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Despite universal vaccination, chronic hepatitis B (CHB) continues to be a major health burden worldwide. Over 400,000 people worldwide are chronically infected with hepatitis B virus (HBV), are at increased risk of developing hepatocellular carcinoma (HCC) and cirrhosis. HBV infected persons need regular lifelong follow-up [1,2]. HBV infection is common with major clinical consequences worldwide. In Asian Americans, the HBsAg carrier rate ranges from 7 to 16%; HBV is the most important cause of chronic hepatitis, cirrhosis, and HCC [3].

Turkey Liver Research Association's throughout Turkey in 2010, according to a survey conducted in Turkey is estimated to be 3 million people with chronic hepatitis B. Hepatitis B virus carriers, representing 4% of HBsAg, hepatitis B virus immune status of anti-HBs 32%, anti-HDV positivity was found to be 2.7%. HBsAg positivity rates by region of HBV is most commonly seen in regions of Central and Southeastern Anatolia Region, at least seen the eastern regions of the Aegean and Central Anatolian region [4,5].

Of the estimated 50 million new cases of HBV infection diagnosed annually, 5-10% of adults and up to 90% of infants will become chronically infected, 75% of these in Asia where hepatitis B is the leading cause of chronic hepatitis, cirrhosis and HCC. Prevention of HBV infection thorough vaccination is still, therefore, the best strategy for decreasing the incidence of hepatitis B-associated cirrhosis and HCC [6].

e diagnosis of CHB is made using a combination of serological, virologic, biochemical, and histologic markers. e natural history of HBV infection can be divided into four phases: immune tolerance, immune clearance (HBeAg-positive chronic hepatitis B), inactive HBsAg carrier, and reactivation (HBeAg-negative chronic hepatitis B). Patients in the immune clearance and reactivation phases, with elevated alanine aminotransferase (ALT) and HBV DNA levels, are candidates for antiviral therapy [7].

e presence of HBV replication markers--hepatitis B e antigen (HBeAg) or HBV DNA--is associated with continuing hepatitis activity or intermittent hepatitis ares and subsequent disease progression, including hepatic decompensation and development of liver cirrhosis or HCC. e average rate of spontaneous HBeAg seroconversion is 10% per year. About 2.1% of patients with chronic type B hepatitis develop cirrhosis each year. e ultimate outcome of chronic HBV infection appears to depend on the duration and severity of liver injury during the immune clearance phase. About 2.1% of patients with chronic type B hepatitis develop cirrhosis each year.. e development of HCC related to the severity of the underlying liver disease. e annual incidence of HCC is only 0.1% in asymptomatic HBsAg individual, 1% in patients with chronic hepatitis B, but increases to 3-10% in patients with cirrhosis. e outcome of HBV-infected persons with 'spontaneous' seroclearance of HBsAg is usually favourable, though progress to cirrhosis and HCC is still possible [8,9].

Natural history and outcome, severity of liver damage and need for liver biopsy and antiviral treatment di er signi cantly between these groups of patients. It is not always easy to distinguish between inactive HBV carriers and patients su ering from HBeAg-negative chronic hepatitis with transient disease remission, as they share similar biochemical (normal serum ALT values) and virological (HBeAg negativity and low HBV DNA levels) features. In clinical practice, it is very important to di erentiate inactive carriers from patients with chronic hepatitis B with spontaneous transient remission [10]. Liver biopsies usually were scored by using the modi ed histology activity index score of Knodell and the Ishak brosis score [11].

e primary determinant of treatment outcomes for CHB is suppression of serum HBV DNA, and long-term suppression of viral replication is likely to reduce progression to cirrhosis and HCC [7]. Candidates for antiviral therapy include patients with moderate-to-severe liver disease as determined by elevated alanine aminotransferas and/or liver biopsy and elevated HBV DNA levels above 2000 IU/mL, per evidenced-based guidelines [3].

e approval of potent oral antiviral agents has revolutionised hepatitis B treatment since 1998. Current antiviral treatment options for CHB include interferon alfa-2b, peginterferon alfa-2a, lamivudine, adefovir, entecavir, telbivudine, and tenofovir. In patients with HBeAg-positive CHB, antiviral treatment is indicated when the serum HBV DNA level is 20 000 IU/mL and the ALT level is elevated. For HBeAg-negative (en)4 (o)12 (f)9 (o)16 (v)- l ,m Hwals (v)hndapos (s in)4 (di

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