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Research Article

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Risk Factors Associated with Dyslipidemia and Cardiovascular Disease among People Living with HIV on HAART at Machakos Level V Hospital, Kenya

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Abstract

The introduction of highly active antiretroviral therapy (HAART) has transformed HIV infection into a manageable chronic condition. However, the long-term use of antiretrovirals has been associated with metabolic complications such as dyslipidemia and an increased risk of cardiovascular disease (CVD). These emerging comorbidities threaten to offset the gains achieved in HIV management, particularly in low-resource settings. This study aimed to determine the risk factors associated with dyslipidemia and cardiovascular disease among PLWHIV on HAART at Machakos Level V Hospital, Kenya. A hospital-based cross-sectional study employing systematic sampling was conducted among 406 HIV-positive adults on HAART. Blood samples were collected for lipid profile analysis, and the Framingham Risk Score was used to assess 10-year cardiovascular risk. The Framingham model incorporated seven parameters: total cholesterol, high-density lipoprotein cholesterol (HDL-C), systolic blood pressure, age, sex, duration on antiretrovirals, and cigarette smoking. Additional demographic and clinical data were obtained through structured questionnaires. Descriptive statistics summarized prevalence rates, while bivariable and multivariate analyses identified exposure variables significantly associated with dyslipidemia and cardiovascular risk ($p \leq 0.05$).

The overall prevalence of dyslipidemia was 74.1% ($n = 301$). Age ($p = 0.005$), elevated systolic blood pressure ($p = 0.049$), and hypertension ($p < 0.001$) were significantly associated with dyslipidemia. The total cholesterol-to-HDL ratio was significantly linked to antiretroviral regimen ($p < 0.05$), with patients using protease inhibitors being four times more likely to have an elevated ratio compared to those on non-nucleoside reverse transcriptase inhibitors (OR = 4.19, 95% CI: 1.03–17.02). According to the Framingham Risk Score, 71.2% of participants had low CVD risk, 18.5% moderate, 9.8% moderately high, and 0.5% high risk. Age, low HDL-C, smoking, high systolic pressure, gender, and duration on HAART were significantly associated with higher Framingham risk ($p \leq 0.001$). These findings demonstrate a high burden of dyslipidemia and considerable cardiovascular risk among individuals on HAART. Integrating lipid monitoring, blood pressure control, and lifestyle interventions into routine HIV care is essential to mitigate long-term metabolic and cardiovascular complications in this population.

Keywords: Dyslipidemia, Cardiovascular Disease, Framingham Risk Score, HAART, HIV

1. Introduction

The introduction of highly active antiretroviral therapy (HAART) has revolutionized the management of human immunodeficiency virus (HIV) infection, transforming what was once a fatal condition into a chronic, manageable disease. With improved access to treatment, the life expectancy of people living with HIV (PLHIV) has significantly increased worldwide. According to UNAIDS (2024), over 39 million people globally are living with HIV, and nearly 30 million are receiving antiretroviral therapy. While this represents a remarkable public health success, prolonged exposure to HAART has been associated with a growing burden of metabolic complications, notably dyslipidemia and cardiovascular disease

(CVD). These emerging comorbidities threaten to erode the gains achieved in HIV care, particularly in sub-Saharan Africa, where infectious and non-communicable diseases increasingly coexist.

HIV infection itself alters lipid metabolism through chronic immune activation, inflammation, and cytokine dysregulation. The virus downregulates lipoprotein lipase activity and impairs cholesterol transport, leading to increased triglycerides and decreased high-density lipoprotein cholesterol (HDL-C). However, the introduction of antiretroviral therapy has added a new layer of metabolic disturbance. Certain drug classes—especially protease inhibitors (PIs) and some non-nucleoside reverse transcriptase inhibitors (NNRTIs)—exacerbate lipid abnormalities by increasing total cholesterol, low-density lipoprotein cholesterol (LDL-C), and triglycerides while further lowering HDL-C. This constellation of changes, collectively termed HAART-induced dyslipidemia, contributes to accelerated atherosclerosis and heightened cardiovascular risk in this population.

Globally, the pattern of morbidity among PLHIV is shifting from opportunistic infections to chronic diseases such as hypertension, diabetes, and cardiovascular disorders. A growing body of evidence demonstrates that HIV-positive individuals on HAART experience higher rates of metabolic syndrome and CVD compared to the general population. A meta-analysis by Zhou et al. estimated that approximately 54% of PLHIV on antiretroviral therapy develop dyslipidemia, with variations based on regimen type, duration of therapy, and population characteristics. Cardiovascular risk among HIV patients has also been shown to be two- to three-fold higher than that of HIV-negative counterparts, underscoring the dual challenge of infection control and chronic disease prevention.

