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SEROPREVALENCE OF HEPATITIS B VIRUS INFECTION AMONG THE STUDENTS OF NASARAWA STATE UNIVERSITY, KEFFI, NIGERIA.

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Abstract: *Hepatitis B virus is a serious global health problem, which account for about 2 billion infected cases and 400 million chronic infections worldwide, accounting for about for about 1.2 million-mortality rate annually. This study was carried out to detect the Seroprevalence of hepatitis B surface antigen (HBsAg) among Students of Nasarawa University, Keffi. The samples were collected and tested using one-step rapid strip (Royal care), and Combo Test Device (Skytech USA) was used to confirm the positive samples. Out of the 168 samples tested, 12 (7.1%) were found to be HBsAg positive. The distribution of hepatitis B surface antigen in relation to age group was higher in the age group 31-above years with seroprevalence of 9 (7.3%), and lower in the age group 18-30 years with 3 (6.7%). The distribution of hepatitis B surface antigen in relation to gender was higher in males 8 (11.6%) compared to females 4 (4.0%). The study found a seroprevalence of 7.1%, which shows that the students are at risk for hepatitis B viral infection. The Nasarawa State government through the ministry of health should consider a vast or mass vaccination and treatment program that will cover undergraduate students of Nasarawa State University to curtail further spread of the virus among other students.*

Keywords: Seroprevalence, Hepatitis, Virus, Infection, disease,

1.0 Introduction

Hepatitis is general term use for describing an infection or inflammation of the liver (Arnold, 1992). Hepatitis or inflammation of the liver is cause mainly by viruses, autoimmune diseases, chemical agents or irradiation. However, conventionally, the word hepatitis refers to the disease caused by viruses primarily affecting the liver (Ochei and Kolhatkar, 2007).

Viral hepatitis is a general term reserved for infections affecting the liver caused by one of at least five distinct group of hepatitis virus (A,B,C,D and E)(Bernard *et al*., 1996). Hepatitis B virus (HBV) apart from causing hepatitis it is also a major risk factor in the development of liver cirrhosis and hepatocellular carcinoma (HCC) (Cancer of the liver) in humans. HBV is caused by

a double stranded DNA virus of the hepadnaviridae family measuring 42nm in size. It is also refer as Dane particle (Arnold, 1992; Ochei and Kolhatkar, 2007).

The DNA genome composed of a complete negative strand and an incomplete positive strand. The virion contains a DNA polymerase which completes the synthesis of a closed circular positive strand DNA. It contains a nucleocapsid core, the polypeptide component of which is known as core antigen. The core antigen is surrounded by an outer coat, called envelope. Contain in the envelope is the hepatitis B surface antigen (HBsAg), formally known as the Australian antigen (AU). Another antigen, the hepatitis B envelope (HBeAg) is also associated with the core of the virus. These antigens HBsAg and HBeAg may be detected in the sera of infected individuals. These antigens and there homologous antibodies are considered to be specific markers of hepatitis B virus infection (Arnold, 1992; Ochei and Kolhatkar, 2007; Jawetz *et al*., 2013). Hepatitis B viral infection is a highly contagious liver disease that results from infection with hepatitis B virus (Bernard *et al*., 1996; Jinlin *et al*., 2003), and it is 50 to 100 times more infective than HIV (Gebere *et al*., 2013; WHO, 2015). HBV have an incubation period which varies widely from four weeks to six months (CDC, 2015). After infection, there is a variable incubation period during which the virus replicates in the liver tissue but do not cause overt pathology, shortly before the onset of symptoms; the virus is detectable in the blood (Arnold, 1992).
When an individual is exposed to HBV, the person can develop acute infection which can range in severity from mild illness with few or no symptoms to a serious condition requiring hospitalization (CDC, 2015; Joanah *et al*. 2016). It is very pathetic to know that many of those infected individuals are asymptomatic and hence failed to seek for appropriate medical help, therefore progressing to the chronic liver disease, and liver cancer (Bello *et al*., 2011). HBV chronically infect approximately 350 million people worldwide. Without intervention about 15%-40% of the chronically infected individuals will eventually develop cirrhosis, end-stage liver disease or hepatocellular carcinoma, or require liver transplantation (Mel *et al*., 2005).

Acute hepatitis refer to the first six months after exposure to HBV, in which the patient present with fever, malaise, nausea, fatigue, loss of appetite, jaundice (dark urine due to deposition of bile pigment). Chronic replication of the virus in the liver can destroy the organ, it also carry a high risk of hepatocellular carcinoma. But an effective vaccine has been developed and is extensively in use in many countries. HBV have an extensive interaction with its host, resulting in slow and progressive disease, this chronic and active growths of the virus often result in liver cancer. Hepatitis is the most common cause of liver cancer in the world especially in Africa and Asia (Arnold, 1992).

Hepatitis B viral infection can be spread by blood, body fluids, and close personal contact, mother to child, unprotected sex, sharps and penetrating objects and through sharing of cups and spoons (Cheesbrough, 2006). Implementation of effective vaccination measure can reduce the global prevalence of hepatitis B viral infection. Vaccination is an economically advantageous option both in term of cost effectiveness and benefit cost. The world health organization in 1991 recommended that all countries should introduce a policy of universal hepatitis B vaccination to prevent and control HBV infection and its long term menace on the global scale (Ndako *et al*., 2016).

