

# Hepatitis B and C infection: Clinical implications in dental practice

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## ABSTRACT

Health-care workers have an occupational risk of infection with hepatitis B virus (HBV) and hepatitis C virus (HCV). Since dental healthcare professionals have numerous patients and are exposed to blood, they are likely to have the maximum risk. HBC and HCV are transmitted by skin prick with infected, contaminated needles and syringes or through accidental inoculation of minute quantities of blood during surgical and dental procedures. HBV can be prevented by strict adherence to standard microbiological practices and techniques, and routine use of appropriate barrier precautions to prevent skin and mucous membrane exposure when handling blood and other body fluids of all patients in healthcare settings and pre-exposure vaccines. Despite many publications about programs and strategies to prevent transmission, HBV and HCV infections remain a major public health issue. Oral clinical manifestations can be observed, such as bleeding disorders, jaundice, fetor hepaticus, and xerostomia. The most frequent extrahepatic manifestations mostly affect the oral region in the form of lichen planus, xerostomia, Sjögren's syndrome, and sialadenitis. The present paper highlights some of the important oral manifestations related to hepatitis B and C infection and various post-exposure protocols that can be undertaken to minimize the risk of infection.

## Key words

Dentistry, hepatitis, management, transmission

## INTRODUCTION

The liver has a broad range of functions in maintaining homeostasis and health. It synthesizes the most essential serum proteins (albumin, transporter proteins, blood coagulation factors V, VII, IX, and X, prothrombin, and fibrinogen,<sup>[1]</sup> as well as many hormone and growth factors), produces bile and its transporters (bile acids, cholesterol, lecithin, phospholipids), intervenes in the regulation of nutrients (glucose, glycogen, lipids, cholesterol, amino acids), and metabolizes and conjugates lipophilic compounds (bilirubin, cations, drugs) to facilitate their excretion in bile or urine. Liver dysfunction alters the metabolism of carbohydrates, lipids, proteins, drugs, bilirubin, and hormones.<sup>[2]</sup> Accordingly, liver disease is characterized by a series of aspects that must be taken into account in the context of medical and dental care.<sup>[3]</sup>

Hepatitis B virus (HBV) is a DNA virus and one of many unrelated viruses that cause viral hepatitis. The disease was originally known as “serum hepatitis”.<sup>[4]</sup> Hepatitis C is a hepatotropic viral infection caused by hepatitis C virus (HCV), which is a major cause of acute hepatitis and chronic liver disease. It is characterized by inflammation of the liver and in many cases permanent damage to liver tissue. The most common types of hepatitis are hepatitis A, B, C, D, E, and G. Hepatitis B and C can lead to permanent liver damage and in many cases death.<sup>[4]</sup> There are more than 2 billion people worldwide having evidence of recent or past HBV infection and 350 million are chronic carriers. In the Southeast Asia region, there are estimated 80 million HBV carriers (about 6% of the total population).<sup>[5]</sup> India has the intermediate endemicity of hepatitis B, with hepatitis B surface antigen prevalence between 2% and 10% among the population studied. The number of carriers in India has been estimated to be over 40 million.<sup>[6]</sup>

HBV and HCV infections are serious public health problems that can have consequences in terms of psychological and occupational diseases. HBV and HCV are common causes of occupational diseases transmitted from patients to health care workers (HCWs) and *vice versa*, and also to HCWs' families.<sup>[7]</sup> It has been estimated that 14.4% and 1.4% of hospital workers are infected with HBV and HCV, respectively.<sup>[8]</sup> Physicians, dentists, nurses, laboratory

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staff, and dialysis center personnel are at high risk of acquiring infection. HCV prevalence varies widely among countries, with the highest being in several African and eastern Mediterranean countries.<sup>[9]</sup> The frequency of exposure to HBV was the highest among dental healthcare workers according to a study conducted in Japan.<sup>[10]</sup> In another study, nurses were most commonly exposed to infection (41%), followed by physicians (31%) among healthcare workers.<sup>[11]</sup> Prevention of hepatitis B and C transmission through the medical care setting is an important public health issue.<sup>[12]</sup> Even after the introduction of many programs and strategies, hepatitis infection continues to remain a health problem in dental settings. Therefore, the present study considers aspects such as common oral manifestations of hepatitis B and C infection for the clinician to be able to appropriately diagnose, prevent, and manage the transmission and progression of this fatal disease.

