Case 1

Answers:

1. This is a typical pattern of hepatocellular injury, compatible with acute hepatitis of viral, drug-induced, or ischemic etiology.

2. Hepatitis viruses A,B, and C; possibly acetaminophen level although if normal or within therapeutic range this would not exclude acetominophen toxicity as a possible cause.

Additional blood test results:

HBsAg negative

Anti-HCV negative

anti-HAV IgG positive

anti-HAV IgM positive

Case 2

Answers:

1. Acute-on-chronic alcoholic liver disease (alcoholic hepatitis and cirrhosis), possibly spontaneous peritonitis or hepatocellular CA.

   Less likely, viral, drug, autoimmune, metal-induced, genetic, vascular, or obstructive liver disease.

2. Alcoholic hepatitis and cirrhosis (typically AST > ALT, only modest increase in AST/ALT despite significant liver dysfunction). The reason for this is that ALT and AST both require pyridoxal phosphate as a cofactor, and low levels of pyridoxal are common in alcoholics. ALT is more sensitive to pyridoxal deficiency than AST. A second reason is that AST is mitochondrial as well as cytosolic, and alcoholic hepatitis is associated with mitochondrial injury.

3. Increased sinusoidal pressure causes transudation of relatively protein-rich fluid from the liver through fenestrated sinusoidal endothelium. Hypoalbuminemia and increased portal pressure also results in increased intestinal lymph production (Starling forces) although the protein content is lower than liver-derived lymph because intestinal capillaries have tight junctions and no fenestrae. When lymph flows from both sources exceeds capacity of peritoneal lymphatics, ascites forms. As ascites develops, there is a decreased effective circulating blood volume, which stimulates renin/aldosterone release. Other mechanisms involved in the abnormal sodium handling include prostaglandins, sympathetic tone, and atrial natriuretic factor. There is also evidence for a direct role of cirrhosis in triggering sodium retention in the absence of actual or effective hypovolemia.

Case 3 Answers

Additional results:

ferritin >1500 (normal <250)

liver biopsy: cirrhosis, ++++ parenchymal iron

genetic test for hemochromatosis: homozygous for Cys282Tyr mutation

Case 4 Answers

1. The rash and polyarthralgias are suggestive of an immune-complex phenomenon. This can occur with hepatitis B as well as with hepatitis C.

2. The edema is caused by hypoalbuminemia. This seems out of proportion to other parameters of liver function, and suggests excess albumin loss rather than impaired albumin synthesis to be responsible

3. Additional test results:

   HBsAg positive
   anti-HBc positive
   HBeAg positive

   liver biopsy: mild periportal inflammatory infiltrate, no piecemeal necrosis, no fibrosis

   urine protein 10 g/day

   renal biopsy: membraneous GN with positive immunoperoxidase staining with anti-HBc

4. Either lamivudine or interferon could be considered.

Case 5

Answers:

1. The GGT elevation in this case is quite nonspecific. However, it is perhaps somewhat higher than what one would expect from mild hepatocellular damage alone, and so suggests possible steatosis (diabetes, obesity) or alcohol effect.
2. The mild chronic elevation of transaminases and GGT is compatible with many conditions including inflammatory, genetic, toxic/metabolic, infiltrative and neoplastic. In this clinical setting, chronic viral hepatitis (esp. hepatitis C), steatosis/alcoholic liver disease, and hemochromatosis would be the leading considerations.

3. HBsAg, anti-HCV, ferritin or serum iron/TIBC, abdominal ultrasound, repeat enzymes after 3 months complete abstemiousness from alcohol. One could include ceruloplasmin, alpha-1 antitrypsin, ANA, immunoglobulin quantitation in the initial test battery. A liver biopsy should not be necessary to establish the diagnosis in this case, but if requested by students before the serology is ordered, a biopsy shows "mild portal fibrosis, moderate portal inflammatory infiltrates with mild piecemeal necrosis, moderate steatosis. Iron stain normal."

   Additional blood test results:

   HBsAg negative
   anti-HCV positive
   ANA negative
   ceruloplasmin normal
   alpha-1 antitrypsin normal