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Impact of immunological, hematological and biochemical markers on discordant partners of Hepatitis B infection in Enugu State, Eastern Nigeria

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Abstract

Globally, hepatitis B infection is a serious health problem as its morbidities are difficult to handle with the commonly used antivirals. This study therefore aims to evaluate the serological, biochemical and hematological markers of Hepatitis B virus infection in a discordant partner in an attempt to aid proper diagnosis, management and control of the infection. This is a cross-sectional study consisting of 150 subjects recruited using a simple random sampling technique. HBsAg was screened using a rapid ELISA diagnostic strip and rescreened later for confirmation using Ichroma Fluorescence Immunoassay (FIA). HBV Serological markers were determined using HBV 5 panel test, hematological parameters were assessed using Mindray BC 10 automated counter, while liver enzymes were estimated using COBAS 111 analyzer. Statistical analysis was performed using Graph Pad Prism. This study demonstrated a varying percentage detection rates of HBV serological markers in both groups: Discordant and Concordant partners (HBsAg- 62%, HBsAb- 4.2%, HBeAg- 2.5%, HBeAb- 50%, HBcAb-60.8%), and Control partners (HBsAg-0%, HBsAb-23.3%, HBeAg-0%, HBeAb-0%, and HBcAb- 6.7%). The liver enzymes showed significant mean values ($P < 0.001$) for both ALT and AST positive partners when compared to their negative counterparts. Hematological parameters, only hemoglobin showed a significant mean ($P < 0.001$) on male subjects as against females, while white blood cell and platelets were not statistically significant. There is a need for hepatitis B panel test inclusion in the routine investigation for Hepatitis B viral infection which will contribute immensely in proper clinical management and control of the infection in conjunction with vaccination, while periodic assessment of liver enzymes will ensure proper management of chronic hepatitis B infections.

Keywords: Hepatitis B; Serological markers; Immunity; Discordant partners; Immunoassay

1. Introduction

Infection with hepatitis B virus (HBV) is a serious global public health problem [1], with the reported prevalence ranging from 0.1% to 20% and approximately 350-400 million people worldwide are chronically infected facing off the high risk for developing cirrhosis, fulminant hepatitis, end-stage liver disease and hepatocellular carcinoma [2]. HBV prevalence is highest in Sub-Saharan Africa and East Asia, where between 5–10% of the adult population is chronically infected accounting for 500,000–1.2 million deaths per year and is the tenth leading cause of mortality worldwide [3].

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Hepatitis B Virus is an enveloped, hepatotropic and non-cytopathic small DNA virus with a partially double-stranded DNA of about 3.2 kb, belonging to the family Hepadnaviridae. Hepatitis B infection is caused by the hepatitis B virus (HBV) and it affects the liver. Infection with Hepatitis B Virus results in acute hepatitis followed by recovery in about 85%-90% of human adults [4]. Recovery occurs when the host mounts adequate immune responses against the virus. Such responses include production of protective and neutralizing antibodies against the HBV surface antigen (HBsAg) [5,6], activation of strong and diversified CD4 and CD8 T-cells [7], expression of antiviral cytokines in the liver, such as gamma interferon and tumour necrosis factor-alpha [8,9,10], and generation of cells that are protected from re-infection [11,12]. In contrast, progression to chronic HBV infection occurs predominantly in immune-compromised adults and unvaccinated infants [13]. Such individuals exhibit weak and inefficient humoral and cellular immune responses, resulting in continual viral replication and high HBV surface antigenemia [14]. The progression to chronicity and complication is directly related to high viral replication demonstrable serologically by the presence of markers of pathogenicity, infectivity and chronicity. These serological markers include Hepatitis B surface antigen (HBsAg), and its corresponding antibody (anti-HBs), hepatitis B core antigen (HBcAg) and antibodies to the core antigen (anti-HBc IgM and IgG), hepatitis B envelope antigen (HBeAg) and hepatitis B envelope antibody (anti-HBe) and hepatitis B virus DNA (HBV DNA). Hepatitis B surface antigen (HBsAg) also known as Australian antigen is the surface antigen of the hepatitis B virus and it serves as an important serological marker in the diagnosis and monitoring of patients infected with hepatitis B virus [15]. The presence of HBsAg indicates that the person is infectious and it can be detected in high levels in serum during acute or chronic hepatitis B infection. Individuals with IgG variety of anti-HBc may not be infectious as they may have sufficiently high titres of antibodies to HBsAg which are protective and the affected individuals may be disease-free [16, 17]. It has been demonstrated that some patients with no detectable HBsAg in them but showing HBc IgM antibody in their sera continue to replicate HBV [18]. Thus, the absence of HBsAg in the blood sample may not be enough to prove the absence of an ongoing HBV infection [18]

