



Insulin Resistance and Renal Function Tests Alterations in People Living With HIV on Antiretroviral Therapy at Shanahan University Teaching Hospital, Onitsha, Nigeria

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DOI: <https://doi.org/10.68050/JAMS.2026.569>

Received: 07 Sept 2026; Accepted: 14 Sept 2026; Published: 29 September 2026

ABSTRACT

The increasing survival of people living with HIV (PLWH) following widespread access to antiretroviral therapy (ART) has shifted clinical attention toward long-term metabolic and renal comorbidities. Insulin resistance and impaired renal function may occur through a complex interaction involving persistent immune activation, HIV-associated metabolic abnormalities, traditional cardiometabolic risk factors and adverse effects of selected antiretroviral agents. This study assessed insulin resistance and renal function test alterations among people living with HIV receiving ART at Shanahan University Teaching Hospital, Onitsha, Nigeria. A comparative study involving 120 participants divided equally into three groups of 40 was undertaken: HIV-negative controls, HIV-positive participants not receiving ART, and HIV-positive participants receiving ART. Glycaemic and insulin-resistance indices included fasting glucose, glycated haemoglobin (HbA1c), fasting insulin, homeostatic model assessment of insulin resistance (HOMA-IR) and quantitative insulin sensitivity check index (QUICKI). Renal indices included serum urea, creatinine, sodium, potassium, bicarbonate and chloride. Group means were compared using analysis of variance (ANOVA), with statistical significance set at $p < 0.05$. Marked differences in metabolic indices were observed across the three groups. Fasting glucose was 4.59 ± 0.29 mmol/L in controls, 5.61 ± 0.55 mmol/L in HIV-positive participants not on ART and 5.27 ± 0.42 mmol/L in those receiving ART. Corresponding fasting insulin concentrations were 6.21 ± 1.05 , 13.33 ± 5.86 and 8.68 ± 2.40 μ IU/mL, while HOMA-IR values were 1.27 ± 0.29 , 3.49 ± 1.82 and 2.22 ± 1.23 , respectively. QUICKI demonstrated the opposite pattern, with values of 0.37 ± 0.01 , 0.34 ± 0.04 and 0.39 ± 0.06 , respectively. Renal assessment showed increased serum creatinine among both HIV groups compared with controls: 82.70 ± 11.33 μ mol/L in controls, 92.58 ± 10.89 μ mol/L in HIV-positive participants not on ART and 90.18 ± 10.42 μ mol/L in participants on ART. Urea was highest among participants not on ART (5.72 ± 1.02 mmol/L) and declined to 4.81 ± 0.81 mmol/L among those receiving ART, compared with 4.59 ± 0.56 mmol/L in controls. The findings demonstrate significant metabolic and biochemical differences between HIV-positive and HIV-negative participants, particularly in insulin sensitivity and serum creatinine. HIV-positive participants not receiving ART demonstrated the greatest degree of insulin resistance, whereas ART-treated participants showed partial improvement in insulin-resistance indices but retained evidence of altered renal biochemical parameters. The findings support integration of metabolic and renal surveillance into routine HIV care in Nigeria.

Keywords: HIV; antiretroviral therapy; insulin resistance; HOMA-IR; QUICKI; fasting insulin; renal function; creatinine; urea; Nigeria.

INTRODUCTION

The introduction of effective antiretroviral therapy (ART) has fundamentally changed the natural history of



human immunodeficiency virus (HIV) infection. People living with HIV now survive substantially longer, and HIV infection is increasingly managed as a chronic condition rather than an immediately life-threatening disease. By the end of 2025, approximately 41 million people were estimated to be living with HIV globally, with 78% receiving ART. The WHO African Region continues to carry the greatest burden, accounting for more than two-thirds of people living with HIV worldwide (World Health Organization [WHO], 2026).

The success of ART has consequently exposed a new dimension of HIV medicine: the prevention and management of chronic non-AIDS comorbidities. Diabetes, obesity, hypertension, dyslipidaemia, cardiovascular disease and chronic kidney disease (CKD) have become increasingly important determinants of long-term morbidity among people living with HIV. The contemporary metabolic burden of HIV is multifactorial, reflecting the interaction between HIV-related inflammation, ageing, lifestyle factors, restoration of health following ART initiation and the metabolic effects of individual antiretroviral agents (Mahanani et al., 2026; Wearne et al., 2023; WHO, 2025a).

Insulin resistance represents an important early abnormality in the development of type 2 diabetes mellitus. It is characterized by diminished biological responsiveness to insulin, resulting in compensatory hyperinsulinaemia before overt hyperglycaemia becomes established. Consequently, fasting insulin and calculated indices such as HOMA-IR may identify metabolic disturbances that are not readily apparent from fasting glucose or HbA1c alone. Conversely, QUICKI is an insulin-sensitivity index that generally changes inversely to HOMA-IR. These complementary measures are therefore useful for characterizing early abnormalities in glucose metabolism.

