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March 2008 "Update: Hepatitis" 707-000-08-003-H01-P



**UPDATE:  
HEPATITIS**

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**HAVE YOU RECENTLY MOVED? PLEASE NOTIFY US.**

This lesson takes a look at 5 types of hepatitis. As pharmacists, we receive a lot of inquiries from patients about this disease. Our goal is to provide information that can be shared with patients. This lesson provides 1.25 hours (0.125 CEUs) of credit, and is intended for pharmacists in all practice settings. **The program ID # for this lesson is 707-000-08-003-H01-P. Pharmacists completing this lesson by March 31, 2011 may receive full credit.**

To obtain continuing education credit for this lesson, you must answer the questions on the quiz (70% correct required), and return the quiz. Should you score less than 70%, you will be asked to repeat the quiz. Computerized records are maintained for each participant.

If you have any comments, suggestions or questions, contact us at the above address, or call toll free 1-800-323-4305. (In Alaska and Hawaii phone 1-847-945-8050). **Please write your ID Number (the number that is on the top of the mailing label) in the indicated space on the quiz page (for continuous participants only).**

**The objectives of this lesson are such that upon completion the participant will be able to:**

1. Define hepatitis.
2. Differentiate between the various types of hepatitis.
3. Describe tests used to diagnose the disease types.
4. Discuss modes of transmission.
5. Discuss pre-exposure & post-exposure prophylactic methods used to prevent hepatitis.

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## REVIEW OF HEPATITIS

Hepatitis, which is inflammation of the liver, is a common disease that affects many people worldwide. This lesson focuses on five viral hepatitis types. Once developed, hepatitis exhibits itself by symptoms like nausea, vomiting, loss of appetite, abdominal pain, hepatomegaly, and fatigue; increase in serum bilirubin and aminotransferase; presence of a hepatitis viral genome in the liver and blood, as well as the appearance of antibodies to viral antigens; and the detection of varying degrees of hepatocellular necrosis and inflammation. From a clinical point of view, all types of viral hepatitis are similar, but can be differentiated by serological examinations.

Viral hepatitis is caused by one or a combination of the following viruses: hepatitis A (HAV), hepatitis B (HBV), hepatitis C (HCV), hepatitis D (HDV), hepatitis E (HEV) and hepatitis G (HGV). Although the number of cases reported annually in the U.S. is about 56,000, the actual figure is probably much higher. Viral hepatitis is normally a self-limited disease, and the number of fatalities is usually low. It can, however, cause pain, discomfort, all types of suffering and excessive absences from work. Infections begin with an incubation period. After establishment, the infective viral particles appear in the blood and other body secretions, body fluids and tissues. Throughout the incubation period, (duration depending on the viral type), the patient is asymptomatic. Viral hepatitis may cause inflammatory necrosis of the liver. This liver injury may be due to host immune response or direct damaging effect on the hepatocytes. The acute hepatitis phase occurs after the incubation period and may manifest itself by flu-like symptoms, anorexia, nausea, fatigue and malaise. This occurs before the emergence of jaundice or cellular liver damage. In most cases, the acute hepatitis phase produces mild symptoms only, with minimal hepatocyte injury and mild elevation of liver enzyme levels. However, since patients may develop hepatocellular damage, this may result in clinical symptoms such as jaundice, fever, right upper-quadrant abdominal pain, nausea, vomiting, dark urine, stools that are almost colorless, and a significant increase in liver enzyme levels. Viral serologic tests, as well as blood antibodies in such patients will appear. Depending on the virus type, most patients recover without the development of chronicity or complications.

