AJPMHS GLOBAL RESEARCH NETWORK

AMERICAN Journal of Pediatric Medicine and Health Sciences
Volume 3, Issue 3, 2025 ISSN (E): 2993-2149

Changes in the Blood in Liver Diseases and Modern Clinical Diagnostic Methods

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Abstract: Many liver biochemical tests can assess liver function by measuring the amount of enzymes released into the bloodstream (e.g., aminotransferase from damaged liver cells, alkaline phosphatase in cholestasis) or hepatobiliary excretion (e.g., bilirubin). Other tests are used to assess the synthetic function of the liver (e.g., prothrombin time [PT], usually international normalized ratio [INR]; albumin).

The most useful tests for screening for various liver diseases are serum aminotransferase levels (the most commonly measured liver function tests), bilirubin, and alkaline phosphatase. Certain biochemical abnormalities can help distinguish hepatocellular pathology from biliary tract disorders (cholestasis - see table of most common laboratory abnormalities). Tests for the diagnosis of viral hepatitis, liver inflammation, or immune disorders include serological tests for markers of hepatitis viruses and measurements of immunoglobulins, antibodies, and autoantibodies.

Keywords: Liver pathologies, pathogenesis, prevention, prognosis and treatment.

Introduction: A systematic approach including laboratory tests, imaging, and liver biopsy should be used to determine the etiology of abnormal liver function tests.

Some laboratory tests are diagnostic or highly indicative in themselves; these include :

a. IgM antibodies to hepatitis A virus (anti-HAV) in acute hepatitis A;

b. Hepatitis B surface antigen (HBsAg) in acute and/or chronic hepatitis B.

c. Antibodies to hepatitis C virus (anti-HCV) and HCV - RNA in acute and/or chronic hepatitis C

d. Antimitochondrial antibodies in primary biliary cholangitis (formerly called primary biliary cirrhosis)

e. In Wilson's disease, serum ceruloplasmin (decreased) and urinary copper (elevated)

f. Serum alpha-1 antitrypsin levels and genetic testing for alpha-1 antitrypsin deficiency

Other causes of liver disease are diagnoses of exclusion, made based on characteristic laboratory test results, as well as the patient's history and exclusion of other causes.

Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are released from damaged hepatocytes; thus, these enzymes are sensitive indicators of hepatocyte damage. True

normal ALT values are 29 to 33 IU/L in men and 19 to 25 IU/L in women, which are lower than the values used as reference values by many commercial laboratories.

Significantly elevated values (> 500 IU/L), indicating acute hepatocellular necrosis or injury, are usually caused by:

Methods and Materials: Elevated levels usually persist for several days or weeks, depending on the etiology of the injury. The degree of elevation may not reflect the extent of liver injury. Serial measurements are more predictive of severity and prognosis than a single study. A return to normal levels, unless accompanied by an increase in bilirubin and prothrombin time (PT) or international normalized ratio (INR), indicates recovery, which may indicate acute liver failure, as well as fulminant liver failure. In acute liver failure, enzyme levels may normalize because fewer hepatocytes remain; thus, such normalization does not indicate improvement in liver function.

Aminotransferase levels may also be significantly elevated in the following cases:

a. exacerbation of autoimmune hepatitis;

b. reactivation of chronic hepatitis B;

c. Liver disease caused by drugs or toxins

d. passage of a stone through the common bile duct

e. acute Budd-Chiari syndrome;

f. acute fatty liver of pregnancy;

g. Acute Wilson's disease

Moderate elevations of ALT and AST levels (300-500 IU/L) are seen in chronic liver disease (e.g., chronic hepatitis), biliary obstruction, except for the passage of a stone through the common bile duct, which can cause a transient significant increase in enzyme levels.

