International Journal of Innovative and Applied Research [2023]

(Volume 11, Issue 04)



Journal home page: http://www.journalijiar.com

INTERNATIONAL JOURNAL **OF INNOVATIVE AND APPLIED RESEARCH**

RESEARCH ARTICLE

Article DOI: 10.58538/IJIAR/2019 **DOI URL:** http://dx.doi.org/10.58538/IJIAR/2019

HEPATITIS B VIRAL INFECTION: OCCUPATIONAL HARZARS

*Emmanuel Ifeanyi Obeagu¹ and Calister Ndidi Adike²

1. Department of Medical Laboratory Science, Kampala International University, Uganda.

2. Department of Medical Laboratory Science, Nnamdi Azikiwe University, Nnewi Campus, Nnewi, Anambra State, Nigeria.

Manuscript Info	Abstract
Manuscript History Received: 11 March 2023 Final Accepted: 23 April 2023 Published: April 2023 <i>Keywords:</i> Hepatitis B, Infection, Hazards, Occupation	Hepatitis B is a viral infection that attacks the liver and can cause both acute and chronic disease. Hepatitis B is a potentially life-threatening liver infection caused by the hepatitis B virus. Hepatitis B prevalence is highest in sub-Saharan Africa and East Asia, where between 5–10% of the adult population is chronically infected. Hepatitis B is an important occupational hazard for health workers. However, it can be prevented by currently available safe and effective vaccine. Hepatitis B has a potential of causing life-threatening liver infection caused by the hepatitis B virus.

*Corresponding Author:- Emmanuel Ifeanyi Obeagu

.

Introduction:-

Hepatitis B is a viral infection that attacks the liver and can cause both acute and chronic disease. Hepatitis B is a potentially life-threatening liver infection caused by the hepatitis B virus. It is a major global health problem. It can cause chronic infection and puts people at high risk of death from cirrhosis and liver cancer [1-5].

Globally, some 240 million people have chronic hepatitis B virus with the highest rates of infection in Africa and Asia. People with chronic hepatitis B infection are at increased risk of dying from cirrhosis and liver cancer[6].

The sub-Saharan Africa is ranked highly endemic, Africa has the second largest number of chronic HBV carrier rate after Asia, with over 50 million people being life time carriers. It has been estimated that over 12 million people will die due hepatitis B- induced liver disease, representing a 25% risk among carrier [7].

In sub-Saharan Africa, the Pacific, and particularly Asia, HBV infection is highly endemic, with the majority of individuals becoming infected during childhood [6].

Hepatitis B prevalence is highest in sub-Saharan Africa and East Asia, where between 5-10% of the adult population is chronically infected. High rates of chronic infections are also found in the Amazon and the southern parts of eastern and central Europe. In the Middle East and the Indian subcontinent, an estimated 2-5% of the general population is chronically infected. Less than 1% of the population of Western Europe and North America is chronically infected [7].

04-08

(Volume 11, Issue 04)

Hepatitis B is also spread by percutaneous or mucosal exposure to infected blood and various body fluids, as well as through saliva, menstrual, vaginal, and seminal fluids. Sexual transmission of hepatitis B may occur, particularly in unvaccinated men who have sex with men and heterosexual persons with multiple sex partners or contact with sex workers. Infection in adulthood leads to chronic hepatitis in less than 5% of cases. Transmission of the virus may also occur through the reuse of needles and syringes either in health-care settings or among persons who inject drugs. In addition, infection can occur during medical, surgical and dental procedures, through tattooing, or through the use of razors and similar objects that are contaminated with infected blood [8].

