



Management of the Pregnant Woman with Chronic Hepatitis B: A Literature Review

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Abstract

Chronic hepatitis B (CHB) remains a major public health problem, and during pregnancy it carries particular significance because of the risk of vertical transmission, which in most cases occurs during labour and delivery. Although pregnancy is generally well tolerated in women with hepatitis B virus (HBV) infection, the peripartum and postpartum periods may be associated with hepatic flares, and the absence of neonatal prophylaxis results in a substantial burden of new infections. This narrative review synthesises current evidence on the clinical management of CHB in pregnancy, with emphasis on the pathophysiology of HBV infection in gestation, initial maternal assessment, stratification of the risk of vertical transmission, universal antenatal screening, indications for and timing of antiviral therapy, the antivirals available during pregnancy, intrapartum and delivery management, breastfeeding, neonatal prophylaxis and maternal postpartum follow-up. The maternal viral load and hepatitis B e antigen (HBeAg) status are the principal determinants of transmission risk, and antiviral prophylaxis with tenofovir, initiated in the late second or third trimester in women with HBV DNA above 200,000 IU/mL, is an effective adjunct to neonatal immunoprophylaxis. Timely administration of the hepatitis B vaccine and hepatitis B immunoglobulin to the exposed newborn remains the cornerstone of prevention. Care should be understood as a continuous pathway extending from antenatal screening through delivery, the neonatal period, breastfeeding and long-term maternal and infant follow-up, requiring coordinated multidisciplinary management to support the goal of eliminating hepatitis B as a public health threat.

Keywords: Chronic hepatitis B; Pregnancy; Vertical transmission; Antiviral prophylaxis; Tenofovir; Neonatal Immunoprophylaxis

Abbreviations

AASLD: American Association for the Study of Liver Diseases; ACOG: American College of Obstetricians and Gynecologists; AFP: Alpha-Fetoprotein; AGA: American Gastroenterological Association;

on; ALT: Alanine Aminotransferase; APRI: Aspartate Aminotransferase-To-Platelet Ratio Index; AST: Aspartate Aminotransferase; CHB: Chronic Hepatitis B; EASL: European Association for the Study of the Liver; HBeAg: Hepatitis B E Antigen; HBIG: Hepatitis B Immunoglobulin; HBsAg: Hepatitis B Surface Antigen; HBV: He-

patitis B Virus; HCC: Hepatocellular Carcinoma; IDSA: Infectious Diseases Society of America; INR: international Normalised Ratio; SMFM: Society for Maternal-Fetal Medicine; TAF: Tenofovir Alafenamide; TDF: Tenofovir Disoproxil Fumarate; ULN: Upper Limit Of Normal; WHO: World Health Organization.

Introduction

Chronic hepatitis B (CHB) constitutes a major public health problem, with a prevalence of approximately 3.2% to 3.5% of the world population [1]. In pregnancy, HBV infection has significant repercussions, above all because of the risk of vertical transmission, which in most cases occurs during labour and delivery [2]. From the maternal standpoint, pregnancy is generally well tolerated in women with HBV, despite the risk of exacerbation, particularly in the postpartum period [3], so that adequate follow-up is required to assess the disease, quantify the viral load, evaluate the safety of antiviral therapy and adopt measures to prevent maternal-fetal transmission. From the neonatal standpoint, the absence of prophylaxis results in a substantial impact on the number of infections. The management of CHB in pregnancy is therefore relevant for synthesising and systematising the available evidence, in order to optimise measures to prevent vertical transmission and to improve maternal and neonatal follow-up, favouring better outcomes for the maternal-fetal dyad.

Methods

This is a narrative review of the literature on the clinical management of CHB during pregnancy, with emphasis on prevention of vertical transmission, antiviral prophylaxis, maternal-fetal monitoring and management at delivery and in the puerperium. Medical literature databases were consulted, including direct searches in PubMed, the Cochrane Library and SciELO, together with national and international guidelines such as those of the WHO and FEBRASGO and the OpenEvidence platform (which aggregates guidelines, clinical trials, systematic reviews and package inserts from regulatory agencies such as the FDA, as well as data from bodies such as the CDC). Search descriptors included “chronic hepatitis B” AND “vertical transmission” OR “mother-to-child transmission”. Inclusion criteria were original studies, systematic reviews, meta-analyses and clinical guidelines addressing diagnosis, risk stratification, antiviral prophylaxis, timing of initiation and discontinuation of treatment, mode of delivery and neonatal care in pregnant women

with CHB, published between 2016 and 2026. Exclusion criteria comprised studies dealing exclusively with acute hepatitis B unrelated to pregnancy and isolated case reports without relevance to practical recommendations.

