Society for Maternal-Fetal Medicine (SMFM) Consult Series: 
#38: Hepatitis B in pregnancy screening, treatment, and prevention of vertical transmission

Society for Maternal-Fetal Medicine (SMFM); Jodie Dionne-Odom, MD; Alan T. N. Tita, MD, PhD; Neil S. Silverman, MD

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Between 800,000-1.4 million people in the United States and more than 240 million people worldwide are infected with hepatitis B virus (HBV). Specific to pregnancy, an estimated prevalence of 0.7-0.9% for chronic hepatitis B infection among pregnant women in the United States has been reported, with >25,000 infants at risk for chronic infection born annually to these women. Vertical transmission of HBV from infected mothers to their fetuses or newborns, either in utero or peripartum, remains a major source of perpetuating the reservoir of chronically infected individuals globally. Universal screening for hepatitis B infection during pregnancy has been recommended for many years. Identification of pregnant women with chronic HBV infection through universal screening has had a major impact in decreasing the risk of neonatal infection. The purpose of this document is to aid clinicians in counseling their patients regarding perinatal risks and management options available to pregnant women with hepatitis B infection in the absence of coinfecion with HIV. We recommend the following: (1) perform routine screening during pregnancy for HBV infection with maternal HBsAg testing (grade 1A); (2) administer hepatitis B vaccine and HBV immunoglobulin within 12 hours of birth to all newborns of HBsAg-positive mothers or those with unknown or undocumented HBsAg status, regardless of whether maternal antiviral therapy has been given during the pregnancy (grade 1A); (3) In pregnant women with HBV infection, we suggest HBV viral load testing in the third trimester (grade 2B); (4) in pregnant women with HBV infection and viral load >6-8 log 10 copies/mL, HBV-targeted maternal antiviral therapy should be considered for the purpose of decreasing the risk of intrauterine fetal infection (grade 2B); (5) in pregnant women with HBV infection who are candidates for maternal antiviral therapy, we suggest tenofovir as a first-line agent (grade 2B); (6) we recommend that women with HBV infection be encouraged to breast-feed as long as the infant receives immunoprophylaxis at birth (HBV vaccination and hepatitis B immunoglobulin) (grade 1C); (7) for HBV infected women who have an indication for genetic testing, invasive testing (eg amniocentesis or chorionic villus sampling) may be offered—counseling should include the fact that the risk for maternal-fetal transmission may increase with HBV viral load >7 log 10 IU/mL (grade 2C); and (8) we suggest cesarean delivery not be performed for the sole indication for reduction of vertical HBV transmission (grade 2C).

Key words: antiviral therapy, breast-feeding, chronic hepatitis, hepatitis B, immunoprophylaxis, vertical transmission, viral load

Introduction
Obstetric providers are challenged continuously with the evaluation of the potential benefits and harms of new diagnostic and therapeutic procedures or treatments for patients (mother and fetus), often in the setting of limited high-quality data (eg, from randomized clinical trials). The purpose of this document is to aid clinicians in counseling their patients regarding the risk and management options available after a positive hepatitis B surface antigen (HBsAg) test result.

What risks and potential impact does hepatitis B infection present during pregnancy?
Between 800,000-1.4 million people in the United States and >240 million people worldwide are infected with hepatitis B virus (HBV).1 From a global public health perspective, chronic HBV infection is the major source of hepatocellular carcinoma, leading to 50% of cases worldwide and 80% in high-endemic areas for HBV. Specific to pregnancy, an estimated prevalence of 0.7-0.9% for chronic hepatitis B infection among pregnant women in the United States has been reported,2-3 with >25,000 infants at risk for chronic infection born annually to these women.4 While transmission through sexual intercourse and intravenous drug abuse are the major risk factors for acquisition of hepatitis B among adults in the...
United States, perinatal transmission is responsible for up to 50% of HBV infection worldwide (Table 1). Vertical transmission of HBV from infected mothers to their fetuses or newborns, either in utero or peripartum, remains a major source of perpetuating the reservoir of chronically infected individuals globally. It has been demonstrated that prenatal risk factor–based screening alone will miss many chronic HBV infections among pregnant women, thereby missing the opportunity to interrupt perinatal transmission via established newborn protocols.