Statement of Problem

There is high HBV carrier rate found in sub Saharan Africa, Kalahari Desert areas and Asia. Most people become infected at birth or at childhood, or by sexual contact, and up to 20 percent become chronic carriers, particularly those infected between 1 to 5 years (Cheesbrough, 2006). Worldwide, there are estimated 257 million people living with hepatitis B virus, in 2015 , hepatitis resulted in 887,000 deaths, mostly from complications (including cirrhosis and hepatocellular carcinoma) (WHO, 2018).

In West Africa, it has been estimated that 40% children, will be infected by age 2 years and above 90% by the age 10 years. Carrier rate is 20% in these children. According to WHO, any carrier rate above 7% in a population is considered endemic (Emechebe *et al*., 2009). Nigeria is classified among the countries endemic for Hepatitis B viral infection, accounting for about 18 million cases and has reach hyper-endemic level with sero-prevalence of HbsAg estimated to to range from 10-40% (Jombo, *et al*, 2005).

Hepatitis B is the leading cause of liver cirrhosis and hepatocellular carcinoma and most death related hepatitis. Hepatitis B has become a major public health problem globally occurring endemically in most areas of the world especially in sub Saharan Africa. Most research have shown that Nigeria have a 10 percent higher prevalence rate than South Africa (Alao *et al*., 2009).

Justifications of the Study

Several work have been carried out on hepatitis B virus in many parts of the country, but there is not yet any scientific documented research work carried out on students of Nasarawa State University, Keffi knowing fully well that hepatitis B viral infection is one of the major silent killer disease in the world this motivated the research work, as early detection and treatment can save lives.

Significance of the Study

This study will provide baseline data for further research work in the study area or the state and the findings will also add to the body of knowledge.

Research Question

What Is The Prevalence of Hepatitis B Infection Among Students of Nasarawa State University?

Hypotheses

Null Hypothesis (HO): Hepatitis B viral infection is not highly prevalent among students of Nasarawa State University.

Alternative Hypothesis (HI): Hepatitis B viral infection is highly prevalent among students of Nasarawa State University

Aim of the Study

To determine the seroprevalence of hepatitis B infection among the students of Nasarawa State University, Keffi, Nigeria.

Objectives of The Research Work

1. To determine the seroprevalence of hepatitis B virus among Students in the study area.
2. To determine the seroprevalence of HBV in relation to some demographic data.
3. To confirm the active replicative state of the virus using combo test device.
4. To determine the risk factor associated with the prevalence of hepatitis B infection among the students.

2.0 LITERITURE REVIEW

Historical Background of Hepatitis B Virus

Liver diseases associated with the inflammation of the hepatocytes had been in existence right from the time of Hippocrates, which is usually marked by jaundice. The existence of a parenterally transmitted form of hepatitis was documented by Lurman in 1885; he reported the development of jaundice in 15% of 1,289 shipyard workers in Bremen 2−8 months after receiving Smallpox vaccine prepared from human lymph. Others was trace to the improper sterilization of syringes and needles, especially among patients from veneral disease clinic receiving subcutaneous, intra muscular, intra venous injection of therapeutic agents (Bernard *et al*., 1998).

The chain of events that culminated the discovery of hepatitis B virus was a tortuous one. It began in 1965, when Baruch Blumberg of the National Institute of Health, began examining thousands of blood samples from diverse populations in a study designed to look for inherited polymorphic traits in different geographic areas of the world, to detect novel antigens, sera from multiply transfused haemophilia patients were used because it was postulated that such sera might contain antibodies against these unique protein. In the course of the study he discover the Hepatitis B virus in the serum sample from an Australian aboriginal contained that reacted specifically with the antibody from an American haemophilia; the Australian antigen which was also known as hepatitis B surface antigen (HBsAg), which is found in blood(Alter and Blumberg 1966; Bernard *et al*., 1998).

In 1970 Dane *et al*. Detected the complete HB virion a 42nm double shelled particle that consist of an outer envelope, the other viral particles such as the sub viral spherical particles and the sub viral filamentous were also discovered. The World Health Organization named this virus Hepatitis B virus. The first vaccines for Hepatitis B virus was later discovered and was being tested in December 1980 (Bernard *et al*., 1998).

The discovery of the first specific marker of viral hepatitis−Australian antigen (AUAg), now referred to as hepatitis B surface antigen (HBsAg) or sometimes called serum Hepatitis became

the driving force for the development of modern virus diagnostic, vaccine and this also allowed for the screening of blood from donors before successful and effective transfusion (Blumberg *et al*., 1965; Brigham *et al*., 2003; Gerlich. 2013).

