepatic (like bile duct obstruction).
2. **Hepatocellular Pattern:**
   - Marked increase in **AST and ALT** with normal or mildly increased **ALP and GGT**.
   - Typical of **viral hepatitis** or **toxic liver injury**.

Why the Answer is A (Primary Biliary Cholangitis):

- **PBC** is a **cholestatic liver disease** commonly seen in **middle-aged women**.
- It involves **autoimmune destruction of the small intrahepatic bile ducts**, leading to elevated **ALP and GGT**.
- Mild elevation of **AST and ALT** can also be present.

Why Other Options Are Incorrect:

- **b. Acute viral hepatitis:**
  - Shows a **hepatocellular pattern** with **markedly elevated AST/ALT**, not ALP and GGT.
- **c. Rhabdomyolysis:**
  - Causes **elevated AST and ALT** due to muscle breakdown but does **not increase GGT**.
  - **ALP** is usually **normal**.
- **d. Gilbert's syndrome:**
  - Characterized by **isolated elevation of indirect bilirubin**.
  - Liver enzymes (AST, ALT, ALP, GGT) are **normal**.

Smart Note for Exam:

- **High ALP & GGT with mild AST/ALT rise → Think "cholestasis" (intrahepatic or extrahepatic).**
- **PBC:** Common in **middle-aged women**; check for **anti-mitochondrial antibodies (AMA)**.
- **If ALP is high but GGT is normal → Think "bone disease."**
- **Mnemonic: "PBC" = "P**ositive **B**ile **C**holestasis" (high ALP & GGT).

Question:

A young man's tests reveal:

- **Total bilirubin:** 3.2 mg/dL (↑)
- **Conjugated bilirubin:** Normal
- **AST, ALT, ALP, GGT:** All normal

Which diagnosis fits best?
a. Dubin-Johnson syndrome
b. Rotor syndrome
c. Hemolytic anemia
d. Acute viral hepatitis
Answer: c. Hemolytic anemia

Explanation:

Understanding Hyperbilirubinemia:

1. **Unconjugated (Indirect) Hyperbilirubinemia:**
   - Caused by **overproduction** of bilirubin or **impaired conjugation**.
   - **Liver function tests (LFTs)** (AST, ALT, ALP, GGT) are typically **normal**.
   - Seen in **hemolytic anemia** due to increased breakdown of red blood cells (RBCs).
2. **Conjugated (Direct) Hyperbilirubinemia:**
   - Indicates **defective excretion** of conjugated bilirubin.
   - Often associated with **cholestatic or hepatocellular diseases**.

Why the Answer is C (Hemolytic Anemia):

- The patient shows **elevated total bilirubin** but **normal conjugated bilirubin**, indicating **indirect (unconjugated) hyperbilirubinemia**.
- **LFTs are normal**, ruling out liver or biliary disease.
- **Hemolytic anemia** causes **increased unconjugated bilirubin** due to excessive RBC breakdown.

Why Other Options Are Incorrect:

- **a. Dubin-Johnson syndrome:**
  - Causes **conjugated hyperbilirubinemia** due to impaired excretion.
  - **Dark liver on biopsy** is characteristic.
- **b. Rotor syndrome:**
  - Also causes **conjugated hyperbilirubinemia** but without liver pigmentation.
- **d. Acute viral hepatitis:**
  - Shows **hepatocellular pattern** with **elevated AST and ALT**.
  - Can cause **mixed hyperbilirubinemia**.

Smart Note for Exam:

- **Elevated total bilirubin + normal conjugated + normal enzymes → Unconjugated hyperbilirubinemia.**
- **Common causes: Hemolytic anemia, Gilbert's syndrome.**
- **If conjugated is high with normal enzymes → Think "Dubin-Johnson" or "Rotor syndrome."**
- **Mnemonic: "H**emolysis = **H**igh indirect bilirubin."

