Insulin Resistance and Liver Biochemical Alterations among People Living with HIV Before and During Antiretroviral Therapy in Onitsha, Southeast, Nigeria

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ABSTRACT

People living with HIV (PLWH) are increasingly surviving into older age because of effective antiretroviral therapy (ART). Consequently, attention has shifted from AIDS-related morbidity to chronic metabolic and cardiovascular complications, including insulin resistance, dyslipidaemia, abnormal glucose metabolism, weight gain and atherosclerotic cardiovascular disease. Both persistent HIV-associated inflammation and exposure to particular antiretroviral agents may contribute to these abnormalities. Contemporary evidence indicates that the metabolic phenotype associated with ART has evolved, with newer regimens producing less severe lipid toxicity than some older regimens but raising concerns about weight gain and glucose deregulation. This study assessed differences in insulin-resistance indices and serum lipid profiles among HIV-negative controls, people living with HIV who were not receiving ART, and people living with HIV receiving ART at Shanahan University Teaching Hospital, Waterside, Onitsha, and South-eastern Nigeria. The study comprised 120 participants, allocated equally into three groups of 40: HIV-negative controls, HIV-positive participants not receiving ART, and HIV-positive participants receiving ART. Glucose, HbA1c, fasting insulin, HOMA-IR and QUICKI were assessed as markers of glucose metabolism and insulin sensitivity, while total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C) and triglycerides (TG) constituted the lipid profile. Group differences were evaluated using analysis of variance (ANOVA), with statistical significance set at $p < .05$. These included the HIV-positive adults aged 18 years and above and who have been on ART for at least 6 months. Pregnant women, patients on steroids or lipid-altering drugs; patients with pre-existing severe liver or kidney disease, or Concurrent malignancy were excluded. Mean glucose concentrations were 4.59 ± 0.29, 5.61 ± 0.55 and 5.27 ± 0.42 mmol/L in the control, HIV-not-on-ART and HIV-on-ART groups, respectively. HbA1c was 5.05 ± 0.18%, 5.43 ± 0.40% and 5.49 ± 0.40%, respectively. Fasting insulin was substantially higher in untreated HIV (13.33 ± 5.86 µIU/mL) than in controls (6.21 ± 1.05 µIU/mL) and remained elevated among ART recipients (8.68 ± 2.40 µIU/mL). Similarly, HOMA-IR was highest in the untreated HIV group (3.49 ± 1.82), followed by the ART group (2.22 ± 1.23) and controls (1.27 ± 0.29). The lipid profile showed higher TC (5.18 ± 0.67 mmol/L), LDL-C (3.22 ± 0.69 mmol/L) and TG (1.73 ± 0.56 mmol/L) but lower HDL-C (1.13 ± 0.22 mmol/L) among participants not receiving ART. ART recipients showed comparatively improved TC (4.70 ± 0.53 mmol/L) and TG (1.17 ± 0.32 mmol/L), although HDL-C (1.27 ± 0.21 mmol/L) remained below the control value. The findings demonstrate substantial metabolic abnormalities among people living with HIV, particularly increased insulin

concentrations and HOMA-IR together with an adverse lipid pattern. The comparatively lower HOMA-IR and improved lipid concentrations among ART recipients suggest that effective HIV treatment may attenuate some metabolic abnormalities associated with untreated HIV, although metabolic risk does not completely disappear after ART initiation. These findings support integration of metabolic screening into routine HIV care in Nigeria.

KEYWORDS

HIV, antiretroviral therapy, insulin resistance, HOMA-IR; QUICK, dyslipidaemia, triglycerides, HDL cholesterol, LDL cholesterol, Nigeria

I. INTRODUCTION

The widespread availability of effective antiretroviral therapy has fundamentally changed the natural history of HIV infection. People living with HIV can now achieve prolonged survival and, with sustained viral suppression, approach the life expectancy of people without HIV. This therapeutic success has consequently shifted the clinical priorities of HIV care toward the prevention and management of chronic non-AIDS conditions. Cardiovascular disease, diabetes, dyslipidaemia, obesity, metabolic syndrome and insulin resistance have become increasingly important components of long-term HIV medicine.

Metabolic disturbances in HIV are biologically complex and cannot be attributed exclusively to ART. Chronic HIV infection is associated with persistent immune activation and systemic inflammation, even among individuals who achieve virological suppression. These processes can influence adipose-tissue function, hepatic lipid metabolism, endothelial function and glucose homeostasis. At the same time, conventional risk factors—including increasing age, obesity, physical inactivity, dietary factors, smoking and hypertension—contribute to cardio metabolic risk. Antiretroviral drugs may further modify this risk through drug-specific effects on adipose tissue, body weight, glucose metabolism and serum lipids.

