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HIV-1 RNA Viral Load, CD4 Count and Some Haematological Parameters of People Living with HIV in the Enugu Metropolis

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Abstract

Background: It is widely known that the human immune-deficiency virus (HIV) induces biochemical and physiological changes in affected persons. Consequently, the overall aim of this study was to evaluate the HIV-1 RNA viral load, CD4 count, and certain haematological parameters among HIV treatment-naïve subjects in the Enugu metropolis of Nigeria. **Materials and Methods**: A total of 252 HIV-infected, ART-native subjects (≥18) attending the University of Nigeria Teaching Hospital (UNTH) in Ituku-Ozalla, Enugu were recruited for this study and were made up of 157 (62.3%) females and 95 (37.7%) males. A total of 250 HIV-negative subjects were used as control subjects (100 males and 150 females). Blood samples were collected from all the participants and their HIV-1 status was confirmed by an immunoblot confirmatory test. Their haematological parameters and CD4 count were evaluated, while the HIV-1 viral load was only assessed on confirmed HIV-positive subjects. **Results**: There was female predominance (62.3%) among these HIV-positive subjects. The mean age of HIV-positive subjects was 39.16 ± 10.08 years while the mean age of the control subjects was 34.8 ± 8.6 years. The age group of 31 - 40 years (102/252 (40.5%)) constituted most of the test subjects. The total white blood cells (TWBC) (6.05 ± 5.46), lymphocyte counts (36 ± 14), haemoglobin concentrations (Hb) (9.85 ± 7.36) and the

CD4 counts (242 ± 228) of the HIV-infected subjects showed a significant difference when compared with their control counterpart values of TWBC (4.5 ± 0.568), lymphocytes (39.67 ± 8.2), Hb (13.48 ± 1.5), and CD4 counts (807 ± 249) ($p < 0.05$). The platelet count (241 ± 103) and the neutrophil count (53.38 ± 15) of the infected subjects, when compared with the control subjects, showed no significant difference ($p > 0.05$). Anaemia, lymphocytopenia, and thrombocytopenia were the haematological abnormalities seen in the HIV-positive subjects. HIV viral load correlated with haemoglobin concentration, CD4 count, lymphocyte count, and neutrophil count ($p < 0.05$). Moreover, the age of the test subjects also correlated with their CD4 counts ($p < 0.05$). **Conclusion**: Prognostic factors, such as haemoglobin concentrations, CD4 counts, lymphocyte counts, and neutrophil counts can be used to monitor patients' viral loads since they correlate with the latter; furthermore, age is a factor that should be considered in the management of HIV-positive patients.

Keywords

HIV-1 RNA, CD4 Count, Haematological Parameters

1. Introduction

HIV is a lentivirus (a member of the retrovirus family) and is the etiological agent of AIDS. This illness was first described in 1981 and HIV-1 was isolated by the end of 1983 [1]. Since then, AIDS has become a worldwide epidemic, ever-expanding in scope and magnitude as HIV infections affect different populations and geographic regions [2]. Its devastating health effects can be seen globally with over 39 million HIV/AIDS-related deaths to date and more than 36 million people currently living with HIV [3]. Despite therapy increase in the use of ARTs since the mid-2000s and the resulting decline in mortality, it remains the most common cause of death in sub-Saharan Africa. In addition, the burden of the global HIV epidemic is disproportionately concentrated in this region where, in 2017, 75% of the world's AIDS-related deaths and 65% of its new infections were recorded. Furthermore, 71% of people living with HIV reside here [4] [5]. Consequently, Nigeria is one of the countries with the highest number of people (1.4%) living with HIV, accounting for 9% of the global HIV burden (HIV) [6] [7].

The risk of HIV acquisition and/or transmission is usually determined by a combination of behavioural, biological, and structural factors, such as the frequency of unprotected sexual intercourse, a high number of concurrent sexual partners, the presence of other sexually transmitted infections (STIs), limited access to prevention, and treatment services, social stigma, and discriminatory legal or regulatory policies [8]. Structural factors that elevate risk among FSW include extreme poverty, concomitant lack of familial and social support, gender inequality, stigma, discrimination, physical and sexual violence, and legal and regulatory policies penalizing sex work. STIs, especially genital ulcers, may play

an important role in HIV transmission and acquisition.

