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CLINICAL CASE

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Acute myocardial infarction in a young adult living with newly diagnosed human immunodeficiency virus

Infarto agudo de miocardio, en un adulto joven con diagnóstico reciente de infección por el virus de la inmunodeficiencia humana

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ABSTRACT

Human immunodeficiency virus (HIV) infection continues to be a major public health problem. However, people living with HIV are surviving to older ages and are presenting with a higher prevalence of cardiovascular disease. HIV infection itself is associated with an increased risk of atherosclerotic cardiovascular disease, most commonly manifesting as acute coronary syndrome, typically at younger ages and often in the absence of traditional cardiovascular risk factors. We present the case of a patient with ST-segment elevation acute coronary syndrome with a history of non-significant tobacco exposure (0.75 pack-years, discontinued six years prior), whose main cardiovascular risk factor was a recent diagnosis of HIV infection. Consequently, this was a treatment-naïve patient with no prior exposure to antiretroviral therapy, consistent with findings from other studies analyzing this population, which describe a low burden of comorbidities, a distinct clinical presentation of the syndrome, and a high thrombus burden at the level of the culprit plaque on coronary angiography.

RESUMEN

La infección por virus de la inmunodeficiencia humana continúa siendo un problema de salud pública. Sin embargo, los pacientes que viven con VIH están sobreviviendo a edades mayores y presentando mayor prevalencia de enfermedad cardiovascular. La misma infección por VIH se asocia con un riesgo incrementado de enfermedad cardiovascular aterosclerótica, que se manifiesta más comúnmente como un síndrome coronario agudo y usualmente a edades más tempranas y sin factores de riesgo tradicionales. Se presenta el caso de un paciente con un síndrome coronario agudo con elevación del segmento ST con antecedente de tabaquismo no significativo (0.75 paquetes-año, suspendido hace seis años), cuyo factor de riesgo principal es diagnóstico reciente de infección por VIH, siendo un paciente sin exposición previa a tratamiento antirretroviral y coincide con otros estudios donde se ha analizado esta población en donde se encuentran con escasas comorbilidades, la forma de presentación del síndrome y la alta carga de trombo a nivel de la placa en la angiografía.

Keywords: myocardial infarction, human immunodeficiency virus, young adult, cardiovascular diseases.

Palabras clave: *infarto del miocardio, virus de la inmunodeficiencia humana, adulto joven, enfermedades cardiovasculares.*

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Abbreviations:
AMI = Acute Myocardial Infarction
ART = AntiRetroviral Therapy
CAD = Coronary Artery Disease
CHIP = Clonal Hematopoiesis of Indeterminate Potential
HIV = Human Immunodeficiency Virus
LVEF = Left Ventricular Ejection Fraction
PLWH = People Living With HIV

INTRODUCTION

Currently, an estimated 40.8 million people worldwide are living with the human immunodeficiency virus (HIV), with approximately 1.3 million new cases per year.[1] By 2024, 630,000 people had died from HIV-related causes; however, efforts in prevention, diagnosis, and early treatment have positively impacted life expectancy in these patients, transforming HIV infection from

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a fatal disease into a complex chronic health condition.[1] People living with HIV (PLWH) are surviving to older ages, and epidemiological studies have demonstrated that they have up to twice the likelihood of developing cardiovascular disease compared with HIV-negative individuals.[2] Coronary artery disease has been the predominant clinical manifestation of cardiovascular disease in PLWH, with a 1.5 to 2.1-fold increased risk of acute myocardial infarction compared with controls.[3] In several cohort studies, the mean age at which PLWH experienced acute myocardial infarction was 48 years, lower than that reported in the general population.[4] We present the case of a patient with acute myocardial infarction who, due to his age, is classified as a young myocardial infarction patient, whose most relevant risk factor for this event is the recent diagnosis of HIV infection.

CASE PRESENTATION

31-year-old male with a bachelor's degree in nursing, with no family history of ischemic heart disease. He had a history of smoking for five years at approximately three cigarettes per day, corresponding to a one pack-year index, discontinued six years earlier. He denied using other substances. He had no prior diagnoses of diabetes mellitus or systemic arterial hypertension.

