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ORIGINAL ARTICLE

The Impact of Hepatitis-B envelope Antigen and Viremia load on Hematological Parameters and Liver enzymes in Chronic HBV Infection

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Abstract

Introduction: Hepatitis B viral (HBV) infection is a major cause of chronic liver diseases and failure. While the general impact of HBV on the liver has been extensively studied, there is a relative paucity of information regarding the specific effect of its envelope-antigen (HBeAg) and viremia load on hematopoiesis. Thus, this study aimed to bridge the gap.

Materials and Method: A hospital-based cross-sectional study was conducted between July and October 2024 at Obafemi Awolowo Teaching Hospital, Ile-ife. 73 HBV participants were categorized based on their viremia load and HBeAg status. Standard laboratory procedures were adopted to evaluate various biomarkers. Continuous and categorical data were expressed in mean ± SD and percentage. The study used statistical analysis software, SPSS version 26.

Results: Both HBeAg and high viremia load are associated with transaminase elevation. Lymphocyte and platelet counts were significantly reduced, neutrophil-to-lymphocyte ratio (p=0.044) and platelet-to-lymphocyte ratio (p=0.007) were elevated in HBeAg+. On the other hand, platelet count was reduced considerably (p=0.000) with a high viremia state.

Conclusion: HBeAg+ was associated with increased systemic and hepatic necrotic inflammation. However, a high viremia state was only associated with thrombocytopenia. Thus, regular monitoring of hematological parameters is vital to reduce the consequence of extra-hepatic complications.

Keywords: HBeAg, envelope-antigen, HBV, viremia load, necro-inflammation

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INTRODUCTION

Chronic hepatitis B virus (CHBV) infection remains a significant global health challenge, with an estimated over 350 million people infected worldwide (1). HBV is one of the major causes of liver-related diseases, including cirrhosis, hepatocellular carcinoma, and liver failure (2). While the broader impact of HBV on clinical outcomes has been extensively studied (3, 4), there is a relative paucity of information regarding the specific effects of two critical viral factors: hepatitis B envelope-antigen (HBeAg) status and the level of viremia on hematological parameters and liver enzymes.

HBeAg, a secretory protein encoded by the pre-core gene of HBV, is a marker of active viral replication and high infectivity (5). People with CHBV infection can be broadly classified based on the status of HBeAg into HBeAg+ and HBeAg- (6, 7). The presence of HBeAg is typically associated with higher levels of viremia, which correlates with increased liver inflammation and damage (3). On the other hand, the level of viremia is a more dependable marker for the activity of the infection, and the level varies meaningfully in different states of HBV infection, which helps diagnose the state of HBV infection (5). Viral load quantification plays a significant role in better managing HBV infection as it helps stage the state of infection, design drug regimens, and monitor antiviral treatment (6).

Given the role of the liver in hematopoiesis and the synthesis of coagulatory proteins, liver dysfunction in HBV-infected patients can potentially influence both hematological and biochemical parameters (8). However, the relationship between HBeAg status, viremia, and their potential impact on hematological profiles remains underexplored. Hematological parameters such as red blood cell (RBC) count, white blood cell (WBC) count, and platelet levels are vital indicators of systemic health. They may reflect underlying liver function or immune response to chronic viral infection (9). Elevated levels of viremia and positive HBeAg status might exacerbate extra-hepatic complications of HBV infection. However, the extent and mechanisms by which these factors contribute to alterations in hematological profiles and liver enzyme levels require further investigation (8).

In light of this gap, the present study aims to evaluate the impact of HBeAg status and the level of viremia on both hematological parameters and liver enzyme profiles in individuals with chronic HBV infection. By elucidating these associations, this research seeks to provide further insights into the pathophysiology of HBV and its systemic effects beyond the liver.

MATERIALS AND METHODS

Study design

This hospital-based cross-sectional study was carried out at the Gastroenterology clinic of Obafemi Awolowo University Teaching Hospital Complex, Ile-Ife, between July and October 2024

Inclusion and exclusion criteria

All patients attending the clinic who were willing to participate and within the age range of 18–55 years were enrolled in the study. Such an individual must be HBeAg positive for at least six months (cases), have no history of malignancy or alcohol intake, be negative for HCV and human immunodeficiency virus (HIV), and have not undergone antiviral treatment during the past six months. Pregnant women, asthmatic patients, and individuals co-infected with tuberculosis were excluded from the study—no history of blood transfusion during the past four months.