The relevance of insulin resistance to HIV care is increasingly recognized. Persistent immune activation and inflammation can influence adipose-tissue function, glucose transport and insulin signalling. In addition, changes in body composition and fat distribution may accompany HIV infection and ART. Older antiretroviral drugs, particularly thymidine analogues and didanosine, have been associated with insulin resistance and lipodystrophy, while contemporary ART regimens may influence metabolic risk through weight gain and changes in body composition (Høgh et al., 2022; Diggins et al., 2022; Mahanani et al., 2026).

Evidence from Africa indicates that dysglycaemia is already an important health problem among PLWH. A systematic review and meta-analysis involving more than 86,000 African participants estimated diabetes and prediabetes prevalence at approximately 5.1% and 15.1%, respectively. Although the analysis did not demonstrate a statistically significant difference according to ART status, the authors emphasized the considerable burden of dysglycaemia and the likelihood of future diabetes resulting from the substantial prevalence of prediabetes (Peer et al., 2023).

Evidence from Nigeria is similarly concerning. Jumare et al. (2023), in a Nigerian cohort, reported metabolic syndrome in approximately 30.7% of people living with HIV compared with 22.8% of HIV-negative participants. Prediabetes, diabetes and low HDL cholesterol were particularly prominent among PLWH. Another Nigerian study found higher metabolic-syndrome prevalence among HIV patients receiving ART and reported significant differences in fasting plasma glucose and other cardiometabolic variables between ART-treated and ART-naïve participants (Jumare et al., 2023; Ojong et al., 2022).

Recent evidence also suggests that the relationship between ART and diabetes is more complex than the assumption that all ART necessarily increases insulin resistance. A 2023 systematic review and meta-analysis of integrase strand transfer inhibitors (INSTIs) found heterogeneous effects on diabetes and insulin resistance, with overall risk patterns varying according to comparator regimen and population. Importantly, African populations demonstrated a distinct risk pattern, emphasizing the need for context-specific evidence rather than extrapolation from predominantly high-income settings (Mulindwa et al., 2023).

More recent longitudinal evidence reinforces the importance of metabolic surveillance. In Ghana, Larrey et al. (2024) reported substantial incidence of type 2 diabetes among PLWH initiating dolutegravir-based ART, while a prospective Ghanaian study demonstrated the emergence of metabolic syndrome among people receiving contemporary ART (Larrey et al., 2024; Torpey et al., 2025). At the same time, a 2026 meta-analysis



of prospective cohorts reported that diabetes incidence among PLWH receiving ART was higher than among those not receiving ART, although the authors emphasized variation by region and diagnostic definitions (Li et al., 2026).

Renal disease represents another major chronic complication of HIV. HIV-associated nephropathy, immune-complex kidney disease, acute kidney injury, CKD and drug-associated nephrotoxicity may all occur in people living with HIV. The renal consequences of HIV are particularly important in populations of African ancestry because genetic susceptibility, including APOL1-associated risk, can modify vulnerability to HIV-related kidney disease (Lucas & Wyatt, 2022; Olabisi & Freedman, 2021).

The widespread use of ART has reduced the incidence of classic HIV-associated nephropathy but has not eliminated renal disease. Indeed, some antiretroviral agents may directly or indirectly affect kidney function. Tenofovir disoproxil fumarate (TDF), for example, can produce proximal tubular toxicity and reductions in renal function in susceptible individuals. More recent evidence suggests that tenofovir alafenamide (TAF) produces lower circulating tenofovir exposure and has a more favourable renal safety profile than TDF (Zhao & Lu, 2024).

The changing renal epidemiology of HIV is particularly relevant in sub-Saharan Africa. A 2024 Ghanaian study found that people living with HIV experienced progressive renal-function decline and that exposure to TDF was associated with a greater annual decline in eGFR than some comparator NRTIs (Chadwick et al., 2024). Similarly, a prospective Tanzanian cohort demonstrated a higher incidence of CKD and more rapid eGFR decline among PLWH receiving TDF-based ART compared with HIV-uninfected controls (Roberts et al., 2025).

Nigerian evidence also demonstrates the importance of renal surveillance. Nwatarali et al. (2023) reported kidney dysfunction among HIV-positive patients receiving HAART at a Nigerian tertiary hospital, while studies in northern Nigeria have demonstrated associations between APOL1 risk variants, proteinuric CKD and HIV-related renal disease (Nwatarali et al., 2023; Wudil et al., 2021).

Current international guidance emphasizes systematic assessment of kidney function using estimated glomerular filtration rate (eGFR) together with urinary albumin assessment rather than relying on serum creatinine alone. The KDIGO 2024 guideline recommends creatinine-based eGFR assessment in adults at risk of CKD and confirmation of abnormal findings through repeat testing and complementary markers where appropriate (KDIGO CKD Work Group, 2024).