Prevalence of Hepatitis B

Globally, Hepatitis B virus (HBV) is seen to be a public burden of causing the world's major infectious diseases of which 350 million people being chronic carriers of the virus. Hepatitis B infection is the 10th leading cause of death worldwide, as a significant number of the chronic carriers go on to develop liver cirrhosis or hepatocellular carcinoma (HCC) and over 1 million die annually from HBV associated liver disease [9]. According to Lavanchy [10] HCC account for 320 000 deaths per year despite the antiviral drugs that are available for HBV infected individuals that may prevent the danger progression of chronic liver disease, which allow for the significance identification of infected individuals and monitoring the prevalence of the disease [11]. More than 750000 deaths are due to HBV-related complications. (Lozano et al., 2010) Early diagnosis and prompt treatment of chronic hepatitis B infection is important for reducing morbidity and mortality.

Hepatitis B is an important occupational hazard for health workers. However, it can be prevented by currently available safe and effective vaccine. Hepatitis B has a potential of causing life-threatening liver infection caused by the hepatitis B virus. It is a major global health problem. It can cause chronic infection with a progress in people putting them at high risk of death from cirrhosis and cancer of the liver [6].

Several well-defined antigen–antibody systems are associated with HBV infection. HBsAg, formerly called Australia antigen or hepatitis-associated antigen, is an antigenic determinant found on the surface of the virus. It also makes up sub viral 22-nm spherical and tubular particles. HBsAg can be identified in serum 30 to 60 days after exposure to HBV and persists for variable periods. HBsAg is not infectious. Only the complete virus (Dane particle) is infectious. During replication, HBV produces HBsAg in excess of that needed for production of Dane particles. HBsAg is antigenically heterogeneous, with a common antigen and 2 pairs of mutually exclusive antigens, resulting in 4 major subtypes: adw, ayw, adr and ayr. The distribution of subtypes varies geographically; because of the common "a" determinant, protection against one subtype appears to confer protection against the other subtypes, and no differences in clinical features have been related to subtype [12].

In Africa ,HIV infection has a major impacts on the course of HBV infection, causing uprising liver disease and increasing mortality up to 8-fold compared to those infected by HIV alone [12]. According to Modi and Feld[13], the prevalence of HBV co-infection among HIV-infected Africans vary from approximately 5 to 25% on estimation but the prevalence and existence of HIV-HBV co-infection is unknown in many sub-Saharan African populations. Improved understanding of the course of HBV infection in the setting of HIV could inform HBV prevention strategies including both vaccination efforts and treatment strategies for HIV/HBV co-infected persons in Africa

Knowledge on Hepatitis B

Viral hepatitis is a common term used referring to the inflammation of hepatocytes with no prior differentiation in clinical, etiology and epidemiological differences with responsible viruses. Hepatitis A (formerly called infectious hepatitis) and hepatitis B (formerly called serum hepatitis) have been recognized as separate entities since the early 1940s and can be diagnosed with specific serologic tests. Delta hepatitis is an infection dependent on the hepatitis B virus (HBV). It may occur as a co-infection with acute HBV infection or as super infection of an HBV carrier.

Epidemic jaundice was described by Hippocrates in the 5th century. The first recorded cases of "serum hepatitis," or hepatitis B, are thought to be those that followed the administration of smallpox vaccine containing human lymph to shipyard workers in Germany in 1883. In the early and middle parts of the 20th century, serum hepatitis was repeatedly observed following the use of contaminated needles and syringes. The role of blood as a vehicle for virus transmission was further emphasized in 1943, when Beeson described jaundice that had occurred in seven recipients of blood transfusions. Australia antigen, later called hepatitis B surface antigen (HBsAg), was first described in 1965, and the Dane particle (complete hepatitis B virion) was identified in 1970. Identification of serologic markers for HBV infection followed, which helped clarify the natural history of the disease. Ultimately, HBsAg was

(Volume 11, Issue 04)

prepared in quantity and now comprises the immunogen in highly effective vaccines for prevention of HBV infection[14].

