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Immune Profiling of Blood Donors with Occult Hepatitis B Virus Infection: Analysis of IL-10, IL-12, TNF-α, and HLA Expression in Abuja, Nigeria

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Abstract

Background: *Occult hepatitis B virus infection (OBI) poses a hidden risk to blood transfusion safety, particularly in regions with high HBV endemicity such as Nigeria. The immunological mechanisms underlying viral persistence in OBI remain poorly defined. Cytokine regulation and HLA molecule expression may play critical roles in shaping host immune responses.*

Methods: *Blood samples from voluntary donors in Abuja, Nigeria, were screened for OBI using molecular assays. Immunological profiling included quantification of interleukin-10 (IL-10), interleukin-12 (IL-12), and tumor necrosis factor-alpha (TNF-α) levels, alongside assessment of HLA class I and II molecule expression.*

Results: *Donors with OBI demonstrated significantly elevated IL-10 concentrations, indicating a skew toward immunosuppressive regulation. In contrast, IL-12 and TNF-α levels were reduced, suggesting impaired pro-inflammatory and antiviral activity. Altered HLA class I and II expression patterns were observed, consistent with potential HBV immune evasion strategies.*

Conclusion: *The immunological profile of OBI donors in Abuja highlights a cytokine imbalance characterized by IL-10 dominance and reduced IL-12/TNF-α activity, coupled with altered HLA expression. These findings suggest that HBV persistence in OBI is facilitated by immune modulation and evasion, underscoring the need for enhanced donor screening and immunologically informed interventions to improve transfusion safety in Nigeria.*

Keywords: *Cytokines, IL-10, IL-12, TNF-α, MHC-HLA*0201 and OBI.*

INTRODUCTION

Occult hepatitis B virus infection (OBI) remains a major challenge in transfusion medicine, particularly in endemic regions such as Nigeria, where serological screening alone may fail to detect hidden carriers [3][32]. The persistence of HBV despite undetectable surface antigen (HBsAg) undersc\ores the need to explore host immunogenetic factors that influence viral control and transmission risk [9][13[. Over 250 million people are chronically infected with the Hepatitis B virus (HBV) worldwide. Sub-Saharan Africa, including Nigeria, bears a disproportionate burden of HBV, where high endemicity and limited access to advanced diagnostics complicate prevention and control efforts [30] [31]. Blood transfusion is a critical component of healthcare delivery [11]. Yet it also represents a potential route of HBV transmission, particularly in regions where screening relies primarily on detection of surface antigen (HBsAg) [11][17].

The persistence of HBV DNA in the absence of detectable HBsAg poses a significant risk and is capable of transmission [16][18]. The molecular basis of OBI involves the persistence of covalently closed circular DNA (cccDNA) within hepatocytes, serving as a stable reservoir for viral replication and reactivation [18][29]. However, host immune factors also play a decisive role in determining whether HBV is cleared, controlled, or persists in occult form [22]. Immunogenetic profiling provides a valuable lens through which to understand these host-virus interactions [18][24]. Immune-modulation through cytokines signaling where

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hypothesis to be the central regulators of antiviral immunity, influencing T-cell activation, inflammatory responses, and viral clearance [1][42]. Elevated IL-10 levels, for instance, may suppress effective immune responses [1], while IL-12 and TNF-α promote antiviral activity [38][46]. In parallel, human leukocyte antigen (HLA) molecules govern antigen presentation and shape the quality of T-cell responses. Specific HLA alleles have been linked to susceptibility or resistance to HBV persistence, underscoring the importance of host genetics in infection outcomes [39][45][49].

In Nigeria, where HBV prevalence remains high and blood transfusion services are vital, understanding the immunogenetic determinants of OBI among blood donors is essential [32][34]. This study therefore investigates cytokine expression patterns (IL-10, IL-12, TNF-α) and HLA molecule distribution in blood donors in Abuja. By integrating molecular detection with immunogenetic profiling, we aim to uncover host factors associated with OBI, thereby contributing to improved transfusion safety and informing public health strategies for HBV control in endemic regions.

