HEPATITIS B

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Author: Diana Harland, BS, CCRC
Editor: Cheryl Duksta, RN, ADN, M.Ed

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About the Author

Diana Harland, BS, CCRC, received her degree in Microbiology from the University of Texas at El Paso. She worked in pre-clinical research in retrovirology while in undergraduate school, and again after graduation at Texas Biomedical Research Institute (formerly Southwest Foundation for Biomedical Research) in San Antonio, Texas. While at SWFBR, she worked in the department of Virology and Immunology at biosafety level 3-4 (BSL-3/BSL-4). She has extensive training in NIH, OSHA, and CDC guidelines for sterility and asepsis in tissue culture and retrovirology. She holds a certificate in bioterrorism from Tulane University School of Public Health and Tropical Medicine and the University of Alabama at Birmingham School of Public Health. She is a member of the Austin Disaster Relief Network (infrastructure for the city of Austin, TX disaster response), and the Association for Clinical Research Professionals.

Editing done by **Cheryl Duksta, RN, ADN, M.Ed.,** is a critical care nurse and current member of the American Association of Critical Care Nurses. She has 15 years of publishing experience, specifically, writing and editing educational and health-related books and journals. A master's-prepared teacher, Cheryl has experience in curriculum development at the state and national level and she also serves as a publishing consultant.

Instructional Objectives

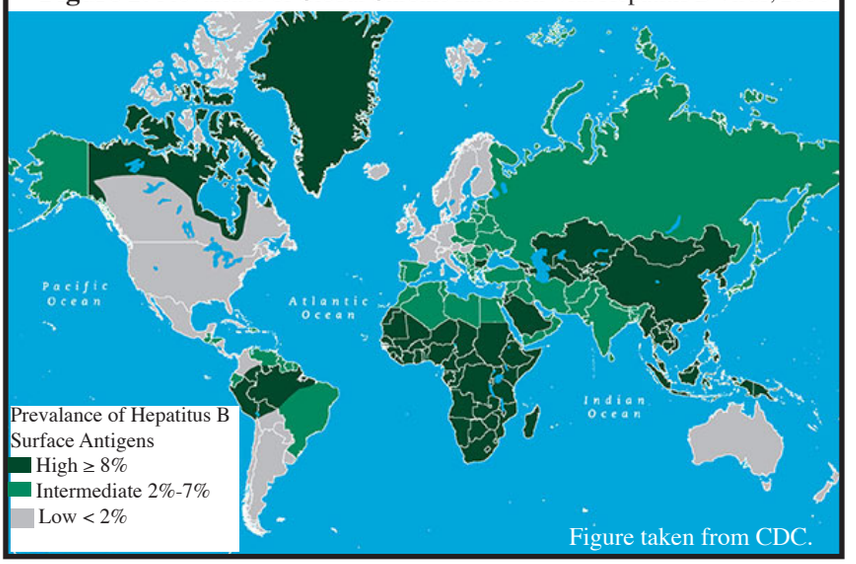
Upon completion of this course, the learner will be able to:

1. Describe the hepatitis B virus and compare its ability to cause infection with the HIV virus.
2. State the regions of the world where hepatitis B is endemic.
3. Relate the mechanisms of transmission of the hepatitis B virus and identify risk factors and persons at risk for contracting the disease.
4. Describe the serological markers used to diagnose and treat hepatitis B.
5. Identify the differences between acute and chronic hepatitis B, and outline the drugs for treatment of chronic HBV infection.
6. Explain why the hepatitis B vaccine is so important in preventing the disease.
7. Summarize the ways healthcare workers can act to reduce transmission of HBV.

Purpose and Goals

The purpose of this course is to give the healthcare professional an overview of the hepatitis B disease, with emphasis on the understanding of serological markers and the treatment of chronic hepatitis.

Figure 1: Prevalence of Global Chronic Infection with Hepatitis B Virus, 2006



Introduction

Hepatitis B is a highly contagious bloodborne viral infection of the liver caused by the **hepatitis B virus (HBV).** Infection can range in severity from mild and short term **(acute hepatitis B)** to severe and long term **(chronic hepatitis B).** Chronic hepatitis B that is left untreated can lead to cirrhosis (scarring) of the liver, **hepatocellular carcinoma (HCC)**, and liver failure.