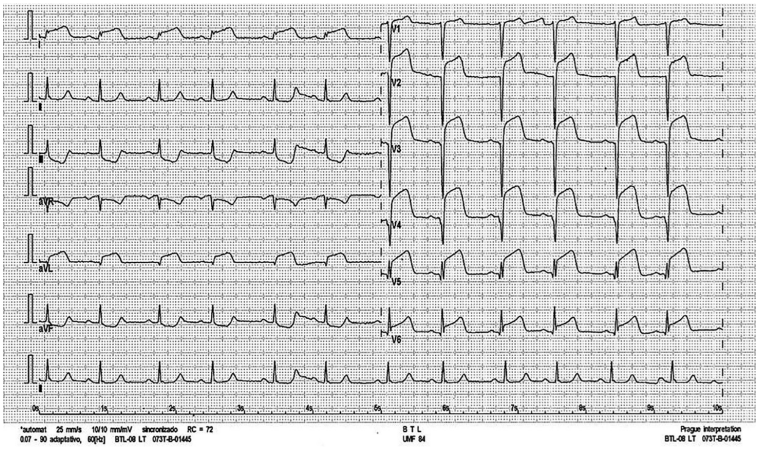

Figure 1: Initial 12-lead electrocardiogram: inversion of the T wave in leads V1 with an elevation of the ST-segment of 2 mm in leads V1-V6, DI, and aVL. aVL = augmented voltage left.

His current condition began in August 2024 with a five-hour evolution characterized by oppressive precordial chest pain, intensity 8/10 on the VAS scale, radiating to the submandibular region, accompanied by dyspnea on moderate exertion. On physical examination: heart rate 82 bpm, respiratory rate 19 breaths/minute, blood pressure 110/78 mmHg, oxygen saturation 98%. There was no respiratory distress with normal heart and respiratory sounds.

Admission laboratory studies showed leukocytes 8.5 × 10^9 /L, hemoglobin 13.7 g/dL, glucose 112 mg/dL, troponin I 14 ng/mL, glycated hemoglobin 5.2%, triglycerids 110 mg/dL, total cholesterol 106 mg/dL, HDL 20 mg, and LDL 64 mg/dL. Electrocardiogram revealed sinus rhythm, axis 48º, ST-segment elevation in leads V1-V6, DI and AVL *(Figure 1)*. Based on these findings, thrombolysis with a 35 mg tenecteplase bolus was performed, with clinical improvement and achievement of reperfusion criteria on the control ECG.

Given the patient's occupational epidemiological background, fourth-generation HIV ELISA testing was requested and resulted in a positive result. To rule out myocarditis as a differential diagnosis, cardiac magnetic resonance imaging *(Figure 2)* was performed, demonstrating subendocardial late gadolinium enhancement compatible with acute myocardial infarction of the anterior and anteroseptal walls at basal, mid, and apical levels, complicated by microvascular obstruction, with infarct size of 28% and left ventricular ejection fraction (LVEF) of 36%.

Within the following 24 hours, the patient underwent percutaneous coronary intervention *(Figure 3)*, where the left anterior descending artery demonstrated a definite thrombotic image in the mid-segment with 70% stenosis. A drug-eluting stent was implanted, achieving TIMI 3 antegrade flow. A glycoprotein IIb/IIIa inhibitor was administered and maintained for 24 hours. The patient remained asymptomatic thereafter. Additional evaluations showed viral load of 29,830 copies/mL and CD4 count of 224 cells/mm^3.Twelve weeks after the event, lupus anticoagulant was 18; anti-β2 glycoprotein I IgM and IgG were 8 SMU U/mL and 12 UGS/mL, respectively; anticardiolipin