Question:

A patient with known cirrhosis suddenly develops a prolonged PT/INR. Which is the most likely explanation?
a. Acute ischemic hepatitis
b. Worsening synthetic failure of the liver
c. Hemolysis
d. Early cholestasis
Answer: b. Worsening synthetic failure of the liver

Explanation:

Understanding PT/INR in Liver Disease:

- **Prothrombin time (PT)** and **International Normalized Ratio (INR)** reflect the **liver's synthetic function**.
- The liver produces **clotting factors II, V, VII, IX, and X**, as well as **fibrinogen**.
- In **cirrhosis**, the liver's ability to produce these factors decreases.
- A **sudden prolongation of PT/INR** suggests a **further decline in liver synthetic capacity**.

Why the Answer is B (Worsening Synthetic Failure of the Liver):

- The patient already has **cirrhosis** (chronic liver disease).
- **Sudden worsening of PT/INR** indicates **acute deterioration** of liver function, reflecting **decompensation**.
- Causes include **acute-on-chronic liver failure**, **superimposed infection**, or **hepatic ischemia**.

Why Other Options Are Incorrect:

- **a. Acute ischemic hepatitis:**
  - Would show **markedly elevated AST and ALT**.
  - PT/INR may increase, but this condition is typically acute and not related to chronic cirrhosis.
- **c. Hemolysis:**
  - Does not affect **PT/INR**; instead, it causes **unconjugated hyperbilirubinemia**.
- **d. Early cholestasis:**
  - Increases **ALP and GGT**, not PT/INR.
  - Coagulopathy is less likely unless there is **severe cholestasis with vitamin K deficiency**.

Smart Note for Exam:

- **PT/INR = Marker of hepatic synthetic capacity**; if prolonged, think **synthetic failure**.
- **Cirrhosis with sudden PT prolongation = Decompensation or acute liver failure.**
- **Mnemonic: "PT Prolonged → Think Poor Protein Production."**

Question:

In acute viral hepatitis, the ratio **ALT:LDH** often helps distinguish it from ischemic injury. A ratio > 4.5 suggests:
a. Acute viral hepatitis
b. Ischemic hepatitis
c. Alcoholic hepatitis
d. Primary sclerosing cholangitis
Answer: a. Acute viral hepatitis

Explanation:

Understanding ALT and LDH Ratios:

1. **ALT (Alanine Aminotransferase):**
   - Significantly elevated in **hepatocellular injury**, especially **viral hepatitis**.
2. **LDH (Lactate Dehydrogenase):**
   - Increases in **cellular damage** but rises more dramatically in **ischemic hepatitis** due to **tissue hypoxia**.

ALT:LDH Ratio Interpretation:

- **ALT/LDH > 4.5:**
  - Suggests **acute viral hepatitis** because ALT rises significantly while LDH changes are less pronounced.
- **ALT/LDH < 1.5:**
  - Suggests **ischemic hepatitis**, where LDH rises more compared to ALT.

Why the Answer is A (Acute Viral Hepatitis):

- **Acute viral hepatitis** causes a **marked rise in ALT**, while **LDH** remains relatively stable or mildly elevated.
- A **ratio greater than 4.5** is **highly suggestive of viral hepatitis**.

Why Other Options Are Incorrect:

- **b. Ischemic hepatitis:**
  - LDH often rises **more significantly** than ALT, resulting in an **ALT/LDH ratio < 1.5**.
- **c. Alcoholic hepatitis:**
  - Shows an **AST:ALT ratio > 2**, but LDH is not typically a distinguishing marker.
- **d. Primary sclerosing cholangitis:**
  - Associated with **elevated ALP and GGT**, not specifically related to **ALT/LDH ratios**.

Smart Note for Exam:

- **ALT/LDH > 4.5 → Think "Acute Viral Hepatitis."**
- **ALT/LDH < 1.5 → Think "Ischemic Hepatitis."**
- **Mnemonic: "V**iral = **V**ery high ALT compared to LDH."