It is estimated that one-third of the world's population (over 2.3 billion people) have been infected with the hepatitis B virus at one point in their lives and that about 350 million people worldwide are chronic carriers of the virus. **Figure 1** shows the most current worldwide prevalence of chronic HBV infection. The CDC estimates that 800,000 to 1.4 million Americans are infected with chronic hepatitis B and as many as two-thirds of them do not know that they are infected and that they can unknowingly spread the virus. For these reasons, hepatitis B is a significant occupational hazard for healthcare workers. Fortunately, rates of acute hepatitis B in the U.S. have been declining since 1990 due to routine vaccination of children.

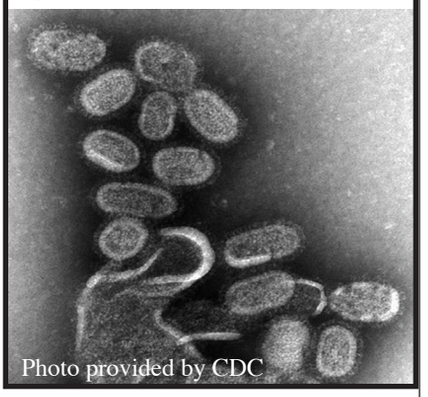
Other viruses that cause hepatitis include **hepatitis A virus (HAV), hepatitis C virus (HCV), and hepatitis D virus (HDV)**. HDV can only cause infection if HBV is first present and can be in the form of a coinfection with HBV or a superinfection that presents after a person has been infected with HBV for some time.

Characteristics and History of the hepatitis B Virus

The HBV virus is classified in the orthohepadnavirus genus and the hepadnavirus family of viruses, "hepa" from the Latin word meaning "liver," and "dna" from the fact that it is a DNA virus. It is one of the smallest animal viruses, consisting of an outer lipid envelope with a protein core. The genetic material (or genome) is composed of circular DNA that is not completely double stranded. **Figure 2** shows a transmission electron micrograph of hepatitis B virions.

In 1965, Nobel Prize-winning scientist Baruch Blumberg was working with fel-

Figure 2:Transmission electron micrograph showing several large round hepatitis B virions.



low scientist Harvey Alter to study the serum from hemophiliac and leukemia patients who had received multiple blood transfusions. They noticed that the serum from one hemophiliac patient reacted with a sample of serum from an Aboriginal Australian. They called the newly discovered antigen the "Australia antigen." In 1968, the link between the Australia antigen as being a part of the virus that caused "serum hepatitis" was confirmed by virologist Alfred Prince, and the antigen was later renamed as the **hepatitis B surface antigen (HBsAg)**. In 1972, laws were passed that required donated blood to be tested for HBsAg, and thereafter, acquiring HBV from blood transfusions became a rare occurrence. Hepatitis B virus was the first human disease-causing virus to be sequenced (identification of the exact order of DNA bases in the genome), making recombinant vaccine research possible. In 1986, following more years of research, a vaccine produced from recombinant HBsAg made from yeast cells (rather than producing HBsAg derived from blood plasma) was approved by the Food and Drug Administration (FDA). This is the current vaccine in use today.

When a person becomes infected with HBV, the virus is transported by the bloodstream to the liver. This is the primary site for viral replication. The highest concentration of HBV is found in blood, and lower concentrations are also found in other body fluids, such as semen, vaginal secretions, wound exudates, and saliva.

The hepatitis B virus is 50-100 times more infectious than the **human immunodeficiency virus (HIV).** Unlike HIV, it is resistant to heat and cold and can survive outside the body on surfaces for a minimum of 7 days and possibly up to weeks, even when visible blood is absent.

Epidemiology and Trends

Because of great efforts to immunize children at a very early age, the rate of new hepatitis B infections in the U.S. has decreased dramatically by 82% since 1991 when routine vaccination of HBV began. In 2009, the CDC estimated that 38,000 persons in the U.S. were newly infected with HBV. Estimates by the CDC must take into account the fact that many HBV infections are either asymptomatic or never reported. Estimated numbers will therefore be tenfold more than actual numbers.

