Journal of Applied Health Sciences and Medicine, 6(9):9–14, September: 2026.
DOI: https://doi.org/10.58614/jahsm693

Journal of Applied Health Sciences and Medicine
ISSN (Online): 2583-1887

Journal homepage: https://jpub.org/journal-details.php?journal-id=39

Research Article

Evaluation of some selected Immunological and Oxidative Stress profiles of HIV Patients in Part of Southern Nigeria

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Article Info

Keywords: *Immunology, Oxidative Stress, HIV, HAART, Antioxidants.*

Received: *29.06.2026;*
Accepted: *10.09.2026;*
Published: *15.09.2026*

Abstract

Human Immunodeficiency Virus (HIV) infection is characterized by chronic immune activation, which plays a pivotal role in disease progression and the depletion of CD4+ T cells. This persistent activation is marked by an elevated state of immune cells, increased release of pro-inflammatory cytokines, and elevated oxidative stress. This study evaluated levels of Interleukin-10 (IL-10), Tumor Necrosis Factor-alpha (TNF-α), Malondialdehyde (MDA), Total Antioxidant Capacity (TAC), White Blood Cell count (WBC), Percentage Lymphocyte (LYM), and Packed Cell Volume (PCV) in HIV-infected individuals undergoing HAART. Participants were categorized into three groups: Stage I HIV patients (30), Stage II HIV patients (28), and HIV-negative controls (30). Serum levels of IL-10, TNF-α, MDA, TAC, WBC, LYM, and PCV were measured and statistically analyzed across these groups. Stage I patients exhibited significantly lower IL-10 levels compared to Stage II ($p=0.021$), while Stage II patients had higher IL-10 levels than controls ($p=0.001$). TNF-α levels were elevated in Stage II compared to both Stage I and controls ($p=0.000$), and higher in Stage I than controls ($p=0.02$). MDA levels were significantly higher in both HIV-positive groups compared to controls ($p<0.05$), indicating increased oxidative stress. TAC levels showed no significant differences across groups. WBC counts were higher in Stage I than Stage II ($p<0.05$) and lower in Stage II compared to controls ($p<0.05$). Percentage LYM were elevated in HIV-positive individuals relative to controls ($p<0.05$). No significant differences in PCV values were observed among the groups. Conclusively, the findings reveal distinct immunological and oxidative stress profiles in HIV-infected individuals on HAART, varying by disease stage. Elevated IL-10 and TNF-α levels in advanced stages suggest heightened inflammatory responses. Increased MDA levels highlight persistent oxidative stress despite therapy. These insights underscore the need for continuous monitoring and tailored therapeutic strategies to optimize patient outcomes.

How To Cite: S.O. Okparaku, K.O. Iyevhobu, G.O. Okparaku, K.O. Obohwemu, A.O. Eigbedion, I.V. Oni. Evaluation of some selected Immunological and Oxidative Stress profiles of HIV Patients in Part of Southern Nigeria. *Journal of Applied Health Sciences and Medicine*, 6(9):9–14, 2026. https://doi.org/10.58614/jahsm693

1. Introduction

Human Immunodeficiency Virus (HIV) infection is characterized by chronic immune activation, which plays a pivotal role in disease progression and the depletion of CD4+ T cells. This persistent activation is marked by an elevated state of immune cells and increased release of pro-inflammatory cytokines [1]. The underlying causes are multifaceted, including factors such as microbial translocation, co-infections, and the continuous presence of replicating virus. Alterations in cytokine levels can significantly influence the trajectory of HIV infection, either promoting or inhibiting viral replication [2]. Assessing these cytokine fluctuations offers valuable insights into the rate of viral replication and the status of immune system damage or recovery, especially during Highly Active Antiretroviral Therapy (HAART) [3].

Furthermore, HIV infection induces oxidative stress through the production of reactive oxygen species (ROS) [4, 5]. This overproduction can lead to cellular damage [6] and has been associated with increased HIV transcription via the activation of nuclear factor κB (NF-κB). The resulting imbalance between pro-oxidative and antioxidative processes exacerbates the oxidative condition in HIV-infected individuals. Studies have shown that oxidative stress serves as an additional predictor of mortality and inflammation, independent of traditional HIV-associated markers like CD4 cell count and viral load according to Bastard *et al*. [7].

