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LETTER TO THE EDITOR

Myocardial Infarction in People Living with HIV (PLWH)

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Abstract

People living with HIV (PLWH) face a two-fold higher risk of myocardial infarction (MI) compared to HIV-negative individuals, often occurring at a younger age. We report the sudden death of a 37-year-old man with a 5-year history of HIV infection on antiretroviral therapy (ART), well controlled on a regimen of dolutegravir, lamivudine, and tenofovir disoproxil fumarate. He was a non-smoker and non-alcohol user with no traditional comorbidities (dyslipidaemia, hypertension, diabetes, smoking, and metabolic syndrome). At autopsy, the heart was enlarged and an erythematous patch was seen on the antero-lateral wall of the left ventricle. Histopathological examination confirmed myocardial infarction with neutrophilic infiltration and 50% occlusion of the lumen of left circumflex artery. This case emphasizes that cardiovascular risk in PLWH persists despite the use of newer, non-dyslipidaemic ART regimens and the absence of traditional risk factors. It stresses the need for aggressive cardiovascular risk assessment in the management of HIV-positive patients as part of their ART care and periodic follow-up.

Keywords: HIV, Myocardial Infarction, Antiretroviral Therapy, Sudden Cardiac Death, Forensic Pathology

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The advent of Antiretroviral Therapy (ART) has shifted the trajectory of HIV from a fatal disease to a manageable chronic condition. However, this increased longevity is accompanied by a rise in non-AIDS-defining illnesses, particularly cardiovascular disease (CVD) [1]. Even when viral suppression is achieved, People Living with HIV (PLWH) exhibit an approximately two-fold higher risk of myocardial infarction (MI) compared to uninfected controls [2]. Furthermore, PLWH who experience MI are typically younger on average by 10 years than their HIV-negative counterparts [3].

Modern ART regimens, specifically those incorporating Integrase Strand Transfer Inhibitors (INSTIs) like Dolutegravir, are generally considered to have a more favourable metabolic profile than older protease inhibitors. Despite this, the risk of MI remains elevated even in patients on these newer therapies [4]. This suggests that the underlying inflammatory milieu and HIV-related vascular injury persist regardless of the ART class. This case report discusses the autopsy findings of a sudden death in a young, HIV-positive male on the newer non-dyslipidaemic ART regimen (metabolically favourable).

A 37-year-old man living with HIV had been on antiretroviral therapy for five years, receiving dolutegravir, lamivudine, and tenofovir disoproxil fumarate. He had no history of tobacco or alcohol abuse and was not known to have hypertension, diabetes mellitus, or any other traditional risk factors for myocardial infarction. He was discovered unconscious in the backyard of his residence after having last been seen alive the previous night when he went to the washroom. He was declared “brought dead” the following morning, following which a medicolegal case was registered, and a forensic autopsy was undertaken to ascertain the cause of the sudden, unwitnessed death.

At autopsy, a laceration measuring 2 cm × 1 cm, scalp-deep with irregular and contused margins, was present over the left occipital region, with a corresponding internal contusion of the scalp. Multiple abrasions measuring 1–2 cm were noted over the forehead and left elbow. The injuries mentioned above are consistent with a terminal fall and there are no other fatal injuries on the body. On further examination, the heart was enlarged, weighed 310g and covered with pericardial fat. A 2.5 cm × 1.5 cm distinct erythematous patch with a focal adherent blood clot was observed over the antero-lateral wall of the left ventricle (Figure 1). Microscopic examination of the left ventricular wall revealed focal sub pericardial neutrophilic infiltration in the interstitium and increased eosinophilia of cardiac muscle fibres, indicative of early ischemic changes consistent with myocardial infarction (Figures 2-4). The left circumflex artery showed an atheromatous plaque resulting in approximately 50% luminal occlusion, with the lumen remaining patent. Chemical analysis of the viscera was negative for ethyl alcohol and common poisons. The cause of death in this case was opined as myocardial infarction.

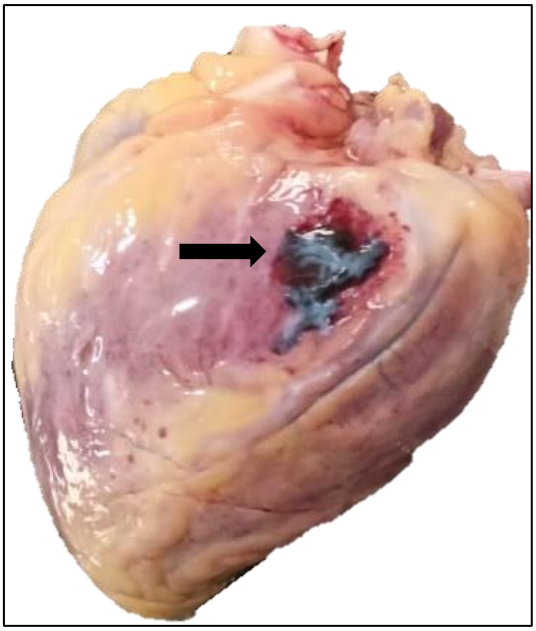

Figure 1. Heart, Gross; distinct erythematous patch with focal adherent blood clot.

