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Acute Coronary Syndrome in HIV Naïve Patient with Low CD4 Count and No Other Significant Risk Factors: Case Report and Literature Review.

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Abstract

Coronary artery disease (CAD) has become the leading cause of mortality in patients with Human Immunodeficiency Virus (HIV). The typical HIV-infected patient presenting with acute coronary syndrome (ACS) is a man in his mid to late 40s. The most common presentation is an acute myocardial infarction (MI), most often with ST segment elevation. Coronary anatomy seems to be variable, with some studies showing a higher prevalence of single-vessel disease and others showing a higher prevalence of 2- and 3-vessel disease than in controls not infected with HIV.

Keywords

Coronary Artery disease, Acute Coronary Syndrome, Human ImmunodeficiencyVirus, Antiretroviral therapy, Myocardial Infarction

Abbreviations

CAD: Coronary Artery Disease; HIV: Human Immunodeficiency Virus; MI: Myocardial Infarction; ART: Antiretroviral Therapy; ECG: Electrocardiography; STEMI: ST Elevation Myocardial Infarction; PCI: Percutaneous Coronary Intervention; SMART: Strategies for Management of Antiretroviral Therapy; ACS: Acute Coronary Syndrome; CABG: Coronary Artery Bypass Grafting

Introduction

Coronary artery disease (CAD)is the leading cause of mortality in patients infected with human immunodeficiencyvirus (HIV).[1] The pathophysiology of atherosclerosis in these patients is very complex, including direct endothelial damage from viremia, inflammation from immune activation, higher prevalence and contribution from traditional atherosclerotic risk factors, and direct effects from

antiretroviral therapy itself. [1] Recent reports on young HIV-infected patients have focused interest on the association between myocardial infarction (MI) and antiretroviral medications[2] It is not yet entirely clear whether there is a direct or indirect association between HIV infection or its treatment and acute coronary syndrome. It was found that although side effects of Antiretroviral therapy (ART) may contribute to some of the increased rate of CAD in patients with HIV; [3,4] untreated HIV infection is also associated with substantial increase in the risk of CAD. [5,6]

Case Presentation

We report a case of a 32 year old African American male who was previously healthy with no risk factors for coronary artery disease admitted to our hospital on account of a four week history of shortness of breath, fever and productive cough, his initial work up pointed toward atypical pneumonia that turned out to bepneumocystis jiroveci pneumonia. His HIV test came back as positive with a very low CD4 Count of 9 cells/mm3,his lipid panel was normal; he was started on 21 days course of Sulfamethoxazole/Trimethoprim. On the 12th day of admission the patient developed another episode of chest pain. Electrocardiography (ECG) showed new ST Elevation in the inferior leads (Figure-1) that was not evident on the original ECG on admission (Figure-2).

A diagnosis of acute ST elevation myocardial infarction (STEMI) was made and the patient was transferred immediately to the catheterization laboratory. Selective coronary angiography showed right dominant coronary system with acute thrombotic partial occlusion of the mid right coronary artery (Figure-3). The left main left anterior descending and circumflex arteries showed no signs of focal coronary artery disease. A coronary spasm was a consideration, however repeated doses of nitroglycerin did not change the RCA lesion. The patient underwent percutaneous coronary intervention (PCI) of the lesion using a drug-eluting stent. Post intervention images showed excellent results with no residual stenosis and TIMI 3 flow (Figure-4). Work-up for coagulopathy and potential thrombotic or embolic source did not establish any obvious source.

Patient was started on dual anti-platelet therapy, beta-blocker, and statin. Echocardiogram showed normal LV systolic function without wall motion abnormality or significant valvular abnormality.

Discussion

Evidence establishing the association between HIV and CAD comes from several epidemiologic data that show that HIV is associated with an increased risk of cardiac death, myocardial infarction (MI), and stable CAD. [5-10] This increased risk is seen earlier in HIV patients thanin the general population. Currier et al compared the incidence of CAD among 28,513 HIV-infected individuals with 3,083,209

controls using data from California Medicaid (Medi-Cal) and found that the incidence of CAD among young men (<34 years) and women (<44 years) with HIV was significantly higher than controls. [7, 8] This suggests a potential acceleration of an atherogenic or thrombotic process in HIV-infected individuals even in relatively younger population.