Therefore, beyond monitoring CD4+ T cell counts, evaluating cytokine profile and oxidative stress markers may provide a more comprehensive understanding of infection progression, potential adverse treatment responses, and the likelihood of developing non-AIDS-related complications [3]. In this study, we shall determine the level of IL-10, TNF alpha, MDA, TAC, WBC, LYM and PCV in HIV Infected subjects on HAART. The result of this research will help to understand the health dynamics of HIV patients.

2. Methods

2.1. Research Design and Study Area

The research was a cross-sectional study to assess the level of IL-10, TNF-alpha, MDA, TAC, WBC, LYM and PCV in HIV Infection using seropositive patients visiting HIV clinic at Irrua Specialist Teaching Hospital and Central Hospital, Uromi Edo State. Information on the clinical state of the study participants were obtained from their medical records. This study was carried out in Esan Central and Esan North-East LGA in Edo State, Nigeria. This area is located between latitude $6°10'$ and $6°45'$ north of the equator and between longitudes $6°10'$ and $6°30'$ east of the Greenwich Meridian [8].

2.2. Study population

A total of 58 HIV sero-positive patients on HAART and 30 apparently health participants (control) were recruited for this study. The CDC staging method with CD4 count was used to classify the study participants; stage 1 (CD4 count $\geq$ 500 cells/µl), stage 2 (CD4 count 200 to 500 cells/µl) [9, 10]. There was limited number of stage III participants (CD4 count $\leq$ 200 cells/µl), hence were excluded from the study.

2.3. Sample Collection

About 10mls of venous blood were collected from each patient and dispensed into plain container (5ml) and EDTA container (5ml). The samples in plain container were centrifuged and serum obtained for the determination of cytokines (IL-10, TNF-α), MDA and Total anti-oxidant Capacity. The sera were separated and stored at $\leq$ -20°C until analyses. The EDTA samples were used for the estimation of some FBC parameters (WBC, LYM and PCV). These samples were analyzed at ONAMEC Research Laboratory Nnewi and Irrua Specialist Teaching Hospital HIV laboratory.

2.4. Sample Analysis

Estimation of Full Blood Count

The Full blood count (FBC) was estimated using an automated cell counting machine (SYSMEX XE-2100^{TM} haematological analyser) [11].
Principle: A blood sample is aspirated and measured, diluted to the specified ratio and stained. The sample is then fed into the flow cell. A semiconductor laser beam is emitted to the blood cells passing through the flow cell. The forward scattered light is received by the photodiode, and the lateral scattered light and lateral fluorescent light are received by the photomultiplier tube. This light is converted into electrical pulses, thus making it possible to obtain blood cell information.

Determination of MDA level

MDA level was determined by the colorimetric method of Kumar et al, [12].
Principle: Malondialdehyde (MDA) is a product of lipid peroxidation. When heated with 2-thiobarbituric acid (TBA) under alkaline condition, it forms a pink-coloured product, which has absorption maximum at 532 nm. The intensity of colour generated is directly proportional to the concentration of MDA in the sample.

Estimation of Total Antioxidant Capacity

Total antioxidant activity was estimated by Ferric Reducing Ability of Plasma (FRAP) method by Benzie and Devaki, [13].
Principle: At low pH, Antioxidant power causes the reduction of ferric tripyridyl triazine (Fe III TPTZ) complex to ferrous form (which has an intense blue colour) that can be monitored by measuring the change in absorption at 593nm. FRAP values are obtained by comparing the absorbance change at 593 nm in mixture (test), with those containing ferrous ion in known concentration (Standard).

Estimation of Human Interleukin 10 (IL-10)

Interleukin 10 (IL-10) was estimated using human Interleukin 10 (IL-10) ELISA Kit [14].

Principle: The kit employs double-antibody sandwich enzyme-linked immunosorbent one-step process. Standard, test sample and HRP-labeled Interleukin 10 (IL-10) antibodies are added to wells which are Pre-coated with Interleukin 10 (IL-10) antibody. After incubation and washing to remove the uncombined enzyme, Chromogen Solution A and B is added. The color of the liquid will change into blue. At the effect of acid, the color finally becomes yellow. The color change is measured spectrophotometrically at a wavelength of 450 nm. The concentration of Interleukin 10 (IL-10) in the samples is then determined by comparing the O.D. of the samples to the standard curve.

