

Prevalence of Hepatitis B Virus in Nigeria: Review Update

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Abstract

Hepatitis B virus is among the common viral infectious agents of public health concern. An estimated two billion people are infected worldwide with approximately 350 million others suffering the chronic form of the disease. Nigeria, a tropical country, has been documented as highly endemic for HBV infection and about 75% of its population is likely to have been exposed to the virus at one time or the other in their lives. Currently about 18 million Nigerians are infected. A prevalence rate of 4.3 % to 23.3% have been reported from different part of the country This paper presented up-date prevalence of hepatitis B virus from different part of Nigeria. The structure, mode of transmission, replication, pathogenesis, diagnosis, prevention and possible treatment of the disease were also mentioned.

Keywords: Hepatitis B; Prevalence; Nigeria; Infections; Blood

Introduction

Hepatitis B virus is among the common viral infectious agents of public health importance globally. An estimated two billion people are infected worldwide with approximately 350 million others suffering the chronic form of the disease [1,2] In Africa, more than 50 million people are chronically infected, with mortality risk of about 25%. The carrier rates of the virus in Sub-Sahara Africa range from 9% - 20% [3]. Hepatitis B virus (HBV) infection is a serious health problem worldwide. Once chronic infection is established, HBV may persist in the liver for lifetime [4], which not only causes severe HBV-related sequelae such as cirrhosis and hepatocellular carcinoma but also constitutes the reservoir of the virus [5].

The spectrum of the symptoms of HBV disease varies from sub-clinical hepatitis to icteric, hyperacute, acute and subacute hepatitis

during the primo-infection phase and from an asymptomatic carrier state to chronic hepatic cirrhosis and hepatocellular carcinoma during the chronic phase. In the acute phase, the incubation period is 1-6 months [6]. Anicteric hepatitis is a predominant form of expression for this disease, at this phase most of the patients are asymptomatic. Patients with anicteric hepatitis have a greater tendency to develop chronic hepatitis. Icteric Hepatitis B is associated with a prodromal period, during which a serum sickness-like syndrome can occur [7].

The predominant routes of transmission is commonly through blood transfusion, blood products, body fluids (urine, semen, sweat, saliva, and tears), use of contaminated needles, vertical transmission (mother to child through infected birth canal), and

sexual contact [8]. Neonates born of chronically infected mothers have a 70–90% risk of the infection progressing to a chronic phase [9]. The demand for safe blood or blood products in life-saving interventions is critical to avoiding non curable infectious diseases [3]. Blood transfusions carry the risk of transfusion-transmitted infections such as hepatitis B. In order to measure their severity, the World Health Organization has recommended a pre-transfusion blood test. The residual risk of infection from HBV is higher than that of hepatitis C virus (HCV) in non-endemic countries [10].

Nigeria, a tropical country, has been documented as highly endemic for HBV infection and about 75% of its population is likely to have been exposed to the virus at one time or the other in their lives [11]. Currently about 18 million Nigerians are infected [12]. A prevalence rate of 4.3 % was reported from Port Harcourt [13], 5.7% from Ilorin [14], 11.6% from Maiduguri [15] and 8.3% from Zaria [16] A seroprevalence of 23.3% was reported among patients attending all clinics at the Aminu Kano Teaching Hospital (AKTH) [17].

Historical Background of HBV

The hepatitis B virus was discovered in 1965 when Blumberg and co-workers found the hepatitis B surface antigen which was originally called the Australia antigen because it was found in serum from an Australian patient [18]. Dr Baruch Samuel Blumberg was awarded the 1976 Noble Prize in Physiology or Medicine for this discovery. The virus was fully described in the 1970s [19]. In recent times, the rapid and continuous discoveries of the viral disease around the whole world have improved our understanding of the complexity of this unusual virus. Although there has not been any substantial decrease in the overall prevalence of HBV, there is the hope that the next generation will see a decline in both the worldwide carrier rate and the incidence of new HBV infections if current HBV vaccinations are intensified [20].

Transmission

The HBI can be transmitted by the same modes as with the human immunodeficiency virus (HIV), even though the HBV is hardier and 50-100 times more infectious than the HIV (WHO, 2008). Unlike HIV, the virus can survive outside the body for at least 7 days. During that time, the virus can still cause infection if it enters into the body of a person who is not infected. Transmission of hepatitis B virus results from exposure to infectious blood or body fluids. Possible modes of transmission include but are not limited to unprotected sexual blood transfusions, re-use of contaminated needles and syringes, and vertical transmission from mother to child during childbirth. Without intervention, a mother who is positive for HBsAg confers a 20% risk of passing the infection to her offspring at the time of birth (WHO, 2008).

This risk is as high as 90% if the mother is also positive for HBeAg. The HBV infection can be transmitted between family members within households, possibly by contact of non-intact skin or mucous membrane with secretions or saliva containing HBV (Petersen et al., 1976). However, at least 30% of reported

hepatitis B among adults cannot be associated with an identifiable risk factor (Shapiro, 1993). In many developed countries (e.g. those in Western Europe and North America), patterns of transmission are different from those mentioned above. Today, most infections in these countries are transmitted during young adulthood by sexual activity and injecting drug use. HBV is a major infectious occupational hazard of health workers (WHO, 2008). HBV is not spread by contaminated food or water and cannot be spread casually in the workplace. The virus incubation period is 90 days on average but can vary from about 30 to 180 days (AASLD, 2007). HBV may be detected 30 to 60 days after infection and persist for widely variable periods of time.