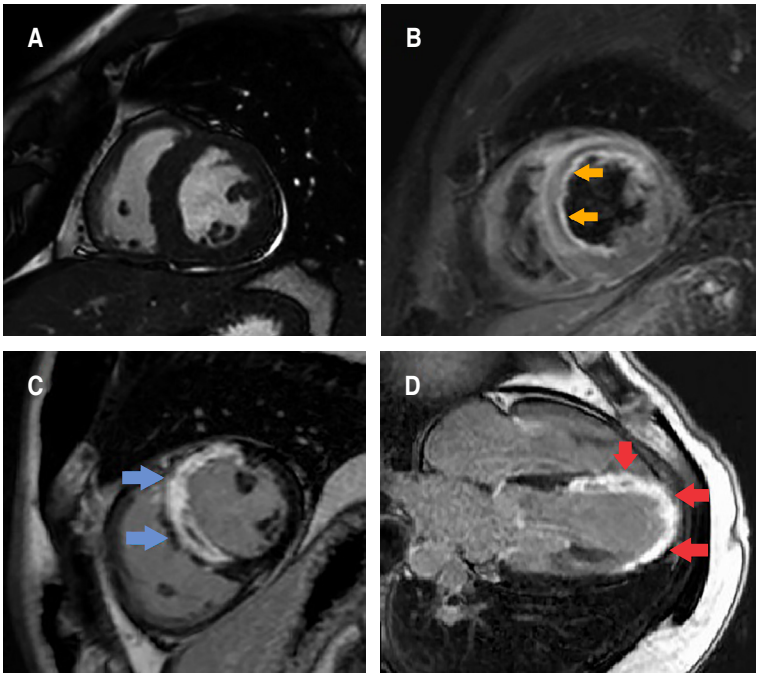

Figure 2: Cardiac magnetic resonance imaging: **A)** Short-axis cine at the mid-ventricular level (SSFP [Steady-State Free Precession]). **B)** Short-axis T2 STIR (Short Tau Inversion Recovery) showing transmural myocardial hyperintensity compatible with edema (yellow arrows). **C)** Short-axis late gadolinium enhancement (T1 GRE I-R [Inversion-Recovery Gradient Echo]) showing transmural enhancement in the anterior and anteroseptal walls (blue arrows). **D)** Four-chamber long-axis late gadolinium enhancement (T1 GRE I-R [Inversion-Recovery Gradient Echo]) involving the septal and lateral segments at the basal, mid, and apical levels (red arrows).
GRE I-R = Inversion-Recovery Gradient Echo. SSFP = Steady-State Free Precession. STIR = Short Tau Inversion Recovery.

IgM and IgG were below reference ranges, therefore negative for antiphospholipid antibody syndrome. Antiretroviral therapy with bictegravir/emtricitabine/tenofovir alafenamide was initiated, and statin therapy was changed to pitavastatin.

DISCUSSION

The association between cardiovascular disease and HIV infection has been recognized for at least two decades, including the increased risk of coronary artery disease (CAD).[5] In the Veterans Aging Cohort Study, a more than 50% higher risk of acute myocardial infarction (AMI) was identified in people living with HIV compared with healthy individuals, even after adjusting for risk factors such as smoking and substance use.[5] Although PLWH have a higher likelihood of traditional cardiovascular risk factors, the pathogenesis is complex and is potentially related to immune activation and chronic inflammation.[3] In the present case, the patient had no previously known cardiovascular risk factors, other than a history of smoking, which was considered clinically insignificant based on the consumption pattern.

Although the use of antiretroviral therapy (ART) in PLWH and its relationship with ischemic heart disease has been described since early studies involving protease inhibitors—associated with elevations in triglycerides, low-density lipoprotein, and lipoprotein(a)—the benefit and protective effect of ART clearly outweigh these risks and have significantly improved survival.[5]

Theoretically, HIV infection itself may independently predispose to premature atherosclerosis through endothelial and microvascular dysfunction, an enhanced proinflammatory and procoagulant state, and dyslipidemia.[3] In relation to this, elevated biomarker levels have been identified (C-reactive protein, interleukin-6, tumor necrosis factor-alpha), monocytic activation (CD14 and CD163), and increased coagulation activity (D-dimer).[2,5] Recent studies have explored the relationship between clonal hematopoiesis of indeterminate potential (CHIP), inflammation, and cardiovascular risk in PLWH; the prevalence of CHIP-associated somatic mutations is enriched in this population and is associated with ART, IL-6 levels, and C-reactive protein.[2] These markers could not be assessed in our patient; however, other differential diagnoses and prothrombotic states—including antiphospholipid antibody syndrome—were investigated and resulted in negative findings.

