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Research Article

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Prevalence of Dyslipidemia and Its Association with HAART Regimens among HIV-Positive Patients at Machakos Level V Hospital

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Abstract

Background: *Highly active antiretroviral therapy (HAART) has transformed HIV management, improving survival and quality of life. However, prolonged use has been associated with metabolic derangements, including dyslipidemia, which elevates cardiovascular disease risk. Evaluating lipid changes across HAART regimens enhances laboratory and clinical understanding of therapy-related complications.*

Objective: *To determine the prevalence of dyslipidemia and its association with HAART regimens among HIV-positive patients attending Machakos Level V Hospital, Kenya.*

Methods: *A cross-sectional study involving 406 HIV-positive adults on HAART was conducted. Data on socio-demographics and clinical characteristics were obtained through questionnaires. Venous blood was collected after overnight fasting, and serum lipid profiles (TC, TG, HDL-C, LDL-C) were analyzed using enzymatic colorimetric assays. Dyslipidemia was classified per NCEP-ATP III criteria. Data were analyzed using SPSS v28, with Chi-square and logistic regression used to test associations at $p < 0.05$.*

Results: *Overall dyslipidemia prevalence was 74.1%. Protease inhibitor (PI)-based regimens were associated with higher mean TC and LDL-C levels and significantly elevated TC/HDL-C ratios ($p = 0.046$). PI users were four times more likely to have a high atherogenic index (OR = 4.19, 95% CI 1.03–17.02). Low HDL-C (58.5%) and high TG (47.4%) were the most frequent abnormalities, while high LDL-C was least common (26.2%). Dyslipidemia prevalence was slightly higher in males and older participants but not statistically significant. Hypertension was significantly associated with dyslipidemia ($p = 0.044$).*

Conclusion: *Dyslipidemia is highly prevalent among HIV-positive patients on HAART, especially those on PI regimens. Strengthening laboratory monitoring, clinician laboratory collaboration, and education on lipid interpretation in HIV care are vital for improved patient outcomes.*

Keywords: HIV, HAART, Dyslipidemia, Lipid Profile, Clinical Chemistry, Laboratory Practice

1. Introduction

Human Immunodeficiency Virus (HIV) infection remains a leading global health concern, affecting over 38 million individuals worldwide and continuing to challenge healthcare systems, especially in sub-Saharan Africa [1]. The introduction of Highly Active Antiretroviral Therapy (HAART) has transformed HIV

infection from a fatal disease into a chronic, manageable condition [2]. Despite its success, long-term HAART use has been linked to metabolic complications, notably dyslipidemia, which increases cardiovascular disease (CVD) risk and compromises overall health outcomes [3,5].

Dyslipidemia is defined by abnormalities in serum lipid levels — elevated total cholesterol (TC), triglycerides (TG), or low-density lipoprotein cholesterol (LDL-C), and/or reduced high-density lipoprotein cholesterol (HDL-C) [6]. In HIV-infected individuals, dyslipidemia arises from both viral effects and drug-induced metabolic alterations. HIV infection promotes chronic inflammation and cytokine dysregulation, which affect lipid metabolism. HAART compounds these effects through direct biochemical interference with lipid synthesis, transport, and clearance [7]. Different antiretroviral drug classes influence lipid metabolism through specific mechanisms. Protease inhibitors (PIs) alter lipid regulation by inhibiting hepatic sterol regulatory element-binding proteins (SREBPs), disrupting triglyceride synthesis and LDL receptor recycling, and increasing circulating LDL-C and TG [8,9]. Non-nucleoside reverse transcriptase inhibitors (NNRTIs), such as efavirenz, induce hepatic cytochrome P450 enzymes that may enhance cholesterol production [10]. Conversely, integrase strand transfer inhibitors (INSTIs), like dolutegravir, generally show neutral or favorable lipid effects [11]. These drug-induced alterations elevate atherogenic indices, including the total cholesterol to HDL-C (TC/HDL-C) ratio, and contribute to early-onset cardiovascular complications [12]. Therefore, monitoring lipid profiles in HAART-treated patients is essential for detecting metabolic disturbances and preventing adverse outcomes.

