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Haemostatic, Haematological and Immunological Parameters of HIV-Positive Patients at Two Hospitals in Ilorin, Kwara State, Nigeria

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ABSTRACT

BACKGROUND: Human immunodeficiency virus (HIV), with its attendant opportunistic infections and malignancies, is characterized by the presence of well-pronounced haemostatic, immunological and haematological abnormalities. **AIM:** This study investigated the haemostatic, immunological and haematological parameters of HIV-positive patients at two hospitals in Ilorin, Kwara State, Nigeria. **METHODS:** A total of 54 subjects were recruited for the study, comprising 34 HIV-infected patients who were on highly active antiretroviral therapy (HAART) and 20 HIV-infected patients who were not receiving the treatment, at Sobi Specialist Hospital and Children Specialist Hospital in Ilorin, Kwara State, Nigeria, for a period of 3 months. $CD4^+$ count was analyzed by flow cytometry using the CyFlow® SL and Partec CD4+ autoanalyzer; full blood count was determined using Flow Cytometry using the Sysmex XP-300™ Haematology Analyzer, while select coagulation variables were analyzed using coagulometry. **RESULTS:** The CD4+ count, platelet count, activated partial thromboplastin time and prothrombin time, packed cells volume, red blood cell count, mean cell volume, mean cell haemoglobin, red cell distribution width and mean platelet volume were all significantly ($p < 0.05$) elevated in HIV-seropositive patients who were receiving HAART, relative to those who were not receiving HAART. Hence, HAART improved the basic haemostatic, immunological and hematological indices of the HIV-infected patients.

Key words: $CD4^+$ Count, HAART, haematological, haemostatic, HIV, immunological
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INTRODUCTION

According to the CDC (1), the human immunodeficiency virus (HIV) is a virus that is spread via certain human body fluids and tends to make the immune system weaker by annihilating the body's disease-fighting cells, specifically CD4 cells (often called T cells). If the infection is not treated, HIV will reduce the population of CD4 cells in the body, and this makes it harder for the immune system to repel contagions and other diseases. HIV can subsequently progress to acquired immunodeficiency syndrome (AIDS). The virus is conveyed through body fluids such as semen, blood and breast milk. It produces its effect through infection of T-helper (CD4) cells and cells of the monocyte lineage. According to Cilliers *et al.* (2), viral entry into human T cells requires that the CD4 molecule and the chemokine receptors (CCR-5 and CXCR-4) are present. The HIV infection has four disease stages, and during these periods, a wide range of haematological changes occur, due to defects in the bone marrow and immune cytopenias. These may result from HIV infection, or opportunistic infections and lymphomas. According to Hoffbrand *et al*. (3) and Tagre and Asantewaa (4), they could also be as a result of the side effects of medication used to treat the HIV itself or other complex opportunistic infections (e.g tuberculosis) or lymphomas. There are various blood and bone marrow alteration that are commonly associated with the disease. According to Tagre and Asantewaa (4), this is due to its diverse influences on the haemopoietic tissues.

The HIV/AIDS pandemic is part of one of the greatest health disasters ever faced by mankind. This pandemic has already killed 20 million people. Tagre and Asantewaa (4) reported that presently, according to UNAIDS, approximately 38 million people are living with HIV and HIV.gov (5) stated that several millions of people have died due to causes related to AIDS since the beginning of the epidemic. In Nigeria, available data according to the NACA (6), indicate that HIV prevalence is 1.4%, with Kwara State having a prevalence of 1.0%. According to Parashar *et al*. (7), each year, three million people die of HIV/AIDS (Report on the global HIV/AIDS epidemic, 2002). However, the same author (7) stated that many of these deaths associated with HIV/AIDS are actually preventable, if the HIV/AIDS patients have access to antiretroviral therapy (ART), especially HAART.