OBI IMMUNOGENIC MECHANISM

The biological interplay between viral persistence and host immunogenetic factors that define occult hepatitis B virus infection (OBI) among blood donors [22][32]. Within hepatocytes, hepatitis B virus covalently closed circular DNA (cccDNA) serves as a stable nuclear reservoir, enabling continuous low-level viral replication even when surface antigen (HBsAg) is undetectable [20][25]. This molecular persistence forms the foundation of OBI, allowing the virus to evade standard serological screening and remain transmissible through blood donation [6][10]. A major contributor to this persistence is cytokine imbalance [1][40]. Elevated interleukin-10 (IL-10) suppresses antiviral immune responses by inhibiting T-cell activation and reducing inflammatory signaling [19][44]. In contrast, diminished levels of interleukin-12 (IL-12) [9][43] and tumor necrosis factor-alpha (TNF-α) weaken the body's ability to mount effective cellular immunity. IL-12 normally promotes Th1-type responses and cytotoxic T-cell activation, while TNF-α facilitates inflammation and the destruction of infected hepatocytes [21][41]. When these cytokines are downregulated, the immune system fails to clear infected cells efficiently, allowing HBV to persist at subclinical levels [37].

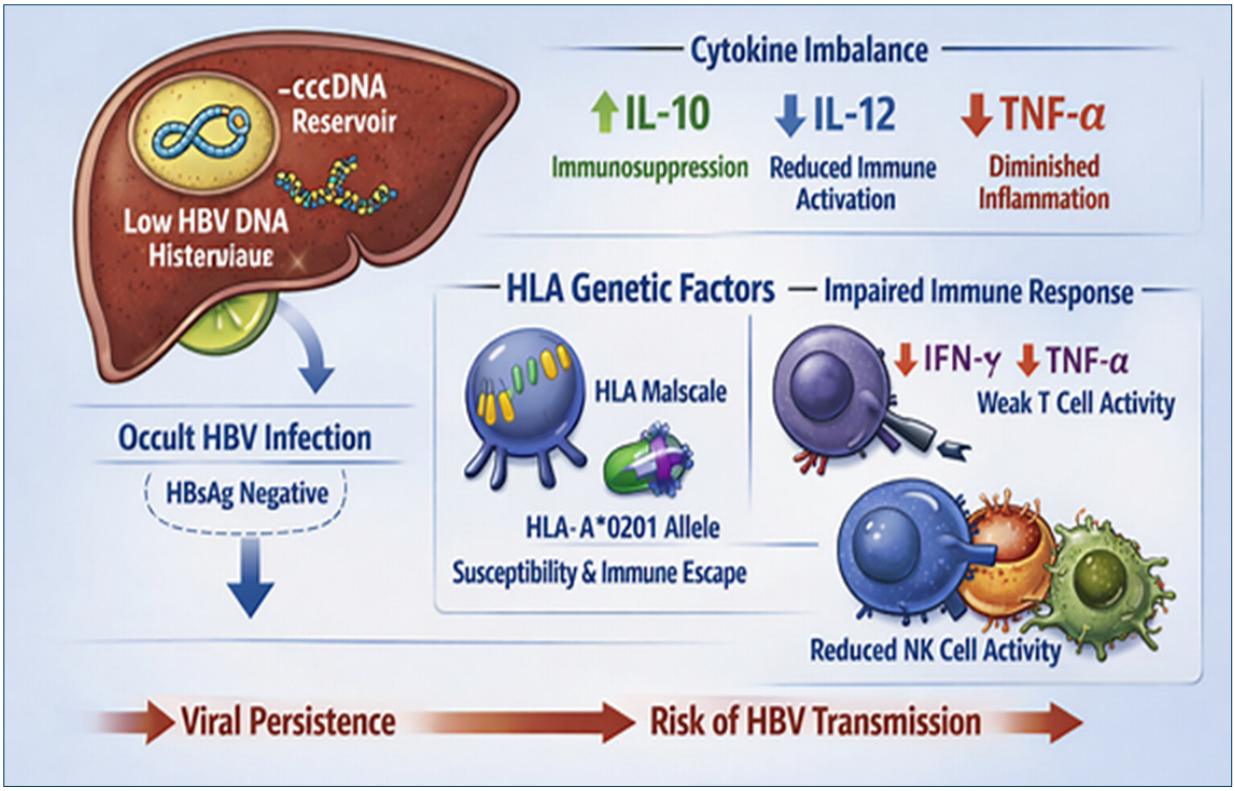


Figure 1. Immunological Bases of Occult Hepatitis B Interaction with Host Immune System Mediated by Cytokine and Expression to HLA Molecules in the Hepatocyte