Stages of HBV Infection

Remarkable progress has been made in the understanding of the three (3) main natural stages of the HBV infection in hosts: acute infection, chronic asymptomatic and chronic symptomatic stages (AASLD, 2007). However, not all HBV-infected patients go through all the three stages. The risk to develop liver-related complications, such as cirrhosis and hepatocellular carcinomas increases as patient progresses from acute to chronic stage of the infection. Indeed, most HBV infections end up at the acute stage (~ 90%) with a few progressing on to the chronic stage.

Acute HBV infection

This is the initial stage of the infection and every HBV- infected patient goes through this, even though not all patients transit beyond this stage. Early phases of this stage of the infection are characterized serologically by the presence of HBsAg, high serum HBV DNA, HBeAg, and normal level of serum aminotransferase level (ALT), and minimal or insignificant inflammation on liver biopsy [21]. A later phase, also called immunity phase, is marked by increased serum titres of anti-HBsAg IgG (HBsAb), anti-HBcAg IgG, lowered or disappearance of HBsAg and HBV DNA, normal liver histology. This is true for those who recover fully from the infection after attaining full and permanent immunity through exposure. The duration of either phase differs among patients but generally lasts between 5-8 months (AASLD, 2007). However, those patients who fail to mobilize adequate immune response factors to combat the infection end up with the fate of living with the disease their entire lifetime. In this case, it is said the disease has become chronic. The physical signs and symptoms, such as jaundice, fever, dark-urine formation, nausea, among others, would occur, even though they will last shortly after which they get resolved following recovery. Generally, transition from the acute stage to the chronic stage depends on several factors including age, gender, viral genotype, and host immune competence.

Chronic HBV infection

This occurs as a progression of the early phase of the acute HBV infection due to the host's failure to mount the necessary immune stimulus to ensure total viral clearance and consequent resolution of the disease. It is serologically marked by relative rise in serum anti- HBcAg IgG, disappearance or lower titres of anti-HBsAg

IgG, and either normal or significant liver damage as shown by ultrasonography (WHO, 2008). Also, this stage of the disease may be characterized by normal or elevated serum aminotransferase levels (aspartate amino transferase (AST) and alanine amino transferase (ALT)) and other markers of hepatic integrity (AASL, 2007).

The serological presence of HBeAg is real in all stages of the disease. The presence of this antigen together with elevated viral load (HBV DNA > 10³ copies/ml) and higher ALT (> 60 IU/l) is a strong indication of viral activity, replication, and infectivity (WHO, 2008). Patients with such manifestations are put on retroviral. A key event in the natural history of HBeAg – positive CHB patients is HBeAg seroconversion (Sharma et al., 2005). It is believed that seroconversion of HBeAg to HBeAb is accompanied with cessation of HBV replication and remission of liver disease. Several studies have shown that seroconversion with a marked reduction in HBV replication is associated with biochemical and histological remission of inflammatory activity in the majority of patients [22].

Prevalence of Hepatitis B Virus in Nigeria

Prevalence of hepatitis B virus among blood donors

Mosley et al., suggested that anti-HBc screening of blood donations might prevent HBV transmission from HBsAg-negative blood donors that are positive for anti- HBc [23] The prevalence of OHB varies significantly between geographical regions as well as among various patient populations tested. Recent Evaluation of hepatitis B virus sero-positivity among 300 voluntary blood donors at a centralized blood service center in Nigeria by [24] revealed that Thirty-three (13.8%) of first-time donors were positive for hepatitis B markers while all retained donors were sero-negative. There were 32 (13.3%) sero- positive reactions to HBsAg and 3 (1.3%) reacted to HBeAg. In another study in Jos, Uneke and others reported a 14.3% HBsAg Seropositivity among their blood donors against a higher 25.9% among patients infected with HIV. They also noted higher infection rate of 44% in donors 51-60 years and 28% frequency within the age bracket of 31-40 years [25,26]. while studying Seroprevalence of hepatitis B e antigen (HBe antigen) and B core antibodies among hepatitis B surface antigen positive blood donors at a Tertiary Centre in Nigeria found a seroprevalence of 8.2% (22 of 267) HBeAg, 4 of 267 (1.5%) were indeterminate while 241 (90.3%) of their subjects tested negative. Only 27 out of 267 donors (10.1%) tested positive to IgM anti-HBcore, 234(87.6%) tested negative, while 6(2.2%) were indeterminate. A higher percentage of 60.7% (162 of 267) tested positive to IgG anti-HBcore, while 39.3% (105 of 267) tested negative. They concluded that there is a low seroprevalence rate of HBeAg-positive chronic hepatitis and relatively high IgG anti-HBcore and IgM anti-HBcore rates in South West Nigeria [26].

Another study among blood donors, in North Central Nigeria, at the Bishop Murray Medical Centre in Makurdi, age group prevalence of HBV was reported at 11.90%, 13.05% and 6.53% within the age ranges of 18-22, 23-27 and 28-32 years respectively [27]. Jeremiah and others reported a prevalence of 8.6% HBsAg in Maiduguri, Northeast Nigeria with anti HBc IgM in 18.4% suggesting that

donors negative for HBsAg are not necessarily uninfected with HBV and recommended the mandatory screening of HBC in donor blood [28].