Hepatitis B is endemic in China and regions of Asia. Asians and Pacific Islanders are particularly at risk for hepatitis B. In the United States, this group of people makes up less than 5% of the population but accounts for more than 50% of Americans living with chronic hepatitis B. Most were infected as young children. Liver cancer from HBV is a leading cause of death among Asians and Pacific Islanders, and they are 7 times more likely to die from hepatitis B than are Caucasians.

Persons infected with HBV may also be **coinfected** with HIV. These persons are at increased risk for developing chronic HBV, and are at increased risk for serious medical complications including liver-related morbidity and mortality. Liver disease from coinfection of HIV and HBV or HCV is now the most common non-AIDS related cause of death in this population.

Transmission of hepatitis B

Ways hepatitis B is transmitted:

- Exposure to blood and/or blood products from an infected individual
- Sexual contact with an infected partner—this is the most common method of spreading the disease among adults in the U.S.
- Use of contaminated needles or other drug-injection paraphernalia by injecting drug users
- Sharing personal items such as toothbrushes or razors with an infected person
- Perinatal transmission—passed from infected mother to infant at birth
- Contact with open sores or contaminated body fluids of an infected individual
- Accidental needlesticks
- Contact with contaminated surfaces

Ways hepatitis B is NOT transmitted:

- Through food or water
- Shared eating utensils
- Breastfeeding
- Hugging, kissing, or holding hands
- Coughing or sneezing
- Casual touching
- Casual contact in the workplace

Risk factors for hepatitis B infection:

- Having unprotected sex
- Having multiple sex partners (> 1 sex partner in the previous 6 months)
- Men having sex with men (MSM)
- Having a sex partner who has other multiple sex partners or is a MSM
- Having a sex partner who is an injection steroid or injection drug user
- Being an injection drug user
- Sharing needles or any equipment that is used to inject drugs (cookers, filtration cotton, etc.)
- Engaging in a business that exchanges sex for money or drugs
- Having a recent sexually transmitted disease(STD)
- Being born to a mother who is infected with hepatitis B
- Being a household contact of a person with chronic HBV
- Being a hemodialysis patient
- Working in a healthcare or public safety position where there is risk for occupational exposure to blood or contaminated body fluids
- Working as a staff member or being a resident of a facility for developmentally disabled persons
- Having dental, medical, or cosmetic procedures done with needles or other equipment that may be contaminated with blood
- Traveling to a country with

intermediate or high prevalence of HBV infection

STDs and Hepatitis B

Statistics from the CDC show that 10-40% of persons seeking treatment for STDs have evidence of previous or current hepatitis B infection. A CDC study showed that 39% of adults diagnosed with HBV had previously sought care for an STD or had been screened for an STD. For these reasons, the CDC highly recommends vaccination of at-risk persons who have or are seeking treatment for an STD.

Identification of Persons with Chronic HBV

It is important to identify persons with chronic HBV as soon as possible because treatment to delay or prevent onset of liver disease can be initiated. Also, further spread of the disease to others can be halted because chronic HBV carriers are the primary source of new HBV infections.

According to the CDC, identifying persons with chronic HBV permits the following:

1. Clinical evaluation to detect onset and progression of liver disease
2. Antiviral treatment that can delay or reverse progression of liver disease
3. The ability to obtain a baseline **alpha-fetoprotein (AFP)** result and obtain a periodic ultrasound to detect HCC at a potentially treatable stage. Early intervention and procedures that include ablation of small localized tumors, resection, and transplantation have resulted in long-term tumor-free survival.
4. The ability to make interventions that can reduce progression of liver injury. Such interventions include vaccination against hepatitis A (because chronic liver disease will increase morbidity and mortality of hepatitis A) and counseling to avoid excessive alcohol consumption (because use of greater than 25-30ml of alcohol per day is associated with progression of HBV-related disease).

The AFP tumor marker

Alpha-fetoprotein is a protein produced in high levels by the fetal liver. After birth, levels decline rapidly, and in healthy non-pregnant adults, AFP is detectable only in very low levels. The AFP test is used to diagnose cancers of the liver and monitor patients with chronic liver disease because levels can be elevated with liver damage and liver cancers. This test is also ordered periodically to monitor patient response to therapy for HCC and to check for any cancer recurrence. The AFP test result is an indicator and not a diagnostic marker. Sharp increases are more important than numerical values. Because moderate elevations can be seen in other conditions, testing can yield false positives. About 60% of patients with liver cancer will have an elevated AFP, and 40% of patients with liver cancer will have an AFP test in the normal range.