Human leukocyte antigen (HLA) molecules further influence this dynamic by determining how viral peptides are presented to immune cells [35][50]. Certain HLA alleles are associated with increased susceptibility to HBV persistence, as they may present viral antigens less effectively or allow immune escape through altered peptide recognition [48][49]. This genetic variability among donors contributes to differences in immune control and the likelihood of developing OBI [45][47]. Effector immune cells, particularly $CD8^+$ cytotoxic T lymphocytes and natural killer (NK) cells, play a crucial role in viral clearance [8][50]. In OBI, these cells often exhibit reduced cytotoxic activity and impaired cytokine

production, such as lower interferon-gamma (IFN-γ) and TNF-α levels [36][40]. This functional exhaustion limits their ability to eliminate infected hepatocytes, perpetuating the cycle of low-level replication and viral persistence [32] [47]. Collectively, these mechanisms, cccDNA maintenance, cytokine dysregulation, HLA-mediated immune modulation, and weakened effector cell function, create a biological environment conducive to occult infection [12][15]. The result is a silent but significant risk of HBV transmission through blood transfusion, emphasizing the need for molecular and immunogenetic screening strategies to complement traditional serological tests in donor populations [7][38] [44]. The clinical relevance of these immune markers is critical in determining viral persistence versus clearance [2][10]. IL-10 is immunosuppressive, IL-12 promotes Th1 responses, TNF-α drives inflammation, and HLA molecules regulate antigen presentation [26][38][42]. Understanding how these immune factors influence OBI among blood donors in Abuja can improve transfusion safety and guide targeted interventions [4][5][21].

MATERIAL AND METHODS

A cross-sectional study was conducted among blood donors in Abuja. 7ml of samples were collected, centrifuged (3ml in a plain tube, 4ml in an EDTA container) from 250 HBsAg-negative blood donors. Both separated samples were stored at -20°C or lower until analysis to prevent degradation of viral markers. 4th generation Arial ELISA was deployed for evaluation of HBsAg, HCV while Determine test kit was used to establish HIV negativity, and HBV-DNA was determined using Tianlong techniques for NAT-PCR, with results expressed in (IU/ml). Data obtained were collated using an Excel spreadsheet and later analyzed using SPSS version 20. Student t-test and Chi-square test were conducted, with a *p*-value of 0.005 considered statistically significant.

Study Area

The study was conducted in Abuja comprising of secondary health and tertiary health institution in all the district general hospital of the area councils as depicted in the figure 2 below.

