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Easl hep b guidelines 2017

ALT (alanine aminotransferase), HBeAg (hepatitis B virus envelope antigen), HBsAg (hepatitis B virus surface antigen), HBV (hepatitis B virus), HCC (hepatocellular carcinoma), ULN (upper limit of normal)Despite revolutionary advances in the treatment of hepatitis C virus infection, treatment of hepatitis B virus (HBV) infection remains a clinical challenge. Chronic HBV infection is typically indolent, and treatment is most often indefinite and suppressive. Thus, not all infected patients need to be treated. However, treatment should be offered to patients with a specific potential benefit. HBV treatment is with nucleos(t)ide analogues with a high barrier to resistance (eg, entecavir or tenofovir). Additional information on treatment options can be found in the major hepatology society guidelines.1Yuen M.F. Hou J.L. Chen C.J. et al.Asian-Pacific clinical practice guidelines on the management of hepatitis B: a 2015 update., . We present the following case to illustrate the salient characteristics that inform decisions about when to treat chronic HBV infection.An otherwise healthy 25-year-old woman is seen in consultation for abnormal hepatic biochemical tests. She is asymptomatic, drinks no alcohol, and has no history of parenteral exposure risk to hepatitis B or C. There is a maternal history of hepatocellular carcinoma (HCC). She has an unremarkable physical exam (body mass index 20 kg/m2, without jaundice, ascites, edema, or stigmata of chronic liver disease). Her alanine aminotransferase (ALT) level is 35 U/L (the laboratory provided normal range is 14–54 U/L); on review of the record, ALT is noted to have varied from 35 to 48 U/L during the past 5 years. Hepatitis B virus surface antigen (HBsAg) is present. During the same period, HBV DNA has been 3–4 log IU/mL (1055–10,200 IU/mL). Hepatitis B virus envelope antigen (HBeAg) is not detected. Genetic analysis reveals a basal core-promoter mutation (A1762T/G1764A). An ultrasound of the liver is unremarkable, and liver stiffness measured by vibration-controlled transient elastography is 7.0 kPa.Assessment of the patient with HBV infection includes confirmation of active infection marked by the presence of HBsAg and HBV DNA, staging of hepatic fibrosis via liver biopsy or noninvasive modalities (eg, elastography or serologic markers), and estimate of hepatic inflammatory activity by liver biopsy or serum markers (eg, ALT). The presence of additional serologic markers such as HBeAg also informs treatment decisions. Furthermore, HBV infection is dynamic and has a fluctuating clinical and virologic course. Thus, a treatment decision should be deferred until the patient meets criteria for treatment that are based on at least 2 and preferably 3 evaluations about 3 months apart.The intention of treatment is to prevent or forestall complications of HBV, which may increase in prevalence with patient age/duration of infection, fibrosis stage, inflammatory activity, and HBV DNA level (Figure 1).4Jen C.-L. Lu S.-N. Yang H.-I. et al.Prediction models of long-term cirrhosis and hepatocellular carcinoma risk in chronic hepatitis B patients: risk scores integrating host and virus profiles.Crossref PubMed Scopus (197) Google Scholar The foremost indication for treatment is mitigation of progressive liver disease. The patient described in the vignette above is young with unknown duration of infection. Her estimated liver fibrosis stage is likely near or just below the “moderate/significant” threshold on the basis of liver stiffness measurement. At least periportal fibrosis with few septae formation (F2) by biopsy staging is generally accepted as an indication for treatment. Her HBV DNA level has been variable but at times elevated (ie, >2000 IU/mL). Although there is no histologic assessment provided, the elevated ALT may suggest necroinflammatory activity, which also typically indicates treatment. Notably, although this patient’s ALT falls within the laboratory-reported normal range, the true upper limit of normal (ULN) for a healthy woman is 19–25 U/L. Furthermore, there is no other apparent etiology for the abnormal ALT. Last, she has a family history of HCC, which is associated with a markedly increased risk of HCC among patients with chronic HBV infection (hazard ratio, 32).Figure 1Treatment is indicated by evidence of significant and/or progressive fibrosis or necroinflammatory activity or in special circumstances (eg, immunosuppression, pregnancy) when the risk of adverse outcomes is especially high. ALT, alanine aminotransferase; CP, core promoter; HBeAg, hepatitis B envelope antigen; HBV, hepatitis B virus; HCC, hepatocellular carcinoma; HCV, hepatitis C virus; HIV, human immunodeficiency virus; INR, international normalized ratio; PC, precore; ULN, upper limit of normal. *Examples of moderate immunosuppression include treatment with anti-tumor necrosis factor or other cytokine/integrin inhibitors, tyrosine kinase inhibitors, or less than 10 mg/day prednisone (or equivalent) for at least 1 month. **Examples of marked immunosuppression include treatment with anthracycline derivatives or 10–20 mg prednisone/day (or greater) for at least 1 month.Although less well-defined, there are other viral parameters associated with increased risk of cirrhosis and/or HCC, including elevated quantitative HBsAg level, the presence of a pre-core or basal core promoter mutation, and HBV genotype