HIV infection increases the risk of many risk factors of CAD. HIV has been associated with a significantly higher prevalence of CAD risk factors including hypertension (21.2% vs. 15.9%), diabetes (11.5% vs. 6.6%), and dyslipidemia (23.3% vs. 17.6%) than the non- HIV cohort (P<0.0001 for each comparison). [9] In addition to the traditional risk factors, the risk of CAD in HIV patients seems to be linked to a CD4 count less than 500 cells/mm3. [10]

Although ART contributes to increased risk of CAD;[3,4] there is strong evidence that not being on ART therapy is a risk factor in itself. This evidence comes from the Strategies for Management of Antiretroviral Therapy (SMART) trial. [5] This study randomized 5472 HIV-positive patients with CD4 counts higher than 350 cells/mm3 on therapy to a management strategy of continuous ART versus intermittent use of ART depending on their CD4 counts. Participants in the intermittent arm would stop taking ARTs when their CD4 cell count reached greater than 350 cells/mm3 and restart therapy once the CD4 count reached 250 cells/mm3 or less. The intermittent therapy arm had a significant higher risk of nonfatal cardiovascular events (RR=1.5 with 95% 1.0–2.5).[6]

In another large population study, HIV infection was associated with significantly higher incidence of MI [11.13 per 1,000 vs.6.98 per 1,000-patient years; Relative risk RR= 1.75 (95% CI, 1.51–2.02; P< 0.0001)]. This trend was even more significant among women. [9]. This risk of ACS could be related to increased thrombotic activity in these patients, evidenced by lower protein C level, higher factor VIII levels and higher frequencies of anticardiolipin and antiprothrombin antibodies when compared to non-HIV patients. [11] Another risk factor that may be responsible for the risk of MI is pneumonia; recent studies showed that the inflammatory state associated with pneumonia is associated with increased risk with cardiovascular events including MI. [12, 13]

The incidence of ACS in the HIV population trends to start in younger age and has some special characteristics. The typical HIV-infected patient presenting with ACS is a man in his mid to late 40s. The most common presentation is ST segment elevationMI. [6]. Coronary anatomy seems to be variable, with some studies showing a higher prevalence of single-vessel disease and others showing a higher prevalence of 2- and 3-vessel disease than controls not infected with HIV [6]. Compared to non-HIV patients, HIV-infected patients with a first episode of ACS had a higher incidence of recurrent ACS and

urgent PCI during the first year, this is increased risk of recurrent ACS is more linked to both increased risk of thrombosis and coronary restenosis. [14, 15]

The management of ACS in HIV patients is similar to its management in non-HIV patients. PCI with and without stenting as well as coronary artery bypass grafting(CABG) seems to be safe, effective and feasible option in HIV patients, but it is associated with a higher incidence of repeat revascularization in the long-term. [16-21]

Conclusion

HIV increases the risk of CAD especially in treatment naïve patients with low CD4 counts.

Management of CAD in these patients should be the same as for the general population, while keeping in mind that this group tends to have a higher incidence of recurrence especially in the first year.

Figures

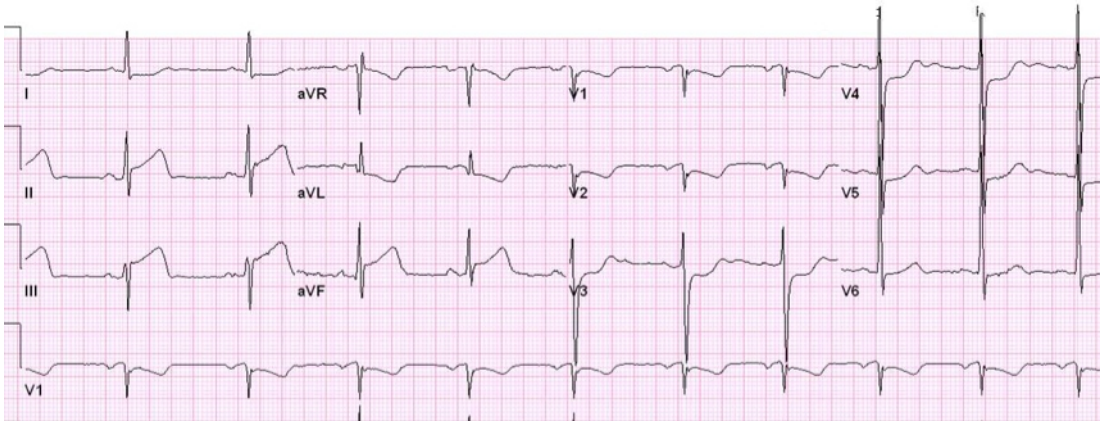

Figure 1: ECG on day 12 of admission shows new ST Elevation MI in inferior leads II,III,aVF

