

Prevention and Management of Viral Hepatitis in Pregnancy



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KEYWORDS

- Hepatitis A • Hepatitis B • Hepatitis C • Hepatitis D • Hepatitis E • Viral hepatitis
- Pregnancy

KEY POINTS

- Hepatitis A virus infection is an acute self-limiting infection with a benign course during pregnancy.
- Hepatitis B virus (HBV) infection can cause both acute and chronic hepatitis. Although HBV does not seem to adversely affect pregnancy outcomes, vertical transmission is a risk that is significantly reduced by immunoprophylaxis of the newborn.
- Hepatitis C virus (HCV) infection is the most common blood-borne infection in the United States. Vertical transmission seems to be related to degree of maternal viremia, and efforts for vaccine development are promising.
- Hepatitis D virus (HDV) requires coinfection with HBV for propagation, and tends to have a more rapid progression to cirrhosis despite suppressing HBV viremia. Prevention of HBV is the mainstay of the prevention of HDV.
- Hepatitis E virus is the most common cause of acute hepatitis worldwide, and portends a 20% risk of maternal mortality during pregnancy. Two vaccines are available for at-risk populations in China, but studies are needed in pregnant populations before widespread use becomes viable.

HEPATITIS A

Introduction

Disease description

Hepatitis A virus (HAV) is a single-stranded 27-nm RNA picornavirus. HAV infection is usually a self-limiting illness that does not lead to chronic infection, and HAV immunoglobulin G (IgG) provides lifelong immunity. The average incubation period is 4 weeks. The virus can be detected in blood and feces 10 to 12 days after initial infection. A person is most contagious 14 to 21 days before the onset of symptoms and

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continues to be so 1 week after symptoms begin. Symptomatic infection depends on age of acquisition. Approximately 10% of children who become infected are symptomatic. In countries of high endemicity, clinical disease is rare, as greater than 90% of people are infected as children and become subsequently immune by adulthood. In countries of lower endemicity, symptomatic infection is seen in adolescents and adults of high-risk groups, such as injection drug users, men having sex with men, travelers to high-prevalence areas, and members of closed religious communities.¹ As a result, HAV presents a significant economic burden to countries of low prevalence, such as the United States.¹

Early infection is characterized by malaise, fatigue, fever, anorexia, nausea, and abdominal pain, followed by jaundice and dark urine.^{2,3} Symptoms usually last no more than 2 months, but can persist or relapse up to 6 months after initial infection in 10% to 15% of patients.⁴ Fulminant hepatitis occurs in 0.01% of cases. Liver function rapidly deteriorates, and fatality rates are high when this occurs.

Risk factors

Humans are the only reservoir for HAV. Transmission occurs via the fecal-oral route, person-to-person contact, or contaminated food and water. Much less commonly, cases have been reported after intravenous drug use, blood transfusion, and sexual contact, although usually during the early stages of disease when HAV viral loads are highest.³ Travel to endemic areas, household contacts of infected persons, and day care and health care settings are all known risk factors, although up to half of patients have no identifiable risk factor.

Prevalence/incidence/mortality rates

The incidence of HAV varies according to the socioeconomic development of the area. Globally approximately 1.4 million new cases of hepatitis A are diagnosed each year.¹ In 2011, the overall incidence rate in the United States was 0.4 per 100,000 population.⁵ Since the introduction of the HAV vaccine in 1995, the incidence has decreased by 95% in the United States.⁶ Mortality from acute infection is less than 1% but can be higher in the setting of preexisting liver disease.^{3,7}

Clinical Outcomes (Pregnancy/Maternal/Fetal)

Acute HAV infection during pregnancy is rare. As a result, incidence during pregnancy is difficult to ascertain. Most cases reported in the literature are those requiring hospitalization and/or recorded in countries endemic for HAV where acute infection in the adult population is low.² For example, Elinav and colleagues² found that only 13 of 79,458 pregnancies admitted to an Israeli hospital over a 25-year period were diagnosed with acute HAV. During the HAV epidemic in Tennessee from 1994 to 1995, only 4 of the 1700 cases reported were pregnant women.⁸

In general, maternal and fetal outcomes are excellent in developed nations. Preterm birth has been associated with HAV infection in the second and third trimesters, in addition to neonatal cholestasis.^{2,9} In the same series from Israel,² the average gestational age at delivery in women with HAV was 34 weeks. When fever and hypoalbuminemia (defined as albumin <30 g/L) was present, delivery was significantly earlier, at a mean gestational age of 32 weeks.² Hepatitis A is not teratogenic, and transmission to a fetus antepartum, intrapartum, and postpartum via breast milk is rare.

Management

Diagnosis

Diagnosis requires serologic testing. The presence of HAV immunoglobulin M (IgM) is diagnostic of acute infection and persists for several months. HAV IgG predominates

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