In sub-Saharan Africa, dyslipidemia prevalence among HAART users ranges between 60–80% [13–15], with Kenyan studies reporting similar figures [16]. However, there remains limited data on regimen-specific lipid effects in semi-urban populations such as Machakos County. Beyond clinical implications, this issue has substantial relevance to clinical chemistry education and practice. Laboratory professionals play a critical role in lipid testing, quality assurance, result interpretation, and communication with clinicians. Enhancing awareness of HAART-induced biochemical changes among medical laboratory scientists can improve diagnostic accuracy and patient management. This study therefore aimed to determine the prevalence of dyslipidemia and its association with HAART regimens among HIV-positive patients attending Machakos Level V Hospital, Kenya, while drawing practical insights for laboratory practice and clinical chemistry education.

2. Materials and Methods

2.1. Study Design and Setting

A cross-sectional study was conducted at the Comprehensive Care Clinic (CCC) of Machakos Level V Hospital, a major referral facility serving Machakos County and neighboring regions.

2.2. Study Population

The study population comprised HIV-positive adult patients on HAART for at least three months. Participants with diabetes, acute illness, or those on lipid-lowering medication were excluded.

2.3. Sample Size and Sampling Technique

A total of 406 participants were selected using systematic random sampling from the CCC registry.

2.4. Data and Sample Collection

Structured questionnaires were used to gather demographic and clinical data. After overnight fasting, 5 mL of venous blood was collected, serum separated, and analyzed immediately or stored at −20 °C until testing.

2.5. Laboratory Analysis

Lipid parameters (TC, TG, HDL-C, and LDL-C) were analyzed using enzymatic colorimetric methods with the H600 autoanalyzer system. LDL-C was calculated using the Friedewald formula. Internal quality control was maintained using commercial control sera before every analytical run.

2.6. Definition of Dyslipidemia

Dyslipidemia in this study was defined according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III) criteria. It was characterized by one or more abnormalities in serum lipid levels, including total cholesterol (TC) greater than 5.2 mmol/L, triglycerides (TG) greater than 1.7 mmol/L, or low-density lipoprotein cholesterol (LDL-C) greater than 3.4 mmol/L. High-density lipoprotein cholesterol (HDL-C) levels less than 1.0 mmol/L for males or less than 1.3 mmol/L for females were also considered abnormal. Participants who exhibited any one or more of these lipid abnormalities were classified as having dyslipidemia.

2.7. Data Analysis

Data were analyzed using SPSS v28. Descriptive statistics summarized demographic and lipid data. Associations between dyslipidemia and HAART regimens were tested using Chi-square, and odds ratios (ORs) were estimated at 95% confidence intervals. A p-value < 0.05 denoted statistical significance.

2.8. Ethical Considerations

Ethical approval was obtained from Kenyatta University Ethics Review Committee (KU-ERC). Permission was granted by the National Commission for Science, Technology, and Innovation (NACOSTI). Written informed consent was obtained from all participants.

3. Results

A total of 406 HIV-positive participants were included, with a female majority and most aged between 35–54 years. The mean duration on HAART was 7.3 years, and a large proportion were on first-line regimens.

3.1. Lipid Profiles by HAART Regimen

Participants were categorized into NNRTI-, PI-, and INSTI-based regimen groups. The TDF/3TC/DTG (INSTI-based) regimen accounted for the largest proportion (approximately 89.7%).

Across all regimens, the mean serum concentrations for TC, TG, LDL-C, and HDL-C varied, but patients on PI-based regimens consistently displayed higher mean TC and LDL-C levels compared to NNRTI or INSTI users. The mean TC/HDL-C ratio also differed significantly among regimen groups (p = 0.046), indicating an elevated atherogenic risk among PI users. Logistic regression confirmed that PI users were four times more likely to present with high TC/HDL-C ratios (OR = 4.19, 95% CI = 1.03–17.02). Other lipid parameters showed no statistically significant difference across HAART regimens (*see Table 1*).