Transmission risks of hepatitis B

The hepatitis B virus can survive outside the body for at least 7 days. Several factors influence the risk of transmission of HBV infection, including the viral load of the source in a healthcare occupational context, the level that is regarded as "high" for a viral load differs in various regions. In America and Ireland, HCWs who are infected with HBV but have a circulating viral burden <104 genome equivalents/ ml are allowed to continue working unrestricted [15].Transmission of HBV via percutaneous route is considered unlikely at HBV DNA levels below 107genome equivalents/ml [16].

Health Occupational Predisposition

Needle stick injuries

Those who are e antigen positive generally have higher viral loads, and the transmission rate of HBV following a needle stick injury from a source who is e antigen positive is estimated to be between 30% and 62%. The same injury with exposure to blood from a source who is antigen negative is associated with 6-37% risk of serological evidence of HBV infection in the recipient[17]. Some patients are infected with pre-core mutant viruses. This is associated with a high viral load in the absence of the e antigen, and thus is also associated with a high risk of HBV transmission risk from needle stick injuries in the community is more difficult to estimate and the exact incidence of needle stick injuries and the transmission rate is unknown. The limited published case reports would indicate that there is a very low risk of HBV transmission associated with community acquired needle stick injuries [18].

Healthcare setting exposures

Spring loaded lancets have been implicated in the transmission of HBV to patients (Polish et al., 2010) as have reusable sub-dermal EEG electrodes [19]. There is a report of transmission of HBV to a patient during an endoscopic procedure, although no biopsies were taken, but bleeding gastric ulceration was identified. The presumed source was HBeAg positive [20].

Cleveland et al report that HBV infection prevalence in dentist's increases with longer duration in practice [20]. Although rates in a reference control population were not included in this report, increasing prevalence with longer duration of practice indicates that there is potential for transmission to dentists during their work.

Personal behavioral factors

Percutaneous exposures

There are case reports documenting the transmission of HBV among butchers, these are attributed to small hand cuts, and sharing knives, which can carry the virus on the handle. It is also thought that HBV can be transmitted via small cuts acquired in barber shops [22]. HBV is transmitted by percutaneous and mucous membrane exposures to infectious body fluids, such as serum, semen, and saliva [23].

Body fluid exposures

HBV DNA has been detected in body fluids apart from blood, including saliva, urine, nasopharyngeal fluid, semen, cervicovaginal fluids and tears [24].HBV transmission can occur following exposure to non-intact skin and mucous membranes

Human bite

HBV virus can be transmitted via a human bite, when associated with the skin being broken [25].

Sexual exposure

The prevalence of HBV in heterosexuals is increased in those with multiple sexual partners and those who have markers for HIV or syphilis, an infection rate of is seen in regular heterosexual partners of HBV infected patients In addition, female commercial sex workers with a history of having anal intercourse had an increased risk of HBV infection, the risk of developing HBV infection is particularly high among men who have sex with men.

(Volume 11, Issue 04)

Treatment challenges to hepatitis B

According to Zoulim and Durantel [26], the prevention and control of Chronic Hepatitis B (CHB) virus infection have greatly improved .Despite different available anti-HBV reagents and the updated variable guidelines, the control of HBV becomes more difficult [27].Now a days, available therapies popularly used in the whole world are safe, well tolerated, and highly effective in anti-HBV therapy, both reducing HBV viremia and improving clinical course and prognosis [28]. However, due to antiviral resistance and HBV, long-term administration remains a clinical challenge: only long-term virologic control, elimination of HBV and the recovery of CHB patients are not possible [29]

IgM anti-HBc manifests in persons with acute disease about the time of illness onset and indicates recent HBV infection. IgM anti-HBc can be detected from 4 to 6 months after onset of illness and is the best serologic marker of acute HBV infection. A negative test for IgM-anti-HBc together with a positive test for HBsAg in a single blood sample identifies a chronic HBV infection. HBV DNA assays are used to monitor response to treatment, assess the likelihood of maternal-to-child transmission of HBV, and to detect the presence of occult HBV infection (i.e. infection in someone who tests HBsAg negative) [30].