Genotype and Serotype of Hepatitis B Virus

The virus is divided into four serotypes (adr, adw, ayr, ayw) based on antigenic epitopes present on it envelope proteins, and into eight genotypes (A−H) according to the nucleotide sequence variation of the genome. The genotypes have a distinct geographical distribution and are used in tracing the evolution and transmission of the virus. Differences between genotypes affect the disease severity, course and likelihood of complications, and response to treatment and possibly vaccination (Magnius *et al*., 1995). Genotypes differ by at least 8% of there sequence and were first reported in 1988 when six of the genotype where initially described A−F (Norder *et al*., 1994). Two other types have since been described G and H (Shibayama *et al*., 2005). Most genotypes are now divided into sub genotypes with distinct properties (Schaefer *et al*., 2005).

Genotype A is most commonly found in the Americas, Africa, India and Western Europe. Genotype B is most commonly found in Asia and in the United States. Genotype B1 dominates in Japan, B1 dominate in China and Vietnam while B3 is confined to Indonesia. B4 is confined to Vietnam. All these strains specify the genotype ayw1, B5 is most common in the Philippines. Genotype C is common in Asia and the United States. Sub genotype C1 is common in Japan, Korea and China. C2 is common in China. Southeast Asia and Bangladesh and C3 in Oceania. All these strains specify the serotype adrq. C4 specifying ayw3 is found in Aborigines from Australia (Kurbanov *et al*., 2010). Genotype D is most commonly found in southern Europe, India and the United States and has been divided into eight subtypes (D1−D8). In Turkey genotype D is also the most common type. A pattern of defined geographical distribution is less evident with D1−D4 where these sub genotypes are widely spread within Europe, Africa and Asia. This may be due to this divergence having occurred before than of genotype B and C. D4 appears to be the oldest split and is still the dominating sub genotype of D in the Oceania. Type E is most commonly found in West and Southern Africa. Type F is most commonly found in Central and South America and has been divided into two sub groups (F1 and F2). Genotype G has an insertion of 36 nucleotides in the core gene and is found in France and the United States (Stuyver *et al*., 2000). Type H is most commonly found in Central and South America and California in United States. Africa has five genotype (A−E). Of these the predominant genotype are A in Kenya, B and D in Egypt, D in Tunisia, A−D in South Africa and E in Nigeria (Kurbanov *et al*., 2010). Genotype H is probably split off from genotype F within the new world (Arauz−Ruiz *et al*., 2002).

Morphological Characteristics of Hepatitis B Virus

Hepatitis b virus consist of three types of particles; double shelled particles with diameter of 42 nm (Dane particle), spheres of about 20nm diameter, usually present in a 10,000 to 100,000 fold

excess over Dane particles, and relative to complete virus that are smaller quantity of filaments of 20 nm diameter and variable length often measuring about 200nm (Emechebe *et al*., 2009). The Dane particle contains an envelope riched in hepatitis B surface protein. The envelope surrounds the inner nucleocapsid which is made up of 180 hepatitis B core proteins arranged in an icosahedral pattern. The nucleocapsid also contain at least one hepatitis B polymerase protein (p) along with hepatitis B virus genome .the sphere contain both middle and smaller hepatitis surface protein, whereas the filament also include large hepatitis b protein. The absence of the hepatitis B core, polymerase and genome cause the particle to have a non-infectious nature (Jawazt *et al*., 2013).

The nucleocapsid contains pores that allow the diffusion of nucleotides during the synthesis of the DNA genome. The C-terminal amino acids of the core protein play a role in the packaging of the pregenome –polymerase complex within the nucleocapsid (Bruss, 2007). The DNA genome is composed of between 3182-3248 nucleotides depending on the genotype. The genome consist of a complete minus (-) DNA strand with a short terminal and a shorter plus (+)DNA strand that leaves a single stranded gap of variable length , in mature nucleocapsids and released viruses. Base pairing of plus- and minus- cohesive overlap region of the genome maintain the circular configuration. The 5 end of the minus strand is linked to the N- terminal portion of he viral polymerase. At its 5 end of the RNA pregenome and serve as a primer for plus- strand –DNA synthesis (Bruss, 2007).

Hepatitis B Virus Genome

The genome of hepatitis B virus is made up of circular DNA that is partially double stranded, that form a covalently close circle with 5end of the full length minus strand which is linked to the viral DNA polymerase. The genome is 3020-3320nucleotide long (Sagar, 2015). The negative sense non coding is complementary to the viral mRNA. The viral DNA is rendered fully double stranded by the completion of the (+) sense strand and a short sequence of RNA from the (+) sense strand. Non coding base are removed from the end of the (-) sense strand and the end are rejoined. There are four genes encoded by replication is preceded by an upstream in frame AUG start codon for which the pre-core protein is produced. HBeAg is produced by proteolytic processing of the pre-core protein. The DNA polymerase is encoded by P gene. Gene S is the gene that code for the surface antigen (HBsAg). The HBsAg gene is one long open reading frame but contain three in frame “start” (ATG) codons that divide the gene into three sections, pre-S1, pre-S2 and S. because of the multiple start codons polypeptides of three different sizes called large, middle and small (pre-S1+ pre-S2+S, pre-S2+S or S) are produced (Beck *et al*., 2007). The function of the protein coded for by gene X is not fully understood but it has been suggested to be associated with the development of liver cancer (Field *et al*., 1995). It stimulates genes that promote cell growth and inactivates growth regulating molecules (Li *et al*., 2010).