	High n (%)	**Low n (%)**	**OR (95%CI)**	**P-value**
HAART regimen	**TC**			
INSTI	108(91.5)	260(90.3)	0.30(0.04 - 2.44)	0.26
PI	9(7.6)	20(6.9)	0.28(0.03 - 2.57)	0.259
NNRTI	1(0.8)	8(2.8)	Ref	
	TG			
INSTI	139(88.0)	229(92.3)	2.06(0.544 - 7.80)	0.288
PI	14(8.9)	15(6.0)	1.34(0.30 - 6.02)	0.703
NNRTI	5(3.2)	4(1.6)	Ref	
	HDL			
INSTI	161(87.5)	207(93.2)	1.61(0.43 - 6.08)	0.485
PI	18(9.8)	11(5.0)	0.76(0,17 - 3.47)	0.271
NNRTI	5(2.7)	4(1.8)	Ref	
	LDL			
INSTI	93(92.1)	275(90.2)	0.85(0.17 - 4.14)	0.835
PI	6(5.9)	23(7.5)	1.10(0.18 - 6.70)	0.922
NNRTI	2(2.0)	7(2.3)	Ref	
	TC/HDL			
INSTI	119(86.9)	249(92.6)	2.83(0.59 - 13.63)	0.194
PI	12(8.8)	17(6.3)	4.19(1.03 - 17.02)	0.046
NNRTI	6(4.4)	3(1.1)	Ref	
	TG/HDL			
INSTI	275(89.3)	93(94.9)	1.18(0.24 - 5.80)	0.835
PI	26(8.4)	3(3.1)	0.40(0.06 - 2.91)	0.368
NNRTI	7(2.3)	2(2.0)	Ref	
	LDL/HDL			
INSTI	100)88.5)	268(91.5)	3.35(0.88 - 12.73)	0.076
PI	8(7.1)	21(7.2)	3.28(0.70 - 15.41)	0.132
NNRTI	5(4.4)	4(1.4)	Ref	

TC: Total cholesterol, **TG:** Triglycerides, **HDL:** High density lipoprotein, **LDL:** Low density lipoprotein, **PI:** Protease inhibitor, **INSTI:** Intergrase inhibitor, **NNRTI:** Non- nucleoside reverse transcriptase inhibitor, **OR:** Odds ratio

Table 1: Lipid Profiles According to Haart for Hiv-Positive Patients on Haart at Machakos Level V Hospital

3.2. Prevalence of Dyslipidemia

The overall prevalence of dyslipidemia was 74.1% (301/406). Dyslipidemia was more frequent among males (75.9%) than females (73.4%), although the difference was not statistically significant ($p = 0.707$). Prevalence increased with age, peaking at 79.7% among participants aged ≥60 years, and was highest among obese participants (79.4%) compared to those with normal BMI (70.1%). By regimen, dyslipidemia was present in 79.3% of PI users, 73.1% of INSTI users, and 100% of NNRTI users, though not statistically significant (see Table 2). Hypertensive participants recorded a significantly higher prevalence (84.6%) compared to non-hypertensive ones ($p = 0.044$).

