



**HAEMATOLOGICAL AND INFLAMMATORY BIOMARKER DYSREGULATION
AMONG PEOPLE LIVING WITH HIV BEFORE AND DURING ANTIRETROVIRAL
THERAPY: A COMPARATIVE STUDY AT SHANAHAN UNIVERSITY TEACHING
HOSPITAL, WATERSIDE, ONITSHA, NIGERIA**

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ABSTRACT

The introduction of antiretroviral therapy (ART) has transformed human immunodeficiency virus (HIV) infection from a life-threatening illness into a chronic manageable condition. However, prolonged survival has been accompanied by increasing recognition of non-AIDS comorbidities, including metabolic disorders, cardiovascular disease, obesity, dysglycaemia and chronic systemic inflammation. This study compared selected haematological and inflammatory biomarkers packed cell volume (PCV), total white blood cell (WBC) count, CD4+ T-cell count and C-reactive protein (CRP)—among HIV-negative controls, HIV-positive participants who had not commenced ART, and HIV-positive participants receiving ART at Shanahan University Teaching Hospital, Onitsha, Nigeria. A comparative hospital-based study involving 120 participants, comprising three groups of 40 participants each, was conducted. The study groups were: HIV-negative controls, HIV-positive participants not receiving ART, and HIV-positive participants receiving ART. PCV, WBC count, CD4+ T-cell count and CRP were determined using standard laboratory procedures. Differences among the three groups were evaluated using analysis of variance (ANOVA), with statistical significance set at $p < .05$. Mean PCV values were 0.44 ± 0.03 , 0.42 ± 0.02 and 0.44 ± 0.03 for controls, HIV-positive participants not receiving ART and HIV-positive participants receiving ART, respectively. Mean WBC counts were $6.53 \pm 0.48 \times 10^9/L$, $5.13 \pm 0.18 \times 10^9/L$ and $6.22 \pm 0.60 \times 10^9/L$, respectively. Mean CD4+ T-cell counts were $1,149.00 \pm 132.22$, 548.75 ± 145.61 and 644.75 ± 118.07 cells/ μL , respectively. Mean CRP concentrations were 1.10 ± 0.48 , 2.85 ± 6.53 and 3.41 ± 1.56 , respectively. Thus, compared with controls, HIV infection was characterized by a substantial reduction in CD4+ T-cell count and WBC count, together with increased CRP. ART-treated participants demonstrated partial recovery of WBC and CD4+ T-cell counts but retained elevated CRP concentrations. PCV showed little variation between groups. Conclusion The findings demonstrate persistent immunological and inflammatory dysregulation among HIV-positive individuals, including those receiving ART. The increase in CRP despite improvement in CD4+ T-cell and WBC measures suggests that restoration of immunological status with ART may not necessarily coincide with complete resolution of systemic inflammation. These findings are consistent with contemporary evidence that residual inflammation may persist during treated HIV infection and contribute to long-term cardiometabolic morbidity. However, the present biomarkers do not directly establish insulin resistance. Future studies at this centre should incorporate fasting glucose, fasting insulin, HOMA-IR, HbA1c, lipid profile, anthropometric indices and HIV viral load to directly characterize the relationship between insulin resistance, ART exposure and chronic inflammation.

KEYWORDS: HIV; antiretroviral therapy; CD4 count; C-reactive protein; white blood cells; packed cell volume; inflammation; metabolic dysregulation; insulin resistance; Nigeria.

1. INTRODUCTION

Human immunodeficiency virus (HIV) infection remains an important global public-health challenge, although the widespread availability of effective antiretroviral therapy (ART) has dramatically reduced HIV-associated morbidity and mortality. Contemporary HIV management emphasizes early and sustained ART, viral suppression and long-term management of comorbidities rather than treatment of HIV infection as an isolated infectious disease (World Health Organization [WHO], 2021; Gandhi *et al.*, 2025).

The epidemiological transition associated with successful ART has consequently changed the clinical profile of people living with HIV (PLWH). As life expectancy increases, metabolic and cardiovascular disorders have become increasingly important causes of morbidity. Current evidence identifies obesity, dyslipidaemia, diabetes, hypertension, metabolic syndrome and cardiovascular disease as important concerns in long-term HIV care (HIV Medicine Association [HIVMA]/Infectious Diseases Society of America [IDSA], 2024; National Institutes of Health [NIH], 2025).