Transmission of hepatitis B virus results from exposure to infectious blood or body fluids containing blood. It is 50 to 100 times more infectious than HIV [19]. Possible forms of transmission include sexual contact, blood transfusion and transfusion with other human blood products, re-use of contaminated needles and syringes and vertical transmission from mother to child transmission (MTCT) during childbirth. Without intervention, a mother that is HBsAg positive has a 20% risk of passing the infection to her offspring at the time of birth, but the risk may be as high as 90% if the mother is equally positive for HBeAg [20]. At least 30% of reported hepatitis B among adults cannot be associated with an identifiable risk factor. The incidence of HBV infection rises after the onset of sexual activity in puberty and peaks in the age group of 25-29 years [21]. Unprotected sexual intercourse is associated with an estimated risk of infection of at least 20% [22]. Importantly, it is associated with the lack of pre-emptive vaccination of sexual partners. Thus, recent HBV guidelines strongly recommended vaccination of sexual partners to prevent infection [5]. However, there are healthy seronegative sexual partners of patients with chronic hepatitis B who have never been vaccinated but are negative for HBsAg, anti-HBs, anti-HBc and HBV-DNA and much is not known about this phenomenon. Therefore, this study was designed to evaluate the serological, hematological and biochemical markers and their impact on discordant partners of hepatitis B infection.

2. Material and methods

2.1. Study Area

This study was done at the Laboratory division of the department of Medical Enugu State University Teaching Hospital, (ESUTH) Parklane, Enugu, in Enugu State, Nigeria.

2.2. Research Design

This is a cross-sectional study performed to profile the serological, hematological and biochemical markers of hepatitis B viral infection in discordant couples. A simple random sampling technique was employed in collecting the sample after informed consent.

2.3. Specimen Collection

A total of 150 subjects were recruited for this study including 30 uninfected partners (controls), 90 discordant and 30 concordant partners of hepatitis B virus infection. Blood samples were collected from the subjects in EDTA bottles and plain tubes. The samples in plain tubes were allowed to be clotted before the separation of the serum through centrifugation at 3,000 rpm for 5 minutes, while the EDTA tubes were used immediately for hematological tests.

2.4. Sample Analysis

2.4.1. Serological Screening

The samples were first screened for HBsAg using a rapid diagnostic strip (LabAcon, Biotest Biotech Co. Ltd, China). The test strip is a qualitative solid-phase, two-site sandwich immunoassay that detects HBsAg in serum or plasma. The tests were done according to the manufacturer's instruction and positive samples were further screened for hepatitis A, C, and HIV and positive samples were excluded.

2.4.2. HBsAg Assay

An additional test on HBsAg was done to confirm the positivity of the screened hepatitis B samples using Ichroma Fluorescence Immunoassay (FIA) technique. The test uses a sandwich immunodetection method and dried antibodies once diluted with the diluent, bind with antigens in the sample to form an antigen-antibody complex. The complex then migrates through the nitrocellulose matrix and are captured by another set of immobilized antibodies on the test line. The more the antigens HBsAg in the sample serum, the more the antigen-antibody complexes, which leads to a stronger fluorescence signal. The signal is then interpreted by the reader and results are displayed on the screen. The results were interpreted as follows:

