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Viral Hepatitis B In Pregnant Women

Yusupov Sh.R., Otajanov Sh.Z., Saparova Q.K.
Urgench state medical institute

Abstract. Chronic hepatitis B (CHB) remains a major global public health concern. This review outlines current approaches to managing CHB in pregnant individuals. The rate of HBV infection in pregnancy generally mirrors that of the wider population in a given region. Universal screening for hepatitis B surface antigen (HBsAg) is essential during pregnancy. For those who test positive, further assessment may involve measuring hepatitis B e antigen, HBV DNA levels, alanine aminotransferase, and HBsAg quantitation. Clinical management is guided by the phase of HBV infection, which should ideally be established prior to conception. For individuals with CHB who may become pregnant, antiviral therapy serves two key purposes: to treat active liver disease and to prevent mother-to-child transmission. Tenofovir is the recommended antiviral agent during pregnancy for both indications. To prevent vertical transmission, every newborn to an HBsAg-positive mother should receive hepatitis B immunoglobulin and the first dose of hepatitis B vaccine within 12 hours of birth. Breastfeeding is not contraindicated for mothers with CHB.
Key Words: Chronic hepatitis B; Hepatitis B viral load; Pregnancy; Antiviral treatment.

Introduction. Chronic hepatitis B (CHB) remains a major global public health concern. According to World Health Organization (WHO) estimates, approximately 257 million individuals worldwide were living with CHB in 2015 [1,2]. The prevalence of hepatitis B virus (HBV) infection shows marked geographic variability, with regions classified as having high (>8%), intermediate (2%–8%), or low (<2%) endemicity. The clinical spectrum of CHB ranges from inactive hepatitis B surface antigen (HBsAg) carriage to progressive disease characterized by liver fibrosis, cirrhosis, and the development of hepatocellular carcinoma (HCC). Although widespread hepatitis B vaccination programs have significantly reduced HBV transmission, mother-to-child transmission (MTCT) remains a major route of infection. Each year, an estimated 4–5 million children acquire HBV infection from mothers with CHB [3]. In highly endemic areas, more than half of CHB cases result from infection occurring at birth or during early childhood [4]. This mode of transmission is particularly concerning because HBV infection acquired early in life leads to chronic infection in the majority of cases, whereas fewer than 20% of adults infected with HBV develop CHB.

In the absence of preventive measures, the risk of MTCT is strongly influenced by maternal hepatitis B e antigen (HBeAg) status. Transmission rates are approximately 70%–90% among HBeAg-positive mothers, compared with 10%–40% among HBeAg-negative mothers [5]. In 2016, the WHO established the goal of eliminating viral hepatitis as a major public health threat by 2030. Achieving this objective requires effective prevention of vertical HBV transmission. Therefore, implementing comprehensive strategies for the management of pregnancy in women with CHB is essential to reduce the risk of HBV MTCT.

In both developed and developing countries, universal screening of pregnant women for hepatitis B surface antigen (HBsAg) is routinely implemented. Limiting screening to so-called high-risk groups—such as intravenous drug users, individuals with multiple sexual partners, sex workers, or those with sexual contact with HBsAg-positive individuals—has proven insufficient, as this approach fails to identify up to 50% of pregnant women with CHB. Special attention should be paid to women in whom CHB is diagnosed for the first time during pregnancy, as acute hepatitis B must be excluded in these cases.

The scope of additional investigations for pregnant women with CHB varies by region. Recommendations from leading hepatology societies regarding the evaluation of pregnant women with CHB are summarized in Table 1. Most guidelines emphasize the importance of assessing HBV viral load to determine the need for antiviral therapy during pregnancy; however, they differ with respect to the optimal timing of both testing and treatment initiation. In general, HBV DNA levels should be measured no later than the 30th week of gestation.

Currently, quantitative determination of HBsAg levels during pregnancy is included only in European clinical practice guidelines for CHB management. Nevertheless, available evidence demonstrates a significant association between maternal HBsAg levels and the risk of mother-to-child transmission.

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Compared with viral load, HBsAg levels remain more stable throughout pregnancy and are less costly to measure. Consequently, HBsAg quantification may serve as a useful predictor of vertical HBV transmission, particularly in resource-limited settings. In pregnant women with low HBsAg levels, additional HBV DNA testing may not be necessary.

The prevalence of hepatitis B virus (HBV) infection among pregnant women generally reflects the prevalence observed in the overall population within the same geographic region. In China, the prevalence of HBV infection among women of reproductive age ranges from 2% to 8%, whereas in the United States it is approximately 0.4%. Data on the proportion of HBsAg-positive pregnant women in selected countries are presented in Table 2. Currently, HBV prevalence among pregnant women remains high in many African countries, while relatively low rates are reported in Europe and the Americas. Notably, even in China—historically a region of high HBV endemicity—a substantial decline in the proportion of HBsAg-positive pregnant women has been observed in recent years.