The relationship between HIV and metabolic dysfunction is complex. HIV itself can induce persistent immune activation, systemic inflammation, oxidative stress, mitochondrial dysfunction and disturbances of adipose-tissue biology. ART may independently modify body weight and metabolic risk depending on the drug class, duration of exposure and characteristics of the individual. Contemporary integrase strand-transfer inhibitor (INSTI)-based regimens, for example, have been associated in several studies with weight gain, although the magnitude and clinical implications vary among populations and treatment regimens (Wood & Huhn, 2021; Milic *et al.*, 2022; Palella *et al.*, 2023; Valenzuela-Rodriguez *et al.*, 2023).

Importantly, the metabolic consequences of HIV extend beyond adiposity. Insulin resistance represents a key pathophysiological pathway linking visceral adiposity, inflammation, altered glucose metabolism and cardiovascular risk. In PLWH, insulin resistance may reflect the combined effects of chronic HIV-associated inflammation, altered adipokine signalling, mitochondrial dysfunction, changes in body composition, traditional metabolic risk factors and ART exposure. Recent literature continues to describe a close interaction between immune activation and metabolic pathways in treated HIV infection (Okine *et al.*, 2025; Yoshino *et al.*, 2026).

Evidence from Africa demonstrates that metabolic disease is increasingly relevant among PLWH. Peer *et al.* (2023), in a systematic review and meta-analysis of African studies, estimated diabetes and prediabetes prevalences of approximately 5.1% and 15.1%, respectively, although substantial heterogeneity existed

among studies. More recent African evidence indicates that metabolic syndrome occurs in approximately one-fifth or more of PLWH, depending on the diagnostic criteria employed (Peer *et al.*, 2023; Asgedom *et al.*, 2024).

This concern is particularly relevant in Onitsha, Nigeria, where increasing ART coverage has resulted in longer survival among PLWH and a corresponding need to address non-communicable diseases. Nigerian studies have documented substantial metabolic syndrome among HIV-positive populations. In a study from the University of Calabar Teaching Hospital, metabolic syndrome was more frequent among patients receiving ART than among ART-naïve participants, while a separate Nigerian study reported metabolic syndrome in 30.7% of PLWH compared with 22.8% of HIV-negative individuals (Ojong *et al.*, 2022; Jumare *et al.*, 2023).

In addition to metabolic disturbances, HIV produces characteristic immunological and haematological abnormalities. CD4⁺ T-cell depletion is a central feature of HIV pathogenesis, while alterations in circulating leukocyte populations can persist even during long-term ART. Van de Wijer *et al.* (2021) demonstrated persistent abnormalities in multiple circulating immune-cell populations among virologically suppressed PLWH, including alterations involving CD4⁺ T cells, CD8⁺ T cells, monocytes and other immune subsets. Similarly, African cohort data have demonstrated persistent immune activation among treated PLWH (Streeck *et al.*, 2022).

The haematological consequences of HIV are also clinically important. Anaemia is among the most common haematological abnormalities observed in PLWH, with a large systematic review estimating substantial prevalence among adults with HIV (Cao *et al.*, 2022). Changes in leukocyte profiles may reflect HIV disease activity, opportunistic infections, medication effects, bone-marrow suppression and immune recovery. Consequently, routine haematological indices remain useful adjuncts to conventional HIV monitoring, particularly in resource-constrained settings (Cao *et al.*, 2022).

Inflammation is another major component of the HIV disease process. Although ART substantially suppresses viral replication, immune activation and inflammation may persist. CRP is a widely available acute-phase inflammatory marker and has been investigated as an indicator of residual inflammatory burden and cardiometabolic risk. A 2024 systematic review and meta-analysis found that hs-CRP was frequently elevated among PLWH receiving ART, with particularly notable findings in African populations (Mabhida *et al.*, 2024).

Experimental and longitudinal studies further support the concept that HIV acquisition itself increases systemic inflammatory biomarkers and that early ART can

attenuate, but not necessarily eliminate, inflammatory activation (Kosmider et al., 2024). Chronic inflammation in treated HIV has been associated with cardiovascular, renal, hepatic and other non-AIDS complications (Lv et al., 2021; Yoshino et al., 2026).