Persons with acute or chronic HBV infections should prevent their blood and other potentially infective body fluids from contacting other persons. They should not donate blood or share toothbrushes or razors with household members [31].

Several factors have been associated with nonresponse to hepatitis B vaccine. These include vaccine factors and host factors. Older age, male sex, obesity, smoking, and chronic illness have been independently associated with nonresponse to hepatitis B vaccine. Additional vaccine doses for persons who receive post-vaccination testing and who fail to respond to a primary vaccination series administered in the deltoid muscle produce adequate response in 15% to 25% of vaccines after one additional dose and in 30% to 50% after three additional doses [32].

Conclusion:-

Hepatitis B is an important occupational hazard for health workers. However, it can be prevented by currently available safe and effective vaccine. Hepatitis B has a potential of causing life-threatening liver infection caused by the hepatitis B virus.

References:-

- 1. WHO. Facts about hepatitis, July 2016, World Health organization. Fact Sheet. Hepatitis B. 2016.
- 2. Ifeanyi OE, Amilo Grace I, Uzoma OG, Nnatuanya Isaac N. Human Immunodeficiency Virus infection and cardiovascular. Int. J. Curr. Res. Med. Sci. 2017;3(9):9-37.
- Nwovu A, Ifeanyi OE, Uzoma OG, Nwebonyi NS. Occurrence of Some Blood Borne Viral Infection and Adherence to Universal Precautions among Laboratory Staff in Federal Teaching Hospital Abakaliki Ebonyi State. Arch Blood Transfus Disord. 2018;1(2). Ifeanyi OE, Leticia OI, Nwosu D, Chinedum OK. A Review on blood borne viral infections: universal precautions. Int. J. Adv. Res. Biol. Sci. 2018;5(6):60-6.
- 4. Obeagu E, Chima CO, Nwosu DC, Opara AU, Dike-Ndudim JN, Ahiara CO, Obeagu EI. STUDIES ON HEPATITIS B VIRUS INFECTION IN EBONYI STATE NIGERIA USING HBSAG AS MARKERS: RAPID ASSESSMENT SURVEY. Madonna University journal of Medicine and Health Sciences ISSN: 2814-3035. 2022 Mar 3;2(1):185-203.
- Obeagu E, Chima CO, Nwosu DC, Dike-Ndudim JN, Ahiara CO, Obeagu EI. Comparative Studies on Hepatitis B Virus Infection in Ebonyi State Nigeria Using HBsAg as indices. Madonna University journal of Medicine and Health Sciences ISSN: 2814-3035. 2022 Mar 3;2(1):159-84.
- 6. WHO. Guidelines for the prevention, care and treatment of persons living with chronic hepatitis B infection. 2015.
- 7. Kiire CF. The epidemiology and prophylaxis of hepatitis B in sub-Saharan Africa: a view from tropical and sub-tropical Africa. Gut. 2015; 38:S5-12.
- 8. Centre for Disease Control Prevention. prevention. Updated U.S. Public Health Service guidelines for the management of occupational exposures to HBV, HCV, and HIV and recommendations for post exposure prophylaxis, MMWR. 2016; 50(RR-11):1-52.
- 9. Wright., T. Introduction to chronic hepatitis B infection. Am J Gastroenterol. 2009; 101(Suppl 1): S1–S6.
- 10. Lavanchy D. Hepatitis B virus epidemiology, disease burden, treatment, and current and emerging prevention

(Volume 11, Issue 04)

and control measures., J Viral Hepat. 2011; 11: 97–107.