Research plays an important role in how clinicians usually assess the risk of HIV disease progression from a combination of CD4 cell count and HIV-1 RNA plasma viral load or viral load alone. The CD4 cell count is generally regarded as a marker of the degree of immune deficiency, while the VL is considered a marker of disease activity or the rate at which the CD4 cell count is likely to decrease. The above-mentioned study indicated that VL provides prognostic information about patients' risk of developing conditions that are part of active AIDS, independent of CD4 cell count [9].

Individuals with a higher VL during the asymptomatic phase in the absence of ART were more likely to rapidly progress to AIDS. Plasma HIV-1 RNA VL levels of HIV-infected persons are a strong predictor of sexual and perinatal transmission of HIV, the rapidity of achieving viral suppression with ART, and the probability of progression to AIDS or death. Pre-ART HIV RNA VL levels can be exploited in the strategic timing of ART, particularly in settings of limited resources [10]. An African study that modelled the relationship between VL and heterosexual transmission risk in discordant couples, concluded that 90% of new HIV infections could be eliminated by treating only people with HIV VL > 10,000 copies/mL [11] [12] [13].

Haematological abnormalities are common complications of HIV infections and increase as the disease progresses. In ART-treated and untreated individuals alike, various types of haematological abnormalities are common [14]. Of these, anaemia, leukopenia, and thrombocytopenia are the most common occurrences of an HIV infection. The use of anti-retroviral drugs could positively or negatively affect these disorders. Thus, a specific diagnosis and the determination of haematological and immunological parameters are required to initiate and implement early treatment to avert the disease's advance [14] [15]. Later research Enawgaw *et al.*, 2014 [14] also recommends that HIV patients be investigated for haematological and immunological changes followed by appropriate therapeutic interventions.

However, despite numerous studies on the use of HIV-1 plasma viral load and CD4 cell count in HIV prognosis, studies that have integrated HIV-1 VL, CD4 count, and haematological parameters in determining HIV prognosis are severely lacking. Therefore, this work aims to determine HIV-1 RNA VL, CD4 count, and certain haematological parameters of people living with HIV in the Enugu State of Nigeria. Furthermore, it seeks to correlate the haematological findings with HIV VLs and CD4 counts.

2. Materials and Methods

2.1. Study Area

This study was conducted among HIV/AIDS patients attending the UNTH, Ituku-Ozalla, Enugu, which is supported by the Centre for Clinical Research of USA President's Emergency Plan for AIDS Relief (PEPFAR).

2.2. Study Population

A total of 252 HIV patients whose serological reactivity was determined by enzyme immunoassay and confirmed by Western blot analysis were recruited into the study. Among the probands comprised, there were patients with AIDS diagnosed by immunological or clinical criteria based on the CDC1993 revised classification for HIV infection. This particular study was conducted from March 2013 to February 2016, after meeting the inclusion criteria for the ART programme and obtaining the subjects' informed consent. The inclusion criteria for patients were HIV seropositive status (confirmed by the Western blot test) and informed (written) consent. The exclusion criteria were age (<18 years), severe renal or hepatic failure, a prior history of highly active anti-retroviral therapy (HAART) usage, any bleeding disorders, and the inability to provide consent or non-consent.

2.3. Study Design

This study was prospective, with an observational cohort. The (252) HIV/AIDS patients that were enrolled in this study comprised 95 (37.7%) males and 157 (62.3%) females.

2.3.1. Sample Size

The sample size was determined using the established formula by Rosie Cornish *et al.* [16].

2.3.2. Sample Collection

Two specimen bottles were used for each subject, namely an anticoagulant bottle (containing potassium EDTA for $CD4^+$count and full blood count) and a plain bottle for HIV screening and confirmation. Samples were collected after receiving patients' consent and assuring them of the utmost confidentiality. Blood samples of 8 ml were collected by a clear vein puncture via the antecubital vein,4 mls were then dispensed into the already labelled, different bottles. Subsequently, serum was extracted from the blood clot in the plain tubes and stored in a cryovial at −80˚C until they were needed. The blood samples in potassium EDTA were never stored, but were used within 6 hours to estimate the $CD4^+$ T-cell count and full blood count. The EDTA sample was spun at 1200 g and the plasma was separated and stored at −80˚C in freezers for VL estimation.

2.3.3. Ethical Clearance

Ethical approval was obtained from the ethics review committee of the UNTH Ituku-Ozalla, Enugu for the study to be carried out (NHREC/05/01/2008B-1RB00002323).