Samples and data collection

7mL of whole blood was collected from the eligible participants into plain and EDTA bottles. The samples in plain bottles were allowed to clot, retracted, and centrifuged at 3000 rpm (Uniscope Inc, USA), and serum was used to assess ALT, AST, and HBeAg. The EDTA was used for hematological and viral load assays. All the participant's medical records were retrospectively reviewed for relevant data.

HBsAg and HBeAg status evaluation

HBsAg and HBeAg were analyzed using a commercial ELISA kit (Bio-Inteco, UK, and Monobond Inc. USA). All CHB participants were eligible, and 142 ultimately underwent HBsAg and HBeAg testing.

HBV load assay

The tests were performed according to the HBV DNA quantitative detection kit and gene amplification apparatus procedures, with a sensitivity of > 500 copies/mL.

Hematology assessment

A 5 ml venous blood sample was taken into an EDTA specimen bottle, and the full blood count was analyzed using a Sysmex hematology analyzer.

Liver enzymes Assay

Serum ALT and AST levels were determined using a commercial kit (Agape Diagnostic Switzerland Gmbh). All participants were eligible, and 222 ultimately underwent ALT and AST testing.

Ethical clearance

Ethical approval was obtained from the ethical review committee of the Osun State Ministry of Health with reference number OSHREC/PRS/569T/626. The research was carried out in line with the ethics governing the use of human samples and in accordance with Helsinki declaration. Ethical practices such as participant consent, confidentiality, and safety laboratory practice were observed during the study. The questionnaire was used to gather socio-demographic data such as age, gender, marital status, family type, history of blood transfusion, presence of tattoo, number of partners, and maternal HBsAg status.

Statistical analysis

Our data were analyzed using the IBM SPSS version 26 for Windows software (SPSS Inc. Chicago, IL USA). Quantitative and qualitative variables were presented as mean ± SD and percent, respectively. Descriptive analysis and a one-way Student's test were used to compare data; p-value < 0.05 were considered significant. The Pearson correlation coefficient was used to test the association between variables, as appropriate.

RESULTS

The demographic and clinical characteristics of enrolled participants are presented in Table 1. 73 chronic HBV-infected participants were recruited; they comprised 61 (83.6%) males and 12 (16.4%) females. The two groups were HBeAg-Positive (n = 7) and HBeAg-Negative (n = 66). The male-to-female ratio in the two groups was 4:5.8 respectively. The mean ages for the two groups were similar and comparable. Also, the body mass index (BMI) of HBeAg positive and negative were computed and comparable. All participants were of Black ethnicity. Virtually all the participants involved in this study were asymptomatic and in a chronic state.

Figure 1 shows the distribution of the envelope antigen and viral load among the participants. Out of 73 CHB-infected participants recruited for the study, 7 (9.6%) had a positive history of HBV envelope antigen, and the remaining 90.4% were negative for envelope antigen. Among the participants, those with high viral loads were 28.8%.

As Table 2 shows, there were no significant differences in WBC, neutrophil, eosinophil, basophil, RBC counts, HGB, and PCV levels between the HBeAg+ and HBeAg- groups. However, HBeAg+ participants showed significantly higher PLR and NLR, while platelet counts and lymphocyte levels were elevated in the HBeAg- group. AST and ALT levels were significantly higher in the HBeAg+ group, although the AST/ALT ratio showed no significant difference.

Table 3 revealed no significant differences in WBC, neutrophil, eosinophil, basophil, RBC counts, HGB, PCV levels, PLR, and NLR between the HVL and LVL groups. However, while platelet count was significantly reduced in HVL, AST/ALT was reduced considerably in the LVL group.