The World Health Organization (WHO), in September 2003, stated that the failure to make antiretroviral therapy available to patients in developing countries, would very likely lead to a public health emergency that would affect the whole world. Consequently, the WHO (8) partnered with the Joint United Nations Programme on HIV/AIDS (UNAIDS) and partners, to achieve the goal of giving three million people in emerging economies antiretroviral therapy by the end of 2005 (also known as the "3 by 5" Initiative). This was the initial target of the partnership; although the WHO (8) stated that its long term objective was for every HIV/AIDS patient in the world to have access to ART. The earlier author (8) also stated that the major goal of antiretroviral therapy is to increase the survival rate, and also improve the quality of life of persons who are suffering from HIV/AIDS. By reducing the HIV viral load to sustainable untraceable levels, it is hoped that ART will significantly play a part in the prevention of HIV. Medical laboratory support is paramount in every area relating to the diagnosis and management of HIV. The diagnosis of HIV cannot be established by any means other than blood tests in the laboratory. CD4 lymphocyte count is a necessary diagnostic criterion for the commencement of antiretroviral therapy

and for monitoring the outcome of treatment. Thus, both immunological and hematological ART monitoring are therefore, primarily reliant on an effective laboratory services. Hoffbrand *et al*. (3) reported that certain antiretrovirals used to treat HIV-infected patients have been associated with myelotoxic side effects and implicated as a possible cause of hematological dysfunction, though the possible hematological, haemostatic and Immunological side effects of HAART have not been studied in Ilorin, Kwara State, Nigeria. Hence, the aim of this study was to evaluate selected haemostatic, immunological and haematological parameters of HIV-positive patients placed on HAART and those who were not receiving the treatment at two hospitals (Sobi Specialist Hospital and Children Specialist Hospital) in Ilorin, Kwara State, Nigeria.

MATERIALS AND METHODS

Study area
The study was carried out at Sobi Specialist Hospital and Children Specialist Hospital, both located in Ilorin, Kwara State, Nigeria, while the analysis of the samples was done in the Department of Haematology, Sobi Specialist Hospital, Ilorin, Kwara State.

Ethical considerations
Ethical approval was obtained from the Ethics and Research Committee of the Kwara State Ministry of Health, Ilorin, Kwara State, Nigeria (ref no. MOH/KS/EU/777/386).

Study design
The study design was a comparative cross-sectional study.

Subjects
After an informed consent was obtained, a total of 54 HIV-positive patients comprising 34 HIV-positive patients who were on HAART and 20 HIV-positive patients who were not on HAART, were randomly selected for the study. There were a total of 33 females and 21 males who participated in the Study, giving a total of 54 subjects. A total of 32 (18 female and 14 male) subjects from Sobi Specialist Hospital and 22 (13 female and 9 male) subjects from Children Specialist Hospital Ilorin, participated in the Study. Both groups of subjects were attending clinic at Sobi Specialist Hospital and Children Specialist Hospital Ilorin, Kwara State.

Inclusion criteria
The inclusion criteria were HIV-seropositive patients of both sexes, aged between 18 and 50 years, placed on HAART and HIV-seropositive patients not placed on HAART.

Exclusion criteria
Patients at the extremes of age, pregnant women, HIV and TB co-infected subjects and those on anticoagulant therapy, through their responses to the administered questionnaires, were excluded from the study. In addition, those on cytotoxic chemotherapy and radiotherapy were excluded, since they may have weakened immune system.

Sample collection
A portion of 6 mL of blood was collected aseptically from the antecubital vein by clean venipuncture from each participant, with each subject in a sitting position. The blood sample was aliquoted as follows: 1.8 mL of blood was dispensed into plastic containers containing 0.2 mL of 3.2% sodium citrate anticoagulant to give a final blood:citrate ratio of 9:1. The blood sample was centrifuged immediately at room temperature at 2000 g for 10 minutes to obtain platelet-poor plasma, which was separated for activated partial thromboplastin time test and prothrombin time test. Then 4 mL was

dispensed into bottles containing the di-potassium salt of ethylene diamine tetra-acetic acid (K_2 EDTA) at a concentration of 1.5 mg/mL of blood for the $CD4^+$ Count and full blood count.

