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Research Article

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Acute Coronary Syndrome Profile in Human Immunodeficiency Virus (Hiv) Patients at A Tertiary Hospital in South India: A Retrospective 10-Year Study

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Abstract

Individuals with HIV are increasingly affected by non-communicable diseases such as coronary artery disease. 6.5-15% of mortality in this population is attributable to cardiovascular disease. The angiographic pattern of coronary artery disease (CAD) in patients with HIV is unknown Data from high-income countries show a different risk factor profile. However, such data is lacking in the Indian population. Therefore, we sought to describe the angiographic features and burden of CAD in patients with HIV compared to those without HIV infection.

Objective: To describe the angiographic features and burden of CAD in patients with HIV compared to those without HIV infection.

Methodology: We conducted a single centre retrospective case-control study of all patients presenting to KLES Dr Prabhakar Kore Hospital, Belagavi, Karnataka, India, from January 2013 to January of 2023with ACS. KLES Dr Prabhakar Kore Hospital is the largest private teaching hospital in the northern Karnataka state of India.

Results: results: This is a retrospective, single-centre study comparing 76 patients with HIV infection who underwent coronary angiography between 2013 and 2023 with 100 control patients without HIV infection matched for age (±3 years), gender and BMI. Gensini score was used to assess the severity of the lesions in both groups. The median age for the populations was 50.8 years in the HIV group and 53.9 years in the non-HIV group. Patients with HIV were more likely to present with ST-segment elevation myocardial infarction (STEMI), lower LDL levels and high Triglyceride levels. However, lesion severity was lesser compared to the non-HIV group.

Conclusion: Conclusions: While HIV+ patients were more likely to present with STEMI, angiographic analysis revealed less severe lesions. This suggests alterations in the genesis and progression of atherosclerosis in this clinical setting.

Keywords: Acute coronary syndrome, HIV infection, coronary intervention, etc

Introduction

The worldwide estimated prevalence of HIV at the end of 2018 was 37.9 million individuals, and the incidence was 1.7 million the same year [1]. After introducing antiretroviral therapy (ART) in 1996, the spectrum of CVD shifted from dilated cardiomyopathies, pericardial effusions, conduction

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system abnormalities, pulmonary hypertension and neoplastic infiltration to atherosclerosis [2, 3]. Manifestations of atherosclerosis, specifically myocardial infarction and stroke, become prominent, and heart failure, atrial fibrillation and sudden cardiac death have emerged. Atherosclerosis in these individuals occurs a decade earlier when compared to their non-infected counterparts. This suggests an alternate pathogenesis in the HIV population. The relative risk of patients living with HIV is 2.16 (95% CI) compared to uninfected counterparts [4]. CAD accounts for 8-22 % of deaths among the HIV population [5].

Many studies from the West describe the prevalence of HIV in ACS, risk factors and angiographic profiles in these patients. However, there is limited data in south-east Asia. Most studies demonstrated that HIV patients with ACS tend to be a decade younger, predominantly male and more likely to be smokers. The prevalence of traditional risk factors is more in the HIV population receiving ART as compared with non-infected people. The increased incidence of ACS in the HIV population suggests additional mechanisms such as ongoing inflammation, immune activation and ART-induced metabolic derangements, mainly dyslipidaemia. Chronic inflammation associated with HIV tends to give rise to unstable plaques. Association of C-reactive protein (CRP), IL-6 and D-dimer contribute to pro-inflammatory and procoagulant state and strongly predict CV events and all-cause mortality in individuals with HIV infection [6]. The clinical presentation of HIV patients with CAD seems similar to the non-infected population, including both ACS and Stable angina. ACS is the primary clinical manifestation, in which ST-elevation myocardial infarction STEMI is the most common subtype. Among the non-infected population, NSTEMI and unstable angina are more common [7, 8]. CAD is no longer a disease that affects high-income countries. As per the results of the Global Burden of Disease study, the age-standardized CVD death rate of 272 per 100000 population in India is much higher than that of global average of 235 [9]. HIV prevalence in India is 0.21%. Of the total HIV population of the country, 55 % is made up of four south Indian states, including Andhra Pradesh, Karnataka, Tamil Nadu and Maharashtra. These HIV patients are at an increased risk from two epidemics which affect the quality of life and mortality [10]. We conducted a ten-year cross-sectional retrospective case-control study in a tertiary care centre in south India presenting with ACS to describe the risk factors, patient and angiographic profile in patients with HIV compared to the uninfected population presenting with ACS.

