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RESEARCH ARTICLE

ALTERATION IN PLATELET INDICES AS LABORATORY TOOLS FOR MONITORING HIV PATIENTS ON ANTIRETROVIRAL THERAPY IN A SOUTH-SOUTH TERTIARY HOSPITAL, NIGERIA

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Abstract

Background: Human Immunodeficiency Virus (HIV) infection is associated with haematological abnormalities, including changes in platelet count and platelet indices, which may persist despite antiretroviral therapy.
Objective: To assess platelet count, platelet indices, CD4 count, and viral load among PLHIV receiving Highly Active Antiretroviral Therapy (HAART) in Calabar, Nigeria, and to compare these parameters with HIV-seronegative controls.
Methods: A comparative cross-sectional study was conducted among 60 participants aged 22–45 years, comprising 30 HIV-positive individuals receiving HAART and 30 HIV-seronegative controls. Ethical approval was obtained, informed consent was secured, and structured questionnaires were administered. Full blood count was performed using an automated haematologyanalyser to determine platelet count and indices, while CD4 T-cell count was measured using the PartecCyFlow counter.
Results: Females constituted 57% of participants and males 43%, while 63% were married. Among PLHIV, 70% received Tenofovir/Lamivudine/Dolutegravir (TLD) and 30% received Abacavir/Lamivudine/Dolutegravir (ALD), with approximately 60% reporting adherence to therapy. Platelet distribution width (PDW) was significantly higher among PLHIV than controls (16.1% vs. 14.6%), while CD4 count was significantly lower.
Conclusion: HIV infection was associated with significant alterations in PDW, platelet-related parameters, CD4 count, and viral load among individuals receiving HAART. Although platelet parameters remained within reference ranges, their changes may provide clinically useful indicators for monitoring haematological status and treatment response in HIV care.
Keywords: HIV, highly active antiretroviral therapy, platelet distribution width, Plateletcrit (PCT), Nigeria.

INTRODUCTION

Platelets play a vital role in formation of aggregate or primary haemostatic plug that stems blood loss at the site of injury[1]. They also supply the cellular surface on which complexes like tennase and prothrombinase assemble on[2-4]. Their functions include adhesion and aggregation with formation of haemostatic plug, release of platelet activating and procoagulant molecules and provision of a procoagulant surface for reactions of the coagulation system[5]. The platelets contain alpha granules whose content include coagulation factors like Factor V, VIII, Fibrinogen[6]. They also contain dense granules with contents like calcium, serotonin, adenosine diphosphate and adenosine triphosphate[7]. These substances cause vasoconstriction and reduce blood flow to injured area[8]. When a vessel wall is damaged the sub-endothelial structures e.g. collagen and micro fibrils are exposed and also surface bound vWF help in adhesion of platelets to these exposed structures[9,10]. Human Immunodeficiency Virus infection and HAART impair

liver function by inducing hepatotoxicity that diminishes the function and synthesis of coagulation factors[11]. Chronic immune activation and inflammatory state in both untreated and treated HIV infection can result in abnormal haemostatic changes[12]. Immune-mediated destruction of platelets by antibodies, impaired megakaryocytes, hypersplenism, opportunistic infections, malignancy, and toxic and myelo-suppressive effects of HIV medications may lead to thrombocytopenia and alter the haemo-static system in patients with HIV[13,14]. Evidence suggests that HIV replication may upregulate the tissue factor pathway via innate immune activation[15]. Untreated HIV replication leads to short-term increases in some procoagulants as well as decreases in key anticoagulants. Abnormalities consistent with a hyper-coagulable state have been reported in HIV patients[16,17]. Haemostatic derangement contributes to HIV-related morbidity and affects patients from every risk group independent of age, sex, or stage of infection[18]. Considering the gains achieved with current HAART in suppressing viral replication[19], it is not yet clear whether haemostatic derangements persist in PLWH. Evaluation of platelet parameters in these subjects has become necessary, hence the need for this study.