An additional consideration is the relationship between metabolic and renal abnormalities. Insulin resistance and diabetes can promote kidney injury through glomerular hyperfiltration, endothelial dysfunction, oxidative stress, inflammation and progressive microvascular damage. Conversely, renal dysfunction may alter insulin metabolism and contribute to abnormalities of glucose homeostasis. Thus, metabolic and renal complications in PLWH should not be regarded as isolated phenomena but as potentially interconnected components of long-term cardiometabolic risk (Wearne et al., 2023; American Diabetes Association Professional Practice Committee, 2025).

Despite the increasing importance of these complications, there remains limited locally generated evidence simultaneously evaluating insulin resistance and renal biochemical alterations among HIV-positive populations in southeastern Nigeria. The present study therefore examined fasting glucose, HbA1c, fasting insulin, HOMA-IR, QUICKI and selected renal-function/electrolyte parameters among HIV-positive participants receiving ART, HIV-positive participants not receiving ART and HIV-negative controls at Shanahan University Teaching Hospital, Onitsha.

EXPERIMENTAL DESIGN

Subjects and Samples

A formal approval for this thesis was obtained from the Ethical committee of the Shanahan University



Teaching Hospital,(Holy Rosary Specialist Hospital & Maternity, Water-Side) SUTH, Onitsha. Informed consent was obtained from the patients prior to their inclusion in this study. Three categories of subjects were used in this PROSPECTIVE CONHORT study: The first category was made up of forty (40) adults confirmed HIV-positive patients (males and females) who have been on standard ART for at least six (6) months at SUTH, Onitsha. The second category was made up of forty (40) adults confirmed HIV-positive patients (males and females) at SUTH, Onitsha who were NOT on ART. The third category was the control subjects made up of apparently healthy, forty (40)adults confirmed HIV-Negative (males and females)volunteers, from hospital workers, students and traders within Onitsha city.

Inclusion criteria: These included the HIV-positive adults aged 18 years and above who have been on ART for at least 6 months.

Exclusion criteria: Pregnant women, patients on steroids or lipid-altering drugs; patients with pre-existing severe liver or kidney disease, or Concurrent malignancy were excluded.

MATERIALS AND METHODS

Venous blood collection was under aseptic conditions and the serum separated immediately. The separated serum samples were analyzed using an Automated biochemical analyzer- MINDRAY produced by Mindray Bio-Medical Electronics Co., Ltd, China; and also FINECARE FLUORESCENCE IMMUNOASSAY ANALYZER (FIA) produced by Guangzhou Wondfo Biotect Co., Ltd, China.

Serum Glucose test was done by Glucose-Oxidase Method [Burtisn C.A. et al (2012)] with the product kit from Randox Laboratories Ltd, UK.

Glycated Hemoglobin (HbA1c) test was done by Immunoassay Method [(Oguz, E.F. et al (2022)], using the product from Guangzhou Wondfo Biotect Co., Ltd, China.

Serum Insulin was estimated using Chemiluminenscent Immunoassay (CLIA) method [Rosli, N. et al (2022)].

HORMA-IR and QUICKIwere calculated .(Willig & overton, 2016)

For BUN (Blood Urea Nitrogen)-the Methods of Analysis was Enzymatic Urease-based [Kaplan, L.A. et al (2003)] with the reagent kit produced by Biosystems S.A. Costa Brava, Barcelona, Spain.

Serum Creatinine analysis was carried out using Jaffe Method (Colorimetric)[Tietz, N.W. (2018), with kit produced by Randox Laboratories Ltd, UK.

Serum Electrolytes analysis were done using the Methods of Ion Selective Electrode (ISE) according to Burtis, C.A.et al(2019) using the kit produced by Labio Scientific co.,India.

STATISTICAL ANALYSIS

Data obtained from the experiments were analyzed using the statistical package for social sciences software for windows version 27 (SPSS 27, Inc, Chicago, Illinois, USA. Descriptive statistics were used to summarize the glycaemic/insulin resistance indices and the renal function parameters of the study participants. The results were expressed as Mean $\pm$ Standard Deviation (SD).

Independent t-test comparison was done using ANOVA-ONE WAY Tests to determine if a significance difference exists between the mean of the HIV-positive patients on ART and the HIV-positive patients not on ART, and also that of the control subjects. The level of significance was set at $p < 0.05$.

RESULTS

The three-group comparison revealed a consistent metabolic pattern.

1. Glycaemic / Insulin Resistance Indices

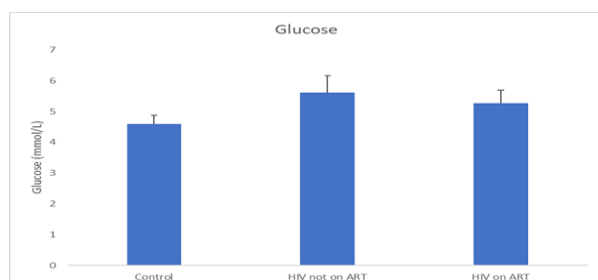


Figure 1: Glucose

There was a significant increase ($p < 0.05$) in the fasting blood glucose of the HIV patients on ART and HIV patients not on ART compared to the Control (figure 1).