### HEPATITIS A VIRUS (HAV)

Hepatitis A virus is a 27 nm RNA hepatovirus that is a member of the Picornaviridae family. Once in circulation, the virus reaches the liver where it replicates and settles in the hepatocyte cytoplasm. It is mostly secreted in the bile, but some virus can reach the serum. HAV may cause epidemic or sporadic cases of hepatitis. The disease is **highly contagious** and transmission occurs mainly by the fecal-oral route, in particular where sanitary conditions are inadequate. HAV can be transmitted from person to person or through ingestion of contaminated food and water. The virus is considered the **most common** cause of acute hepatitis in the U.S., occurring primarily from person to person transmission rather than in epidemic outbreaks. Incidence of HAV in the U.S. is 10.8 per 100,000 people, mostly involving outbreaks in lower socioeconomic groups and sporadically in places such as day care centers. Ingestion of shellfish from contaminated water may trigger infections. Transmission of the disease in contaminated blood is rare. International travelers to countries where HAV infection rate is high are at risk. Children in day care centers, male homosexuals and institutionalized individuals are also at high risk. Those who are recommended for HAV vaccine include children who reside in places where the rate of HAV is at least twice the national average, international travelers to countries with high or intermediate rate of endemic outbreaks, male homosexuals or patients with chronic liver disease such as hepatitis B and C. The average incubation period of the disease is about 30 days. An infected person will excrete HAV in feces for up to two weeks before the appearance of clinical symptoms. After the first week of illness or rise in liver enzymes, stools will show no viral particles. Replication of HAV

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March 2008

takes place in the liver and liver antigens are encountered in the hepatocytes' cytoplasm during the incubation period. The virus remains in the blood throughout the period when liver enzymes are elevated.

Symptoms of HAV occur at the end of the incubation period. The infection may be asymptomatic depending on the patient's age. Children are usually asymptomatic or may experience mild symptoms such as flu-like illness without clinical jaundice. On the other hand, 70% of adults and children six years of age and older experience clinical symptoms and signs such as jaundice and rise in liver enzyme levels. Morbidity rate is low, and the vast majority of patients completely recover. Fulminant (occurring suddenly, rapidly, and with great severity or intensity, usually with pain) hepatitis rarely occurs following HAV. The infection is responsible for only about 100 deaths a year in the U.S. It may be encountered in patients with chronic hepatitis C. **Chronic carriers of HAV do not exist and patients do not become carriers of the disease.** The infection produces self-limiting symptoms that disappear approximately two months following the onset of the disease. A low percentage of patients may experience an increase in the liver enzyme level for up to six months. The mortality rate of HAV is about 0.3%. The rate is higher among older patients and in those who have chronic HCV.

Antibody (anti-HAV) response to HAV appears in all patients before the onset of the symptoms, usually when the virus begins to disappear from the stool. The antibodies IgM and IgG anti-HAV can be detected in blood 5 to 10 days before onset of symptoms. Later on, the antibody level rises to high titer and may persist for life. The presence of anti-HAV confirms either immunity to HAV or presence of current infection. A blood level of antibody IgM peaks during the first five to 10 days of the onset of clinical manifestation, and disappears within 3 to 6 months. However, the IgM antibodies are replaced by IgG antibodies, which normally persist for life, and their presence indicates immunity to HAV. Diagnosis of acute HAV is confirmed by the detection of IgM anti-HAV in serum of patients with clinical signs of hepatitis. The presence of IgG anti-HAV points to previous exposure to HAV, non infectivity and immunity to the disease. It has been estimated that one third of the U.S. population has been previously exposed to the infection. Other signs that indicate acute HAV are characteristic symptoms of elevation of aminotransferase and bilirubin.

Hepatitis A vaccine is recommended for individuals who are at high risk of exposure to the virus, including travelers to endemic areas, male homosexuals, parenteral drug users, hepatitis researches, patients with chronic liver disease and hemophiliacs. Hepatitis A vaccine consists of inactivated hepatitis A antigen purified from cell culture. The vaccine is considered safe with insignificant (0.1%) complications and adverse reactions. Immune globulin may be used as post exposure prophylaxis in patients who have intimate contact with infected individuals. There is no specific method for treating HAV, since the disease is self-limiting and benign. Recovery is usually achieved within two months after the onset of symptoms. Hospitalization is unnecessary unless the patient develops hepatic insufficiency such as encephalopathy and hemorrhage. Patients should continue their normal daily routine, but rest and avoidance of stress are recommended. In case of the presence of frequent vomiting, fluid and electrolyte replacement should be initiated. Patients should refrain from alcohol consumption, at least during the acute stage of the infection.