In Southwest Nigeria Salawu and others reported the occurrence of other HBV markers in HBsAg negative blood donors and recommended the inclusion of routine testing of markers such as antibody to hepatitis B core (HBC) antigen in donor blood before transfusion [29]. Japhet and his co-workers found an overall prevalence of transfusion transmissible infections of 32.6% in their study with 19.6% HBsAg positivity, 13.0% HBC antibody reaction and 8.9% hepatitis B envelop antigen (HBeAg) detection which marks infectivity of the virus and appears in blood after HBsAg [30].

In Benin City of Nigeria, Mutimer and others reported an overall 14% prevalence of TTIS. They concluded that screening of blood routinely may not reduce the incidence of HBV infections (Mutimer et al., 1994) Far in the North Eastern Nigeria, Harry and colleagues reported a high 22.0% HBsAg and 6.64% HBeAg among blood donors. They found only 11.6% and 1.39% of pregnant women subgroup of their study reactive for HBsAg and HBeAg respectively [15].

Prevalence of hepatitis B virus among pregnant women

Adabara et al., 2012 evaluated the Prevalence of Hepatitis B Virus among Women Attending Antenatal Clinic in the General Hospital, Minna, Niger State, there results revealed that Thirteen (6.5%) out of the 200 subjects investigated were found to be positive for hepatitis B infection. On the basis of age, the distribution of HBV infection among the subjects revealed that the age group 20-29 has the highest rate of infection of 10.3% followed in descending order by 40-49 (4.5%), 30-39 (4.2%) and 10-19 (0.0%). The authors linked the prevalence of the virus to low level of awareness and the poor standard of living observed among the subjects [31] carry out a cross-sectional study over a 3-month period (August-October 2009). On Prevalence and pattern of hepatitis B among 480 women attending antenatal clinics in Nnewi, Nigeria was done by simple random sampling using computer generated random numbers. Of these, 40 tested positive to HBsAg, accounting for 8.3% of the sample population. The age of the subjects studied varied from 14 to 45 years (mean age - 24.3 years) while the mean parity was 2.18. The HIV/HBV co-infection rate was 4.2%.

Agarry and Lekwot also evaluated the prevalence of hepatitis B virus surface antigen (HBsAG) and hepatitis C (HCV) antibody amongst 200 pregnant women attending ante-natal clinic in Gwagwalada, Abuja. Of the 200 blood samples tested, 19 (9.5%) and 1 (0.5%) were positive for the presence of hepatitis B and C respectively. No mixed infection of both viruses was observed in the pregnant women tested [32,33]. While studying the seroprevalence of hepatitis B virus (HBsAg) antibodies in pregnant women In Akure, Ondo State found that out of Eight hundred and sixty pregnant women. Only forty (4.7%) were positive while eight hundred and twenty (95.3%) were negative, indicating an overall prevalence of 4.7% [33].

The prevalence of Hepatitis B Virus (HBV) carrier and infectivity status among three hundred (300) pregnant women in Makurdi were evaluated [34]. Maternal HBV infectivity status was determined by testing all HBsAg positive samples for the presence of hepatitis B e antigen (HBeAg). Overall, 33 (11%) pregnant women were identified as carriers of HBV and 10 of the 33 (30.3%) pregnant women identified as HBV carriers tested positive for HBeAg. Hence, 3.3% of the entire study population was found to have high viral replication as well as high risk of transmitting HBV to their neonates.

Prevalence of hepatitis B virus co-infections with other disease

Rescently Ejeliogu [35], evaluated the Prevalence of Hepatitis B Virus Co-infected Nigerian Children (2 months to 15 years) with Human Immunodeficiency Virus. Out of 452 Children that were screened, three hundred and ninety-four (87.2%) were mono-infected with HIV while 58 (12.8%) were co-infected with HIV and HBV (HIV/HBV). Egah et al while studying seropositivity to hepatitis B, C and the human immunodeficiency viruses among clergy men in training, in a seminary in Jos, found a 15.5% hepatitis B surface antigen positive reaction among their subjects who were a low risk blood donor group. They also documented a crude transfusion transmissible infection prevalence of 22.1% and HIV/HBV co-infection rate of 0.4% in their study [36].

In the year 2011 Omalu et al., evaluated the Seroprevalence of Malaria and Hepatitis B (HBsAg) with Associated Risk Factors among Pregnant Women Attending Antenatal Clinic in General Hospital Minna, North-Central Nigeria. Out of the 269 pregnant women screened 216(80.30%) were positive for malaria, 22(8.18%) for hepatitis B and 21(7.81%) were co-infection of malaria and hepatitis B and 10 were negative, while non-pregnant women had 51(51.00%), 8(8.00%) and 6(6.00%) for malaria, hepatitis B and co-infection of both out of 100 screened [37,38] found out that out of 1535 sampled individuals analyzed for Hepatitis B Virus (HBV), 1319 (85.9%) showed a serological evidence of exposure to HBV infection, some through natural infection (22.7%) and others (13.0%) through vaccination; 12% of the exposed were inferred to be currently infected and 91.2% chronically infected. Hepatitis delta virus (HDV) antigen was also detected in 2.7% of the HBsAg positive individuals; and was encountered more (6.7%) in those with acute hepatitis than those with chronic disease.