Insulin resistance is particularly important because it may precede clinically overt diabetes by many years. HOMA-IR, calculated from fasting glucose and fasting insulin, is widely used as a surrogate index of insulin resistance in research, while QUICKI provides an inverse measure of insulin resistance and therefore generally increases as insulin sensitivity improves. In HIV, insulin resistance may be influenced by chronic inflammation, changes in adipose distribution, mitochondrial effects, lip dystrophy, weight gain and antiretroviral exposure. Contemporary reviews indicate that newer integrase strand-transfer inhibitors (INSTIs) and tenofovir alafenamide (TAF) can be associated with weight gain, while the effect on glucose metabolism is less uniform and may vary according to patient characteristics and the accompanying ART regimen.

The relationship between ART and metabolic abnormalities is therefore not necessarily linear. Earlier ART regimens, particularly some protease-inhibitor-containing combinations and older nucleoside analogues, were strongly associated with dyslipidaemia and body-fat redistribution. In contrast, contemporary INSTI-based regimens generally have a more favourable lipid profile but have generated concern regarding weight gain and, in some studies, impaired glucose homeostasis. A 2026 systematic review and meta-analysis reported that the metabolic burden associated with ART has shifted over time from

predominantly lipid abnormalities toward weight- and glucose-related outcomes, with ART-treated adults having higher odds of metabolic syndrome than ART-naïve adults, although residual confounding remains important.

Dyslipidaemia remains clinically important because PLWH have an elevated risk of atherosclerotic cardiovascular disease (ASCVD). A 2024 systematic review and meta-analysis found higher risks of dyslipidaemia, coronary artery disease and myocardial infarction among PLWH compared with people without HIV. In sub-Saharan Africa, this issue is particularly important because the epidemiological transition toward obesity, diabetes and cardiovascular disease is occurring alongside the continuing burden of HIV infection. A recent Ethiopian meta-analysis reported a pooled dyslipidaemia prevalence of approximately 69% among adults receiving ART, illustrating the substantial metabolic burden documented in African HIV populations.

Recent African studies further demonstrate that metabolic abnormalities remain clinically relevant in the modern ART era. In Ethiopia, dyslipidaemia has been reported among PLWH receiving routine HIV care, while comparative data have demonstrated substantial dyslipidaemia and abnormal glucose profiles among people receiving dolutegravir- and efavirenz-based regimens. Similarly, recent Ghanaian data demonstrated a high prevalence of dyslipidaemia among PLWH on ART, with BMI, physical inactivity and alcohol use among relevant associated factors.

Nigeria is undergoing a similar epidemiological transition, yet contemporary locally generated evidence integrating both insulin resistance and lipid abnormalities across untreated and ART-treated HIV groups remains comparatively limited. This is clinically important because metabolic alterations may occur even when conventional glucose concentrations remain within apparently acceptable ranges. Assessment of fasting insulin and insulin-resistance indices may therefore identify early metabolic dysfunction that would be missed by glucose or HbA1c alone. Moreover, recent HIV primary-care guidance recommends metabolic assessment—including glucose, HbA1c and lipid evaluation—as part of comprehensive long-term HIV management.

The present study therefore compared glucose metabolism, insulin resistance and lipid profiles among three groups of adults—HIV-negative controls, HIV-positive individuals not receiving ART, and HIV-positive individuals receiving ART—at Shanahan University Teaching Hospital, Onitsha. The central hypothesis was that HIV infection would be associated with adverse metabolic changes and that ART exposure would modify, but not necessarily completely reverse, these abnormalities.

II. MATERIALS AND METHODS

Venous blood collection was under aseptic conditions and the serum separated immediately. The separated serum samples were analysed using an Automated biochemical analyser-MINDRAY (BA 88A) produced by Mind ray Bio-Medical Electronics Co., Ltd, China; and also FINECARE FLUORESCENCE IMMUNOASSAY ANALYZER (FIA) produced by Guangzhou Wondfo Biotech Co., Ltd, China.

- Serum Glucose test was done by Glucose-Oxidase Method [Burtis 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 QUICKI were calculated. (Willig & overton, 2016)
- For Serum Urea -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.
- Lipid profile parameters (Triglyceride, Total Cholesterol, HDL-Cholestrol, LDL-Cholesterol) analysis were by enzymatic colorimetric techniques [Burtis, C.A.et al (2012)] using the kit produced by Randox Laboratories Ltd, UK.