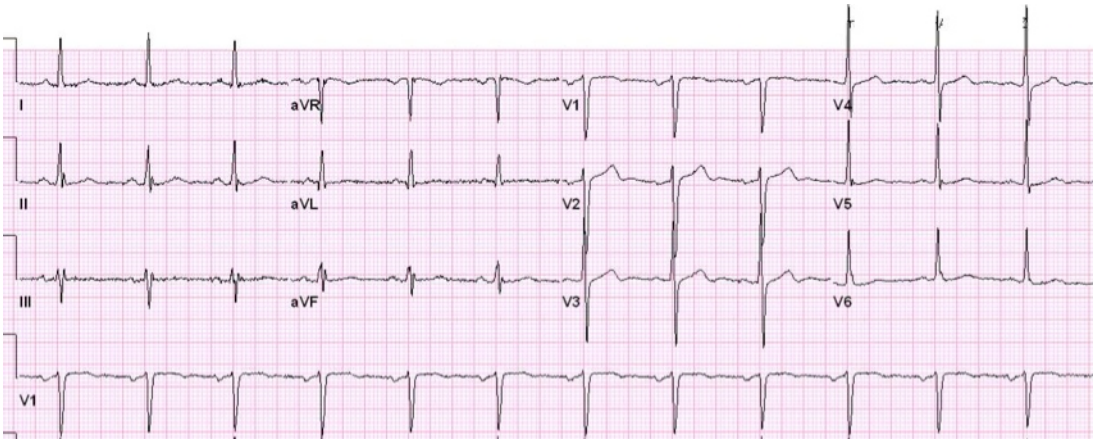

Figure 2: ECG on admission, no signs of prior MI

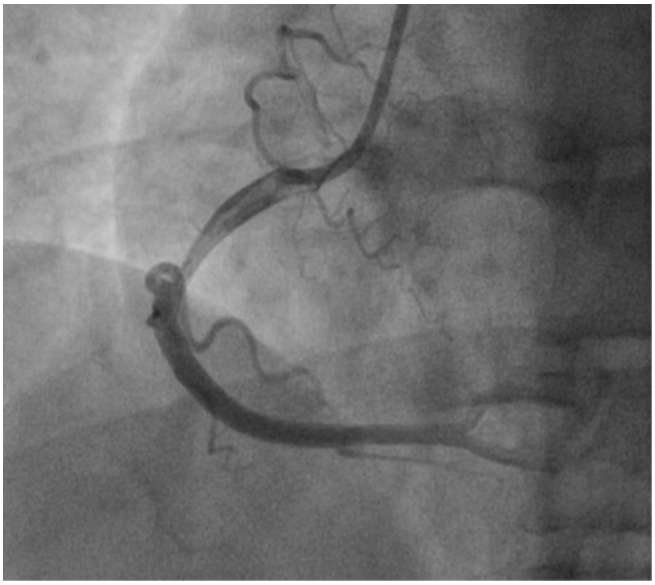

Figure 3: Right Coronary Artery before intervention

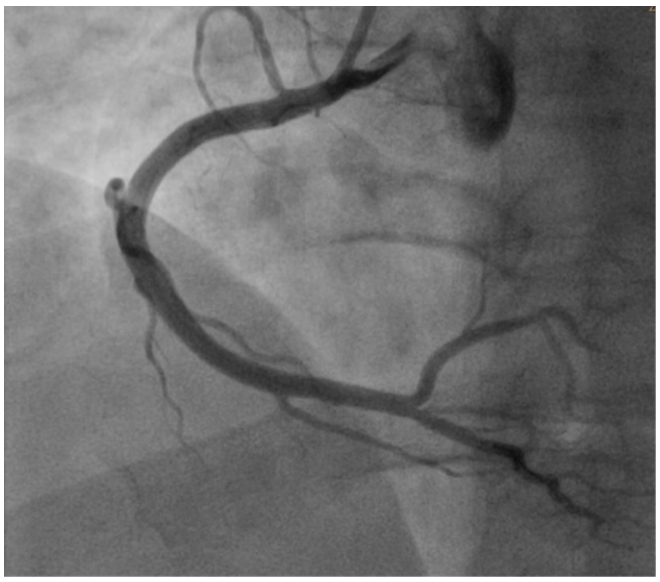

Figure 4: Right Coronary Artery after intervention

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