In sub-Saharan Africa, where nearly 70% of the global HIV burden resides, the situation is particularly complex. The region has made substantial progress in scaling up HAART coverage, but systematic screening and management of non-communicable comorbidities remain limited. Studies from Nigeria, Uganda, and South Africa have reported a high prevalence of dyslipidemia and elevated cardiovascular risk among patients receiving HAART, largely attributed to prolonged therapy, urbanization, and lifestyle factors. In Kenya, HIV prevalence remains significant, estimated at 4.5% in adults (Kenya Ministry of Health), with over 1.3 million individuals on HAART. While national guidelines emphasize routine monitoring of viral load and CD4 counts, lipid and cardiovascular risk assessments are rarely implemented as part of standard HIV care.

Dyslipidemia among PLHIV on HAART manifests as elevated total cholesterol, LDL-C, triglycerides, and reduced HDL-C levels. The resulting atherogenic profile predisposes patients to early-onset atherosclerosis and ischemic heart disease. Mechanistically, HAART-induced metabolic disturbances arise from mitochondrial toxicity, oxidative stress, and altered adipocyte differentiation. Protease inhibitors such as lopinavir and ritonavir interfere with lipid-regulating enzymes and transcription factors, leading to lipodystrophy and hyperlipidemia. NNRTIs like efavirenz, though less severe, have also been associated with increased total cholesterol and triglycerides. Furthermore, traditional CVD risk factors—including age, hypertension, obesity, cigarette smoking, and sedentary lifestyle—interact synergistically with HAART-related metabolic derangements, compounding cardiovascular risk.

The Framingham Risk Score (FRS) remains one of the most widely used predictive tools for assessing 10-year cardiovascular risk. It integrates multiple parameters such as age, sex, systolic blood pressure, smoking status, total cholesterol, HDL-C, and diabetes to estimate overall cardiovascular disease probability. Several studies have validated its applicability among HIV-infected populations, though emerging data suggest that conventional models may underestimate actual risk in PLHIV due to persistent inflammation and immune dysregulation. Nevertheless, FRS remains a practical and accessible tool for resource-limited settings, including Kenya, to identify high-risk individuals and guide preventive strategies.

Recent Kenyan studies have highlighted the metabolic consequences of HAART but remain limited in scope and generalizability. For instance, Oduor et al. reported that over 60% of HIV patients on HAART at Kenyatta National Hospital exhibited at least one lipid abnormality, with females and older adults more affected. However, few studies have simultaneously assessed both dyslipidemia and calculated cardiovascular risk using standardized models such as the Framingham Risk Score. Moreover, data from semi-urban and rural Kenyan populations remain sparse, yet these populations often have distinct sociodemographic and nutritional characteristics that influence metabolic health.

Machakos County, a semi-urban region in Eastern Kenya, has a high HAART uptake rate and represents a typical middle-income Kenyan population transitioning toward lifestyle-related health risks. Understanding the interplay between HIV, antiretroviral therapy, and cardiovascular risk in this setting is essential for developing context-specific prevention strategies. Assessing both biochemical and clinical determinants of dyslipidemia and CVD can inform local health policies and strengthen the integration of non-communicable disease (NCD) monitoring into HIV care programs.

Given the increasing longevity of people living with HIV and the expanding coverage of HAART, there is a pressing need to characterize metabolic and cardiovascular risk profiles in diverse Kenyan settings. Identifying modifiable risk factors can guide clinicians and policymakers in optimizing care protocols and reducing long-term morbidity. Therefore, this study aimed to determine the risk factors associated with dyslipidemia and cardiovascular disease among PLWHIV on HAART at Machakos Level V Hospital, Kenya. The findings provide crucial insights for integrating metabolic health surveillance into HIV management, supporting the shift toward holistic, patient-centered care that addresses both infectious and chronic disease risks.

2. Materials and Methods

2.1 Study Design and Setting

This was a cross-sectional study conducted at Machakos Level V Hospital, Kenya, between April 2022 to June 2022. Machakos Level V Hospital is a referral facility serving both urban and rural populations in Machakos County and neighboring regions. The hospital has a comprehensive HIV care and treatment center offering clinical, laboratory, and pharmaceutical services to over 10,000 registered PLWHIV. The setting was selected due to its large patient base, established HAART program, and accessibility to both urban and peri-urban communities.

2.2 Study population

The study population comprised HIV-positive adults receiving HAART at the facility. Patients aged 20 years and above, who had been on HAART for at least six months, were eligible. Pregnant women, critically ill patients, and those with incomplete clinical records were excluded to avoid confounding due to pregnancy-related metabolic changes or missing data.

2.3 Sample Size Determination

The sample size was calculated using Fisher's formula for cross-sectional studies, based on an estimated prevalence of dyslipidemia among HAART users of 59.9% (to maximize sample size), a 95% confidence interval, and a 5% margin of error. The minimum required sample was 384 participants. Allowing for non-response, a total of 406 participants were recruited, ensuring adequate statistical power.