For this reason, universal screening for hepatitis B infection during pregnancy at the first prenatal visit has been recommended for many years by both the American Congress of Obstetricians and Gynecologists and the US Preventative Services Task Force.\(^5,6\)

In contrast to HBV acquisition in adulthood, which more commonly leads to acute resolved infection and immunity, perinatal/neonatal HBV is more likely to lead to chronic infection and its long-term disease risks. Chronic hepatitis B infection will develop in up to 90% of exposed neonates who do not receive appropriate immunoprophylaxis, in contrast to 10-25% of infected children and only 5-10% of exposed immunocompetent adults. Among all individuals with chronic HBV infection, regardless of the timing of infection, 20% will eventually die from complications of HBV infection including cirrhosis, end-stage liver disease, and liver cancer.\(^7\)

With the exception of the major risk of perinatal transmission (see below), data are insufficient to suggest that acute or chronic HBV infection is associated with adverse pregnancy outcomes such as preterm birth, low birth weight, or gestational diabetes. However, cirrhosis due to chronic HBV may be associated with increased maternal and perinatal death, gestational hypertension, abruptio, preterm birth, and fetal growth restriction.\(^7,13\)

### How are HBV-infected pregnant women identified and what have been traditional approaches to their pregnancies?

Identification of pregnant women with chronic HBV infection through universal screening has had a major impact in decreasing the risk of neonatal infection. Recent data demonstrate that 95% of pregnant women are currently screened prior to delivery for evidence of chronic HBV infection, with rates of perinatal transmission decreasing significantly over the past 2 decades.\(^14\)

The presence of HBsAg in maternal blood more commonly represents chronic infection than acute infection. While some adults will be identified because of symptomatic illness, the vast majority of chronically infected adults are asymptomatic. The diagnosis of the chronic carrier state is confirmed with persistence of HBsAg and the absence of hepatitis B surface antibody (HBsAb), which is a neutralizing antibody that can be detected after HBV infection has been cleared. HBsAb and HBsAg essentially do not exist together. HBsAb is also detected after successful immunization with the HBV vaccine. Therefore, we suggest performing routine screening during pregnancy for HBV infection with maternal HBsAg testing (GRADE 1A). Hepatitis B core antibody, on the other hand, develops in the setting of natural infection, never from immunization, and persists regardless of whether the acute infection is cleared or becomes chronic (Table 2). It is emphasized strongly that pregnancy is not a contraindication to hepatitis B vaccination. Pregnant women who are identified as being at risk for HBV infection during pregnancy (eg, having >1 sex partner during the previous 6 months, been evaluated or treated for a sexually transmitted disease, recent or current injection drug use, or having had an HBsAg-positive sex partner) should be vaccinated.

The most common risk for perinatal HBV infection occurs when the infant comes into contact with infected vaginal blood and secretions at the time of delivery. Invasive procedures during labor and delivery (including internal monitors, episiotomy, and operative vaginal delivery) may theoretically increase the risk of transmission. However, the availability of neonatal HBV immunoprophylaxis is thought to ameliorate these risks, and current opinions do not support altering regular obstetric practices. Elective cesarean delivery has also been discussed as one way to reduce vertical transmission, but it is not recommended since available data are conflicting and of poor quality.\(^15\) We suggest cesarean delivery not be performed for the sole indication for reduction of vertical HBV transmission (GRADE 2C). Similarly, in the setting of neonatal HBV immunoprophylaxis, breast-feeding is not contraindicated.\(^16,17\) Studies have documented no difference in rates of infection between breast-fed and formula-fed vaccinated infants born to HBV-infected women, with rates in both groups between 0-5%.\(^17,18\) We recommend that women with HBV infection be encouraged to breast-feed as long as the infant receives immunoprophylaxis at birth (HBV vaccination and hepatitis B immunoglobulin) (GRADE 1C).
### TABLE 2
**Interpretation of hepatitis B serologic test results (from [www.cdc.gov](http://www.cdc.gov/hepatitis/hbv/pdfs/serologicchartv8.pdf)**

**Interpretation of Hepatitis B Serologic Test Results**

Hepatitis B serologic testing involves measurement of several hepatitis B virus (HBV)-specific antigens and antibodies. Different serologic "markers" or combinations of markers are used to identify different phases of HBV infection and to determine whether a patient has acute or chronic HBV infection, is immune to HBV as a result of prior infection or vaccination, or is susceptible to infection.