Life Cycle/Replication of Hepatitis B Virus

In order to replicate, the HBV, most first attach onto a cell which is capable of supporting it replication, although hepatocyte are known to be the most effective cell type for replication of hepatitis B virus, other cell types in the human body are also found capable of supporting its replication but to a lesser degree(Garces *et al*., 2002).

The virion attach to the liver cell membrane by cell associated heparin sulfate proteoglycan, the viral particle bind specifically to an unknown hepatocyte specific preS1-receptor. The DNA is then injected/released into the host cell by endocytosis and direct fusion of the viral envelope with the plasma membrane. To release/uncoating into the cytoplasn and transport of the nucleocapsid t the nucleus, the partially double stranded viral relaxed DNA (rcDNA) is repaired by the viral polymerase and in another step the viral polymerase and RNA –primers used for DNA plus- strand synthesis are removed by circular enzyme. Eventually, covalently used circular DNA (cccDNA) is formed by covalent ligation of both DNA strands (Daniel *et al*., 2011). The cccDNA is crucial in the persistence of Hepatitis B virus infection. Viral ccc DNA serves as template for RNA synthesis. All viral RNA are transcribed from the ccc DNA using the cellular transcriptional mechanism (Flint *et al*., 2002).

The viral DNA contain four major reading frames, which are: Pre core/core genes coding for the nucleocapsid protein and for the secreted non-structural pre core protein, the HBeAg; The polymerase gene coding for the reverse transcriptase, RNase and terminal protein domains; The pre S1/L- pre S2/m- and surface/S-gene, coding for three envelope proteins and The X gene coding for regulatory X-protein (Daniel *et al*., 2011).

Diseases of Hepatitis B Virus

Acute Hepatitis B: it is ca condition in which when a person is exposed to the virus begin to develop the sign and symptoms of the viral hepatitis, which may be asymptomatic or result in varying degree of acute liver injury. Although such conditions can be severe, vast majority of adults will resolve the primary infection. This period of time is called the incubation period, is an average of 90 days, but could be shorter as 45 days and as long as 6 months (Bernard *et al*., 1995).

Chronic Hepatitis B: Is a major stage in the viral infection and occurs when a person has acute infection but is unable to resolve the infection on its own. The disease can be chronic or completely resolved depending on the age of the infected person; about 90% of infants infected at birth will progress to chronic disease (Straus *et al*., 2002). However, as a person ages, the risk of chronic infection decreases such that between 20%-50% of children or less than 10% of older children or adults will progress from acute to chronic infection (Terrautt *et al*., 2005).

Clinical Sign and Symptoms

Most people with acute viral hepatitis do not experience any symptoms. However, some people have acute illness with symptoms that last for several weeks, including yellowing of the eye and skin (Jaundice), dark urine, extreme fatique, nausea, vomiting and abdominal pain. A small subset of people with acute hepatitis B can develop acute liver failure, which can lead to death (WHO, 2018).
Chronic infection with Hepatitis B virus may either be asymptomatic or may be associated with chronic liver inflammation (chronic hepatitis), leading to cirrhosis after several years. This type of infection leads to increase in incidence of hepatocellular carcinoma (liver cancer). Clinical sign and symptoms may be related to the stage in which the disease is currently operating. Acute or chronic, the general sign and symptoms of Hepatitis B virus infection include; fever, fatique, nausea, vomiting, jaundice and hepatomegaly, loss of appetite, dark urine, abdominal pain and joint pain (Jawetz *et al*., 2013).

Epidemiology of Hepatitis B Virus

The world health organization has estimated that there are more than 2 billion Hepatitis B virus infected people and about 378 million chronic carriers worldwide (Elisabetha *et al*., 2012). It was estimated that more than 50% of liver cancer were attributed to Hepatitis B infection. The prevalence of HBV infection, according to the geographical area may be high (8%), intermediate (2-7%) or low (<2%) (Maddrey, 2000).

Hepatitis B is highly endemic in developing countries with large populations such as South east Asia, China, Sub-Saharan Africa, and the Amazon basin (Sharma *et al*., 2005),where at least 8.0% of the population were HBV chronic carriers (Alter, 2003). Chronic infection with HBV occurs in 90% of infants infected at birth, 30% of children infected at 1-5 years and 6% of persons infected above 5 years (CDC, 2013). The prevalence of 11% was reported in Uganda among medical students in Central Africa (Bongomin *et al*., 2005).

In sierra Leone in 20018, Rashid *et al*., found out that HBV is highly endemic with a percentage of 13.7% in Bo. A much lower prevalence of 5.3% was found among secondary school in Abidjan (Outattara *et al*., 2019).

