

# **Liver Disease**

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Recently I have been getting a flurry of patients that have some serious liver complications. This week's literature review will be the dental management of the patients with liver disease specifically hepatitis.

Liver dysfunction may be attributed to several causes; including lifestyle habits, acquired infections and conditions. The reason why this is important to us as dentists is because the liver plays a vital role in metabolic functions, including secretion of bile needed for fat absorption, conversion of sugar to glycogen and excretion of bilirubin. Also, and more importantly to us is the liver's role in drug metabolism and synthesis of coagulation factors.

## **Hepatitis**

Hepatitis is inflammation of the liver that may result from infectious sources or other causes. Examples of hepatitis with infectious causes are viral hepatitis, infectious mononucleosis, secondary syphilis and tuberculosis. Noninfectious sources are toxic substances like certain drugs and alcohol abuse.

### **Viral Hepatitis:**

#### **Etiology**

There are five different types of infectious hepatitis (A, B, C, D and E). Surprisingly they have little in common other than they target the same organ and some epidemiologic characteristics.

#### **Epidemiology**

More than 325 million people around the world have Hepatitis B or C and majority of those do not have access to life saving medications. In 2015, viral hepatitis caused 1.34 million deaths, a number comparable to deaths caused by tuberculosis and HIV. But while mortality from tuberculosis and HIV has been declining, deaths from hepatitis are on the increase.

#### **Hepatitis A**

This virus is an RNA virus that replicates in the liver, secreted in the bile and shed in stool. Transmission of HAV occurs almost exclusively through fecal contamination of food and water. Hep A is a common disease with serologic evidence of infection noted in approximately 40% of the urban populations in the United States. Its incubation period ranges from 15 to 50 days and averages 25 days. Hep A tends to be of mild severity and self-limiting; it lasts a couple of weeks and often goes undiagnosed. Hep A may be

diagnosed by signs and symptoms such as fatigue, fever, lymphadenopathy, gastrointestinal upset and nausea, night sweats and possibly jaundice. No carrier state is known and recovery usually conveys immunity against reinfection. HAV infection may be effectively prevented by administration of the HAV immune globulin prophylactically or within two weeks.

### **Hepatitis B**

This virus is a DNA virus that replicates in hepatocytes and to a lesser extent in stem cells in the pancreas, bone marrow and spleen. This virus is transmitted by percutaneous and permucosal exposure; with the most frequent form of transmission being sexual transmission. Fecal and airborne spread is not possible with HBV. The lifetime risk of exposure to HBV is low for the general population; however, groups such as dentists and healthcare providers have a higher risk of transmission. HBV tends to cause greater morbidity and mortality especially for the very young and older populations.

### **Hepatitis C**

Is a RNA virus and is similar to HBV in terms of behavior and characteristic? The incubation period of HCV ranges from 2 weeks to 6 months with a median of 50 days. Most of the cases are from transmission by blood and blood products. Those at greatest risk for this disease are injection drug users.

### **Hepatitis D**

HDV is a negative-strand RNA virus that requires HBsAg for its viral envelope and transmission. However, once HDV are within the infected cells, HDV can replicate without the need of the HBV. Hep D only occurs as a coinfection with acute hep B or as a superinfection in carriers of HBV. HDV is reported to only occur primarily in drug addicts and persons with hemophilia.

### **Hepatitis E**

HEV is an RNA virus that is clinically similar to HAV. Transmission is similar via fecal/oral contamination however the incubation period ranges from 15-60 days.

## **Pathophysiology and Complications**

Hepatitis viruses replicate in hepatocytes ultimately damaging the host cell. Commonly, acute viral hepatitis is characterized by ballooning degeneration and necrosis of the liver cells. The entire liver lobule becomes inflamed and consists of lymphocytes and mononuclear phagocytes.