Methods

We conducted a single centre retrospective case-control study of all patients presenting to KLES Dr Prabhakar Kore Hospital, Belagavi, Karnataka, India, from January 2013 to January of 2023 with ACS. KLES Dr Prabhakar Kore Hospital is the largest private teaching hospital in the northern Karnataka state of India. The hospital caters to populations belonging to all social and economic backgrounds. Patients were identified using a medical record database (MRD) with a diagnosis of acute coronary syndrome, including STEMI, NSTEMI and UA. The diagnosis of myocardial infarction (MI) at our centre was made according to the Fourth Universal Definition [11]: acute myocardial injury with clinical evidence of acute myocardial ischaemia and a rise of cardiac troponin levels with at least one value >99th percentile of the upper reference limit and at least one of the following: symptoms of myocardial ischaemia, new ischaemic changes on the ECG, pathological Q-waves on the ECG, imaging evidence of new loss of myocardium or new localised wall motion abnormality in a pattern indicative of ischaemic causes, presence of a coronary thrombus by angiography+. UA is diagnosed by the presence of ischaemic symptoms suggestive of ACS, without elevated biomarkers or ECG changes suggestive of infarction +. The diagnosis of ACS was confirmed by reviewing discharge summaries and cross-checking the admission records, electrocardiograms (ECGs), troponin levels, echocardiograms and angiogram reports of each patient. Patients with repeated ACS events within the year were only counted once, and their first ACS event within the year was described.

We extracted the HIV status of all patients with a clinical diagnosis of ACS from MRD records and physician case records. Patients who did not undergo an angiogram or were admitted for heart failure and stabilization were excluded from the study. HIV-positive patients were classified as CD4 cell count <200 cells/mm3, 201-499 cells/mm3 and > 500 cells/mm3 as per CDC guidelines [12]. In the case-control analysis of our study, we compared the CAD risk factors, lipid profile, ACS type and angiographic severity of the lesion. In order to do this, we selected one hundred HIV-negative patients (confirmed HIV-negative test results). The HIV-negative patients were chosen by convenience sampling. Our method allowed for matching baseline characteristics such as age, gender and body mass index (BMI). In addition, we extracted the following CAD variables from their medical records: comorbidities (hypertension, dyslipidaemia, diabetes mellitus), family history of CAD, social risk factors (tobacco-smoking, body mass index > 25 kg/m2, ethanol-misuse), the type of ACS (STEMI, NSTEMI and UA), the culprit coronary artery, the region of myocardium involved, single- or multivessel coronary artery disease, echocardiographic findings, as well as their serum creatinine concentration on presentation. We identified medical comorbidities using discharge summary diagnoses, prescription data and medical records from the MRD. Diagnosis of hypertension requires at least three blood pressure readings ≥140/90 at least two days apart. Diagnosis of diabetes mellitus is made based on the following: symptoms of hyperglycaemia or metabolic decompensation with any one single test that confirms a random plasma glucose ≥200mg/dl, fasting plasma glucose

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≥126 mg/dl, HbA1c ≥ 6.5% or 2-h post-load glucose ≥200mg/dl. Dyslipidaemia is diagnosed based on the finding of abnormal serum lipid levels.

The data was tabulated on excel spreadsheet and master chart was prepared (Annexure III). The data was analysed using statistical software SPSS version 20.0. Continuous variables were analyzed for normality by the Shapiro Wilk test. The data was expressed in terms of mean±standard deviation (SD) for the data that followed normal distribution and the data which followed skewed distribution was expressed as median and interquartile range (IQR). The Comparison of categorical data was done using Chi-square test or Fisher's exact test. The comparison of median Apo B to A ratio was done using Wilcoxon Ranksum test. Spearman's correlation coefficient (ρ) was used to determine correlation between Genisini score and CD4 count. Kruskal Wallis test was used to compare more than two median values. At 95% confidence interval (CI), a probability value ('p' value) of less than or equal to 0.050 was considered to be statistically significant.