Transmission

The hepatitis B Virus is highly contagious and is present in blood and body fluids, including semen and vaginal secretions. The saliva of people with hepatitis B may contain evidence of the virus but in such small concentration that kissing does not spread HBV 2018). The virus is transmitted when blood, semen or another body fluid from an infected person enters the body of another individual. Because the virus is extremely infectious-50 to 100 times more infectious

than HIV – even brief, direct contact could be enough to cause infection (Charles, 2018). The hepatitis B virus can survive outside the body for at least 7 days. During this time, the virus can still cause infection if it enters the body of a person who is not protected by the vaccine. The incubation period of the hepatitis B virus is 75 days on average, but can vary from 30 to 180 days. The virus may be detected within 30 to 60 days after infection and can persist and develop into chronic hepatitis B (Charles, 2018).

In highly endemic areas, hepatitis B is most commonly spread from mother to child at birth (perinatal transmission), or through horizontal transmission (exposure to infected blood), especially from an infected child to an uninfected child during the first 5 years of life. The development of chronic infection is very common in infants infected from their mothers or before the age of 5 years.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 (WHO, 2018). Toothbrush, towel and bed sharing are also risk factors for HBV transmission (Atmore C, *et al*., 1989).

Diagnosis of Hepatitis B Viral Infection

It is not possible, on clinical grounds to differentiate hepatitis B from hepatitis cause by other viral agents and hence, laboratory confirmation of the diagnosis is essential. A number of blood test are available for the diagnosis and to monitor people with hepatitis B. they can be use to distinguish acute and chronic infection. Laboratory diagnosis of hepatitis B infection focuses on the detection of HBsAg. Acute HBV infection is characterized by the presence of HBsAg and immunoglobulin M (IgM) antibody to the core antigen, HBcAg. During the initial phase of the infection, patients who are also seropositive for HBeAg. HBeAg is usually a marker of high level replication of the virus. The presence of HBeAg indicates that the blood and body fluid of the infected individual is highly infectious. Chronic infection is characterized by the persistence of HBsAg for at least 6 months (with or without concurrent HBeAg). Persistence of HBsAg is the principal marker of risk for developing chronic liver disease (hepatocellular carcinoma) later in life (WHO, 2018).

Clinical presentation/impression can lead the clinician request for investigations in the laboratory. Elevated liver enzyme in the blood is an indication of hepatocyte damage. Serologic markers in patient with HBV can reveal the following parameters:

The HBsAg is a general indicator of hepatitis viral infection, HBsAb is an indicator of recovery or immunity to Hepatitis B viral infection, the Anti-HBcIgG indicates past or chronic infection, Anti-HBcIgM is the marker for acute infection, HBeAg indicates an active replication of the viral DNA and it is also a marker of infectiveness of the virus and Anti-HBeAb is an indicator for period of no replication, Detection of DNA by polymerase chain reaction (PCR), Indicates active replication (Liang, 2009).

Treatment of Hepatitis B viral infection

Clinically, the antiviral therapy should improve the quality of life and survival of chronically HBV infected patients by preventing disease progression to cirrhosis, decompensate cirrhosis, end-stage liver disease, HCC, and death. Current therapies aim for a sustained suppression of viral replication that usually results in a reduced histological activity of chronic hepatitis and biochemical remission. Consequently, the risk of progression to cirrhosis decreases together with the incidence of HCC in non-cirrhotic and to a lesser extent also in cirrhotic patients (Daniel *et al*., 2011). The end point of HBV therapy differs between patient groups. The European Association for the Study of the Liver (EASL) in 2008 distinguishes between three serological constellations:

1. In HBeAg positive and HBeAg negative patients, the ideal end point is sustained HBsAg loss with or without seroconversion to anti-HBs.
2. In HBeAg positive patients, durable seroconversion to anti-HBe is a satisfactory end point.
3. In HBeAg positive patients who do not achieve an anti-HBe seroconversion, a maintained undetectable HBV DNA level on treatment with NUCs or a sustained undetectable HBV DNA level after interferon-α (IFN-α) therapy is the next most desirable end point.

In theory, the ideal goal of antiviral therapy for chronically HBV infected patients would be a complete HBV elimination including HBsAg loss and seroconversion to anti-HBs and complete eradication of cccDNA from infected hepatocytes. However, the elimination of viral cccDNA from the nucleus of infected hepatocytes cannot be achieved by any of the currently available drugs. In line with this, a leading hypothesis is that the clinical resolution of HBV infection does not necessarily require the complete eradication of the virus from the liver. Rather, control of viral replication by the host's immune system is thought to play a crucial role (Daniel *et al*., 2011).

Prevention and control of Hepatitis B virus

The prevention and control of hepatitis B virus is considered into two dimensions Viz: The primary and secondary prevention activities. Primary prevention activities include; creating awareness of hepatitis B viral infection, vaccination; 1^{st}, 2^{nd} and3^{rd} dosage, using attenuated virus and making sure its successful, implementation of blood safety strategies, safety precaution in health care setting and or facilities. The secondary activities are meant for those that are already

infected with the disease and trying to prevent progression of the disease to cirrhosis and hepatocellular carcinoma. Avoidance of all the risk factors is one of the best way to prevent the infection Viz; use of condom during sex, avoid sharing of needles, razor, toothbrushes and other sharps with other people of unknown health status, use of latex or plastic gloves when handling blood, fluids from people of unknown medical history (CDC, 2017). Hepatitis B treatment is relatively expensive and challenging, some people with chronic hepatitis B do not respond well to treatment at all. Hepatitis B do not generally spread through food and water, sharing eating utensils, breast feeding, hugging, kissing, hand holding, cough or sneezing (CDC, 2016, CDC, 2018).