2.4 Sampling Technique

Systematic random sampling was employed. The daily patient attendance list at the HIV clinic served as the sampling frame. A sampling interval was determined by dividing the expected daily patient flow by the number required per day to meet the target sample. Every nth patient meeting eligibility criteria was invited to participate until the sample size was achieved.

2.5 Data Collection Instruments

Data were collected using a structured, pretested questionnaire administered by trained research assistants. The questionnaire captured sociodemographic variables (age, sex, education, marital status), clinical information (duration on HAART, current regimen, comorbid conditions), lifestyle behaviors (smoking, alcohol use, physical activity), and anthropometric measurements.

2.6 Anthropometric and Clinical Measurements

Weight was measured using a calibrated digital scale with participants wearing light clothing and no shoes. Height was measured using a stadiometer, and BMI was calculated as weight (kg) divided by height in meters squared (m^2). BMI categories were defined according to WHO criteria: underweight (<18.5 kg/m^2), normal weight (18.5–24.9 kg/m^2), overweight (25–29.9 kg/m^2), and obese (≥30 kg/m^2). Blood pressure was measured using a mercury sphygmomanometer after participants had rested for at least five minutes. Hypertension was defined as systolic blood pressure ≥140 mmHg and/or diastolic ≥90 mmHg, or current use of antihypertensive medication.

2.7 Laboratory Analysis

Venous blood samples were collected after an overnight fast of 8–12 hours. Serum was separated and analyzed for lipid profiles, including total cholesterol (TC), triglycerides (TG), HDL-C, and LDL-C, using an automated clinical chemistry analyzer following standard protocols. Internal quality control was performed daily, and the laboratory was enrolled in an external quality assurance program to ensure reliability of results. Dyslipidemia was defined according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) guidelines as one or more of the following: TC ≥200 mg/dL, LDL-C ≥130 mg/dL, TG ≥150 mg/dL, or HDL-C <40 mg/dL for men and <50 mg/dL for women. Participants with at least one abnormal parameter were classified as dyslipidemic.

2.7 Data Management and Analysis

Data were checked for completeness and accuracy before entry, then cleaned, coded, and analyzed using the Statistical Package for the Social Sciences (SPSS) version 25.0. Descriptive statistics were used to summarize the data: categorical variables were presented as frequencies and percentages, while continuous variables were expressed as means and standard deviations (SD).

A bivariable analysis was first conducted to identify potential exposure variables associated with dyslipidemia and cardiovascular disease risk. Categorical variables were analyzed using the chi-square test, and continuous variables were compared using independent t-tests. Variables that showed an association with the outcome at a significance level of $p \leq 0.20$ in the bivariable analysis were subsequently included in a multivariate logistic regression model to control for possible confounding factors and determine independent predictors. Adjusted odds ratios (AORs) with corresponding 95% confidence intervals (CIs) were computed to estimate the strength and direction of associations between risk factors and the dependent variables (dyslipidemia and cardiovascular disease risk). Statistical significance was determined at the 95% confidence level with a p-value ≤ 0.05 considered significant.

2.8 Ethical Considerations

Ethical approval for this study was obtained from the Kenyatta University Ethical Review Committee (KU-ERC) (Ref: PKU/2402/11536). A research permit was also granted by the National Commission for Science, Technology, and Innovation (NACOSTI) (License No: NACOSTI/P/22/15859). Authorization to conduct the research within the facility was provided by the Machakos County Referral Hospital administration. Written informed consent was obtained from all participants prior to their enrollment in the study. Participation was entirely voluntary, and refusal to consent did not affect the provision of any medical or laboratory services. Confidentiality and anonymity were maintained throughout the research process. All participants received their laboratory results during subsequent follow-up visits at the Comprehensive Care Centre (CCC) for appropriate

clinical management and counseling.

3. Results

3.1 Sociodemographic and Clinical Characteristics

A total of 406 HIV-positive adults receiving highly active antiretroviral therapy (HAART) at Machakos Level V Hospital were included in the study. The study population was predominantly female, accounting for 290 (71.4%) participants, while males comprised 116 (28.6%). The median age of the respondents was 46 years (interquartile range [IQR]: 38–55 years). More than half of the participants, 224 (55.2%), were aged between 41 and 59 years, 100 (24.6%) were aged 25–40 years, 59 (14.5%) were 60 years and above, and 23 (5.7%) were aged 24 years or below. These findings indicate that the majority of patients on long-term HAART in this setting are middle-aged adults, reflecting the maturing HIV population in Kenya.

Educational attainment varied, with 180 (44.3%) of participants having completed primary education or below, 167 (41.1%) having secondary education, and 59 (14.5%) possessing tertiary-level qualifications. In terms of marital status, 217 (53.4%) were single and 189 (46.6%) were married. The predominance of single individuals may reflect the social effects of HIV infection, including stigma, widowhood, or marital dissolution.