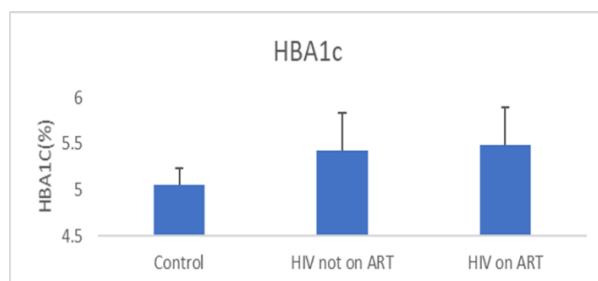


Figure 2: HBA1c: There was a significant increase ($p < 0.05$) in the HBA1c of the HIV patients on ART and HIV patients not on ART compared to the Control .

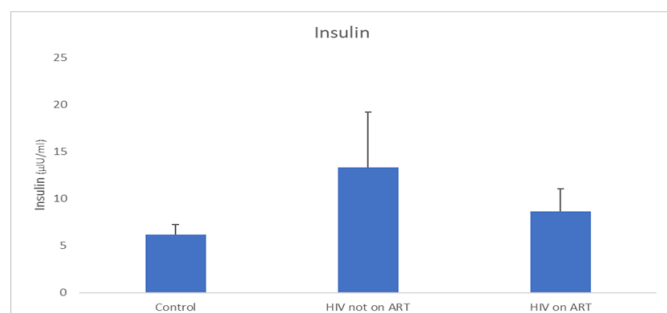


Figure 3: Insulin: There was a significant increase ($p < 0.05$) in the insulin level of the HIV patients not on ART and HIV patients on ART compared to the Control

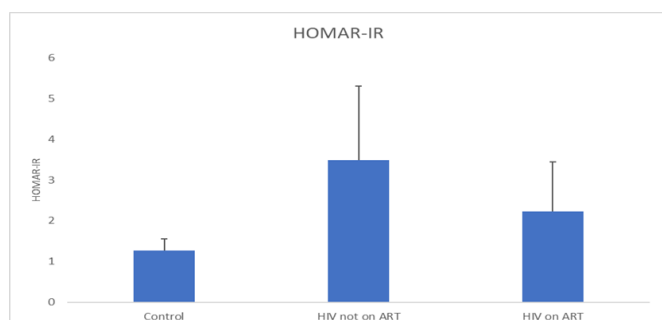


Figure 4: Homar-IR: There was a significant increase ($p < 0.05$) in the HOMAR-IR level of the HIV patients not on ART and HIV patients on ART compared to the Control

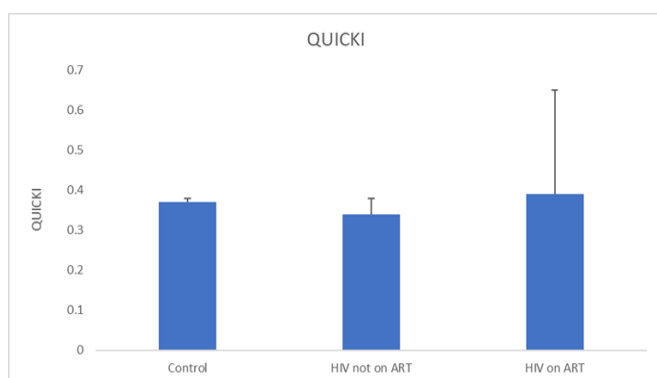


Figure 5: QUICKI: There was no significant difference ($p < 0.05$) in the QUICKI level of the HIV patients not on ART and HIV patients on ART compared to the Control

Table 1: Glycaemic/Insulin Resistance Indices

Group	Glucose (mmol/L)	HbA1c (%)	Insulin (μ IU/mL)	HOMA-IR	QUICKI
Control	4.59 $\pm$ 0.29	5.05 $\pm$ 0.18	6.21 $\pm$ 1.05	1.27 $\pm$ 0.29	0.37 $\pm$ 0.01
HIV not on ART	5.61 $\pm$ 0.55	5.43 $\pm$ 0.40	13.33 $\pm$ 5.80	3.49 $\pm$ 1.82	0.34 $\pm$ 0.04
HIV on ART	5.27 $\pm$ 0.42	5.49 $\pm$ 0.40	8.68 $\pm$ 2.40	2.22 $\pm$ 1.23	0.39 $\pm$ 0.26