Vaccination

Several vaccines have been developed for the prevention of hepatitis B viral infection. They rely on the use of viral envelope proteins (HBsAg). The vaccine was originally prepare from plasma obtained from people who had a long standing infection. However, currently it is made using synthetic recombinant DNA technology that does not contain blood products, one cannot be infected with hepatitis B from this vaccine. Following vaccination, hepatitis B surface antigen may be detected in serum for several days; this is known as vaccine antigenaemia (Martin-Ancell *et al*., 2004).

The vaccine is administered in two, three, or four dose schedule, into infants and adults, which provide protection for 85-90% of individuals. Protection have been observed to last for 12 years in individuals who show adequate initial response to the primary course of vaccination, and that immunity is predicted to last at least 25 years (Vandamme *et al*., 2007).

Hepatitis B vaccine is the most effective way to prevent the infection. The vaccine is up to 95% effective against the hepatitis B virus if all the 3rd or 4rd shorts of the vaccine are taken. These vaccines provide protection against the virus for at least 20 years. There are times that post vaccination testing is ordered to ensure that immunity is fully developed against the virus. It was normally given to those who have impaired immunity or immune system, health workers, and sex partners of those who have chronic infection (CDC, 2017).

MATERIAL AND METHODOLOGY

Study Area

The study was carried out among students of Nasarawa State University. It has a total area of. It is located at the Keffi Town, Abuja Road, Nasarawa State in Nigeria. Whereas Lafia is the capital of Nasarawa State but Keffi is the second largest city in the state.

Study Population

No age or gender restriction was imposed, both male and female were included and treated alike, youth and adults were well defined as those aged 18-30 years while adults as those age 30 years and above. Basic demography, medical history and history of blood transfusion were documented using questionnaires for each individual.

Study Design

This was a cross sectional study aimed at determining the seroprevalence and an indicator of hepatitis B viral infection. Sample were collected using random sampling techniques. A questionnaire was used to capture some socio demographic and clinical information of the eligible subjects. The structured questionnaire was administered to only consented subjects to gain their demographic data and clinical information of male and female eligible subjects of the various age groups.

Sample Size/Statistical Analysis

The Thrusfield formula described in 1997 was used to obtained the sample size. This was calculated as follows:

$$N = \frac{(1.96)^2 \text{ X pexp } (1 - \text{pexp})}{d^2}$$

Where
N = Number of sample
Pexp = Expected prevalence rate
d = desired absolute precision of 5%

Expected prevalence of 11.5% (Joanah *et al*., 2016)

$$N = \frac{(1.96)^2 \text{ X } 0.115(1 - 0.115)}{(0.05)^2}$$

$$N = \frac{3.842 \text{ X } 0.115(0.884)}{0.0025}$$

$$N = \frac{0.39057772}{0.0025}$$

N = 156.2
N = 156
The minimum sample size is 156 participants, but 10% attrition rate was added to give 172 as the number of samples that was collected.

Sample Collection and Processing

Three milliliters (3ml) of blood sample was collected from each participant into an EDTA container by venipuncture using a sterile syringe and needle. Each sample was properly labeled with the number corresponding to the number assigned to consenting participant. The intended participant was interviewed to obtain information on their demographic data. The samples collected were separated by centrifugation at 2000rpm for five minutes to obtain plasma. The separated plasma samples were then aliquated and store in refrigerator at -20^0c until ready for use.

Detection of HBV SERO Markers

Royal Care One Step Strip Style HBSAG Rapid Test Kit

(Immunochromatography)

Principle of the assay: The HBsAg one step surface antigen test strip (whole blood) is a qualitative lateral flow immunoassay based on the principle of sandwich immunoassay for the determination of HBsAg in whole blood.

Assay Procedure

The sealed pouch was opened by tearing along the notch to remove the strip, which was laid on a flat, clean, dry, nonabsorbent surface, and a drop of whole blood was applied into the sample pad. Then two drops of the buffer was applied into the sample pad as well, after which the result was read within 10-20 minutes, as the result becomes invalid after 30 minutes.

Principle and Procedure Hbv5-In 1 Markers Rapid Test Panel (Colloidial Gold Chromatography)

Principle of assay: the specimen mixing up Colloidal-Gold monoclonal antibody move along the membrane of the T-line, and from the T-line when the human serum/plasma and whole blood contains HBsAg, HBsAb, HBeAg, according to the principle of double antibody sandwich method and Gold immunochromatography assay, which is a positive result. Unreacted markers move forward continually to combine with anti-mouse antibody and form the control line. If the test line does not appear, it is a negative result.

Assay Procedure: The reagent was kept at room temperature for at least 30 minutes before the commencement of the test to return to the room temperature (20˚c).

The outer packing was taken off, and cassette put on to the desk with the sample window up. Two drops of plasma (50ul) was drop vertically into the circular groove of the cassette, and 1 drop of buffer (50ul) was added into the circular groove of the cassette. Test result was observed immediately within 15 minutes, the result is invalid over 20 minutes.

