TABLE 41-3 INTERPRETATION OF DIAGNOSTIC MARKERS IN HEPATITIS B

	HBsAg	HBeAg	ANTI-HBc IgM	ANTI-HBc IgG	ANTI-HBs	ANTI-HBe	BLOOD HBV DNA
Acute infection	+	+	+	+	–	+/–	High
Acute self-limited infection	–	–	+	+	+	+/–	–
Vaccinated	–	–	–	–	+	–	–
Chronic infection							
HBeAg positive	+	+	–	+	–	–	High
HBeAg negative	+	–	–	+	–	+	Low
Immune escape	+	–	–	+	–	+	High
Occult infection	–	–	–	+	–	+/–	Very low
Reactivation of chronic infection	+	+	+/–	+	–	+/–	High

anti-HBc IgG, Immunoglobulin G antibody against hepatitis B core antigen; anti-HBc IgM, immunoglobulin M antibody against hepatitis B core antigen; anti-HBe, antibody against hepatitis B e antigen; anti-HBs, antibody against hepatitis B surface antigen; HBeAg, hepatitis B e antigen; HBsAg, hepatitis B surface antigen; HBV DNA, hepatitis B virus deoxyribonucleic acid.

within 4 to 5 weeks after biochemical abnormalities are discovered. Importantly, these are not neutralizing antibodies and do not indicate immunity. At onset of symptoms, 30% of patients will be missed if checked by serum enzyme immunoassay (EIA) for HCV antibody alone.

Commercial EIAs for hepatitis E to detect both IgM and IgG class antibodies are also available but may lack sensitivity and specificity. Diagnosis of HEV infection should be established by PCR assays in immunosuppressed patients, because serologic testing is unreliable and seroconversion might never occur.

Complications

Cholestatic Hepatitis

In some patients, most commonly during HAV infection, a prolonged but self-limited period of cholestasis occurs that is characterized by marked conjugated hyperbilirubinemia, elevation of ALP, and pruritus. Further investigation may be required to rule out biliary obstruction (see Chapter 40).

Fulminant Hepatitis

Massive hepatic necrosis occurs in fewer than 1% of patients with acute viral hepatitis; it leads to a devastating and often fatal condition called acute liver failure. This condition is discussed in detail in Chapter 42.

Chronic Hepatitis

Hepatitis A does not progress to chronic liver disease, although occasionally it has a relapsing course. Persistence of elevated levels of ALT and AST, viral antigens, or nucleic acids beyond 6 months in patients with hepatitis B or C suggests evolution to chronic hepatitis, although slowly resolving acute hepatitis may occasionally exhibit such test abnormalities for up to 12 months with eventual complete resolution. About 60% of organ transplant recipients infected with HEV fail to clear the virus and go on to develop chronic hepatitis. Chronic hepatitis is considered in detail later in this chapter.

Rare Complications

Acute viral hepatitis may rarely be followed by aplastic anemia, which tends to affect mostly male patients and results in a mortality rate greater than 80%. Pancreatitis, myocarditis, pericarditis, pleural effusion, and neurologic complications including Guillain-Barré syndrome, aseptic meningitis, and encephalitis have also been reported. Cryoglobulinemia and glomerulonephritis are associated with hepatitis B and C, and polyarteritis nodosa is associated with hepatitis B. These manifestations are more common in patients who fail to clear acute HBV or HCV and develop chronic hepatitis.

Management

Unless complicated by fulminant hepatitis, cases of acute hepatitis A, B, and E are usually self-limited and are managed by supportive care including rest, maintenance of adequate hydration and dietary intake, and avoidance of alcohol use. Hospitalization may be needed for patients who cannot tolerate oral intake and for those with evidence of deteriorated liver function, such as hepatic encephalopathy or coagulopathy.

In general, hepatitis A and E may be regarded as noninfectious after 3 weeks, whereas hepatitis B is potentially infectious to sexual contacts throughout its course, although the risk is low once HBsAg has been cleared. Studies of antiviral therapy in acute hepatitis B have not shown clear benefit, although some experts advocate the use of nucleos(t)ide analogues, specifically in the setting of acute liver failure due to hepatitis B. Treatment of acute hepatitis C is not fully defined because it is often asymptomatic, although early treatment within 12 weeks of diagnosis with pegylated interferon-α–based therapy induces high sustained virologic response rates (>90%) and in responders may prevent the development of chronic infection.

Prevention

In patients with hepatitis A or E, both feces and blood contain virus during the prodromal and early icteric phases. General hygiene measures should include handwashing by contacts and careful handling, disposal, and sterilization of excreta, contaminated clothing, and utensils. HAV vaccination is appropriate for children older than 12 months of age, travelers to endemic areas, individuals with immunodeficiency or chronic liver disease, and those with high-risk behaviors or occupations. Since 2007, HAV vaccination has been preferred over immunoglobulin for postexposure prophylaxis, based on results from randomized trials. With the availability of two candidate vaccines, one of which is already licensed for use in China, HEV prevention through vaccination is now a realistic possibility.

HBV is rarely transmitted by body fluids other than blood; however, it is highly infectious, and strict adherence to universal precautions is mandatory. Efforts at preventing hepatitis B have involved the use of hepatitis B immunoglobulin (HBIG) and