Jaundice is associated with hepatitis in approximately 10% of hep A, 60 -70% of hep C, and 70 to 90% of hep B. This is caused by the accumulation of bilirubin in the plasma, epithelium and urine. Most cases of viral hepatitis especially types A and E resolve with no complications. HBV, HCV and HDV may persist and can replicate in the liver when the virus is not completely cleared from the organ. The consequences of hepatitis include recovery, persistent infection, dual infection, cirrhosis, hepatocellular carcinoma and death.

Chronic hepatitis is associated with liver abnormalities but is often asymptomatic for 10 to 30 years. Nonspecific symptoms of chronic hepatitis hep C (loss of weight, easy fatigue, sleep disorder, difficulty in concentrating, right upper quadrant pain and liver tenderness) may not appear until hepatic fibrosis or hepatocellular carcinoma.

Extra hepatic immunological disorders associated with chronic HCV result from the production of autoantibodies and can result in two oral manifestations which include lichen planus and Sjogren's- like syndromes.

Signs of advanced disease include bleeding esophageal varices, ascites, spider angioma and dark urine

HDV infection often results in severe acute hepatitis or rapidly progressive chronic liver disease.

## **Treatment**

Treatment therapy is palliative and supportive. bed rest and fluids may be prescribed especially during the acute phase. Alcohol and drugs metabolized in the liver are not to be ingested. Viral antigens and ALT levels should also be monitored for 6 months so it can be determined if the hepatitis is resolving.

Chronic hepatitis rarely resolves and standard therapy for these patients is interferon alfa- 2b given 3x times weekly for 6 months to a year.

## **Dental Management**

Identification of potential or actual carriers of HBV, HCV and HDV is problematic because in most instances, carriers cannot be identified by history. Therefore, all patients with a history of viral hepatitis must be managed as though they are potentially infectious. Current recommendations for infection control practices in dentistry are the standard of care for preventing cross-infection. In addition, these organizations recommend that all dental health care providers should receive vaccination against HBV.

### **Patients with Active Hepatitis**

No dental treatment should be rendered except for emergency. If surgery is necessary at that time than preoperative prothrombin time and bleeding time should be obtained.

### **Patients with a History of Hepatitis**

For those that have a positive history of hepatitis additional historical information is needed to determine the type of virus. No special precautions are necessary except for our daily universal precautions that are applied for all our patients.

### **Drug Administration**

No special drug considerations are required for a patient who has completely recovered from viral hepatitis.

If a patient has chronic active hepatitis or is carrier of the HBsAg or HCV and has impaired liver function, then dose modification is necessary. Dose modification should be performed if one of more of the following are present:

- Aminotransferase levels elevated to greater than 4x normal values
- Serum bilirubin elevated to above 2 mg/dl
- Serum albumin levels lower than 35 mg/l
- Signs of ascites and encephalopathy and prolonged bleeding time.
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The proper reduction for many of the common drugs that are prescribed within dentistry can be properly looked up at the time of prescription writing. Additionally, the safe amount of lidocaine to give a patient that has chronic hepatitis with impaired liver function is approximately 120 mg or three cartridges.

## **Oral manifestations and Complications**

The most common complications associated with patients with hepatitis is abnormal bleeding. This results from abnormal synthesis of blood clotting factors, abnormal polymerization of fibrin, inadequate fibrin stabilization, excessive fibrinolysis or thrombocytopenia associated with splenomegaly. Before any major surgery the platelet count should be obtained and should confirm the INR is less than 3.5. If the INR is greater than 3.5 the risk for severe postoperative bleeding exists. In these cases, the surgical procedure should be postponed. If surgery is necessary, then a shot of vitamin K usually corrects the problem and the surgery can go ahead as planned. Also, platelet function analysis may indicate whether platelet replacement may be required before surgery.

## **Take Home Message**

Hepatitis is a very common disease that doesn't require really any dental modifications until the disease has really progressed and has become advanced. With proper history taking and ordering the correct blood work, no complications should occur when treating this patient population.

## **References**

Little, J. W. (2013). Dental management of the medically compromised patient. St. Louis, Mo: Elsevier/Mosby.