III. RESULTS

A. Glucose and insulin-resistance indices

The results demonstrate a clear difference in glucose-insulin homeostasis among the three study groups.

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).

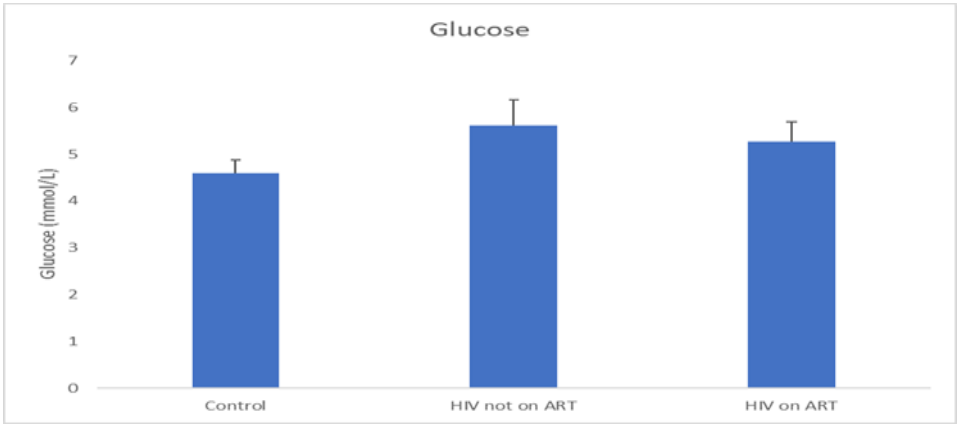

Figure 1: Glucose

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 2).

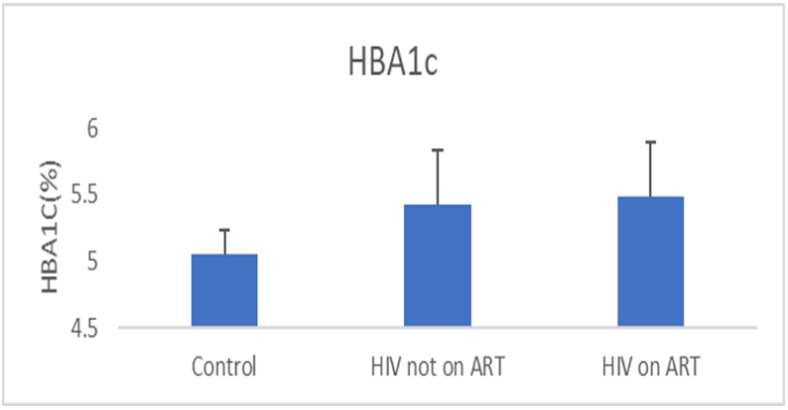

Figure 2: HBAIc

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 3).

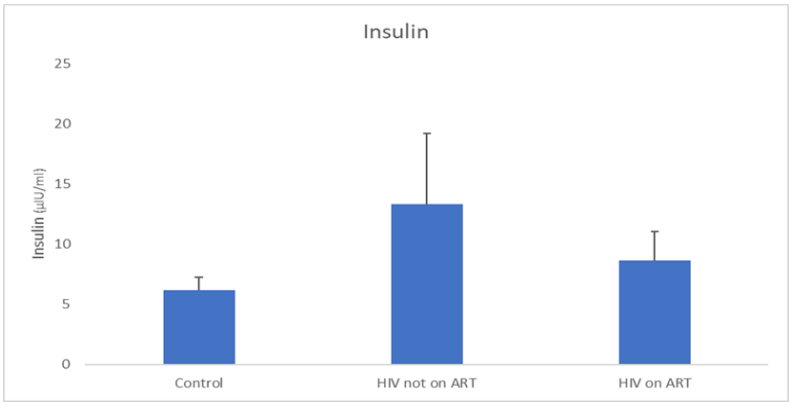

Figure 3: Insulin

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 4).

