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CATCH ME IF YOU CAN! SIMPLE STEPS SAVES LIVES - DENTAL PERSPECTIVE

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Abstract
Hepatitis – B virus is a double-stranded deoxy ribo nucleic acid virus belonging to the hepadna viridae family. HBV is the most common blood-borne pathogen affecting dental and medical professionals and also placing healthcare workers at high occupational risk. The most common inflammatory infection of the liver is caused by Hepatitis- B virus. There are many possible ways by which the disease is transmitted such as unprotected sexual contact, re-usage of contaminated needles, and vertical transmission. Percutaneous exposure (needle stick injuries) and contact with the saliva or blood of the infected patients is the most common mode of transmission during dental practice. It has been reported in certain studies that saliva and gingival crevicular fluid play a major role as there is a possibility of HBV transmission when dentists are exposed to it and thus oral health professionals are more vulnerable to hepatitis infection. This review is to discuss the transmission of the Hepatitis- B virus and the prevention strategy among dental practitioners.
keywords: Hepatitis- B, Vaccination, Sharps safety, Antiviral therapy

INTRODUCTION:

Around 350 millions of people are chronic carriers of the HBV infection, which has infected approximately two billion people worldwide[1],[2]. HIV is less contagious than HBV infections[1],[3]. Hepatitis – B infection can become persistent and it can lead to cirrhosis of the liver and at times to liver cancer. It is most commonly acquired through blood products and contaminated needles and sometimes has diffuse infection patterns[4]. Among all the healthcare workers, dentists are actual carriers and are placed in high-risk group categories.

Hepatitis is referred to the inflammation of the liver tissue. Hepatitis – B infection is also known as a 'silent epidemic' as many people do not have symptoms when they are infected initially or when people have the infection for a chronic period. Thus, these people can unknowingly spread the infection. Chronically infected patients don't have any symptoms, but the liver is still silently damaged, which can cause liver cirrhosis and liver cancer[5].

The most important fact is that hepatitis – B infection is preventable and treatable. Hepatitis- B vaccination is one of the effective methods to prevent the disease; a simple blood test is enough to diagnose the infection. Thus, vaccination plays a major role as it helps to prevent the disease at the earliest.

THE ROLE OF LIVER AND HEPATITIS- B:

- The liver is such a vital organ that humans can only survive for one or two days if it shuts down. If the liver fails, the rest of the organs in the body will continue to work. The liver can still work even if up to 80% of it is damaged[5]. This is due to its remarkable capacity to regenerate itself using the healthy liver cells that are still present. Hepatitis infection is acute if it resolves within six months and chronic if it lasts more than six months.

There are various functions of the liver of which the following are the most important. They are as follows:

- Processing of nutrients from the food
- Storage of sugar for later use
- Liver produces bile
- It helps in removing various toxins from the body and combating infections
- It helps in the processing and storage of vitamins
- It plays a major role in protein synthesis

- It helps in maintaining the levels of fats, amino acids, and glucose in the blood.
- It plays a major role in regulating hormones including those that help in platelet formation.

TYPES OF HEPATITIS VIRUSES:

There are five types of hepatitis viruses which include

- Hepatitis A
- Hepatitis B
- Hepatitis C
- Hepatitis D
- Hepatitis E

Certain other viruses can also cause liver inflammation including the Epstein-Barr virus, Cytomegalovirus, and yellow fever virus.

Through contaminated food and water, hepatitis A and E virus spread. Of all these types Hepatitis A, B, and D are preventable with immunization[5].

STRUCTURE OF HEPATITIS B VIRUS:

Hepatitis B virus is a DNA virus and it belongs to the hepadnaviridae family.

Group: Group VII (dsDNA-RT)
Family: Hepadnaviridae
Genus: Orthohepadnavirus
Species: Hepatitis B virus

The virus contains the outer envelope, the core (27 nm), and the icosahedral nucleocapsid
(4 nm) and the phrase "surface antigen" or "HBsAg" refers to the outside membrane, which is made up of lipids and proteins.