The relationship between inflammation, immune restoration and metabolic disease is therefore clinically important. The persistence of inflammatory activity despite improved CD4⁺ T-cell counts raises the possibility that conventional measures of HIV treatment response may not fully capture the biological processes underlying long-term metabolic complications.

Against this background, the present study compared PCV, WBC count, CD4⁺ T-cell count and CRP among HIV-negative controls, HIV-positive individuals not receiving ART and HIV-positive individuals receiving ART at Shanahan University Teaching Hospital, Onitsha. The study was designed to characterize differences in haematological and inflammatory status across these clinically distinct groups and to provide locally relevant evidence for understanding HIV-associated biological dysregulation.

2. PROCEDURES

Venous blood collection was under aseptic conditions and the serum separated immediately.

Packed Cell Volume (PCV): PCV was determined by centrifuging anticoagulated blood in capillary tubes at high speed using a micro-hematocrit centrifuge [Turgeon, M.L. (2018)] using Haematospin 1400 Micro-Hematocrit Centrifuge manufactured by Hawksley and

Sons Ltd, 25 Marlborough Road, Lancing, West Sussex BN15 8TN, United Kingdom.

Total WBC count was measured using an automated hematology analyzer[Keohane, E.M. et al (2020)], Sysmex XP-300 Automated Hematology Analyzer manufactured by Sysmex Corporation. 1-5-1 Wakinoama-Kaigandori, Chuo-ku, Kobe 651-0073, Japan.

CD4 count was determined using flow cytometry method[Brown, M. et al (2019)] with BD FACSCount System machine manufactured by Becton, Dickinson and Company. 1 Becton Drive, Franklin Lakes, New Jersey 07417, United States.

Serum CRP was analyzed by immunoassay method (Pepys, M.B. et al 2003) using FIA machine produced by Finecare Fluorescence Immunoassay Analyzer (FIA) produced by Guangzhou Wondfo Biotect Co., Ltd, China.

RESULTS

CD4⁺ T-cell depletion and immune dysfunction

The present study demonstrates distinct differences in immunological, inflammatory and selected haematological biomarkers across HIV-negative controls, HIV-positive participants not receiving ART and HIV-positive participants receiving ART. The most striking findings were the marked reduction in CD4⁺ T-cell count among HIV-positive participants, reduction in WBC count particularly among those not receiving ART, and elevation of CRP in both HIV-positive groups.

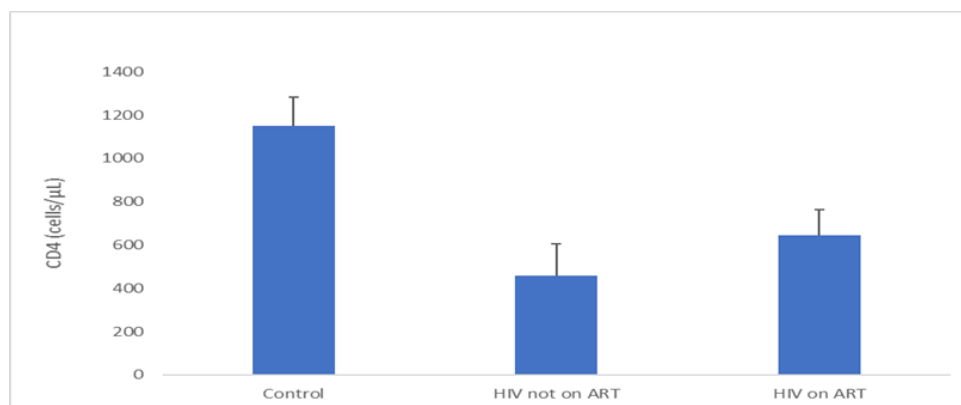


Figure 1: CD4.

White blood cell alterations

Mean WBC count decreased from $6.53 \times 10^9/L$ in controls to $5.13 \times 10^9/L$ among HIV-positive participants not receiving ART. The ART-treated group

demonstrated a higher mean WBC count of $6.22 \times 10^9/L$, approaching the control value.