Statistical Analysis

The data was tabulated on excel spreadsheet and master chart was prepared (Annexure III). The data was analysed using statistical software SPSS version 20.0. Continuous variables were analyzed for normality by the Shapiro Wilk test. The data was expressed in terms of mean±standard deviation (SD) for the data that followed normal distribution and the data which followed skewed distribution was expressed as median and interquartile range (IQR). The Comparison of categorical data was done using Chi-square test or Fisher's exact test. The comparison of median Apo B to A ratio was done using Wilcoxon Ranksum test. Spearman's correlation coefficient (ρ) was used to determine correlation between Genisini score and CD4 count. Kruskal Wallis test was used to compare more than two median values. At 95% confidence interval (CI), a probability value ('p' value) of less than or equal to 0.050 was considered to be statistically significant.

Results

Prevalence of HIV in ACS

We identified 18000 patients who presented to our centre from January 2013 to January 2023 with the diagnosis of ACS, of which 76 patients were HIV-positive to give a 10-year prevalence of 0.003%

Baseline Characteristics

Seventy-six HIV-positive ACS patients were compared with 100 randomly selected HIV-negative ACS patients, with the following findings: The HIV-positive and non-HIV patients had a mean age of 50.8 years and 53.9 years, respectively. Patients were predominantly male in both groups (HIV- 82.89% and non-HIV- 74%). HIV patients were less likely to have diabetes mellitus (21.05%vs.57%, p<0.01) and hypertension (32.89% vs. 48%,p0.031). In addition, they were more likely to be smokers (32.89% vs 7%,p0.001). Patients with HIV had lower LDL levels (93.66 ± 21.50 mg/dl vs 107.44±43.09 mg/dl, p0.006) and high triglyceride levels (197.82±71.59 vs 173.95±138.57,p<0.001). Total cholesterol levels were higher in the HIV group (327±69.88 vs 176.62±48.74, p 0.001). There was no difference in HDL noted between the groups.

Single vessel disease was more common in the HIV group than in the non-HIV group (46.05% vs 41%, p0.015), while DVD was common in the non-HIV group (35% vs 21.05%, p 0.015). The groups for TVD remained the same. There was no difference in the region of myocardium involved between the two groups (p 0.283). The severity of the lesion,

Table 1: Clinical profile of the patients with HIV

Variables	Mean (n=77)		Median		Range		p value
	Mean	SD	Median	IQR	Minimum	Maximum	
Age (Years)	50.83	9.47	50.5	13.5	34	76	**0.032**
Body mass index	27.62	3.95	27	5.15	21	38.67	0.051
Low density lipoprotein (mg/dL)	93.66	21.5	91.5	32.75	54	148	0.17
Triglycerides (mg/dL)	197.82	71.59	197	89.75	46	394	0.2
High density lipoprotein (mg/dL)	35.89	8	34.5	10	18	59	0.096
Total cholesterol (mg/dL)	327.37	69.88	322	75.5	190	489	0.2
Total cholesterol of HDL ratio	9.72	3.74	9.07	4.07	4.22	25.89	**0.008**
LDL to HDL ratio	2.71	0.79	2.55	1.2	1.14	4.41	0.097
Ejection fraction	48.93	8.7	45	18.75	30	69	**<0.001**
Gensisni Score	41.26	37.81	35.5	36.25	1	208	**<0.001**
CD4 count	481.75	206.87	467	271.25	39	1341	**0.2**

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Mayana SK, et al., Cardiol Cardiovasc Med 2024
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Table: 1:2 Clinical profile of the patients in control group

Variables	Mean (n=100)		Median		Range		p value
	Mean	SD	Median	IQR	Minimum	Maximum	
Age (Years)	56.71	10.7	58	14.5	26	81	0.2
Body mass index	27.98	5.21	27.3	6.41	18.7	44	**0.024**
Low density lipoprotein (mg/dL)	107.44	43.09	107	63.75	26	232	**0.015**
Triglycerides (mg/dL)	173.95	138.57	145	99.5	47	1143	**<0.001**
High density lipoprotein (mg/dL)	37.8	8.55	37.5	12.75	14	59	0.2
Total cholesterol (mg/dL)	176.62	48.74	170.5	76.75	92	333	0.2
Total cholesterol of HDL ratio	4.83	1.52	4.49	2.05	2.38	10.74	**0.002**
LDL to HDL ratio	2.92	1.21	2.67	1.72	0.7	6.63	**0.008**
Ejection fraction	51.4	7.88	50	15	30	60	**<0.001**
Gensisni Score	47.02	27.32	42.5	31.13	3.5	128	**0.01**