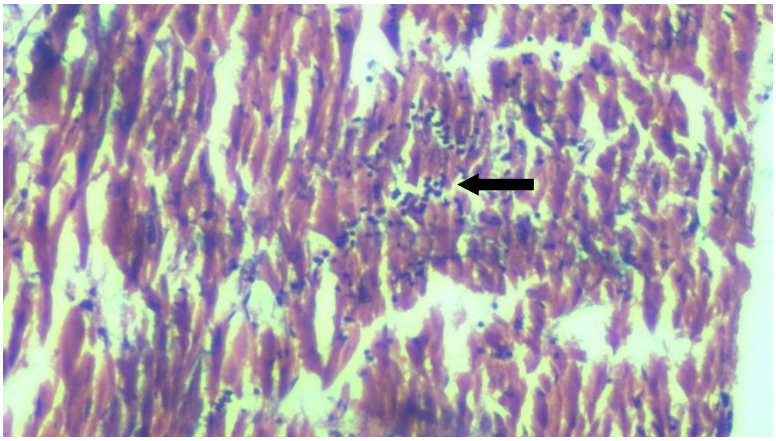

Figure 2. (H&E staining, Magnification – 200) Neutrophilic infiltration in the interstitium of cardiac muscle bundles.

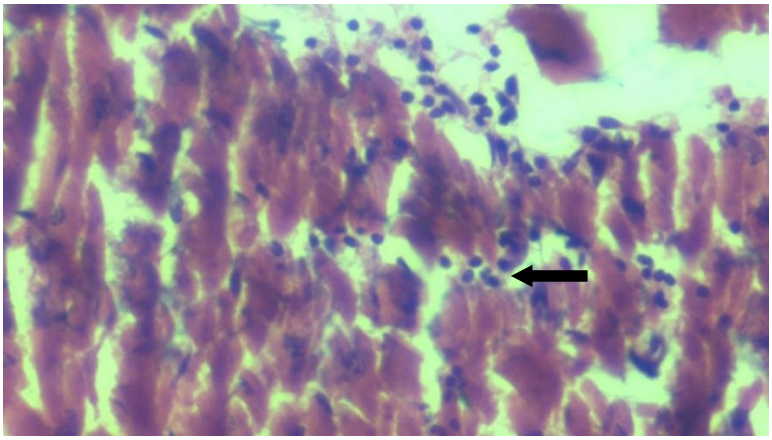

Figure 3. (H&E staining, Magnification – 400) Neutrophilic infiltration in the interstitium of cardiac muscle bundles.

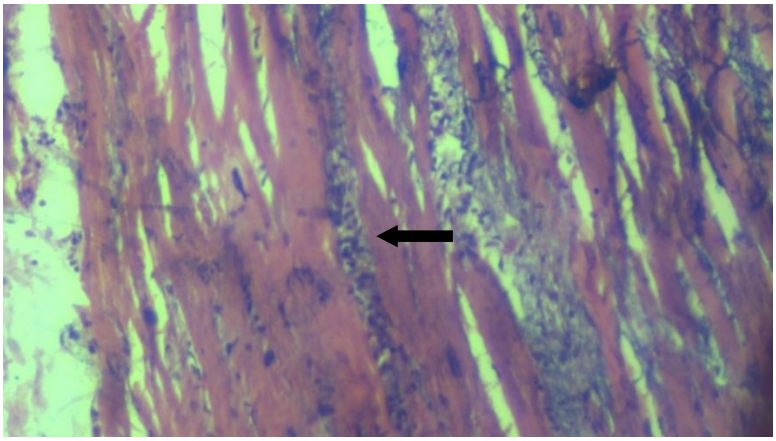

Figure 4. (H&E staining, Magnification - 400) Increased eosinophilia of cardiac muscle bundles along with neutrophilic infiltration.

The autopsy findings in this 37-year-old male confirm that sudden cardiac death (SCD) in HIV-positive individuals can occur in the absence of traditional risk factors such as smoking or diabetes, even in patients on modern, non-dyslipidaemic ART regimens. The presence of ischemic changes and partial coronary occlusion (50%) suggests that HIV-associated myocardial infarctions often involve mechanisms beyond simple plaque rupture, including endothelial dysfunction and supply–demand mismatch [5].