Factors	Dyslipidemia		P-value
	Yes n (%)	No n (%)	
Age			
≤24 years	13(56.5)	10(43.5)	
25 - 40 years	78(78.0)	22(22.0)	0.262
41 - 59 years	163(72.8)	61(27.2)	
≥60 years	47(79.7)	12(20.3)	
Gender			
Male	88(75.9)	28(24.1)	0.707
Female	213(73.4)	77(26.6)	
BMI			
Normal	145(73.6)	52(26.4)	
Underweight	17(63.0)	10(37.0)	0.408
Overweight	85(74.6)	29(25.4)	
Obese	54(79.4)	14(20.6)	
Systolic pressure			
>=140 mmHg	62(88.6)	8(11.4)	0.002
<140 mmHg	239(71.1)	97(28.9)	
Diastolic pressure			
>=90 mmHg	58(84.1)	11(15.9)	0.049
<90 mmHg	243(72.1)	94(27.9)	
Hypertension			
Yes	55(84.6)	10(15.4)	0.044
No	246(72.1)	95(27.9)	
Cigarette smoking			
Yes	15(68.2)	7(31.8)	0.616
No	286(74.5)	98(25.5)	
History of CVD			
Yes	20(71.4)	8(28.6)	0.823
No	281(74.3)	97(25.7)	
Duration of ARV drug use			
Less than 5 years	66(73.3)	24(26.7)	
5 – 10 years	103(69.1)	46(30.9)	0.129
>10 years	132(79.0)	35(21.0)	
Treatment line			
First line	278(73.7)	99(26.3)	0.661
Second line	23(79.3)	6(20.7)	
HAART regimen			
PI			
Yes	23(79.3)	6(20.7)	0.661
No	278(73.7)	99(26.3)	
INSTI			
Yes	269(73.1)	99(26.9)	0.173
No	32(84.2)	6(15.8)	
NNRTI			
Yes	9(100)	0	1.00
No	292(74.1)	105(25.9)	

CVD: Cardiovascular disease, **BMI:** Body mass index, **ARV:** Antiretrovirals, **HAART:** Highly active antiretroviral therapy, **PI:** Protease inhibitor, **INSTI:** Intergrase inhibitor, **NNRTI:** Non- nucleoside reverse transcriptase inhibitor

Table 2: Prevalence of dyslipidemia according to patient characteristics

3.3. Frequency of Specific Lipid Abnormalities

Among the dyslipidemic participants, **low HDL-C** was the most common abnormality (58.5%), followed by **high triglycerides (47.4%)** and **elevated total cholesterol (35.9%). High LDL-C** was least frequent (26.2%). Combined lipid abnormalities (≥2 deranged parameters) were observed in **60.8%** of cases, while 39.2% showed single-parameter changes *(see Table 3*). These findings indicate that the majority of patients experienced **mixed dyslipidemia**, suggesting broad metabolic effects of long-term HAART use.

Lipid Abnormality	Female N (%)	Male N (%)	Difference (%)	Total N (%)
No lipid abnormality	77(73.3)	28(26.7)	46.6	105(25.9)
Isolated dyslipidemias				
One abnormality				
TC	92(78.0)	26(22.0)	56.0	118(29.1)
TG	114(72.2)	44(27.8)	44.4	158(38.9)
HDL	122(66.3)	62(33.7)	32.6	184(45.3)
LDL	78(77.2)	23(27.8)	44.4	101(24.9)
TC/HDL ratio	84(70.6)	35(29.4)	41.2	119(29.3)
Mixed dyslipidemia				
Two abnormalities				
TG + low HDL	54(65.1)	29(34.9)	30.2	83(20.4)
LDL+ low HDL	32(74.4)	11(25.6)	48.8	43(10.6)
TC + TG	51(77.3)	15(22.7)	54.6	66(16.3)
TC + LDL	73(78.5)	20(21.5)	57.0	93(22.9)
Three abnormalities				
TC + TG + HDL	21(67.7)	10(32.3)	35.4	31(7.6)
TC + TG + LDL	37(78.7)	10(21.3)	57.4	47(11.6)
Four abnormalities				
TC + TG + HDL +LDL	16(66.7)	8(33.3)	33.4	24(5.9)
TC + TG + HDL +LDL+TC/HDL ratio	16(66.7)	8(33.3)	33.4	24(5.9)
Dyslipidemia	213 (70.8)	88(29.2)	41.6	301(74.1)

TC: Total cholesterol, **TG:** Triglycerides, **HDL:** High-density lipoprotein, **LDL:** Low-density lipoprotein