### HEPATITIS B VIRUS (HBV)

Hepatitis B virus is a member of the Hepadnaviridae family. HBV infection is common worldwide, and it is a major public health problem. In the U.S. its prevalence is estimated to be 4.9%. In North America, Australia, Europe and temperate South America the disease is transmitted via intimate contact or parenteral drug use. People at high risk include homosexual and bisexual men, workers at hemodialysis centers, health professionals, injection drug users, and workers at blood banks. In the U.S., transmission occurs mostly by contact with blood products or body secretions such as saliva, vaginal fluid and semen. Although saliva contains infectious HBV, studies have indicated that kissing typically may not be a major transmission route. However, biting that causes injuries could result in transmission of the virus. Sexual transmission of HBV accounts for 30 to 60% of new cases annually. About 0.2% of the population in the U.S. is HBsAg positive. (This is the hepatitis B antigen that indicates viral replication). The disease is responsible from 1% to 14% of chronic liver disease.

Anti-HBc: IgM anti HBc antibody is detected in the patient's serum within a short period of time after the emergence of HBsAg. The presence of IgM anti-HBc, which persists for 3-6 months or more, is used as a mean of diagnosis of acute

hepatitis B. IgG anti-HBc appears in the early stages of hepatitis B and remains in the blood indefinitely.

**HBeAg:** This antigen is found in the serum only in the presence of HBsAg. It is formed during the incubation period, almost immediately after the appearance of HBsAg. Detection of HBeAg shows replication of the virus and the presence of an infection. Usually HBeAg lasts only a few months. Its presence in the blood for more than 3 months is a sign that the patient may have, or is in a state of developing, chronic hepatitis B. Its disappearance, and the appearance of anti-HBe, is a sign that viral replication, as well as infectivity, has been reduced.

**HBV DNA:** Usually HBV DNA and HBeAg are simultaneously present in the serum. Its presence signifies viral replication and infectivity, more so than the presence of HBeAg. Small amounts of HBV DNA may remain in the serum after recovery from acute hepatitis B without being infectious. However, in some patients who have chronic hepatitis B the HBV DNA may be found in high levels.

**Clinical manifestations** of HBV infection are similar to those of other viral hepatitis types, and may range from asymptomatic infection to fulminant hepatic failure. In a typical case, the patient may experience nausea, vomiting, anorexia, jaundice, weakness, malaise, fatigue and arthralgia. In general, acute infections in children are asymptomatic, whereas a significant percentage of adults experience symptoms, and only 1% develop fulminant hepatic failure.

Diagnosis of HBV is confirmed by the presence of HBsAg in serum. The level of HBsAg in a small percentage of patients may be so low that it is difficult to detect. Furthermore, the presence of IgM-anti HBc antibody in blood indicates previous HBV infection. As indicated earlier, specific serological tests may give clues as to the cause of the disease. About 2 to 7% of patients with this virus develop chronic HBV. Chronicity is more common in men, immunosuppressed persons, newborns infected with the virus, infants and to a much lesser extent, adults. Chronic hepatitis B is a major cause of cirrhosis in the U.S. and plays a role in triggering liver cancer.

Vaccination against HBV is available and recommended to newborns, children, and adults especially those who are at high risk of acquiring the infection. Post exposure prophylaxis by injecting hepatitis B immune globulin (HBIG) is recommended. The CDC suggests that persons who have had sexual contact with HBV carriers should be given post exposure prophylaxis.

#### HEPATITIS D VIRUS (HDV, A DELTA AGENT)

HDV is a small, single stranded, defective RNA virus that is usually present in the nuclei of infected hepatocytes and is often encountered in some HBV infected patients. **The presence of HBV, namely HBsAg, is needed for replication of HDV.** It disappears once HBsAg is no longer in circulation. Acquiring HDV infection by patients with chronic HBV may result in complications, including fulminant hepatitis B or cirrhosis. Thus infection with HDV may exist as a simultaneous infection with HBV (co-infection), as a super infection that is acquired after exposure to HBV, or as HDV chronic infection. About 5% of HBV- HDV co-infections and the vast majority of HDV super infections result in chronic HDV and other chronic liver disease. It has been estimated that 15 million cases of HDV occur annually worldwide. It is endemic in the Amazon basin and central Africa; and is common in the Mediterranean countries and Eastern Europe. Approximately 7,500 cases of HDV are reported annually in the U.S., and occur primarily in intravenous drug abusers. The incidence is common among parenteral drug users and hemophiliacs. Even though HDV may be transmitted sexually, it occurs less frequently than HBV. Since HDV exists in association of HBV, preventing infection of the later will prevent HDV.