Question:

A young female presents with mild right upper quadrant (RUQ) pain and elevated liver enzymes. Lab results show:

- **Increased INR**
- **AST > ALT**
- **Serum pH < 7.3**

What is the most likely cause of her liver injury?
a. Autoimmune hepatitis
b. Paracetamol toxicity
c. Viral hepatitis
d. Alcoholic hepatitis
Answer: b. Paracetamol toxicity

Explanation:

Key Features of Paracetamol Toxicity:

1. **Liver Enzymes:**
   - **AST > ALT:** In severe toxicity, **AST** is often **higher** than **ALT** due to **centrilobular necrosis**.
2. **Coagulation Abnormalities:**
   - **Increased INR** reflects **impaired hepatic synthesis** of clotting factors.
3. **Metabolic Acidosis:**
   - **Serum pH < 7.3** indicates **severe metabolic acidosis**, suggesting **acute liver failure**.
4. **Mechanism:**
   - **Paracetamol overdose** depletes **glutathione**, leading to **accumulation of toxic metabolites** and **hepatic necrosis**.

Why the Answer is B (Paracetamol Toxicity):

- The combination of **elevated INR, AST > ALT**, and **metabolic acidosis** is **highly characteristic of paracetamol toxicity**.
- **Acute liver failure** and **centrilobular necrosis** are hallmarks of severe overdose.
- **Confirmatory Test: Serum paracetamol level** and **LFTs**.
- **Treatment:** Administer **N-acetylcysteine (NAC)** promptly.

Why Other Options Are Incorrect:

- **a. Autoimmune hepatitis:**
  - Typically shows **ALT > AST** and no metabolic acidosis.
  - INR may be elevated if liver failure develops, but not as abruptly.
- **c. Viral hepatitis:**
  - Also presents with **ALT > AST**.
  - **Normal pH** unless fulminant hepatitis occurs.
- **d. Alcoholic hepatitis:**
  - Usually shows **AST:ALT ratio > 2**, but without **metabolic acidosis**.
  - INR may increase with **chronic liver dysfunction**, not acutely.

Smart Note for Exam:

- **Paracetamol toxicity:**
  - **↑ INR, AST > ALT, low serum pH** = Acute liver failure.
  - **Confirm with serum paracetamol level.**
  - **Antidote: N-acetylcysteine (NAC)**.
- **Mnemonic: "P**aracetamol = **P**oor Prognosis with **P**H < 7.3."

Question:

A 30-year-old female presents with fatigue, jaundice, and arthralgia. Lab results show:

- **ALT > AST**
- **Positive ANA and Anti-Smooth Muscle Antibody (ASMA)**
- **Elevated IgG**
- **Normal ALP and GGT**

What is the most likely diagnosis?
a. Alcoholic hepatitis
b. Autoimmune hepatitis
c. Viral hepatitis
d. Hemochromatosis
Answer: b. Autoimmune hepatitis

Explanation:

Key Features of Autoimmune Hepatitis (AIH):

1. **Liver Enzymes:**
   - **ALT > AST:** Indicates a **hepatocellular pattern**.
   - **Normal ALP and GGT:** Rules out **cholestatic injury**.
2. **Autoantibodies:**
   - **Positive ANA (Antinuclear Antibody)**
   - **Positive ASMA (Anti-Smooth Muscle Antibody)**
   - These are typical **serological markers** of AIH.
3. **Immunoglobulin Levels:**
   - **Elevated IgG (hypergammaglobulinemia)** is a hallmark of AIH.
4. **Clinical Presentation:**
   - Common in **young females**.
   - Symptoms include **fatigue, jaundice, and arthralgia**.

Why the Answer is B (Autoimmune Hepatitis):

- The combination of **ALT > AST**, **positive ANA/ASMA**, and **elevated IgG** strongly points to **autoimmune hepatitis**.
- **Diagnosis Confirmation:**
  - **Autoantibody panel** (ANA, ASMA, anti-LKM1).
  - **Liver biopsy:** Shows **interface hepatitis** (lymphoplasmacytic infiltration).
- **Treatment:**
  - **Immunosuppressive therapy** with **corticosteroids** and **azathioprine**.

Why Other Options Are Incorrect:

- **a. Alcoholic hepatitis:**
  - Shows **AST > ALT** (usually AST:ALT ratio > 2).
  - **No autoantibodies** or **hypergammaglobulinemia**.
- **c. Viral hepatitis:**
  - Typically presents with **ALT > AST**, but lacks **autoantibodies** and **IgG elevation**.
  - Diagnosis confirmed by **viral serologies (HBV, HCV)**.
- **d. Hemochromatosis:**
  - **Increased ferritin** and **iron saturation**, not IgG.
  - Typically **chronic liver injury** without positive autoantibodies.