C.1Yuen M.F. Hou J.L. Chen C.J. et al.Asian-Pacific clinical practice guidelines on the management of hepatitis B: a 2015 update., . Basal core promoter and pre-core mutations impair transcription or formation of HBeAg, respectively, and thus may result in undetectable HBeAg despite active HBV replication and a fluctuating ALT level. Presence of these mutations may increase risk of cirrhosis or HCC. Among genotypes common in the United States (A, B, and C), HBV genotype C appears to be associated with more necroinflammatory activity, more rapid progression of fibrosis, and higher incidence of HCC. It is also associated with a lower rate of HBeAg seroconversion.Major hepatology societies provide relevant guidance for the management of HBeAg-negative chronic HBV infection, with general agreement (Table 1).1Yuen M.F. Hou J.L. Chen C.J. et al.Asian-Pacific clinical practice guidelines on the management of hepatitis B: a 2015 update., . Overall, treatment is recommended for patients with a certain threshold for HBV DNA, ALT elevation, fibrosis, necroinflammatory activity, or age or in patients with a family history of significant liver disease (ie, cirrhosis or HCC).Table 1Recommendations for Treatment of HBeAg-negative Chronic Hepatitis B InfectionALT, alanine aminotransferase; HBeAg, hepatitis B virus envelope antigen; HBV, hepatitis B virus; HCC, hepatocellular carcinoma; ULN, upper limit of normal.For patients with HBeAg-positive disease there are several additional indications for treatment: elevated HBV DNA (>20,000) and age >30 years and/or ALT >2× ULN or significant fibrosis or inflammatory activity.1Yuen M.F. Hou J.L. Chen C.J. et al.Asian-Pacific clinical practice guidelines on the management of hepatitis B: a 2015 update., . In addition, treatment is indicated if HBV DNA is 2000–20,000 and ALT >2× ULN for 6 months.In cases of acute HBV infection or reactivation complicated by severe acute liver injury, marked by elevation of the international normalized ratio (≥2.0, ALT ≥10× ULN, and bilirubin ≥3.0 mg/dL) or acute liver failure (international normalized ratio ≥1.5 with hepatic encephalopathy), immediate HBV treatment may be initiated., The benefit of such treatment is uncertain, but the risk of short-term treatment appears low and the potential benefit is high if death or liver transplantation can be avoided or if HBV DNA is suppressed before liver transplantation.Additional indications for treatment include reduction of risk caused by chronic, high morbidity complications, such as cirrhosis or HCC, or amelioration of extrahepatic complications, such as vasculitis or glomerulonephritis., All patients with cirrhosis and detectable HBV DNA and positive HBsAg should be treated.1Yuen M.F. Hou J.L. Chen C.J. et al.Asian-Pacific clinical practice guidelines on the management of hepatitis B: a 2015 update., . Even in the absence of other HBV treatment indications, HBV treatment may be considered after resection of HCC, which may reduce the risk of recurrence.7Wu C.Y. Chen Y.J. Ho H.J. et al.Association between nucleoside analogues and risk of hepatitis B virus-related hepatocellular carcinoma recurrence following liver resection.Crossref PubMed Scopus (592) Google Scholar.8Chan A.C.Y. Chok K.S.H. Yuen W.K. et al.Impact of antiviral therapy on the survival of patients after major hepatectomy for hepatitis B virus-related hepatocellular carcinoma.Crossref PubMed Scopus (89) Google ScholarRisk of transmission of HBV may be more likely from patients with elevated HBV DNA level. Treatment is thus indicated to reduce the potential for transmission among pregnant women (HBV DNA >200,000 IU/mL beginning at 28–32 weeks’ gestation)., It is recommended for healthcare workers (HBV DNA >1000 IU/mL for those with decreased skin integrity or who participate in procedures with increased risk of puncture injury).11Frieden T.R. Jaffe H.W. Stephens J.W. et al.Updated CDC recommendations for the management of hepatitis B virus-infected health-care providers and students.Last, treatment of patients with detectable HBsAg or HBV DNA is indicated by ongoing or expected immunosuppression, eg, anti-rejection treatment for organ transplantation, prolonged corticosteroid use (equivalent of ≥10 mg prednisone daily for ≥1 month), or the use of B-cell depleting agents (eg, rituximab or ofatumumab) or tumor necrosis factor-α inhibitors. Post-transplant prophylaxis is also indicated for liver transplant recipients who have detectable HBsAg before transplantation or receive an organ from a donor with detectable HBsAg or anti-HBV core antibody. Prophylactic treatment of patients with detectable anti-HBV core antibody without detectable HBsAg or HBV DNA is often indicated in those with ongoing or expected major immunosuppression, and guidance on this topic has been previously published.,Many patients with chronic HBV infection do not require treatment, although all should receive periodic monitoring consisting of at least measurement of ALT and HBV DNA in addition to clinical assessment. Patients with “immune tolerant” chronic HBV infection, marked by undetectable HBeAg, persistently normal ALT, and elevated HBV DNA (>2× 107 IU/mL) generally do not require treatment. Similarly, patients in a phase of chronic hepatitis B with low viral replication (often referred to as an “inactive” state), characterized by undetectable HBeAg/detectable HBeAb, persistently normal ALT, and low HBV DNA (

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