The core particle, also known as "HBcAg," is an inner protein shell that houses the viral DNA and the enzymes needed for viral replication (referred to as "DNA polymerase").

Hepatitis B e antigen, or(HBeAg), is an antigenic determinant that is closely related to the HBV nucleocapsid. In serum, it also circulates as a soluble protein.

Electron microscopy is used to identify three different viral particle types in infected serum.

- Diameter of 42 nm (Dane particle- Virion)
- Diameter of 20 nm (Spherical Structures)
- Diameter of 22 nm (Filamentous Particles)

GENOME OF HEPATITIS B VIRUS:[6]

The 5′ end of the full-length minus strand of the genome, which is connected to the viral DNA polymerase, creates a covalently closed circle with other ends of the double-stranded DNA.

Size: Circular, non-segmented, about 3.2 kilobases (kb).

The genome has two lengths: 1700–2800 nucleotides and 3020–3320 nucleotides (for the entire length strand) (and for the short-length strand).

The compact organization with no non-coding areas and four overlapping reading frames
flowing in the same direction.

SYMPTOMS:

The various symptoms of hepatitis infection include the following:

- Fever
- Fatigue
- Dark-colored urine
- Nausea, vomiting
- Jaundice
- Upper right abdomen pain (due to inflamed liver)

REPLICATION OF VIRUS :

As it adheres to cells, the infectious virion sheds its coating and the viral genome's partly double-stranded form is transformed into covalently closed circular double-stranded DNA in the nucleus (cccDNA).

All viral transcripts, including a 3.5-kb pregenome RNA, are templated by the cccDNA and the freshly created HBcAg encapsidates the pregenome RNA.

A negative-strand DNA copy is created inside the cores by reverse transcription and it is carried out by the viral polymerase. Though it begins, the polymerase does not finish creating the positive DNA strand. Pre-Golgi membranes allow for the budging of cores, which may then take up and eventually shed HBsAg-containing envelopes.

As an alternative, cores can be imported again into the nucleus to start fresh replication in the same cell[6].

PATHOGENESIS OF HEPATITIS B VIRUS:

Injuries to liver cells caused by HBV infections appear to be caused by three different pathways.

The first is an HLA class I restricted cytotoxic T-cell (CTL) response focused on HBcAg/HBeAg on HBV-infected hepatocytes.

A second potential mechanism is a direct cytopathic effect of HBcAg expression in infected hepatocytes. A third potential mechanism is HBsAg's high level of expression and ineffective secretion.

In acute hepatitis, the portal tracts exhibit symptoms of inflammation; the infiltrate is mostly lymphocytic. Infected hepatocytes in the liver parenchyma inflate and develop acidophilic (Councilman) bodies as they expire.

In chronic hepatitis, the damage spreads from the portal tracts, creating the appearance of piecemeal necrosis. There is also some lobular inflammation. Fibrosis and ultimately cirrhosis appear as the illness worsens.

The immune response might not be the only cause of liver damage in people with hepatitis B. Post-liver transplant hepatitis B patients on immunosuppressant therapy also have hepatitis B-associated damage. The histological pattern that results from this illness is known as fibrosing cholestatic hepatitis, and it is believed to be linked to a significant amount of HBsAg exposure. This supports the theory that hepatitis B may still be harmful despite an immune system response.

HISTOPATHOLOGICAL FINDINGS:

Hepatitis B infection that is acute is characterized by "lobular disarray, ballooning degeneration, numerous apoptotic bodies, Kupffer cell activation, and lymphocyte-predominant lobular and portal inflammation," according to histologic results[7].

Lymphocyte-predominant portal inflammation along with interface hepatitis and patchy lobular inflammation characterize chronic hepatitis B infection.

