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Original Research Article

Integrated Serological, Hematological, and Inflammatory Marker Profiles of Hepatitis B Virus Infection among Individuals in Abakaliki Metropolis

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Abstract: ***Background*:** Hepatitis B virus (HBV) infection remains a major public health challenge in sub-Saharan Africa, including Nigeria. In addition to hepatic damage, HBV disrupts haematopoiesis, iron metabolism, and immune regulation, resulting in altered haematological and inflammatory profiles. Understanding these changes may improve disease monitoring and patient management. ***Objective*:** This study evaluated the serological status of HBV and the associated variations in haematological parameters, serum ferritin, and interleukin-6 (IL-6) among individuals in Abakaliki metropolis, Ebonyi State, Nigeria. ***Methods*:** A descriptive cross-sectional study was conducted among 1,000 adults recruited from selected healthcare facilities in Abakaliki. Venous blood samples (6 mL) were collected and separated into EDTA and plain tubes for haematological and biochemical analyses. HBV screening was carried out using a rapid diagnostic test, and serological markers were determined using ELISA. Haematological parameters were analysed using an automated analyzer, while ferritin and IL-6 were measured using ELISA and fluorescence immunoassay methods, respectively. Statistical analysis was performed at a significance level of $p < 0.05$. ***Results*:** Active HBV infection occurred in 7.5% of participants, while 30.8% had inactive infection, 27.5% were vaccinated, and 16.7% were uninfected. Significant variations were observed in RBC, Hb, PCV, MCV, MCH, MCHC, RDW, TWBC, and platelet count across the groups ($p < 0.05$). The active infection group showed the lowest RBC and platelet counts but the highest TWBC and IL-6 levels. Ferritin was lowest in active cases and highest in vaccinated individuals. Gender analysis revealed similar trends in males and females, though lymphocyte and monocyte counts were significantly higher in males with active infection. ***Conclusion*:** HBV infection significantly alters haematological indices and inflammatory markers, particularly in individuals with active disease. Routine monitoring of these parameters may support improved clinical assessment and disease management.

Keywords: Hepatitis B virus, Haematology, Ferritin, IL-6, Abakaliki, Nigeria.

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INTRODUCTION

Hepatitis B virus (HBV) is an enveloped virus with a partially double-stranded circular DNA genome and belongs to the *Hepadnaviridae* family [1]. The virus replicates within hepatocytes, disrupting normal liver function and activating the host immune system, which attempts to eliminate the infection but also contributes to liver inflammation. Globally, HBV remains a major public health problem, infecting millions and causing both acute and chronic liver diseases, including cirrhosis, liver failure, and hepatocellular carcinoma (HCC), which may be fatal [2, 3]. HBV possesses three key antigens—surface antigen (HBsAg), core antigen (HBcAg), and e-antigen (HBeAg) [1]. Electron microscopy typically reveals three structural forms of HBV: small spherical particles (20 nm), filamentous forms (20 × 200 nm), and the Dane particle (42 nm), the infectious viral particle [4].

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Modes of Transmission

Most HBV-infected individuals appear healthy and asymptomatic, yet they may be highly infectious. HBV is extremely contagious and can be transmitted through several routes, including perinatal transmission, horizontal transmission among children, sexual contact, and injection drug use[5]. Although improved screening and infection-control measures have reduced transmission through contaminated blood and unsafe medical procedures, healthcare-associated infections remain a concern globally [6]. Major transmission routes include:

- **Vertical Transmission**, especially common in highly endemic regions such as East Asia and sub-Saharan Africa.
- **Horizontal Transmission** through close contact, contaminated instruments, exposure to infected blood, unprotected sex, and unscreened blood transfusion.

HBV has been detected in saliva, tears, breast milk, sweat, and urine; however, transmission through these fluids is rare unless blood is present. Breastfeeding does not significantly increase the risk of infection. The virus cannot penetrate intact skin or mucous membranes; even minor breaches facilitate entry [5]. HBV is capable of surviving on environmental surfaces (e.g., razors, tabletops) for up to one week without losing infectivity. The virus infects hepatocytes through low-affinity binding between HBsAg and heparan-sulfate proteoglycans (HSPG), followed by high-affinity binding of the pre-S1 region to the NTCP receptor, enabling viral entry.