Most investigators recognize five distinct phases in the natural history of chronic hepatitis B. The initial phase, referred to as the immune-tolerant phase, typically develops following perinatal infection and is characterized by a prolonged, minimally symptomatic course, normal serum alanine aminotransferase (ALT) levels, and minimal histological liver changes. As shown in Table 3, patients in this phase are HBeAg-positive and usually exhibit very high levels of HBV DNA (approximately 10^8–10^9 IU/mL). In individuals infected during adulthood, this phase is generally short-lived.

The second phase, known as the immune-active or immunoreactive phase, most commonly occurs in patients infected at birth or during early childhood and typically begins after two to three decades. This phase is marked by intermittent elevations in ALT levels and a moderate reduction in HBV DNA concentrations compared with the immune-tolerant phase, reflecting increased host immune activity against the virus. The age at which this phase develops varies according to HBV genotype and geographic region. For example, in Taiwan, approximately 90% of HBeAg seroconversion occurs before the age of 40 years, with earlier seroconversion observed in patients infected with genotype B compared with genotype C. In Europe, fewer than 30% of patients remain HBeAg-positive beyond the age of 40 years [30]. This observation is clinically relevant, as earlier pregnancy increases the likelihood that a woman is in the immune-tolerant phase, characterized by high viral replication and, consequently, an increased risk of mother-to-child transmission.

The third phase, referred to as the inactive HBsAg carrier state, is defined by persistent HBsAg positivity, absence of HBeAg, and low (<2000 IU/mL) or undetectable HBV DNA levels. ALT levels remain within the normal range, fibrosis progression is minimal or absent, and spontaneous HBsAg seroclearance may occur. This phase may persist for decades and is associated with a low risk of vertical transmission.

The fourth phase, termed HBeAg-negative chronic hepatitis B, is characterized by a fluctuating clinical course with periodic ALT elevations. HBV DNA levels vary widely, whereas HBsAg concentrations tend to remain relatively stable [33]. HBeAg is absent, liver fibrosis gradually progresses, and the risk of hepatocellular carcinoma increases. In this phase, the likelihood of vertical transmission largely depends on the level of viral replication.

The fifth and final phase, known as the HBsAg-negative or occult HBV infection phase, is characterized by the loss of detectable HBsAg despite ongoing low-level viral replication within the liver. Clinical manifestations are usually mild or absent, and ALT levels are typically normal. Reactivation of CHB may occur, particularly in the setting of immunosuppression, including the physiological immunosuppression associated with pregnancy. Although only a limited number of cases of HBV reactivation during pregnancy have been reported [34,35], the overall risk of vertical transmission in this phase remains low.

The management of pregnancy in women with HBV infection depends largely on the phase of the disease. However, many women are diagnosed with CHB for the first time during pregnancy. Therefore, screening for viral hepatitis markers prior to conception is strongly recommended. Accurate staging of CHB during pregnancy is challenging, as several laboratory and clinical parameters change physiologically during gestation. For instance, alpha-fetoprotein levels increase early in pregnancy, and conditions such as early-pregnancy toxicosis or hyperemesis gravidarum may lead to elevated cytolytic markers, complicating the

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interpretation of ALT fluctuations. Furthermore, certain diagnostic tools, including transient elastography, may yield unreliable results due to pregnancy-related changes in circulating blood volume. Consequently, determining the phase of CHB before pregnancy is preferable whenever possible.

In general, women of reproductive age do not present with advanced liver fibrosis or cirrhosis. However, with the increasing age at first pregnancy and the historical predominance of vertical transmission prior to the widespread implementation of neonatal hepatitis B vaccination, cases of chronic hepatitis B (CHB) with advanced fibrosis are not uncommon. Pregnancy in women with liver cirrhosis is associated with an elevated risk of maternal complications.

In most cases, CHB does not worsen during pregnancy, and markers of cytolytic activity typically normalize. Nevertheless, sporadic cases of CHB exacerbation during pregnancy have been reported, including rare instances of fulminant hepatic failure. HBV viral load may fluctuate during pregnancy, and reactivation of CHB has been documented. In one study, HBV DNA was undetectable in the first trimester but became detectable in 19.6% of women during the second trimester and in 30.4% during the third trimester. Another study demonstrated an increase in viral load during pregnancy followed by a decline after delivery.

Several investigations have also reported hepatitis flares in the early postpartum period. While ALT levels tend to decrease during pregnancy in most women, a marked rise in cytolytic activity is commonly observed after childbirth. For example, ALT elevations of threefold or greater occurred in approximately 45% of women within six months postpartum. HBeAg seroconversion during pregnancy has been described in 12.5%–17% of patients.