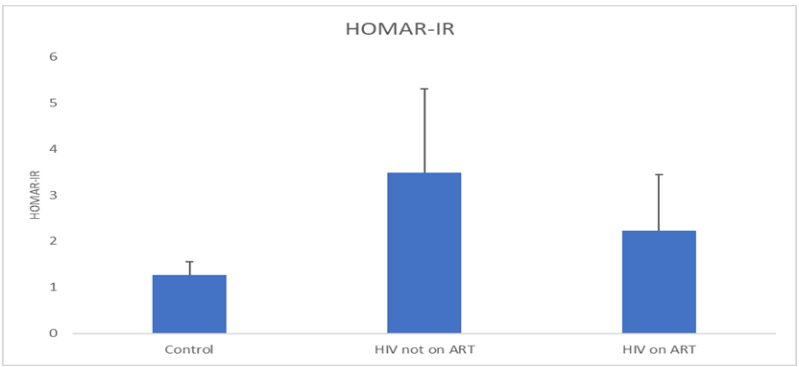

Figure 4: Homar-IR

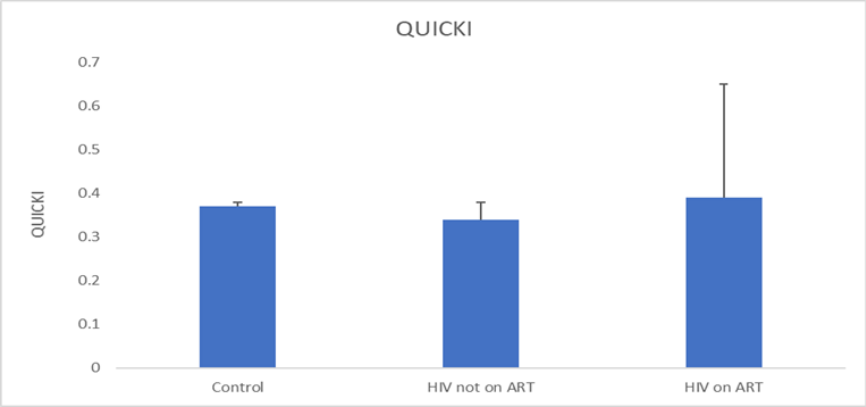

Figure 5: QUICKI

Table 1: Glycaemic Index

Group	Glucose (mmol/L)	HBA1c (%)	Insulin (µIU/ml)	HOMA-IR	QUICKI
Control	4.59 ± 0.29	5.05 ± 0.18	6.21 ± 1.05	1.27 ± 0.29	0.37 ± 0.01
HIV not on ART	5.61 ± 0.55	5.43 ± 0.40	13.33 ± 5.86	3.49 ± 1.82	0.34 ± 0.04
HIV on ART	5.27 ± 0.42	5.49 ± 0.40	8.68 ± 2.40	2.22 ± 1.23	0.39 ± 0.26

The data demonstrate a graded metabolic pattern across the three groups, with the untreated HIV group generally showing the greatest metabolic disturbance.

Fasting glucose was 4.59 ± 0.29 mmol/L in controls, increased significantly to 5.61 ± 0.55 mmol/L among HIV-positive participants not receiving ART, and was 5.27 ± 0.42 mmol/L among HIV-positive participants receiving ART.

HbA1c followed a similar pattern, increasing from 5.05 ± 0.18% in controls to 5.43 ± 0.40% among untreated HIV-positive participants and 5.49 ± 0.40% among ART-treated participants.

The most pronounced difference occurred in fasting insulin. Mean insulin concentration was 6.21 ± 1.05 µIU/mL among controls compared with 13.33 ± 5.86 µIU/mL among HIV-positive participants not receiving ART. ART-treated participants had an intermediate insulin concentration of 8.68 ± 2.40 µIU/mL.

HOMA-IR increased from 1.27 ± 0.29 in controls to 3.49 ± 1.82 in HIV-positive participants not receiving ART and 2.22 ± 1.23 in HIV-positive participants receiving ART. The study's superscript notation indicates significant increases relative to controls and a significant decrease in the ART-treated group relative to the untreated HIV group.

QUICKI was 0.37 ± 0.01 among controls and 0.34 ± 0.04 among untreated HIV-positive participants. The ART group was reported as 0.39 ± 0.26. Because the standard deviation in this cell is unusually large, the original dataset should be checked before publication.

Overall, the pattern of elevated fasting insulin and HOMA-IR among HIV-positive participants, particularly those not receiving ART, is consistent with reduced insulin sensitivity.

B. Lipid Profile

A distinct dyslipidaemia pattern was observed among HIV-positive participants not receiving ART.