Pathophysiology of HBV infection in pregnancy

Natural history of hepatitis B

CHB is a dynamic infection whose course is conventionally divided into sequential phases defined by hepatitis B e antigen (HBeAg) status, serum HBV DNA level, alanine aminotransferase (ALT) activity and the presence or absence of hepatic necroinflammation [4]. According to the nomenclature of the European Association for the Study of the Liver (EASL), four phases are recognised: HBeAg-positive chronic infection (formerly termed “immune-tolerant”; high HBV DNA, normal ALT, minimal inflammation), HBeAg-positive chronic hepatitis (“immune clearance”; fluctuating ALT and active necroinflammation), HBeAg-negative chronic infection (“inactive carrier”; low HBV DNA, normal ALT) and HBeAg-negative chronic hepatitis (variable HBV DNA, elevated ALT, frequently associated with pre-core/basal core promoter variants) [4]. A substantial proportion of individuals cannot be readily assigned to a single phase and are described as being in an “indeterminate” or “grey-zone” state [4]. These distinctions are important in the obstetric context because most women of childbearing age are in the HBeAg-positive chronic infection phase, characterised by a high viral load with preserved hepatic function; in this group, therapeutic decisions are aimed primarily at preventing vertical transmission rather than at treating maternal liver disease [5,6].

Immunological changes of pregnancy

Pregnancy is accompanied by profound immunological adaptations that allow maternal tolerance of the semi-allogeneic fetus. This tolerance is initiated and maintained at the maternal-fetal interface through decidual natural killer cells, macrophages and dendritic cells and, in the adaptive compartment, through a moderate expansion and functional enhancement of regulatory T cells (Treg) [7]. Expression of indoleamine 2,3-dioxygenase and HLA-G by trophoblasts and immune cells, together with a shift of the Th1/Th2 balance towards Th2 and of the Th17/Treg balance towards Treg predominance, further favours the tolerogenic environment [7]. Although they protect the fetus from rejection, these changes also modulate hepatic immunity in women with CHB: the relative attenuation of Th1-type cytotoxic responses tends to redu-

ce immune-mediated hepatocellular injury while permitting viral replication, so that ALT levels frequently remain stable or decrease whereas HBV DNA may rise over the course of pregnancy [5,7].

Effect of pregnancy on liver disease

In women without advanced fibrosis, CHB exerts little influence on the course of pregnancy, and pregnancy is not associated with an increased incidence of acute liver failure [8]. The main virological consequence of the gestational tolerogenic state is a tendency towards lower ALT and comparatively higher viral load, reflecting reduced immune pressure on the virus rather than true disease quiescence [5,7]. In the minority with cirrhosis or advanced fibrosis, however, the haemodynamic and volume demands of pregnancy may precipitate hepatic decompensation, variceal haemorrhage and adverse maternal and fetal outcomes, and these women require multidisciplinary co-management with hepatology [6,8].

Postpartum changes

Delivery is followed by rapid reconstitution of the pre-pregnancy immune state, and this reversal is the main trigger of postpartum hepatitis flares [7]. Loss of maternal-fetal tolerance, local uterine inflammation, the abrupt fall in circulating cortisol and re-establishment of Th1/Th17 predominance converge to reactivate HBV-specific CD8⁺ T-cell responses, which may trigger immune-mediated hepatocellular injury [7]. ALT flares occur in approximately 3.5% to 25% of women during the first three months after delivery or after discontinuation of antiviral therapy [5]. Most are asymptomatic and self-limiting, but women with pre-existing advanced fibrosis may develop clinically significant decompensation [5]. HBeAg-positive women appear more prone than HBeAg-negative women to postpartum flares and to phase transitions of the underlying infection [7].