HBsAg

≤0.9 Negative	>0.9, <1.0 Indeterminate	≥1.0 Positive	Linearity limitation is between 0 – 500 miu/ml.
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2.4.3. HBV Panel test

HBsAb, HBeAg, HBeAb, HBcAb, and HBcAb were serologically screened using HBV panel test cassette by BioRad, Marnes la Coquette, France. It is a qualitative test for the detection of hepatitis B serological markers and the kit uses colloidal gold and membrane chromatography technology. HBsAg and HBeAg were measured using a dual antibody sandwich, HBsAb was through dual antigen sandwich method, while HBeAb and HBcAb used neutralization competitive inhibition method. The assay was done by strictly following the manufacturer's protocols.

2.4.4. Haemoglobin (Hb), Total White Blood Cell (WBC) and Platelets:

Hematological parameters (Hb, WBC and Platelets) were estimated using Mindray BC10 autoanalyser.

2.4.5. Liver enzymes

((Alanine Transferase (ALT) and Aspartate Transaminase (AST)) were estimated using COBAS 111.

2.5. Data Analysis

All continuous variables were expressed as mean ± standard deviation (SD) or range, and categorical variables were presented as frequencies or percentages. Differences in variables or comparison between groups were analyzed using analysis of variance and Student's t -tests (for normally distributed data) or the Kruskal-Wallis χ^2 and Mann-Whitney U -tests (for non-normally distributed data). The level of statistical significance was set at $p < 0.05$. Statistical analysis was performed using Graph Pad Prism version 6.0 software.

3. Results

3.1. Demographic and Baseline Characteristics of the Study Population

Table 1 shows the demographic and baseline characteristics of the study population. The mean age of the participants was 36.60 years ± 7.06 years. Majority of the participants (control- 56.7%; discordant- 62.2%; concordant- 50%) were of the age group 31-40 years. Fifty per cent of the subjects were males and fifty per cent were females in all the study groups. Regarding their smoking habit, the majority are non-smokers in all the groups (control, 86.7%, discordant, 96.7% and concordant, 90%). A greater percentage of both discordant (51.1%) and concordant (60%) groups drink alcohol. The majority of the participants (control, 90%; discordant, 92.2%; concordant, 100%) had no history of HBV

vaccination. Fifty per cent of the discordant group and 100% of the concordant group were infected with hepatitis B virus.

Table 1 Demographic and baseline characteristics of the study population represented as Mean \pm SD or n (%)

Characteristics	Discordant Partners (n = 90)	Concordant Partners (n = 30)	Uninfected Control (n = 30)	All (n = 150)
Age (yrs)	36.31 \pm 6.67	37.30 \pm 7.62	35.26 \pm 7.69	36.30 \pm 7.06
20-30	16 (17.8%)	5 (16.7%)	9 (30%)	30 (20%)
31-40	56 (62.2%)	15 (50%)	17 (56.7%)	88 (58.7%)
>40	18 (20%)	10 (33.3%)	4 (13.3%)	32 (21.3%)
Sex				
Males	45 (50%)	15 (50%)	15 (50%)	75 (50%)
Females	45 (50%)	15 (50%)	15 (50%)	75 (50%)
Smoking Habit				
No	87 (96.7%)	27 (90%)	26 (86.7%)	140 (93.3%)
Yes	3 (3.3%)	3 (10%)	4 (13.3%)	10 (6.7%)
Drinking Habit				
No	46 (51.1%)	18 (60%)	11 (36.7%)	75 (50%)
Yes	44 (48.9%)	12 (40%)	19 (63.3%)	75 (50%)
Vaccination				
No	83 (92.2%)	30 (100%)	27 (90%)	140 (93.3%)
Yes	7 (7.8%)	0 (0%)	3 (10%)	10 (6.7%)
HBV Status				
Negative	45 (50%)	0 (0%)	30 (100%)	75 (50%)
Positive	45 (50%)	30 (100%)	0 (0%)	75 (50%)