Table 1: Demographic and clinical characteristics of Chronic HBV infected patients

Characteristics		HBeAg-Positive	HBeAg-Negative
Frequency (Prevalence)		7 (9.6%)	66 (90.4%)
Age (years)		38.4 ± 15.65	41.34 ± 18.45
Gender	Male	5 (6.8%)	56 (76.7%)
	Female	2 (2.7%)	10 (13.7%)
BMI (Kg/m^2)		23.3 ± 2.89	24.6 ± 2.11
Viremia load	Low	1 (1.4)	59 (72.6)
	High	6 (8.2)	7 (17.8)
HBsAg status		Positive	Positive
HBeAg status		Positive	Negative
Other viral infections		Negative	Negative
Duration of HBV infection		> 6 months	> 6 months
History of antiviral treatment		Negative	Negative

The values are frequency (%) and mean ± standard deviation

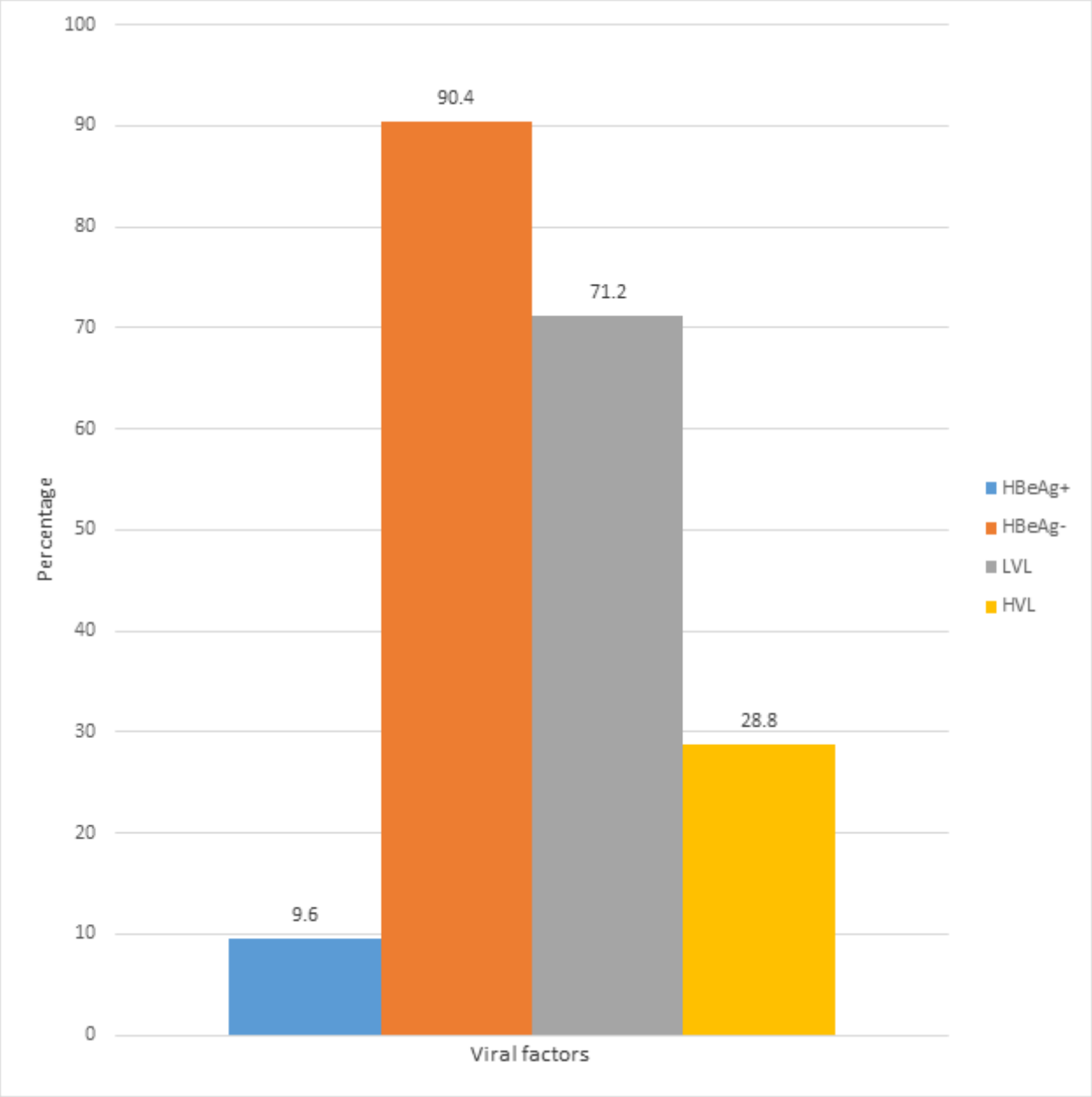

Figure 1: Bar chart showing the percentage distribution of HBeAg and viral load among the studied groups.