DIAGNOSIS OF HEPATITIS B:

The diagnosis of hepatitis B virus is by proper history taking, physical examination, laboratory investigations, and imaging.

Usually, the initial symptoms are non-specific. The evaluation of serum biomarkers plays a major role in the diagnosis of the infection.

Hepatitis B surface antigen serves as the main viral marker in the serology for the disease, which is typically detectable 1–12 weeks after initial infection (HBsAg). Rarely does the presence of HBsAg last longer than six months after infection, and it frequently occurs before detectable levels of the matching antibody to surface antigen (Anti-HBsAg). The window period or "serological gap" refers to the interval of time between the removal of HBsAg and the emergence of Anti-HBsAg. The viral serology could not be detected during the window period.

Usually, liver enzyme levels rise toward the end of the infection's replicative phase. Because of ongoing inflammatory processes, liver transaminases might also be within the normal range. Therefore, liver transaminases shouldn't be the only factor considered when making a hepatitis B infection diagnosis.

In contrast to HBeAg, which may suggest a persistent infection condition, HBsAg antibodies show immunization status. The emergence of antibodies to HBeAg on their own is known as seroconversion, and it is the process by which an acute, immune-active phase changes into an inactive carrier state. With repeated episodes of reactivation and remission, later

seroconversion is more likely to develop problems such as liver cirrhosis, which leads to less favorable results. Earlier seroconversion has been linked to better outcomes. [8] Acute hepatitis B infection is distinguished from chronic hepatitis B infection by the persistence of serum HBsAg for 6 months or more[9].

INTERPRETATION OF SEROLOGICAL MARKERS:[8]

- HBsAg: Acute or chronic infection
- Anti-HBs: Immunity from vaccination or recovery after acute infection.
- Anti-HBe is a Low replicative phase
- Anti-HBc IgM: Acute infection is the sole marker visible throughout the window period and may be evident when a chronic infection is exacerbating.
- Anti-HBc IgG: Infection exposure, chronic infection (if present along with HBsAg), acute infection recovery (if present with anti-HBs). The occult infection may represent occult infection.
- Hepatitis B genotyping helps to know the disease progression and response to interferons[10].

STEPS TO BE FOLLOWED IN CASE OF ACCIDENTAL EXPOSURE DURING ROUTINE DENTAL PROCEDURES:

The wound should be washed gently without rubbing for several minutes with soap and water or a disinfectant that is effective against the virus. This helps to prevent the infection from spreading to deeper tissues (iodine solutions or chlorine formulations). To remove any potentially important debris, it is important to cause bleeding by applying pressure beneath the level of the cut. But this fact has not received a lot of support. The final target of all these measures is to lower the number of viral units below the threshold count which is needed to trigger infection (the infectious dose). Diluting a substance with water may cause the virus to count to fall below this cut-off value. A thorough record of the patient's medical and clinical history must be kept to comply with the law.

HISTORY OF HBV VACCINES AND RECOMMENDATIONS:

The prevention of HBV infection depends on vaccination. Over the years, there have been three generations of HBV vaccinations released. The first generation of HBV vaccines, which comprise concentrated plasma from HBV-infected persons and contain HBsAg, was developed as a result of Krugman's observation regarding the immunogenicity of HBsAg and the protective effects of the anti-HBS antibody. This vaccine was created concurrently by Merck and the Pasteur Institute and was authorized by the FDA in 1981[11]. The Saccharomyces cerevisiae yeast, which also contains HBsAg, was used to create the second generation of HBV vaccinations using recombinant DNA technology. Pre-S1 and pre-S2 antigens are used in the third-generation vaccines, which are significantly more immunogenic. Third-generation vaccinations were created using mammalian cells and recombinant DNA technologies.