Epidemiology of Hepatitis B Virus Infection

According to WHO, an estimated 254 million people worldwide live with chronic hepatitis B, representing a global prevalence of 3.8% [7, 8]. Approximately 1.5 million new cases and 1.1 million HBV-related deaths occur annually, mainly due to cirrhosis and hepatocellular carcinoma. Prevalence varies geographically: sub-Saharan Africa, East Asia, and the Pacific exhibit high prevalence (≥8%); Eastern Europe, the Middle East, and South America show intermediate rates (2–7%); while North America, Western Europe, and Australia remain below 2% [8].

WHO (2022) further reports regional burdens of 97 million cases in the Western Pacific, 65 million in Africa, 61 million in Southeast Asia, 15 million in the Eastern Mediterranean, 11 million in Europe, and 5 million in the Americas. In Nigeria, HBV prevalence is substantial, estimated at 8.1% among individuals aged 15–64 years [9], placing the country among the top ten nations most affected by hepatitis B globally. Variability exists across regions, but the southeastern zone aligns with national patterns, partly due to inadequate awareness [10].

In Ebonyi State, a 2023 study reported a 5.3% prevalence of HBsAg among pregnant women, with 6.3% coinfected with HIV and HBV [11]. A statewide survey found a 6.9% overall HBV prevalence; vaccinated individuals had only 1.0% prevalence, while unvaccinated individuals showed 8.5% [12]. Among donkey butchers in the state, HBV prevalence was reported at 8.0% [13].

Stages of Hepatitis B Virus Infection

HBV infection occurs in two major clinical forms—acute and chronic—which reflect the dynamic interaction between the virus and host immunity and guide diagnosis and management.

Acute Hepatitis B Infection

Acute HBV occurs after initial exposure, with an incubation period of 1–4 months. Key stages include:

1. **Incubation Period (30–180 days):** Silent replication in hepatocytes; HBV DNA and HBsAg detectable; minimal immune response.
2. **Prodromal (Pre-icteric) Phase (days to 2 weeks):** Symptoms such as malaise, anorexia, fever, nausea, myalgia; positive HBsAg and HBeAg; rising HBV DNA.
3. **Icteric Phase:** Jaundice, dark urine, pale stools; elevated liver enzymes; active immune-mediated hepatocyte destruction.
4. **Convalescent/Recovery Phase:** 90–95% of adults clear the infection, losing HBsAg and developing anti-HBs; anti-HBc IgG persists. A small proportion progresses to chronic infection.

Chronic Hepatitis B Infection

Chronic HBV is defined by persistence of HBsAg for ≥6 months. Disease progression depends on viral factors and host immunity and follows five evolving phases:

1. **Immune-Tolerant Phase:** High viral replication with minimal liver damage; HBeAg-positive, high HBV DNA, normal ALT. Common in perinatally infected individuals[14].
2. **Immune-Active Phase:** Strong immune attack on infected hepatocytes, causing inflammation and fibrosis; elevated ALT, high HBV DNA, HBeAg-positive; risk of cirrhosis and HCC [14].
3. **Inactive Carrier Phase:** Low viral replication following HBeAg seroconversion; low/undetectable HBV DNA, normal ALT; minimal inflammation, but reactivation is possible.
4. **Reactivation Phase (HBeAg-Negative Chronic Hepatitis):** Mutant HBV strains allow active replication without HBeAg; high HBV DNA and liver inflammation; high risk of cirrhosis.

5. **Resolved/Occult Infection:** HBsAg disappears, anti-HBs and/or anti-HBc present; HBV DNA is undetectable or very low. In occult infection, HBsAg is negative but low-level HBV DNA persists.

Hepatitis B Infection Status

Hepatitis B infection status describes an individual's infectiousness and clinical condition based on serological markers, viral load, and clinical history. It identifies whether a person is susceptible, acutely infected, chronically infected, vaccinated, immune, or carries occult infection. Highly infectious individuals are typically HBsAg-positive, HBeAg-positive, and have high HBV DNA levels [15].

1. **Susceptible:** Never infected or vaccinated; HBsAg, anti-HBs, and anti-HBc all negative; not infectious but at high risk if exposed.
2. **Acute Infection:** Recent infection (<6 months); HBsAg and anti-HBc IgM positive; high HBV DNA; may progress to chronicity.
3. **Chronic Active Infection:** Ongoing viral replication with liver inflammation; highly infectious; increased risk of cirrhosis and HCC.
4. **Other stages** include inactive carrier, resolved infection, immune after vaccination, and occult infection, each defined by specific antigen/antibody patterns and degree of infectivity.