Initial assessment of the pregnant woman

Diagnosis

Universal screening for HBV is recommended for all pregnant women at the first antenatal visit, in every pregnancy, ideally with the triple panel of HBsAg, total anti-HBc and anti-HBs; this universal approach is endorsed by the great majority of international clinical guidelines [9]. HBsAg-negative women without markers of immunity should be considered for vaccination, which is safe during pregnancy [5]. HBsAg is the principal marker of active in-

fection; its persistence for more than six months defines chronic infection, and a newly detected positive result in pregnancy requires complete serological and virological assessment [6,10]. HBeAg positivity indicates high viral replication and infectivity and identifies the pregnancies at greatest risk of transmission, whereas anti-HBe positivity generally reflects lower replication but does not exclude active disease, since HBeAg-negative chronic hepatitis due to pre-core variants may maintain significant viraemia [4,6]. Total anti-HBc denotes past or current exposure; IgM anti-HBc suggests acute infection or reactivation. Isolated anti-HBc positivity (HBsAg-negative, anti-HBs-negative) generally reflects resolved past exposure and requires additional management only when immunosuppression is planned [4]. Quantitative HBV DNA is the fundamental test that stratifies the risk of transmission and defines eligibility for antiviral prophylaxis, and should be obtained in every HBsAg-positive pregnant woman [10,26].

Assessment of liver disease

Serum ALT and AST establish the degree of hepatic inflammatory activity, contribute to phase classification and identify women with an indication for treatment independent of pregnancy [6]. Non-invasive assessment of fibrosis requires caution in this context: physiological pregnancy may increase hepatic stiffness through plasma volume expansion and relative cholestasis, reducing the specificity of transient elastography, so that conventional cut-off points are unreliable in the second and third trimesters; serum indices such as APRI and FIB-4 undergo similar interference [6]. When fibrosis staging is indispensable, findings should be interpreted conservatively and, whenever possible, deferred to or confirmed in the postpartum period [6]. Abdominal ultrasonography is the imaging method of choice in pregnancy, being radiation-free and safe; it is used to assess hepatic morphology, screen for focal lesions and detect signs suggestive of cirrhosis and portal hypertension, such as nodular contours, splenomegaly and associated thrombocytopenia [6,11]. Surveillance for hepatocellular carcinoma (HCC) should be maintained during pregnancy in patients who already meet screening criteria, including those with cirrhosis and selected high-risk non-cirrhotic patients. Six-monthly ultrasonography is recommended, with or without alpha-fetoprotein (AFP) measurement. AFP should be interpreted with caution, given its physiological elevation during pregnancy, which reduces its specificity as a tumour marker [10,11]. Although HCC in pregnancy is rare, it carries high maternal and neonatal mortality; in a systematic re-

view of 63 pregnancies, HBV (47%) and cirrhosis (22%) were the most frequent predisposing conditions, and incidental diagnosis in asymptomatic high-risk women or after an abnormal maternal AFP was associated with better maternal outcomes [11].

Risk of vertical transmission

Role of viral load

Maternal viraemia is the strongest modifiable determinant of vertical transmission. Contemporary guidelines converge on a threshold of HBV DNA $\geq 200,000$ IU/mL ($\geq 5.3 \log_{10}$ IU/mL) as the level above which antiviral prophylaxis is indicated; in a WHO meta-analysis, no transmission was observed below this threshold when the newborn received the timely dose of vaccine and hepatitis B immunoglobulin (HBIG) [12,13]. Nevertheless, the risk of transmission increases progressively with the maternal viral load, which helps to explain the higher risk observed in younger mothers, in whom rates of viral replication and HBeAg positivity are greater [13,14].

Importance of HBeAg

In the absence of neonatal immunoprophylaxis or maternal antiviral therapy, an estimated 70% to 90% of infants of HBeAg-positive mothers acquire HBV, a proportion markedly higher than the 5% to 10% observed with HBeAg-negative mothers [1]. HBeAg positivity therefore identifies the highest-risk pregnancies and, in settings where HBV DNA testing is unavailable, serves as an accepted surrogate for eligibility for antiviral prophylaxis, an approach adopted by the 2024 WHO guideline [9,12].

Main mechanisms of transmission

Vertical transmission occurs predominantly in the peripartum period, through exposure of the neonate to maternal blood and secretions during labour and delivery. The high efficacy of neonatal immunoprophylaxis corroborates the predominance of this route of transmission [15]. A smaller fraction results from intrauterine transmission, which is not prevented by postnatal immunoprophylaxis and thus accounts for a large part of residual failures. Among the proposed intrauterine routes are paracellular transplacental leakage, transport of the virus by peripheral blood mononuclear cells, germline infection and direct placental infection [15].