### Search strategy

A meticulous review of the literature was carried out, which engaged most of the articles published in peer-reviewed journals relating to the subject of hepatitis B and C in dentistry. The review itself began with the search of relevant key words linked with hepatitis B and C infection like hepatitis transmission, oral manifestations of hepatitis, and dental management of hepatitis B and hepatitis C exposure in various search engines including PubMed. Reports published only in the English language were included in the review. The spot light of the present review is on the prevalence of hepatitis B and C infection and its various oral manifestations, implications, and clinical management. The search also targeted infection control measures and preventive strategies toward transmission and post-exposure prophylaxis of HBV and HCV virus infection.

### The virus and its transmission

HBV is a DNA virus belonging to the family Hepadnaviridae. It is a complex 42 nm double-shelled particle. The outer surface or envelop of the virus contains hepatitis B surface antigen (HbsAg). It encloses an inner icosahedral 27 nm nucleocapsid (core), which contains hepatitis B

core antigen (Hbc Ag). Inside the core, there is a circular double-stranded DNA and a DNA polymerase.<sup>[13]</sup>

Hepatitis C virus is a RNA virus belonging to family Flaviviridae. Genetically distinct viral groups have evolved with 9 different genotypes of hepatitis C and 40 different subgroups.<sup>[14]</sup> The sources of contagion include blood transfusion, percutaneous exposure through contaminated instruments, and occupational exposure to blood.<sup>[7]</sup> The individuals at the greatest risk are hemophiliacs, patients on dialysis, and parenteral drug abusers. Other transmission routes are sexual contact and the perinatal and idiopathic routes. The main characteristics of HBV and HCV are summarized in Table 1.<sup>[15]</sup>

### Clinical presentation of the disease

Around 30-50% of adults and children develop clinical illness typical of hepatitis B after initial exposure to HBV. The incubation period for hepatitis B usually ranges from 60 to 150 days.<sup>[15]</sup> Early symptoms that occur before jaundice include constitutional symptoms like malaise, fatigue, and anorexia for a period of 1-2 weeks. In the acute phase, the typical clinical signs and symptoms include nausea, vomiting, abdominal pain, and jaundice. In some cases, skin rashes, joint pain, and arthritis may occur. Acute hepatitis B progresses to chronic HBV infection in 30-90% of people infected as infants or young children and during adolescence or adulthood around <5% of people infected may develop chronic infection. Chronic infection with HBV results in chronic liver disease, including liver cirrhosis and hepatocellular carcinoma.<sup>[15]</sup>

In most of the cases, the onset of hepatitis C infection is unrecognized because the clinical symptoms are often mild and clinically not apparent. In symptomatic cases, clinical features include malaise, nausea, vomiting, abdominal discomfort, pale stools, dark urine, and jaundice. Between 70% and 80% develop chronic infections. Chronic infection is defined as infection persisting for more than 6 months with some evidence of hepatitis. The term chronic relates to the duration of infection and not to the severity of the disease. Chronic

**Table 1: Characteristics of the hepatitis B and C virus**

Feature	Hepatitis B virus	Hepatitis C virus
Genome	DNA	RNA
Nomenclature	Hepadnaviridae	Flaviviridae
Mode of transmission	Parenteral, sexual, perinatal, oral fluids	Parenteral, sexual, may get transmitted by oral fluids
Antigens in blood	HbsAg, HbeAg	HCV
Antibodies in blood	Anti-HBs, Anti-Hbe, Anti-HBc	Anti-HCV
Profile	Can be mild, severe, acute or chronic. Less than 5% of adult HBV infections become chronic	Hepatitis C is likely to become a chronic condition in 70 to 80% of infected people, with 10% developing severe liver disease
Passive immunization	Hepatitis B immune globulin	Not available
Active immunization	Recombivax, Engerix and Twinrix	Not available