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

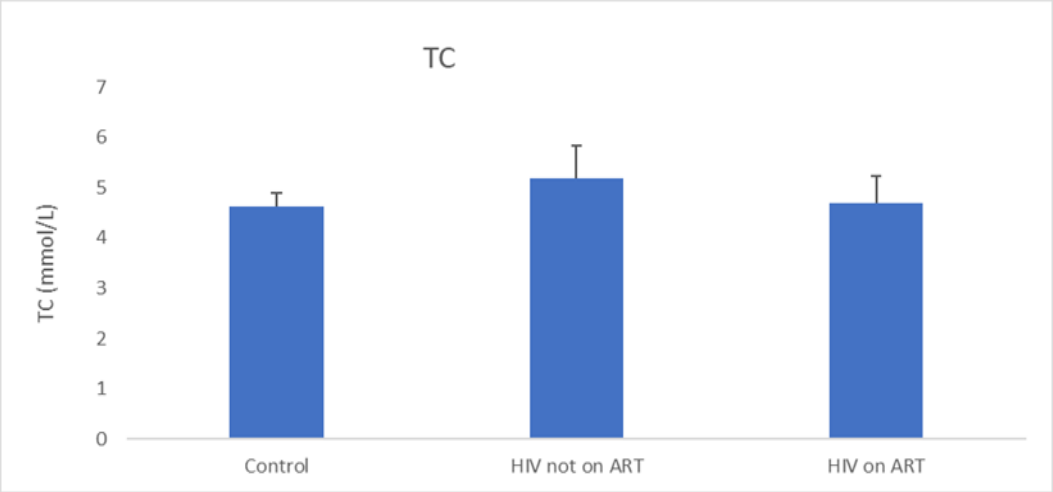

Figure 6: TOTAL CHOLESTEROL (TC)

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

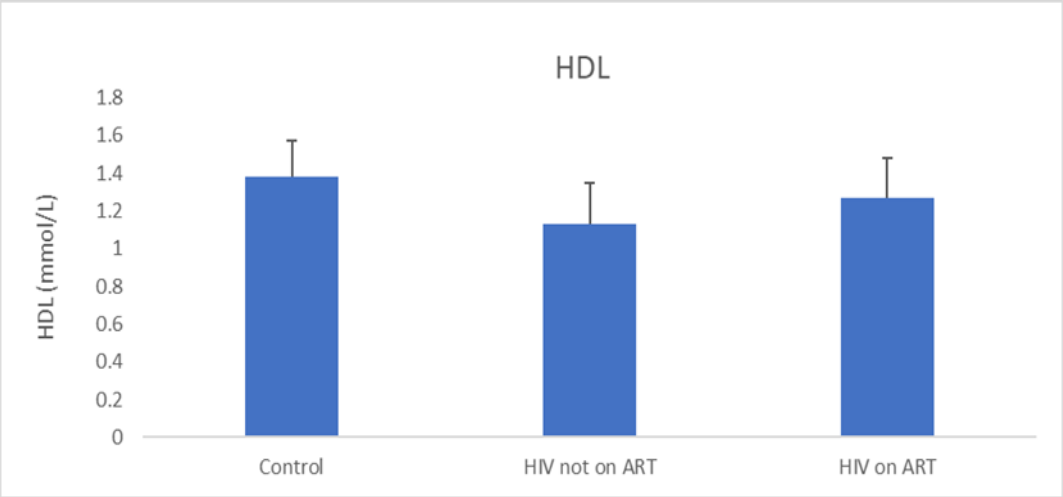

Figure 7: HDL-CHOLESTEROL

There was a significant increase ($p<0.05$) in the LDL concentration of the HIV patients not on ART compared to the HIV patients on ART and Control (figure 8).