Additional risk factors

Beyond high viraemia and HBeAg positivity, several factors independently modulate the risk of transmission. In a popu-

lation-based study conducted in Guangdong (2021-2023), maternal age below 25 years, absence of antenatal antiviral therapy and delay in administration of HBIG and vaccine beyond six hours after birth were associated with a greater risk of transmission [14]. To these factors are added obstetric conditions linked to intrauterine infection, such as the performance of invasive procedures in the presence of high viraemia and threatened preterm labour, which favour contact of the fetus with maternal blood before birth [15]. With regard to amniocentesis in particular, the available cohort data suggest that the procedure does not substantially increase the risk of transmission in women with low viraemia, whereas caution remains warranted in HBeAg-positive women with a high viral load [30,31]. Caesarean section, in turn, has not demonstrated a consistent reduction in vertical transmission and should not be performed solely for this indication [9,14]. Furthermore, breastfeeding is not contraindicated, even in the presence of nipple fissures, since the neonate is protected by the vaccine and HBIG and there is no evidence of transmission through breast milk when preventive measures are followed [16]. Thus, vaginal delivery and breastfeeding are encouraged by most guidelines [9,16].

Universal screening in pregnancy

In Brazil, screening for hepatitis B should be carried out through HBsAg testing in all pregnant women, by means of a rapid test or immunoassay, ideally at the first antenatal visit and repeated in the third trimester [9,17]. For pregnant women with a negative result but no history of previous vaccination, a three-dose vaccination schedule is recommended. In pregnant women with reactive HBsAg, complementary assessment with HBeAg, ALT and HBV DNA viral load should be performed. In cases where the viral load exceeds 200,000 IU/mL, tenofovir (TDF) is indicated, started in the third trimester and to be maintained until 12 weeks postpartum [17].

Indications for antiviral treatment

The indications for antiviral treatment of hepatitis B fall into two distinct contexts: treatment for maternal benefit and treatment for prevention of vertical transmission. In the context of maternal benefit, pregnant women with hepatitis B should be assessed on the basis of ALT, the presence of significant viral replication and evidence of liver disease such as fibrosis and cirrhosis. Women with advanced liver disease should not interrupt treatment because of the risk of flare and decompensation [5,6]. Elevated ALT and detectable HBV DNA: treatment is indicated when ALT is $\geq 2 \times$ the ULN

with HBV DNA above the threshold corresponding to HBeAg status. Cirrhosis with active viral replication: treatment is recommended regardless of the trimester, with a risk of decompensation if suspended. Pregnant women on antiviral treatment prior to pregnancy: treatment should be maintained, preferably with TDF. In the context of treatment for prevention of vertical transmission, the indication is independent of maternal hepatic activity and is based exclusively on the viral load (HBV DNA). When treatment is given solely for prevention of vertical transmission, without a maternal indication of its own, it may be discontinued at delivery or up to 12 weeks postpartum, with ALT monitoring every one to three months up to six months because of the risk of flare after discontinuation [6,10]. HBV DNA > 200,000 IU/mL ($> 5.3 \log_{10}$ IU/mL): antiviral prophylaxis with tenofovir (TDF 300 mg/day or TAF 25 mg/day) is indicated, started between 28 and 32 weeks of gestation as an adjunct to neonatal immunoprophylaxis. Very high viral loads ($\geq 7 \log_{10}$ IU/mL) may justify earlier initiation of the antiviral, from the 16th week, to ensure adequate viral suppression by delivery.

Antivirals in pregnancy

Tenofovir disoproxil fumarate (TDF)

At a dose of 300 mg once daily, TDF is the drug of choice for the treatment of hepatitis B during pregnancy and may be used both for treatment of maternal disease and for prophylaxis of vertical HBV transmission. It has high viral-suppression potency and a high genetic barrier to resistance. Although it shows high placental transfer, the available data do not demonstrate an increased risk of congenital malformations associated with its use. Moreover, the pharmacokinetic changes observed during pregnancy do not justify adjustment of the usual dose. TDF is the antiviral with the greatest experience and volume of accumulated safety data during pregnancy, including evidence from its wide use in pregnant women with HIV [6,8,21].