2.4. Laboratory Analysis

2.4.1. HIV Testing

Testing for HIV was done using the methods of [17] who utilised Abbot Determine™ HIV-1/2 serum/plasma rapid assay (Abbot, Max-Planck-Ring2, Wies-

baden, Germany) based on antigen-antibody bound complex formation, which is detected with the use of a chromogen substrate. An amount of 50 µl of serum or plasma was applied to the sample pad and briefly incubated for 15 - 60 minutes. The appearance of a red bar in both the control and test window of the strip was interpreted as positive while one red bar in the patient window of the strip was considered negative, and, no bar in the control window was regarded as invalid, leading to repetition of the test.

2.4.2. HIV Confirmatory Test

A HIV confirmatory test was done using Qualicode™ HIV 1/2 kit cat No, DK-C 152-024 (Immunoetics Inc Boston and MA USA), a qualitative immunoblot assay based on the Western Blotting principle in the following manner: firstly, the assay was performed on an immunoblot membrane containing HIV-1 viral lysate proteins (HTLV-111 B strain) and a recombinant HIV-2 protein. The procedure was carried out by following the manufacturer's instructions. The result was interpreted by aligning the positive control strip and the test strip bands with the matching bands on the kit's reference card. It was then scored as follows: gp160 outer ENV glycoprotein precursors, gp120 outer ENV glycoprotein, p66, and p51 reverse transcriptase components of POL, gp41 transmembrane ENV glycoprotein, p31 endonuclease component of POL, and p24, p17 GAG protein [18] [19].

2.4.3. Viral Load (VL) Estimation

The Amplicor HIV-1 Monitor PCR Test kit (Roche Diagnostic Systems, Mannheim Germany) was used for the VL estimation and the manufacturer's instructions were followed. The viral estimation involved five major processes, namely: specimen preparation, reverse transcription (RT) of target RNA to generate complementary DNA (cDNA), PCR amplification of target cDNA using HIV-1 specific complementary primers, hybridisation of the amplified products to oligonucleotide probes specific to the target(s), and the detection of the probe bound amplified products by means of calorimetric determination.

2.4.4. Estimation of $CD4^+$ T- Lymphocyte Counts

The $CD4^+$ T-lymphocyte count estimation was done by using a flow cytometry automated machine (CyflowPartec, Germany) according to its manufacturer's instructions. This test is based on the intensity of the fluorescence emitted by fluorescing complexes formed by fluorescent antibodies and anticoagulated blood when introduced into a flow cytometer. The intensity is directly proportional to the number of $CD4^+$ cells present. Briefly, 20 µl of anticoagulated blood was pipetted into a test tube followed by 20 µl of CyflowPartec (Germany) CD4 beads. The set-up was then mixed properly by gentle inversion and incubated in the dark for 15 minutes. Furthermore, 800 µl of no-lyse buffer was added and the sample was read using the Partec flow cytometer. The $CD4^+$ count was recorded and expressed in cells/µl [20].

2.4.5. Full Blood Count

Peripheral full blood counts were determined using Mindray BC-3200 (Shenzhen Mindray Bio-Medical Electronics Co., Ltd China) haematology auto analyser instrument [21].

2.5. Statistical Analysis

This study made use of descriptive statistics to obtain the mean and standard deviation of the age groups and certain test results. The differences between groups were analysed using students' t-tests. The means of more than two groups were analysed using ANOVA and a post hoc comparison was then done using Tukey. Next, categorical variables were compared using contingency tables and the Chi-square (χ^2). Differences between groups were considered significant at a p-value < 0.05. The statistical package for social sciences (SPSS) for windows 15.0 version was used for all statistical analyses.

3. Results

Table 1 presents the demographic status of the study population and their control counterparts. A total of 252 HIV-infected, ART-naïve subjects, consisting of 157 (62.3%) females and 95 (37.7%) males, were recruited. A total of 250 HIV-negative subjects were used as control subjects, consisting of 100 (40%) males and 150 (60%) females. There was a female predominance among the HIV-positive subjects. The mean age of HIV-positive subjects was 39.16 ± 10.08 years, while the mean age of the control subjects was 34.8 ± 8.6 years. When compared to their control counterparts, no significant difference was seen ($p > 0.05$). The age group of 31 - 40 years (102 /252 (40.5%) constituted most test subjects, which suggests that this age group is more prone to be affected by HIV.