Laboratory analysis

Prothrombin time (PT) testing was done using a water bath and reagents according to the method of Cheesbrough (9). A volume of 0.1 mL of the patient's plasma was dispensed into a pre-warmed Khan tube placed in a water bath at 37°C. Pre-warmed 0.2 mL of thromboplastin-calcium reagent was added. A control was set alongside with the test. Stop watch was started immediately and the time taken for the mixture to clot was recorded.

For the activated partial thromboplastin time (APTT) test, the method of the earlier author (9) was applied. A volume of 0.1 mL of the subject's plasma was dispensed into a pre-warmed glass tube placed in a water bath at 37°C. Then 0.1 mL of kaolin-phospholipids commercially prepared solution was added. The mixture was allowed to stand in a water bath at 37°C for 3 minutes, after which 0.1 mL of pre-warmed $CaCl_2$ was added and stop watch was started immediately. A control was set alongside the test. The time taken for the mixture to clot was recorded.

The full blood count was performed according to the method of Sysmex Corporation (10), using the Sysmex XP 300 automated analyzer to assess the haematological parameters including the haemoglobin (Hb) concentration, total white blood cell (WBC) count, packed cells volume (PCV), red blood cell (RBC) count, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC), red cell distribution width (RDW), mean platelet volume (MPV) and platelet count.

The CD4+ count was done with Cyflow counter and Partec CD4+ easy count kit according to Fryland *et al*. (8). Fluorescent-labeled antibodies directed against the human cell surface marker CD4+ were used for labelling cells in whole blood samples, applying the CD4+ easy count kit (Partec Code No. 05-8401) and the Cyflow® Counter (Partec Code No. CY-S-3022).

Statistical analysis

Data collected were analyzed using the IBM SPSS statistic software (version 25). The students' t-test for independent samples was used to test for the significance of differences between the two groups. $P < 0.05$ was considered statistically significant.

RESULTS

Table 1 presents the age and gender distribution among the HIV-positive patients who were on HAART therapy and those who were not on HAART therapy. There were total of 54 subjects, comprising 34 HIV-positive subjects who were on HAART and 20 control subjects who were not on HAART. Out of the 54 subjects, 22 (65%) were females while 12 (35%) were males for the HIV-positive subjects who were on HAART. Among the 20 controls, 11 (55%) were females while 9 (45%) were males. The age distribution of the HIV-positive subjects who were on HAART include< 20 years: 1 subject; 20-29 years: 5 subjects; 30-39 years: 12 subjects; 40-49 years: 15 subjects and ≥ 50 years: 1 subject. For the HIV-positive subjects who were not on HAART, their ages were: < 20 years: 0 subject; 20-29 years: 8 subjects; 30-39 years: 7 subjects; 40-49 years: 5 subjects; and ≥ 50 years: 0 subject.

TABLE 1: Age and gender distribution among HIV-positive patients who were on HAART therapy and those who were not on HAART therapy

Age/Gender	HIV-positive on HAART	HIV-positive not on HAART
Age (Years)	<20 (3%) N=1	<20 (0%) N=0
	20-29 (15%) N=5	20-29 (40%) N=8
	30-39 (35%) N=12	30-39 (35%) N=7
	40-49 (44%) N=15	40-49 (25%) N=5
	≥50 (3%) N=1	≥50 (0%) N=0
Gender	Female (65%) N=22	Female (55%) N=11
	Male (35%) N=12	Male (45%) N=9

Table 2 presents the mean levels of the CD4+ count, and total WBC count in the HIV-positive patients on HAART and those not on HAART (control). The mean of the CD4+ count was significantly higher in the test group (546.55±395.82 cells/mm^3) compared to that of the control (177.70±111.95 cells/mm^3) (p value = 0.001). However, there was no significant difference between the average total WBC count in the test subjects (6.21±3.06 X10^9/l) compared to the control group (4.94±1.68 X10^9/l) (p = 0.140).