Due to the association of HBV and HDV, HDV infection causes a biphasic increase in the transaminase level. The first increase is due to the HBV infection, and the second elevation is due to the HDV infection. Later on, the disappearance of HBsAg in serum indicates that both infections no longer exist, and instead antibodies to both viruses emerge. The antibody anti-HBs provides lasting immunity to HBV and HDV. In cases of HDV super infection, the virus replicates quickly due to the already present HBV infection which can be a good source of HBsAg as well as infectivity. The diagnosis of acute HDV infection can be made by the presence of symptoms of acute hepatitis with HBsAg as well as antibody anti-HDV and the absence of IgM and anti-HBc in blood. Another mode of diagnosis is to test for serum HDV RNA by means of PCR (polymerase chain reaction test) as well as for the presence of HDV antigen. The antigen also may be found in the

liver. The existence of co-infection or super infection of HDV is determined by the existence or absence of anti-HBc IgM. Acute co-infection with HBV is diagnosed by the presence of anti-HDV IgM, HDV RNA, or anti-HBc IgM.

Because HDV requires HBV for replication in the liver, consequently HDV can be prevented by eliminating HBV. Hepatitis B vaccination is recommended in areas of the world where HDV is present in endemic proportions. Immunoprophylactic treatment for the prevention of HDV hepatitis in patients who are HBsAg carriers is also recommended.

### **HEPATITIS C VIRUS (HCV)**

HCV is a single stranded 50 nm RNA that belongs to the Flaviviridae family and is within the genus Hepacivirus. Prior to coining the name HCV, the majority of post-transfusion hepatitis was known as non-A, non-B (NANB) hepatitis. HCV exists in at least six types of genomes and many subtypes. Genomes are identified by number 1 through 6, and the subtypes by letters starting with a. These genotypes differ from each other. The most commonly encountered genotype in the U.S. is type "1", accounting for more than 70% of cases, and subtype "a" is the most common. One of the characteristics of this virus is its constant mutation. Replication of the virus is not well known, but it is believed that viral uptake into the liver cells occurs via membrane fusion and receptor-mediated endocytosis.

HCV is transmitted mainly through parenteral injection (50% to 60% of cases) and contaminated blood products (4%). In the past blood transfusions were estimated to cause 90% of cases. The decrease in this number is due to improved procedures for blood donor selection and screening. HCV is considered the most prominent cause of chronic blood-borne infection in the U.S. About 40% of chronic liver disease and an estimated 8,000 to 10,000 deaths that occur annually are due to HCV. About 4 million Americans have had the disease during their life time and about 3 million have chronic infections. The risk of transmission through perinatal exposure is low, while that through sexual contact is rare. However, in certain groups of patients with HCV RNA who have multiple sexual partners, the risk is high. A significant percentage of HIV patients may acquire HCV. Patients receiving dialysis and kidney transplant recipients have a high incidence of HCV infection compared to the general population.

The average duration of the incubation period is about 50 days. During the first one to two weeks of exposure, the virus gains access to hepatocytes; HCV RNA is detectable in the serum, and remains as such throughout the clinical course of the infection. Once in the hepatocytes, the virus releases the genome and starts the replication process. Formation of antibody starts late, sometimes after the start of the clinical manifestations and signs, such as a rise in the aminotransferase. Antibodies to a certain genotype will not confer resistance to another type. When the infection resolves, the HCV RNA becomes undetectable in serum.