Data analysis

The result obtained from the sample analyses were subjected to statistical analysis using statistical package for social science (SPSS) software version 21 (USA) in order to determine the level of significance in infection based on considered variables. General descriptive analysis was used to analyze variables and results were expressed as percentages. The Chi square test was used to compare categorical data at 95% confidence interval, significance level was taken at $P<0.05$.

4.0 ANALYSIS OF RESULT

Out of the 168 samples examined, 12(7.1%) prevalence was obtained, and 69 were male with 8 (4.7%) while female were 99 with 4 (2.4%). $X^2=3.498$; P=0.061 (Table 1).

Age group 18-30 years recorded the highest prevalence of 9 (7.3%), and age group 30-above years, 3(6.7%). $X^2=0.021$; P=0.885 (Table 2).

In term of sex history, those with previous sex history have higher prevalence of 3 (14.3%), even though they were few but compared with 9 (6.1%) of those without any previous sex history. $X^2=1.846$; P=0.174 (Table 3).

Prevalence of HBsAg in relation to exposure to sharps shows that 10 (7.3%) had ever been exposed to sharps while 2 (6.2%) have never been exposed to sharps. $X^2=0.027$; P=0.869 (Table 3).

Distribution of HBsAg in relation to alcohol consumption indicate that 3(13.6%) take alcohol while 9(6.2) don't drink alcohol. $X^2=1.609$; P=0.05 (Table 3).

The prevalence of HBsAg in relation to history of blood transfusion shows 12(7.7) had never been hospitalized while non, 0 (0.0) had ever been transfused. $X^2=0.994$; P=0.319 (Table 3).

Table 3 shows a distribution of 12(7.4%) of those who had never been screened for the infection while none of those ever screened is positive for HBsAg 0 (0.0). $x^2=0.396$ P=0.529.

Prevalence of HBsAg in relation to those sharing toiletries shows that, people with history of sharing toiletries had a lower prevalence of 6(6.9%) compared to 6(7.4%) of those who had never share toiletries. $X^2=0.017$; P=0.898 (Table 3).

Table 1; Distribution of HBsAg among Nasarawa State University, in relation to Gender

Gender	No. Examined	HBsAg Rapid test. No. Positive (%)	Combo rapid test (%)	X^2	df	P-value
Male	69	8 (11.6)	8 (11.6)	3.498	1	0.061
Female	99	95 (4.0)	4 (4.0)			
Total	168	12(7.1)	12 (7.1)			

Table2; Distribution of HBsAg among Students of Nasarawa State University, in relation to Age group

Age group (years)	No. Examined	HBsAg Rapid test No. Pos. (%)	Combo Rapid test (%)	X^2	df	P-value
18 – 30 yrs	45	3(6.7)	3 (6.7)	0.021	1	0.885
31 – above yrs	123	9(7.3)	9 (7.3)			
Total	168	12(7.1)	12 (7.1)			

Table 3. Prevalence of HBV in relation to some risk factors

Risk factor	No. examined	HBsAg Rapid Test No.Pos. (%)	Combo rapid Test (%)	X^2	DF	P-Value	Odd ratio
Previous Sex History							
Yes	21	3(14.3)	3(14.3)	1.846	1	0.174	0.371
No	147	9(6.1)	9(6.1)				
History of exposure to sharp object							
Yes	137	10(7.3)	10(7.3)	0.027	1	0.869	0.876
No	31	2(6.5)	2(6.5)				
Alcohol consumption							
Yes	22	3(13.6)	3(13.6)	1.609	1	0.205	0.416
No	146	9(6.2)	9(6.2)				
History of blood transfusion							
Yes	12	0(0.0)	0(0.0)	0.994	1	0.319	1.00
No	156	12(7.7)	12(7.7)				
Previous HBsAg Screening							
Yes							

No	5	0(0.00)	0.00	0.396	1	0.529	1.00
	163	12(7.4)	12(7.4)				
Sharing of toilets							
Yes	87	6(0.9)	6(6.9)	0.017	1	0.898	1.00
No	81	6(7.4)	6(7.4)				

X^2= Chi-square test

DF= Degree of Freedom

5.0 DISCUSSION

The 7.1% seroprevalence of Hepatitis B Virus obtained in this study is in tandem with 9.2% reported by Isa *et al*. (2015) among students of Ahmadu Bello University, Zaria. This study is higher than 1.2% reported by Joanah *et al*. 2016 among school children in calabar, Nigeria and 5.3% obtained by Ouattara *et al*., (2019) in a Secondary School in Abidjan. It is however lower compared to the 16.7% reported by Odinachi *et al*., (2014), among newly admitted students of University of Jos, Nigeria, 31.5% as reported by Tula *et al*. (2015) among healthy Students of Federal Polytehnic Mubi, 11.0% as reported by Bongomin and Magid (2005) among Makerere University students Uganda, 11.4% as reported by Mustafa *et al*. (2015) among students attending University of Maiduguri clinic, Borno, Nigeria and 12.5% was reported by Aminu *et al*. (2013) among healthy students of ABU Zaria.