3.2. Serological Markers of Hepatitis B Virus Infection

Table 2 Distribution of HBV serological markers in (A) Discordant and concordant partners, and (B) Control partners

A: Discordant and Concordant partners		
Marker	No	% (n=120)
HBsAg	75	62.5
HBsAb	5	4.2
HBeAg	3	2.5
HBeAb	60	50
HBcAb	73	60.1
B: Control partners		
Marker	No	% (n=30)
HBsAg	-	0
HBsAb	7	23.3
HBeAg	-	0
HBeAb	-	0
HBcAb	2	6.7

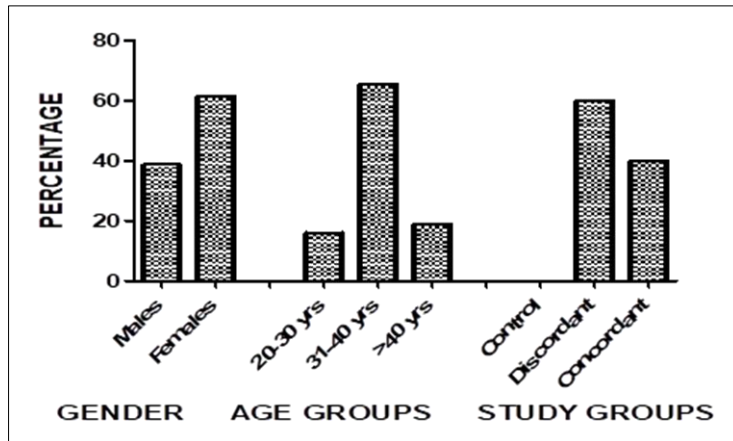


Figure 1 Distribution of HBV carriers in study population (n = 75) according to sex, age and study groups

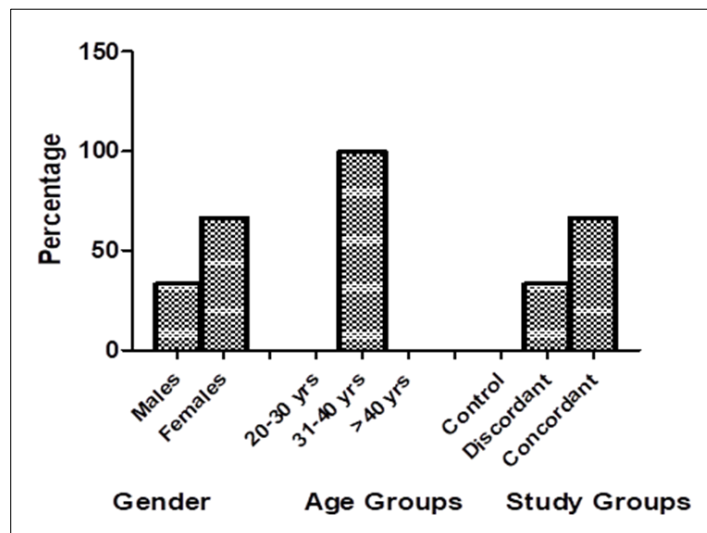


Figure 2 Distribution of individuals with active HBV replication (HBsAg (+ive) + HBeAg (+ive); n = 3) according to sex, age and study groups

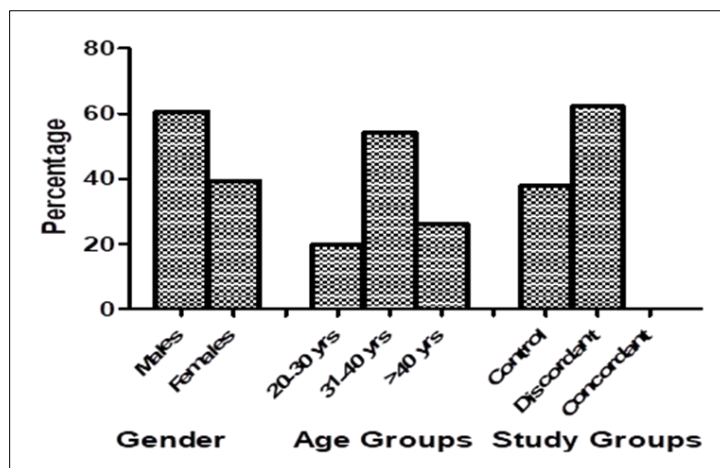


Figure 3 Distribution of HBV susceptible population (HBsAg (-ve), HBeAg (-ve), HBsAb (-ve), HBeAb (-ve), HBcAb (-ve)) according to sex, age and study groups