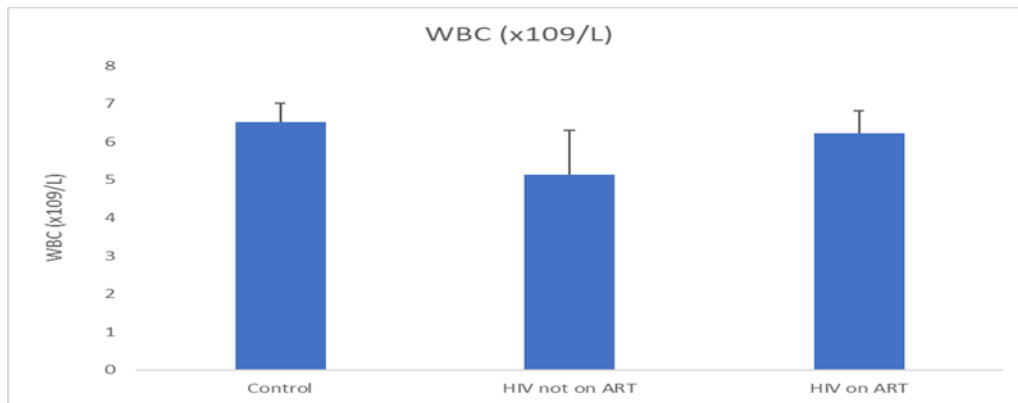


Figure 2: WBC.

Packed cell volume

PCV demonstrated comparatively little variation between groups, with mean values of 0.44 ± 0.03 , 0.42 ± 0.02 and

0.44 ± 0.03 among controls, untreated HIV-positive participants and ART-treated participants, respectively.

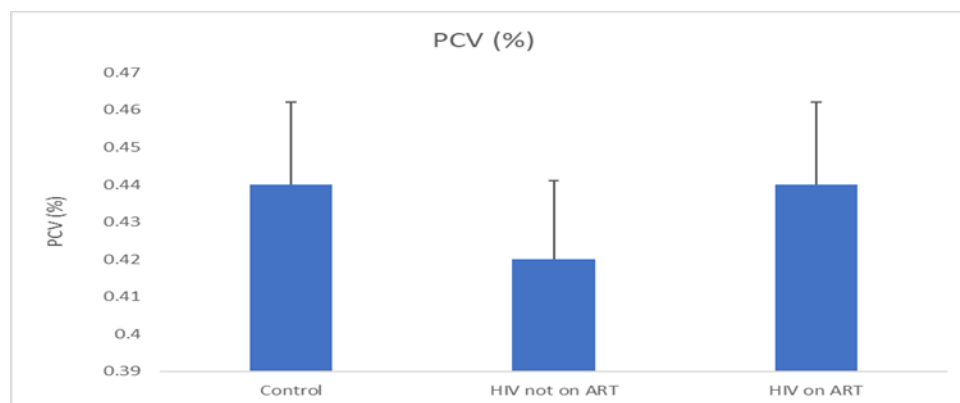


Figure 3: PCV.

Elevated CRP and persistent inflammation

The CRP findings are arguably the most important observation from the perspective of the proposed metabolic-dysregulation framework. Mean CRP

increased from 1.10 ± 0.48 in controls to 2.85 ± 6.53 among HIV-positive participants not receiving ART and 3.41 ± 1.56 among those receiving ART.

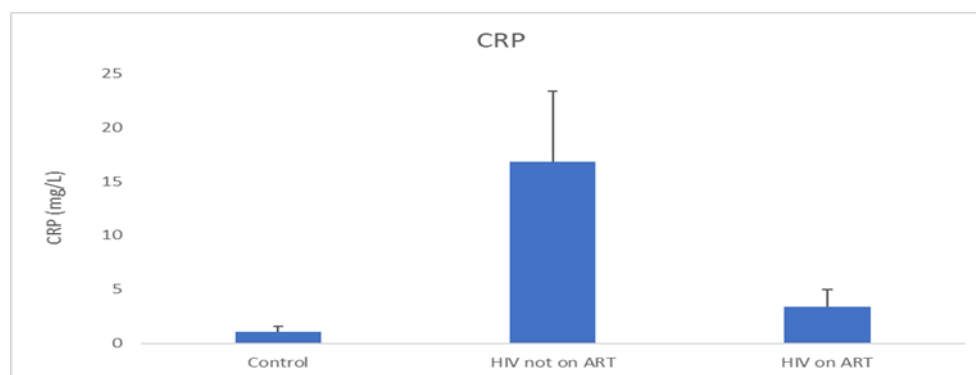


Figure 4: CRP.