Clinical Presentation

Acute hepatitis

The incubation period for hepatitis B from initial exposure can range from 6 weeks to 6 months, with an average of 2-3 months. Symptoms of HBV infection will vary by age, health, and stage of infection. In persons ≥5 years, 30-50% will show symptoms of acute disease lasting from several weeks to 6 months. Infants < 5 years and immunosuppressed individuals (e.g. HIV patients or hemodialysis patients) are usually asymptomatic. Acute HBV symptoms can range from mild to fulminant hepatitis in rare cases. Symptoms may include fever, fatigue, abdominal pain, nausea, vomiting, loss of appetite, joint pain, dark urine, and pale, clay-colored stools. Other symptoms include jaundice, liver tenderness, and possible hepatomegaly and splenomegaly. Symptoms are more severe among persons over age 60. Fatality rates, as determined by CDC surveillance, are 0.5% to 1% of cases.

Acute hepatitis can be self-limiting and may resolve with viral elimination from the blood and lasting immunity from reinfection, or the disease may progress to chronic hepatitis. Having had a resolved primary infection is not a risk factor for later occurrence of either chronic liver disease or HCC.

Chronic hepatitis

In cases of chronic HBV, as time progresses, the virus destroys the hepatic cells of the liver, giving them the appearance of ground glass **(ground glass hepatocyte or GGH)** when viewed microscopically. Macroscopically, the diseased liver becomes fibrous and orange-yellow in color as healthy cells are replaced by scar tissue and nodules.

Progression to chronic hepatitis B is inversely related to age at the time of infection. Greater than 90% of infants, 25-50% of children age 1-5, and < 5% of older children and adults will develop chronic hepatitis. Only 0.5% of chronic cases will resolve annually, and this is indicated by undetectable HBsAg levels and normal serum ALT levels.

Although some people can experience symptoms similar to those of acute hepatitis, most persons with chronic HBV will have no symptoms and no evidence of liver disease for as many as 20 to 30 years. Symptoms can range, however, from asymptomatic to systom associated with cirrhosis or liver cancer in 15-25% of patients.

Diagnosis

Diagnosis of hepatitis B is made by patient history, lab testing and evaluation of liver function tests (LFTs), and serologic testing. Patients with hepatitis B will have high levels of **alanine aminotransferase (ALT or SGPT)** and **aspartate aminotransferase (AST or SGOT)**. ALT and AST may be greater than 10 times normal levels in cases of acute hepatitis. In cases of chronic hepatitis, ALT and AST may be less than four times normal values or can vary between normal and slightly increased.

Serological Markers and Their Meanings

There are a number of serologic tests for HBV, which alone or in combination can help the clinician identify the presence and stage of the hepatitis B disease:

1. **HBsAg (hepatitis B surface antigen)**. This protein is located on

the surface of the virus. Because the natural immune response to hepatitis B infection is to make antibodies to HBsAg, it is used to make the hepatitis B vaccine. The presence of HBsAg indicates a person is infectious. It can be detected by an average of 4 weeks postinfection (range of 1-9 weeks). It can be found in high levels during:

a. Acute infection
b. Chronic infection

All persons with positive HBsAg should be considered infectious. Persons who do not become chronically infected will be HBsAg negative by 15 weeks after symptom onset.

Testing for hepatitis B surface antigen (HBsAg) is recommended by the CDC for:

- Persons with elevated liver enzymes with no known cause
- Men who have sex with men (MSM)
- Persons born in the U.S. who have not been vaccinated and whose parents are from an area with > 8% prevalence of HBsAg
- Healthcare workers who have had an accidental exposure through needlesticks, cuts, etc.
- Persons with suppressed immune systems
- Anyone infected with HIV
- Any close contacts of persons infected with HBV
- Pregnant women
- Persons born in areas of the world with a greater than 2% prevalence of HBsAg including most of Asia and Africa

