



Assessment of Some Hematological Parameters in Hepatitis B Subjects Co-Infected with HIV on Therapy, Attending Imo Specialist Hospital, Owerri

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Abstract

Infections with the human immunodeficiency virus (HIV) and the hepatitis B virus (HBV) are serious public health problems around the world, especially in sub-Saharan Africa, where they are common and spread in similar ways. Co-infection with these viruses hastens illness progression, complicates therapy, and negatively impacts haematological markers. This study assessed certain haematological parameters in HIV-infected patients co-infected with HBV on antiretroviral therapy (ART) at Imo Specialist Hospital, Owerri. A cross-sectional study was performed including 100 participants aged 18 to 60 years, including 50 HIV-infected individuals and 50 co-infected with HIV and HBV. Five millilitres of venous blood were obtained in EDTA tubes and examined using an automated haematology analyser. The parameters that were looked at were haemoglobin (Hb), packed cell volume (PCV), red blood cell count (RBC), white blood cell count (WBC), lymphocyte percentage, and platelet count. The presence of hepatitis B surface antigen (HBsAg) verified the HBV infection. We used SPSS version 25.0 to look at the data, and we set the significance level at $p < 0.05$. The average levels of Hb, PCV, and RBC in individuals co-infected with HIV and HBV were considerably lower ($p < 0.05$) compared to those infected alone with HIV. Total WBC and lymphocyte counts were reduced in co-infected patients, signifying mild leukopenia and lymphocytopenia. The platelet count was reduced in the HIV/HBV co-infected group relative to persons alone infected with HIV. HIV/HBV co-infection adversely affects haematological parameters, indicating bone marrow suppression, decreased erythropoiesis, and immunological dysregulation. It is advised to conduct continuous haematologic monitoring of co-infected patients to enhance therapeutic results and avert haematologic problems linked to antiretroviral and antiviral therapy.

Keywords: HIV, Hepatitis B, Co-infection, Haematological parameters, Antiretroviral therapy, Owerri.

Introduction

HIV and hepatitis B virus (HBV) infections are still major public health problems around the world, especially in sub-Saharan Africa, where both diseases are common and continue to cause a lot of sickness and death. The World Health Organisation (WHO) estimates that in 2023, around 39 million people around the world had HIV, and more than 250 million people were chronically infected with HBV [1]. The co-endemic presence of these illnesses in numerous developing countries is mostly due to overlapping transmission pathways, insufficient healthcare access, and the limited execution of preventative measures, including immunisation and safe blood screening [2]. HIV predominantly attacks CD4+ T cells, which weakens the immune system over time and makes the body more likely to suffer opportunistic infections and cancers. HBV, conversely, is a hepatotropic virus that targets hepatocytes and can lead to acute or chronic liver damage, cirrhosis, and hepatocellular cancer. Because both viruses can be spread in the same ways, such as through unprotected sex, contact with contaminated blood or blood products, intravenous drug use, and transmission from mother to child, co-infection is common, especially among high-risk groups like healthcare workers, intravenous drug users, and

people with multiple sexual partners [4]. HIV/HBV co-infection poses distinct clinical and therapeutic problems. The presence of both viruses can affect how the disease progresses and how well treatment works. People with HIV who also have HBV had greater levels of HBV DNA, delayed HBeAg seroconversion, and a higher risk of chronicity [5]. On the other hand, HIV infection can make liver damage caused by HBV worse because it makes it harder for the immune system to manage the virus's reproduction. Moreover, hepatotoxicity linked to antiretroviral therapy (ART) and certain anti-HBV drugs may exacerbate management challenges and adversely affect prognosis [6]. Both HIV and HBV infections have significant impacts on the haematopoietic system. Haematological abnormalities are prevalent problems in HIV-infected individuals and frequently signify disease progression [7]. These include anaemia, leukopenia, and thrombocytopenia, which occur due to multifactorial mechanisms, including direct viral suppression of bone marrow progenitor cells, immune-mediated destruction of circulating blood cells, opportunistic infections infiltrating the marrow, and drug-induced myelosuppression. Anaemia has been identified as a significant independent predictor of morbidity and mortality in patients infected with HIV [8]. In HBV infection, hepatic impairment is pivotal in haematological changes. The liver makes a number of important haematopoietic growth hormones, including erythropoietin and thrombopoietin. It also plays a role in storing and using iron. Damage to liver tissue in chronic HBV infection might thereby hinder the manufacture of these hormones, resulting in secondary bone marrow suppression. Hypersplenism caused by portal hypertension may also make cytopenias worse by making blood cells more likely to be destroyed or kept in the body [9]. HIV/HBV co-infection is anticipated to adversely affect haematopoiesis due to the synergistic effects of viral replication, immunological dysregulation, and hepatocellular damage. Research indicates that co-infected individuals have more pronounced haematological abnormalities compared to those infected with a single virus [10]. Additionally, ART, although life-saving, has been linked to varied levels of bone marrow suppression, exacerbating haematologic problems. These changes not only affect the prognosis of the disease, but they also affect treatment options, since some antiretroviral drugs need to be changed or adjusted in dose when the liver is not working properly [11]. In light of these clinical issues, evaluating haematological markers in HIV/HBV co-infected individuals yields significant insights into disease pathogenesis, treatment-associated problems, and overall prognosis. This kind of evaluation can help find patients who are at risk of severe anaemia, thrombocytopenia, or leukopenia, so that timely intervention and better clinical care can be done [12]. Consequently, this study aimed to assess certain haematological parameters in HIV patients co-infected with HBV on antiretroviral therapy at Imo Specialist Hospital, Owerri. The aim was to ascertain the degree of haematologic changes in this population, investigate potential associations with illness progression and treatment status, and furnish data that could optimise patient monitoring and enhance therapy outcomes.