The risk of MI in PLWH is multifactorial. While traditional risk factors such as smoking and dyslipidaemia may also be prevalent in this population, HIV-specific factors including chronic immune activation, inflammation, and endothelial dysfunction play a critical independent role [6]. Although this patient was on a newer ART regimen (dolutegravir/tenofovir) known for a safer lipid profile compared with older protease inhibitors or abacavir [7, 8], the risks associated with these agents require further scrutiny. Biomarkers such as IL-6 and hsCRP remain elevated in PLWH and correlate with mortality [9]. This supports the hypothesis that chronic low-grade inflammation and immune activation drive accelerated atherosclerosis and plaque instability, even in patients with viral suppression [10].

People living with HIV continue to exhibit a 20%–100% increased relative risk of myocardial infarction compared with those without HIV. In addition to myocardial infarction, HIV infection has been associated with a heightened risk of stroke, sudden cardiac death, heart failure, pulmonary hypertension, and myocardial fibrosis [5]. Chronic immune activation and persistent systemic inflammation, even in virally suppressed individuals, contribute to accelerated atherosclerosis. Elevated inflammatory cytokines and markers of immune activation promote endothelial dysfunction, lipid oxidation, and plaque instability, thereby increasing susceptibility to acute coronary events [6].

A distinct clinical entity, myocardial infarction with non-obstructive coronary arteries (MINOCA), is characterized by evidence of acute myocardial infarction in the absence of obstructive coronary artery disease. In people living with HIV,

MINOCA may be associated with mechanisms such as microvascular ischemia or transient coronary thrombosis without fixed coronary obstruction [11].

Beyond myocardial infarction, HIV infection has been linked to a range of cerebrovascular complications. Studies have reported an increased risk of stroke in people living with HIV, including cases of juvenile ischemic stroke, suggesting that HIV can accelerate vascular disease at a younger age. The underlying mechanisms include blood clotting disorders, emboli from infections, opportunistic CNS infections, and direct damage to blood vessels by the virus. These factors illustrate that HIV can affect the cardiovascular system broadly, not just the heart, and may explain the reason why people with HIV remain at higher risk for serious vascular events like myocardial infarction [12].

A postmortem study by Tseng et al. (2021) found that HIV-positive individuals had more than double the rate of presumed sudden cardiac death compared with those without HIV. Moreover, HIV-positive hearts showed higher levels of myocardial fibrosis, indicating structural changes that may increase vulnerability to fatal cardiac events like arrhythmias responsible for sudden cardiac death [13].

India's HIV epidemic remains low, with an adult HIV prevalence of 0.20% in 2024; however, the country still has approximately 25.61 lakh people living with HIV (PLWH), representing the second largest PLWH population globally. As ART coverage improves and individuals with HIV live longer, attention is shifting from opportunistic infections to systemic diseases like cardiovascular disease [14].

The 2021 National Guidelines for HIV Care and Treatment emphasize early, lifelong ART to mitigate inflammation, but sudden cardiac death related to MI remains a concern and requires proactive monitoring. The national programme under NACO provides ART free of cost, and there is a clear policy movement toward integrated HIV–Non communicable Diseases (NCD) care. Clinicians are therefore encouraged to look beyond rigid algorithms by systematically screening for hypertension, diabetes, and dyslipidaemia, selecting ART regimens with lower metabolic risk, and ensuring linkage of PLWH to primary NCD programs to reduce the burden of preventable cardiovascular morbidity and mortality [15].

In conclusion, this case illustrates the increased risk of myocardial infarction in HIV-positive individuals, even when they are receiving treatment. The inflammatory burden of chronic HIV infection remains a potent driver of atherothrombosis and sudden cardiac death, necessitating continued cardiovascular vigilance and comprehensive risk assessment, including in patients on modern, non-dyslipidaemic ART regimens. Importantly, virologic suppression and the absence of traditional cardiovascular risk factors do not confer immunity from fatal cardiac events in people living with HIV, and HIV itself should be recognized as an independent cardiovascular risk accelerant.

Limitations

Detailed premortem information on HIV disease characteristics, including CD4 count, viral load, and overall immune status, was not available. Furthermore, non-modifiable cardiovascular risk determinants, such as family history and underlying genetic predisposition, could not be ascertained.

Conflicts of interest

The authors declare that they do not have conflict of interest.

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Ethics committee approval

Consent for conducting autopsy in this case was obtained from law enforcement authorities and all ethical concerns including consent from the next of the kin were addressed by the authors.

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