MATERIALS AND METHODS

Ethical approval (UCTH/HREC/33/Vol.III/301) was duly obtained from the University of Calabar Teaching Hospital Health Research Ethics Committee, while an informed consent was obtained from each participant. A total of 60 male and female subjects of age range 22-45 years, were enrolled which consist of 30 HIV subjects on HAART and 30 seronegative subjects as controls. A case-control study design was adopted for this study. Confirmed HIV status was considered for inclusion of consenting test and control participants. In addition, being on HAART was also an inclusion criterion for persons living with HIV. Those on other medications apart from HAART, HIV patients who did not give consent, as well as those who were pregnant were excluded. A well-structured questionnaire was administered to each study participant for bio-data and information related to HIV infection and management. Blood sample was appropriately obtained from each subject for testing of full blood count and CD4 count. Full blood count (FBC) and CD4 count were determined by automation using Mindray BC-5000 Haematology analyzer, and Partec Cy Flow counter (Sysmex Corporation, Japan). Data generated were analysed using Statistical Package for Social Sciences (SPSS) software version 22.0. Statistical significance was drawn at a $p \leq 0.05$.

RESULTS

Table 1 show features of HIV subjects where 70% (21) of HIV subjects were placed on TLD combination with only 30% (9) of them on ALD combination. Those whose treatment duration was up to 5 years were 53.3% (16), and above 5 years was 46.7% (14). It was observed that 60% (18) of them adhered to HAART treatment and 40% (12) did not adhere, 30% (9) of them did not adhere to treatment because of intolerance, while 10% (3) was as a result of forgetfulness. Table 2 shows platelet parameters and CD4 count of HIV subjects and control. The measured parameters include; platelet (PLT), plateletcrit (PCT), platelet distribution width (PDW) and mean platelet volume (MPV). There was no significant difference in PLT, MPV, and PCT parameters of HIV subjects when compared with the control subjects. However, PDW, was observed to be higher (p=0.002) in HIV subjects (16.07±0.25%) than the control subjects (14.63± 0.25%). CD4 count was significantly reduced (503.40± 91.40 cells/ml) in HIV subjects compared with control group (792.23±137.03 cells/ml).

Table 1: Features of HIV subjects.

Variables	Numbers (%) (n=30)	*p*-value
Gender		
Male	13 (43.3)	0.866
Female	17 (56.7)	
Marital Status		
Single	7 (23.3)	
Married	19 (63.3)	0.001*
Widowed	1 (3.3)	
Divorced	3 (10)	
Types of HAART		
TLD	21 (70)	0.028*
ALD	9 (30)	
Duration of treatment		
Up to 5 years	16 (53.3)	0.715
Above 5 years	14 (46.7)	
HAART adherence status		
Adhered	18 (60)	0.273
Non-adhered	12 (40)	
Reason for non-adherence		
Intolerance	9 (30)	0.083
Forgot	3 (10)	

ALD =Abacavir Lamivudine Dolutegravir, TLD = Tenofovir Lamivudine Dolutegravir

Table 2: Platelet parameters and CD4 count of HIV subjects and controls.

Parameters	HIV Subjects (n=30)	Controls (n=30)	*p*-value
PLT (150-400 x 10^9/l)	191.37±58.91	199.83±53.43	0.563
MPV (9.4-12.3fl)	9.53±1.43	9.63±0.61	0.728
PDW (10.0-17.9 %)	16.07±.025	14.63±2.51	0.002*
PCT (1.08-2.82 %)	1.77±0.78	1.89±0.54	0.326
CD4 Count (>500 cells/ml)	503.40±91.40	792.23±137.03	0.001*

PLT = Platelet, MPV = Mean platelet volume, PDW = Platelet distribution width, PCT = Plateletcrit, CD4= Cluster of differentiation. *Significant difference.

Table 3 shows Platelet parameters, CD4 count and Viral load of HIV subjects based on treatment duration. Here, PCT (1.48±0.74 %) was observed to be lower in value in those on less than 5 years treatment compared with those whose treatment duration was above 5 years (2.01±0.46 %). Again, the Viral Load mean value of HIV subjects on treatment duration less than 5 years (74.38±68.40 cells/mL) was higher than those on treatment duration above 5 years (33.07±11.49 cells/ mL). The PDW was not significantly affected by gender or adherence in the studied population. In certain conditions like HIV infection, both the disease pathophysiology and therapeutic drugs mask normal gender-influenced physiological differences in blood cell parameters.