2. **Anti-HBs (hepatitis B surface antibody)**. Presence of this antibody indicates:
   a. Recovery and immunity from HBV infection
   b. Successful vaccination against HBV
3. **Anti-HBc (Total hepatitis B core antibody)**. This antibody will be present in:
   a. Acute hepatitis (and will remain for life)
   b. Any previous or ongoing infection (with no defined time frame)
4. **IgM anti-HBc (IgM antibody to hepatitis B core antigen)**. This antibody is present in:
   a. Recent infection of HBV < 6 months
   b. Acute infection
5. **HBeAg (hepatitis B e antigen)**. The presence of this antigen indicates high levels of HBV and good viral replication. It should be noted that certain strains of HBV (common in the Middle East and Asia) do not make an e antigen, making this test of no use for these strains. This antigen is present in:
   a. Acute hepatitis
   b. Chronic hepatitis
6. **HBeAb or anti-HBe (hepatitis B antibody)**. This antibody is found during:
   a. Acute infection—produced temporarily
   b. Viral replication burst—produced consistently during or after
7. **Hepatitis B viral DNA**. This test detects the presence of HBV viral DNA in blood. It is primarily used to monitor drug efficacy in chronic HBV infections. Hepatitis B viral DNA is present during active infection.

Table 1 shows CDC guidelines for the interpretation of serologic test results.

Table 1: Interpretation of Hepatitis B Serologic Test Results

HBsAg **anti-HBc** **anti-HBs**	negative negative negative	Susceptible
HBsAg **anti-HBc** **anti-HBs**	negative positive positive	Immune due to natural infection
HBsAg **anti-HBc** **anti-HBs**	negative negative positive	Immune due to hepatitis B vaccination
HBsAg **anti-HBc** **IgM anti-HBc** **anti-HBs**	positive positive positive negative	Acutely infected
HBsAg **anti-HBc** **IgM anti-HBc** **anti-HBs**	positive positive negative negative	Chronically infected
HBsAg **anti-HBc** **anti-HBs**	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

Adapted from CDC

Classifying and Reporting hepatitis B

The CDC has developed criteria to provide uniform clinical and lab testing criteria for identification and reporting of hepatitis B cases**. Table 2** shows the case definition criteria for identification and reporting of hepatitis B as outlined by the CDC.

Treatment

Acute HBV

Currently, there is no medication for treatment of acute HBV. Treatment consists of supportive therapy including rest, nutrition, fluids, and hospitalization when necessary.

Chronic HBV

Chronic HBV can be treated with **interferon alpha-2b (IFNa-2b), PE-Gylated interferon alpha-2a (PEG-IFNa-2a)**, or other antiviral drugs, such as the nucleoside/nucleotide analog drugs **lamivudine, tenofovir, adefovir dipivoxil, telbivudine, and entecavir.** Goals for treatment of chronic HBV in-

Table 2: CDC Case Definition Criteria for Identification and Reporting of Hepatitis B

	Acute Hepatitis B	Chronic Hepatitis B		
Clinical Symptoms	Acute illness with: (a) discreet onset of symptoms **AND** (b) Jaundice **or** serum ALT > 200 IU/L	No symptoms required; no evidence of liver disease or may show a spectrum of disease ranging from chronic hepatitis to cirrhosis or liver cancer		
Laboratory Criteria		**Criteria A OR Criteria B**	Any combination of the tests below performed at least 6 months apart	
IgM anti-HBc (IgM antibody to Hepatitis B core antigen)	Positive	Negative **AND A Positive to one of the following below:**		
HBsAg (Hepatitis B surface antigen)	Positive	Positive	Positive 2X at least 6 months apart	
IgM anti-HAV	Positive if done			
HBeAg (Hepatitis B e antigen)		Positive	Positive 2X at least 6 months apart	
HBV DNA (Hepatitis B virus DNA)		©NCCE Adapted from CDC		

clude stopping viral replication, reducing chances of further liver damage and the occurrence of HCC, and reducing infectivity. In patients who are in the latest stages of HBV disease, liver transplantation may be the only treatment option.