Materials and Methods

Study Area

This study was conducted at the Imo Specialist Hospital, Owerri, Imo State, Nigeria. It is a tertiary health facility providing specialized care for HIV/AIDS and viral hepatitis patients.

Study Population

100 adults aged 18–60 years were enrolled and categorized into two groups: Group 1 (Control): 50 HIV infected patients on ART as well as Group 2: 50 HIV/HBV co-infected patients on ART (n = 50), All participants had been on ART for at least 6 months prior to recruitment.

Ethical Approval

The study was approved by the Ethical and Research Committees of the Specialist Hospital Owerri used in the study. Informed consent was also obtained from all participating patients.

Collection of Blood Samples

Blood samples were collected aseptically by venopuncture, using a 5ml sterile disposable syringe and needle from all the subjects and was then dispensed into EDTA container for hematological estimation

Laboratory Procedures

The following parameters Hemoglobin concentration (Hb), Packed Cell Volume (PCV), Red Blood Cell count (RBC), Total White Blood Cell count (WBC), Differential count (Neutrophils, Lymphocytes) and Platelet count were analyzed using an automated hematology
HBV infection was determined by detecting HBsAg using rapid diagnostic test kits.

Statistical Analysis

All data generated in this study was subjected to statistical analysis using SPSS version 23. Mean and standard deviation, student t-test and correlation were determined. The level of significant will be taken at $p < 0.05$.

Results

Table 1: The Mean and Standard Deviation of the Levels of Hematological Parameters (Hb, PCV, RBC, WBC, Neutrophils, Lymphocytes, and Platelets) in Hepatitis B Subjects Co-infected with HIV on Therapy, Attending Imo Specialist Hospital, Owerri.

Parameter	HIV	HIV + HBV	p-value
Hemoglobin (Hb, g/dL)	12.1 ± 1.2	10.8 ± 1.4	0.011
Packed Cell Volume (PCV, %)	37.1 ± 3.5	33.2 ± 2.8	0.001
Red Blood Cell count (RBC, ×10 ¹² /L)	3.6 ± 0.7	2.9 ± 0.8	0.001
Total White Blood Cell count (WBC, ×10 ⁹ /L)	4.8 ± 1.1	4.4 ± 1.4	0.010
Neutrophils (%)	56.5 ± 7.1	50.1 ± 4.4	0.012
Lymphocytes (%)	33.9 ± 7.4	28.3 ± 6.1	0.015
Platelets (×10 ⁹ /L)	203 ± 32	155 ± 30	0.007

Discussion

The findings of this investigation demonstrated notable haematological changes in HIV/HBV co-infected patients in contrast to those only infected with HIV. The decrease in haemoglobin concentration and packed cell volume (PCV) in co-infected individuals unequivocally signifies the existence of anaemia, a common haematologic disorder linked to both HIV and chronic HBV infections. Anaemia in these patients may arise from various interacting causes, including direct suppression of bone marrow by HIV, persistent systemic inflammation, hepatic dysfunction related to HBV infection, and the myelosuppressive effects of antiretroviral therapy (ART). The liver is essential for erythropoietin production and iron metabolism; hence, hepatic damage caused by HBV can severely disrupt erythropoiesis, resulting in normocytic or microcytic anaemia [13].