The nutritional and anthropometric assessment revealed a median body mass index (BMI) of 24.4 kg/m² (IQR: 21.2–28.3). Nearly half of the participants, 197 (48.5%), had a normal BMI, while 27 (6.7%) were underweight, 114 (28.1%) were overweight, and 68 (16.7%) were obese. This distribution illustrates a double burden of malnutrition among HIV-positive individuals, characterized by coexistence of underweight and overweight, consistent with nutritional and metabolic shifts observed in populations undergoing long-term HAART.

Clinical assessment revealed that 62 (15.3%) of respondents had a previous history of hypertension, while 344 (84.7%) reported no such history. Additionally, 28 (6.9%) participants had a self-reported history of cardiovascular disease, whereas 378 (93.1%) did not. Cigarette smoking was uncommon in this cohort; only 22 (5.4%) participants reported a history of smoking compared to 384 (94.6%) who had never smoked. When categorized by blood pressure status, 337 (83%) had normal blood pressure, while 69 (17%) were hypertensive at the time of study.

The duration of HAART use among participants ranged from six months to twenty years, with a median duration of nine years (IQR: 5–13 years). The majority, 299 (73.6%), had been on therapy for more than five years, reflecting substantial long-term treatment exposure. Further classification by duration indicated that 90 (22.2%) of participants had been on HAART for less than five years, 149 (36.7%) for five to ten years, and 167 (41.1%) for more than ten years. This extended treatment duration provides valuable insight into the long-term metabolic effects of antiretroviral therapy.

Regarding treatment regimens, 378 (93.1%) of the participants were on first-line HAART, while 28 (6.9%) were on second-line therapy. The most frequently prescribed regimen was tenofovir/lamivudine/dolutegravir (TDF/3TC/DTG), used by 364 (89.7%) of participants. Smaller proportions were on zidovudine/lamivudine/atazanavir-ritonavir (AZT/3TC/ATV/r) at 13 (3.2%), tenofovir/lamivudine/efavirenz (TDF/3TC/EFV) at 6 (1.5%), abacavir/lamivudine/dolutegravir (ABC/3TC/DTG) at 3 (0.7%), and abacavir/lamivudine/atazanavir-ritonavir (ABC/3TC/ATV/r) at 2 (0.5%).

Variable	Category	Frequency (n)	Percent (%)
Gender	Male	116	28.6
	Female	290	71.4
Age (Median, IQR)	46 (38–55) years		
	≤ 24 years	23	5.7
	25–40 years	100	24.6
	41–59 years	224	55.2
	≥ 60 years	59	14.5
Marital status	Single	217	53.4
	Married	189	46.6
Education level	Primary or lower	180	44.3
	Secondary	167	41.1
	Tertiary	59	14.5
Occupation	Unemployed	76	18.7
	Employed	68	16.7
	Self-employed	262	64.5

Body Mass Index (BMI) (Median, IQR)	24.4 (21.2–28.3) kg/m²		
	Underweight	27	6.7
	Normal	197	48.5
	Overweight	114	28.1
	Obese	68	16.7
Cigarette smoking	Yes	22	5.4
	No	384	94.6
History of hypertension	Yes	62	15.3
	No	344	84.7
History of cardiovascular disease	Yes	28	6.9
	No	378	93.1
Systolic blood pressure	≥140 mmHg	69	17.0
	<140 mmHg	337	83.0
Diastolic blood pressure	≥90 mmHg	69	17.0
	<90 mmHg	337	83.0
Duration on HAART (Median, IQR)	9 (5–13) years		
	<5 years	90	22.2
	5–10 years	149	36.7
	>10 years	167	41.1
HAART treatment line	First line	377	92.9
	Second line	29	7.1
HAART regimen type	ABC/3TC/ATV/r	2	0.5
	ABC/3TC/DTG	3	0.7
	AZT/3TC/ATV/r	13	3.2
	AZT/3TC/DTG	1	0.2
	AZT/3TC/LPV/r	1	0.2
	AZT/3TC/NVP	1	0.2
	AZT3TC/LPV/r	1	0.2
	D4T/3TC/NVP	1	0.2
	TDF/3TC/ATV/r	12	3.0
	TDF/3TC/DTG	364	89.7
	TDF/3TC/EFV	6	1.5
	TDF/3TC/NVP	1	0.2

Table 1: Sociodemographic and Clinical Characteristics of PLWHIV on HAART at Machakos Level V Hospital (n = 406)