2. Renal Function Tests

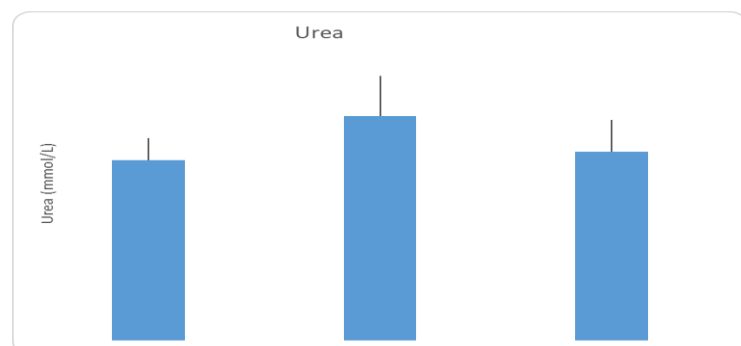


Figure 6: UREA

There was a significant increase ($p < 0.05$) in the urea level of the HIV patients not on ART compared to the HIV patients on ART and Control (figure 6).

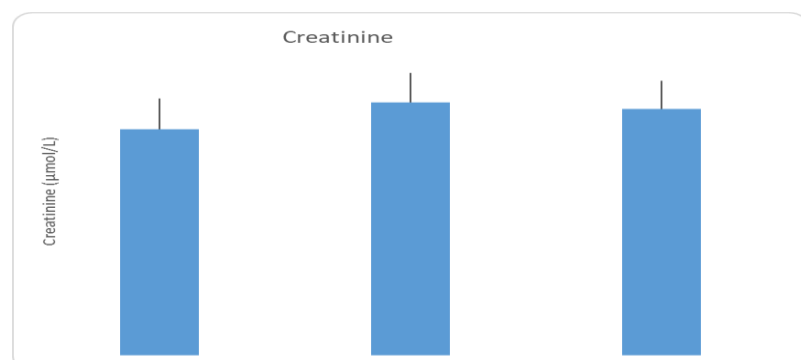


Figure 7: CREATINE

There was a significant increase ($p < 0.05$) in the creatinine level of the HIV patients not on ART and the HIV patients on ART compared to the Control (figure 7).

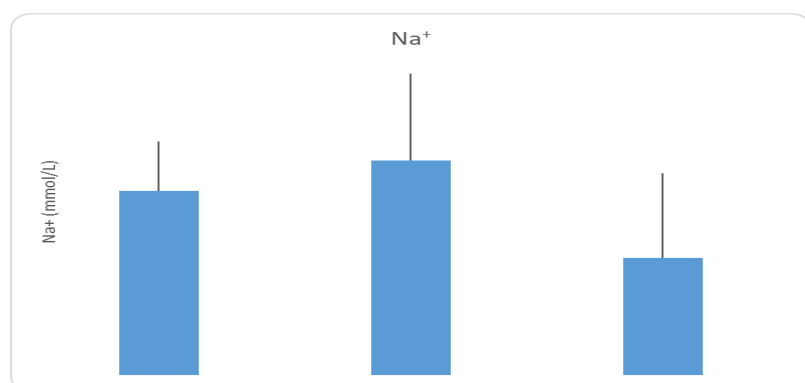


Figure 8: Na⁺

There was a significant decrease ($p < 0.05$) in the sodium ion level of the HIV patients on ART compared to the HIV patients not on ART and Control (figure 8).

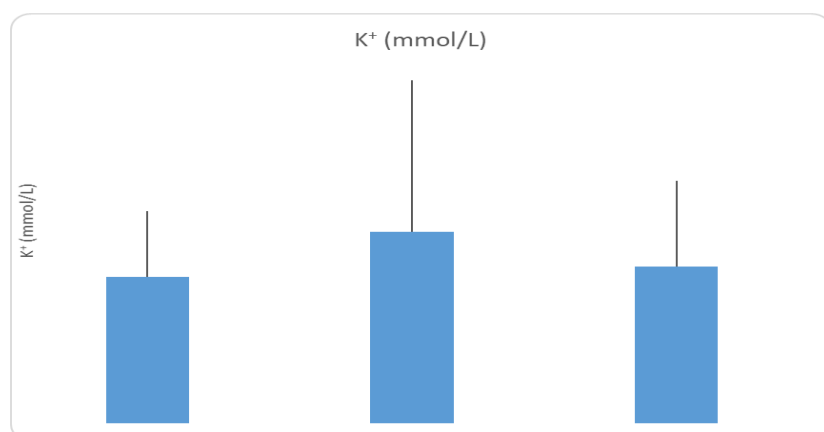


Figure 9: K⁺

There was no significant difference ($p > 0.05$) in the potassium ion level of the groups compared (figure 9).