<table>
<thead>
<tr>
<th>HBsAg</th>
<th>anti-HBc</th>
<th>anti-HBs</th>
<th><strong>Result</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>negative</td>
<td>negative</td>
<td>negative</td>
<td>Susceptible</td>
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</tbody>
</table>

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<tr>
<th>HBsAg</th>
<th>anti-HBc</th>
<th>anti-HBs</th>
<th><strong>Result</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>negative</td>
<td>positive</td>
<td>positive</td>
<td>Immune due to natural infection</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>HBsAg</th>
<th>anti-HBc</th>
<th>IgM anti-HBc</th>
<th>anti-HBs</th>
<th><strong>Result</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>negative</td>
<td>negative</td>
<td>positive</td>
<td>positive</td>
<td>Immune due to hepatitis B vaccination</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>HBsAg</th>
<th>anti-HBc</th>
<th>IgM anti-HBc</th>
<th>anti-HBs</th>
<th><strong>Result</strong></th>
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</thead>
<tbody>
<tr>
<td>positive</td>
<td>positive</td>
<td>positive</td>
<td>negative</td>
<td>Acutely infected</td>
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</table>

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<th>anti-HBc</th>
<th>IgM anti-HBc</th>
<th>anti-HBs</th>
<th><strong>Result</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>positive</td>
<td>positive</td>
<td>negative</td>
<td>negative</td>
<td>Chronically infected</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>HBsAg</th>
<th>anti-HBc</th>
<th>anti-HBs</th>
<th><strong>Result</strong></th>
</tr>
</thead>
</table>
| negative | positive | negative | Interpretation unclear; four possibilities: 
1. Resolved infection (most common) 
2. False-positive anti-HBc, thus susceptible 
3. “Low level” chronic infection 
4. Resolving acute infection |

- **Hepatitis B surface antigen (HBsAg):**
  A protein on the surface of hepatitis B virus; it can be detected in high levels in serum during acute or chronic hepatitis B virus infection. The presence of HBsAg indicates that the person is infectious. The body normally produces antibodies to HBsAg as part of the normal immune response to infection. HBsAg is the antigen used to make hepatitis B vaccine.

- **Hepatitis B surface antibody (anti-HBs):**
  The presence of anti-HBs is generally interpreted as indicating recovery and immunity from hepatitis B virus infection. Anti-HBs also develops in a person who has been successfully vaccinated against hepatitis B.

- **Total hepatitis B core antibody (anti-HBc):**
  Appears at the onset of symptoms in acute hepatitis B and persists for life. The presence of anti-HBc indicates previous or ongoing infection with hepatitis B virus in an undefined time frame.

- **IgM antibody to hepatitis B core antigen (IgM anti-HBc):**
  Positivity indicates recent infection with hepatitis B virus (≤6 mos). Its presence indicates acute infection.

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Concerns have also been raised regarding invasive diagnostic procedures during pregnancy, such as amniocentesis, since these would occur well before the timing for immunoprophylaxis. However, the majority of reported earlier series did not demonstrate an increased risk for in utero infection after amniocentesis in women with chronic HBV infection.19-23 These series were conducted before the routine use of HBV viral load testing as a disease marker; therefore, it may not apply to women with very high viral load as will be defined in a later section. In fact, a recent series did demonstrate an increase in risk for in utero infection after amniocentesis in women with viral titers >7 log 10 copies/mL, compared to those women with titers below that cutoff (50% vs 4%; odds ratio, 21.3; P = .006).22 Such emerging data may have an impact on counseling surrounding invasive prenatal testing as data accumulate from more series using maternal HBV viral load titers. For HBV-infected women who have an indication for genetic testing, invasive testing (eg amniocentesis or chorionic villus sampling) may be offered. Counseling should include the fact that the risk for maternal-fetal transmission may increase with HBV viral load >7 log 10 copies/mL (GRADE 2C).

What has been the traditional approach to preventing neonatal HBV infection?
The mainstay of perinatal HBV infection prevention is a combination of active and passive immunization for exposed infants. Before the development of an HBV vaccine, HBV immunoglobulin (HBIG) alone, administered within 12 hours of delivery, was shown to be effective in providing transient passive immunity, but 25% of infants became infected through household contact by 1 year of age.24 When the vaccine became available in the 1980s, it was subsequently shown that a combination of HBV vaccine and HBIG given within the first 12 hours after birth gave the greatest degree of durable protection, conferring long-term immunity in 85-95% of cases.25 Immunoprophylaxis is also recommended for infants born to mothers with unknown or undocumented HBsAg status. Completion of the full 3-dose HBV vaccine series following the birth-dose vaccine is important for the newborn to gain maximal protection and is recommended for all infants irrespective of maternal HBV infection status. This approach has shown significant impact on longer-term disease outcome measures for newborns who received prophylaxis in areas that are endemic for HBV infection. In Taiwan, the institution of a universal screening and immunization program lowered the rate of chronic HBV infection among children from 10% to 1% during a 10-year period.27 Concurrently, the rate of childhood hepatocellular carcinoma was lowered by half in the same population, from 0.7 to 0.36 per 100,000.28 We recommend administering hepatitis B vaccine and HBIG within 12 hours of birth to all newborns of HBsAg-positive mothers or those with unknown or undocumented HBsAg status, regardless of whether maternal antiviral therapy has been given during the pregnancy (GRADE 1A).