Smart Note for Exam:

- **Autoimmune hepatitis:** Think of **young females** with **ALT > AST**, **positive ANA/ASMA**, and **↑ IgG**.
- **Diagnostic markers: ANA, ASMA, anti-LKM1**, and **elevated IgG**.
- **Treatment: Steroids + Azathioprine**.
- **Mnemonic: "A**utoimmune **H**epatitis = **A**NA + **H**igh IgG."

Question:

A patient presents with elevated liver enzymes. Which of the following lab findings would specifically point toward **paracetamol toxicity** rather than **autoimmune hepatitis**?
a. Positive ANA
b. AST > ALT in thousands
c. Elevated IgG
d. ALT > AST
Answer: b. AST > ALT in thousands

Explanation:

Key Features of Paracetamol Toxicity:

1. **Massive Enzyme Elevation:**
   - Both **AST and ALT** can rise dramatically, often into the **thousands (1000-5000 U/L)**.
   - **AST > ALT** is typical in severe cases.
2. **Mechanism:**
   - Paracetamol overdose leads to **centrilobular hepatic necrosis**, causing a **massive release of transaminases**, predominantly **AST**.

Key Features of Autoimmune Hepatitis (AIH):

- **ALT > AST** (reflecting hepatocellular injury).
- Associated with **positive autoantibodies (ANA, ASMA)**.
- **Elevated IgG** (hypergammaglobulinemia).
- Enzyme levels typically do not reach the **thousands** as in paracetamol toxicity.

Why the Answer is B (AST > ALT in thousands):

- **Paracetamol toxicity** is characterized by **extremely high transaminase levels**, often reaching the **thousands**, with **AST typically higher than ALT**.
- This pattern **distinguishes it from autoimmune hepatitis**, where **ALT > AST** and elevations are usually more moderate.

Why Other Options Are Incorrect:

- **a. Positive ANA:**
  - Indicates **autoimmune hepatitis**, not paracetamol toxicity.
- **c. Elevated IgG:**
  - Characteristic of **autoimmune hepatitis**.
- **d. ALT > AST:**
  - Common in **autoimmune and viral hepatitis**, not paracetamol toxicity.

Smart Note for Exam:

- **Paracetamol toxicity:**
  - **AST and ALT > 1000 U/L**, with **AST > ALT**.
  - **Think massive hepatocellular necrosis.**
- **Autoimmune hepatitis:**
  - **ALT > AST**, **positive ANA/ASMA**, **↑ IgG**.
- **Mnemonic: "P**aracetamol = **P**eak AST (in thousands)."

Question:

A case of mild elevation of ALT, AST, and highly elevated ALP and GGT, which of the following is **not** included in the differential diagnoses?
a. Autoimmune hepatitis
b. Fatty liver disease
c. Hemochromatosis
d. Wilson's disease
Answer: d. Wilson's disease (Wilson's has normal ALP)

Explanation:

To understand why the answer is Wilson's disease, let's break down the liver function test (LFT) patterns and the reasoning behind each option.

Liver Enzyme Patterns:

1. **ALT (Alanine Aminotransferase) and AST (Aspartate Aminotransferase)**:
   - Mild elevation indicates **hepatocellular damage** (injury to liver cells).
2. **ALP (Alkaline Phosphatase)**:
   - Significantly elevated in cases of **cholestasis**, **biliary obstruction**, or **infiltrative liver diseases** (like primary biliary cirrhosis or metastatic liver disease).
   - Also elevated in **bone diseases**, but GGT helps differentiate between liver and bone origin.
3. **GGT (Gamma-Glutamyl Transferase)**:
   - Elevates in **biliary obstruction**, **cholestasis**, and **alcoholic liver disease**.
   - Unlike ALP, GGT is not elevated in bone disorders, indicating that the elevation of ALP is liver-specific when GGT is also elevated.