Estimation of Tumor Necrosis Factor α (TNF-α)

Tumor Necrosis Factor α (TNF-α) was estimated using human Tumor Necrosis Factor α (TNF-α) ELISA Kit [14].

Principle: The kit employs double-antibody sandwich enzyme-linked immunosorbent one-step process. Standard, test sample and HRP-labeled Tumor Necrosis Factor α (TNF-α) antibodies are added to wells which are Pre-coated with Tumor Necrosis Factor α (TNF-α) antibody. After incubation and washing to remove the uncombined enzyme, Chromogen Solution A and B is added. The color of the liquid changes into blue. At the effect of acid, the color finally becomes yellow. The color change is measured spectrophotometrically at a wavelength of 450 nm. The concentration of Tumor Necrosis Factor α (TNF-α) in the samples is then determined by comparing the O.D. of the samples to the standard curve.

2.5. Statistical Analysis

The results were presented as mean±standard deviation. The data obtained were analyzed using ANOVA. SPSS statistical package version 22 was used in data analysis. Significant levels were considered at $P<0.05$

3. Results

The present study determine the level of IL-10, TNF alpha, MDA, TAC, WBC, LYM and PCV in HIV Infected subjects on HAART

Table 1 shows the socio-demographic characteristics of the study population. As indicated in the table, the participants comprised of 58 HIV sero-positive and 30 HIV sero-negative (control) participants. The HIV participants were classified according to the Centre for Disease Control pattern, into stage I and II with 30 and 28 study participants respectively. The age range of the participants was 20 years and above, with majority between 41 to 60 years for the sero-positive groups and 20-40 years for the control group. Stage I had 7 (23.3%) male and 23 (76.7%) female, stage II had 6 (21.4%) male and 22 (78.6%) female, while control group had 10 (33.3%) male and 20 (66.7%) female.

Table 2 shows the level of IL-10, TNF-α , MDA, and TAC in various stages of HIV and control groups (Mean±SD). As indicated in the table, the mean values of IL-10 was significantly ($p = 0.021$) lower in stage I (105.13±9.16 pg/ml) when compared to stage II (113.48±3.89 pg/ml) group. Similarly, stage II subjects showed significantly ($p = 0.001$) higher mean IL-10 values when compared to the control group (99.93±8.28 pg/ml). There was no significant difference ($p = 0.282$) between the IL-10 values obtained in stage I and control groups. The table equally shows that the sero-positive groups had significantly ($p<0.05$) higher mean TNF-α values than the control group (14.57±1.30 pg/ml). Also stage II subjects (20.25±1.74 pg/ml) revealed significantly ($p<0.05$) higher mean TNF-α values than stage I subjects (17.04±1.90). Malondialdehyde (MDA) values between stages I (1.84±0.11 nmol/ml) and II (1.85±0.14 nmol/ml) showed no significant difference ($p = 1.000$). When compared to the control group, both stage I and II showed significantly ($p = 0.000$) higher MDA values than the control group (1.55±0.08 nmol/ml). The TAC values across the groups showed no significant difference ($p > 0.05$). However, while stage I showed the highest TAC values (895.87±128.49 µmol/l), stage II recorded the lowest values (806.35±150.86 µmol/l).

Table 3 shows the level of WBC, LYM, and PCV in the HIV sero-positive and control groups. Values are expressed as mean±SD. As indicated in the table, the mean WBC for stage II group (4.17±1.07 x109/l) was significantly ($p<0.05$) lower than that of stage I (5.55±1.38 x109/l) and control groups (6.14±1.59 x109/l). The LYM showed no significant difference between stages I (47.45±8.80%) and II (45.27±13.53%), but the sero-positive groups showed statistically significant ($p<0.05$) higher values than the control (33.30±6.65%) group. There was no significant ($p > 0.05$) difference across the groups for PCV. Stage II PCV however, showed the lowest value (34.64±3.50%) whereas the control group showed the highest values (38.30±2.50%).