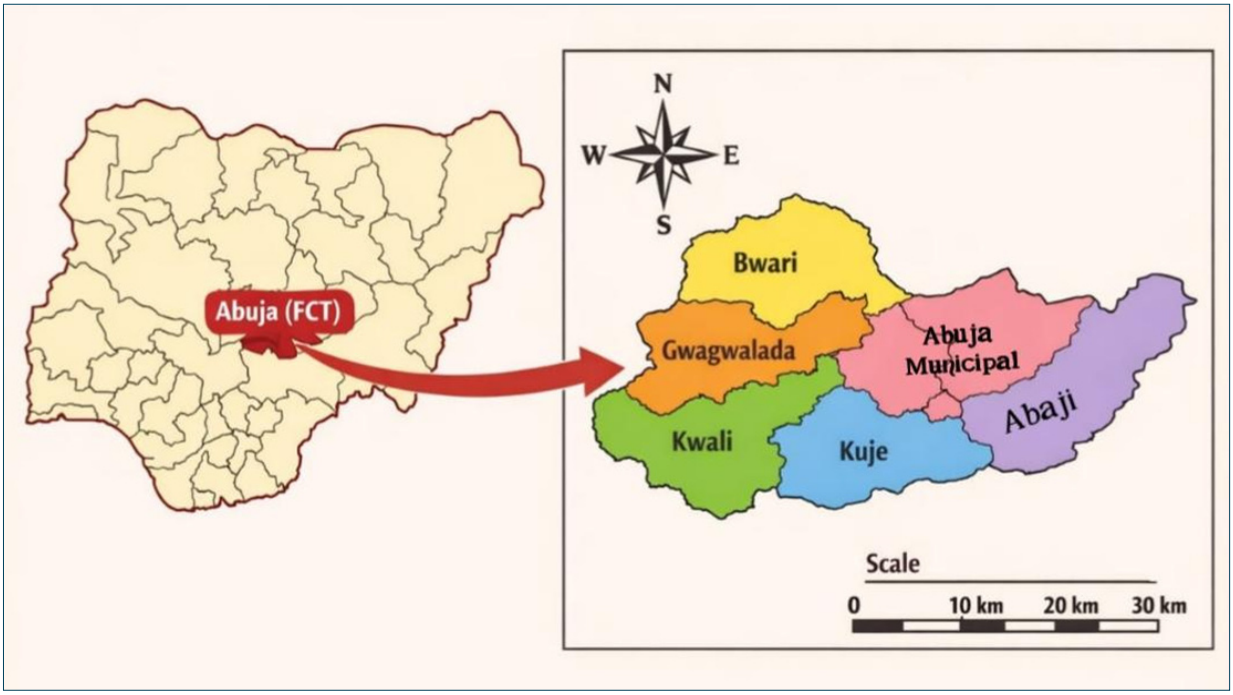


Figure 2. Map of Abuja, Nigeria showing the Location of the Health Institution Enrolled for this work in all the Abuja District

Ethical Approval

This study was approved by the ethical committee of the Federal Capital Territory Health Research Ethics Committee (FHREC/2024/01/73/14-03-2)

Inclusion and Exclusion Criteria

The study focused on apparently healthy, consenting blood donors aged 18–60 years who were negative for HIV, HCV, HBsAg, and syphilis, with special emphasis on HBsAg-negative individuals. Donors were excluded if they had serious medical conditions,

Were on chemotherapy or anti-TB drugs, were pregnant, had positive viral markers, were donating autologously, or lacked documented HBV vaccination records.

Data Analysis

Statistical analyses were conducted using SPSS version 29:2023. The chi-square test for independence, Fisher's exact test to compare the proportion of marker positivity between the groups and 2 x k test were used to determine whether the differences in health institution, sociodemographic, and location are statistically associated with OBI occurrence. Student's t-test was used to compare mean values of continues variable. A p-value of 0.05 was considered statistically significant. ANOVA was used to assess associations between

continuous variables, expressed as mean ± SD. Pearson correlation was used to evaluate the strength analysis measured the strength of relationships between variables, with correlation coefficients (r)

ANALYTICAL METHODS

Estimation of Human Serum Cytokines (IL-10, IL-12, and TNF-α)

Serum concentrations of interleukin-10 (IL-10), interleukin-12 (IL-12), and tumour necrosis factor-alpha (TNF-α) were determined using enzyme-linked immunosorbent assay (ELISA) kits according to manufacturers' instructions and established protocols (Walker et al., 2022; Chard, 1990). IL-10 and IL-12, ELISA kits from Enzo Life Sciences (UK) were employed. All reagents, standards, and samples were equilibrated to room temperature for 30 minutes before use. A blank well received 100 µl of diluent, while 100 µl of standards (1–7) were added to designated wells. Serum samples (50 µl) were pipetted into test wells, followed by 100 µl of antibody solution (except blank). Plates were incubated for 1 hour at room temperature on a shaker (500 rpm), then washed three times with 400 µl wash buffer. Subsequently, 100 µl of yellow antibody was added, incubated for 1 hour, and washed three times. Next, 100 µl of blue conjugate was added, incubated for 30 minutes, and washed three times. Substrate solution (100 µl) was added, incubated for 15 minutes, and stopped with 100 µl stop solution. Absorbance was measured at 450 nm using a Genesis MR 1200 microplate reader. Net optical density (OD) values were calculated by subtracting blank OD, and cytokine concentrations were interpolated from standard curves. TNF-α, a sandwich ELISA kit (WKEA Med Supplies Corp, China) was used. Fifty microliters of standards, controls, and serum samples were added to wells, mixed, covered, and incubated for 30 minutes at 37 °C. Wells were emptied, washed five times with 350 µl wash buffer, and blotted dry. Enzyme conjugate (50 µl) was added, incubated for 30 minutes at 37 °C, and washed five times. Substrate A (TMB) and substrate B (HRP) (50 µl each) were added, incubated for 15 minutes, and stopped with 50 µl stop solution. Absorbance was measured at 450 nm, and concentrations were extrapolated from calibration curves.