The clinical manifestations of HCV are similar to those of other types of hepatitis. They are usually mild or asymptomatic, but characterized by fluctuating levels of aminotransferase, (either low or high). Because HCV is mostly asymptomatic, patients become aware of its existence during routine physical examinations. Fulminant disease is rarely encountered. However, the major complication is the development of chronicity. During the chronic state, HCV RNA is detectable, and the level of aminotransferase becomes variable.

Diagnosis of HCV is made by monitoring hepatic transaminase level, presence of clinical symptoms, and finding of anti-HCV (antibodies) in serum. It should be kept in mind that the antibodies may not be detectable until weeks or months after the appearance of the clinical symptoms. The presence of antibodies does not confer immunity to the patient. It merely indicates that HCV is the cause of the formation of the antibodies.

There are no reliable means of effective vaccines to prevent HCV infection. Prevention measures should focus on high-risk healthy persons such as medical personnel to counsel them on how to reduce the risk for acquiring the disease. HCV positive patients should never be allowed to donate blood, organs, or semen. The use of condoms is recommended; sharing razors and tooth brushes as well as needle exchange in a household with an infected person should be stopped.

### HEPATITIS E VIRUS (HEV)

Hepatitis E virus is a 29 to 32 nm non-enveloped, single stranded virus that belongs to the Caliciviridae family. This viral infection is rare in the U.S., but is encountered in endemic areas such as India, Thailand, Afghanistan, Africa, Mexico, and Central America. The disease is self-limited, has low secondary complication rate and infectivity. Transmission of HEV occurs via the fecal-oral route, especially via drinking contaminated water. Sexual and parenteral injection use is not considered a mode of transmission. However, the disease may be transmitted from infected mother to fetus. Acquiring the infection during pregnancy may result in spontaneous abortion. Carrier state of HEV has not been documented. Neither vaccine nor immunophylactic measures are available for HEV. Preventive measures such as improving sanitary conditions, only drinking bottled water in endemic areas, or not eating uncooked shellfish and unpeeled, exposed fruits and vegetables, should be followed.

### HEPATITIS G VIRUS (HGV)

The term Hepatitis G virus is applied to the Flaviviridae family, and is usually detected in intravenous drug users, homosexual men, hemophiliacs, and in patients who are undergoing hemodialysis or suffering from chronic hepatitis (HBV or HCV). The infection and viremia (presence of a virus in the blood) may stay in the blood for up to 10 years, but it does not appear to cause hepatitis or hepatocellular damage or interfere with treatment of chronic HBV or HCV. Reports indicate that viremia is found in 1 to 4% in healthy persons in Europe and North America. The rate in South America and Africa is much higher.

### CHRONICITY

Acute viral hepatitis may become chronic in nature. It is a major cause of cirrhosis and hepatitis failure worldwide. Chronic liver disease is the tenth leading cause of death among adults in the U.S.

### CHRONIC HEPATITIS B

This disease affects 1.25 million Americans and over 400 million people worldwide. It may occur and is detected as a continuation of acute HBV or as a result of rise in liver enzymes (aminotransferase). During the early stages of the disease, the patient's serum levels will contain HBsAG, HBeAG and HBV DNA and gradually rise in titer. Usually the symptoms appear after a mean incubation period of 25 days at a time when the virus is at its peak. Symptoms of chronic HBV are usually mild, non specific and not accompanied by jaundice. In general, the cause of progressively chronic HBV is variable. Some patients may have HBeAG and HBV DNA in their serum, indicating active viral replications, as well as the appearance of liver damage such as cirrhosis. In others, the progression of the disease is insidious, and signs of liver injury may appear after many years. In a large percentage of patients, clinical improvement takes place, and the disease may enter into remission. Remission may coincide with a decrease in the level or disappearance of HBV DNA and HBeAG and anti HBe. The disappearance of HBeAG and the decrease in the level of the virus are indicative of transition from chronic HBV to chronic state, where the disease is inactive and the symptoms are no longer present. It should be borne in mind that the loss of HBeAG is not indicative of resolution of the infection, as reactivation may occur once HBeAG emerges. In some cases a replicating HBV develops but without having the capability of producing HBeAG. Patients with HBV mutant may develop severe complications