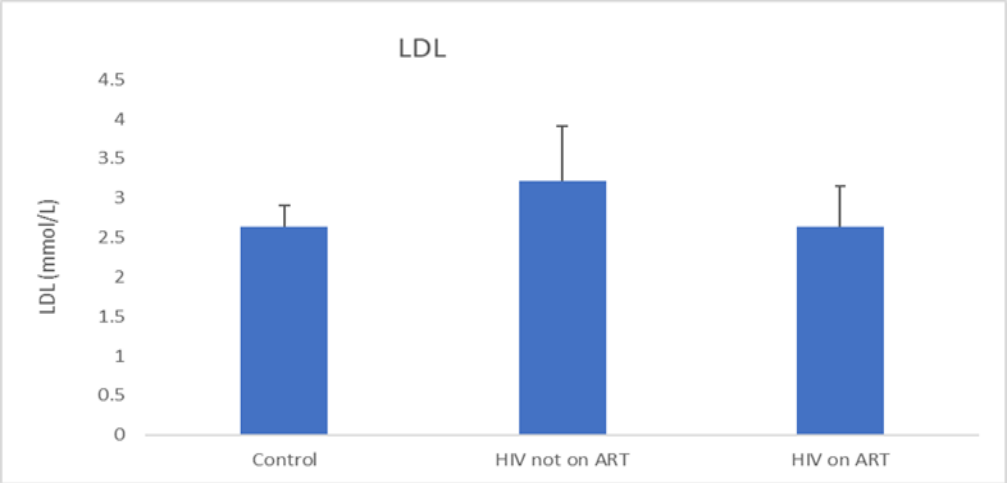

Figure 8: LDL-CHOLESTEROL

There was a significant increase ($p<0.05$) in the TG concentration of the HIV patients not on ART compared to the HIV patients on ART and Control (figure 9).

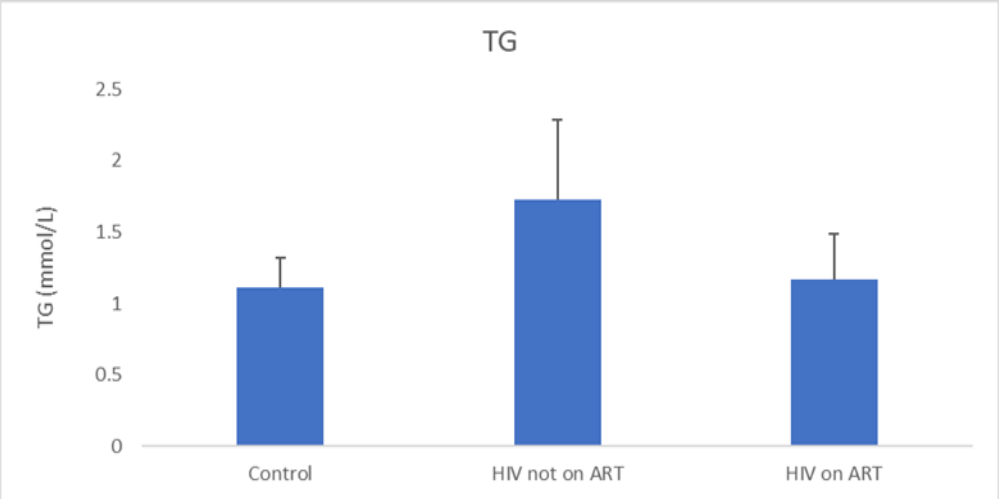

Figure 9: TTRGGLYCERIDE

Table 2: Lipid Profile

	TC (mmol/L)	HDL (mmol/L)	LDL (mmol/L)	TG (mmol/L)
Control	4.62 ± 0.28	1.38 ± 0.19	2.64 ± 0.27	1.11 ± 0.2
HIV not on ART	5.18 ± 0.67	1.13 ± 0.22	3.22 ± 0.69	1.73 ± 0.56
HIV on ART	4.70 ± 0.53	1.27 ± 0.21	2.64 ±0.51	1.17 ± 0.32

Total cholesterol increased from 4.62 ± 0.28 mmol/L among controls to 5.18 ± 0.67 mmol/L among HIV-positive participants not receiving ART. In the ART group, total cholesterol decreased to 4.70 ± 0.53 mmol/L. HDL-C was lower among untreated HIV-positive participants (1.13 ± 0.22 mmol/L) than among controls (1.38 ± 0.19 mmol/L). ART-treated participants had an intermediate HDL-C concentration of 1.27 ± 0.21 mmol/L. LDL-C increased from 2.64 ± 0.27 mmol/L in controls to 3.22 ± 0.69 mmol/L among untreated HIV-positive participants. The ART group had 2.64 ± 0.51 mmol/L, essentially returning to the control mean. Triglycerides showed the largest lipid difference: 1.11 ± 0.20 mmol/L in controls, 1.73 ± 0.56 mmol/L among untreated HIV-positive participants and 1.17 ± 0.32 mmol/L among ART-treated participants. Thus, the untreated HIV group demonstrated a pattern characterized by higher TC, LDL-C and TG and lower HDL-C, whereas ART was associated with substantial attenuation of these abnormalities.