Table 2 shows the distribution of hepatitis B serological markers in both groups. (A) Part of the table contains both the discordant and concordant partners and it reveals that only 3 or 2.5% were actively replicating (HBeAg positive), while 5 or 4.2% had been seroconverted or immunized (HBsAb positive), while greater numbers (73 or 60.1%) were in chronicity (HBcAb positive). (B) Part of the table shows the serological markers of the control partners and it indicates that about 23.3% were either naturally immunized or had been vaccinated (HBsAb positive). Figure 1 shows the distribution of HBV carriers (HBsAg (+ive)) in the study population according to sex, age and study groups. Data indicated that a greater percentage of those identified as HBV carriers were females (61.3%, n = 46) compared with the males (38.7%, n = 29). The HBV carriers was more in the age-group of 31-40 years (65.3%, n = 49), followed by >40 years (18.7%, n = 14) and 20-30 years (16%, n = 12). Figure 2 shows the distribution of individuals with active HBV replication (HBsAg (+ive) + HBeAg (+ive)) according to sex, age and study groups, and the result showed that the incidence of active HBV replication was more in females (66.7%, n = 2) compared with males (33.3%, n = 1). Active HBV replication was found in individuals of age 31-40 years (100%, n = 3) and a greater percentage of individuals with the active HBV replication was found in concordant (66.7%) compared with the discordant (33.3%). The distribution of HBV susceptible population according to sex, age and study groups is shown in Figure 3. Results indicated that a greater percentage of the males (60.7%, n = 37) were susceptible to HBV infection compared with the females (39.3%, n = 24). The HBV susceptibility was highest in individuals between the age group of 31-40 years (54.1%, n = 33), followed by those greater than forty years (>40 years, 26.2%, n = 16) and 20-30 years (19.7%, n = 12). The discordant group (62.3%, n = 38) had more susceptible cases compared with control (37.7%, n = 23).

3.3. Liver Enzymes (ALT, AST) – Biochemical Markers

Table 3 shows the mean ALT and AST levels of HBV infected and non-infected individuals. The results indicated that HBV positive individuals had significantly greater mean ALT ($p < 0.001$) and AST ($p < 0.001$) compared with the HBV negative individuals. Also, Table 4 shows the mean concentrations of some liver function parameters in HBV infected and non-infected individuals according to their gender. In males, results indicated significantly greater mean ALT ($P < 0.001$) and AST ($p = 0.001$) in HBV infected compared with uninfected individuals. Also in females, the HBV infected individuals indicated significantly higher mean ALT ($p = 0.001$) compared with the uninfected individuals. But in contrast, no significant difference was observed in AST ($p = 0.096$) between the two groups. Table 5 shows the mean concentrations of some liver function parameters in HBV infected and non-infected individuals according to their age. In the age group 20-30 years, there is a significantly greater mean ALT ($P = 0.018$) compared with uninfected individuals. However, no significant difference was observed in AST ($p = 0.240$) between the two groups. In the 31-40 years age group, the HBV infected individuals also indicated significantly higher mean ALT ($p < 0.001$) and AST ($p = 0.002$) compared with the uninfected individuals. In the age group >40 years, the HBV infected individuals indicated significantly greater mean ALT ($P = 0.012$) compared with uninfected individuals. In contrast, no significant difference was observed in AST ($p = 0.109$) between the two groups.