How has the approach to treating HBV infection in general changed recently?
As was shown with the evolution of management of HIV-related illness, the use of HBV viral load as a predictor of disease progression and as a measure of treatment response has been a major factor regarding development of treatment models for HBV-related disease. This has resulted in development of treatment protocols for lowering and even eliminating viremia in HBV-infected adults, with evolving corollary implications for management during pregnancy. HBV viral load has been shown to be directly related to the risk of disease progression in infected adults. In interpreting studies reporting outcomes and indications for treatment in relation to viral load, the results are inconsistently reported in relation of HBV units. Some studies provide data in the form of copies/mL, while others report in IU/mL, despite the fact that the World Health Organization has recommended that HBV DNA be expressed in terms of IU/mL. Conversion is straightforward: to convert from IU/mL to copies/mL, the IU/mL value should be multiplied by 5.6 (or the copies/mL value similarly divided).29

In a large prospective cohort from Taiwan, an HBV-DNA level >4 log 10 copies/mL was associated with significantly higher rates of cirrhosis, hepatocellular carcinoma, and death, independent of hepatitis B e-antigen status as a surrogate marker of viremia.30,31 Randomized controlled trials were subsequently conducted evaluating the use of antivirals in HBV-infected adults in an attempt to lower viremia and, in turn, lower long-term disease risks. Some of the single-agent antivirals studied had been used to treat HIV infection, specifically lamivudine and tenofovir. One of the earlier trials using lamivudine demonstrated significantly less progression of hepatic fibrosis and cirrhosis over 32 months compared to placebo, but also that drug resistance developed in a high proportion of patients.32 Subsequent trials using tenofovir and entecavir, another reverse transcriptase inhibitor, showed sustained viral suppression below detectable levels and reversal of hepatic histopathology without similar levels of resistance.33 As a result, the American Association for the Study of Liver Diseases issued revised guidelines in 2009 for the treatment of chronic HBV infection, moving tenofovir and entecavir to first-line therapies, with lamivudine not a first-line agent due to resistance concerns.34 More recent reports have demonstrated that in chronically infected adults, tenofovir monotherapy has maintained HBV-DNA suppression while used for up to 6 years of continuous treatment, with no evidence of tenofovir resistance, even in patients whose virus became resistant to lamivudine.35,36 Table 3 outlines the characteristics, results, and resistance risks of currently available and studied HBV antivirals.

How has the approach to HBV in pregnancy been affected by its treatment in nonpregnant adults? What new measures now need to be considered?
While the use of HBIG and HBV vaccine neonatally has shown a dramatic impact
in lowering rates of perinatal HBV transmission, concern persisted regarding the 5-15% of newborns who are infected despite receiving appropriate neonatal immunoprophylaxis. This subgroup has been thought to represent a cohort of newborns infected in utero but, until recently, no measures had been shown to have an impact on HBV viremia in infected individuals. The accomplishments using antivirals in adults with HBV infection led investigators to explore whether a comparable model might apply to in utero infection. Using HIV as a model, hypotheses evolved exploring the use of antivirals, particularly those with established fetal safety profiles when used in HIV-infected pregnant women, to effectively treat nonpregnant HBV-infected adults. To date, small, mostly nonrandomized series have been published studying the use of lamivudine, telbivudine, entecavir, and tenofovir for this purpose. Lamivudine has been the agent used most frequently in recent trials, due to its better-established safety profile in pregnancy in the setting of maternal HIV infection, although tenofovir also has an enlarging body of registry-based data supporting its use in pregnancy.