HBeAg+ = HBV envelope positive, HBeAg- = HBV envelope negative, LVL = low viral load, HVL = high viral load

Table 2: Comparison of Hematological and Liver Parameters between HBeAg+ and HBeAg- Chronic-HBV Infection

Parameters	**HBeAg-Negative (n=66)**	**HBeAg-Positive (n=7)**	**P-value**
WBC (x 10^9/L)	5.99 ± 1.59	4.86± 2.56	0.382
Neutrophil (%)	43.13 ± 10.29	47.80 ± 15.40	0.755
Lymphocytes (%)	54.79 ± 10.50	46.60 ± 4.38	0.009*
NLR	0.81 ± 0.52	1.03 ± 0.74	0.013*
Eosinophil (%)	3.97 ± 1.61	3.25 ± 1.50	0.440
PCV (%)	40.05 ± 3.49	36.20 ± 5.67	0.204
HBG (g/dL)	13.32 ± 1.15	12.04 ± 1.89	0.205
RBC (x 10^{12}/L)	4.35 ± 0.50	4.44 ± 0.18	0.958
Platelet (x 10^9/L)	182.40 ± 25.86	141.48 ± 33.17	0.024*
PLR	2.81 ± 0.54	3.76 ± 1.34	0.011*
AST/PLT	0.007 ± 0.005	0.002 ± 0.001	0.019*
AST	27.79 ± 15.78	78.80 ± 27.67	0.038*
ALT	20.47 ± 11.07	83.40 ± 34.08	0.003*
AST/ALT	0.89 ± 0.51	1.45 ± 0.86	0.318

The values are mean ± standard deviation. A Student t-test was used to compare the means, and p = 0.005. WBC = white blood cell count, NLR = Neutrophil: lymphocyte ratio, PCV = Packed cell volume, HBG = hemoglobin level, PLT = Platelet, PLR = platelet/Lymphocyte ratio, and RBC = red blood cell.

Table 3: Comparison of Hematological Parameters between high and low viremia load Chronic-HBV Infection

Parameters	**High viremia (n=13)**	**Low viremia (n=60)**	**P-value**
WBC (x 10^9/L)	5.86 ± 1.79	5.96± 1.67	0.821
Neutrophil (%)	45.25 ± 11.84	47.78 ± 10.14	0.367
Lymphocytes (%)	50.60 ± 10.43	55.11 ± 10.86	0.387
NLR	1.05 ± 0.52	0.96 ± 0.54	0.439
Eosinophil (%)	3.72 ± 2.61	4.04 ± 2.16	0.718
PCV (%)	39.55 ± 5.06	39.92 ± 3.20	0.710
HBG (g/dL)	13.15 ± 1.68	13.27 ± 1.06	0.761
RBC (x 10^{12}/L)	4.35 ± 0.42	4.54 ± 0.38	0.345
Platelet (x 10^9/L)	146.45 ± 35.6	183.48 ± 27.51	0.044*
PLR	3289.1 ± 807.9	3877.2 ± 1448.4	0.081
AST/PLT	0.005 ± 0.004	0.001 ± 0.006	0.081
AST (IU/L)	48.65 ± 32.44	16.65 ± 7.36	0.000*
ALT (IU/L)	47.80 ± 29.24	25.50 ± 18.32	0.000*
AST/ALT	1.09 ± 0.61	0.79 ± 0.35	0.011*

The values are mean ± standard deviation. A Student t-test was used to compare the means, and p = 0.005. WBC = white blood cell count, NLR = Neutrophil: lymphocyte ratio, PCV = Packed cell volume, HBG = hemoglobin level, PLR = platelet/Lymphocyte ratio, and RBC = red blood cell.