Table 1: Haematological/Inflammatory Tests.

	PCV (%)	WBC (x109/L)	CD4 (cells/μL)	CRP (mg/L)
Control	0.44 ± 0.03	6.53 ± 0.48	1149.00 ± 132.22	1.10 ± 0.48
HIV not on ART	0.42 ± 0.03	5.13 ± 1.18	458.75 ± 145 .61	16.85 ± 6.53
HIV on ART	0.44 ± 0.03	6.22 ± 0.60	644.75 ± 118.10b	3.41 ± 1.56

Control (n=40) HIV, ART-naïve (n=40) HIV, ART-treated (n=40), p-value = ≤ 0.05

DISCUSSION

Figure 1 shows the mean CD4⁺ T-cell count was 1149.00 cells/ μ L among controls but decreased to 548.75 cells/ μ L among HIV-positive participants not receiving ART and 644.75 cells/ μ L among HIV-positive participants receiving ART. Relative to controls, these represent approximate reductions of 52.2% in the ART-naïve group and 43.9% in the ART-treated group.

The lower CD4⁺ count among HIV-positive participants is biologically consistent with the fundamental pathogenesis of HIV infection. HIV progressively disrupts CD4⁺ T-cell homeostasis through direct and indirect mechanisms, including viral cytopathic effects, immune-mediated destruction, chronic immune activation and altered lymphocyte turnover. Persistent immune activation can continue even after viral suppression (Lv et al., 2021; Streeck et al., 2022).

The higher mean CD4⁺ count in the ART-treated group compared with the untreated HIV-positive group is consistent with immune recovery following ART. However, the fact that the mean CD4⁺ count remained below that of the control group suggests incomplete immunological normalization. This is consistent with evidence that restoration of CD4⁺ T-cell numbers does not necessarily imply complete restoration of immune function or elimination of immune dysregulation (Van de Wijer et al., 2021; Gandhi et al., 2025).

An important implication is that CD4⁺ recovery and inflammatory recovery may occur along different biological trajectories. Contemporary HIV medicine increasingly recognizes that viral suppression and CD4⁺ restoration do not completely reverse the inflammatory consequences of HIV infection.

This pattern may suggest partial haematological recovery following ART. Leukopenia in untreated HIV may result from several mechanisms, including immune-cell destruction, impaired haematopoiesis, bone-marrow dysfunction, nutritional deficiencies, opportunistic infections and chronic inflammatory processes. ART-associated improvement may occur as HIV replication is controlled and immune function improves.

The observed findings are compatible with research demonstrating substantial alterations in circulating immune-cell populations in treated HIV. Van de Wijer et al. (2021) reported persistent dysregulation across both innate and adaptive immune-cell compartments despite stable ART, highlighting the complexity of interpreting total WBC count as a surrogate of immune status.

Thus, while the increase in WBC count from $5.13 \times 10^9/L$ to $6.22 \times 10^9/L$ following ART exposure is encouraging, total WBC count alone cannot distinguish beneficial immune reconstitution from alterations in specific leukocyte subsets.

This finding suggests that substantial PCV alteration was not a major feature of the study population. Nevertheless, this should not be interpreted as evidence that haematological abnormalities are absent in HIV. Anaemia remains a common complication of HIV globally and in African populations, with causes including chronic inflammation, nutritional deficiencies, opportunistic infections, renal disease and medication-related marrow suppression (Cao et al., 2022).

The relatively preserved PCV in the present study may reflect the characteristics of the study population, access to care, treatment status or exclusion of individuals with severe comorbid disease. It also reinforces the importance of assessing multiple haematological parameters rather than assuming that HIV-associated immune dysfunction necessarily manifests as reduced PCV.

Thus, CRP was approximately 159% higher in the ART-naïve HIV group and approximately 210% higher in the ART-treated HIV group relative to controls.