A recent metaanalysis compiling data on the use of lamivudine during pregnancy for this purpose included 10 trials, although only 3 were placebo-controlled. Compared to placebo, treatment with lamivudine starting at 24-32 weeks of gestation through 4 weeks postpartum resulted in a significant 80% decrease in MTCT of HBV (odds ratio, 0.2; 0.10-0.39; P < .001, 95% CI). Concerns are still present, however, regarding the use of lamivudine as a single agent for this purpose due to its high rate of resistance developing, reported as up to 32% after 1 year, and its potential implications for treatment of the woman after delivery should it become necessary. In addition, in one larger recent series, 62% of women treated during pregnancy with lamivudine experienced a significant postpartum flare in their liver function test (LFT) results when their medication was stopped.

Other antiviral agents with much lower resistance rates have also been studied for their impact on MTCT rates for HBV, although these series have been smaller and less rigorous. The use of tenofovir, 300 mg/d, was initially reported in an observational case series of 11 women with a mean HBV viral load of 8.9 log 10 copies/mL, with medication started at 28-52 weeks of gestation and continued until delivery. The mean maternal viral load was significantly lower at delivery than in women who received placebo.

### TABLE 3

<table>
<thead>
<tr>
<th>Resistance data</th>
<th>Clinical issues</th>
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</thead>
<tbody>
<tr>
<td><strong>Adefovir</strong></td>
<td>0–3% at 1–2 y 11–18% at 3–4 y</td>
</tr>
<tr>
<td><strong>Entecavir</strong></td>
<td>Virologic breakthrough rare in NA-naive patients Resistance 1–2% in naive patients up to 5 y of treatment Resistance high (51%) in lamivudine-refractory patients</td>
</tr>
<tr>
<td><strong>Lamivudine</strong></td>
<td>14–32% after 1 y 60–70% after 5 y</td>
</tr>
<tr>
<td><strong>Telbivudine</strong></td>
<td>2–5% after 1 y 11–25% after 2 y</td>
</tr>
<tr>
<td><strong>Tenofovir</strong></td>
<td>No resistance after 6 y of continuous therapy, despite low rates of viral breakthrough</td>
</tr>
</tbody>
</table>

#### Resistance risks and clinical issues for currently available hepatitis B virus antivirals

As a result of the poor performance of HBIG as an intervention to lower the risk of in utero HBV infection, researchers turned to evaluating the use of HBV antivirals during pregnancy to potentially lower maternal viremia and reduce MTCT as a result. This research drew on the use of antivirals during pregnancy in HIV-infected women to effectively accomplish the same result, as well as the published results of trials using the same antivirals to treat nonpregnant HBV-infected adults. To date, small, mostly nonrandomized series have been published exploring the use of antivirals in utero HBV infection. Researchers turned to evaluating the use of HBV antivirals during pregnancy to potentially lower maternal viremia and reduce MTCT as a result. This research drew on the use of antivirals during pregnancy in HIV-infected women to effectively accomplish the same result, as well as the published results of trials using the same antivirals to treat nonpregnant HBV-infected adults. To date, small, mostly nonrandomized series have been published exploring the use of antivirals in utero HBV infection.
lowered, to 5.2 log 10 copies, with 55% of the women achieving a level <6 log 10 copies/mL. All infants were HBsAg and HBV-DNA negative at 36 weeks post-delivery, and none of the treated women experienced a postpartum LFT flare. In a more recent multicenter prospective observational study, HBV antiviral therapy was given to pregnant women with elevated HBV DNA levels (>7 log 10 IU/mL) after 32 weeks of gestation. Lamivudine was used initially in the study then changed to tenofovir partway through the trial, due to emerging evidence in 2010 of increasing lamivudine resistance rates in nonpregnant adults; controls were those women who declined therapy. All newborns received recommended active and passive immunization.

Based on these studies and others, the use of lamivudine, tenofovir, or telbivudine after 28-32 weeks of gestation for HBV-infected women with high viral load (>6-8 log 10 copies/mL) has been suggested, in addition to administration of both HBV vaccine and HBIG within 12-24 hours of birth, to minimize in utero infection and to maximize neonatal HBV prevention. It has also been demonstrated, in a recent study employing a decision-tree model that perinatal antiviral prophylaxis is cost-effective across a wide range of assumptions when either a positive hepatitis B e-antigen or high maternal viral load (>6-8 log 10 copies/mL) is present. In this analysis, the researchers demonstrated that single-agent anti-HBV therapy during pregnancy remained cost-saving unless the reduction in perinatal transmission was <18.5%, which is higher than shown in any antiviral study to date. In addition, this treatment arm would prevent 9.7 cases of chronic hepatitis B in newborns for each 100 women treated and save $5184 per 100 women treated. Other decision-analysis model-based studies have drawn similar conclusions regarding the cost-effectiveness of single-agent anti-HBV therapy during pregnancy. In one, for every 100 women treated with lamivudine, $337,000 was
saved, 314 quality-adjusted life-years were gained, and 21 cases of hepatocellular carcinoma and 5 liver transplants were prevented in offspring.53