Jibrin & Mustapha [39], screened, two hundred consecutively recruited HIV-infected individuals comprising 97 males and 103 females for HBsAg using ELISA. A total of Fifty-three of the patients tested positive for HBsAg giving an overall prevalence rate of 26.5% which was significantly higher ($p < 0.001$) than the 10.4% recorded among non-HIV-infected individuals. Co-infection rate in males (24.7%) did not differ significantly from that of females (28.2%). Co-infection was highest in the 40-49 years age group (41.6%), while no case of co-infection was recorded in the ≤ 19 years. Among the different occupational groups businessmen had the highest co-infection rate (44%) followed by long distance drivers (39.5%). In relation to marital status, divorcees/widows had the highest

proportion of those with coinfection (53%) followed by those who were unmarried (32.5%) and those married (21.6%). The authors confirm the high prevalence rate of HBV co-infection in HIV-infected patients compared to the non-HIV- infected population. Therefore, there is a need to screen all HIV-infected patients for HBV infection.

According to Taiwo et al. [40] among patients in Lagos State University Teaching Hospital (LASUTH), Dual presence of HBsAg and anti-HCV was observed in 4(3.9%) of HIV infected patients, while 29(28.4%) and 15(14.7%) were repeatedly reactive for HBsAg and anti-HCV respectively. HIV negative blood donor controls have HBsAg and anti-HCV prevalence of (22) 6.0% and (3) 0.8% respectively. The prevalence of hepatitis co infection is higher among the male study patients 16(50%) than the female 32 (45.7%). Salawu et al. [41] studied the Prevalence and trends of HBsAg, anti-HCV, anti-HIV and VDRL in blood donors in the last three and a half years in a tertiary health care facility in Ile-Ife, Nigeria. The screening records of all blood donors from January 2006 to June 2009 were evaluated with respect to screening outcome for HBsAg, anti-HIV, anti-HCV and VDRL. Of the total 14,500 donors bled, 7.50% were positive for HBsAg, 0.96% for anti-HIV, 0.86% for anti-HCV and 2.61% for VDRL. There was a gradual decline in the prevalence rate of HBsAg from 9.20% in 2006, to 8.37 in 2007 and 6.25% in 2008; with a rise in the first half of 2009 to 6.32%. Similarly, HIV prevalence declined from 1.44% in 2006 to 0.94% in 2007 and 0.66% in 2008 but rose to 0.96% in the first half of 2009. HCV prevalence fluctuated throughout the period under study. Prevalence of syphilis declined from 2.93% in 2008 to 1.92% in 2009.

Bola et al. (2016) evaluated the sero-prevalence of HCV in HIV sero-positive children in Lagos, Nigeria. A total of 132 blood HIV sero-positive children aged 1-15 years were serological assay for HCV. Out of the 132 HIV sero-positive samples, 6 were positive for HCV with a prevalence of 4.54%. Zero prevalence was recorded between age groups 1-3 years while a sero-prevalence of 20% was found among age groups 12-15 years. Ejeliogu et al. [35], evaluated the Prevalence of Hepatitis B Virus Co-infected Nigerian Children (2 months to 15 years) with Human Immunodeficiency Virus. Out of 452 Children that were screened, three hundred and ninety-four (87.2%) were mono-infected with HIV while 58 (12.8%) were co-infected with HIV and HBV (HIV/HBV).

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Similarly, Hamza et al. (2013) evaluated the prevalence of HIV-HBV- patients in Kano State and find out that 54/440 were HB-HIV coinfectd [17], also evaluated the prevalence of HIV-HBV- patients

in Kano State and find out that 211/300 were HB-HIV coinfecting Udeze et al. (2015), evaluated the prevalence rate of HB and C infections among HIV-infected patients accessing healthcare at HIV and AIDS section of University of Ilorin Teaching Hospital, Ilorin, Nigeria. Of the 356 HIV-infected participants, 114 (32.0%) and 14 (3.9%) were respectively positive for HBsAg and anti-HCV antibody.

Prevalence of hepatitis B virus among healthy individuals

James et al., 2011 carried out a study to assess the seroprevalence of hepatitis B surface antigen (HBsAg) and associated risk factors among students of a secondary school in Jagindi Tasha, Kaduna State, Nigeria. Out of One hundred and ninety (190) apparently healthy students that were screened for HBsAg, 35 (18.4%) were sero-positive. Subjects aged 13-15 years recorded 6.8% positivity and male subjects had 25.5% positivity compared to 10.9% positivity for females. Risk factors such as blood transfusion was 32.0% among male subjects compared to 30.0% in females.

Moses et al., (2010), evaluated the prevalence of Hepatitis B virus infections in apparently healthy urban Nigerians. Of the 1,891 participants, 957 (50.6 %) were males and 934 (49.4%) were females. Overall 114 (6.0%) were positive, of whom 71 (7.4%) were males and 43 (4.6%) females. Those aged 21–30 years had the highest infection rate, and males were more likely to be infected with the virus than females. According to Gambo et al. [42] out of 182 Fulani nomads in Toro, North-Eastern Nigeria the gender-specific seroprevalence of HBsAg was found to be in the ratio of about 2:1 male-female. Infection rate was found to be higher in those between 25 and 29 years (8.2%) followed by those the age group 30-37 years (6.0%).