Patients on any antiviral therapy will undergo long periods of time on these drugs and must be monitored regularly for progression of liver disease and efficacy of treatment. Response to treatment may vary with each individual and may depend in part on the genotype of the virus and each individual's own immune system. Success of treatment is measured by:

1. **sustained virologic response (SVR).** This is said to be achieved when the virus can no longer be detected in the blood for at least 6 months after stopping treatment.
2. **normalization of ALT levels.**
3. **seroconversion of HBeAg.** This occurs when there is loss of HBeAg in blood followed by production of detectable levels of HBeAb in blood. The CDC recommends that treatment be continued in patients who have lost HBeAg for at least 6 additional months. Relapse rates for these individuals are 80-90% if treatment is stopped in 1-2 years.
4. **viral load.** This is a measurement of the amount of viral DNA in the blood. The goal is to have HBV DNA levels < 10,000 copies/ml.
5. **Improvement in liver biopsy results.**

What Are Interferons?

Interferons are low molecular weight, heat stable glycoproteins that are produced by cells that have been exposed to viruses, bacteria, or chemicals. They function as a first-line of defense in response to infection. They "interfere" with viral replication. Patients with chronic hepatitis have been shown to have decreased levels of interferon production when compared to persons who are not infected; therefore, boosting interferon levels in these patients is the reasoning behind interferon treatment.

Different interferons will affect the body differently. **Alpha interferons** are produced in the body by leukocytes and function to inhibit viral replication, suppress cell proliferation, and regulate immune response. Alpha interferons are used to treat viral infections and cancers. **Beta interferons** are produced by in the body by fibroblasts and tissue type cells and are used in the treatment of multiple sclerosis. **Gamma interferons** are produced by lymphocytes and are used to treat chronic granulomatous disease. Interferons can also be made in the lab from recombinant DNA by taking cells grown in tissue culture and inserting genes for interferon synthesis into them. **Pegylated interferons** have an additional **polyethylene glycol (PEG)** added to the interferon through a process called **pegylation** (covalently coupling PEG to a larger molecule). The addition of PEG enhances the half-life of the drug, making it longer acting and enabling patients to take weekly doses rather than daily doses. When pegylated interferon alpha 2a is taken, many patients show a greater chance of achieving SVR. Individuals who have taken pegylated interferon alpha 2a have also shown improvements in liver histology. Other research has shown that alpha interferon therapy can reduce liver cirrhosis and development of HCC.

Side effects of therapeutic interferons (aches, fever, and flu like symptoms) are similar to the side effects produced by naturally made interferons in the body. Symptoms may be mild or more pronounced. The most common side effect is fatigue. Other symptoms include muscle and joint pain, malaise, flu like symptoms, fever, headache, skin irritation, rashes, hair loss, nausea, vomiting, decreased appetite, sadness, depression, insomnia, and mood changes. Side effects seen in the blood may include anemia, neutropenia, and thrombocytopenia, which should all reverse when treatment is concluded.

Reactivation of Hepatitis B

Patients with chronic HBV who have had clearance of HBsAg in serum may still later show reactivation of a latent viral infection. Although there may be clearance of HBsAg from serum, HBV DNA may still be detectable in the liver, in bodily secretions, and in mononuclear cells of peripheral blood. This suggests that HBV can remain in a state of low level replication or in a state that can be reactivated to again become infectious. Immunocompromised individuals (steroid use or contracting the HIV virus) are at greatest risk of relapse. **Virologic recurrence** is defined as an increase of HBV DNA to > 10,000 copies/ml in a person that had previously shown seroconversion and HBV DNA < 10,000 copies/ml (confirmed by consecutive samples). **Serologic recurrence** is defined as reappearance of HBeAg positivity (confirmed by consecutive samples) after a previous seroconversion. Current research indicates that antiretroviral drugs that are reverse transcriptase inhibitors (nucleotide and nucleoside analogs) will enable only temporary HBeAg seroconversion in most patients.