Table: 1.3 Comparison of baseline and demographic characteristics

Variables	Sub groups	Group HIV (n=76)		Control Group (n=100)		p value
		Number	Percentage	Number	Percentage	
Sex	Male	63	82.89	74	74	0.2
	Female	13	17.11	26	26	
Age group (Years)	21 to 29	0	0	2	2	
	30 to 39	6	7.89	4	4	
	40 to 49	26	34.21	17	17	
	50 to 59	31	40.79	33	33	0.355
	60 to 69	9	11.84	33	33	
	70 to 79	4	5.26	9	9	
	80 to 89	0	0	2	2	
Median age (Years)	Mean±SD	50.82	9.46	53.91	10.98	0.052
ACS	NSTEMI	14	18.42	35	35	
	NSTEMI SP PTCA	2	2.63	0	0	
	STEMI	34	44.74	41	41	
	STEMI SP PTCA	3	3.95	0	0	**0.003**
	USA	18	23.68	24	24	
	USA SP CABG	1	1.32	0	0	
	USA SP PTCA	4	5.26	0	0	
Diabetes mellitus	Present	16	21.05	57	57	
	Prediabetes	0	0	7	7	**<0.001**
	Absent	60	78.95	36	36	
Other risk factors	Hypertension	25	32.89	48	48	**0.031**
	Smoking	25	32.89	7	7	**<0.001**
	Alcohol consumption	0	0	5	5	0.057
Body mass index	Normal	6	7.89	12	12	
	At risk	13	17.11	21	21	
	Overweight	37	48.68	36	36	0.384
	Obese	20	26.32	31	31	
Body mass index (Kg/m^2)	Median and IQR	27	5.15	27.3	6.41	0.963

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LDL (mg/dL)	< 100	44	57.89	44	44	**0.047**
	≥ 100	32	42.11	56	56	
LDL (mg/dL)	Mean±SD	93.66	21.5	107.44	43.09	**0.006**
Triglycerides (mg/dL)	< 150	19	25	55	55	**<0.001**
	≥ 150	57	75	45	45	
Triglycerides (mg/dL)	Mean±SD	197.82	71.59	173.95	138.57	0.14
HDL (mg/dL)	Normal	57	75	70	70	0.501
	Abnormal	19	25	30	30	
HDL (mg/dL)	Mean±SD	35.89	8	37.8	8.55	0.131
Dyslipidemia	Present	15	19.74	19	19	0.526
	Absent	61	80.26	81	81	
Total cholesterol (mg/dL)	< 200	1	1.32	64	64	**<0.001**
	≥ 200	75	98.68	36	36	
Total cholesterol (mg/dL)	Mean±SD	327.37	69.88	176.62	48.74	**<0.001**
Total cholesterol to HDL ratio	Median and IQR	9.07	4.07	4.49	2.05	**<0.001**
LDL to HDL ratio	Median and IQR	2.55	1.2	2.67	1.72	0.473
Coronary artery disease	DVD	16	21.05	35	35	
	MILD CAD	4	5.26	0	0	
	MYOCARDIAL BRIDGING	1	1.32	0	0	
	RECANALISED LAD	2	2.63	0	0	**0.015**
	RECANALISED RCA	1	1.32	0	0	
	SVD	35	46.05	41	41	
	TVD	17	22.37	24	24	
Regional wall motion abnormalities	Ant. Wall	34	44.74	34	34	
	Inf Wall	19	25	24	24	
	LAD	0	0	1	1	0.283
	Lat. Wall	0	0	1	1	
	Nr. Severe MR	1	1.32	0	0	
	NRWA	22	28.95	40	40	
Ejection fraction	Mild LVD	20	26.32	28	28	
	Moderate LVD	18	23.68	11	11	0.058
	Severe LVD	1	1.32	0	0	
	Preserved LVD	37	48.68	61	61	
Ejection fraction	Median and IQR	45	18.75	50	15	**0.045**
Gensini score	Mild	28	36.84	16	16	
	Intermediate	26	34.21	43	43	**0.007**
	High	22	28.95	41	41	
Gensisni Score	Median and IQR	35.5	36.25	42.5	31.13	**0.02**
CD4 count	<200	4	5.26			
	200 to 499	37	48.68			
	≥ 500	35	46.05			
Duration of disease (Years)	Newly diagnosed	25	32.89			
	Upto 5	37	48.68			
	>5 to 10	12	15.79			
	≥ 10	2	2.63			

