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DIAGNOSIS OF ACUTE AND CHRONIC HEPATITIS

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Abstract

If the period of inflammation or hepatocellular injury lasts for less than six months, characterized by normalization of the liver function tests, it is called acute hepatitis. In contrast, if the inflammation or hepatocellular injury persists beyond six months, it is termed chronic hepatitis

Keywords: chronic hepatitis, immunoglobulin, hepatocellular.

Ask your doctor for the following blood tests: hepatitis b surface antigen (hbsag): tells if you have chronic hepatitis b. Only the hbsag blood test can tell if you have chronic hepatitis b. Hepatitis b surface antibody (anti-hbs): tells if you are protected against hepatitis b. If you know you've been exposed to the hepatitis B virus, call your health care provider immediately. It is important to know whether you have been vaccinated for hepatitis B. Your health care provider will want to know when you were exposed and what kind of exposure you had.

An injection of immunoglobulin (an antibody) given within 24 hours of exposure to the virus may help protect you from getting sick with hepatitis B. Because this treatment only provides short-term protection, you also should get the hepatitis B vaccine at the same time if you never received it. If your provider determines your hepatitis B infection is acute — meaning it is short lived and will go away on its own — you may not need treatment. Instead, your provider might recommend rest, proper nutrition, plenty of fluids and close monitoring while your body fights the infection. In severe cases, antiviral drugs or a hospital stay is needed to prevent complications.

Most people diagnosed with chronic hepatitis B infection need treatment for the rest of their lives. The decision to start treatment depends on many factors, including: if the virus is causing inflammation or scarring of the liver, also called cirrhosis; if you have other infections, such as hepatitis C or HIV; or if your immune system is suppressed by medicine or illness. Treatment helps reduce the risk of liver disease and prevents you from passing the infection to others.

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Antiviral medications. Several antiviral medicines — including entecavir (Baraclude), tenofovir (Viread), lamivudine (Epivir), adefovir (Hepsera) and telbivudine — can help fight the virus and slow its ability to damage your liver. These drugs are taken by mouth. Your provider may recommend combining two of these medications or taking one of these medications with interferon to improve treatment response.

Interferon injections. Interferon alfa-2b (Intron A) is a man-made version of a substance produced by the body to fight infection. It's used mainly for young people with hepatitis B who wish to avoid long-term treatment or women who might want to get pregnant within a few years, after completing a finite course of therapy. Women should use contraception during interferon treatment. Interferon should not be used during pregnancy. Side effects may include nausea, vomiting, difficulty breathing and depression. Liver transplant. If your liver has been severely damaged, a liver transplant may be an option. During a liver transplant, the surgeon removes your damaged liver and replaces it with a healthy liver. Most transplanted livers come from deceased donors, though a small number come from living donors who donate a portion of their livers. Acute hepatitis is a term used to describe a wide variety of conditions characterized by acute inflammation of the hepatic parenchyma or injury to hepatocytes resulting in elevated liver function indices. In general, hepatitis is classified as acute or chronic based on the duration of the inflammation and insult to the hepatic parenchyma. If the period of inflammation or hepatocellular injury lasts for less than six months, characterized by normalization of the liver function tests, it is called acute hepatitis. In contrast, if the inflammation or hepatocellular injury persists beyond six months, it is termed chronic hepatitis. The most common infectious cause of acute hepatitis is secondary to a viral infection (acute viral hepatitis). Nevertheless, acute hepatitis can result from a wide variety of noninfectious causes that include but not limited to are drugs (drug-induced hepatitis), alcohol (alcoholic hepatitis), immunologic (autoimmune hepatitis, primary biliary cholangitis) or as a result of indirect insult secondary to biliary tract dysfunction (cholestatic hepatitis), pregnancy-related liver dysfunction, shock or metastatic disease. The histopathology of acute hepatitis is determined by the underlying etiology causing the hepatocellular injury. Acute hepatitis secondary to acetaminophen overdose demonstrates characteristic histological features such as central to central bridging necrosis and minimal inflammatory cell infiltrates. The histopathology features of acute hepatitis secondary to viral infections usually show intranuclear viral

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inclusions and surrounding neutrophils. Classical historical features of autoimmune hepatitis demonstrate portal inflammation and interface hepatitis formally known as piecemeal necrosis which is essentially the presence of portal inflammatory cells between the portal and liver parenchyma[10]. Diffuse microvesicular steatosis, Mallory bodies, fibrosis, or cirrhosis of the typical findings seen in alcohol-related liver injury[18]. Iron accumulation with hepatocellular hemosiderin pigment and increase hepatic copper concentrations and liver biopsy samples are the classical histopathological findings in patients with hereditary hemochromatosis and Wilson's disease respectively[19]. The microscopic changes in PSC and PBC are not pathognomonic for the condition. PBC is characterized by classical findings of florid duct lesion which is essentially granulomatous and lymphocytic portal inflammation centered around the interlobular bile ducts. The presence of concentric rings of fibrosis known as onion skin fibrosis is the hallmark historical features of PSC.

The clinical presentation of acute hepatitis depends on the underlying etiology. It can clinically manifest with various clinical signs and symptoms, ranging from asymptomatic elevated liver function tests to acute liver failure requiring liver transplantation. Hence, ascertaining the etiology of acute hepatitis is of utmost importance in its clinical management, making it very crucial to obtain a detailed history that should include the duration of the presenting illness, travel history, and assessing for high-risk activities like IV drug use, alcohol consumption, sexual history, prior blood-product transfusion history, or recent food intake. It is also imperative that drug history include not only recent or current prescription medications but also over-the-counter medications, acetaminophen (paracetamol), common cough/cold medications that contain acetaminophen, multivitamins and herbal/nutritional supplements. Patients with acute viral hepatitis commonly present with symptoms such as fever, malaise, fatigue, loss of appetite, vomiting, diarrhea, and abdominal pain. Patients may also report yellowish discoloration of their sclera (icterus) and /or skin (jaundice), dark-colored urine, and light-colored stools. Depending on the underlying etiology, physical exam findings can range from the presence of icterus and jaundice to signs of acute encephalopathy, seizures, bleeding diathesis, hypotension, and other manifestations related to multiple organ failure[5][12]. Signs of chronic liver disease such as caput medusae, spider nevi, palmar erythema, ascites, Dupuytren contracture, gynecomastia, and hepatic encephalopathy can be seen in patients presenting with acute on chronic liver disease.

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