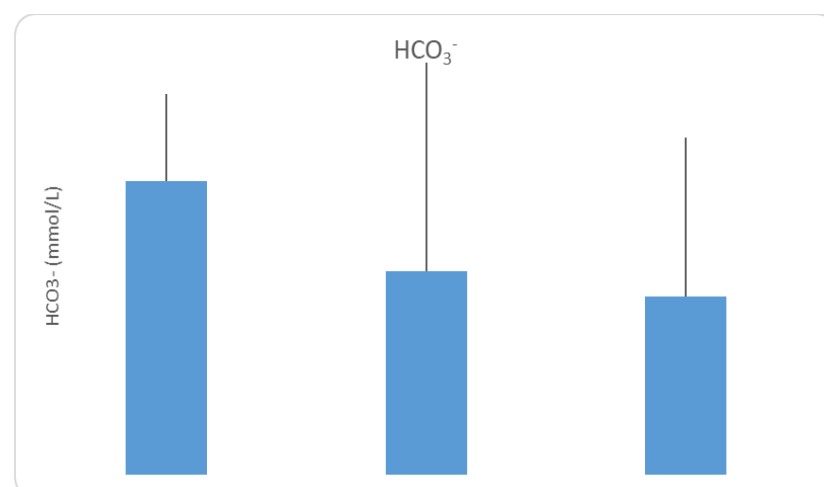


Figure 10: HCO₃⁻

There was a significant decrease ($p < 0.05$) in the bicarbonate level of the HIV patients on ART compared to the HIV patients not on ART and Control (figure 10).

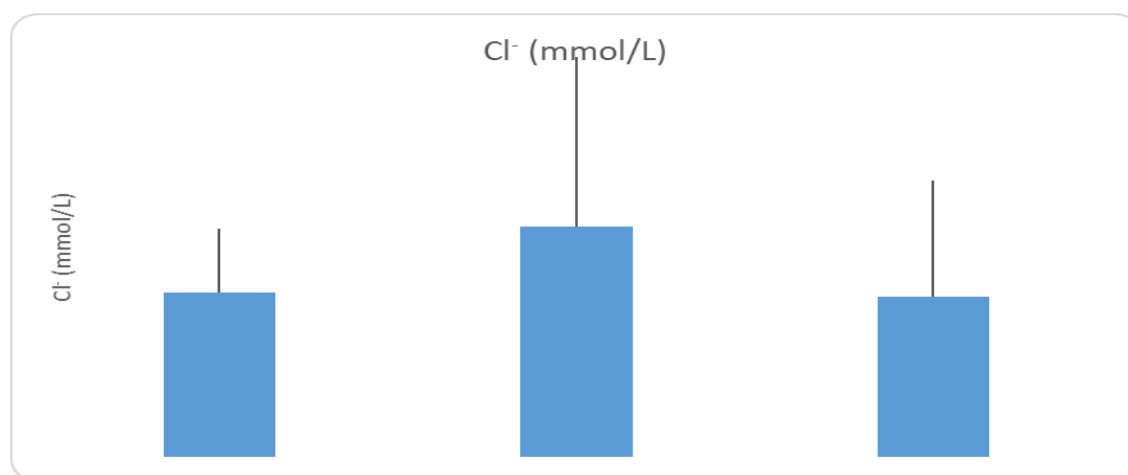


Figure11: Cl-

There was a significant decrease ($p < 0.05$) in the bicarbonate level of the HIV patients on ART compared to the HIV patients not on ART and Control (figure 11).

Table 2: Renal Function Tests

Group	Urea (mmol/L)	Creatinine (μ mol/L)	Na ⁺ (mmol/L)	K ⁺ (mmol/L)	HCO ₃ ⁻ (mmol/L)	Cl ⁻ (mmol/L)
Control	4.59 ± 0.56	82.70 ± 11.33	139.80 ± 1.29	4.19 ± 0.13	24.85 ± 0.70	102.13 ± 1.22
HIV not on ART	5.72 ± 1.02	92.58 ± 10.89	140.60 ± 2.25	4.28 ± 0.30	24.13 ± 1.67	103.40 ± 3.23
HIV on ART	4.81 ± 0.81	90.18 ± 10.42	138.05 ± 2.21	4.21 ± 0.17	23.93 ± 1.27	102.05 ± 2.22

DISCUSSION

Insulin Resistance among the Study Groups

The most important metabolic observation in this study was the marked elevation of fasting insulin and HOMA-IR among HIV-positive participants who were not receiving ART. This pattern suggests that HIV infection itself, or the biological consequences associated with untreated infection, may contribute substantially to insulin resistance. The observation is important because it prevents the metabolic consequences of HIV from being attributed exclusively to ART.

The HOMA-IR value of 3.49 ± 1.82 in the untreated HIV group was substantially greater than the 1.27 ± 0.29 observed in controls. This finding is biologically plausible because chronic HIV infection is accompanied by immune activation, systemic inflammation and alterations in adipose-tissue biology, all of which can interfere with insulin signalling. Høgh et al. (2022) similarly found that insulin resistance among PLWH was associated with previous immunodeficiency and exposure to older antiretroviral agents, supporting the concept that HIV-related disease history and treatment exposure can both influence glucose metabolism.