Table 2 below is a summary of the haematological profiles, VLs, and CD4 counts of HIV-positive subjects and their control counterparts. The TWBC of the HIV-infected subjects were significantly higher than those of their control counterparts ($p < 0.05$). The haemoglobin (Hb) concentration test of HIV-positive subjects was also significantly higher than that of the control group ($p < 0.05$). In contrast, the neutrophils (NEUT) of the test subjects were not statistically significant when compared to the control group's statistics while the lymphocyte (LYMP) counts of the participants were significantly higher than those of the control group ($p < 0.05$). The platelet count difference was not statistically significant ($p > 0.05$), and CD4 counts were significantly lower than their control group counterparts ($p < 0.05$).

Table 3 below shows the comparison of haematological parameters based on HIV infection stages. HIV infection (Stage 1) was comprised of HIV-positive subjects with CD4 counts equal to or greater than 500 cells/μl. Stage 2's participants had CD4 counts ranging from 200 to 499 cells/μl while Stage 3 subjects had CD4 counts of fewer than 200 cells/μl (see Table 4). When comparing the TWBC of the HIV infection in Stage 1 (5.90 ± 1.69), Stage 2 (6.10 ± 2.93) and Stage 3 (6.06 ± 7.02), respectively, no significant difference could be detected

($p > 0.05$). In addition, when the haemoglobin concentration of Stage 1 (10.54 ± 1.67), Stage 2 (9.92 ± 2.07) and Stage 3 (9.64 ± 9.85) were compared, no significant difference was seen ($p > 0.05$). However, a significant difference was seen when the neutrophil counts of Stage 1 (2.72 ± 0.61), Stage 2 (2.90 ± 0.80) and Stage 3 (3.55 ± 0.91) were compared ($p < 0.05$). A significant difference was again seen when the lymphocyte count of Stages 1 (2.60 ± 0.57), 2 (2.58 ± 0.74,) and 3 (1.86 ± 0.86) were compared ($p < 0.05$). No significant differences were detected when the means of platelets were compared while, the VL of the three infection stages were significant ($p < 0.05$).

Table 1. Demographic characteristics of HIV-positive subjects and their control counterparts.

	Test			Control			
Age ranges	**Total n = 252**	**Male n = 95**	**Female n = 157**	**Total n = 250**	**Male n = 100**	**Female n = 150**	**P-value**
Mean age	39.16 ± 10.08			34.8 ± 8.60			0.188
17 - 30	52	15	37	50	14	36	
31 - 40	112	50	62	114	52	62	
41 - 50	55	20	35	72	30	42	
>50	32	10	22	26	11	15	

Table 2. Summary of the haematological profiles, VLs, and CD4 counts of HIV-positive subjects and their control counterparts.

Parameters	Test n = 252	Control n = 250	F-value	P-values
TWBC ($\times 10^9$/L)	6.05 ± 5.46	4.5 ± 0.568	20.02	0.000*
Hb (g/dl)	9.85 ± 7.36	13.48 ± 1.50	22.21	0.000*
NEUT ($\times 10^9$/L)	3.23 ± 0.91	2.35 ± 0.37	0.54	0.246
LYMP ($\times 10^9$/L)	2.18 ± 0.85	1.79 ± 0.37	26.74	0.00*
Platelet ($\times 10^9$/L)	241 ± 103	228.67 ± 54.80	1.17	0.095
CD4 (Cells/ul)	242 ± 228	807 ± 249	21.11	0.000*
Viral load (RNA copies/ml)	407282 ± 1040185	0.00	20.60	0.000*

*Statistically significant.

Table 3. Comparison of disease stages and haematological parameters of HIV-positive subjects.

	Stage 1	Stage 2	Stage 3	P-values
WBC	5.90 ± 1.69	6.10 ± 2.93	6.06 ± 7.02	0.987
HB	10.54 ± 1.67	9.92 ± 2.07	9.64 ± 9.85	0.800
NEUT	2.72 ± 0.61	2.90 ± 0.80	3.55 ± 0.91	0.000*
LYMPH	2.60 ± 0.57	2.58 ± 0.74	1.86 ± 0.86	0.000*
PLT	222.97 ± 92.67	233.95 ± 89.32	249.32 ± 112.06	0.367
VL	75535.14 ± 133407	256414.62 ± 447530	579031.34 ± 1341031	0.027

*Statistically significant.

Table 4. Spearman's correlation of the HIV VL of disease stages with haematological profiles and CD4 counts of HIV-positive subjects.