The clinical presentation of CHB in pregnant women is dominated by asthenic and dyspeptic symptoms, reported in approximately 63% of cases. Hemorrhagic manifestations, such as gingival bleeding, occur in about 15% of patients, while hepatomegaly is observed in roughly 10%. The impact of chronic maternal HBV infection on pregnancy outcomes remains incompletely understood, and published data are inconsistent. Some studies have found no association between maternal CHB and adverse pregnancy outcomes, whereas others report generally favorable perinatal outcomes, with the exception of lower Apgar scores in newborns.

Conversely, several studies have identified an increased incidence of adverse outcomes—including fetal distress, preterm labor, and meconium peritonitis—among HBV-infected women and their infants. A large cohort study conducted in China demonstrated that HBsAg-positive pregnant women had a higher risk of gestational diabetes mellitus, postpartum hemorrhage, and intrahepatic cholestasis of pregnancy. Additionally, a recent study identified a significant association between maternal HBV viral load and blood glucose parameters, including fasting glucose, 2-hour postprandial glucose, and hemoglobin A1c levels. No statistically significant associations were observed between HBsAg positivity and pre-eclampsia or placenta previa.

Overall, HBsAg positivity during pregnancy has been linked to an increased risk of multiple adverse maternal outcomes. A large case–control study from China showed that maternal HBsAg carriage was associated with pregnancy-induced hypertension, fetal distress, cesarean delivery, and macrosomia. The same study also reported a significant association between high maternal viral load during the second trimester and an increased risk of preterm birth. Although several previous studies support an association between maternal HBV infection and preterm delivery, contradictory findings have also been reported. Some investigations have described a higher frequency of bleeding during labor among women with CHB, whereas others have suggested a lower incidence of hypertension and pre-eclampsia in this population.

At present, available therapeutic strategies for CHB are unable to achieve complete eradication of HBV. Therefore, treatment goals should be individualized and may include: (1) suppression of viral replication; (2) reduction of hepatic inflammation; (3) regression or stabilization of liver fibrosis; (4) prevention of cirrhosis and hepatocellular carcinoma; and (5) reduction of the risk of vertical transmission of HBV.

When selecting antiviral therapy, both the safety and efficacy of the agents, as well as the potential for the development of antiviral resistance, must be carefully considered. In women of reproductive age with chronic hepatitis B (CHB), antiviral treatment generally serves two primary purposes: management of active CHB and prevention of mother-to-child transmission (MTCT) (Table 4). The indication for treating inactive

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HBsAg carriers remains controversial. Currently, such treatment is recommended only by the Asia-Pacific Association for the Study of the Liver (APASL), whereas the European Association for the Study of the Liver (EASL) and the American Association for the Study of Liver Diseases (AASLD) do not support this approach.

Evidence from large clinical trials demonstrates a reduction in HBV transmission and HBsAg positivity among infants born to HBsAg-positive mothers treated with telbivudine or lamivudine. Furthermore, a systematic review has shown that antiviral therapy with nucleos(t)ide analogues (NAs), including lamivudine, telbivudine, and tenofovir, significantly reduces maternal HBV DNA levels during pregnancy. Among these agents, tenofovir is currently considered the preferred option in pregnant women because of its potent antiviral activity and low risk of resistance development. Tenofovir is generally well tolerated during pregnancy and effectively suppresses viral replication prior to delivery.

NA prophylaxis may also be beneficial for HBeAg-negative women with high HBV DNA levels but normal alanine aminotransferase (ALT) values. Initiation of NA therapy at 28–30 weeks of gestation results in a rapid decline in viral load by the time of delivery, thereby significantly reducing the risk of vertical transmission. However, discontinuation of therapy is typically followed by a rapid rebound in HBV DNA levels.

Several studies have demonstrated the efficacy of telbivudine initiated during the third trimester in women with high viral loads. In one study, telbivudine therapy reduced HBV DNA to undetectable levels at delivery in 33% of treated women, whereas no such reduction was observed in the control group. Moreover, no cases of MTCT were reported among infants born to treated mothers, compared with an HBsAg positivity rate of 8% at seven months of age in the control group. Similarly, a large prospective study involving 450 HBeAg-positive women with high viral loads reported zero cases of vertical transmission in the telbivudine-treated group, whereas 14.7% of infants in the untreated group were HBsAg-positive six months after birth.

When antiviral therapy is administered solely to prevent MTCT, it is generally discontinued after delivery. However, there is no consensus regarding the optimal timing of treatment cessation. According to AASLD guidelines, therapy may be stopped shortly after delivery; EASL recommends discontinuation at delivery or within the first three months postpartum; and APASL suggests continuing treatment for 4–12 weeks after childbirth.