Table 1: Socio-Demographic Characteristics of the Study Population

Variables		**CDC Stage I n(%)**	**CDC Stage II n(%)**	**Control n(%)**
Age (years)	20-40	13(43.3)	7(25.0)	24(80)
	41-60	16(53.3)	17(60.7)	6(20)
	≥61	1(3.3)	4(14.3)	0(0)
	Total	**30(100)**	**28(100)**	**30(100)**
Gender	Male	7(23.3)	6(21.4)	10(33.3)
	Female	23(76.7)	22(78.6)	20(66.7)
	Total	**30(100)**	**28(100)**	**30(100)**

Keys: CDC = Centre for Disease Control; n = Number of subjects

Table 2: IL-10, TNF-α, MDA and TAC values of the study groups (mean±SD)

Group(n)	IL-10 (pg/ml)	TNF-α (pg/ml)	MDA (nmol/ml)	TAC (µmol/l)
Stage I(30) (A)	105.13±9.16	17.04±1.90	1.84±0.11	895.87±128.49
Stage II(28) (B)	113.48±3.89	20.25±1.74	1.85±0.14	806.35±150.86
Control(30) (C)	99.93±8.28	14.57±1.30	1.55±0.08	873.83±77.89
f- value	7.902	28.201	26.934	1.884
p-value	0.001*	<0.0001*	<0.0001*	0.165
A vs B	0.021*	<0.0001*	1.000	0.181
A vs C	0.282	0.002*	<0.0001*	1.000
B vs C	0.001*	<0.0001*	<0.0001*	0.676

Keys: MDA= Malondialdehyde; TAC= Total anti-oxidant capacity; IL-10= Interleukin 10; TNF- α = Tumor necrosis factor- α; SD=Standard Deviation; Significant level (P<0.05); n = Number of subjects; * = Significant values.

Table 3: WBC, LYM and PCV values of the study population (mean±SD)

Group(n)	WBC (x10^9/l)	LYM (%)	PCV(%)
Stage I(30) (A)	5.55±1.38	47.45±8.80	36.55±5.35
Stage II(28) (B)	4.17±1.07	45.27±13.53	34.64±3.50
Control(30) (C)	6.14±1.59	33.30±6.65	38.30±2.50
f-value	6.028	7.358	1.810
p-value	0.005*	0.002*	0.177
A vs B	0.028*	1.000	0.746
A vs C	0.776	0.002*	0.911
B vs C	0.006*	0.024*	0.195

Key: WBC= Total white blood cells count; PCV= Packed cell volume; LYM = % Lymphocytes count; SD = Standard Deviation; Significant level (P<0.05); n = Number of subjects; * = Significant values

4. Discussion

In this study, we determined the level of IL-10, TNF alpha, MDA, TAC, WBC, LYM and PCV in HIV Infected subjects on HAART. Data from the mean IL-10 values revealed that stage I had considerable (p=0.021) lower mean values than stage II. Furthermore, comparing stage II to the control group, the value was considerably (p=0.001) higher in stage II. This could be the consequence of either a negative feedback reaction from greater pro-inflammatory cytokine release and subsequent inflammation due to increased disease severity observed in stage II compared to stage I or it could be the result of a favorable response to HAART in stage I. The mean values of IL-10 for stage I and control groups did not, however, differ significantly (p=0.282). According to Osuji *et al.* [3], HAART has been observed to cause a noteworthy and steady rise in the count of CD4+ T cells, a reduction in viral load, and levels of IL-4, IL-6, and IL-10 that are comparable to those of people who are HIV negative.

According to the TNF-α statistical data, the mean values of stage II were significantly (p=0.000) higher than those of stage I and the control group. Stage II patients may have higher levels of pro-inflammatory cytokines as a result of a more severe illness and ensuing rise in inflammation. The mean TNF-α values of stage I group was significantly (p=0.02) higher than the control group. When a person has HIV infection, the infected cells and immune cells produce cytokines. According to Balaji *et al.* [15] these cytokines influence viral replication and modulate immune response. Via their ability to either stimulate or inhibit HIV replication, cytokine changes in HIV-positive persons might influence immune system performance and potentially have a direct effect on the course of the disease [2]. The findings from this study are consistent with the conclusions of earlier research, which maintained that the majority of cytokines have been used to assess patients' responses to antiretroviral therapy and as indicators of how the disease is progressing [3].