TABLE 2: CD4$^+$ and total WBC counts in HIV positive patients on HAART and HIV-positive patients not on HAART

Parameters	HIV-positive on HAART (N=34)	HIV-positive not on HAART (N=20)	*p*-value
CD4+ count (cells/mm^3)	546.55±395.82	177.70±111.95	0.001*
Total WBC count (X10^9/l)	6.21±3.06	4.94±1.68	0.140

*p-value is significant at 0.05. N = 54.
Key: CD4+ count= CD4+ count (Normal range: 500-1200 cells/mm^3);
Total WBC count = Total white blood cell count (Normal range: 4.0- 11.0 X 10^9/l)

Table 3 presents the mean levels of the platelet count, activated partial thromboplastin time (aPTT), and prothrombin time (PT) in HIV-positive patients on HAART (test) and HIV-positive patients not on HAART (control). The mean level of the platelet count was significantly ($p < 0.05$) higher in the test group (224.00±145.55 X10^9/l) compared to that of the control (138.70±33.88 X10^9/l) (p = 0.001). However, the mean level of the aPTT was significantly reduced in the test group (36.55±5.29 secs) compared to that of the control (75.05±16.74 secs) ($p < 0.001$). Likewise, the mean value of the PT was significantly lower in the test group (12.90±1.92 secs) compared to that of the control (20.85±4.43 secs) ($p < 0.001$).

TABLE 3: Platelet count, activated partial thromboplastin time, and prothrombin time in HIV positive patients on HAART and HIV-positive patients not on HAART

Parameters	HIV-positive on HAART (N=34)	HIV-positive not on HAART (N=20)	*p*-value
Platelet count ($X10^9$/l)	224.00±145.55	138.70±33.88	0.001*
aPTT (seconds)	36.55±5.29	75.05±16.74	<0.001*
PT (seconds)	12.90±1.92	20.85±4.43	<0.001*

**p*-value is significant at 0.05. N = 54.
Key:
Platelet count = Platelet count (Normal range: 150-400$X10^9$/l);
aPTT = Activated partial thromboplastin time (Normal range: 21-38 seconds);
PT = prothrombin time (Normal range: 10-15 seconds);

Table 4 presents the mean levels of the Hb, PCV, RBC, MCV, MCH, MCHC, RDW, MPV in HIV-positive patients on HAART and HIV-positive patients not on HAART (control). The mean value of the Hb concentrations were comparable (p = 0.060) in the HIV subjects who were on HAART (10.07±1.26 g/dl) and in the HIV subjects who are not on HAART (9.07±1.75 g/dl). However, the mean value of the PCV was significantly higher in the test group (33.91±3.75%) compared to that of the control (27.58±6.64%) (p = 0.001). Similarly, the mean level of the RBC count was significantly higher in the test group (4.44±0.67 $X10^{12}$/l) compared to that of the control (3.62±0.71 $X10^{12}$/l) (p < 0.001). The mean level of the MCV was also significantly (p = 0.003) higher in the test group (77.85±4.89 fl) when compared with the control group (71.84±6.03 fl). In contrast, the mean levels of the MCH and RDW (23.04±2.01 pg and 37.63±2.45 fl, respectively) were significantly (p = 0.002) reduced in the test group compared to those of the control group (26.43±3.35 pg and 56.40±4.92, respectively). However, the mean level of the MCHC did not vary significantly (p = 0.479) in the test group (29.57±1.28 g/dl) compared to that of the control (29.14±1.98 g/dl). Conversely, the mean level of the MPV was significantly (p < 0.001) increased in the test group (9.25±0.59 fl) compared to that of the control (6.98±0.58 fl).