According to Olokoba et al., 2009, Five hundred and ninety-five consecutively recruited voluntary blood donors in Yola, Nigeria that were screened for hepatitis B and hepatitis C virus infections. Only 14 donors (male) each (2.4%) were positive for HBsAg and anti-HCV. The authors concluded that the seroprevalence of hepatitis B and C virus infection is low among voluntary blood donors in Yola, Nigeria [43], evaluated the Prevalence of Hepatitis B surface Antigen among the Newly Admitted Students of University of Jos, Nigeria. Out of the 300 newly admitted students that were screened, 50 (16.7%) were seropositive to HBsAg. The prevalence of HBsAg was higher in males 34(11.33%) compared to 16(5.33%) in females. Age specific prevalence was significantly higher in the age bracket 25-29, with 16(28.57%) and the lowest was found in the age bracket 15 -19 years with 12(17.39%).

In a study conducted by Ndako et al. [44], a total of 188 Health personnel, which constitutes Nurses, Doctors, Medical Laboratory Scientists, Technicians/Assistants, Pharmacists and Ward Assistance in Uyo Metropolis, were screened for HBV surface antigen (HBsAg). Out of the one hundred and Eighty-eight (188) respondents screened. Thirty-two (32) representing 17.0% were found to be seropositive, female subjects recorded (17.3%) prevalence compared to (16.7%) recorded by the Male subjects. Frank et al., 2004 carried out epidemiology study of HBV infection

among 124 unvaccinated Dutch missionaries and family members who lived in a rural area of Nigeria. Antibodies to hepatitis B core antigen were found in 5 (9.8%) of 51 adults (incidence rate, 1.7 per 1000 person-months at risk [PMAR]) and 9 (12.3%) of 73 children (incidence rate, 2.8 per 1000 PMAR).

Prevention and Treatment of Hepatitis B

Even though HBV has become a major source of health concern worldwide, we should also be reminded by the good news that it is the only STD that can be prevented by vaccination (CDC, 2005). The prevention of HBV globally has become one of the topmost priorities of major political actors and decision makers in recent years. The disease is prevented using safe and effective vaccine which became available in 1982 through funding and implementation of hepatitis B immunization programs. Measures for HBV prevention have been geared towards avoidance of unsafe blood exposure or blocking of transmission before the advent of the vaccine. Unsafe blood transfusion has been a major force in the transmission of HBV globally [45].

The enactment of a law for the donation and management of blood in blood banks across the world has aggressively fought this channel of HBV transmission. This notwithstanding, current researches have showed that blood transfusion is regaining its position as one of the major risk factors for HBV transmission globally. This finding is attributed to the presence of occult HBV infection (OHBVI) among blood donors [46]. It is also worth mentioning that the global acceptance of the auto-disposable syringes (ADS) has considerably reduced the incidence of HBV infections that occur due to unsafe injections. Also, as a result of the extensive use of invasive medical procedures, iatrogenic HBV infections are no longer frequent. There have also been speculations that dental care operations which are capable of causing oral mucous membrane injuries is becoming a major route to HBV transmission if steps are not taken to prevent it [47].

HBV per se does not have a permanent treatment therefore, the surest antidote to the global epidemic is prevention. There has not been any universal agreement on drugs used for the temporary treatment of the HBV in the world even though two therapeutic agents such as interferon alpha (IFNa) and lamivudine are currently used by many countries for the treatment of the disease. Interferon-alpha is a potent cytokine with antiviral and immunomodulating actions which is produced in response to viral infection [48]. Temporary treatment of the disease is therefore aimed at suppressing viral replication, reducing the risk of progressing to advanced liver disease or inflammation of the liver and the development of complications such as liver failure or liver cancer [49-55]. Chronic hepatitis B is therefore easily managed rather than treated. Some of the general management strategies for HBV recommended by medical experts include the avoidance of:

- Heavy alcohol consumption.
- Unprotected sexual intercourse with partners who are not vaccinated.

- Sharing of needles or other items that potentially contain blood such as shavers or toothbrushes.
 - Donation of blood or organs.
2. Screening of family members and sexual partners for HBV infection and vaccination of those who are sero-negative [56-65].
 3. Patient education and long-term follow-up with regular testing of liver biochemistry and surveillance of hepatocellular carcinoma in high risk groups [20, 66-70].

Conclusion

In conclusion, this review sheds light on many important aspects of HBV epidemiology in Nigeria, the prevalence of hepatitis B virus is high and varies with geographical region and gender different been higher in pregnant women. However, Reduction in hepatitis B virus infection could be achieved by public enlightenment campaign, mass immunization of the children and adults at risk while antiviral drugs and immunostimulatory therapy should be provided for those already infected.

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Conflict of Interest

The author declares no conflict of interest exist.