Haematological parameters provide essential information about blood composition and are vital for diagnosing numerous medical conditions [16]. A Complete Blood Count (CBC) evaluates hemoglobin levels, red and white cell numbers, platelet counts, and cell morphology, offering insight into both hematologic and systemic disorders [17]. Blood cell production begins in fetal tissues but becomes confined to the bone marrow after birth, where pluripotent stem cells differentiate into erythroid, myeloid, lymphoid, and megakaryocytic lineages [18]. Because the liver regulates clotting, thrombopoietin production, and iron metabolism, liver diseases such as hepatitis B significantly alter hematopoiesis and peripheral blood indices [18].

Hepatitis B virus (HBV) infection affects blood formation indirectly through immune-mediated liver injury, systemic inflammation, cytokine release, and metabolic disturbances. Changes commonly observed include anemia, leukopenia or leukocytosis, thrombocytopenia, and prolonged coagulation times, with severity depending on disease stage, cirrhosis, hypersplenism, and antiviral therapy [19]. HBV also influences red cell indices (MCV, MCH, MCHC) and white cell differentials, while platelet reduction often results from reduced thrombopoietin, splenic sequestration, or immune destruction[20]. Ferritin levels rise in HBV due to inflammation, hepatocyte damage, altered hepcidin regulation, and, in some patients, true hepatic iron overload, making ferritin a biomarker for liver injury, fibrosis, and hepatocellular carcinoma risk [20].

Interleukin-6 (IL-6) plays a central role in HBV infection by mediating inflammation, acute-phase responses, and immune activation. While IL-6 can inhibit HBV entry and replication through NTCP downregulation and reduced cccDNA formation, elevated IL-6 is also associated with liver injury, fibrosis, and poor outcomes in severe disease. HBV antigens and the HBx protein stimulate IL-6 production via TLR activation and NF-κB/STAT3 pathways, reflecting its dual protective and pathogenic functions. Overall, the interaction between HBV, hematopoiesis, iron metabolism, and IL-6 signaling shapes the clinical hematological profile of infected individuals [21].

MATERIALS AND METHODS

Study Area

The study was carried out in Abakaliki Metropolis, the capital of Ebonyi State in South-East Nigeria. The area lies around latitude 6°19´N and longitude 8°06´E and comprises major urban communities such as Ugwuachara, Amike-Aba, and Azuiyiokwu. These locations host key healthcare facilities—including Mile Four Hospital, Alex Ekwueme Federal Teaching Hospital, and the Mother and Child Health Centre—which provide comprehensive clinical and laboratory services for infectious diseases, making the area suitable for HBV surveillance.

Study Population

A total of 1,000 adult participants were recruited from outpatient clinics, antenatal units, and HBV follow-up clinics of the selected hospitals in Abakaliki.

Study Design

A descriptive cross-sectional design was employed to determine HBV prevalence, investigate likely causes of infection, and evaluate associated serological, haematological, inflammatory, and iron-regulatory biomarkers among residents of the metropolis.

Sampling Technique

Convenience sampling was used.

Sample Size Determination

Sample size was calculated using Araoye (2004). Using an HBV prevalence of 8.5%, a 95% confidence level (Z = 1.96), and a 5% margin of error, the minimum required sample size was approximately 120.

Ethical Consideration

Ethical approval was obtained from the relevant hospital ethics committees, and informed written consent was secured from each participant.

Selection Criteria

Inclusion: HBV-positive adults (drug naïve or on treatment), individuals with co-infections, and those not vaccinated.

Exclusion: HBV-negative individuals, children, and clinically unsuitable controls. Healthy HBV-negative individuals served as controls.

Sample Collection: Six millilitres of venous blood were collected from each participant—4 mL into plain tubes for ferritin and IL-6 assays, and 2 mL into EDTA tubes for haematological analysis. Serum samples were centrifuged at 3000 rpm for 10 minutes and stored at –80°C before analysis.

Laboratory Analysis

HBV Screening

Initial screening was done using the Anbio Rapid Test Kit based on rapid diagnostic methods, identifying 100 HBV-positive individuals.