Table 3: Frequency of dyslipidemia in HIV infected patients

4. Discussion

This study established that 74.1% of HIV-positive patients on HAART at Machakos Level V Hospital exhibited dyslipidemia. This finding aligns with studies in Kenya [13,16]. Ethiopia , and Cameroon, which report prevalence rates of 60–80% [17]. The consistently high burden of lipid abnormalities among HAART users across Africa reflects the dual influence of prolonged therapy and emerging non-communicable disease risks in this population [18]. Patients on protease inhibitor (PI) regimens had significantly higher mean TC and LDL-C levels and elevated TC/HDL-C ratios compared to those on NNRTI or INSTI regimens. The TC/HDL-C ratio was a significant marker of atherogenic risk ($p = 0.046$). These findings support earlier observations by Taramasso et al., Ji et al. , and Su et al., who linked PI therapy with elevated cholesterol and triglycerides due to hepatic lipid dysregulation [19, 20].

Biochemically, PIs disrupt lipid homeostasis by inhibiting SREBPs and LDL receptor recycling, increasing VLDL secretion and TG synthesis. They also reduce peripheral lipolysis, promote insulin resistance, and induce lipodystrophy. These mechanisms collectively explain the high dyslipidemia prevalence observed in PI users. Conversely, INSTI-based regimensparticularly dolutegravir-based combinationsshowed more favorable lipid patterns, consistent with Bahar et al. and other multicenter studies that found INSTIs metabolically safer. Although differences by sex and age were not statistically significant, dyslipidemia tended to be higher among males and older adults (≥60 years), consistent with reports by Lloyd-Jones et al.. Obesity and hypertension were also posi-

tively associated, suggesting clustering of CVD risk factors in this population. The strong link between hypertension and dyslipidemia ($p = 0.044$) highlights the importance of integrated metabolic screening in HIV care.

From a clinical chemistry perspective, these results emphasize the importance of routine lipid monitoring for patients on HAART, particularly those on PI-based regimens. Accurate lipid analysis requires adherence to pre-analytical standards (e.g., fasting samples, proper centrifugation) and validated enzymatic colorimetric methods. Laboratories must maintain rigorous internal and external quality control, use traceable calibrators, and adhere to standardized reporting formats to ensure diagnostic accuracy. Result interpretation should not stop at numerical reporting. Laboratory professionals should flag atherogenic ratios and provide interpretive comments guiding clinicians toward patient-centered management. Integration of biochemical and clinical data enhances multidisciplinary decision-making and aligns with evidence-based care principles. This study also holds significance for medical laboratory education and capacity building. Training programs should integrate HAART-related metabolic case studies into clinical chemistry curricula to strengthen understanding of drug-lipid interactions. Laboratory trainees should be exposed to interpretive reporting, cardiovascular risk assessment, and communication skills to support clinical teams. Continuing Professional Development (CPD) workshops can further improve competence in lipid testing and result interpretation.

These educational efforts would not only improve diagnostic services but also foster critical thinking among laboratory professionals. Given Kenya's growing population of PLHIV on lifelong therapy, HAART-related dyslipidemia represents an emerging public health challenge. Integrating routine lipid testing into HIV programs could facilitate early identification of high-risk individuals and reduce long-term CVD burden. Public health interventions should emphasize lifestyle modification, periodic biochemical screening, and regimen adjustment where feasible.

In summary, the high dyslipidemia prevalence found underscores the metabolic toll of HAART, especially PI-based therapy. INSTI regimens appear more lipid-friendly, offering safer long-term options. Strengthening laboratory systems, ensuring analytical reliability, and embedding clinical interpretation in laboratory education are essential to addressing this biochemical complication of HIV therapy.

5. Conclusion and Recommendations

Dyslipidemia is prevalent among HIV-positive patients on HAART at Machakos Level V Hospital, particularly in those on PI-based regimens. The TC/HDL-C ratio serves as a practical indicator of atherogenic risk.

Recommendations:

1. Integrate regular lipid profiling into HIV management protocols.
2. Provide continuous laboratory staff training on lipid testing and interpretation.
3. Include HAART-induced metabolic monitoring in undergraduate and CPD curricula.
4. Conduct longitudinal research linking lipid changes with cardiovascular outcomes.

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Conflict of Interest and Funding

The authors declare no conflict of interest.

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