DNA - De-oxyribonucleic acid; RNA - Ribonucleic acid; HCV - Hepatitis C virus; HBsAg - Hepatitis B surface antigen; HBeAg - Hepatitis B e antigen; HBV - Hepatitis B virus

hepatitis C infection leads to a wide spectrum of liver diseases, ranging from mild hepatitis to cirrhosis, liver cancer and finally to liver failure in 10% of the infected individuals.<sup>[15,16]</sup>

### Hepatitis B and C in oral cavity

HBV infection is the most important infectious occupational hazard in the dental profession. Vectors of infection with HBV in dental practice include blood, saliva, and nasopharyngeal secretions.<sup>[16]</sup> Intraorally, the greatest concentration of hepatitis B infection is the gingival sulcus.<sup>[17]</sup> In addition, periodontal disease, severity of bleeding, and bad oral hygiene were associated with the risk of HBV. In Egypt, patients with periodontal disease showed a higher detectability rate of HBsAg, anti-HBc, anti-HCV, or both anti-HCV and/or anti-HBc in whole unstimulated saliva than in the controls.<sup>[18]</sup> HCV-RNA has been detected in saliva and in salivary glands from patients with sialadenitis.<sup>[19]</sup> Most HCV patients (77%) had higher HCV RNA levels in their gingival sulcus than in their saliva.<sup>[20]</sup> Leao *et al.* found HCV-RNA in tooth brushes used by hepatitis C patients.<sup>[21]</sup>

### Oral manifestations of hepatitis B and C infection

Manifestations in the oral cavity include lichen planus, Sjögren's syndrome, and sialadenitis, some forms of oral cancers may also be seen.<sup>[16]</sup> Furthermore, cirrhotic patients may have thrombocytopenia due to hypersplenism or treatment with interferon.<sup>[22]</sup> In patients with liver disease, the resultant impaired hemostasis can be manifested in the mouth as petechiae or excessive gingival bleeding with minor trauma. This is especially suggestive if it occurs in the absence of inflammation. Therefore, special care must be taken during any type of surgery, oral or otherwise; severe hemorrhage can ensue as a result of the paucity of clotting factors. An interesting correlation exists between the increased prevalence of diabetes in patients with chronic liver disease due to the severity of liver disease or to the treatment with interferon. HCV may act as an independent diabetogenic factor.<sup>[22]</sup> For the dentist this association has important implications because diabetes is associated with significant changes in the oral cavity such as increased frequency of periodontal disease, stomatitis, candidiasis, cheilitis, oral leukoplakia, and dental caries.

### Oral lichen planus and hepatitis

Lichen planus is a mucocutaneous disease of uncertain cause that affects the oral mucosa. It is well documented that the disease represents a cell-mediated immune response.<sup>[23]</sup> Its prevalence in the general population ranges from 0.1% to 2.2%,<sup>[24]</sup> and the diagnostic criteria for OLP are based on clinical and histopathologic features of the condition.<sup>[25]</sup> It is classified as reticular, plaque, atrophic, erosive, or bullous according to the clinical presentation.<sup>[25]</sup> Emotional stress, immunological disturbances, neurological dysfunctions, and viruses are

possible etiological factors.<sup>[26,27]</sup> The prevalence of OLP and pitted keratolysis in the HBs Ag carrier group has been found to be significantly high.<sup>[28]</sup> HBs Ag positivity may induce or cause proneness to OLP and pitted keratolysis with some mechanism that needs to be elucidated.<sup>[28]</sup> Many studies and reports suggest a positive correlation between the prevalence of hepatitis C and OLP, but some of them still remain controversial.<sup>[29-32]</sup> A recent case report suggests a correlation between the drugs and interferons used for the treatment of hepatitis C with the extrahepatic manifestation such as OLP.<sup>[33]</sup> Some authors suggest that patients with HCV should undergo periodic oral examinations and patients with OLP should undergo screening tests for HCV infection.<sup>[34]</sup> The epidemiological relationship between OLP and hepatitis C has been reported<sup>[35,36]</sup> notably in the erosive type<sup>[37-39]</sup> and asymmetric lesions on the buccal mucosa. It has been seen that the prevalence of OLP associated with hepatitis C presents geographical variations.<sup>[36-38,40,41]</sup> Genetic variations among the populations seem to be the main factor accounting for these differences.<sup>[42]</sup>