IV. DISCUSSION

A. Insulin resistance was most pronounced in untreated HIV

The principal finding of this study is the substantially higher fasting insulin and HOMA-IR among participants with HIV who were not receiving ART. This suggests that untreated HIV infection in this population was associated with reduced insulin sensitivity, even though mean glucose and HbA1c concentrations remained below the conventional diagnostic range for diabetes. This finding has important clinical implications. Insulin resistance often develops before overt hyperglycaemia; consequently, relying exclusively on fasting glucose or HbA1c may underestimate early metabolic dysfunction. The present results suggest that apparently modest differences in glucose can coexist with substantial compensatory hyperinsulinaemia. This is particularly relevant in HIV populations, in whom HbA1c may have reduced sensitivity for detecting dysglycaemia.

The biological explanation is likely multifactorial. Persistent HIV-associated immune activation can promote inflammatory signalling that interferes with insulin signalling. Altered adipose-tissue distribution and adipocyte dysfunction may further increase free fatty-acid flux, impair insulin action and contribute to hepatic insulin resistance. These mechanisms can interact with conventional risk factors such as obesity, physical inactivity and dietary patterns. Current NIH guidance similarly identifies chronic inflammation, immune activation, traditional cardio metabolic risk factors and possible antiretroviral toxicities as overlapping contributors to metabolic disease in people with HIV.

The observed pattern also agrees with the broader contemporary literature. Diggins et al. (2022) concluded that ART can affect weight, adipose tissue, glucose and lipid metabolism, while Savinelli et al. (2024) noted emerging evidence of insulin resistance and diabetes in some individuals receiving INSTI-based therapy. More recently, a 2026 prospective-cohort meta-analysis found a substantial incidence of diabetes among PLWH and reported higher diabetes incidence among ART-exposed than ART-unexposed individuals, although differences in populations and diagnostic definitions remain important (Li et al., 2026).

B. ART appeared to attenuate, but not eliminate, insulin-resistance abnormalities

An important finding is that the ART group did not simply show a continuation of the metabolic deterioration seen in untreated HIV. Instead, fasting insulin fell from 13.33

μIU/mL in untreated HIV to 8.68 μIU/mL among ART recipients, while HOMA-IR declined from 3.49 to 2.22.

This observation should be interpreted cautiously. Because the three groups were compared at one point in time, the study cannot establish that ART itself caused the improvement. Differences could reflect duration of HIV infection, disease severity, nutritional status, body composition, ART regimen, adherence, viral suppression, CD4-cell recovery, age, sex or other unmeasured factors. Nevertheless, the intermediate metabolic phenotype of the ART group is compatible with the possibility that effective treatment reduces some metabolic consequences of uncontrolled HIV infection.

This distinction is particularly important because ART remains essential and should not be withheld because of potential metabolic adverse effects. Contemporary international guidance continues to recommend ART for all people with HIV, with INSTI-based regimens such as dolutegravir- or bictegravir-containing combinations recommended for most individuals.

The literature also demonstrates why ART effects must be considered regimen-specifically. Palella et al. (2023) reported that switching to INSTI- or TAF-containing regimens was associated with subsequent increases in BMI, while Capeau et al. (2024) described weight gain as particularly associated with dolutegravir, bictegravir and TAF. Conversely, Burns et al. (2022) found no overall difference in the rate of weight gain according to ART regimen in another cohort. These apparently contrasting findings reinforce the importance of considering the individual regimen and patient characteristics rather than treating "ART" as a single metabolic exposure.

C. Lipid abnormalities were greatest in untreated HIV

The lipid results reveal another important pattern. Compared with controls, participants with untreated HIV had higher: TC: 5.18 vs. 4.62 mmol/L; LDL-C: 3.22 vs. 2.64 mmol/L; TG: 1.73 vs. 1.11 mmol/L;

While HDL-C was lower: HDL-C: 1.13 vs. 1.38 mmol/L. Thus, the untreated HIV group exhibited a classic adverse lipid pattern characterised by higher atherogenic cholesterol and triglycerides together with lower HDL-C.

The approximate 56% elevation in TG is particularly notable, while the approximately 22% elevation in LDL-C indicates an important increase in atherogenic lipoprotein burden. The approximately 18% reduction in HDL-C further worsens the overall lipid profile. Although the present study did not directly calculate ASCVD risk, this combination is clinically relevant because dyslipidaemia is an established contributor to cardiovascular risk.

The finding is consistent with contemporary reviews describing dyslipidaemia in HIV as a multifactorial consequence of HIV infection, inflammation, lifestyle factors, body composition and ART exposure.