Tenofovir alafenamide (TAF)

TAF is an emerging alternative, with growing safety data and efficacy equivalent to TDF in reducing vertical transmission, without an identified increase in the risk of adverse gestational outcomes or congenital malformations. It shows lower systemic exposure and lower placental transfer compared with TDF, while maintaining adequate antiviral activity. Although safety data in pregnancy are less numerous than those available for TDF, the accumulated evidence is favourable. Patients already using TAF before becoming

pregnant should maintain TAF during pregnancy, and a switch to TDF is not required [6,18].

Entecavir

The use of entecavir is not recommended during pregnancy because of insufficient data establishing its safety for the maternal-fetal dyad [8]. Women being treated with entecavir who become pregnant should have their antiviral regimen replaced by TDF or TAF [8,19]. Indiscriminate interruption of antiviral therapy should be avoided, especially in patients with advanced liver disease, because of the risk of viral reactivation and exacerbation of hepatitis [8,19,20].

Other antivirals

Other available antivirals include lamivudine, telbivudine, adefovir and peginterferon (IFN/PEG-IFN) [8,20]. Lamivudine and telbivudine have already demonstrated efficacy and safety in recent studies. However, because of their low genetic barrier and the greater risk of developing viral resistance, they are not considered preferred options [8]. Adefovir and peginterferon, antivirals also used outside pregnancy, should not be used in pregnancy, the latter being contraindicated and teratogenic [8,19].

Timing of initiation and discontinuation of treatment

Antiviral prophylaxis should ideally be initiated at 28 weeks of gestational age in patients with an indication for treatment to prevent vertical HBV transmission. Evidence shows that medication started between the 28th and 32nd weeks of gestation reduces the maternal-fetal transmission rate from 18% to 5% [5]. In pregnant women who enter antenatal care late, beyond this time window, therapy should be instituted promptly, in line with the AASLD/IDSA recommendation that prophylaxis with TDF or TAF be started immediately even after the 28th week, to mitigate the risk of vertical transmission [10]. In patients with markedly elevated viraemia—HBV DNA equal to or greater than $7 \log$ IU/mL—or in scenarios where it is known in advance that HBIG will not be available for the neonate, earlier antiviral prophylaxis is recommended, with the aim of ensuring adequate virological suppression by the time of delivery. In this context, a randomised clinical trial published in the Journal of the American Medical Association demonstrated non-inferiority of the strategy of TDF started at the 16th week of gestation combined with neonatal vaccination alone, compared with the standard regimen—TDF started at the 28th week combi-

ned with vaccination and administration of HBIG to the newborn. Maternal-fetal transmission rates were 0.76% (1/131) in the experimental group versus 0% (0/142) in the standard-care group (intention-to-treat analysis), with a between-group difference within the pre-specified non-inferiority margin of 3%. This simplified regimen may be particularly relevant in resource-limited settings where HBIG is not available [22].

In the puerperium, therapy may be discontinued if the only therapeutic indication is prophylaxis of vertical transmission. The biochemical exacerbations, also called flares, that may occur in this period tend to be mild and to resolve spontaneously, although rare cases of acute liver failure in this context have been documented. However, prolonging antiviral prophylaxis in the puerperium has not demonstrated benefit in preventing exacerbations, a finding that supports the recommendation to suspend therapy at delivery [23]. As to the criteria for maintaining long-term treatment—independent of the obstetric indication—the general criteria for treatment of CHB recommended by the AASLD/IDSA apply, which recommend antiviral therapy in patients with cirrhosis, as well as in those without cirrhosis who present persistently elevated HBV DNA associated with ALT elevation above the upper limit of normal and evidence of significant fibrosis ($\geq F2$) on biopsy or non-invasive methods. Treatment is also indicated in patients with a family history of hepatocellular carcinoma or cirrhosis and in those who will require immunosuppressive therapy or chemotherapy [10]. After discontinuation of therapy, serial monitoring of HBV DNA and ALT is recommended for early detection of exacerbations, every one to three months for up to six months postpartum [10,23]. Reinstitution of treatment is indicated if an ALT elevation equal to or greater than five times the upper limit of normal is observed, configuring a clinically significant flare—a criterion used for patients in general and, by extension, for the obstetric scenario.