Based on the result of the study, the prevalence of HBsAg in relation to gender showed that males (11.6%) were more infected than females (4.0%), despite the high number of females that were examined when compared to their males counterpart. This could be as a result of multiple sex partners and promiscuity mostly occurring in males than in their female counterparts as reported by United Nations in Nigeria (2001). There was no significant association between HBsAg distributions with gender, (P=0.061).

The distribution of HBsAg in respect to age showed that, age group 31-above years had the highest prevalence of 7.3% then followed by the age group 18-30 years with 6.7%. This could be as a result of lifestyle, youthful exuberances which increases their risk of exposure, such as sexual activities, sharing of contaminated sharp objects for fashion. According to WHO, the age

at which an individual acquires the infection is the major determinant of the incidence and prevalence rate (WHO, 2015).

The prevalence HBsAg in relation to sex history showed that those with a previous sexual history had the highest prevalence of 14.3% as against those that had no previous sexual history with 6.1% prevalence. The prevalence rate of Hepatitis B Viral infection observed among those with pervious sex history could be attributed to unprotected sexual activities which might have exposed them to the infection as compared against those who had no previous sex history had lower prevalence despite the fact that they are more in number.

The prevalence rate in relation to the use of sharps indicated that those who had history of injuries sustained from sharps recorded the highest prevalence of 7.3% compared to those who have never been exposed to sharp objects who recorded a lower prevalence of 6.5%. This could be that the objects exposed to could have been pre-contaminated with the virus.

The prevalence of HBsAg based on alcohol consumption; showed, the highest prevalence of 13.6% for those who are into alcohol consumption as against 6.2% for those who do not take alcohol. These can be due to the effect of alcohol on the behavior of a person that lead to some abnormal behaviours such as fighting, gangsterism and sexual promiscuity which can predisposed them to the infection.

The distribution of HBsAg in regards to history of blood transfusion indicated that those who had a history of blood transfusion recorded no evidence of HBsAg, this could be due to guidelines for safe transfusion of blood and blood products, as against those who had never been transfused who recorded a high prevalence of 7.7%. The high prevalence among those with no history of transmission indicated that they might have been infected through other risk factors.

The prevalence based on the history of sharing of toiletries showed a slight variation as those who share toiletries had a prevalence of 6.9% as against those who do not share toiletries (7.4%).

A comparative study of the work to other studies within the country showed a prevalence of 31.5% among Students of Federal Polytechnic Mubi (Tulsa *et al*., 2015), 9.2% among Students of Ahmadu Bello University, Zaria (Isa *et al*., 2015), a prevalence of 18.4% was reported by Ndako *et al*., (2011) among secondary school Students in North-central Nigeria. Odusanya *et al*., 2007, reported a prevalence of 3.2% among medical students in the South-western region of the country. Ugwuja *et al*., (2008), reported a prevalence of 3.9% among secondary school students in Abakaliki. A prevalence of 12% was reported by Edia-Asuke *et al*. (2015) among public tertiary institution in Kaduna State, Nigeria.

This variation is possibly due to increase awareness and vaccination against the viral infection. It could also be attributed to differences in the study selection. This study was carried out among apparently healthy school children, whereas others were carried out among patients and other groups.

Considering the result of the positive cases, 12 samples were HBsAg positive, which affirmed a viral infection, but 11 samples were HBeAg positive. This is an indicator of the active replication of the DNA of the virus and is a mark of the infectiveness of the virus (Liang, 2009).

Conclusion

The result of this research work bring to limelight the endemicity of HBsAg among the students of Nasarawa State University. This may not be unconnected to low level of awareness on the route of transmission, youthful exuberances, prevention and control of the viral infection as well as poor vaccination coverage among the age group.

The study also show that majority of the study population were unaware of their HBsAg status. This study has provided additional information on the burden of HBV infection to the existing data in Keffi, Nasarawa State, Nigeria.

Vaccination and prevention of infection still remain the hallmark of activities in the prevention of the transmission of the infection. However, adolescents are usually not targeted for vaccination programmes and coupled with their high risk behaviour such as lack of awareness, increase unprotected sexual activities, sharing of razor blades, tattooing e t c.

Recommendations

Based on the findings of the study, there is need for intensive public sensitization campaign on the routes of transmission, prevention and control of this silent killer disease among students of this school and environs. Thus, the Nasarawa State Government through the Ministry of Health should consider wide vaccination coverage and treatment that will include students in schools, so as to curtail the vast spread of the virus among others due to close personal contact. There should also be special programme as regards to this disease to monitor the success and implementation of measures aimed at achieving a maximum success in the prevention, control, management and treatment of the infection just as in the case if HIV.

Since when infected it is hard to get rid of the virus completely from the body, I therefore recommend that more researchers should develop interest in this research area to explore more ways the virus can be managed and possibly be eradicated.

The government and the NGOs should create more awareness on the ways of prevention and control of the viral infection. They can also subsidies the cost of treatment to these individuals.

Individuals should be advice to go for the screening to know their status; this will greatly reduce the mortality and other medical complications caused by this virus.

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