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Regimen	NEW	20	26.32			
	NO	2	2.63			
	TEE	1	1.32			
	TLD	11	14.47			
	TLE	18	23.68			
	ZLN	24	31.58			

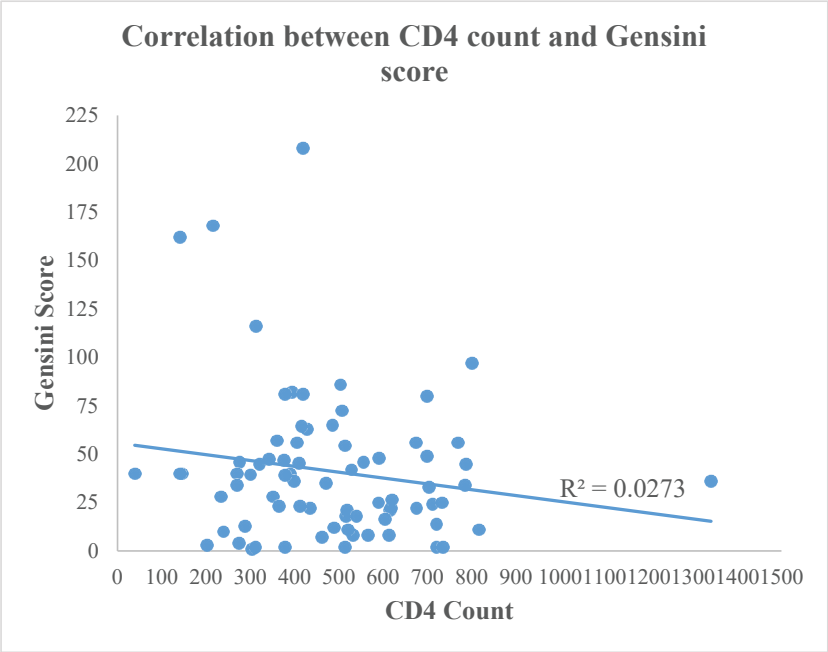

Fig:1 Spearman's Correlation coefficient (ρ)=-0.123; R2=0.027; p=0.291

Table: 1.4 Comparison of CD4 count with respect to duration of HIV

Duration	Number of patients	CD4 count	
		Median	IQR
Newly diagnosed	25	507	352.5
Upto 5	37	471	291
>5 to 10	12	454.5	241.75
≥ 10	2	357	-
p value		0.379	

Table: 1.5 Comparison of CD4 count with respect to HIV regimen

HIV regimen	Number of patients	CD4 count	
		Median	IQR
NEW	20	561.5	333.75
NO	2	296.5	-
TEE	1	-	-
TLD	11	516	399
TLE	18	500	284.25
ZLN	24	410.5	212
p value		0.26	

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as assessed by the Gensini score, was more in the non-HIV group (35.50 vs 42.50, p 0.020). The systolic function was impaired more in the HIV group (45% vs 50%,p 0.045). HIV-specific information in those with available CD4+ results in the HIV-positive group was 481.75 cells/mm3. Four patients (4/76;5.26%) had a CD4+ count <020cells/mm3. Thirty-seven patients (37/76;48.68%) had a CD4+ count of 200-499 cells/mm3, and thirty-five patients (35/76;46%) had a CD4+ count > 500 cells/mm3. Fifty-six patients (73.6%) were on ART at the time of ACS, and twenty (26.3%) were treatment naïve. ZLN regimen was the most commonly prescribed ART regimen (31.58%). There was no correlation between CD4 count and Gensini score (p 0.291).