ROLE OF DENTAL HEALTH CARE PROFESSIONALS AND CROSS-INFECTION:

Microorganisms can be spread in dental care facilities in a variety of ways, including direct contact with patients' bodily fluids or blood, indirect contact with infected tools, or ambient surfaces. Another method of transmission is by the contact of the mucosa of the nasal, conjunctival, or oral cavity with droplets containing germs. Previous studies have demonstrated that saliva contains HBsAg particles as well as contagious viruses. However, even in HBsAg-positive blood, the virus count is modest. Dentistry's most frequent occupational hazards are needle sticks from anesthetic needles or cutting equipment. In a survey conducted by the American Dental Association, private practitioners stated that they suffer an average of 3.2 injuries annually. Institutions teaching dentistry have also reported substantially higher rates. These reports indicate that there is a chance that HBV will be transmitted to dental health care personnel (DHCP). The prevalence of HBV serologic markers among dentists has been estimated to range from 16 to 28 percent in certain previously published research[12],[13]. Only emergency dental care should be provided to HBV patients, according to the guidelines that are currently available.

GUIDELINES FOR SHARPS SAFETY IN DENTAL SETTINGS :

The sharp items such as needles, scalers, burs, lab knives, and wires that are contaminated with the saliva and blood of the patient are to be considered potentially infective and engineering controls are to be established in work practices to prevent injuries.

The used needles should not be recapped by both hands or other technique that involves directing the point of the needle toward the parts of the body.

Usage of a one-handed scoop technique or a mechanical device for holding the needle cap for recapping the needles.

Placed the needles, and syringes in a puncture-resistant container located as close as possible[14].

KEY STEPS FOR SAFE INJECTION PRACTICES:

Injections are to be prepared using an aseptic technique in a clean area.

The rubber septum is to be disinfected with alcohol before piercing.

The needles are not to be used for more than one patient.

Single-dose vials for parenteral medications are recommended.

Leftover contents of single-use vials are not to be combined for later use.

Multidose vials when first opened are to be discarded within 28 days unless it is specified by a manufacturer for a shorter or longer date for that opened vial.

The fluid infusion bags are not to be used for more than one patient[15].

ANTIBODY LEVEL STATUS AFTER VACCINATION:

According to several studies, immunological memory still provides an adequate level of protection after the initial dose even if the antibody titer results as not protective (anti-HBs < 10 UI/L). Thus a booster dose is unnecessary[16],[17].

PREVENTION STRATEGY:

In 95 percent of healthy adults, acute infection is self-cleared. In severe acute cases, antiviral treatment is recommended. Identification of HIV, hepatitis C, and hepatitis D coinfection, the status of the hepatitis B virus replicating, and the severity of the disease are all important components of managing chronic hepatitis B[18].

Entecavir combination medications were not shown to be any more successful than entecavir monotherapy, according to a 2018 meta-analysis based on 24 studies comparing entecavir polytherapy with monotherapy[19].

Tenofovir administration in HBsAg-positive and/or HBeAg-positive mothers were shown to be beneficial in lowering ALT levels in mothers and lowering newborn HBsAg levels at 6 months postpartum in 2015 prospective, multicentre trial[20]. The first substance that effectively reduced viral numbers was lamivudine, but it was also highly medication resistant[21].

The double-blind, placebo-controlled 2013 GAHB trial contrasted Lamivudine and a placebo. With lamivudine medication, most patients could clear their HBsAg, although the study's overall strength was compromised by the small number of participants (n = 35)[22].

According to a randomized controlled trial, the viral load was suboptimally controlled, secondary to high circulating levels of HBV DNA [23].

While there was inadequate data for statistical significance for HBsAg seroconversion in the 2018 POTENT trial, there was no discernible difference between sequential therapy and monotherapy[24].

CONCLUSION:

One of the best methods for preventing the spread of Hepatitis B is the vaccination of dental healthcare practitioners. Immunization significantly lowers the number of dental healthcare workers who are frequent exposed to illnesses , risk of illness spreading to other dental healthcare workers and patients. Consequently, oral healthcare professionals must have a vaccination plan, and doses should be taken at a stipulated time

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