References

1. WHO (2009) Hepatitis B Vaccines? Weekly Epidemiological Record.
2. Ott JJ, Stevens GA, Groeger J, Wiersma ST (2012) Global Epidemiology of Hepatitis B Virus Infection: New Estimates of Age-Specific HBsAg Seroprevalence and Endemicity. *Vaccine* 30(12): 2212-2219.
3. Walana W, Hokey P, Ahiaba S (2014) Sero-Prevalence of Hepatitis B Virus Infection among Blood Donors: A Retrospective Study in the Kintampo Municipal Hospital, Ghana. *Open Journal of Medical Microbiology* 4: 64-69.
4. Jia JD, Zhuang H (2007) A Wining War Against Hepatitis B Virus Infection in China. *Chin Med J (Engl)* 120(24): 2157-2158.
5. Zou S1, Zhang J, Tepper M, Giulivi A, Baptiste B (2001) Enhanced Surveillance of Acute Hepatitis B and Acute Hepatitis C in Four Health Regions in Canada, 1998 to 1999. *Can J Infect Dis* 12(6): 351-356.
6. James A Ndako, Obinna ON, Georgebest ON, Echeonwu SAJ, Onyeka A, et al. (2012) Studies on Prevalence and Risk Factors for Hepatitis B Surface Antigen among Secondary School Students in North-central, Nigeria. *Sierra Leone Journal of Biomedical Research* 3(3): 163-168.
7. Greenwood D, Slack RCB, Peutherer JF (2000) *Medical Microbiology* (15th edn), Churchill Livingstone, An Imprint of Harcourt Publishers Limited, pp. 441.
8. Brooks GF, Carroll KC, Butel JS, Morse SA (2007) *Medical Microbiology*. (24th edn), International edition. McGraw Hill Publishers, New York, USA, pp. 425-443.
9. Tong S, Kim KH, Chante C, Wands J, Li J (2005) Hepatitis B virus e antigen variants. *International Journal of Medical Science* 2(1): 2-7.
10. Kwon SY, Lee CH (2011) Epidemiology and prevention of hepatitis B virus infection. *Korean J Hepatol* 17(2): 87-95.
11. Sirisena ND, Njoku MO, Idoko JA, Isamade E, Barau C, et al. (2002) Carriage rate of hepatitis B surface antigen (HBsAg) in an urban community in Jos, Plateau State, Nigeria. *Niger Postgrad Med J* 9(1): 7-10.
12. Jombo GT, Egah DZ, Banwat EB (2005) Hepatitis B virus infection in a rural settlement of Northern Nigeria. *Niger J Med* 14(4): 425-428.
13. Akani CI, Ojule AC, Opurum Hc, Ejilemele AA (2005) Seroprevalence of hepatitis B surface antigen HBsAg in pregnant women in Port Harcourt Nigeria. *Niger Postgrad Med J* 12 (4): 266-270.
14. Agbede OO, Iseniyi JO, Kolawole MO, Ojuowa A (2007) Risk factors and seroprevalence of hepatitis B surface antigenemia in mothers and their preschool age children in Ilorin, Nigeria. *Therapy* 4(1): 67-72.
15. Harry TO, Bajani MD, Moses AE (1994) Hepatitis B Virus infection among blood donors and pregnant women in Maiduguri Nigeria. *East Afr Med J* 71(9): 596-597.
16. Jatau ED, Yabaya A (2009) Seroprevalence of hepatitis B virus in pregnant women attending a clinic in Zaria, Nigeria. *Science World journal* 4: 7-9.
17. Nwokedi EE, Emokpae MA, Taura AA, Dutse AI (2006) The trends of hepatitis B surface antigenemia among teaching hospital patients in Kano. *African Journal of Clinical Experimental Microbiology* 7: 143-147.
18. Blumberg BS (1977) Australia antigen and the biology of hepatitis B. *Science* 197(4298): 17-25.
19. Dane DS, Cameron CG, Briggs M (1970) Virus-like particles in serum of patients with Australia antigen-associated hepatitis. *Lancet* 1: 696-698.
20. Batholomew C (2011) Knowledge, Attitude and Practices (KAP) concerning Hepatitis B among Adolescents in the Upper West Region of Ghana. The Rural Urban Gradient. Thesis Department of Public Health and Clinical Medicine Umeå University, Sweden.
21. Altiparmak E, Koklu S, Yalinkilic M, Yuksel O, Cicek B (2005) Viral and host causes of fatty liver in chronic hepatitis B. *World J Gastroenterol* 11 (20): 3056-3059.
22. McMahon BJ (2005) Epidemiology and natural history of hepatitis B. *Semin Liver Dis* 1: 3-8.
23. Mosley JW, Stevens CE, Aach RD, Hollinger FB, Mimms LT, et al. (1995) Donor screening for antibody to hepatitis B core antigen and hepatitis B virus infection in transfusion recipients. *Transfusion* 35(1): 5-12.
24. Damulak OD, Ogbenna V Ma'an O, Rufai SD, Kut T, Bodund A (2013) hepatitis B virus sero-positivity among voluntary blood donors at a centralized blood service centre in Nigeria. *International Journal of Medical and Applied Sciences* 2(3): 10-18.
25. Uneke CJ, Ogbu O, Inyama PU, Anyanwu GI, Njoku MO, et al. (2005) Prevalence of hepatitis B virus among blood donors and human immunodeficiency virus infected patients in Jos, Nigeria. *Mem Inst Oswaldo Cruz* 100(1): 13-6.
26. Altiparmak E, Olajumoke OO, Owolabi AD, Titilope AA, Adewumi A, et al. (2012) Seroprevalence of hepatitis B e antigen (HBe antigen) and B core antibodies (IgG anti-HBcore and IgM anti-HBcore) among hepatitis B surface antigen positive blood donors at a Tertiary Centre in Nigeria. *BMC Res Notes* 5: 167.
27. Aernan PT, Sar PT, Torkula SH (2011) Prevalence of Plasmodia and Hepatitis B Virus co-infection in Blood donors at Bishop Murray medical centre Makurdi, Benue State, Nigeria. *Asian Pac J Trop Med* 4(3): 224-226.
28. Jeremiah AZ, Idris H, Ajayi BB, Ezimah AC, Malah MB, et al. (2011) Isolated anti-HBc-IgM antibody among blood donors in the semi-arid region of Nigeria. *Human antibodies* 20(3-4): 77-82.
29. Salawu L, Adegoke AO, Aboderin AO, Haraina HA (2011) Hepatitis B Virus markers in surface antigen negative blood donors: the need to look beyond antibody negativity. *West Africa Medicine* 30(4): 292-295.
30. Japet MO, Adesina OA, Danbraye E, Adewumi MO (2011) Hepatitis B core IgM antibody (anti-HBcIgM) among hepatitis B surface antigen (HBsAg) negative blood donors in Nigeria. *Virol J* 8: 513.
31. Eke AA, Uzoamaka AE, Charles IO, Ifeanyichukwu UE, Chukwuanugo O (2011) Prevalence, correlates and pattern of hepatitis B surface antigen in a low resource setting. *Virol J* 8: 12.