IQR: Interquartile range, BMI: Body mass index, **ARV**: Antiretroviral, **IQR**: Interquartile range, **HAART**: Highly active antiretroviral therapy, **ABC**: Abacavir, **3TC**: Lamivudine, **ATV**/r: Atazanavir/ritonavir, **DTG**: Dolutegavir, **AZT**: Azidothymidine, **LPV/r:** Lopinavir/ritonavir, **D4T**: Stavudine, **NVP**: Nevirapine, **TDF**: Tenofovir disoproxil fumarate, **EFV**: Efavirenz

3.2 Risk Factors Associated with Dyslipidemia Among PLWHIV on HAART

The risk factors associated with dyslipidemia among HIV-positive individuals receiving HAART were analyzed, and the results are summarized in Table 2. Multivariate logistic regression identified several variables that were significantly associated with dyslipidemia. Participants aged 60 years and above were approximately four times more likely to have dyslipidemia compared to those aged 24 years and below (adjusted odds ratio [AOR] = 3.61, 95% confidence interval [CI]: 1.21–9.00, p = 0.005). Similarly, participants with elevated systolic blood pressure (≥140 mmHg) were about twice as likely to present with dyslipidemia compared to those with normal systolic pressure (<140 mmHg) (AOR = 2.41, 95% CI: 1.21–5.78, p = 0.049). A significant relationship was also observed between hypertension history and dyslipidemia. Participants with a previous history of hypertension were three times more likely to have dyslipidemia compared to those without such a history (AOR = 3.11, 95% CI: 1.22–7.81, p < 0.001).

Risk factors	Dyslipidemia		OR (95%CI)	P-value	AOR (95%CI)	P-value
	Yes n (%)	No n (%)				
Gender						
Male	88(29.2)	28(26.7)	1.14(0.69 - 1.87)	0.616		
Female	213(70.8)	77(73.3)	Ref			
Age						
≤24 years	13(4.3)	10(9.5)	Ref		Ref	
25 - 40 years	78(25.9)	22(21.0)	1.47(0.73 - 2.95)	0.284	1.37(0.67 - 2.82)	0.387
41 - 59 years	163(54.2)	61(58.1)	1.11(0.50 - 2.44)	0.805	0.97(0.43 - 2.21)	0.946
≥60 years	47(15.6)	12(11.4)	3.01(1.07 - 8.52)	0.038	3.61(1.21 - 9.00)	0.005
BMI						
Normal	145(48.2)	52(49.5)	Ref			
Underweight	17(5.6)	10(9.5)	1.38(0.71 - 2.70)	0.341		
Overweight	85(28.2)	29(27.6)	2.27(0.85 - 6.03)	0.211		
Obese	54(17.9)	14(13.3)	1.32(0.64 - 2.71)	0.457		
Systolic						
≥140 mmHg	62(20.6)	8(7.6)	3.15(1.45 - 6.82)	0.002	2.41(1.21 - 5.78)	0.049
<140 mmHg	239(79.4)	97(92.4)	Ref		Ref	
Diastolic						
≥90 mmHg	58(19.3)	11(10.5)	2.04(1.03 - 4.06)	0.049	1.20(0.55 - 2.60)	0.645
<90 mmHg	243(80.7)	94(89.5)	Ref		Ref	
Hypertension						
Yes	55(18.3)	10(9.5)	2.1(1.04 - 4.34)	<0.001	3.11(1.22 - 7.81)	<0.001
No	246(81.7)	95(90.5)	Ref			
Cigarette smoking						
Yes	15(5.0)	7(6.7)	0.73(0.29 - 1.85)	0.616		
No	286(95.0)	98(93.3)	Ref			
History of CVD						
Yes	20(6.6)	8(7.6)	0.86(0.37 - 2.02)	0.823		
No	281(93.4)	97(92.4)	Ref			
Treatment line						
First	278(92.4)	99(94.3)	0.73(0.29 – 1.85)	0.166		
Second	23(7.6)	6(5.7)	Ref			

Table 2: Risk Factors Associated with Dyslipidemia Among PLWHIV on HAART (N = 406)

Reference category (Ref) = category of the independent variable which each other category is compared. **OR**: Odds ratio, **AOR**: Adjusted odds ratio, **BMI**: Body mass index, **CVD**: Cardiovascular disease

3.3 Risk Factors Associated with Cardiovascular Disease among PLWHIV on HAART

The risk factors associated with cardiovascular disease (CVD) among HIV-positive individuals receiving HAART were analyzed, and the results are summarized in Table 3.

Male participants were found to have a markedly higher likelihood of developing CVD compared to female participants (AOR = 5.24, 95% CI: 2.43–11.28, $p < 0.001$). Age was also a significant determinant of CVD risk. Participants aged 25–40 years (AOR = 3.21, 95% CI: 1.11–8.31, $p < 0.001$), 41–59 years (AOR = 5.11, 95% CI: 3.22–11.54, $p < 0.001$), and those aged 60 years and above (AOR = 6.60, 95% CI: 2.95–14.76, $p < 0.001$) demonstrated a progressively higher risk of CVD compared to those aged 24 years and below.