It is believed that HCV acts locally, altering the function of the epithelial cells, or that the immune response of the host to HCV is responsible for the development of OLP. Thus, host factors rather than geographic factors may be more important in the pathogenesis of HCV-related OLP.<sup>[43]</sup>

### Salivary gland disorders and hepatitis

The main salivary gland disorders associated with HCV infection are xerostomia, Sjögren's syndrome, and sialadenitis. Xerostomia increases patient vulnerability to caries and oral soft tissue disorders,<sup>[44]</sup> which, in combination with deficient hygiene, in turn facilitate the development of candidiasis. It has not yet been demonstrated whether HCV infection causes disease similar to primary Sjögren's syndrome or whether it is directly responsible for the development of Sjögren's syndrome in certain types of patients. However, notoriously some subjects can present a triple association of HCV infection, Sjögren's syndrome, and sialadenitis or salivary gland lymphoma.<sup>[45]</sup> Although bacteria are the main cause of sialadenitis, viruses such as HCV have been implicated as causes of sialadenitis associated with xerostomia.<sup>[45]</sup> The role of saliva includes cleaning, lubrication, chemical protection, and cell-mediated and antibody mediated immunity. Decrease in salivary flow may lead to dry mouth (particularly at night), halitosis, dental decay, and difficulty in talking, eating, and swallowing.<sup>[45]</sup> To make the patient comfortable, one must take measures to effectively manage such cases. The patient should be asked to take frequent sips of water or sugar-free candies, which act as sialogogues and help increase salivary flow. Prophylactic care including application of fluorides to prevent dental decay must be taken. Patients may be advised to avoid hot and spicy food and use a non-foaming toothpaste to increase oral

comfort. Dietary counseling must be provided to control the frequency and amount of carbohydrate.

### Management of patients with hepatitis B and C infection in dental office

The most important and frequent problems associated with hepatitis B and C in dental settings include the risk of viral contagion on the part of the dental professionals and rest of patients (cross-infection), the risk of bleeding in patients with serious liver disease, and alterations in the metabolism of certain drug substances that increases the risk of toxicity.<sup>[1]</sup> It has been found that HBV and HCV exist on various surfaces in the dental operatory even many days after treating patients positive with hepatitis B and C.<sup>[46]</sup> HCV can remain stable at room temperature for over 5 days.<sup>[47]</sup> Therefore, standard precautions, i.e., the use of barrier methods, with correct sterilization and disinfection measures, must be followed.<sup>[1]</sup> The conventional sterilization techniques usually eliminate specific proteins and nucleic acids (HBV DNA and HCV RNA) from dental instruments previously infected with HBV and HCV.

In case there is an accidental exposure, follow these steps:

1. Carefully wash the wound without rubbing, as this may inoculate the virus into deeper tissues, for several minutes with soap and water, or using a disinfectant of established efficacy against the virus (iodine solutions or chlorine formulations). Some authors suggest that pressure should be applied beneath the level of the wound to induce bleeding and thus help evacuate any possible infectious material.<sup>[48]</sup> However, no such fact has been strongly validated. The rationale behind these measures is to reduce the number of viral units to below the threshold count required to cause infection (the infectious dose). In this sense, dilution with water may lower the viral count to below this threshold.<sup>[49]</sup>
2. A complete detailed medical and clinical history of the patient must be recorded to rule out possible risks.<sup>[50]</sup>