Estimation of MHC I-Strep HLA-A*0201

Serum levels of recombinant human MHC class I allele *HLA-A0201**, fused to a Twin-Strep-tag® (MHC I-Strep), were quantified using a sandwich ELISA kit (IBA Lifesciences GmbH, Goettingen, Germany) following the procedure described by Walker et al. (2022). Fifty microliters of appropriately diluted serum standards, controls, and samples were dispensed into designated microplate wells, mixed, covered, and incubated for 30 minutes at 37 °C. After incubation, wells were emptied and washed five times with 200 µl of diluted wash buffer, then blotted dry. Subsequently, *50 µl of HLA-A0201 enzyme conjugate** was added to each well, incubated for 30 minutes at 37 °C, and washed five times. Equal volumes (50 µl each) of substrate A (TMB) and substrate B (HRP) were added, followed by incubation for 15 minutes. The reaction was terminated with 50 µl stop solution, mixed gently, and absorbance was measured at 450 nm using a microplate reader. The average net optical density (OD) for each sample was obtained by subtracting the blank OD from the sample OD. These values were plotted against known concentrations of HLA-A*0201 standards (pg/ml) to generate a standard curve, from which sample concentrations were interpolated (Sanau et al., 2023).

RESULT

Table 1. Distribution of Sociodemographic Characteristics of Occult Hepatitis B Infection among Blood Donors in Abuja, Nigeria

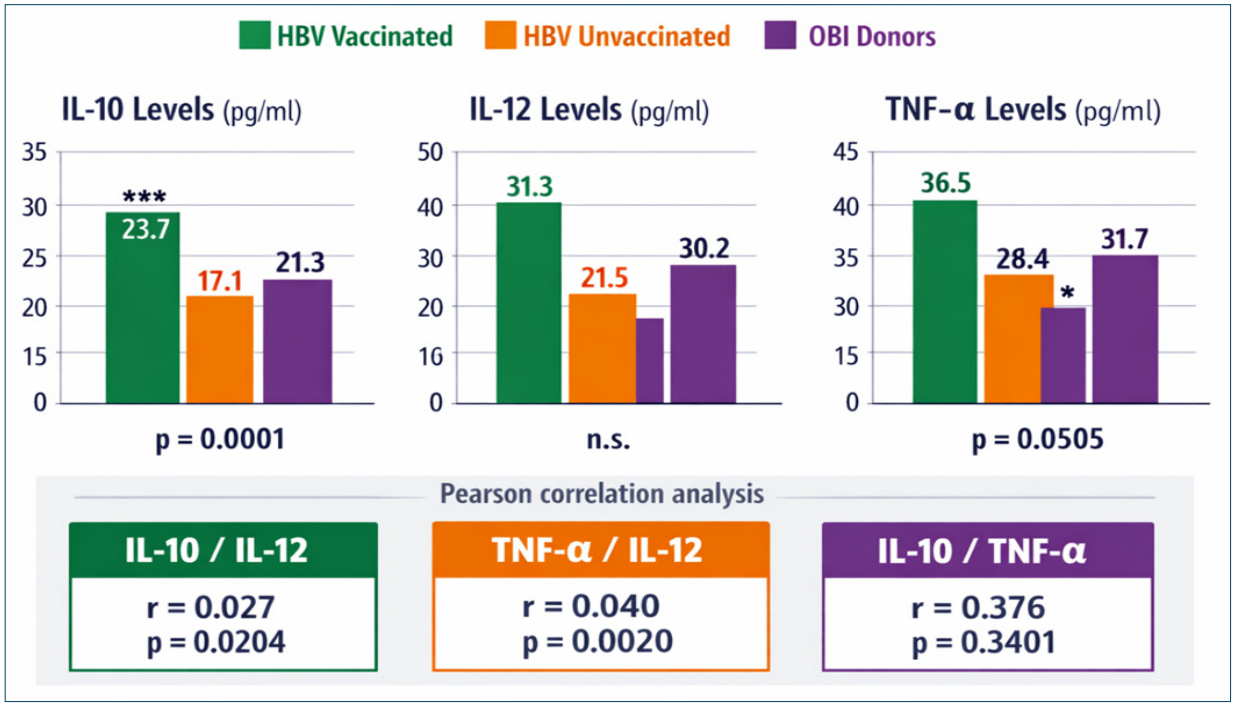
Predictor variable n=250	OBI Positive	OBI Negative (%)	Reference Predicted Group	Odds Ratio (aOR)	Z^2 or 95%CI	*p*-value
Age (years)						
>40	4 (1.6)	30(12.0)		3.07	1.8932	0.059
≤ 40	9 (3.6)	207 (82.8)	≤ 40 years	-	-	-
Gender						
Female	0 (0.0)	9 (3.6)		2.16	0.67 – 8.47	0.570
Male	13 (5.2)	228 (91.2)	Male	-	-	-
Education						
> Secondary	9 (3.6)	168 (67.2)		1.08	0.32-3.63	0.858
≤ Secondary	4 (1.6)	69 (27.6)	≤ Secondary	-	-	-
Type of donor						
Commercial	9 (3.6)	99 (39.6)		3.14	0.94-10.46	0.063
Non-commercial	4 (1.6)	138 (55.2)	Noncommercial	-	-	-