- 11. Weinbaum CM, Mast EE. Recommendations for identification and public health management of persons with chronic hepatitis B virus infection. Hepatology. 2009; 49: S35–S44.
- 12. CDC. Recommendations for identification and public health management of persons with chronic hepatitis B virus infection., MMWR 2008; 57(RR-8); 9-11.
- 13. Modi AA. Viral hepatitis and HIV in Africa. AIDS Rev. 2007; 9(1):25-39.
- CDC. Acomprehensive immunization strategy to eliminate transmission of hepatitis B virus infection in the United States: recommendations of the Advisory Committee on Immunization Practices (ACIP). Part 1: Immunization of infants, children, and adolescents., MMWR 2005; 54(No. RR-16):1–32.
- 15. Henderson Dk, Dembry I, Fishman No, Grady C, lundstrom T, Palmore N. SHEA guideline for management of healthcare workers who are infected with hepatitis B virus, hepatitis C virus, and/or human immunodeficiency virus. Infection Control HospEpidemiol 2010; ; 31(3):203-32.
- 16. Buster EH, van der Eijk AA. Doctor to patient transmission of hepatitis B virus: implications of HBV DNA levels and potential new solutions., Antiviral Research. 2011; 60(2):79-85.
- 17. Department, of H. andChildre. The prevention of transmission of blood-borne diseases in the health-care setting 2005. Blood borne diseases. 2008.
- 18. Res SBF. Acute hepatitis B infection following a community-acquired needle stick injury. J infect 2011; 62(6):487-9.
- 19. CMAJ. An outbreak of hepatitis B associated with reusable sub dermal electroencephalogram electrodes. Hepatitis B outbreak investigation team. 2010; 162(8):1127-31.
- 20. Birnie GG, Quigley EM, Clements GB, Follet EA. Endoscopic transmission of hepatitis B virus., Gut 2014; 24(2):171-4.
- 21. Cleveland Jl, Siew C, lockwood SA, Gruninger SE, Gooch BF. Hepatitis B vaccination and infection among U.S. dentists. J Am Dent Assoc 2009; 127(9):1385-90.
- 22. Mariano A, Mele A, tosti ME, Parlato A, Gallo G, Ragni P. Role of beauty treatment in the spread of parenterally transmitted hepatitis viruses in italy. J Med Virol 2008; 74(2):216-20.
- 23. Murray CLA. The global burden of disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 2010 and projected to 2020. Cambridge, Mass: Harvard University Press. 2010.
- 24. k idd-ljunggren k, Holmberg A, Blackberg J, lindqvist B. High levels of hepatitis B virus DNA in body fluids from chronic carriers., J Hosp infect 2006; 64(4):352-7.
- 25. Hui AY, Hung IC, tse PC, leungWk, Chan Pk. transmission of hepatitis B by human bite--confirmation by detection of virus in saliva and full genome sequencing. J ClinVirol, 2009;33(3):254-6.
- 26. Zoulim FDD. Antiviral therapies and prospects for a cure of chronic hepatitis B. Cold Spring HarbPerspect Med. 2015; 5.
- 27. Huang Z. Aberrant expression and dysfunction of TLR2 and its soluble form in chronic HBV infection and its regulation by antiviral therapy. Antiviral Res 2015;, 118: 10-9.
- Zoulim FLS. Hepatitis B virus resistance to nucleos(t)ide analogues. Gastroenterology 2009; 137: 1593-608 e1-2.
- 29. Ohara T. Efficacy of double filtration plasmapheresis with pegylated interferon/ribavirin therapy for intractable chronic hepatitis C patients and hepatitis C patients with combined liver cirrhosis by HBV, leading to early viral elimination. Hepato-gastroenterolog. 2011.
- 30. Lewis E, Shinefield HR, Woodruff BA. Safety of neonatal hepatitis B vaccine administration. Pediatr Infect Dis J 2009, 20:1049–54.
- 31. Poland GA. Clinical practice: prevention of hepatitis B with the hepatitis B vaccine. N Engl J Med 2012; 351:2832–8.
- 32. CDC. Use of hepatitis B vaccination for adults with diabetes mellitus. Recommendations of the Advisory Committee on Immunization Practices (ACIP)., MMWR 2011; 60(50):1709-11.