### Diagnosis of the disease

The disease can be diagnosed by quantifying the levels of HBV DNA, HBs Ag, and the antigen/antibody ratio by means of immuno-enzymatic assays. Different enzyme-linked immunosorbent assay and recombinant immunoblot assay techniques have been developed for the diagnosis, though the diagnostic gold standard remains detection of the viral genome using real time-polymerase chain reaction (RT-PCR) technology. When the disease has developed and the infection is well established, a liver biopsy must be performed to establish the amount of fibrosis and the severity of the inflammation. These findings help the hepatologist determine the treatment needs of the patients and help establish wise treatment decisions.<sup>[16]</sup>

### Management of exposures to hepatitis B virus

Any blood or body fluid exposure to an unvaccinated person should lead to the initiation of the hepatitis B vaccine series (Recombivax HB® 10 mcg or Energix-B® 20 mcg IM at 0, 1, and 6 months). When hepatitis B Immune Globulin (HBIG) is indicated, it should be administered as soon as possible after the exposure (preferable within 24 h, but it is recommended up to 1 week following an occupational exposure). Hepatitis B vaccine can be administered simultaneously with HBIG but at a separate site. Test for anti-HBs must be performed 1-2 months after the last dose of vaccine.<sup>[51-53]</sup> Anti-HBs cannot be ascertained if HBIG has been administered within the previous 6 weeks [Table 2].

### Management of exposures to hepatitis C virus

On exposure to HCV, test for anti-HCV must be carried out for the source. Baseline testing for anti-HCV and Alanine aminotransferase activity (ALT) should be carried out for the exposed person. The most recent assays are based on the use of RT-PCR). They can detect minute amounts of HCV RNA (down to 10 international units (IU)/ml) and accurately quantify HCV RNA levels up to approximately 107 IU/ml.<sup>[54]</sup> Follow-up testing for anti-HCV and ALT activity and HCV RNA by PCR at 4-6 weeks must be carried out for

**Table 2: Recommendations for post exposure prophylaxis for hepatitis B**

Vaccination status of exposed person	HBsAg status of the source		
	HBsAg positive	HBsAg negative	HBsAg status unknown
Unvaccinated	HBIG x1; start HBV vaccine series	Start HBV vac series	Start HBV vac series
Vaccinated: Responder <sup>a</sup>	No treatment	No treatment	No treatment
Vaccinated: Non-responder <sup>a</sup>	HBIG and start HBV vac series <sup>b</sup> or HBIG x 2 <sup>c</sup>	No treatment	If known high risk for HBV, treat as if source is HBsAg positive
Vaccinated: Response status unknown	Test for anti-HBs If responder: No treatment If non-responder: HBIG x 1; vaccine booster <sup>c</sup>	No treatment	Test for anti-HBs If responder: No treatment If non-responder: Vaccine booster; re-check anti-HBs in 1-2 months

<sup>a</sup>Responder=anti-HBs>10 m IU/ml; non-responder=anti-HBs<10 m IU/ml. Do not repeat anti-HBs if previous results are available. <sup>b</sup>HBIG can be administered simultaneously with HBV vaccine at different sites. HBIG dose=0.06 mg mL/kg IM. <sup>c</sup>If non-responder has received 2 full series of HBV vaccine; then administer a second dose of HBIG one month after initial dose. HBsAg - Hepatitis B surface antigen; HBIG - Hepatitis B immune globulin; HBV - Hepatitis B virus



early detection. Results reported positive by enzyme immunoassay with supplement test (e.g., recombinant immunoassay or HCV RNA by PCR) should be confirmed<sup>[51]</sup> [Table 3].