Discussion

In this retrospective case-control analysis of patient records at a large southern Indian tertiary hospital in India, the ten-year period prevalence of HIV in those hospitalized with ACS was 0.003%. A lower prevalence of comorbidities such as diabetes and hypertension were noted in the HIV population after adjusting for age, gender, and BMI. STEMI and single vessel disease were more commonly experienced by the HIV population [13]. The relative absence of traditional risk factors in these patients points to additional pathogenic mechanisms, one of which is dyslipidemia. Autopsy studies in HIV patients have shown premature CAD even before ART initiation due to dyslipidemia patterns such as decreased LDL, HDL and apolipoprotein B [14]. Despite a non-significant diagnosis of dyslipidemia between the two populations, lipid profiles reveal a higher prevalence of subclinical dyslipidemia. Our study found a higher prevalence of raised triglycerides and low levels of LDL in the HIV population. Previous studies have shown similar results, which are thought to be due to metabolic complications of ART [15]. Recent metanalysis has confirmed how different types of ART drugs modulate metabolic pathways with varying results on lipid parameters. Evidence supports early initiation of ART for cardiovascular protection. There is also evidence that Art interruption increases the risk of CAD and other comorbidities [16]. Hence it may be concluded that the combined effect of HIV and certain ART medications results in complex dyslipidemia patterns and thereby increasing the risk of CAD and resultant ACS. Another mechanism for CAD in HIV patients is immune dysfunction, which can be assessed by measuring the number of CD4 lymphocytes. Lichtenstein et al. demonstrated that a CD4 count of less than 500 cells/mm3 was an independent risk factor for CAD. They demonstrated an attributable risk factor of approximately 20%, similar to other risk factors [17]. The majority (53.9%) of our HIV population had a CD4 count of less than 500 cells/mm3, which suggests an increased risk in these patients. This population's current detectable viremia is an additional risk factor, given its contribution to a chronic persistent inflammatory state [18]. Even patients with viral suppression have high inflammatory markers than those without HIV, thereby predisposing them to CAD [19].

Our HIV population had a predominance of STEMI and single vessel disease, even after adjusting for age, gender, and BMI, which is most probably due to the unique coronary plaque features. Virtual histology using intervascular ultrasound has shown a high prevalence of unstable plaque morphology rich in necrotic tissue and less calcific. These lesions end up having a thicker fibrous cap than traditional CAD [5]. These non-calcified plaques are more prone to rupture when compared to calcific or mixed plaques, thereby putting these patients at a higher risk of single vessel STEMI when compared to the non-HIV population [20]. This higher plaque vulnerability is thought to be caused by the chronic inflammatory process caused by HIV itself. Studies have shown that HIV patients with single-vessel disease had a higher Gensini score [20], as shown by Moran et al [21] indicating more severe stenosis in them. In contrast, our study showed that the HIV population had a lower Gensini score when compared to the non-HIV population. This finding of a lower Gensini score does not suggest low risk, but rather it may represent the presence of a plaque with high-risk features. Our findings overall were consistent with the literature. The lipid profile and risk factors were consistent with the literatures. Also, the type of ACS and the number of coronary arteries involved matched with studies described in the literature. An interesting finding in our study was the prevalence of low Gensini scores in the HIV population. This finding contrasts with some other studies which demonstrate a higher Gensini score. Our study has several limitations. First, the nature of retrospective observational design limits its ability to control for unmeasured confounders. Second, our limited sample size did not allow for sub-group analyses. Third, we may have underestimated the prevalence of HIV within the ACS population due to the relatively infrequent reporting of HIV results. Fourth, our sample was from a single center and may not be representative of India. Last, we were confronted with missing data that were only sometimes collected as part of routine clinical care. For example, CD4+ counts and viral loads were only available for some HIV-positive patients and, when available, were not always taken during the same admission as for the ACS event. As a result, we may have missed cases with our retrospective design. However, this risk was minimized by using electronic database systems for our case identification and data extraction.

Conclusion

In conclusion, we observed that HIV-positive patients with ACS were more likely to present with STEMIs and single-vessel disease and had limited traditional ACS risk factors. It was also noted that HIV patients had low LDL and high Triglyceride levels. This suggests additional pathogenic mechanisms for the development of CAD and ACS in patients with HIV, possibly attributed to the inflammation and

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immune activation caused by infection with HIV. However, ART therapy may also have a role to play. To date, ART is an essential treatment for HIV and highlighting the importance of prompt initiation of ART in patients with HIV cannot be overemphasized [22].

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