Table 4: The combined effect of HBeAg and viremia load on hematological parameters and liver enzymes in chronic HBV infection

	Hematological abnormality		
	Frequency (%)	$\chi 2$	P
HBeAg+ + high viral load	3 (4.1)	0.6	0.102
HBeAg- + low viral load	56 (76.7)		
HBeAg+ + low viral load	4 (5.5)		
HBeAg- + high viral load	10 (13.7)		
Hepatic enzymes abnormality			
HBeAg+ + high viral load	6 (8.2)	3.4	0.001*
HBeAg- + low viral load	59 (80.8)		
HBeAg+ + low viral load	1 (1.4)		
HBeAg- + high viral load	7 (9.6)		

The values are finding (%). Chi-square test was adopted to determine if each category has an equal likelihood, at p = 0.05 (95% confidence level) with degree of freedom 1 (3.8).

DISCUSSION

The demographic and clinical data of the studied groups were compared between HBeAg+ and HBeAg- on one side and between high-viral load and low-viral load on the other with respect to hematological and hepatotoxic parameters. Other chronic and viral infections were ruled out using appropriate methods. The ages of the two groups were comparable. An examination of gender distribution revealed that approximately 85% of the participants were males. This finding contradicted the notion that HBV affects men and women similarly (10). It should be noted that a higher female ratio has been reported by (11). Thus, there is no fixed pattern for HBV infection; the distribution largely depends on the pattern of exposure to risk factors among the population studied.

Viral load and envelope antigen status remain essential in HBV infection therapeutic decisions and treatment response monitoring (12). Out of the eligible 73 participants, 7 (9.6%) were positive for HBeAg, while 13 (17.8) had an incidence of high viral load (Figure 1). This prevalence is lower for the envelope antigen, compared with 17.3% earlier reported in Kisi, Oyo State, Nigeria by (7) and 12% reported in Asia by (13). The demographic variation in e-antigen could be connected with the type of HBV genotype and strain that predominates among the population study. The degree and rate of e-antigen expression differ among different strains of HBV due to the region of nonsense mutation, which could be in the pre-core, core promoter, stop codon, or combination of those regions (14). There is a paucity of information in available journals to support our findings on the prevalence of viremia load.

The lack of significant differences in WBC, neutrophil, eosinophil, basophil, and RBC counts, HGB, and PCV between HBeAg+ and HBeAg- participants, as revealed in Table 2 suggests that HBeAg status may not directly impact these general hematological markers. This is consistent with findings from (9), who reported that routine hematological parameters often do not vary significantly with HBeAg status in CHB patients. However, these common hematologic markers may not fully capture the underlying inflammatory activity associated with HBeAg positivity. The significantly higher lymphocyte counts in HBeAg- patients may suggest a more regulated immune environment, as the absence of HBeAg is associated with a lower degree of immune activation. Research by (15) indicates that HBeAg- CHB patients may have a more controlled immune response, with lower levels of inflammation and cytotoxicity compared to HBeAg+ patients. Lymphocyte elevation in the HBeAg- group may reflect a relative balance in immune function compared to the elevated neutrophil and inflammatory response in HBeAg+ patients. The significantly higher PLR and NLR observed in HBeAg+ patients indicate a heightened inflammatory and immune response in this group. PLR and NLR are markers of systemic inflammation, with higher values often associated with viral hepatitis and liver disease progression. Studies by (8, 9) found that elevated PLR and NLR values were associated with increased inflammation and fibrosis risk in CHB patients, especially those with active viral replication, which is more common in HBeAg+ individuals. This suggests that HBeAg+ status may enhance inflammatory response and immune activation in CHB. The finding of elevated platelet aligns with other studies, such as one by (16), which showed that CHB patients with active viral replication, often seen in HBeAg+ status, tend to have elevated platelet counts. This increase could result from elevated interleukin-6 (IL-6) and other cytokines associated with CHB infection.