Option Analysis:

- **a. Autoimmune hepatitis:**
  - Typically shows **elevated ALT and AST** with possible mild elevation in ALP and GGT, especially if there is **bile duct involvement**.
  - **Included** in differential diagnosis.
- **b. Fatty liver disease:**
  - Commonly presents with **mild ALT and AST elevation**, while ALP and GGT may be mildly elevated, especially in cases with **alcohol use**.
  - **Included** in differential diagnosis.
- **c. Hemochromatosis:**
  - Can present with **mild ALT and AST elevation** due to hepatocyte damage.
  - **ALP can be mildly elevated** in advanced cases with fibrosis or cirrhosis.
  - **Included** in differential diagnosis.
- **d. Wilson's disease (Correct answer):**
  - Primarily shows **mild elevation in ALT and AST**.
  - **ALP is typically normal or low**.
  - Therefore, **not included** in the differential diagnosis when both ALP and GGT are highly elevated.

Smart Note for Exam:

- **Key pattern:** Elevated **ALP and GGT** indicate **cholestasis or biliary disease**.
- **Wilson's disease:** Does **not** cause ALP elevation, unlike autoimmune hepatitis, fatty liver disease, and hemochromatosis.
- **Tip:** When ALP is elevated, always check **GGT** to determine if the origin is hepatic (high GGT) or bone (normal GGT).
- **Remember:** If ALP is high but GGT is normal, think **bone pathology** instead of liver.

Quick Exam Summary:

1. **Paracetamol Toxicity:**
   - **AST > ALT** (often in the thousands).
   - Massive enzyme elevation (AST > ALT).
   - **Confirm with serum paracetamol level**.
   - **Treatment: N-acetylcysteine (NAC)**.
2. **Autoimmune Hepatitis (AIH):**
   - **ALT > AST**.
   - **Positive ANA/ASMA** (autoantibodies).
   - **Elevated IgG**.
   - Common in **young females**.
   - **Treatment: Corticosteroids + Azathioprine**.
3. **Alcoholic Hepatitis:**
   - **AST > ALT** (AST:ALT > 2).
   - **No positive autoantibodies** or **hypergammaglobulinemia**.
   - **No metabolic acidosis**.
4. **Viral Hepatitis:**
   - **ALT > AST**. / ALT:LDH > 4.5
   - Confirm with **viral serologies (HBV, HCV)**.
   - **No autoantibodies**.
5. **Hemolysis (Indirect Hyperbilirubinemia):**
   - **Elevated total bilirubin** with **normal conjugated bilirubin**.
   - **Normal liver enzymes** (AST, ALT, ALP, GGT).
   - **Diagnosis:** Look for **hemolysis markers** (e.g., reticulocytes).
6. **Primary Biliary Cholangitis (PBC):**
   - **Elevated ALP + GGT**.
   - **Mildly elevated AST/ALT**.
   - Confirm with **AMA** (anti-mitochondrial antibody).
7. **Wilson's Disease:**
   - **Elevated AST/ALT** with **normal ALP/GGT**.
   - **Low serum ceruloplasmin**, **high urinary copper**.
   - **Treatment: Penicillamine** or **Zinc**.
8. **Cholestatic Pattern:**
   - **ALP + GGT elevated** (e.g., in PBC, obstructive jaundice).
   - **Mild elevation of AST/ALT**.
9. **Acute Liver Failure (ALF):**
   - **Marked elevation of AST/ALT**.
   - **Increased INR** and **low pH**.
   - **Paracetamol overdose** can lead to **acute liver failure**.

Key Patterns for Exam:

- **AST > ALT (thousands) → Paracetamol toxicity**.
- **ALT > AST → Autoimmune hepatitis, Viral hepatitis, Alcoholic hepatitis**.
- **Elevated ALP + GGT → Cholestatic pattern** (PBC, biliary obstruction).
- **Increased INR → Synthetic liver failure** (e.g., cirrhosis, acute liver failure).

Mnemonic:

- **P**aracetamol = **P**eak AST.
- **A**utoimmune hepatitis = **A**LT > AST, **A**NA, **A**nti-Smooth Muscle Antibodies.