Postexposure prophylaxis

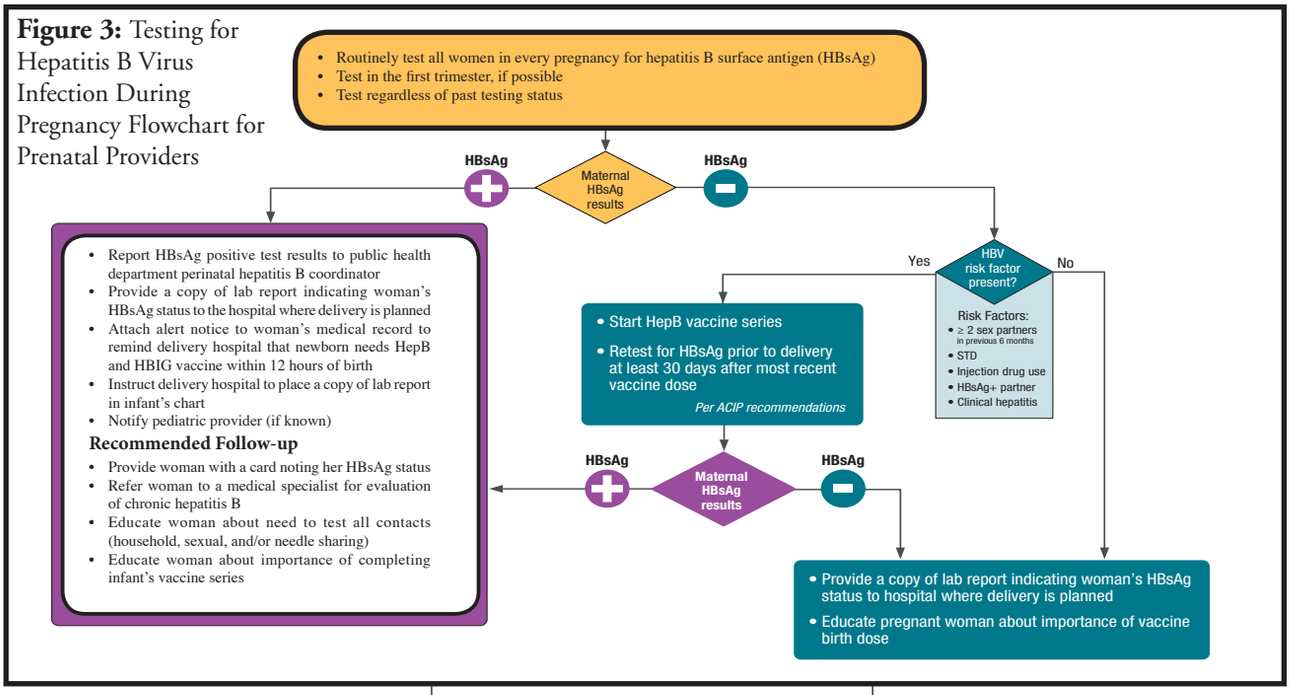
Persons exposed to HBV may be able to prevent HBV infection if given a hepatitis B vaccination and **hepatitis B immune globulin (HBIG)** within 24 hours of exposure. HBIG is made from the plasma of human donors who have high levels of HBsAg. Temporary immunity (for approximately three to six months) to HBV is provided by means by passive transfer of immunoglobulin. HBIG can be used for postexposure prophylaxis in conjunction with the hepatitis B vaccine in persons who are not previously vaccinated, or it can be used alone in persons who do not respond to the vaccine. A person who is exposed to an HBsAg positive source who is able to provide documented evidence of vaccination series but does not have post-vaccination testing should receive a single vaccine booster dose.

Vaccination

The hepatitis B vaccine is a very effective vaccine that provides greater than 90% protection to persons who have received all three doses. Vaccination is the single most important way to prevent hepatitis B. Since 1982 when it became available, over one billion people have been vaccinated worldwide with no serious side effects reported. The vaccine is contraindicated in persons who have had a previous reaction or are allergic to any of its components and in persons who are allergic to yeast because yeast is used in vaccine production.

Levels of protective antibody that can

Figure 3: Testing for Hepatitis B Virus Infection During Pregnancy Flowchart for Prenatal Providers



be made by the body are proportional to age at vaccination. Protective antibody levels of > 95% can be achieved in infants, children, and young adults. After age 40, protective levels are achieved in less than 90% of those vaccinated. At age 60, only 65-75% of those vaccinated will show protective levels.