Table 4: Hb concentration, PCV, RBC, MCV, MCH, MCHC, RDW, MPV in HIV-positive patients on HAART and HIV-positive patients not on HAART

Parameters	HIV-positive on HAART (N=34)	HIV-positive not on HAART (N=20)	*p*-value
Hb (g/dl)	10.07±1.26	9.07±1.75	0.060
PCV (%)	33.91±3.75	27.58±6.64	0.001*
RBC (X10^{12}/l)	4.44±0.67	3.62±0.71	<0.001*
MCV (fl)	77.85±4.89	71.84±6.03	0.003*
MCH (pg)	23.04±2.01	26.43±3.35	0.002*
MCHC (g/dl)	29.57±1.28	29.14±1.98	0.479
RDW (fl)	37.63±2.45	56.40±4.92	<0.001*
MPV (fl)	9.25±0.59	6.98±0.58	<0.001*

*p-value is significant at 0.05. N = 54.

Key:
Hb = Haemoglobin concentration (Normal range: Females → 12-16 g/dl; Males → 14-18 g/dl)
PCV = packed cell volume (Normal range: Females → 35-47%; Males → 40-52%)
RBC = Red blood cell count (Normal range: 4.0-6.2 X 10^{12}/l)
MCV = mean cell volume (Normal range: 82-93 fl)
MCH = mean cell haemoglobin (Normal range: 25-34 pg)
MCHC = mean cell haemoglobin concentration (Normal range: 31-36 g/dl)
RDW = red cell distribution width (Normal range: 39-46 fl)
MPV = mean platelet volume (Normal range: 7.5- 12.0 fl)

Table 5 presents the effect of gender on selected immunological, haematological and haemostatic parameters in HIV-positive patients on HAART and HIV-positive patients not on HAART.

Male subjects who were on HAART had a reduced $CD4^+$ count, total WBC count, platelet count, aPTT, MCV, MCH, MCHC and MPV compared to the females (p = 0.848, 0.272, 0.580, 0.823, 0.410, 0.922, 0.216 and 0.080 respectively), but had increased PT, Hb, PCV, RBC count and RDW compared to the females (p = 0.798, 0.285, 0.291, 0.359 and 0.432 respectively).

For male subjects who were not on HAART, they had decreased total WBC count, aPTT, RBC count, MCH and RDW compared to the females (p = 0.794, 0.909, 0.878, 0.794 and 0.084 respectively), but they possessed elevated $CD4^+$ count, platelet count, PT, Hb, PCV, MCV, MCHC and MPV in comparison with the females (p = 0.221, 0.405, 0.744, 0.908, 0.643, 0.460, 0.687 and 0.926 respectively).

Table 5: Effect of gender on selected immunological, haematological and haemostatic parameters in HIV-positive patients on HAART and HIV-positive patients not on HAART

	HIV-positive patients on HAART			HIV-positive patients not on HAART		
Parameters	**Male (n=12)**	**Female (n=22)**	***p-value***	**Male (n=9)**	**Female (n=11)**	***p*-value**
CD4+ count (cells/mm^3)	517.75±245.00	542.05±395.58	0.848	212.22±149.45	149.45±63.21	0.221
Total WBC count (X10^9/l)	5.56±1.74	6.62±3.03	0.272	4.82±1.75	5.03±1.70	0.794
Platelet count (X10^9/l)	214.08±94.99	238.91±136.38	0.580	145.89±41.44	132.82±26.87	0.405
aPTT (seconds)	36.33±5.94	36.77±5.13	0.823	74.56±17.60	75.45±16.87	0.909
PT (seconds)	13.00±1.95	12.82±1.98	0.798	21.22±5.54	20.55±3.53	0.744
Hb (g/dl)	10.50±0.98	10.09±1.08	0.285	9.12±1.93	9.03±1.70	0.908
PCV (%)	35.03±3.75	33.85±3.26	0.291	28.37±7.12	26.93±6.51	0.643
RBC (X10^{12}/l)	4.59±0.72	4.36±0.68	0.359	3.59±0.82	3.65±0.65	0.878
MCV (fl)	77.93±6.78	79.96±6.75	0.410	72.98±5.17	70.91±6.74	0.460
MCH (pg)	24.13±3.11	24.24±3.03	0.922	26.20±4.91	26.61±1.40	0.794
MCHC (g/dl)	28.96±1.46	29.54±1.18	0.216	29.34±2.56	28.97±1.45	0.687
RDW (fl)	37.31±1.91	36.53±3.08	0.432	54.30±3.69	58.12±5.27	0.084
MPV (fl)	8.86±0.58	9.27±0.67	0.080	6.99±0.65	6.96±0.54	0.926