Z^2 = Trend Z Chi statistic, n= number of tests, %= percentage, OBI=occult hepatitis B infection, OR=odd ratio, CI= confidence interval, >= greater than, ≤= less than or equal to

Table 2. A Comparison of Anti-inflammatory, Pro-Inflammatory Cytokines and MHC-HLA-A0201 Concentration among Vaccinated, Unvaccinated, and OBI Donors in Abuja.

Cytokines and HLA	HBV vaccinated Blood donor (n=37)	HBV unvaccinated Blood donor (n=58)	OBI donors (n=13)	*F*-test	*p*-value
IL-10 (pg/ml)	23.7 ± 1.3	17.1 ± 1.6	21.3 ± 3.3	7.3410	0.0001
IL-12 (pg/ml)	31.3 ± 2.3	21.5 ± 1.1	30.2 ± 2.1	0.8672	0.4234
TNF-α (pg/ml)	36.5 ± 2.4	28.4 ± 2.1	31.7 ± 2.8	4.0763	0.0505
MHC-HLA*0201 (pg/ml)	23.4 ± 3.1	20.7 ± 2.5	21.2 ± 1.3	1.1507	0.3209
Pearson correlation analysis	r(p)	r(p)	-		-
IL-10 /IL-12	0.027(0.0204)	0.059 (0.0802)	-		-
TNF-α/ IL-12	0.040 (0.0020)	0.048 (0.0691)			
IL-10/ TNF- α	0.376 (0.3401)	0.042 (0.0462)			

IL=interleukin, TNF-α = interferon alpha, n= number (%) = percentage, HBV = hepatitis B virus * = significant association, pg/ml= pico-gram per liter, r = regression coefficient, MHC = major histocompatibility complex, HLA = human leukocyte antigen, µL = microliter, ml= milliliter



DISCUSSION

The association of occult hepatitis B infection with demographic characteristics revealed that age and sex -related analysis (p >0.005) did not reach statistical significance. However, the trend suggested that older donors were more likely to harbor occult infection, reflecting cumulative lifetime exposure to HBV and declining immunity over time. Although not statistically significant, data from this work indicate that occult hepatitis B infection prevalence is observed across all educational levels and ages, with a slight increase among males and being most notable among commercial donors. The observed trends offer valuable epidemiological insights.

The main findings suggest that older age and commercial donor status are the strongest sociodemographic indicators of occult hepatitis B infection among blood donors in Abuja, with both variables showing elevated odds ratios and borderline statistical significance. These results highlight the need for targeted interventions, including increased HBV vaccination and screening among older and commercial donors, while continuing to promote voluntary, non-remunerated blood donation to improve blood safety. This report agrees with a report by Ahmad et al. (2019), who found the highest age distribution among blood donors to be in the 30- to 40-year age group. A mean age of 50 ± 15 years was reported in Germany (Xialian et al., 2021). Research by Peter et al. (2020) reported a mean age of 19.5 ± 1.8 years among school-going adolescent voluntary blood donors in Kenya, focusing on occult hepatitis B virus infection and risk.

The mean concentration of serum anti-inflammatory (IL-10)

and proinflammatory cytokines (IL-12, TNF-α), and human leukocyte antigen (MHC-HLA-A0201). Our findings revealed the serum concentration of IL-10 (ng/l) as 23.7 ± 1.3 among vaccinated blood donors, 17.1 ± 1.6 in unvaccinated blood donors, while the concentration of IL-10 (ng/l) among blood donors with occult hepatitis B virus infection is computed at 21.3 ± 3.3 (ng/l with a value <0.001. Similarly, serum concentration of IL-12 (ng/l) showed a concentration of 31.3 ± 2.3 among vaccinated blood donors, 21.5 ± 1.1 among unvaccinated blood donors, while the concentration of IL-12 (ng/l) among blood donors with occult hepatitis B virus infection is computed at 30.2 ± 2.1 (ng/l), respectively, with p=0.423. Serum tumor necrosis factor-alpha (TNF-α) reveals a serum concentration of 36.5 ± 2.4 among vaccinated blood donors, 28.4± 2.1 among unvaccinated blood donors, while the concentration of TNF-α (ng/l) among blood donors with occult hepatitis B virus infection is computed at 31.7 ± 2.8 (ng/l), respectively, with p=0.4234. Conversely, the measurement of serum concentration of MHC-HLA0201 (ng/l) reveals the mean concentration of 23.4 ± 3.1 among vaccinated blood donors, 20.7 ± 2.5 in unvaccinated, and 21.2 ± 1.3 among blood donors confirmed with OBI, with p = 0.321.