32. Agarry OO, Lekwot GZ (2010) Prevalence of hepatitis B virus and hepatitis C virus in ante-natal patients in Gwagwalada- Abuja, Nigeria. *Report and Opinion* 2(7): 48-50.
33. Ojo OO, Anibijuwon II (2009) Determination of Antibodies to Hepatitis B Virus in Pregnant Women in Akure, Ondo State, Nigeria. *Continental Journal of Microbiology* 3: 6-10,
34. Mbaawuaga EM, Enebebeaku MNO, Okopi JA, Damen JG (2008) Hepatitis B Virus (HBV) Infection among Pregnant Women in Makurdi, Nigeria. *African Journal of Biomedical Research* 11(2008): 155-159.
35. Ejeligiou EU, Oguche AO, Ebonyi ES, Okpe ES, Yiltok MO, et al. (2014) Prevalence and Laboratory Profile of Hepatitis B Virus Co-infected Nigerian Children with Human Immunodeficiency Virus. *International Journal of TROPICAL DISEASE and Health* 4(7): 773-781.
36. Egah DZ, Banwat EB, Audu ES, Iya D, Mandong BM, et al. (2007) Hepatitis B Surface antigen, hepatitis C virus and human immunodeficiency virus antibodies in a low risk blood donor group in Nigeria. *East Mediterranean Health Journal* 13(4): 961-966.
37. Omalu ICJ, Jibrin A, Olayemi IK, Hassan SC, Mgbemena C, et al. (2012) Seroprevalence of Malaria and Hepatitis B (HBsAg) with Associated Risk Factors among Pregnant Women Attending Antenatal Clinic in General Hospital Minna, North-Central Nigeria. *Annual Review and Research in Biology* 2(4): 83-88.
38. Emmanuel M, Mbaawuaga CUI, Anthony CI (2014) Hepatitis B Virus (HBV) Serological Patterns in Benue State, Nigeria. *Open Journal of Medical Microbiology* 4: 1-10.
39. Jibrin YB, Mustapha SK (2004) The Prevalence of Hepatitis B Surface Antigenemia in Patients with Human Immunodeficiency Virus (HIV) Infection in Gombe, Nigeria. *Annals of African Medicine* 3(1): 10-12.
40. Taiwo MB, Samuel E, Emmanuel FO (2012) HIV, Hepatitis B and C viruses' coinfection among patients in a Nigerian tertiary hospital. *Pan Afr Med J* 12: 100.
41. Salawu L, Bolarinwa RA, Adegunloye AB, Muraina HA (2010) HBsAg, anti-HCV, anti-HIV and VDRL in blood donors: Prevalence and trends in the last three and a half years in a tertiary health care facility in Ile-Ife, Nigeria. *International Journal of Medicine and Medical Sciences* 2(11): 335-341.
42. Gambo IM, Rabiu AM, Muhammad MB, Shugaba AI (2012) Seroprevalence of HBsAg among Fulani nomads in Toro, North-Eastern Nigeria. *Global Advanced Research Journal of Medicine and Medical Sciences* 1(8): 214-217.
43. Odinachi OE, John DM, Augustine U, Ogbonnaya O, Felicia N, et al. (2014) Prevalence of Hepatitis B surface Antigen among the Newly Admitted Students of University of Jos, Nigeria. *American Journal of Life Sciences* 2(1): 35-39.
44. Ndako JA, Onwuliri EA, Adelani Akande T, Olaolu DT, Dahunsi SO, et al. (2014) Screening for Hepatitis B Surface Antigen (Hbsag) Among Health Care Workers (Hcw) In an Urban Community South, South Nigeria. *IJBPAS* 3(3): 415-425.
45. Wang ST, Wong XZ (1960) Report of 5 cases of post transfusion serum hepatitis. *J Peking Univ (Health Sci) (Chin)* 1: 63-66.
46. Shang G, Seed C, Wang F, Nie D, Albert F (2007) Residual risk of transfusion-transmitted viral infections in Shenzhen, China 2001 through 2004. *Transfusion* 47(3): 529-539.
47. Zhang H, Yin J, Li Y, Li C, Ren H, et al. (2008) Risk factors for acute hepatitis B and its progression to chronic hepatitis in Shanghai, China 57(12): 1713-1720.
48. Sen G, Ransohoff R (1993) Interferon-induced antiviral actions and their regulation. *Adv Virus Res* 42: 57-102.