The increase in fasting insulin in the untreated HIV group is particularly noteworthy. The simultaneous elevation of fasting glucose and insulin suggests a compensatory pancreatic response to reduced peripheral insulin sensitivity. In early insulin resistance, pancreatic β -cells may increase insulin secretion sufficiently to maintain glucose concentrations within or near the normal range. Consequently, a relatively modest increase in fasting glucose should not be interpreted as absence of metabolic dysfunction when fasting insulin and HOMA-IR are substantially elevated.

The current results are also compatible with the observation that insulin resistance may precede clinically diagnosable diabetes by several years. The relatively high HOMA-IR in the absence of overtly diabetic mean glucose concentrations may therefore represent an early metabolic phenotype. This is clinically important because identification of insulin resistance provides an opportunity for lifestyle intervention and risk-factor modification before persistent hyperglycaemia develops.

An important and somewhat unexpected finding was that the ART group had lower fasting insulin and HOMA-IR than the HIV-positive group not receiving ART. HOMA-IR declined from 3.49 ± 1.82 in untreated HIV to 2.22 ± 1.23 among ART-treated participants. Similarly, fasting insulin decreased from 13.33 ± 5.86 to 8.68 ± 2.40 $\mu\text{IU/mL}$.

This finding argues against a simplistic conclusion that ART uniformly causes insulin resistance. Rather, ART may reduce some of the metabolic consequences of uncontrolled HIV infection through suppression of viral replication and reduction of HIV-associated immune activation. At the same time, individual antiretroviral agents can have different metabolic effects.

This distinction is supported by contemporary evidence. A 2026 systematic review found that ART-treated PLWH had substantially greater odds of metabolic syndrome than ART-naïve individuals, although the association with type 2 diabetes itself was not statistically conclusive across all studies (Mahanani et al., 2026). A separate 2026 meta-analysis of prospective cohorts, however, found higher diabetes incidence among ART-exposed participants and an association between longer ART exposure and diabetes incidence (Li et al., 2026).

Therefore, the lower HOMA-IR observed in the ART group in the present study could be as an association rather than evidence that ART protects against insulin resistance. Because this was a three-group comparative study rather than a randomized longitudinal intervention, differences may also reflect duration of HIV infection, nutritional status, disease severity, age, body composition, ART regimen, treatment duration and other factors.

Accordingly, the metabolic effects observed in the Onitsha cohort should not be generalized to all ART regimens without knowing the specific drugs, treatment duration and adherence patterns used by participants.

HbA1c and Glucose Findings

HbA1c was $5.05 \pm 0.18\%$ among controls, $5.43 \pm 0.40\%$ in HIV-positive participants not on ART and $5.49 \pm 0.40\%$ in HIV-positive participants receiving ART. These values suggest higher average glycaemic exposure among the HIV-positive groups, although the group means remain below the conventional diagnostic threshold for diabetes.

The interpretation of HbA1c in HIV requires some caution. The American Diabetes Association recognizes HIV among conditions in which the relationship between HbA1c and glycaemia may sometimes be altered and recommends use of plasma glucose criteria when there is discordance between HbA1c and measured glucose (American Diabetes Association Professional Practice Committee, 2024).

This is relevant to the present study because fasting insulin and HOMA-IR demonstrate metabolic abnormalities that are more pronounced than might be inferred from mean glucose or HbA1c alone. Consequently, reliance on HbA1c alone could potentially underestimate early metabolic dysfunction in some PLWH.

RENAL FUNCTION FINDINGS

Serum Creatinine

Serum creatinine was 82.70 ± 11.33 $\mu\text{mol/L}$ in controls, compared with 92.58 ± 10.89 $\mu\text{mol/L}$ among HIV-positive participants not on ART and 90.18 ± 10.42 $\mu\text{mol/L}$ among those receiving ART.



The thesis table identifies the increases in both HIV groups relative to the control group as statistically significant. Thus, HIV-positive participants demonstrated higher circulating creatinine irrespective of ART status.

This finding is consistent with the established observation that HIV can affect the kidney through several pathways. HIV-associated nephropathy, immune-complex disease, HIV-associated immune activation, comorbid hypertension and diabetes, opportunistic infections, and nephrotoxic medications can all contribute to renal abnormalities (Lucas & Wyatt, 2022; Wearne & Davidson, 2024).

The finding is also relevant to the Nigerian setting. Nwatarali et al. (2023) reported kidney dysfunction among HIV-positive patients receiving HAART at a Nigerian tertiary hospital. Furthermore, Nigerian research has demonstrated that genetic susceptibility involving APOL1 may contribute to proteinuric kidney disease among people living with HIV (Nwatarali et al., 2023; Wudil et al., 2021).