The serum concentration of IL-10 (ng/l) reveals a statistically significant difference (P=0.001) in IL-10 levels among the three groups, with vaccinated donors having higher IL-10 levels compared to unvaccinated and OBI donors. IL-10 is an anti-inflammatory cytokine, suggesting that vaccination may enhance anti-inflammatory responses. The observed no statistically significant difference (p=0.4234) in IL-12 (gn/l) indicates the pro-inflammatory cytokine, and similar levels across groups indicate that vaccination and OBI status do not significantly alter IL-12 concentrations. Determination of tumor necrosis factor-alpha (TNF-**α**) shows that there is a statistically significant difference (p=0.05) in TNF-α levels among the groups, with vaccinated donors having higher TNF-α levels compared to unvaccinated donors. A significant level of TNF-α is a pro-inflammatory cytokine, suggesting that vaccination may enhance pro-inflammatory responses, which are part of the immune response. The MHC-HLA0201: No statistically significant difference (p=0.3215) in MHCHLA0201 levels among the groups. MHC-HLA0201 is involved in antigen presentation and immune response regulation.

Similar levels across groups suggest that vaccination and OBI status do not significantly alter MHC-HLA0201 concentrations. The significant differences in IL-10 and TNF-α levels between vaccinated and unvaccinated donors highlight the impact of HBV vaccination on cytokine responses. The increase in both anti-inflammatory (IL-10) and proinflammatory (TNF-α) cytokines suggests a balanced immune modulation by the vaccine. The lack of significant differences in IL-12 and MHC-HLA0201 levels indicates that these cytokines are not as affected by vaccination status. These findings contribute to our understanding of the immunological effects of HBV vaccination and its potential implications for blood transfusion safety.

The result further demonstrated Pearson correlation analysis of anti-inflammatory and pro-inflammatory cytokines among vaccinated and unvaccinated blood donors. The correlation of IL-10 and IL-12 reveals that there is a weak positive correlation (r = 0.027) that is statistically significant (p = 0.020), suggesting a minimal but significant association between IL-10 and IL-12 levels among vaccinated donors. Also, a weak positive correlation (r = 0.059) with not statistically significant (p = 0.080), indicating no significant association between IL-10 and IL-12 levels among unvaccinated donors. Correlation data of TNF-α / IL-12 also demonstrated a weak positive correlation (r = 0.040) that is statistically significant (p = 0.002), indicating a significant association between TNF-α and IL-12 levels among vaccinated donors. Similarly, our findings show that there is a weak positive correlation (r = 0.048) that is not statistically significant (p =0.0691), indicating no significant association between TNF-α and IL-12 levels among unvaccinated donors.

Data obtained from the correlation of IL-10 / TNF-α reveals a moderate positive correlation (r = 0.376) that is not statistically significant (p=0.3402), suggesting no significant association between IL-10 and TNF-α levels among vaccinated donors. the correlation among HBV Unvaccinated shows a weak positive correlation (r = 0.042) that is statistically significant (p = 0.0462), indicating a significant but minimal association between IL-10 and TNF-α levels among HB-unvaccinated donors. Cytokines study from the research revealed that HB-vaccination was found to be significantly increased in both IL-10 (pg/ml) and TNF-α (pg/ml) levels in blood donors, indicating enhanced anti-inflammatory and pro-inflammatory responses. This dual modulation suggests that vaccination supports viral clearance while limiting immune-mediated damage.

Pearson correlation analysis (r/p - value) demonstrates the impact of HB vaccination on cytokine responses. The data presented highlighted a significant correlation among vaccinated blood donors, with statistically significant correlations found in vaccinated donors (IL-10 / IL-12 and TNF-α / IL-12). This suggests that HBV vaccination may enhance the coordinated immune response, promoting a balanced regulation between anti-inflammatory and pro-inflammatory cytokines. This balance is crucial for effective immune regulation and avoiding excessive inflammation. The absence of significant correlations in unvaccinated donors suggests that their cytokine interactions were less coordinated. This might be due to a lack of vaccination-induced immune regulation, leading to a more erratic or less controlled immune response.