### CHRONIC HEPATITIS C

The HCV is responsible for causing chronic hepatitis C. It has been reported that the **vast majority of patients with HCV develop chronic hepatitis C**. Development of chronicity of hepatitis C occurs upon exposure to the virus and the detection of HCV RNA in serum. The HCV RNA appears during the acute phase and beyond. Only one third of the patients complain of symptoms and signs, including jaundice. Aminotransferase levels are normal in 40% of the patients, even though the HCV RNA remains in serum. In those patients only biopsy can reveal the chronicity of the disease. About

20% of patients with chronic hepatitis C for 20 years or more may develop cirrhosis especially in male patients who drink more than 2 fluid ounces of alcoholic beverages a day. The rate of development of cirrhosis in patients with chronic hepatitis C and whose aminotransferase levels are constantly normal is low. Chronic hepatitis C is diagnosed by the presence of anti-HCV, higher levels of aminotransferase, and liver biopsy. In general the cause of chronic hepatitis C is diverse. Some patients experience severe infections that progress to cirrhosis, while others may not.

### **CHRONIC HEPATITIS D**

As indicated earlier, HDV occurs in the presence of HBV. It is capable of replicating only in the presence of HBsAg. The HDV is superimposed on chronic HBV infection. Infection with HDV may cause severe chronic hepatitis whose outcome is rapid development of cirrhosis and probable death. Diagnosis of chronic HDV is confirmed through detection of antibody (anti-HDV) in the serum. The majority of patients with chronic HDV have HBsAg without serologic indication of the presence of active viral replication.

### **COMPLICATIONS**

#### **Cirrhosis**

Cirrhosis is a degenerative process that occurs as a result of sustained injury to the liver which may be caused by viral infection or other factors such as excessive alcohol consumption and leads to hepatocyte necrosis and liver cellular replacement with nodular fibrous tissue. The outcome of this continuous process is accumulation of fibrous tissue in the liver of the healthy hepatocytes. The liver color becomes yellow-orange and hence the name (Kirros, Greek meaning orange-colored). Normally it is an irreversible process that may lead to death. In fact, it is the eleventh leading cause of death in the U.S. (26,000 annually), and affects about 0.36% of the population. The clinical manifestations vary from one individual to another depending on the nature and severity of the disease. Such manifestations may range from absence of symptoms to liver failure. About 40% of cirrhosis patients are asymptomatic. The symptoms are insidious in nature but may occur abruptly. The most common are fatigue, insomnia, muscular aches, loss of appetite, weight loss, nausea and occasional vomiting. Abdominal discomfort may be encountered due to hepatomegaly. Other symptoms include amenorrhea, impotence, loss of libido and sterility. The main signs of cirrhosis which are present in the majority of patients are hepatomegaly, liver firmness, appearance of the spider nevi and erythema on the palm. At the early stages mild jaundice may occur, but becomes more prominent in advanced cases. In the late stages of the disease encephalopathy accompanied by tremor, dysarthria, delusion, drowsiness and finally coma may occur. When fever occurs, it is normally due to peritonitis or other infections.

#### **JAUNDICE (ICTERUS)**

Jaundice, which is a state of discoloration of the skin, mucus membrane and the white portion of the eyes, is carried by accumulation of predominantly unconjugated or conjugated bilirubin in the blood. Bilirubin, which is a reddish pigment, is found normally in the serum (0.2-1.2mg/dl), and it is a product of heme metabolism. Excessive amounts may be due to hepatic or non hepatic causes. Jaundice becomes recognizable when its concentration in serum is 3.0 mg/dl or more. Conjugated hyperbilirubinemia may occur due to hepatocellulose disease as in hepatitis. Malaise, anorexia, low grade fever and pain in the upper quadrant of the abdomen, dark urine, jaundice, and menstrual irregularities in particular amenorrhea, enlarged liver, feeling of tenderness and vascular spiders are frequent.