Table 3 Mean ALT and AST levels of HBV infected and non-infected individuals

Liver Enzyme	HBV Status	N	Mean \pm SD	T - Statistics	P- Value
ALT	Positive	75	11.04 \pm 6.05	5.58	<0.001
	Negative	75	6.65 \pm 3.10		
AST	Positive	75	12.64 \pm 6.82	3.80	<0.001
	Negative	75	9.08 \pm 4.36		

Table 4 Evaluation of liver function status of HBV infected and non-infected individuals according to their sex

Variables	Sex	HBV Positive (n = 75)		HBV Negative (n = 75)		P-Value
		Mean \pm SD	N	Mean \pm SD	N	
ALT (IU)	Males	10.51 \pm 5.89	29	6.52 \pm 3.05	46	<0.001
	Females	11.36 \pm 6.19	46	6.86 \pm 3.23	29	0.001
AST (IU)	Males	12.51 \pm 6.50	29	8.39 \pm 3.80	46	0.001
	Females	12.71 \pm 7.08	46	10.17 \pm 5.01	29	0.096

Table 5 Evaluation of liver function status (ALT, AST) of HBV infected and non-infected individuals according to their age

Variables	Age (yrs)	HBV Positive (n = 75)		HBV Negative (n = 75)		P- Value
		Mean \pm SD	N	Mean \pm SD	N	
ALT (IU)	20-30	12.50 \pm 8.79	12	6.77 \pm 3.45	18	0.018
	31-40	11.02 \pm 5.77	49	6.69 \pm 3.01	39	<0.001
	>40	9.85 \pm 4.07	14	6.44 \pm 3.12	18	0.012
AST (IU)	20-30	12.50 \pm 8.77	12	9.72 \pm 3.70	18	0.240
	31-40	13.04 \pm 6.83	49	9.02 \pm 4.62	39	0.002
	>40	11.35 \pm 5.01	14	8.55 \pm 4.54	18	0.109

3.4. Hematological Profile/Markers

Some hematological profile of the discordant subjects is shown in Table 6. The mean and standard deviation for all subjects was 12.98 \pm 1.26 g/dl for Hb concentration, 5.55 \pm 2.03 $\times 10^3$ cells/mm³ for white blood cells and 244.82 \pm 70.55 cells/mm³ for platelets. There was a significantly higher ($p < 0.001$) mean Hb concentration in males compared with the females. In contrast, no significant differences were observed in WBC and platelets between males and females. The hematological profile of concordant couples is shown in Table 7.

Table 6 Hematological profile of discordant couples (n = 90)

Variables	All (n = 90)	Males (n = 45)	Females (n = 45)	P-Value
Hemoglobin (g/dl)	12.98 \pm 1.26	13.89 \pm 0.87	12.07 \pm 0.87	<0.001
White Blood Cells ($\times 10^3$ cells/mm ³)	5.55 \pm 2.03	5.84 \pm 2.22	5.26 \pm 1.80	0.177
Platelets (cells/mm ³)	244.82 \pm 70.55	245.95 \pm 64.96	243.68 \pm 76.46	0.880

Table 7 Hematological profile of concordant couples (n = 30)

Variables	All (n = 30)	Males (n = 15)	Females (n = 15)	P-Value
Hemoglobin (g/dl)	13.19 \pm 1.25	14.18 \pm 0.72	12.20 \pm 0.77	<0.001
White Blood Cells ($\times 10^3$ cells/mm ³)	5.68 \pm 1.54	5.31 \pm 1.24	6.06 \pm 1.76	0.190
Platelets (cells/mm ³)	277.0 \pm 60.16	276.20 \pm 62.49	277.80 \pm 59.93	0.943

Table 8 Hematological profile of control subjects (n = 30)

Variables	All (n = 30)	Males (n = 15)	Females (n = 15)	P-Value
Hemoglobin (g/dl)	13.17 \pm 1.30	14.18 \pm 0.89	12.16 \pm 0.73	<0.001
White Blood Cells ($\times 10^3$ cells/mm ³)	5.31 \pm 1.95	5.52 \pm 1.65	5.10 \pm 2.25	0.572
Platelets (cells/mm ³)	239.93 \pm 59.74	237.26 \pm 51.28	242.60 \pm 68.91	0.812