Hepatitis B Marker Determination

HBV serological markers (HBsAg, HBsAb, HBcAb, HBeAg, HBeAb) were assessed using the B-Panel ELISA-based Colloidal Gold kit following Xiaohua Li (2004). Infection status categories included: uninfected controls (n=20), inactive (n=33), inactive chronic (n=37), active infection (n=9), and indeterminate window period (n=21).

Haematological Analysis

Haematological parameters were measured using the Zybio Z31 Autoanalyzer, applying electrical impedance and optical principles.

Serum Ferritin

Ferritin levels were determined using the AccuBind ELISA Microwell kit following the sandwich ELISA principle.

Interleukin-6 Determination

IL-6 was measured using the Anbio Fluorescence Immunoassay (FIA) rapid test kit based on sandwich immune detection.

Statistical Analysis

Data were expressed as mean ± SD. Yates' chi-square, Wilcoxon, Student's t-test, or Kruskal–Wallis tests were used where appropriate. Pearson correlation assessed relationships among variables, with significance set at $p < 0.05$.

RESULTS

Table 1: Hepatitis B virus infection status of the studied participants

Infection status	Number observed	Percentage (%)
Uninfected (control)	20	16.7
Immune (vaccinated) to infection	33	27.5
Inactive infection	37	30.8
Active infection	9	7.5
Unclear (window period)/ indeterminate HBV infection	21	17.5

There were significant variations in several hematological indices—such as RBC, Hb, PCV, MCV, MCH, MCHC, RDW-CV, RDW-SD, TWBC, and platelet count—among the vaccinated/immune, actively infected, and control groups. Comparable trends were observed for ferritin and interleukin-6 (IL-6), as outlined in Table 2 describing the clinical features of the study population. The vaccinated group recorded the highest mean RBC value (4.55 ± 0.82), whereas individuals with active infection had the lowest mean RBC (3.71 ± 0.89). These differences in RBC between the active group and uninfected, immune, and vaccinated individuals were statistically significant ($p = 0.010$, $p = 0.002$, and $p = 0.030$, respectively).

Mean MCHC levels were reduced in both the inactive infection group (31.40 ± 2.13) and the vaccinated group (31.65 ± 2.02) when compared with the control group (33.42 ± 1.39), and these differences were statistically significant ($p < 0.001$ for both comparisons and $p < 0.006$, respectively). In addition, significant differences were seen in mean RDW-SD and RDW-CV between the actively infected group and the uninfected group (22.29 ± 2.32 vs. 28.39 ± 3.32), between the active and vaccinated groups (22.29 ± 2.32 vs. 31.47 ± 4.35), and between active and inactive infection (22.29 ± 2.32 vs. 27.14 ± 4.67), with all comparisons reaching statistical significance ($p \leq 0.030$ and $p < 0.001$).

The highest mean total white blood cell count (9.32 ± 3.63) was observed in the actively infected group, while the lowest mean platelet count (120.51 ± 69.50) was also recorded in this group. Comparisons between the active infection group and the other groups (uninfected, inactive, and vaccinated) showed statistically significant differences in both TWBC and platelet count ($p < 0.001$ and p ranging from 0.002 to 0.010).

With respect to ferritin and IL-6, the active infection group exhibited the lowest mean ferritin concentration (26.18 ± 18.42) and the highest mean IL-6 level (108.71 ± 122.52). The difference in ferritin between the active and vaccinated groups was statistically significant ($p < 0.001$), whereas differences with the other groups were not. In contrast, IL-6 levels

were significantly higher in the active infection group compared with the uninfected, vaccinated, inactive, and indeterminate groups ($p < 0.001$ in all comparisons, except vaccinated where $p = 0.001$).

Table 2: Haematological, ferritin and IL-6 levels of studied participants based on their infection status