Parameters	Stage 1			Stage 2			Stage 3		
	Mean	Coefficient	P-value	Mean	Coefficient	P-value	Mean	coefficient	P-value
WBC ($\times 10^9$/L)	5.90 ± 1.69	−0.049	0.779	6.10 ± 2.93	0.354**	0.001	6.06 ± 7.02	0.067	0.438
HB (g/dL)	10.54 ± 1.67	−0.281	0.102	9.92 ± 2.07	−0.140	0.221	9.64 ± 9.85	0.023	0.789
NEUT. ($\times 10^9$/L)	2.72 ± 0.61	0.092	0.597	2.90 ± 0.80	−0.129	0.256	3.55 ± 0.91	0.116	0.178
LYMPH ($\times 10^9$/L)	2.60 ± 0.57	−0.126	0.469	2.58 ± 0.74	0.151	0.184	1.86 ± 0.86	−0.086	0.319
PLT ($\times 10^9$/L)	222.97 ± 92.67	0.067	0.701	233.95 ± 89.32	−0.073	0.522	249.32 ± 112.06	−0.002	0.983
CD4 (cells/ul)	≥500	−0.057	0.743	200−499	−0.407**	0.000	<200	−0.006	0.948

**. Correlation is significant at the 0.01 level (two-tailed). *. Correlation is significant at the 0.05 level (two-tailed).

Table 4 indicates Spearman's correlation of HIV VLs in the different disease stages with the haematological profiles and CD4 counts of HIV-positive subjects. No relationship was seen between disease Stage 1 (CD4 < 200 cells/µl) and their WBC (5.900 ± 1.69), Hb (10.54 ± 1.67), neutrophil counts (2.72 ± 0.61), lymphocyte counts (2.60 ± 0.57), and platelets (222.97 ± 92.67) with correlation coefficients of −0.049, −0.081, 0.092, −0.126 and 0.067, respectively ($p > 0.05$). Furthermore, there was no relationship between disease Stage 3 (CD4 ≥ 500 cells/µl) and their WBC (6.06 ± 7.02), Hb (9.64 ± 9.85), neutrophil counts (3.55 ± 0.91), lymphocyte counts (1.86 ± 0.86), and platelets (249.32 ± 112.06) with correlation coefficients of 0.067, 0.023, 0.116, −0.086 and −0.006, respectively ($p > 0.05$). However, there was a positive correlation between the total WBC counts (6.10 ± 2.93) and the VLs of the disease Stage 2 (CD4 200 - 499) with a coefficient value of 0.354 ($p < 0.05$). The VL of Stage 2 also correlated with the CD4 (200 - 499 cells/ul) with a coefficient value of −0.407 and p-value of 0.000.

4. Discussion

A high VL, decreased $CD4^+$ T-cells, anaemia, leukopenia (especially neutropenia), and thrombocytopenia were common findings among the HIV patients in the present study.

Furthermore, there was a female predominance among HIV-positive subjects indicating that the sex most affected by HIV infections is female, as previously reported by Anejo-Okopi *et al.* [11]. However, the results contradict studies by Sterling *et al.* [22] and Parinitha *et al.* [23] who presented a predominance of males. While the mechanism underpinning sex differences in HIV disease progression is unclear, the anatomy of the female reproductive system and other socio-cultural factors may more readily predispose women to HIV infections due to this demographic's higher levels of involvement in sex work. Consequently, gender-based strategies focusing on women are needed in our setting to promote the early identification and integration of women into care since this may potentially reduce HIV-associated mortality in the population. The age group of 31 -

40 years (102 (40.5%)) constituted the majority of HIV-infected test subjects, while the group above 50 years was the smallest with 32 subjects (12.7%). This supports a recent study by Anejo-Okopi *et al.* [11] that reported the median age of HIV patients in their work in Jos Nigeria as 34 years and research by Lima *et al.* [10] who reported it as 39 years (interquartile range as 34 - 46 years).

The results of this study indicated that plasma HIV-1 RNA levels were higher in men than women. This corroborates the work of Addo *et al.* [24] and Sterling *et al.* [22] who noted that these levels were lower in women than in men. Hormonal differences have been cited as possible factors contributing to sex-related differences in VL. Estrogen has been found to inhibit the production of tumour necrosis factor-alpha (TNF-α), which could result in the decreased viral replication found in women [25] [26]. The biological mechanism underlying the lower VLs in women (compared to men) that occur at higher CD4 T-cell counts may reflect a sex-related difference in the state of immune activation. Women generally experience more effective cell-mediated immunity than men (from soon after birth). Therefore, greater control of viral reproduction in the early stages of HIV/AIDS may be a manifestation of this nonspecific sex difference in immunity [27].