Urea

Urea increased from 4.59 ± 0.56 mmol/L in controls to 5.72 ± 1.02 mmol/L among HIV-positive participants not receiving ART. Interestingly, the ART group recorded 4.81 ± 0.81 mmol/L, close to the control value and significantly lower than the non-ART HIV group according to the thesis table's superscript notation.

This pattern may suggest that ART-associated viral suppression and improved clinical status were accompanied by partial normalization of urea metabolism. Nevertheless, serum urea is influenced by hydration, protein intake, catabolic state, gastrointestinal bleeding and renal function and should therefore not be interpreted as a specific marker of glomerular filtration.

The finding nevertheless reinforces the importance of interpreting renal biochemical markers collectively. KDIGO recommends assessment of both filtration and kidney damage rather than relying on a single biochemical parameter (KDIGO CKD Work Group, 2024).

Electrolytes and Bicarbonate

The sodium concentrations were relatively close among the three groups, with 139.80 ± 1.29 mmol/L in controls, 140.60 ± 2.25 mmol/L in HIV-positive participants not receiving ART and approximately 138.05 mmol/L in the ART group; the SD in the latter cell is obscured in the supplied photograph.

Potassium values were similarly close: 4.19 ± 0.13 mmol/L, 4.28 ± 0.30 mmol/L and 4.21 ± 0.17 mmol/L in controls, non-ART HIV and ART groups, respectively. Thus, there was no conspicuous potassium disturbance across the groups.

Bicarbonate was lower in the HIV groups: 24.85 ± 0.70 mmol/L in controls, 24.13 ± 1.67 mmol/L among HIV-positive participants not on ART and 23.93 ± 1.27 mmol/L among those on ART. The reduction may indicate a modest alteration in acid-base homeostasis, although the observed values do not by themselves demonstrate clinically important metabolic acidosis.

The overall electrolyte findings are consistent with the concept that renal involvement in HIV may be subtle and may not always manifest as dramatic electrolyte derangement. Contemporary reviews emphasize that HIV-related renal disease encompasses glomerular, tubular and vascular abnormalities as well as treatment-related renal effects (Wearne & Davidson, 2024).

An important implication of this study is the coexistence of metabolic and renal biochemical abnormalities. The untreated HIV group demonstrated the greatest insulin resistance while both HIV groups had higher creatinine concentrations than controls. The present findings therefore have practical relevance beyond the individual laboratory abnormalities. A patient attending an HIV clinic may simultaneously be developing insulin resistance and early renal dysfunction, yet neither condition may be clinically obvious. Routine



biochemical surveillance could identify these abnormalities before progression to overt diabetes, CKD or cardiovascular disease.

The results of this Onitsha study broadly fit the emerging African literature showing that HIV care has entered an era dominated increasingly by chronic metabolic and renal comorbidities. In Nigeria, Jumare et al. (2023) demonstrated a high prevalence of metabolic syndrome and particularly high rates of prediabetes and diabetes among PLWH. Ojong et al. (2022) similarly demonstrated significant metabolic abnormalities among ART-treated Nigerian HIV patients compared with ART-naïve participants. These findings provide regional support for the metabolic abnormalities observed in the present study.

CONCLUSION

This study demonstrates substantial alterations in glucose-insulin homeostasis and selected renal biochemical parameters among people living with HIV attending Shanahan University Teaching Hospital, Onitsha.

The most pronounced insulin-resistance phenotype occurred among HIV-positive participants not receiving ART, who had the highest fasting glucose, fasting insulin and HOMA-IR and the lowest QUICKI. Participants receiving ART demonstrated lower fasting insulin and HOMA-IR than untreated HIV-positive participants, suggesting partial improvement of insulin sensitivity following treatment; however, their metabolic indices remained distinct from those of HIV-negative controls.

Renal assessment demonstrated higher serum creatinine in both HIV-positive groups compared with controls, while urea was highest among HIV-positive participants not receiving ART and approached control values among those receiving ART. Electrolyte abnormalities were comparatively modest, although bicarbonate concentrations were lower in the HIV groups.

Taken together, these findings suggest that HIV infection and its treatment occur within a complex metabolic–renal continuum. Effective ART remains essential and should not be withheld because of these observations. Instead, HIV care should increasingly incorporate systematic screening for metabolic dysfunction and renal abnormalities.

RECOMMENDATIONS

Integrate metabolic screening into routine HIV care. Fasting plasma glucose or an appropriate glucose-based test should be periodically assessed, particularly in individuals with obesity, hypertension, family history of diabetes or prolonged ART exposure.

Laboratory systems should maintain longitudinal records so that changes in renal and metabolic markers can be detected over time rather than interpreted from isolated measurements.

Larger prospective studies should evaluate changes in HOMA-IR and eGFR before and after ART initiation.

Future studies should incorporate BMI, waist circumference, blood pressure, lipid profile, CD4 count, viral load, duration of ART, ART adherence, eGFR and urinary albumin-to-creatinine ratio.

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