Comparing the mean MDA values across the three groups, it was found that stage I values did not differ substantially from stage II (p=1.000). The sero-positive groups revealed substantially (p<0.05) higher mean MDA values than the control group. This result emphasizes how oxidative stress is brought on by HIV infection. Through the envelope glycoprotein gp120 and the regulatory protein Tat, HIV infection triggers the production of reactive oxygen species (ROS) [16]. A statistical comparison of the three groups' mean TAC values revealed no significant ($p > 0.05$) differences in the mean values across the groups.

Result from the mean total WBC counts for the three groups revealed that the value for stage I was considerably (p<0.05) higher than the mean value for stage II group. Stage II WBC counts was significantly (p<0.05) lower than the control group. This result disagrees with previous finding by Okparaku et al, [17], who showed that total WBC count for HIV participants were significantly (p=0.01) higher than those of their sero-negative counterparts. The sample size and study methodology may have an impact on the discrepancy. Notwithstanding, data from the present study aligns with the detrimental effects of HIV infection on CD4 T cells [18] and the progressive decline in the immune system's capacity to produce new T cells [19]. Following evaluation of the groups' mean relative lymphocyte percentage, the findings revealed that stage I values did not differ substantially ($p > 0.05$) from stage II values. However, the sero-positive group mean values were significantly (p<0.05) higher than those of the control group. This may be linked to the increased activation and proliferation of lymphocytes seen in HIV infection, as it is consistent with previous reports on increased total lymphocyte counts in HIV positive subjects when compared to HIV negative individuals [1, 17, 20]. The packed cell volume result revealed that there was non-significant ($p > 0.05$) difference in mean values across the three groups. The current observations disagree with those of Okparaku *et al.* [17], who found that, in comparison to sero-negative controls (38.83±2.61%), the PCV of HIV subjects decreased significantly (P=0.000) to 29.39±6.34%. The non-significant outcome of the present study, however, might be the result of changes in treatment and management policies for HIV individuals.

5. Conclusion

The findings from this study enhance our understanding of the immunological and oxidative changes occurring in HIV-infected individuals undergoing HAART. The observed alterations in cytokine levels and oxidative stress markers underscore the complex interplay between HIV infection, antiretroviral therapy, and the body's immune and antioxidant systems. These results highlight the importance of continuous monitoring of these biomarkers to inform and tailor therapeutic strategies effectively. Implementing individualized treatment approaches that consider these immunological and oxidative parameters may improve patient outcomes and reduce the risk of therapy-related complications. Further research is required to explore the long-term implications of these changes and to develop interventions that can mitigate oxidative stress, adverse inflammation and support immune function in this patient population.

Article Information

Funding: No external funding was received for this study.

Author Contributions: S.O. Okparaku: Conceptualization, Methodology, Data curation, Supervision; G.O. Okparaku: Methodology, Data curation; K.O. Iyevhobu: Formal analysis, Writing – original draft; K.O. Obohwemu: Formal analysis; A.O. Eigbedion: Writing – review & editing; I.V. Oni: Writing – review & editing.

Ethical Approval: The study was approved by the Edo State Ministry of Health with approval number: ED/HREC/2020/1432.

Informed Consent: Written informed consent was obtained from all participants.

Conflict of Interest: The authors declare no conflict of interest.

Data Availability: Data available on reasonable request.

Disclaimer (Artificial Intelligence): The author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.), and text-to-image generators have been used during writing or editing of manuscripts.

Acknowledgements: The authors would like to acknowledge the management of Irrua Specialist Teaching Hospital (ISTH), Irrua, Edo State, Nigeria, Central Hospital, Uromi Edo State, Nigeria, and Suncare Medical Diagnostics, Uromi, Edo State, Nigeria for creating the enabling environment for this study. Thanks to all the Director and technical staff of St Kenny Diagnostic and Research Consult, Ekpoma, Edo State, Nigeria for their excellent assistance and for providing medical writing support/editorial support in accordance with Good Publication Practice (GPP3) guidelines.

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