The total mean and standard deviation for all subjects was 13.19 ± 1.25 g/dl for Hb concentration, $5.68 \pm 1.54 \times 10^3$ cells/mm³ for white blood cells and 277.0 ± 60.12 cells/mm³ for platelets. Also, there was a significantly higher ($p < 0.001$) mean Hb concentration in males compared with the females. And in contrast, no significant differences were observed in WBC and platelets between males and females. Also, some hematological profile of control subjects is shown in Table 8. The total mean and standard deviation for all subjects was 13.17 ± 1.30 g/dl for Hb concentration, $5.31 \pm 1.95 \times 10^3$ cells/mm³ for white blood cells and 239.93 ± 59.74 cells/mm³ for platelets. Independent sample t-test indicated significantly higher ($p < 0.001$) mean Hb concentration in males compared with the females.

Table 9 shows the logistic regression analysis of risk factors for HBV seropositivity among study participants. The univariate analysis indicated there was a significant negative association ($p = 0.002$) between HBV seropositivity and sex. The males were 9.87 less likely to have hepatitis B viral infection compared with the females. In contrast, no significant associations were observed between HBV seropositivity and age, drinking, smoking and history of previous vaccination.

Table 9 Logistic regression analyses of risk factors for HBV seropositivity among study participants (n = 120)

Risk Factors	N	HBV Seropositivity N (%)	Binomial Logistic Regression		
			Wald	OR (95% CI)	P - Value
Sex					
Males	60	29 (48.3)	9.87	0.285 (0.13-0.62)	0.002
Females	60	46 (76.7)		1 (reference)	
Age (yrs)					
20-30	21	12 (57.1)	0.312	1.33 (0.42-4.16)	0.620
31-40	71	49 (69.0)		2.22 (0.91-5.45)	0.080
Drinking					
No	64	45 (70.3)	3.52	2.05 (0.97-4.34)	0.060
Yes	56	30 (53.6)		1 (reference)	
Smoking					
No	114	70 (61.4)	1.06	0.32 (0.03-2.81)	0.303
Yes	6	5 (83.3)		1 (reference)	
Vaccination History					
No	113	75 (66.4)		1 (reference)	
Yes	7	0 (0)	0	0 (0)	0.998

4. Discussion

The risk of contracting HBV infection in Nigeria is substantial, not only due to low vaccination rate, poverty, sexual and social lifestyles, but also given the fact that as many populace as possible will be exposed. This study has demonstrated a varying percentage of detection rates of HBV markers in discordant and concordant partners (HBsAg - 62.5%, HBsAb - 4.2%, HBeAg - 2.5%, HBeAb - 50%, HBcAb - 60.8%), and in control partners (HBsAg - 0%, HBsAb - 23.3%, HBeAg - 0%, HBeAb - 0%, HBcAb - 6.7). While about 2.5% of both discordant and concordant partners showing active replication (HBeAg positive), the rest, especially the positive partners are in the chronic state (HBsAg +ve, HBcAb +ve and HBsAb -ve), and in rhythm with the predominant IgG response as reported elsewhere [23]. Also from the discordant and concordant partners, very few negative partners (4.2%) were seroconverted or immunized (HBsAg -ve, HBcAb +ve and HBsAb +ve: immune due to natural infection), and (HBsAg -ve, HBcAb -ve, and HBsAb +ve: immune due to vaccination). Development of HBsAb indicates clearance of infection and immunity from HBV infection [24]. Anti-HBs are known to replace HBsAg as acute HBV infection is resolving. About 2.5% of the positive hepatitis B subjects have hepatitis B envelope antigen (HBeAg) and this is an indication of an active replicating state and hence a highly infectious phase that could be a 70-90% chance of both vertical or horizontal transmission of the virus to others [25,26,27]. Also, about 50% of the positive hepatitis B subjects have HBeAb indicating that the patients were no longer in the active multiplying phase of the infection. Meanwhile, the control partners which were all negative to hepatitis B virus infection were all equally negative to HBeAg and HBeAb. About 23.3% of these control groups were positive to HBsAb, revealing that these numbers have been immunized. This partially agrees with the information on the questionnaire but still, few couldn't