As was evident in Table 3, there were no significant differences in all hematological parameters except the platelet count, which was lower in those with high viral load. The absence of significant differences in WBC, lymphocytes, neutrophils, eosinophils, basophils, RBC counts, HGB, PCV, PLR, and NLR suggests that elevated HBV viral load may not strongly affect general hematological parameters in CHB. These findings align with prior research, such as the study by [8], which reported that typical hematological markers often remain stable across different levels of HBV viremia. The results suggest that while HBV viral replication influences liver injury and immune activity, it does not cause substantial fluctuations in standard hematologic indices in most cases. The significantly lower platelet counts in patients with high HBV viral load are notable, as thrombocytopenia is commonly associated with advanced liver disease and active viral replication (15). High HBV viral loads have been linked to enhanced liver inflammation and fibrosis, which may lead to a reduction in platelet production due to impaired liver function and potential hypersplenism. A study by (17) demonstrated that high HBV viremia was associated with lower platelet counts, likely due to progressive liver disease and increased splenic sequestration of platelets. Furthermore, HBV-induced liver fibrosis and portal hypertension can also contribute to reduced platelet counts in high-viremia CHB patients (18).

The elevated AST and ALT levels observed in HBeAg+ participants indicate greater liver inflammation and hepatocellular injury in this group (Table 2). AST and ALT are enzymes released from damaged liver cells, and their elevated levels are commonly seen in active viral hepatitis. HBeAg+ states tend to have higher viral loads and active hepatitis, which can lead to more pronounced liver injury. A

study by (19) showed that CHB patients with positive HBeAg status often had elevated liver enzymes, consistent with increased liver inflammation and immune-mediated hepatocyte damage. Despite the elevated AST and ALT levels, the AST/ALT ratio did not differ significantly between the groups. This suggests that the AST/ALT ratio may not be a sensitive marker for distinguishing liver injury severity between HBeAg+ and HBeAg- CHB states.

The significant elevation of AST, ALT, and the AST/ALT ratio in high-viremia patients indicates more significant hepatocellular injury in this group. The AST/ALT ratio reflects hepatocellular stress in HBV infection, particularly when associated with higher viral replication. Our finding is in line with (20). High viral replication is thought to drive immune-mediated cytotoxicity, leading to increased release of these liver enzymes. The elevation in the AST/ALT ratio in high-viremia patients may further indicate a state of active liver injury rather than stable chronic infection. While the AST/ALT ratio is traditionally used in differential diagnosis, higher ratios may signify more advanced liver disease or inflammatory activity in CHB patients with active replication (21).

The prognostic significance of the combination of the effect of HBeAg and high viremia load was evaluated by determining the incidence of hematological aberration and hepatotoxicity in relation to different combinations of the two parameters. For hematological parameters, the Chi-square value was 0.6 with 1 degree of freedom and $p = 0.102$. Thus, the occurrence of hematological abnormality is independent of a combination of the two parameters. However, for hepatotoxic impact, the combination of the two parameters resulted in a significant relation ($p = 0.001$). Therefore, hepatic damage significantly depends on the combined impact of HBeAg and high viremia state. This finding further elucidates the pathogenicity of each of HBeAg and HBV DNA.

CONCLUSION AND RECOMMENDATION

The study highlights the differential impact of HBeAg status and viremia load on hematological parameters and liver enzymes (AST and ALT). The findings reveal that liver enzyme levels were significantly elevated in participants with HBeAg positivity compared to those with high viremia load. This suggests HBeAg positivity may be a stronger indicator of active hepatic inflammation and liver damage than viral replication alone. Additionally, the derangement in hematological parameters, including potential alterations in red blood cells, white blood cells, and platelets, was more pronounced in HBeAg-positive individuals than in those with high viremia load. This underscores the critical role of HBeAg in driving both hepatic and systemic hematological changes in individuals with hepatitis B infection.

These findings provide valuable insights into the clinical significance of HBeAg status, beyond viremia load, in managing and monitoring hepatitis B patients. They emphasize the need for regular assessment of HBeAg in conjunction with viral load to better understand disease activity, guide treatment decisions, and anticipate complications. Future research is recommended to elucidate further these associations' mechanisms and evaluate their implications for long-term disease outcomes. More so, monitoring the liver enzymes and platelet counts, particularly in HBeAg+ with high-viremia patients, is essential in managing CHB-related liver injury.

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