This aligns with the findings of [14], who noted significantly diminished haematologic indices in HIV/HBV co-infected patients in southeastern Nigeria compared to those infected alone with HIV. Chronic viral hepatitis can exacerbate anaemia and thrombocytopenia in HIV-positive individuals via mechanisms including direct cytopathic effects of HBV on haematopoietic progenitor cells, immune-mediated destruction of circulating blood cells, and the inhibitory effects of proinflammatory cytokines on erythropoiesis. Moreover, chronic infection frequently stimulates the synthesis of hepcidin, a hepatic peptide that diminishes iron availability for erythroid precursors, hence exacerbating anaemia of chronic illness [15].

Leukopenia and lymphocytopenia identified in this investigation are clinically significant. These problems may result from direct HIV infection of bone marrow progenitor or stromal cells, toxic consequences of antiretroviral therapy (especially zidovudine and other nucleoside analogues), or indirect effects of chronic inflammation. HIV specifically targets CD4⁺ T cells and monocytes, resulting in a progressive reduction in lymphocyte populations and compromised immunological functionality. In cases of co-infection, hepatic damage generated by HBV and systemic immunological activation can further aggravate lymphocyte apoptosis and dysfunction. Persistent leukopenia in co-infected patients after antiretroviral therapy (ART) indicates insufficient immune reconstitution and may signify residual virus replication, bone marrow fatigue, or ART-induced damage. This study confirms the results of [16], which indicated a consistently low white blood cell count in HIV/HBV co-infected people, suggesting impaired haematopoietic repair despite ongoing viral suppression [17].

Thrombocytopenia, another significant finding in this study, was more common in HIV/HBV co-infected individuals compared to HIV mono-infected controls. There are a few possible reasons for this observation. Platelet degradation may transpire due to immune-mediated clearance initiated by circulating immunological complexes. Moreover, diminished thrombopoietin synthesis resulting from hepatic damage in chronic HBV infection leads to decreased platelet formation. The research conducted by [18] corroborates this idea, detailing significant platelet breakdown and reduced thrombopoietin production in individuals with persistent HBV infection. Additionally, hypersplenism resulting from portal hypertension in advanced liver illness might augment splenic sequestration of platelets, thereby further decreasing circulating platelet numbers.

These haematological anomalies collectively underscore the synergistic and deleterious impacts of HIV/HBV co-infection on the bone marrow and the immune system, even among patients on ART. The cumulative effects of viral replication, immunological dysregulation, hepatic dysfunction, and medication toxicity create a complex pathophysiological condition that disrupts normal haematopoiesis and immune homeostasis. Anaemia and thrombocytopenia are significant indicators of disease progression and predictors of adverse prognosis in patients infected with HIV. Their presence in co-infected patients may indicate expedited clinical deterioration and an elevated risk of opportunistic infections and bleeding disorders [19].

The recurrence of these haematological abnormalities despite antiretroviral therapy (ART) highlights the necessity for continuous haematologic surveillance and prompt identification of cytopenias in co-infected individuals. Keeping an eye on these factors not only helps figure out how the disease is getting worse, but it also helps doctors decide what medications to give, such as changing ART regimens or adding supportive treatments like erythropoiesis-stimulating drugs or hematinics. These results underscore the necessity for coordinated clinical therapy of HIV and HBV infections, with meticulous consideration of hepatic and haematologic health [20].

Consequently, the current study highlights the detrimental interplay between HIV and HBV infections in modifying haematologic profiles. Patients with dual infections had more severe anaemia, leukopenia, and thrombocytopenia than those with only HIV. This pattern shows how both viruses have a combined effect on bone marrow function, liver-mediated haematopoietic control, and immunological integrity. Ongoing monitoring and swift intervention of haematologic irregularities are vital elements of holistic treatment for patients co-infected with HIV and HBV, intended to enhance therapeutic results and overall quality of life [21].

Conclusion

HIV/HBV co-infection correlates with considerable haematological anomalies, notably anaemia, leukopenia, and thrombocytopenia, in contrast to HIV mono-infection. HIV/HBV co-infected people demonstrated substantial decreases in haemoglobin, packed cell volume, red blood cell, white blood cell, and platelet counts relative to HIV mono-infected subjects, indicating anaemia, leukopenia, and thrombocytopenia. These haematologic abnormalities indicate the synergistic effects of viral co-infection and medication toxicity. These changes could be caused by a combination of viral impacts, medication toxicity, and liver problems. Regular haematologic monitoring should be integrated into the standard care of HIV/HBV co-infected patients to facilitate prompt detection and intervention.

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