Participants with elevated systolic blood pressure (≥140 mmHg) were nine times more likely to have CVD compared to those with

lower systolic pressure (<140 mmHg) (AOR = 8.55, 95% CI: 3.49–20.96, p < 0.001). Likewise, those with a history of hypertension exhibited a significantly elevated risk of CVD (AOR = 9.73, 95% CI: 3.45–27.41, p < 0.001). A very strong association was also noted between CVD and cigarette smoking (AOR = 28.13, 95% CI: 5.90–56.11, p < 0.001), as well as a prior history of cardiovascular disease (AOR = 20.28, 95% CI: 6.07–67.76, p < 0.001).

	Risk					
	High risk n (%)	**Low risk n (%)**	**COR (95%CI)**	**P-value**	**AOR (95%CI)**	**P-value**
Gender						
Male	57(48.7)	59(20.4)	3.71(2.33 - 5.88)	<0.001	5.24(2.43 - 11.28)	**<0.001**
Female	60(51.3)	230(79.6)	Ref		Ref	
Age						
≤24 years	1(0.9)	22(7.6)	Ref		Ref	
25 - 40 years	8(6.8)	92(31.8)	3.62(2.0 - 6.60)	<0.001	3.21(1.11 - 8.31	**<0.001**
41 - 59 years	71(60.7)	153(52.9)	19.34(7.91 - 47.32)	<0.001	5.11(3.22 - 11.54)	**<0.001**
≥60 years	37(31.6)	22(7.6)	37.0(4.66 - 293.90)	0.001	6.60(2.95 - 14.76)	**<0.001**
BMI						
Normal	55(47.0)	142(49.1)	Ref			
Underweight	7(6.0)	20(6.9)	1.24(0.68 - 2.24)	0.488		
Overweight	33(28.2)	81(28.0)	1.37(0.50 - 3.71)	0.54		
Obese	22(18.8)	46(15.9)	1.17(0.61 - 2.25)	0.629		
Systolic						
≥140 mmHg	51(43.6)	19(6.6)	10.98(6.08 - 19.84)	<0.001	8.55(3.49 - 20.96)	**<0.001**
<140 mmHg	66(56.4)	270(93.4)	Ref		Ref	
Diastolic						
≥90 mmHg	39(33.3)	30(10.4)	4.32(2.52 - 7.40)	<0.001	1.09(0.41 -2.92)	**0.861**
<90 mmHg	78(66.7)	259(89.6)	Ref		Ref	
History of hypertension						
Yes	46(39.3)	19(6.6)	9.21(5.08 - 16.69)	<0.001	9.73(3.45 - 27.41)	**<0.001**
No	71(60.7)	270(93.4)	Ref		Ref	
History of cigarette smoking						
Yes	19(16.2)	3(1.0)	18.48(5.35 - 63.81)	<0.001	28.13(5.90 - 56.11)	**<0.001**
No	98(83.8)	286(99.0)	Ref		Ref	
History of CVD						
Yes	18(15.4)	10(3.5)	5.07(2.27 - 11.36)	<0.001	20.28(6.07 - 67.76)	**<0.001**
No	99(84.6)	279(96.5)	Ref		Ref	
HAART regimen						
INSTI	108(92.3)	260(90.0)	Ref			
PI	7(6.0)	22(7.6)	0.69(0.14 - 3.36)	0.644		
NNRTI	2(1.7)	7(2.4)	0.90(0.15 - 5.36)	0.906		

TC/HDL ratio						
High	58(49.6)	79(27.3)	2.61(1.67 - 4.08)	<0.001	3.14(1.41 - 9.90)	**<0.001**
Normal	59(50.4)	210(72.7)	Ref		Ref	
TG/HDL						
High	100(85.5)	208(72.0)	2.29(1.29 - 4.07)	0.005	1.76(0.71 - 4.36)	**0.221**
Normal	17(14.5)	81(28.0)	Ref		Ref	
LDL/HDL ratio						
High	46(39.3)	67(23.2)	2.15(1.35 - 3.40)	0.001	1.25(0.39 - 4.07)	**0.221**
Normal	71(60.7)	222(76.8)	Ref		Ref	

Table 3: The risk factors linked with cardiovascular disease among HIV positive patients on HAART (N = 406)

Reference category (Ref) = category of the independent variable which each other category is compared. **Low risk** = participants having low risk of CVD according to FRS analysis.