Table 3: Platelet parameters, CD4 count and Viral Load of HIV subjects based on treatment duration.

Parameters	≤ 5 years (n=16)	>5 years (n=14)	*p*-value
PLT (150-400 x 10^9/l)	180.00±66.92	204.36±47.26	0.266
MPV (9.4-12.3fl)	9.25±1.52	9.95±1.36	0.198
PDW (10.0-17.9 %)	16.08±0.35	16.09±0.35	0.935
PCT (1.08-2.82 %)	1.48±0.74	2.01±0.46	0.029*
CD4 count (>500 cells/ml)	466.88±67.40	545.14±99.38	0.016*
Viral load (0 copies/ml)	74.38±68.40	33.07±11.49	0.034*

PLT = Platelet, MPV = Mean platelet volume, PDW = Platelet distribution width, PCT = Plateletcrit, CD4= Cluster of differentiation. *Significant difference.

DISCUSSION

The present study focused on platelet parameters of people living with human immunodeficiency virus receiving current antiretroviral therapy at University of Calabar Teaching Hospital in Southern Nigeria. The socio-demographic parameters from the present study recorded a higher percentage of female participation (56.7 %), compared with their male counterpart (43.3 %) for HIV subject. Previous reports had attributed this skewed gender distribution in HIV infection to fact that women could be physiologically more vulnerable to HIV infection through sexual intercourse than men[20-22] and that they also tend to seek health care services more than their male counterpart[23,24]. In the midst of these reasons though, an earlier call made more than a decade ago still remains valid that there is increasing recognition for prevention and treatment programs to address gender inequalities in HIV/AIDS[25]. The study observed that up to 23.3% of the participants were single, 63.3% were married, 3.3% were widowed. It was also observed that intolerance contributed to 30% non-adherence, while forgetfulness contributed to 10% non-adherence (Table 1).

The platelet distribution width (PDW) of the HIV subjects on HAART treatment was significantly higher compared with control subjects (Table 2). The PDW is used to measure the variation in platelet size, it can be increased because the bone marrow is trying to compensate for the lost platelet. This observation is in tandem with earlier report[8]. However, a contrary result of comparable platelet counts in both control subjects and those on HAART treatment has been reported[4]. Platelet count and its indices have been shown to exhibit significant alterations in HIV-infected patients receiving antiretroviral therapy, reflecting ongoing immune activation and potential subclinical haemato-logic effects of the virus and treatment regimen[20]. This could be attributed to ameliorative effect of the HAART treatment. CD4 count was significantly reduced in HIV subjects when compared with control. This could also be as a result of the virus attacking and destroying the CD4 cells and the continuous replication of the virus. The hallmark of HIV infection and subsequently AIDS pathogenesis is a progressive depletion of CD4 T cells population in close association with progressive decline in cellular immunity and cellular susceptibility to opportunistic infection[4]. More interestingly was the observation that CD4 count and Viral Load were seen to be significantly improved in those that have been on treatment for more than five (5) years compared with those less than five (5) years on treatment. Better immune response and suppression of viral replication occurred with longer duration of treatment.

CONCLUSION

This study concludes that Haemostatic parameters were significantly altered in HIV-infected subjects on therapy compared to controls even though there was no derangement in the reference values. Among persons on treatment, those whose treatment duration was over five (>5) years had lower Viral Load, with a higher CD4 count and PCT compared to those whose treatment duration was less than or equal five (≤5) years.

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AUTHOR'S CONTRIBUTIONS

Abunimye DA: formal analysis, conceptualisation, data organisation, writing original draft. **Okpokam DC:** methodology, literature review, manuscript drafting. **Mwankon JP:** data analysis, manuscript writing. **Akwiwu EC:** data analysis, manuscript writing. **Okpokam OE:** literature survey. **Egbe SB:** data analysis. **Ibe OE:** data organisation, literature survey. **Akpotuzor JO:** supervision, critical review. **Obeagu EI:** methodology, conceptualization, scientific validation, manuscript. Final manuscript was checked and approved by all authors.

DATA AVAILABILITY

The related author can provide the empirical data supporting the study's conclusions upon request.

CONFLICT OF INTEREST

There are no conflicts of interest in regard to this project.

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