Before treating a patient infected with hepatitis B or C, a compilation of a detailed clinical history is essential before dental treatment to identify patients posing possible risks,<sup>[50]</sup> together with a thorough oral examination. Consultation with the patient's physician or specialist is advisable to establish a safe and adequate treatment plan adapted to the medical condition of the patient,<sup>[52]</sup> considering the degree of liver functional impairment involved.<sup>[1]</sup> Examination of the oral cavity should assess any signs alerting to the existence of systemic disease. The patient should receive an explanation of the risks associated with treatment, and informed consent is to be obtained. In subjects with chronic hepatitis, it is important to determine the possible existence of associated disorders (autoimmune processes, diabetes, etc.) to prevent their direct complications and problems derived from specific medication use (corticosteroids and/or immune suppressors). Liver disease may often be associated with a decrease in plasma coagulation factor concentrations.<sup>[2,3]</sup> In case any invasive procedure is to be performed in these patients, prior coagulation and hemostasis tests are required, which include complete blood count, bleeding time, prothrombin time/international normalized ratio (INR), thrombin time, thromboplastin time, and liver biochemistry tests<sup>[1,55]</sup> and the hematologist and hepatologist must also be consulted.

Usually in an unfavorable state elective treatment is postponed; however, incase treatment is carried out, the dentist must stock up on local hemostatic agents such as oxidized and regenerated cellulose, as well as antifibrinolytic agents (tranexamic acid), fresh plasma, platelets, and vitamin K.<sup>[1,55]</sup> In some cases antibiotic prophylaxis is suggested, since liver dysfunction is associated with diminished immune competence.<sup>[2]</sup> Liver disease may result in alterations in the metabolism of certain drugs. The physician treating the patient therefore should be consulted to establish which drugs are used, their doses, and their possible interactions.<sup>[53,5]</sup> The administration of certain analgesics, antibiotics, and local anesthetics is generally well tolerated by patients with mild to moderate liver dysfunction, though modifications

might be necessary in individuals with advanced-stage liver disease. In this context, drugs metabolized in the liver may have to be used with caution or their doses reduced and certain substances such as erythromycin, metronidazole, or tetracyclines must be avoided entirely.<sup>[3]</sup> Most of the antibiotics prescribed for oral and maxillofacial infection scan be used in patients with chronic liver disease, and in general the beta-lactams can be administered. Aminoglycosides can increase the risk of liver toxicity in patients with liver disease, and hence should be avoided.<sup>[56]</sup> Non-steroidal anti-inflammatory drugs should be used with caution or avoided, due to the risk of gastrointestinal bleeding and gastritis usually associated with liver disease. Prophylaxis can be provided in the form of antacids or histamine receptor antagonists.<sup>[3,2]</sup> Local anesthetics are generally safe, provided the total dosage does not exceed 7 mg/kg, combined with epinephrine.

## CONCLUSION

Hepatitis is a disease of concern and the management of a patient infected with it can be difficult and challenging. HBV and HCV can be transmitted by skin prick with infected, contaminated needles and syringes or through accidental inoculation of minute quantities of blood during dental procedures. Therefore, proper preventive measures must be adopted with strict protocol to prevent the transmission of the virus from the dental practitioner to the patient and from the patient to the dentist. Hepatitis can also have severe effects on the clotting ability of blood and other significant correlations and extrahepatic oral manifestations. Therefore, preventive oral hygiene measures must be implemented to reduce the need for dental surgical treatments.

In many cases, discrimination and stigma, or fear and past experience can prevent people with hepatitis B or C from accessing dental and other healthcare services. Therefore, one must make an endeavor to ensure a welcoming and nonjudgmental approach to treating all clients, to ensure the provision of effective healthcare and follow-up. Prevention is an important aspect in controlling the spread of this viral infection as an epidemic. Knowing facts, having proper awareness, and proper behavior and attitude toward clinical aspects of the infection and toward the patients are critical to prevent the spread of these infections.

**Table 3: Recommendations for post exposure prophylaxis for hepatitis C**

Baseline (at time of exposure)	Obtain anti-HCV and ALT
4 months post-exposure	If anti-HCV is positive, then obtain HCV RNA. If HCV RNA is positive, then evaluate for treatment
6 months post-exposure	If 4-month anti-HCV is negative, then obtain an anti-HCV and ALT. If anti-HCV is negative, then STOP follow-up. If anti-HCV is positive, then obtain HCV RNA. If HCV RNA is positive, then evaluate for treatment

HCV - Hepatitis C virus; ALT - Alanine aminotransferase; RNAw - Ribonucleic acid

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