High risk = participants having moderate, moderately high and high risk of CVD according to FRS. **CVD**: Cardiovascular disease, **BMI**: Body mass index, **HAART**: Highly active antiretroviral therapy, **PI**: Protease inhibitor, **INSTI**: Intergrase inhibitor, **NNRTI**: Non-nucleoside reverse transcriptase inhibitor, **COR**: Crudes odds ratio, **AOR**: Adjusted odds ratio, **TC**: Total cholesterol, **TG**: Triglycerides, **HDL**: High density lipoprotein, **LDL**: Low density lipoprotein

4. Discussion

This study found a high prevalence of dyslipidemia (74.1%) among HIV-positive individuals on HAART and a substantial proportion of participants with moderate to high ten-year cardiovascular risk based on the Framingham Risk Score (FRS). Factors independently associated with dyslipidemia included older age, elevated systolic blood pressure, and a history of hypertension. For cardiovascular disease (CVD) risk, male sex, increasing age, elevated systolic blood pressure, a history of hypertension, cigarette smoking, and prior cardiovascular disease were significant predictors. These findings highlight the convergence of metabolic and hemodynamic risk factors among people living with HIV (PLHIV) on long-term antiretroviral therapy.

The prevalence of dyslipidemia in this study aligns with other reports from sub-Saharan Africa, which have documented lipid abnormalities ranging from 40% to 75% among patients on HAART [1-3]. These results suggest that metabolic complications remain an important concern in the era of effective viral suppression. Differences in prevalence across studies may reflect variation in ART regimen type, duration on therapy, population age structure, and dietary or lifestyle factors.

Age was a consistent predictor of both dyslipidemia and elevated cardiovascular risk. Participants aged 60 years and above were four times more likely to have dyslipidemia and more than six times more likely to have elevated CVD risk compared to younger counterparts. This finding agrees with studies in Ethiopia and Kenya, where dyslipidemia prevalence increased with age [1,3]. Similarly, Najafipour et al. reported that lipid abnormalities rose progressively with advancing age, particularly among women, while Otsuka et al. and Woldu et al. demonstrated that age-related vascular stiffness and declining metabolic rate predispose older adults to hypertension and atherosclerotic changes [4-6]. Collectively, these observations reinforce that age is a key determinant of metabolic and cardiovascular comorbidities among PLHIV.

Elevated systolic blood pressure and a history of hypertension were significantly associated with dyslipidemia in this study, consistent with findings by Opoku et al. and Otsuka et al., who found that lipid abnormalities predicted the onset of hypertension [5,7]. Dyslipidemia may impair endothelial function and baroreceptor sensitivity, increase oxidative stress, and reduce arterial compliance, thereby promoting hypertension [8]. Hypertension and dyslipidemia are known to act synergistically in accelerating atherosclerosis and increasing cardiovascular risk [9].

The observed relationship between hypertension and dyslipidemia may also reflect shared lifestyle and pharmacologic determinants. In particular, HAART regimens, especially those including protease inhibitors (PIs), are known to induce insulin resistance and promote lipid synthesis [10]. Our study found that the total cholesterol to HDL ratio was significantly associated with antiretroviral regimen type, with PI-based regimens conferring higher risk than non-nucleoside reverse transcriptase inhibitor (NNRTI)–based regimens. This aligns with studies showing that PIs and efavirenz adversely affect lipid metabolism, whereas integrase strand transfer inhibitors (INSTIs) such as dolutegravir (DTG) are relatively lipid-neutral but have been linked to weight gain and emerging cardiometabolic concerns [11,12].

In the current study, no significant associations were observed between dyslipidemia and sex, BMI, cigarette smoking, or HAART duration. This may be due to the predominance of normal BMI and low smoking prevalence in our cohort. Similar findings were reported by Kemal et al., who found no significant association between dyslipidemia and gender, smoking, or HAART duration [13]. In contrast, Achila et al. reported that sex, smoking, BMI, and HAART duration were significant predictors of dyslipidemia,

likely due to higher rates of obesity and tobacco use in their population [14].

Male sex was a strong independent predictor of CVD risk in this study. The likelihood of male participants having CVD was five times higher than that of females, consistent with previous research showing higher CVD risk among men [6,15]. The increased risk in men could be attributed to higher rates of smoking and dyslipidemia, as well as hormonal differences that protect premenopausal women from atherosclerosis through estrogen's cardioprotective effects [16]. Studies from South Africa and Nigeria similarly reported that men exhibited higher FRS scores and higher rates of hypertension than women [17,18].

Age remained a strong predictor of CVD risk, with participants aged 41–59 years and ≥60 years showing more than five- and sixfold increases in risk, respectively. Comparable trends were reported by Maas and Appelman and Woldu et al., who found that CVD risk increases steadily with age due to lipid accumulation, endothelial dysfunction, and the cumulative effects of HAART exposure [6,19]. HIV infection itself accelerates vascular aging through chronic inflammation and immune activation [20,21].