*p-value is significant at 0.05. N = 54.

Key:
CD4+ count= CD4+ count (Normal range: 500-1200 cells/mm^3);
Total WBC count = Total white blood cell count (Normal range: 4.0- 11.0 X 10^9/l)
Platelet count = Platelet count (Normal range: 150-400X10^9/l);
aPTT = Activated partial thromboplastin time (Normal range: 21-38 secs);
PT = prothrombin time (Normal range: 10-15secs);
Hb = Haemoglobin concentration (Normal range: Females → 12-16 g/dl; Males → 14-18 g/dl)
PCV = packed cell volume (Normal range: Females → 35-47%; Males → 40-52%)
RBC = Red blood cell count (Normal range: 4.0-6.2 X 10^{12}/l)
MCV = mean cell volume (Normal range: 82-93 fl)
MCH = mean cell haemoglobin (Normal range: 25-34 pg)
MCHC = mean cell haemoglobin concentration (Normal range: 31-36 g/dl)
RDW = red cell distribution width (Normal range: 39-46 fl)
MPV = mean platelet volume (Normal range: 7.5- 12.0 fl)

DISCUSSION

Odunukwe *et al*. (12) stated that Highly active antiretroviral therapies (HAART) are the benchmarks for HIV disease management. In this study the CD4$^+$ count was significantly higher in the test group compared to that of the control (Table 2). This finding agrees with that of Johnathon *et al*. (13). Initiation of HAART may have interrupted multiple points in the virus life,

causing an increase in CD4+ cells, unlike in those who were not on HAART. According to Moyle (14), Depletion of CD4 lymphocytes in HIV positive patients not on HAART is the characteristic feature of HIV infection and predicts an individual's risk for infection with opportunistic pathogens, as well as other complications of HIV infections. The central feature of HIV disease comprises the opportunistic infection and malignancy resulting from CD4 cell depletion. Vajpayee *et al*. (15) reported that ART helps in the significant reversal of the morbidity and mortality associated with HIV, through the restoration of CD4 cell numbers and function.

There was no significant difference between the total WBC count in the test subjects compared to the control group. This agrees with the finding of Cheesbrough (9). In the test and control subjects, the total WBC counts were within the normal range, and this contradicted the finding of Raman *et al*. (16). The mean level of the platelet count was significantly raised in the test group compared to that of the control (Table 3). This agrees with the findings of Dikshit *et al*. (17) and De Santis *et al*. (18). The incidence of thrombocytopenia was slightly higher in patients not receiving HAART therapy in contrast to those on HAART. This difference is probably due to improvement in disease after the commencement of HAART. Thus, it is evident that the incidence of thrombocytopenia was high with low $CD4^+$ counts among those not on HAART. Thrombocytopenia in HIV infection is also caused by increased destruction of platelets due to circulating immune complexes being deposited on platelets. The presence of specific antiplatelet antibodies and megakaryocytes resulting from HIV infection has also been hypothesized according to Akinbami *et al*. (19).