know their vaccination status or had been naturally immunized. Meanwhile, about 6.7% of the control partners were positive for anti-HBc and since it could be persistence for life [23], shows that these group might have been exposed to the infection in the past. Also, this study has revealed an association ($p = 0.002$) between HBV seropositivity and sex, confirming that sex is a significant risk factor [22].

The mean values of ALT in this study were 11.04 iu/l in positive partners and 6.65 iu/l in negative partners while the mean values for AST are 12.64 iu/l in positive partners and 9.08 iu/l in negative partners, and the results show a significantly greater mean in both ALT and AST in positive subjects ($p < 0.001$) when compare with the negative partners. Overall, the sera tested elevated ALT levels were detected in 10.7% of the patients while AST elevated sera were 8%. The 10.7% incidence of elevated ALT levels in CHB patients gotten in this study was contrary to the work of Tulin *et al.* [28] that got 25.8%, but in consistence to the finding of Jebbar *et al.* [29] which agreed that ALT will be slightly raised in chronic HBV infection, and it is consistent with the previous results [30,31], indicating that in case of chronic HBV infection, 89% of the patients yielded continuously normal ALT levels, while 11% showed at least one ALT value above the normal levels. Previous studies have shown that ALT levels were higher in male patients than in female patients, [32], but this study that found higher ALT and AST levels in females can be attributed to the nature of the study group - discordant HBV partners.

Activities of the enzyme ALT was higher in the age range of 20 -30 years when compared to AST as there is no difference in the activity of AST in the age group. Both ALT and AST activities were increased in the age range of 31 – 40 years in hepatitis B positive partners. Some studies put AST activity slightly higher than ALT, up to 15 and 20 years of age in males and females [29]. In the case of adults, AST activity tends to be lower than that of ALT until age 60 [33].

Some selected hematological findings in this study showed no significant changes in the total WBC, haemoglobin concentration and platelet counts. This indicates that these parameters were relatively normal across all the groups investigated. However, while there was a significant higher Hb concentration among males when compared to the female's counterparts in both discordant, concordant and control partners, there was no significant difference in the mean values of total WBC and platelets. Our finding of normal WBC, Hb and platelets was in agreement with previous works in different locations [34, 35, and 36]. Our finding was however at variance with the findings of Fasola *et al.* and Onwuasoanya *et al.* where the focus of their findings was strictly on patients with acute hepatitis B (AHB) viral infection [37,38] while finding from this study confirmed that the positive hepatitis B participant was on chronic stage, which may be the reason for the discrepant results. During the acute phase of HBV infection in immunocompetent individuals, innate immunity generally plays a central role to limit the success of the virus while initiating the development of an adaptive immune response. Being central innate effector cells in viral infections, natural killer (NK) cell, monocyte and its derivatives, and other non-cellular components respond appropriately to eliminate viral infections by detecting the viral infection, hence the observable changes in hematological parameters (immunocytes) during the acute phase of hepatitis infection, unlike in chronic phase [39].

5. Conclusion

Effective management of any ailment starts with proper diagnosis and hepatitis B panel test inclusion in the routine investigation for of hepatitis B viral infection together with vaccination especially on sexual active individuals will contribute immensely in total eradication and control while periodic assessment of liver enzymes will ensure proper management of chronic hepatitis B infections.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors declare that they have no competing interests.

Statement of ethical approval

Ethical approval was obtained from the ethical committee of ESUT Teaching Hospitals, Parklane, Enugu, following the code of ethics for biomedical research involving human subjects.

Statement of informed consent

Informed consent was obtained from each participating individual and a questionnaire was administered to all the participating subjects.

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