Hypertension and systolic blood pressure were among the strongest predictors of CVD in this study. Patients with elevated systolic pressure (≥140 mmHg) were nine times more likely to develop CVD, while those with a history of hypertension had nearly a tenfold increased risk. This finding corresponds with those of Wu et al. and Niklas et al., who identified hypertension as the leading modifiable risk factor for CVD in both HIV-positive and general populations [22,23]. Given the high overlap between hypertension and dyslipidemia, multifactorial interventions addressing both conditions are essential to reduce CVD risk. Smoking was also a significant risk factor for CVD, with smokers exhibiting a 28-fold increased risk compared to nonsmokers. Tobacco use contributes to atherogenesis through endothelial injury, inflammation, and pro-thrombotic mechanisms [24]. Similar associations have been reported globally [25,26]. Smoking cessation has been shown to markedly reduce myocardial infarction and stroke risk, underscoring the need to incorporate tobacco cessation programs into HIV care [27].

A history of cardiovascular disease was another strong predictor of elevated CVD risk. This association is consistent with the genetic and metabolic predisposition to recurrent CVD among affected individuals [28]. Ruamtawee et al. also reported that a family history of CVD increased cardiovascular risk more than twentyfold among HIV-positive individuals [29]. Although family history was not directly assessed in the current study, these findings highlight the importance of genetic predisposition and shared behavioral risks. The coexistence of dyslipidemia, hypertension, and smoking within this population creates a high-risk profile for future cardiovascular events. Our findings are consistent with Ghamri et al., who reported that 61.2% of hypertensive patients had dyslipidemia, and Grand et al., who identified dyslipidemia, smoking, and hypertension as the most common concomitant risk factors for CVD [9,25]. The clustering of these factors among PLHIV supports integrated care models that address both infectious and non-communicable diseases. Finally, our results reaffirm the importance of continuous cardiovascular risk assessment among PLHIV. The Framingham Risk Score, though validated in general populations, may underestimate absolute CVD risk in HIV cohorts since it does not account for HIV-specific inflammatory or treatment-related factors [30]. Nevertheless, it remains a useful tool for identifying high-risk individuals in resource-limited settings.

5. Strengths and Limitations

This study has several notable strengths. It involved a relatively large sample size of 406 participants, enhancing the statistical power and representativeness of the findings. The use of systematic sampling minimized selection bias, and standardized procedures were followed for biochemical and clinical measurements, including fasting lipid profiles and blood pressure determination. The incorporation of the Framingham Risk Score (FRS) provided a practical, evidence-based framework for estimating ten-year cardiovascular risk among people living with HIV (PLHIV). Furthermore, the study contributes valuable local data on dyslipidemia and cardiovascular risk in Kenya, where limited evidence currently exists on the metabolic consequences of long-term antiretroviral therapy (HAART).

However, certain limitations should be acknowledged. The cross-sectional study design limits causal inference; thus, associations identified between risk factors and outcomes cannot establish temporal relationships. The Framingham Risk Score, while widely used, was developed for non-HIV populations and may underestimate absolute cardiovascular risk in PLHIV, who experience additional HIV- and treatment-related inflammatory effects. Behavioral variables such as cigarette smoking and physical activity were self-reported and may be prone to recall or social desirability bias. Moreover, this was a single-center study conducted at Machakos Level V Hospital, which may limit the generalizability of findings to other populations with different demographic or clinical characteristics. Despite these limitations, the study provides robust baseline evidence on the interplay between dyslipidemia, hypertension, and cardiovascular risk among PLHIV in Kenya. These findings underscore the need for continuous surveillance, integration of metabolic screening into HIV care, and the development of tailored cardiovascular prevention strategies for this high-risk population.

6. Conclusion

This study demonstrated that dyslipidemia and cardiovascular disease (CVD) risk remain major emerging comorbidities among people living with HIV (PLHIV) on long-term highly active antiretroviral therapy (HAART). The overall prevalence of dyslipidemia was high, affecting nearly three-quarters of the study participants, while almost one-third had moderate to high ten-year cardiovascular risk based on the Framingham Risk Score. Age, systolic blood pressure, and a history of hypertension were independent predictors of dyslipidemia, whereas male gender,

advanced age, elevated systolic blood pressure, hypertension, smoking, and prior cardiovascular disease were significant determinants of CVD risk.

The findings emphasize the interplay between traditional cardiovascular risk factors and HIV/HAART-related metabolic alterations. As HIV care continues to evolve toward chronic disease management, addressing cardiovascular risk must become a core component of comprehensive care. The clustering of dyslipidemia, hypertension, and smoking in this population underscores the need for early detection and targeted intervention. This study contributes to growing regional evidence on the cardiometabolic consequences of HAART, reinforcing that sustained viral suppression does not eliminate the need for continuous cardiovascular monitoring. The results also highlight the suitability of the Framingham Risk Score as a practical risk assessment tool in resource-limited settings, even though it may underestimate absolute CVD risk in PLHIV [31-35].

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