D. The lipid profile of ART recipients was comparatively improved

The ART group demonstrated considerable improvement in several lipid variables compared with untreated HIV. TC decreased from 5.18 to 4.70 mmol/L, TG from 1.73 to 1.17 mmol/L, and LDL-C from 3.22 to 2.64 mmol/L. HDL-C also increased from 1.13 to 1.27 mmol/L, although it remained below the control value of 1.38 mmol/L.

This pattern is important because it argues against interpreting ART as universally dyslipidaemic. Effective treatment can suppress HIV replication and reduce the metabolic consequences of uncontrolled infection, while some contemporary ART regimens have relatively favourable lipid effects.

Therefore, the improved lipid profile observed in the ART group in the present study may reflect the metabolic benefit of controlling HIV itself, characteristics of the ART regimens used, or both.

E. Comparison with African evidence

The results are particularly relevant in the African context. A 2024 systematic review and meta-analysis from Ethiopia estimated a pooled dyslipidaemia prevalence of 69.32% among adults with HIV receiving ART. Elevated TG, TC and LDL-C and low HDL-C were all common, with age and BMI associated with several lipid abnormalities (Belete et al., 2024).

Similarly, Assefa et al. (2023) documented dyslipidaemia among PLWH receiving routine HIV care in Ethiopia, while Jemal et al. (2023) demonstrated a high burden of dyslipidaemia in individuals receiving both dolutegravir- and efavirenz-based therapy. More recently, Abdulai et al. (2025) reported a 64.1% prevalence of dyslipidaemia among Ghanaian PLWH on ART and identified physical inactivity, higher BMI and alcohol use as important associated factors.

The present Nigerian findings therefore fit into an emerging African pattern in which HIV care is increasingly complicated by cardio metabolic disease. However, regional differences in diet, ART regimens, socioeconomic circumstances, obesity prevalence, age structure and access to laboratory monitoring mean that findings from Ethiopia or Ghana should not be assumed to apply quantitatively to South-eastern Nigeria.

F. Cardiovascular significance

The lipid abnormalities observed in this study deserve attention because cardiovascular disease has become an increasingly important cause of morbidity among PLWH. Zhu et al. (2024), synthesising data from more than 300,000 PLWH, reported significantly higher risks of dyslipidaemia, coronary artery disease and myocardial infarction compared with HIV-negative populations.

The implications extend beyond simply identifying an abnormal laboratory result. Contemporary HIV guidelines now recommend systematic assessment and management of cardiovascular risk factors. The 2025 U.S. HIV guidelines emphasise that chronic inflammation, traditional risk factors and antiretroviral toxicities may converge to increase ASCVD risk in PLWH.

The findings therefore support the concept of partial metabolic recovery with treatment, rather than complete normalization. This interpretation is compatible with recent evidence that the metabolic effects of HIV and ART are dynamic. Mahanani et al. (2026) found that the metabolic consequences associated with ART have changed over successive treatment eras, with contemporary concerns increasingly involving weight and glucose-related outcomes rather than the severe lipid toxicity associated with some older regimens.

V. CONCLUSION

This study demonstrates significant alterations in insulin sensitivity and lipid metabolism among HIV-positive individuals attending Shanahan University Teaching Hospital, Onitsha.

HIV-positive participants not receiving ART exhibited the most pronounced metabolic abnormalities, including higher fasting glucose, HbA1c, fasting insulin and HOMA-IR, together with an adverse lipid profile characterized by increased total cholesterol, LDL-C and triglycerides and reduced HDL-C.

ART-treated participants demonstrated improvement in several parameters relative to untreated HIV-positive participants, particularly fasting insulin, HOMA-IR, total cholesterol, LDL-C and triglycerides. Nevertheless, metabolic abnormalities were not completely normalized when compared with HIV-negative controls.

The findings support the concept that successful HIV care should extend beyond viral suppression to include systematic assessment of metabolic and cardiovascular health. The growing evidence base indicates that HIV, ART, inflammation, ageing, body composition and traditional cardiovascular risk factors interact to determine long-term cardiometabolic outcomes.

RECOMMENDATIONS

Based on the findings of this study, the following recommendations are proposed:

Routine metabolic screening should be incorporated into HIV care. Fasting glucose, HbA1c and lipid profile should be assessed periodically, particularly in patients with additional metabolic or cardiovascular risk factors.

Lipid abnormalities should be identified and treated early. Patients with persistent dyslipidaemia should receive appropriate dietary, lifestyle and pharmacological interventions according to current HIV and

Longitudinal research is particularly important. Prospective studies beginning before ART initiation and following participants after ART commencement would better distinguish metabolic abnormalities caused by HIV itself from those associated with ART exposure.

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