### **SUMMARY**

Viral hepatitis is a public health problem worldwide that is a source of morbidity and fatality. Even though the clinical manifestations are similar, viral hepatitis is caused by hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), Hepatitis D virus (HDV), and hepatitis E virus (HEV). The clinical manifestation of these types of viral infections include, fatigue, malaise, low grade fever, nausea, discomfort in the upper quadrant of the abdomen, jaundice, anorexia, and liver tenderness. Biochemically the infections are characterized by an increase in aminotransferase levels. The infec-

tion is accompanied by hepatocellular necrosis and inflammation and the development of antibodies to viral antigens. Vaccination is effective in reducing the risk of incidence of these viruses. Behavioral modification and changes in lifestyle are also helpful in minimizing the incidence of the infection. Progression of acute viral infection to a chronic one may occur.

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**Which do you like & prefer? Let us know.**

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**LESSON EVALUATION**

1. Does the program meet the learning objectives?  
 Define hepatitis Yes No  
 Differentiate between between the various types of hepatitis Yes No  
 Describe tests used to diagnose the disease types Yes No  
 Discuss modes of transmission Yes No  
 Discuss pre-exposure & post-exposure prophylactic methods in prevention Yes No
2. Was the program independent & non-commercial Yes No
- |  |      |   |         |   |           |
|--|------|---|---------|---|-----------|
|  | Poor |   | Average |   | Excellent |
|  | 1    | 2 | 3       | 4 | 5         |
|  |      |   |         |   | 6         |
|  |      |   |         |   | 7         |
3. Relevance of topic
4. What did you like most about this lesson? \_\_\_\_\_
5. What did you like least about this lesson? \_\_\_\_\_

**Please Select the Most Correct Answer**

- |   |   |
|---|---|
| <p>1. Which statement is FALSE about viral hepatitis?<br/>                 A. Characterized by increased liver enzymes<br/>                 B. Hepatocellular necrosis rarely occurs<br/>                 C. It always leads to chronicity<br/>                 D. Not contagious</p> <p>2. Which is FALSE about HAV?<br/>                 A. Children usually asymptomatic<br/>                 B. Average incubation period is 30 days<br/>                 C. Patients will excrete virus in feces for entire duration of their infection<br/>                 D. Transmitted via fecal-oral route</p> <p>3. HAV vaccine:<br/>                 A. Consists of active virus<br/>                 B. Prepared from inactivated hepatitis A antigen<br/>                 C. Causes serious side effects in children &amp; young adults<br/>                 D. Should be used for post exposure</p> <p>4. The infectivity of HDV depends on:<br/>                 A. HVC antibiotics<br/>                 B. The presence of HBV<br/>                 C. A previous infection with HAV<br/>                 D. Mode of transmission</p> <p>5. Persons who had sexual contact with an individual with HBV:<br/>                 A. Should be given vaccine after exposure<br/>                 B. Should have blood transfusion<br/>                 C. Should use condom after the 1st exposure<br/>                 D. Should be given Hepatitis B immune globulin</p> | <p>6. What is false about HBV?<br/>                 A. Sexual transmission of HBV occurs in 30 to 60% of new cases<br/>                 B. Diagnosis of HBV is confirmed by the presence of HBeAg<br/>                 C. Presence of HBV DNA indicates no viral replication &amp; absence of infectivity<br/>                 D. HBeAg is found in serum only in the presence of HBsAg</p> <p>7. HDV infection:<br/>                 A. Causes a triphasic increase in transaminase level<br/>                 B. Causes a diminished transaminase level after the incubation period<br/>                 C. Is common in the U.S.<br/>                 D. Causes biphasic increase in the transaminase levels</p> <p>8. A correct statement about HCV:<br/>                 A. Belongs to the flaviviruses<br/>                 B. Exists in more types of genomes<br/>                 C. Most common type in the U.S. is type 3<br/>                 D. Risk of perinatal transmission is high</p> <p>9. What is false about cirrhosis?<br/>                 A. Excessive alcohol contributes<br/>                 B. The disorder is reversible<br/>                 C. Hepatomegaly is a sign<br/>                 D. About 40% of patients are asymptomatic</p> <p>10. Jaundice:<br/>                 A. Manifests with bilirubin of 0.3mg/dl<br/>                 B. Is contagious<br/>                 C. Is a sign of hepatic chronicity<br/>                 D. Manifests with bilirubin of 3.0mg/dl</p> |
|---|---|

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