49. Adabara NU, Ajal OO, Momohjimoh A, Hashimu Z, Agabi AYW (2012) Prevalence of Hepatitis B Virus among Women Attending Antenatal Clinic in the General Hospital, Minna, Niger State. *Shiraz E-Medical Journal* 13(1): 28-32.
50. Amira MS (2011) Prevalence of Hepatitis B Virus DNA Among Blood Donors in Nablus- West Bank. A Thesis is Submitted to An-Najah National University, Nablus, Palestine.
51. Ataallah TA, Khaleel A, Kadoori SM, Alaani AS (2011) Prevalence of hepatitis B and C among blood donors attending the National Blood Transfusion Center in Baghdad Iraq from 2006-2009. *Saudi Med J* 32(10): 1046-1050.
52. Bonino FC, Maran P (1987) Serological markers of HBV infectivity. *Ann Ist Super Sanita* 24(2): 217-223.
53. CDC (2005) A comprehensive immunization strategy to eliminate transmission of hepatitis B virus infection in the United States: recommendations of the Advisory Committee on Immunization Practices (ACIP). Part 2: Immunization of adults. *MMWR*.
54. Chu C, Liaw Y (2007) Predictive factors for reactivation of hepatitis B following hepatitis B e antigen seroconversion in chronic hepatitis B. *Gastroenterology* 133(5): 1458-1465.
55. Colin WS, Edgar PS, Lyn F, Anthony EF, Beth PB (2006) Hepatitis B Virus Infection: Epidemiology and Vaccination. *Epidemiologic Reviews* 28(1): 112-125.
56. Conjeevaram HS, Lok AS (2001) Occult hepatitis B virus infection: a hidden menace? *Hepatology* 34(1): 204-206.
57. Elgouhari HMA, Tamimi TI, Carey WD (2008) Hepatitis B virus infection: understanding its epidemiology, course, and diagnosis. *Cleve Clin J Med* 75(12): 881-889.
58. Frank GJ, Cobelens, Henk J van Schothorst, Pauline ME Wertheim Van Dillen, Robert J. Ligthelm, Ineke S. Paul-Steenstra, et al. (2004). Epidemiology of Hepatitis B Infection among Expatriates in Nigeria. *Clinical Infectious Diseases* 38: 370-376.
59. Hajrullah F, Skender T (2009) Prevalence of HBV and HCV among blood donors in Kosovo. *Virol J* 6: 21.
60. Howard CR (1986) The Biology of Hepadna viruses. *Journal of General Virology* 67: 1215-1235.
61. Huang CF, Lin SS, Ho YC, Chen FL, Yang CC (2006) The immune response induced by hepatitis B virus principal antigens. *Cell Mol Immunol* 3(2): 97-106.
62. James AN, Obinna ON, Georgebest ON, Echeonwu SA, Junaid OA, et al. (2011) Studies on Prevalence and Risk Factors for Hepatitis B Surface Antigen among Secondary School Students in North-central, Nigeria. *Sierra Leone Journal of Biomedical Research* 3(3): 163-168.
63. Juergen B, Michael N (2007) Hepatitis B virus replication. *World J Gastroenterol* 13(1): 48-64.
64. Kim KH, Shin HJ, Kim K, Choi HM, Rhee SH, et al. (2007) Hepatitis B virus X protein induces hepatic steatosis via transcriptional activation of SREBP1 and PPAR- gamma. *Gastroenterology* 132(5): 1955-1967.
65. Lai KN, Li PK, Lui SF, Au TC, Tam JS, et al. (1991) Membranous nephropathy related to hepatitis B virus in adults. *N Engl J Med* 324(21): 1457-1463.
66. Lavanchy D (2002) Public health measures in the control of viral hepatitis: a World Health Organization perspective for the next millennium. *J Gastroenterol Hepatol Suppl*: S452-S459.
67. Locarnini S (2004) Molecular virology of hepatitis B virus. *Semin Liver Dis Suppl* 1: 3-10.
68. Lok AS, McMahon B (2007) Chronic hepatitis B. *Hepatology* 45(2): 507-539.
69. McMahon BJ, Alberts SR, Wainwright RB, Bulkow L, Lanier AP (1990) Hepatitis B-related sequelae. Prospective study in 1400 hepatitis B surface antigen-positive Alaska native carriers. *Arch Intern Med* 150(5): 1051-1054.
70. Milich D, Liang TJ (2003) Exploring the biological basis of hepatitis B e antigen in hepatitis B virus infection. *Hepatology* 38(5): 1075-1086.