However, the mean aPTT and PT were significantly reduced in the test group compared to those of the control group. This finding is in agreement with that of Omoregie *et al*. (20), who also reported an increased PT and APTT in HIV patients not on HAART. Similarly, Jong *et al*. (21) also reported an extended PT and APTT in 6% and 2% of their study subjects respectively. According to Andrade and Cotter (22), the cause of higher PT and APTT could be due to endothelial activation, leading to thrombosis and consumption of coagulation factors. There is increased occurrence of thrombosis in HIV patients due to hypercoagulable states like protein C, protein S, antithrombin III deficiency and presence of antiphospholipid antibodies. Park *et al*. (23) also reported an elevated PT and aPTT in patients with hypercoagulable state. Anti-cardiolipin antibodies and lupus anticoagulant observed in some HIV patients can also lead to elevated aPTT. Presence of circulating coagulation inhibitors has also been reported among HIV patients. Zeichner *et al*. (24) reported the presence of an acquired inhibitor for factor VIII in a patient with co-existing HIV.

Some haematological impediments were observed in this study. The Hb, PCV and red cell values were observed to be reduced in HIV-seropositive subjects on HAART; although they were much more reduced in HIV-seropositive subjects not on HAART (Table 4). This reduction does not concur with the findings of Akinbami *et al*. (19) and Erhabor *et al*. (25), but is consistent with the findings of Amegor *et al*. (26) and that of Mena *et al*. (27). The explanation for this is the widespread effect of HIV/AIDS on erythropoiesis, since the precursor cells are inhibited from differentiating and developing into mature red cells.

This study also showed that the MCH and MCHC values were significantly reduced in

HIV-seropositive subjects compared with the controls. This finding agrees with that of Obirikorang and Yehoah (28). The MCV was found to be significantly increased in HIV-seropositive subjects on HAART compared with those not on HAART, this could be as a result of presence of iron deficiency anaemia, vitamin B12 and folic acid deficiencies observed among HIV-seropositive subjects, leading to reduced erythropoiesis. Also, the RDW of HIV-seropositive subjects not on HAART was significantly higher than the seropositive subjects on HAART. This finding supports that of Gallego *et al*. (29) and could be as a result of high level of anisocytosis among HIV-seropositive subjects, as a result of decreased RBC production or ineffective erythropoiesis.

The mean level of the MPV was significantly increased in the HIV-seropositive subjects on HAART compared to that of the HIV-seropositive subjects not on HAART. According to Mena *et al*. (27), few studies have assessed MPV values in HIV-infected populations. The present study's findings are consistent with those of the earlier author (27), who studied 34 HIV-infected subjects not on HAART and observed two-thirds to have thrombocytopenia, of which 92% had very low mean platelet volume, which may be due to myelosuppressive bone marrow disorders cause by HIV. Haematological alterations in HIV-infected patients have been found to vary among different studies. This, according to Wankah *et al*. (30) may be as a result of factors such as drugs, immune mechanisms, opportunistic infections or direct influence of the virus.

The effect of gender on haematological, haemostatic and immunological parameters, remains a highly debated topic till date according to Thorsteinsson *et al*. (31). There were no significant differences observed between the haematological, haemostatic and immunological parameters of males compared to females. These findings agree with those of Thorsteinsson *et al*. (31), Ko *et al*. (32), Moore *et al*. (33) and Nicastri *et al*. (34).

Conclusion

This study has shown that HIV-seropositive patients who were not receiving HAART therapy are at increased risk of deranged haematological parameters such as the red blood cell count, packed cells volume, total white blood cell count and the red cell indices, consequently leading to anaemia. The $CD4^+$ and platelet counts were higher in the HIV-seropositive patients who were on HAART than in those were not receiving HAART. Since coagulation defects become more severe as the disease advances, the basic coagulation tests like platelet count, PT and especially aPTT can be utilized as prospective screening tests to evaluate severity and progress of HIV infection in settings with limited resources, where $CD4^+$ count is not available. It is also concluded that there is no association between gender and haematological, haemostatic and immunological parameters in those receiving HAART and those not receiving HAART.

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