A $CD4^+$ lymphocyte count is essential for the assessment of immune status in HIV-infected persons as the pathogenesis of AIDS is largely attributed to a decrease in absolute $CD4^+$ cell counts. These cell counts are the criterion for categorising HIV-related clinical conditions by the Centre for Disease Control (CDC) classification system for HIV infection [23] [28]. A comparison of haematological profiles, VLs, and CD4 counts of HIV-positive subjects with their control counterparts revealed a direct relationship between HIV infection and WBC, haemoglobin, and CD4 counts. The low $CD4^+$ counts associated with HIV infections are a result of the fact that CD4 T lymphocyte cells are targeted by the virus to infect its host; consequently, as the disease progresses the number of cells decreases. These CD4 T-lymphocytes are also a component of TWBC and lymphocyte counts. The reduction in these $CD4^+$ cells during HIV infection may account for the reduction in TWBC and total lymphocyte counts. The decline in haemoglobin concentration could be a result of possible opportunistic infections, which lead to the destruction of red blood cells, thereby inducing anaemia. Thus, an HIV infection is strongly associated with leukopenia, lymphopenia, and anaemia. These findings agree with previous research by Nakagawa *et al.* [29] indicating that VL determines the ongoing rate of CD4 cell count depletion to a much greater degree than any previous measure and that the VL continues to rise gradually in ART-naïve individuals [29].

Utilising Spearman's correlation between VLs, haematological profiles, and CD4 counts of HIV-positive subjects, it was established that as the VL increases the values of CD4 count, haemoglobin concentration, and lymphocyte counts decreased. This implies that the neutrophil counts increased as the VLs increased because of possible opportunistic infections caused by suppressed CD4 count.

Consequently, neutrophil and lymphocyte counts in HIV subjects can be used when CD4 count cannot be obtained, especially in poor resource settings, as has been recommended by other researchers [2].

The haematological results obtained from this study further support the research of [30] and other authors who explain that anaemia is a riskfactor for early death in AIDS patients and that the causes of HIV-related anaemia are multifactorial [2] [31]. This might be due to generalised pancytopenia, which is usually caused by a chronic condition such as an HIV infection [32].

A comparison of haematological profiles, VLs, and $CD4^+$ counts of HIV-positive males and female subjects revealed higher TWBC, Hb, and VLs in men while their female counterparts presented with higher neutrophil, lymphocytes, platelets, and $CD4^+$ counts. Interestingly, whereas VL levels tended to be lower in females with higher $CD4^+$cell counts in this study, they remained relatively constant at different $CD4^+$ cell count levels in males, which suggests a higher rate of $CD4^+$ cell decline and a faster progression to AIDS in ART-naïve males compared to females with the same level of viraemia. This contradicts some earlier studies indicating that progression to AIDS is faster in women than in men at the same level of viraemia [33] [34].

Comparisons of disease stages and the haematological parameters of HIV-positive subjects revealed that at different, increasing stages of HIV infection, there is generally also an increase in TWBC. Nevertheless, the difference in TWBC between the three stages was not significant. Similarly, the same was noted in haemoglobin concentrations, which implies a general decrease among the three stages of the disease and reveals a possible opportunistic infection at any stage of the infection. The significant difference seen in the lymphocyte counts is a result of progressive destruction of $CD4^+$ T lymphocyte cells as the disease progresses from Stage 1 to 3. The significant difference seen in neutrophils might be due to most people in Stage 2 are susceptible to opportunistic infections, as suggested by other authors; furthermore, when compared with Stage 1, the difference is significant ($p < 0.05$) [35] [36].

5. Conclusions

This study identified an association between CD4 counts, VLs, age, haemoglobin levels, neutrophil count, and platelet count in HIV-infected subjects. While there was a female predominance in HIV infection and ART-naïve, HIV-1-infected patients, females had significantly lower VLs and higher $CD4^+$ cell counts compared to male participants at the same VL threshold. This suggests that males are more likely than females to present late for treatment in the chosen facility and, therefore, were at a higher risk of rapid progression to AIDS. Therefore, more research pertaining to gender-based strategies that focus on the early identification and integration of males into care are essential to mitigate the rapid progression of AIDS.

Since haematologic manifestations are common in HIV-infected patients, re-

levant individuals should be thoroughly examined and accurately treated for such abnormalities in order to reduce their morbidity. Further studies in African settings are required to characterise the pattern and magnitude of VL, haematological parameters, and CD4 counts that may be unique to males and females in this specific study area.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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