CAD in PLWH most commonly presents as acute coronary syndrome. According to several studies, the typical patient is younger than 50 years (90% of cases), male (81-97%), with an HIV diagnosis of more than eight years, on ART (53-96%), and with dyslipidemia (17-58%).[4] The most frequent presentation is ST-segment elevation myocardial infarction (29-64%).[4]

Single-vessel disease is the most common angiographic finding; however, multivessel disease is more frequently seen in HIV-negative patients.[5] There is limited information regarding CAD in ART-naïve patients. In a study by Becker et al., HIV-positive, untreated patients were found to be younger, with fewer traditional cardiovascular risk factors, less angiographic atherosclerotic burden, but greater thrombotic burden compared with controls.[6] This corresponds to our patient, who had low traditional risk, single-vessel disease, and a high thrombus burden.

The prognosis of PLWH during the acute phase of AMI does not appear to differ from that of non-infected individuals, with reported in-hospital mortality ranging from 0-8%, and without differences in acute heart failure rates.[4] Among patients undergoing percutaneous coronary intervention, rates of early and late stent thrombosis are low and comparable to controls, and stent restenosis has been reported in some studies, ranging from 9-52%.[5] At two-year follow-up, reinfarction rates were higher (9.4%), as well as the need for revascularization (20%).[4] Pharmacologic interactions between CAD therapies—including statins, antiplatelet agents—and ART must be considered.[5,7] For this reason, in conjunction with the ART initiated, statin therapy in our patient was changed to pitavastatin due to its lack of CYP3A4 metabolism.

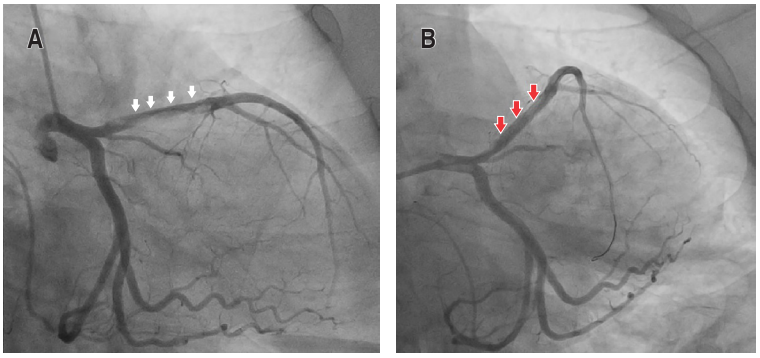

Figure 3: Coronary angiography: **A)** Right anterior oblique (RAO) projection showing a definite thrombus image in the mid-left anterior descending artery with approximately 70% stenosis (white arrows). **B)** Post-drug-eluting stent angiographic control demonstrating good angiographic outcome with TIMI 3 antegrade flow (red arrows).
RAO = right anterior oblique.

Based on the results of the REPRIEVE trial, primary prophylaxis with pitavastatin is recommended for all PLWH aged ≥ 40 years, regardless of cardiovascular risk or LDL levels.[8]

CONCLUSIONS

This case highlights the growing relevance of cardiovascular risk in PLWH as life expectancy increases. Although most reported cases involve previously diagnosed HIV patients on ART, this case involves an undiagnosed and ART-naïve patient, yet consistent with literature describing younger age, absence of traditional risk factors, reduced CD4 count, and high thrombotic burden, implicating HIV infection as a principal etiologic factor. Cardiac MRI findings suggest a poor prognosis. Primary prevention strategies such as statin therapy represent an important intervention for this population according to current evidence.

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Declaration of patient consent: protection of humans and animals. The authors declare that no experiments on humans or animals have been performed for this research. Confidentiality of data. The authors declare that they have followed their center's protocols for the publication of patient data. Right to privacy and informed consent. The authors have obtained the informed consent of the patients and subjects referred to in the article. This document is in the possession of the corresponding author.

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