Our findings differ from the report obtained by Zhang et al. (2022), who observed that IL-10 responses were elevated in

higher infection states, emphasizing its role in HBV-specific cellular immunity and persistence of OBI. Similarly, our findings agree with Sosa-Jurado et al. (2021), who confirmed that TNF-α is a potent cytokine involved in antiviral defense and inflammatory liver injury. In OBI donors, TNF-α was shown to suppress HBV replication, although excessive TNF-α could contribute to hepatocyte damage. This supports our observation that TNF-α plays a dual role in both viral control and liver pathology.

The correlation between IL-10 and TNF-α in HBV-vaccinated donors was not statistically significant; however, the moderate association indicated a potential interplay between anti-inflammatory and pro-inflammatory responses. This balance could be indicative of a more effective immune modulation achieved through vaccination. Consequently, the minimal but statistically significant correlation between IL-10 and TNF-α in HBV-unvaccinated donors pointed towards some level of interaction between these cytokines, although the relationship was weaker compared to HBV-vaccinated blood donors. This aligns with the broader immunopathogenesis framework described by Sultan (2013), who emphasized that elevated IL-10 suppresses antiviral immunity, reduced IL-12 impairs Th1 activation, and TNF-α contributes to viral control but also to inflammatory injury.

Although the vaccine is known to induce a transient increase in proinflammatory and anti-inflammatory cytokines, the time of vaccination was not assessed. This observed difference between vaccinated blood donors may reflect long-term immune memory or underlying immunological variation and not vaccine-induced inflammation (post vaccination inflammatory responses).

Analysis of MHC-HLA0201* revealed no statistically significant difference ($p = 0.3205$) in levels among the groups. Since MHC-HLA0201 is involved in antigen presentation and immune response regulation, similar levels across vaccinated and unvaccinated donors suggest that vaccination and OBI status do not significantly alter the expression of this molecule. This finding is consistent with the report of Ziegler et al. (2014), who noted that HLA0201 expression remains stable across different HBV infection states, underscoring its role as a fundamental antigen-presenting molecule rather than a variable marker influenced by vaccination or occult infection.

Furthermore, immunogenetic studies have emphasized that HLA class I molecules, including HLA-A0201, are critical for presenting HBV peptides to CD8+ T cells. In OBI, the efficiency of antigen presentation by HLA-A0201 influences viral persistence, as strong responses can suppress HBV replication without fully eliminating the virus (Rehermann & Bertoletti, 2015). Similarly, Chen et al. (2019) demonstrated that individuals with OBI often had detectable HBV DNA despite robust HLA-A0201-restricted CD8+ T-cell responses, suggesting that while this allele contributes to viral suppression, it may also facilitate the occult state by maintaining replication at low but persistent levels. In addition, Al-Qahtani et al. (2014) reported that HLA-A0201 was associated with stronger cytotoxic T-cell responses but incomplete viral clearance, supporting its role in maintaining HBV DNA at low levels without HBsAg detection.

CONCLUSION

The immune response to occult hepatitis B infection (OBI) reflects a delicate balance between viral persistence and host immune regulation. Cytokines such as interleukin-10 (IL-10), interleukin-12 (IL-12), and tumor necrosis factor-alpha (TNF-α) play pivotal roles in shaping the immune response. IL-10, an anti-inflammatory mediator, may suppress antiviral immunity and facilitate viral persistence. Conversely, IL-12 and TNF-α are pro-inflammatory cytokines that promote Th1 responses and cytotoxic activity, essential for viral clearance. Dysregulation of these cytokines could explain why HBV remains hidden in OBI cases. Equally important is the role of human leukocyte antigen (HLA) expression, which governs antigen presentation and T-cell activation. Specific HLA alleles and expression patterns may predispose individuals to OBI by modulating immune recognition of HBV epitopes. Thus, profiling cytokine levels and HLA expression provides critical insight into the immunological landscape of OBI.

In Abuja, Nigeria, immune profiling of blood donors with OBI offers a unique opportunity to identify biomarkers of silent HBV carriage, understand host-pathogen interactions, and improve transfusion safety. By analyzing IL-10, IL-12, TNF-α, and HLA expression, this study seeks to unravel the immunological mechanisms underlying occult infection and contribute to strategies for HBV control in high-prevalence regions.

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