Haematolgoical parameters	Uninfected	Vaccinated	Inactive infection	Active infection	Indeterminate infection	p-value
RBC ($x10^{12}$/L)	4.45 ± 0.49	4.55 ± 0.82	4.18 ± 0.65	3.71 ± 0.89[a]	4.21 ± 0.64	0.015
Hb (g/dl)	12.89 ± 1.22	12.84 ± 1.03	12.24 ± 1.65	10.40 ± 2.56[a]	12.00 ± 0.97[b]	<0.001
PCV (%)	40.45 ± 4.53	40.51 ± 4.46	40.44 ± 5.78	35.79 ± 6.46[c]	37.38 ± 4.20	0.035
MCV (fL)	89.75 ± 4.51	86.06 ± 7.55	85.04 ± 6.84[c]	82.49 ± 8.16[c]	84.27 ± 4.49	0.026
MCH (pg)	30.04 ± 2.34	28.86 ± 2.88	29.31 ± 3.13	56.96 ± 27.53[c]	28.41 ± 2.76[d]	<0.001
MCHC (g/dl)	33.42 ± 1.39	31.65 ± 2.02[c]	31.40 ± 2.13[c]	29.78 ± 2.31[c]	31.15 ± 3.05	0.001
RDW -CV (%)	13.34 ± 1.50	13.54 ± 2.34	14.41 ± 1.18	11.30 ± 1.03[a]	13.42 ± 2.02	<0.001
RDW-SD (fl)	41.73 ± 4.82	45.01 ± 6.96	41.55 ± 5.85	33.59 ± 3.35	35.94 ± 4.12	<.001
TWBC ($x10^9$/L)	4.85 ± 1.26	5.54 ± 2.35	5.07 ± 1.88	9.32 ± 3.63[a]	7.84 ± 3.20	<0.001
LYMP.	36.54 ± 8.79	41.69 ± 10.30	31.05 ± 10.88	38.29 ± 10.29	33.71 ± 9.10	0.001
NEUT.	46.60 ± 5.99	49.53 ± 7.77	38.22 ± 8.32	55.07 ± 15.96	41.72 ± 8.78	<0.001
MONO.	7.02 ± 2.87	7.96 ± 3.27	6.77 ± 1.35	10.54 ± 3.78	14.73 ± 4.26	<0.001
PLT ($x10^9$)/L	181.20 ± 59.39	161.06± 52.53	188.03± 58.31	120.51 ± 69.50[a]	188.57± 69.56	0.014
FERRITIN	37.99 ± 24.35	74.67± 45.09	47.57± 31.41	26.18 ±18.42[e]	40.96± 27.21	<0.001
IL-6	11.26 ± 8.94	41.28± 58.46	35.19± 46.31	108.71±122.52[a]	23.63± 32.30	<0.001

RBC – Red Blood Cell, Hb – Haemoglobin, PCV – Packed Cell Volume, MCV – Mean Cell Volume. MCH – Mean Cell Haemoglobin, MCHC – Mean Cell Haemoglobin Concentration RDW-CV – Red Cell Distribution Width (Coefficient of Variation), RDW-SD – Red Cell Distribution Width (Standard Deviation), TWBC – Total White Blood Cell Count, Lym – Lymphocyte, Neut – Neutrophil, Mono – Monocyte PLT – Platelet, IL-6 – Interleukin-6.

Statistical significance keys

[a]($p < 0.05$): Significantly different when compared with the uninfected, vaccinated, and inactive groups in the same row.

[b]($p < 0.05$): Significantly different when compared with the active infection group in the same row.

[c]($p < 0.05$): Significantly different when compared with the uninfected group in the same row.

[d]($p < 0.05$): Significantly different when compared with the active infection group in the same row.

e($p < 0.05$): Significantly different when compared with the vaccinated group in the same row.

Table 3: Gender Based association (male) between Haematological parameters, ferritin and IL-6 level on HBV infection status