A slight increase (< 300 IU/L) is nonspecific and is often observed in the following diseases:

a. Cirrhosis

b. Chronic viral hepatitis

c. non-alcoholic fatty liver disease (NAFLD)

d. cholestatic liver diseases;

e. Hepatocellular carcinoma, cholangiocarcinoma, and metastatic liver cancer

f. Alcoholic liver disease and alcoholic hepatitis

g. Drug-induced liver disease

h. In some liver diseases, aminotransferases may be slightly elevated or even normal, such as

i. Hemochromatosis

j. Drug-induced liver disease (for example, liver damage due to methotrexate or amiodarone)

k. Chronic hepatitis B and chronic hepatitis C

l. NAFLD

m. Infiltrative liver diseases (e.g., hepatic sarcoidosis, amyloidosis)

n. Alpha-1 antitrypsin deficiency

o. Primary biliary cholangitis

p. Primary sclerosing cholangitis

Conclusions : Elevated ALT levels are somewhat specific for liver disease. Since AST is present in the heart, skeletal muscle, kidneys, and pancreas, elevated levels may reflect rhabdomyolysis or damage to one of these organs. In most liver diseases, the ratio of AST to ALT is < 1. However, in alcoholic liver disease, this ratio is usually > 2 because patients with alcoholism are commonly deficient in pyridoxal-5'-phosphate; it is essential for ALT synthesis but less so for AST synthesis. This explains why ALT and AST are usually low in such patients (< 300 IU/L).

Bilirubin, a bile pigment, is produced primarily by the breakdown of hemoglobin in aged red blood cells. Unconjugated (free) bilirubin is insoluble in water and is therefore not excreted in the urine; most of it is bound to albumin in the plasma. Bilirubin is conjugated with glucuronic acid in the liver to form the water-soluble bilirubin diglucuronide. The conjugated bilirubin is then excreted through the bile ducts into the duodenum, where it is metabolized to urobilinogens (some of which are absorbed and re-excreted into the bile) and then to orange urobilinogens (most of which are excreted in the feces). These bile pigments give the stool its characteristic color.

Typically, total bilirubin is expressed as an unconjugated fraction with values of < 1.2 mg/dL (< 20 μmol/L). Fractional measurements estimate the proportion of conjugated bilirubin (i.e., directly, so called because it is measured directly, without the use of solvents). Fractionation is most useful in evaluating jaundice in newborns and in understanding the cause of elevated bilirubin levels when otherwise normal liver function tests indicate that hepatobiliary dysfunction is not the cause.

Unconjugated hyperbilirubinemia is an indirect bilirubin fraction > 85% that reflects increased bilirubin production (e.g., hemolysis) or impaired hepatic absorption or conjugation (e.g., Gilbert's syndrome). In the absence of concomitant liver damage, this increase in unconjugated bilirubin is usually < 5 times normal (< 6 mg/dL [< 100 μmol/L]).

Discussion : Conjugated hyperbilirubinemia (direct bilirubin fraction > 50%) occurs when bile production or excretion (cholestasis) is reduced. In the presence of other abnormalities on liver function tests, elevated serum bilirubin levels indicate hepatocellular and/or biliary dysfunction. Serum bilirubin is not a partial response to liver dysfunction. However, the progression of severe hyperbilirubinemia to primary biliary cholangitis (formerly called primary biliary cirrhosis), primary sclerosing cholangitis, alcoholic hepatitis, and acute liver failure indicates a poor prognosis.

Bilirubinuria indicates the presence of conjugated bilirubin in the urine; bilirubin is present in the urine because its level in the blood is significantly elevated, indicating a serious illness. Unconjugated bilirubin is insoluble in water and binds to albumin, so it is not excreted in the urine. Bilirubinuria can be detected in acute viral hepatitis or other hepatobiliary diseases, even before the onset of jaundice, at the bedside with commercial urine test strips. However, the accuracy of such tests is limited. False negative results may occur if the urine sample has been stored for a long time, if vitamin C has been taken, or if nitrates are present in the urine (for example, due to a urinary tract infection). Similarly, elevated urobilinogen levels are nonspecific and insensitive.

Elevated levels of this hepatocyte enzyme are indicative of cholestasis. The results may be nonspecific because alkaline phosphatase consists of different isoenzymes and has a wide extrahepatic distribution (e.g., in the placenta, small intestine, blood, kidney, and especially bone).