Many pregnant women with HBV and HIV coinfection are already being treated with dually active agents—tenofovir, emtricitabine, or lamivudine—and trials showing efficacy and safety in this population are ongoing.54 A recent analysis of antiretroviral registry data looking specifically at the fetal safety profiles of the subgroup of anti-HIV agents also effective against HBV demonstrated no increase in exposure risk. For tenofovir, for example, the registry had compiled data on a sufficient number of first-trimester exposures to detect at least a 2-fold increase risk in birth defects, with none demonstrated.47,55 Finally, regarding breastfeeding, the use of lamivudine and tenofovir in the postpartum period is not currently recommended solely for HBV prevention until additional data are available.55 Most published study protocols, however, have stopped the maternal HBV therapy at the time of delivery, so this may not be a significant management issue.

No guidelines currently exist in the United States regarding the use of antiviral therapy against HBV during pregnancy specifically for the goal of decreasing the risk of in utero infection and vertical transmission. However, it is being offered increasingly in centers where practitioners already have experience with the use of similar antivirals for the management of HIV-infected pregnant women. Precedent for establishment of universal guidelines exists already in Europe, where both the European Association for the Study of the Liver and the United Kingdom’s National Institute for Health and Care Excellence have published such guidelines in 2012 and 2013, respectively.56-58 Both agencies currently advocate discussion of antiviral therapy with HBV-infected pregnant women with viral loads >6-7 log 10 IU/mL (6.7-7.7 log 10 copies/mL), with treatment to be offered in the third trimester. As more data are published in larger trials, this will inevitably lead to development of perinatal treatment protocols in the United States. HBV-targeted maternal antiviral therapy in the third trimester of pregnancy should be considered to reduce transmission in cases where maternal viral load is >6-8 log 10 copies/mL. In pregnant women with HBV infection, we suggest HBV viral load testing in the third trimester (grade 2B). In pregnant women with HBV infection and viral load >6-8 log 10 copies/mL, HBV-targeted maternal antiviral therapy should be considered for the purpose of decreasing the risk of intrauterine fetal infection (GRADE 2B). In pregnant women with HBV infection who are candidates for maternal antiviral therapy, we suggest tenofovir as a first-line agent (GRADE 2B).

What other issues need to be considered in a pregnant woman diagnosed as a chronic HBV carrier?

Identification of a pregnant woman as chronically HBV infected also presents an important opportunity to counsel her regarding risks to other family and household members. HBV is most easily transmitted via sexual exposure or blood exposure but can also be transmitted through casual shared use of household items such as eating utensils and toothbrushes, as well as through personal contact such as kissing or routine childcare. Therefore, family and household members should be evaluated for HBV status and referred for vaccination if found to be uninfected and nonimmune. The pregnant woman herself should also be assessed for immunity status for hepatitis A and offered vaccination if not immune, since coinfection with another viral hepatitis results in compounded morbidity. The woman should also be counseled regarding exposures to potentially hepatotoxic medications, even those available over the counter, such as acetaminophen, and to avoid the use of alcohol even when not pregnant.

The majority of pregnant women diagnosed with chronic HBV infection will be asymptomatic and identified through routine screening with initial prenatal laboratory tests. To aid in counseling regarding risks and potential management options as outlined above, baseline LFTs should also be drawn when a positive HBsAg test result is obtained, along with a baseline quantitative HBV-DNA level. Consideration should also be given to referral to a maternal-fetal medicine subspecialist or an infectious diseases specialist or hepatologist with experience managing hepatitis B to coordinate care and surveillance for the woman during and after pregnancy. If the baseline HBV DNA polymerase chain reaction testing is negative, it may be repeated in the third trimester, since this is usually the time when consideration is given to beginning antiviral treatment in women with high viral loads. In consideration of cost, baseline HBV-DNA testing during pregnancy can be deferred until the third trimester, especially if the initial LFT results are normal or results prior to pregnancy are available. Even if the maternal viral load is low and antiviral therapy during pregnancy is not recommended, the newborn should still receive standard prophylaxis with HBIG and HBV vaccine within 12 hours of birth, and ongoing surveillance of the woman’s hepatic function after pregnancy is indicated.

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