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Hepatitis B Virus(HBV) infection: Current status

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DESCRIPTION

Hepatitis B Virus (HBV) infection has related to a really large spectrum of clinical presentations which will vary with age and therefore the immunological status. Hepatitis B Virus (HBV) infection may be a lifelong dynamic disease which will be controlled with treatment but cannot be cured yet. Risks of end-stage disease and hepatoma increase with ongoing inflammation and HBV viremia. Initial treatments consist of tenofovir or entecavir. Patients who require the treatment include those with chronic hepatitis, cirrhosis, and HCC or HIV coinfection; patients receiving immunosuppressive treatments; and ladies within the trimester of pregnancy who have elevated HBV DNA level. A number of virology and host immune approaches are being investigated with the aim of achieving HBV eradication.

HBV SERUM MARKERS

In the serum for each intact HBV virion, the sub-particles like HBsAg are present in vast numbers. Thus, HBV DNA is often below limit of detection, with HBsAg present in circulation. With reference to the serologic markers in HBV infection, HBsAg indicates acute or the chronic infection and it is that the first serologic marker to seem. Infection is taken into account chronic if it persisted for greater than 6 months. The presence of HBV e-antigen (HBeAg) indicates active replication of virus. Its absence can indicate absence of ongoing replication; absence also can indicate the mutations within the pre-core region of the e-antigen that prevent production of e-antigen.

Antibody to HBV core antigen (anti-HBc) is present in infection (IgM in acute infection) and with past exposure to HBV. It may be found alone when antibody to HBsAg (anti-HBs) wanes. Anti-HBs indicate recovery or immunity to HBV. It is detectable after immunity conferred by HBV vaccination, and is occasionally seen in chronic carriers. Anti-HBe antibody (anti-HBe) generally indicates that virus is not any longer replicating. However, it is often found in patients with HBeAg mutations (eantigen-negative patients) who have active disease. Patients who are HBsAg-positive and anti-HBc-positive should be mentioned look after HBV infection. Patients who are anti-HBs-positive and anti-HBc-positive have evidence of past infection in these cases; infection is latent but can reactivate in immunocompromised patients, with re-emergence of HBsAg.

Patients who are HBsAg-, anti-HBs-, and anti-HBc-negative lack evidence of immunity and can be vaccinated. Patients who are only anti-HBs-positive are immune or vaccinated.

CONTROL OF HBV

Control of HBV infection includes control of inflammatory components, indicated by normalization of serum alanine aminotransferase level and normal liver biopsy; virology control, indicated by reduction in HBV DNA; and immune control, indicated by seroconversion from HBeAg-positive to anti-HBe-positive and anti-HBs-positive to HBsAb-positive status.

HBV AND HIV

The Coinfection with HIV has been found to increase risk of HBV chronicity (i.e., reduce likelihood of HBsAg clearance in unvaccinated patients), increase antiretroviral-related hepatotoxicity, and increase risk of end-stage liver disease. Patients with coinfection have poorer hospital outcomes and better risk of progression to cirrhosis, HCC, and death than patients with either HIV or HBV mono-infection. Flares in patients with HBV/HIV co-infection are common.

Many HIV medications and much of over-the-counter medications are hepatotoxic, and other causes of ALT elevations in patients with HBV/HIV coinfection should be sought. Flares also can be associated with use of antiretroviral therapy without anti-HBV therapy and with stopping of antiretroviral therapy. Atypical serologic findings occur in patients with HIV coinfection during antiretroviral therapy. Individuals with HBV infection should receive anti-HBV treatment if they need elevated ALT level and elevated HBV DNA level. Treatment should even be given to patients who have cirrhosis and detectable HBV DNA, those are getting to receive chemotherapy or immune suppressive therapy, and people with HIV infection.