Alkaline phosphatase levels increase to ≥4 times normal within 1 to 2 days of the onset of biliary obstruction, regardless of the site of onset. After the biliary obstruction is resolved, levels may remain elevated for several days because the half-life of ALP is approximately 7 days. Elevations of up to 3% of normal levels are common in many liver diseases, including:

Liver-specific gamma-glutamyl transpeptidase or 5-nucleotidase levels differentiate between hepatic and extrahepatic sources of alkaline phosphatase better than its fractionation, which is technically more difficult. Furthermore, in older individuals without other clinically apparent diseases, elevated ALP is usually associated with musculoskeletal problems (eg, Paget's disease) and may not warrant further investigation of liver status.

Elevated levels of this enzyme are as sensitive as alkaline phosphatase in detecting cholestasis and biliary obstruction, but are more specific, and almost always indicate hepatobiliary dysfunction. Because ALP and 5'-nucleodase levels do not always correlate, one may be normal and the other elevated.

The level of this enzyme is increased in hepatobiliary dysfunction, especially in cholestasis, and is freely associated with ALT and 5'-nucleotidase levels. Levels are not increased in bone pathology, childhood, or pregnancy. However, alcohol, some drugs (e.g., some anticonvulsants, warfarin), herbs, and some foods can induce liver microsomal enzymes (cytochrome P-450), which significantly increase GGT levels, thereby somewhat limiting its specificity.

PT can be expressed as time (seconds) or, better, as the ratio of the patient's measured PT to a laboratory control value (INR - see Test). INR is more accurate than PT in monitoring anticoagulation. PT or INR are important indicators of the liver's ability to synthesize fibrinogen and the vitamin K-dependent clotting factors: factors II (prothrombin), VII, IX, and X. Changes can occur rapidly because some of the clotting factors have short biological half-lives (e.g., 6 hours for factor VII). Abnormalities that indicate severe hepatocellular dysfunction are a warning sign of the risk of developing acute liver disease. In chronic liver disease, an increase in INR or PT indicates progressive liver failure. INR or PT levels are not elevated in mild hepatocellular dysfunction and are often within normal limits in compensated cirrhosis.

Prolonged PT and altered INR may result from coagulation disorders such as consumptive coagulopathy and vitamin K deficiency. Fat malabsorption, as in cholestasis, may lead to vitamin K deficiency. Hepatocellular dysfunction in chronic cholestasis can be ruled out if vitamin K (10 mg subcutaneously or intravenously) corrects the PT by ≥ 30% within 24 hours.

Most serum proteins are synthesized by hepatocytes, including alpha- and beta-globulins, albumin, and most coagulation factors (with the exception of factor VIII, which is produced by vascular endothelium, or gamma globulin, which is produced by B cells). Hepatocytes also produce proteins that help diagnose certain diseases:

These proteins are normally elevated in response to various tissue injuries (e.g., inflammation), so the elevation may be nonspecific and unrelated to liver disease. Conversely, serum levels of these proteins may be decreased in cirrhosis.

Summary : In chronic liver disease, serum albumin is usually reduced due to fluid volume redistribution (e.g., ascites), decreased hepatic synthesis, or both. Values < 3 g/dL < 30 g/L indicate decreased synthesis due to one of the following reasons:

Hypoalbuminemia can also result from a large loss of albumin through the kidneys (e.g., nephrotic syndrome), intestines (e.g., protein-losing gastroenteropathies), or skin (e.g., due to burns or exfoliative dermatitis).

Because albumin has a half-life of approximately 20 days, it usually takes weeks for its serum levels to rise or fall, but changes can be rapid in serious illnesses.

Nitrogenous compounds that enter the colon (e.g., ingested protein, excreted urea) are broken down by resident bacteria, releasing ammonia. The ammonia is then absorbed and transported to the liver via the portal vein. A healthy liver removes ammonia from the portal blood and converts it to glutamine, which is metabolized and excreted by the kidneys as urea. In patients with portosystemic shunting and chronic liver disease, the diseased liver is unable to eliminate ammonia, and it quickly enters the systemic circulation, contributing to the development of

portosystemic (hepatic) encephalopathy. Increased ammonia levels are seen in hepatic encephalopathy, but its levels may be abnormally high or low. In cases of severe liver damage, the following can cause elevated ammonia levels:

In acute liver failure, unlike portosystemic shunting, elevated arterial ammonia levels are due to severe acute hepatocyte dysfunction and/or necrosis and may be a sign of poor prognosis.

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