Neonatal hepatitis B vaccination alone reduces the risk of vertical transmission from approximately 90% to 21% in infants born to HBeAg-positive mothers and from 30% to 2.6% in those born to HBeAg-negative mothers. The addition of hepatitis B immunoglobulin (HBIG) further decreases the MTCT risk to 6% and 1%, respectively [60]. To achieve optimal efficacy, this combined prophylaxis must be administered within the first 12 hours after birth.

Conclusion. Despite the sustained decline in the prevalence of chronic hepatitis B (CHB) following the implementation of universal hepatitis B vaccination programs, CHB continues to pose a major global public health challenge. In this review, we have summarized current trends in the management of CHB during pregnancy and proposed evidence-based recommendations aimed at achieving the World Health Organization’s goal of eliminating hepatitis B as a public health threat.The key clinical recommendations are as follows: (1) universal screening of all pregnant women for hepatitis B surface antigen (HBsAg) is essential; further evaluation of HBsAg-positive women may include assessment of HBeAg status, HBV DNA levels, alanine aminotransferase (ALT), and quantitative HBsAg levels; (2) pregnancy management should be guided by the phase of HBV infection, which ideally should be established prior to conception; (3) antiviral therapy in women of reproductive age with CHB serves two principal purposes—treatment of active disease and prevention of mother-to-child transmission—with tenofovir representing the preferred agent during pregnancy; and (4) all infants born to mothers with CHB should receive combined prophylaxis with hepatitis B immunoglobulin and hepatitis B vaccine within the first 12 hours after birth. Under these conditions, breastfeeding is not contraindicated.

REFERENCES

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1. World Health Organization. Global Hepatitis Report 2017. [cited 15 April 2021]. In: World Health Organization [Internet]. Available from: https://www.who.int/hepatitis/publications/global-hepatitisreport2017/en
2. Aslam A, Campoverde Reyes KJ, Malladi VR, Ishtiaq R, Lau DTY. Management of chronic hepatitis B during pregnancy. Gastroenterol Rep (Oxf) 2018; 6: 257-262 [PMID: 30430013 DOI: 10.1093/gastro/goy025]
3. Thio CL, Guo N, Xie C, Nelson KE, Ehrhardt S. Global elimination of mother-to-child transmission of hepatitis B: revisiting the current strategy. Lancet Infect Dis 2015; 15: 981-985 [PMID: 26145195 DOI: 10.1016/S1473-3099(15)00158-9]
4. Alter MJ. Epidemiology of hepatitis B in Europe and worldwide. J Hepatol 2003; 39 Suppl 1: S64S69 [PMID: 14708680 DOI: 10.1016/s0168-8278(03)00141-7]
5. Tran TT. Hepatitis B in Pregnancy. Clin Infect Dis 2016; 62 Suppl 4: S314-S317 [PMID: 27190321 DOI: 10.1093/cid/ciw092]
6. World Health Organization. Global health sector strategy on viral hepatitis 2016–2021. [cited 15 April 2021]. In: World Health Organization [Internet]. Available from: https://apps.who.int/iris/bitstream/handle/10665/246177/ WHO-HIV-2016.06-eng.pdf?sequence=1
7. Jonas MM. Hepatitis B and pregnancy: an underestimated issue. Liver Int 2009; 29 Suppl 1: 133-139 [PMID: 19207977 DOI: 10.1111/j.1478-3231.2008.01933.x]
8. Sarin SK, Kumar M, Lau GK, Abbas Z, Chan HL, Chen CJ, Chen DS, Chen HL, Chen PJ, Chien RN, Dokmeci AK, Gane E, Hou JL, Jafri W, Jia J, Kim JH, Lai CL, Lee HC, Lim SG, Liu CJ, Locarnini S, Al Mahtab M, Mohamed R, Omata M, Park J, Piratvisuth T, Sharma BC, Sollano J, Wang FS, Wei L, Yuen MF, Zheng SS, Kao JH. Asian-Pacific clinical practice guidelines on the management of hepatitis B: a 2015 update. Hepatol Int 2016; 10: 1-98 [PMID: 26563120 DOI: 10.1007/s12072-015-9675-4]
9. European Association for the Study of the Liver. EASL 2017 Clinical Practice Guidelines on the management of hepatitis B virus infection. J Hepatol 2017; 67: 370-398 [PMID: 28427875 DOI: 10.1016/j.jhep.2017.03.021]
10. Terrault NA, Lok ASF, McMahon BJ, Chang KM, Hwang JP, Jonas MM, Brown RS Jr, Bzowej NH, Wong JB. Update on prevention, diagnosis, and treatment of chronic hepatitis B: AASLD 2018