Haematolgoical parameters	Uninfected	Vaccinated	Inactive infection	Active infection	Indeterminate infection	p-value
RBC ($x10^{12}$/L)	4.56 ± 4.41	4.47 ± 0.70	4.33 ± 0.63	4.04 ± 0.73	4.42 ± 0.68	0.633
Hb (g/dl)	13.07 ± 1.10	12.76 ± 1.03	12.45 ± 1.79	11.48 ± 1.25	12.30 ± 0.84	0.270
PCV (%)	43.62 ± 4.74	44.13 ± 3.01	43.99 ± 5.14	39.38 ± 2.49	39.59 ± 3.74	0.020
MCV (fL)	89.11 ± 3.14	85.93 ± 8.20	85.18 ± 6.75	86.52 ± 3.02	84.80 ± 5.54	0.592
MCH (pg)	30.66 ± 2.13	27.72 ± 2.74	28.89 ± 2.60	58.84 ± 27.96	28.07 ± 3.23	<0.001
MCHC (g/dl)	32.74 ± 1.40	31.69 ± 1.86	31.14 ± 1.35	29.70 ± 2.43	31.76 ± 2.86	0.067
RDW (CV) (%)	13.80 ± 1.61	13.58 ± 2.38	14.20 ± 0.98	11.48 ± 0.61	12.84 ± 1.62	0.018
RDW (SD)(fl)	40.64 ± 4.92	44.29 ± 6.72	41.29 ± 6.23	35.08 ± 3.30	36.77 ± 4.89	0.008
TWBC($x10^9$/L)	4.93 ± 1.01	6.12 ± 2.78	5.43 ± 2.12	8.54 ± 2.88	8.21 ± 3.25	0.008
LYMP.	37.94 ± 7.96	46.74 ± 9.20	31.68± 11.01	40.84 ± 12.17	31.77 ± 9.19	<0.001
NEUT.	47.59 ± 6.87	46.06 ± 8.05	40.66 ± 7.82	53.18 ± 18.40	42.17 ± 11.53	0.069
MONO.	6.63 ± 3.24	7.47 ± 3.18	37.95± 12.15	9.32 ± 2.17	14.41 ± 4.44	<0.001
PLT ($x10^9$)/L	196.78 ± 63.67	160.25 ±53.04	178.75±61.92	131.54 ± 47.89	188.70 ± 67.21	0.358
FERRITIN	36.58 ±21.51	81.26± 54.90	57.09± 35.19	35.48 ± 18.24	50.12 ± 32.62	0.044
IL-6	14.36 ± 12.74	31.35± 41.03	36.65 ±52.94	106.72± 47.73	10.41 ± 3.22	0.028

RBC = Red Blood Cell, Hb = Haemoglobin, PCV = Packed Cell Volume, MCV = Mean Cell Volume, MCH = Mean Cell Haemoglobin, MCHC = Mean Cell Haemoglobin Concentration, RDW-CV = Red cell Distribution Width-Coefficient of variation, RDW-SD = Red cell Distribution Width Standard Deviation, TWBC = Total White Blood Cell count, Lym – Lymphocyte, Neut = Neutrophil, Mono – Monocyte, PLT – Platelet, IL-6 = Interleukin-6

Table 4: Gender Based Association (Female) between Haematological parameters, ferritin and IL-6 level on HBV infection status

Haematolgoical parameters	Uninfected	Vaccinated	Inactive infection	Active infection	Indeterminate infection	p-value
RBC ($x10^{12}$/L)	4.35 ± 0.53	4.61 ± 0.93	4.01 ± 0.66	3.30 ± 0.98[a]	4.02 ± 0.56	0.014
Hb (g/dl)	12.74 ± 1.33	12.92 ± 1.06	11.99 ± 1.47	9.05 ± 3.31[a]	11.72 ± 1.02	<0.001
PCV (%)	37.85 ± 2.13	37.12 ± 2.47	36.28 ± 2.13	31.30 ± 7.40[a]	36.05 ± 4.03	0.025
MCV (fL)	90.27 ± 5.47	86.18 ± 7.13	84.86 ± 7.16	77.45±10.22[a]	83.80 ± 3.50	0.019
MCH (pg)	29.53 ± 2.50	29.94 ± 2.66	28.64 ± 3.63	54.60± 31.07[a]	28.72 ±2.35	<0.001
MCHC (g/dl)	33.96 ± 1.19	31.61 ± 2.22[b]	31.71 ± 2.81	29.90 ± 2.52	30.60 ± 3.23	0.017
RDW (CV) (%)	12.95 ± 1.35	13.49 ± 2.38	14.66 ± 1.37[c]	11.07 ± 1.48	13.95 ± 2.28	0.013
RDW (SD)(fl)	42.62 ± 4.77	45.69 ± 7.32	41.86 ±5.55	31.73 ± 2.69	35.19 ± 3.34	<0.001
TWBC($x10^9$/L)	4.77 ± 1.49	5.58 ± 1.92	4.67 ± 2.25	10.30 ± 4.65[a]	7.51 ± 3.26	<0.001
LYMP.	35.39 ± 9.65	36.93 ±9.13	30.30± 11.02	35.10 ± 7.78	35.48 ± 9.07	0.362
NEUT.	45.79 ± 5.48	52.81 ± 6.03	35.37 ± 8.20	57.43 ± 14.64	41.32 ± 5.86	<0.001
MONO	7.35 ± 2.65	8.42 ± 3.37	35.40± 10.53	12.07 ± 5.13	15.03 ± 4.29	<0.001
PLT ($x10^9$)/L	176.64 ± 58.38	161.82 ± 53.66	198.94± 53.51	106.73 ± 24.68	188.45 ± 74.73	0.054
FERRITIN	39.15 ± 27.45	68.46 ±34.01[d]	36.36± 22.40	14.55 ± 11.68	32.64 ± 19.08	0.001
IL-6	8.72 ± 2.51	50.63 ± 71.18	33.47± 38.61	111.20± 04.47	35.65 ± 41.76	0.026