Monitoring during pregnancy

The American College of Obstetricians and Gynecologists holds that isolated CHB, in the absence of associated comorbidities, does not constitute a specific indication for additional antepartum fetal surveillance beyond routine obstetric follow-up, since this infection exerts little influence on the course of pregnancy itself [24,25]. Thus, the central axis of monitoring rests on the maternal viral load (HBV DNA) and hepatic function, as discussed in a previous section, rather than on parameters of fetal well-being. This position is

supported by other reference societies, such as the American Association for the Study of Liver Diseases and the American Gastroenterological Association [5,16]. It is worth noting, for terminological precision, that the Society for Maternal-Fetal Medicine (SMFM) uses a similar formulation, stating that data are insufficient to suggest that acute or chronic HBV infection is associated with adverse gestational outcomes such as preterm birth, low birth weight or gestational diabetes, and therefore recommends routine (rather than additional or intensified) antenatal care and fetal surveillance for pregnant women with CHB [21].

Delivery management

The indication for mode of delivery remains obstetric, since there are no well-designed trials demonstrating the efficacy of caesarean section in reducing the risk of maternal-fetal transmission of hepatitis B [26]. It should be noted that, although this is the official recommendation, meta-analyses of observational studies have suggested a reduction in transmission risk with caesarean section compared with vaginal delivery; however, such findings lack confirmation by multicentre randomised clinical trials, which is why they do not alter standard obstetric management [27,28]. Antiviral therapy, notably TDF, is minimally excreted in breast milk—with a milk concentration corresponding to only about 7% of the maternal plasma concentration and undetectable levels in the infants' plasma—so that its use is considered safe during lactation [5,29]. It is important to note that TAF lacks safety data in lactation at the same level of evidence as TDF.

Neonatal prophylaxis

Prevention of vertical HBV transmission is one of the main strategies for reducing the incidence of chronic hepatitis B and its long-term complications. Infection acquired in the perinatal period has a high probability of becoming chronic, especially compared with infection acquired in adulthood. Identification of maternal infection during antenatal care and timely implementation of preventive measures are therefore essential to interrupt the chain of transmission [4,8,12,21,26]. Prevention of vertical transmission should be understood as a longitudinal strategy involving screening of the pregnant woman, confirmation of infection, assessment of the viral load, identification of women at greatest risk of transmission, indication of antiviral therapy when appropriate, neonatal immunoprophylaxis, completion of the vaccination schedule and subsequent serological assessment of the newborn [4,8,12,21,26].

Neonatal immunoprophylaxis is the principal preventive measure. For newborns of HBsAg-reactive mothers, administration of the hepatitis B vaccine combined with human hepatitis B immunoglobulin (HBIG) is recommended as early as possible after birth, ideally within the first 12 hours of life. The two interventions should be administered at distinct anatomical sites. This recommendation should be followed regardless of whether the mother received antiviral therapy during pregnancy [12,21,26,32,33]. The vaccine provides active immunity, while the immunoglobulin provides immediate passive immunity. The combination of the two measures is particularly important in newborns of women with a high viral load, a situation in which there is a greater residual risk of transmission [12,26,32,33].

The mode of delivery should not be modified solely for the purpose of preventing vertical HBV transmission. In the absence of other obstetric indications, elective caesarean section is not recommended merely on account of maternal HBsAg positivity or high viral load. Intrapartum care should follow the usual obstetric indications, while prevention of transmission should focus mainly on adequate identification of the infected pregnant woman, indication of antiviral therapy when pertinent and, above all, timely administration of neonatal immunoprophylaxis [21,32]. After completion of the vaccination schedule, the exposed newborn should undergo testing for HBsAg and anti-HBs between 9 and 12 months of age or, when the vaccination schedule is delayed, 1 to 2 months after administration of the final dose. Anti-HBs should be interpreted taking a protective immune response as a concentration ≥ 10 mIU/mL. Serological assessment should not be performed before 9 months of age, since the presence of anti-HBs in this period may reflect antibodies passively acquired from the immunoglobulin administered at birth. The recommended interval also increases the probability of correctly identifying any HBV infection acquired in the perinatal period [26,33].

