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Clinical Case

Young Patient with HIV Presented with STEMI and Triple Vessel Disease

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Introduction

Infection with the human immunodeficiency virus is characterized by an acquired and irreversible immunosuppression, which predisposes the patient to multiple infections with opportunistic germs and neoplasms, which can affect the vast majority of organs. The heart, which was initially thought to be exempt from this rule, was shown, to be affected right from the first phase of Human immunodeficiency virus (HIV) infection. Cardiac damage, as the initial sign of HIV infection, is extremely rarely reported, on the other hand, cardiac damage demonstrable by echocardiography and anatomo-pathology is much more frequent than the clinical manifestations suggest [1,2].

It is also known that antiretroviral therapy with protease inhibitors, applied in the treatment of HIV infection, produces disturbances in lipid metabolism and can be associated with early atherosclerosis [3-5].

It is not uncommon for patients with HIV infection to present earlier in life with signs and symptoms of acute coronary syndrome.

We describe a case of a young man with HIV, complicated with left anterior descending (LAD) coronary artery occlusion-related STEMI treated with percutaneous coronary intervention (PCI).

Clinical Case

A 36 years old male with a past medical history of HIV diagnosed in 2012, on treatment with Abacavir 60mg/Dolutegravir 5mg/Lamivudine 30mg (cautions- association of abacavir and high risk of myocardial infarction), was transferred to our Emergency Department with severe chest pain, lasting for 6 hours, suggestive of an acute myocardial infarction. His hemodynamic parameters showed regular heart rate of 60beats/minute and blood pressure of 130/80 mm Hg. Elevated lipid profile values were recorded.

An initial ECG showed normal sinus rhythm and ST elevations in leads V1-V5. Echocardiogram registered normal left ventricle systolic function without wall motion abnormality or significant valvular abnormality.

The patient was given aspirin, ticagrelor, high-intensity statin, and unfractionated heparin and was emergently transported to the cardiac catheterization lab. Diagnostic catheterization revealed triple vessel disease: proximal subtotal occlusion of the left anterior descending (LAD) artery; 80% proximal stenosis of the left circumflex artery; right coronary artery with significant stenosis. The patient underwent percutaneous coronary intervention (PCI) of the culprit lesion (LAD) using two drug-eluting stents- Resolute Onyx 3.5/15mm and Synergy 4.0/24mm with excellent results - no residual stenosis and TIMI 3 flow. At the heart team, the patient was discussed as indicated for subsequent coronary angiography in one month, considering continuing revascularization of the other culprit lesions.

The patient remained hemodynamically stable throughout the hospital stay and remained on goal-directed medical therapy, which included dual anti-platelet therapy, beta-blocker, and statin.

The Repeat ECG showed resolving ST elevations compared to initial ECG and the patient was discharged home and instructed to consult with an infectious disease specialist in order to assess the change of antiretroviral therapy.

One month after discharge, the patient was advised again to undergo coronary angiography.

The antiretroviral therapy was changed to Bictegravir 30 mg/Emtricitabine 120mg/Tenofovir alafenamide 15mg (with no cardiovascular cautions and no evidence of high atherosclerotic risk) and two consecutive procedures were performed. The LCx was treated with PCI and implanting a Resolute Onyx 2.5/22mm drug-eluting stent and the RCA-with PCI and Resolute Onyx 2.5/30mm; 3,5/30mm and 3,5/30mm; with very good outcomes.

The patient was asymptomatic for four months, after that he presented again with accelerated unstable angina, without ECG dynamics and with negative hsTroponinT. After the Heart Team review and discussion, the patient underwent coronary angiography. A preserved result of past stenting in the proximal segment of LAD and LCx was found. RCA- with mid-segment restenosis and high-grade PD stenosis. PTCA of the RCA was performed with satisfactory angiographic result. The patient was discharged hemodynamically stable, with a significant reduction in the values of the lipid profile; with specified medical therapy and a recommendation for a follow-up by a cardiologist.

Conclusion

HIV infection leads to an increased risk of cardiac death and myocardial infarction. It is considered that there is a potential

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acceleration of an atherogenic or thrombotic process in HIV-infected patients, even in relatively younger population.

Our clinical case shows that a lipid profile should be examined and an electrocardiogram should be performed in all HIV-infected patients, especially in the stage of primary infection, when there are no clinical changes suggestive of cardiac dysfunction or these are uncertain. The electrocardiogram changes, especially in young people, raises the suspicion of heart damage from the early stages of HIV infection. Electrical changes in HIV-infected patients should alert the clinician to possible ventricular dysfunction. Cardiac complications of HIV infection are much more common than thought and cause structural and functional damage through complex mechanisms, in which the direct infection of cardiac tissues, the severity of immunodeficiency. Opportunistic infections play an important role. Because of the paucity of the clinical picture, cardiac complications are rarely diagnosed and treated, although they are important for the patient's perspective.

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