RBC = Red Blood Cell, Hb = Haemoglobin, PCV = Packed Cell Volume, MCV = Mean Cell Volume, MCH = Mean Cell Haemoglobin, MCHC = Mean Cell Haemoglobin Concentration, RDW-CV = Red cell Distribution Width-Coefficient of variation, RDW-SD = Red cell Distribution Width Standard Deviation, TWBC = Total White Blood Cell count, Lym – Lymphocyte, Neut = Neutrophil, Mono – Monocyte, PLT – Platelet, IL-6 = Interleukin-6

[a]($p< 0.05$): Significantly difference when compared to uninfected, vaccinated and inactive infection on the same row

[b]($p< 0.05$): Significantly different when compared to uninfected on the same row

[C]($P< 0.05$): Significantly different when compared to uninfected on the same row

[d]($p< 0.05$): Significantly different when compared with uninfected and inactive infection on the same row

Tables 3 and 4 present the gender-based association of haematological parameters, ferritin, and IL-6 according to infection status. In the active infection group, no significant sex-related differences were observed in most haematological indices when compared with the other groups. Both males and females in the active group showed reduced levels of RBC, PCV, MCV, MCHC, RDW-CV, RDW-SD, and ferritin, alongside elevated MCH, TWBC, and IL-6 relative to the other groups. However, lymphocyte and monocyte counts were significantly higher in males than in females within the active group ($p < 0.005$).

DISCUSSION

The present study demonstrates that active hepatitis B virus (HBV) infection significantly alters haematological parameters, ferritin levels, and interleukin-6 (IL-6) concentrations, confirming that HBV is not only a hepatic condition but a systemic disease that affects blood formation, immune regulation, and iron metabolism. These findings both support and contrast existing scientific reports in several key areas.

The observed reductions in RBC, haemoglobin (Hb), PCV, MCV, and MCHC, alongside increased RDW, are consistent with findings from previous studies on chronic hepatitis and other liver disorders. Chronic HBV infection has been shown to impair erythropoiesis due to inflammatory cytokines, hypersplenism, impaired iron utilisation, and reduced erythropoietin activity [22, 23].

Furthermore, increased RDW and reduced MCHC, reflecting anisocytosis and hypochromia, have been widely reported in patients with chronic liver diseases and viral infections [24].

The significantly elevated TWBC, lymphocyte, and monocyte values in the active infection group support earlier findings that viral hepatitis is associated with persistent immune stimulation and inflammation [25, 26]. Lymphocytes and monocytes are activated in response to viral antigens and play essential roles in regulating the host immune response during HBV infection [27].

Therefore, the increased immune cell counts observed in this study align with the known immunopathological processes of HBV infection.

The significantly reduced platelet counts observed in actively infected individuals are consistent with studies reporting thrombocytopenia in chronic liver disease and HBV infection [28]. This reduction is commonly associated with decreased thrombopoietin production, splenic sequestration, and immune-mediated platelet destruction in diseased liver states [29].

Marked elevation in IL-6 levels in active HBV cases strongly supports previous findings that IL-6 is a

key mediator of liver inflammation, immune dysregulation, and fibrosis [30]. IL-6 is commonly elevated in viral hepatitis, cirrhosis, and hepatocellular carcinoma and is correlated with disease severity and disease progression [31].

Thus, the extremely high IL-6 concentrations reported in this study reinforce its role as a reliable marker of disease activity in HBV infection.

The relatively normal haematological and inflammatory profiles in vaccinated individuals align with global findings demonstrating the effectiveness of hepatitis B vaccination in preventing infection and reducing systemic complications associated with the virus [32]. This further emphasizes the importance of strengthening vaccination programs in endemic regions.