Breastfeeding

Maternal HBV infection, in itself, does not constitute a contraindication to breastfeeding. Women with HBV may breastfeed, provided the newborn receives the indicated immunoprophylaxis at birth, and there is no recommendation to delay the start of breastfeeding until the infant vaccination schedule is complete [12,21,34]. Maternal use of TDF is also considered compatible with breastfeeding when there is a clinical indication for its ma-

intenance. Pharmacokinetic data have demonstrated low passage of tenofovir into breast milk and minimal or undetectable systemic exposure in infants [35,36]. Thus, the need to maintain TDF for treatment of maternal hepatitis B or for prevention of mother-to-child transmission does not, in itself, constitute a reason to suspend breastfeeding. The decision should take into account the indication for maternal treatment, the risk of disease progression and the benefits of breastfeeding [10,34,36]. A particular situation is the presence of fissures or bleeding of the nipples or areola. Since HBV can be transmitted through blood, temporary interruption of direct breastfeeding is recommended when there is active bleeding, with expression and discarding of the milk to maintain lactation until healing. After resolution of the fissures or bleeding, breastfeeding may be resumed [34].

Maternal postpartum follow-up

The postpartum period is a particularly important phase for follow-up, since the immunological changes associated ' with the end of pregnancy may favour an increase in hepatic inflammatory activity, with elevation of aminotransferases and the occurrence of hepatitis B exacerbations, frequently termed postpartum flares [4,6,37]. The main factors associated with exacerbations include HBV DNA and ALT levels during pregnancy, HBeAg status and discontinuation of the antiviral in the postpartum period [37]. Although most ALT elevations are self-limiting, clinically significant exacerbations can occur and, in patients with advanced liver disease, may represent a risk of decompensation. For this reason, postpartum follow-up should include clinical and laboratory surveillance, especially during the first months after birth and after discontinuation of antiviral therapy [4,6,37,38]. Postpartum follow-up should include serial assessment of ALT and, depending on the clinical context and the antiviral discontinuation strategy, additional monitoring of viral activity. The Brazilian PCDT is one of the main references for organising this follow-up [6,32,37,38]. In the presence of ALT elevation, assessment should include investigation of hepatitis B activity, particularly through quantification of HBV DNA, in addition to evaluation of liver function with bilirubin, albumin, prothrombin time/INR and other parameters according to clinical severity. Persistent or marked ALT elevations, especially when accompanied by a significant increase in HBV DNA, hyperbilirubinaemia, INR prolongation, symptoms or other signs of impaired liver function, should prompt specialist assessment [4,6,37,38]. Continuity of care is particularly important because some women

identified during pregnancy may have been diagnosed only through obstetric screening and never have received adequate hepatological assessment. The postpartum period should therefore be used to establish longitudinal follow-up, assess the degree of liver disease and determine whether there is an indication for continued treatment [4,6,38].

Conclusions

Prevention of vertical HBV transmission is one of the most effective interventions for reducing the future incidence of chronic hepatitis B and its complications. Its success depends on the integration of screening of the pregnant woman, assessment of the viral load, identification of women at greatest risk, use of antiviral therapy when indicated and, above all, timely administration of the hepatitis B vaccine and HBIG to the exposed newborn [4,8,12,21,26]. Maternal antiviral prophylaxis should be understood as a strategy complementary to neonatal immunoprophylaxis. In pregnant women with a high viral load, tenofovir reduces the residual risk of transmission but does not replace vaccination and immunoglobulin. The combination of measures is fundamental to maximising prevention of mother-to-child transmission [8,12,21,22]. Breastfeeding should be encouraged in HBsAg-reactive women when neonatal immunoprophylaxis is carried out adequately. Maternal HBV infection does not, in isolation, constitute a contraindication to breastfeeding, and the use of TDF does not, in itself, constitute a reason to interrupt breastfeeding when there is an indication to maintain treatment [10,34,36]. Maternal care should not end after delivery. The postpartum period carries an increased risk of ALT alterations and hepatic flares, particularly in women who have undergone discontinuation of antivirals used exclusively for prevention of mother-to-child transmission. Systematic clinical and laboratory monitoring allows early identification of patients who require additional investigation or treatment [4,6,37,38]. Beyond monitoring of viral activity and liver function, the puerperium represents an opportunity to establish longitudinal follow-up of hepatitis B, assess the need for definitive treatment and guide measures to prevent horizontal transmission, including assessment and vaccination of susceptible contacts according to current recommendations [4,12,32]. In summary, the approach to hepatitis B in pregnancy should be understood as a continuous care pathway that begins with antenatal screening and extends through delivery, the neonatal period, breastfeeding and maternal and infant follow-up. Coordination between obstetrics, neonatology, paediatrics,

primary care and hepatology/infectious diseases is essential to ensure the effectiveness of the available strategies and to contribute to the goals of eliminating hepatitis B as a public health problem [4,8,12,21,26,38].

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