Currently, there are five vaccines licensed for use in the U.S.:

1. Engerix-B®: single antigen vaccine.
2. Recombivax®: hepatitis B single antigen vaccine
3. Comvax®: combined hepatitis B and Haemophilus influenza b (Hib) vaccine
4. Pediarix®: combined hepatitis B, diphtheria, tetanus, acellular pertussis (DTaP), and inactivated poliovirus (IPV) vaccine.
5. Twinrix®: combined hepatitis A and hepatitis B vaccine

The vaccine schedule for infants is first dose within 24 hours of birth; a second dose at 1-2 months of age with a minimum interval of 4 weeks between dose 1 and 2; and a third dose (from age 6 months to 18 months) no earlier than age 24 weeks with at least 16 weeks after the first dose. The vaccine schedule for adults is three doses as follows: first dose- month one; second dose-one month after the first dose; and third dose--at least two months after the second dose and at least 4 months after the second dose. Vaccines from different manufacturers may be used to complete the vaccine course because no differences in immune responses have been observed when different manufacturers' vaccines have been used. Extra doses of the vaccine are not harmful.

Booster vaccination is recommended by the CDC for:

- All infants, beginning with first dose at birth
- Any children or adolescents younger than 19 who have not been previously vaccinated or did not receive the entire vaccine series
- All sexually active persons who are not in a longterm mutually monogamous relationship (> one sex partner in the previous 6 months)
- Anyone seeking evaluation or treatment of an STD
- Men who have sex with men (MSM)
- Persons who are injection drug users
- Persons who are close household contacts of someone who is positive to HBsAg
- All healthcare or public safety workers who are at risk for exposure to blood or fluids contaminated with blood
- All dialysis patients
- All staff or residents of facilities for developmentally disabled persons
- Anyone traveling to regions with moderate to high rates of endemic HBV infection
- All persons infected with HIV
- All persons with chronic liver disease
- Anyone in healthcare settings targeted to STDs, HIV testing and treatment, drug abuse treatment and services, services to injection drug users, services to MSMs, hemodialysis facilities or end-stage renal disease programs, correctional facilities, and institutional and nonresidential facilities for developmentally disabled persons
- All unvaccinated persons age 19 to 59 with diabetes mellitus (persons ≥ 60 years may be vaccinated at the clinician's discretion)
- Anyone wanting protection from HBV; a specific risk factor is not required for vaccination

Prevention of Perinatal Transmission

It is important to identify pregnant women who are HBV positive (HBsAg positive) to prevent infection in newborns.(**See Figure 3**) Without intervention, 40% of infants born to HBV positive mothers will develop chronic HBV, and one-fourth of these infants will later die from chronic liver disease.

HBV is not transferred across the

placenta. Infants become perinatally infected during birth when exposed to their mother's blood. Transmission to newborns can be prevented by providing the vaccine and hepatitis immune globulin (HBIG) within 12 hours of birth.

Preventing perinatal transmission is a vital part of the national strategy to eliminate HBV infection in the U.S. By law, many states now require maternal screening and reporting of HBV. A list of states and requirements is available on the CDC website at http://www2a.cdc.gov/nip/StateVaccApp/statevaccsApp/HepatitisScreenandReport.asp.

Disinfection of Hepatitis B in the Environment

The hepatitis B virus can survive outside the body for at least 7 days and still be capable of causing infection. Some sources have reported that the virus is capable of surviving on surfaces for up to 2 weeks. The primary way to prevent occupational exposure to any bloodborne pathogen is to diligently follow standard infection control practices, assuming that all blood and body fluids are potentially infectious. Adhering to individual facility infection control policies, including guidelines for the prevention of needlestick injury, will also prevent exposure to HBV. The practice of proper handwashing cannot be overemphasized because handwashing is still considered to be the single most effective method to prevent transmission of infection. For surfaces, the CDC recommends cleaning any blood spills or dried blood with a 1:10 dilution of household bleach. As with disinfection of all bloodborne pathogens, gloves should also be used during any decontamination procedures. For further information on infection control, please see the NCCE course entitled Science of Infection Control Principles.

The CDC *Guidelines for Isolation Precautions for Preventing Transmission of Infectious Agents in Healthcare Settings* is available on the CDC website at http://www.cdc.gov/hicpac/2007IP/2007isolat

Your Role as a Healthcare Provider

Protecting yourself and your patient from transmission of hepatitis B through proper disinfecting techniques is only one of your responsibilities as a healthcare provider. You must also educate your patients on common routes of transmission and options for routine vaccinations. Your role as educator also includes identifying those at risk for the disease and providing them with information so they can protect themselves from infection. Working together, healthcare providers can continue to protect each other and the public from this disease.

Notes

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