Unlike many studies that report elevated ferritin in liver disease due to inflammatory response and iron accumulation, this study recorded lower ferritin levels in actively infected individuals [33].

This discrepancy may be explained by differences in:

a. Nutritional iron status
b. Severity of liver damage
c. Socioeconomic and dietary patterns
d. Altered hepcidin regulation

This suggests that ferritin dynamics in HBV may be region-specific and influenced by population characteristics.

The finding that males with active HBV infection exhibited higher lymphocyte and monocyte counts than females is not universally reported elsewhere. Some studies show minimal gender differences, while others suggest that females mount stronger adaptive immune responses [34].

This variation may relate to:

a. Hormonal differences
b. Genetic immune response variation
c. Behavioural and environmental exposure factors

The proportion of inactive carriers (30.8%) exceeding active cases (7.5%) differs from studies where chronic active cases dominate [35]. This could indicate:

a. Improved immune control in the population
b. Increased vaccination coverage
c. Early detection of infection

The observation of markedly elevated MCH in active infection contrasts studies that usually report normal or reduced MCH in chronic disease anaemia [36].

Possible explanations include:

a. Macrocytic changes from folate or vitamin B12 deficiency
b. Reticulocytosis
c. Liver-induced changes in erythrocyte morphology

This unusual finding highlights a potential area for further investigation.

In conclusion, the present study corroborates global findings that active HBV infection is characterised by anaemia, thrombocytopenia, and heightened inflammatory activity marked by elevated IL-6. However, the contradictory patterns observed in ferritin levels, MCH elevation, and gender-based immune response highlight the importance of population-specific research in HBV management and interpretation[36].

These unique findings expand current knowledge and underscore the need for regionally-tailored monitoring and intervention strategies.

Overall, the study provides strong evidence that active HBV infection is associated with significant haematological disturbances and increased inflammatory burden. These findings underscore the relevance of integrating haematological and inflammatory marker assessment into routine HBV monitoring and management strategies, especially in endemic settings like Abakaliki.

CONCLUSION

This study demonstrates that hepatitis B virus infection, particularly in its active form, significantly alters haematological indices, serum ferritin, and interleukin-6 levels among individuals in Abakaliki metropolis. Active infection was characterised by reduced red blood cell parameters, low platelet counts, and markedly elevated inflammatory markers, especially IL-6. These changes reflect impaired erythropoiesis, immune dysregulation, and ongoing hepatic injury.

The significantly higher TWBC, lymphocyte, and monocyte counts further confirm an exaggerated immune and inflammatory response in actively infected individuals. Vaccinated participants showed relatively normal haematological profiles, highlighting the protective role of immunisation against both infection and its systemic effects. Gender-based analysis revealed comparable trends in both sexes, although males demonstrated greater elevations in specific immune cell subsets.

In summary, HBV infection extends beyond liver pathology, significantly affecting blood composition and inflammatory status. Routine assessment of haematological and inflammatory markers should therefore be considered an essential component of patient evaluation, monitoring, and management in HBV-endemic regions.

Recommendations

Based on the findings of this study, the following recommendations are proposed:

1. **Routine Monitoring:** Haematological parameters, serum ferritin, and IL-6 levels should be incorporated into regular clinical monitoring of HBV-infected patients to provide early warning signs of disease progression and complications.
2. **Strengthen Vaccination Programs:** Expanded and sustained hepatitis B vaccination campaigns should be prioritised, particularly for high-risk groups, as vaccinated individuals demonstrated better haematological and inflammatory profiles.
3. **Early Screening and Diagnosis:** Routine HBV screening should be promoted in healthcare facilities and communities in Ebonyi State to facilitate early detection and intervention, thereby reducing long-term complications.
4. **Integrated Clinical Management:** Healthcare providers should adopt a multidisciplinary approach that includes immunological, haematological, and biochemical evaluation in the management of HBV patients.
5. **Public Health Education:** Awareness campaigns should be intensified to educate the public on HBV transmission routes, prevention methods, and the importance of regular screening.
6. **Further Research:** Longitudinal and multi-centre studies with larger sample sizes are recommended to better define the prognostic value of IL-6, ferritin, and haematological changes in HBV infection and treatment response.

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