These findings are consistent with contemporary evidence demonstrating persistent systemic inflammation among PLWH, including those receiving ART. Mabhida et al. (2024), in a systematic review and meta-analysis of hs-CRP among PLWH receiving ART, found that most included studies reported increased hs-CRP compared with controls, with particularly prominent findings in African studies.

The finding of higher CRP in ART-treated participants deserves careful interpretation. It would be inappropriate to conclude that ART caused increased inflammation because this was a comparative observational analysis rather than a randomized longitudinal study. ART-treated individuals may have differed from ART-naïve participants in duration of HIV infection, disease history, age, immune status, ART regimen, comorbidities, nutritional status and other factors.

Moreover, ART itself is not expected simply to increase inflammatory burden in a uniform manner. Rather, current evidence indicates that although effective ART markedly reduces HIV replication, residual immune activation may persist because of mechanisms including microbial translocation, immune dysregulation, latent viral reservoirs, co-infections and altered metabolic pathways (Lv et al., 2021; Kosmider et al., 2024; Okine et al., 2025; Yoshino et al., 2026).

ART and contemporary metabolic risk

The literature regarding ART and metabolic outcomes is evolving. Some newer ART regimens, especially INSTI- and tenofovir alafenamide-containing regimens, have been associated with increased weight gain in certain populations. However, the relationship is heterogeneous and may be influenced by baseline nutritional status, sex, age, ART history, drug combinations and the

phenomenon of “return to health” after treatment initiation (Wood & Huhn, 2021; Palella et al., 2023; Valenzuela-Rodriguez et al., 2023). Milic et al. (2022) further demonstrated that weight gain and insulin resistance are related in people switching to INSTI-based therapy, although their findings emphasize the complexity of this relationship rather than a simple causal effect of INSTIs alone.

Recent systematic evidence similarly suggests that metabolic outcomes associated with ART have changed over time and that ART-treated populations may have higher odds of metabolic syndrome than ART-naïve populations, while also emphasizing substantial confounding and heterogeneity (Mahanani et al., 2026). The present study therefore fits within an increasingly important research question: whether successful control of HIV infection can coexist with persistent inflammatory and metabolic dysregulation.

3.6 Relevance to Nigeria and sub-Saharan Africa

The Nigerian context is particularly important because the burden of HIV intersects with increasing prevalence of obesity, hypertension, diabetes and other non-communicable diseases. Evidence from Nigeria has already demonstrated clinically important metabolic syndrome among PLWH. Ojong et al. (2022) reported substantially greater metabolic syndrome among ART-treated patients, while Jumare et al. (2023) found a higher prevalence of metabolic syndrome among PLWH than HIV-negative Nigerians.

At the continental level, systematic reviews have estimated a pooled metabolic syndrome prevalence exceeding 20% among PLWH in sub-Saharan Africa (Asgedom et al., 2024). More recent African analyses identify BMI, sex, alcohol use and other conventional metabolic factors as important determinants (David et al., 2025). The present findings therefore have potential relevance beyond HIV disease monitoring. They suggest that HIV clinics may provide an important platform for integrated screening for metabolic and inflammatory risk. The study also provides locally generated data from Onitsha, southeastern Nigeria, a setting for which additional evidence on the intersection of HIV, inflammation and metabolic health is valuable.

4. CONCLUSION

This comparative study of 120 participants demonstrates important differences in immunological, haematological and inflammatory biomarkers among HIV-negative individuals, HIV-positive individuals not receiving ART and HIV-positive individuals receiving ART.

The principal findings were marked CD4⁺ T-cell depletion in HIV-positive participants, reduced WBC count particularly among ART-naïve participants, relative preservation of PCV, and increased CRP among both HIV-positive groups. ART-treated participants showed evidence of partial immunological and

haematological recovery, reflected by higher CD4⁺ and WBC values than the ART-naïve group, but inflammatory activity remained elevated.

These findings support the concept that immunological recovery and inflammatory normalization are not necessarily synonymous. Persistent inflammation may represent an important biological link between treated HIV infection and the growing burden of non-AIDS cardiometabolic disease.

The clinical implication is not to reduce ART exposure but to move toward integrated HIV and non-communicable-disease care, with routine assessment of metabolic risk alongside virological and immunological monitoring. This approach is increasingly supported by contemporary HIV guidelines and African evidence concerning the emerging cardiometabolic burden of treated HIV.

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