Case Reports

Acute Coronary Thrombosis and Multiple Coronary Aneurysms

in a 22-Year-Old Man with the Human Immunodeficiency Virus

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The human immunodeficiency virus (HIV) can cause diverse cardiovascular complications. In HIV patients on antiretroviral therapy, the prevalence of myocardial infarction has steadily increased over the years. Young patients who are naïve to antiretroviral therapy and who experience coronary events are not well represented in the medical literature. We describe the case of a 22-year-old man, infected with HIV for 4 years and never treated with antiretroviral therapy, who emergently presented with a non-ST-segment-elevation myocardial infarction. Coronary angiograms revealed thrombosis and multiple coronary artery aneurysms; however, no areas of atherosclerotic stenosis were apparent. He was successfully treated with coronary stenting, antiplatelet therapy, and anticoagulation. Nine months after the initial presentation, he exhibited excellent exercise capacity, and no ischemia was evident. We discuss the various therapeutic approaches in this case. ***(Tex Heart Inst J 2014;41(2):208-11)***

Key words: *Anticholesteremic agents/therapeutic use; anticoagulants/therapeutic use; cardiovascular diseases/diagnosis/etiology/therapy; coronary aneurysm/diagnosis/drug therapy/epidemiology/etiology; HIV infections/complications; risk factors; treatment outcome; vasculitis/physiopathology/virology*

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Patients with the human immunodeficiency virus (HIV) can present with dilated cardiomyopathy, myocarditis, pericarditis, endocarditis, pericardial effusion, and vasculitis. Premature coronary atherosclerosis is more frequently seen in this population because of traditional risk factors for coronary artery disease (CAD), improved survival rates from antiretroviral therapy (ART), and metabolic disturbances related to ART, including lipodystrophy, dyslipidemia, and insulin resistance.[1-3] The prevalence of myocardial infarction (MI) has increased 4-fold in this population, according to an analysis of almost 5,000 patients from the Frankfurt HIV cohort after ART was instituted.[1] Some HIV patients develop coagulation abnormalities with arterial and venous thromboembolism. Combined HIV and ART might be associated with accelerated atherosclerosis and endothelial dysfunction.[1,3] The development of coronary artery aneurysms (CAA) and acute MI in HIV patients has not been reported amidst other manifestations.[4,5] We report the case of a young man with HIV who had a myocardial infarction with coronary thrombosis and CAAs without atherosclerotic stenosis, and we discuss the various therapeutic options.

Case Report

In June 2011, a 22-year-old black homosexual man, with a 4-year history of HIV infection, CD4 counts above 500 cells/μL, a low viral count, and no history of ART, emergently presented after experiencing severe substernal chest pain (Universal Pain Assessment Tool score, 10/10). He had no history of Kawasaki disease during childhood, did not smoke or use illicit drugs, had no family history of premature CAD or sudden cardiac death, and previously had no traditional risk factors of CAD.

At the current presentation, an electrocardiogram showed deep T-wave inversions in leads V_2 through V_5. A lipid profile yielded a low-density-lipoprotein cholesterol level of 64 mg/dL, a high-density-lipoprotein cholesterol level of 35 mg/dL, and triglycerides at 26 mg/dL. The patient's homocysteine level was normal (9.5 μMol/L), and his cardiac troponin I level peaked at 5.64 ng/mL. The diagnosis was non-ST-segment-elevation MI.

The patient was urgently taken to the cardiac catheterization laboratory. Coronary angiograms obtained through right radial artery access showed aneurysmal left

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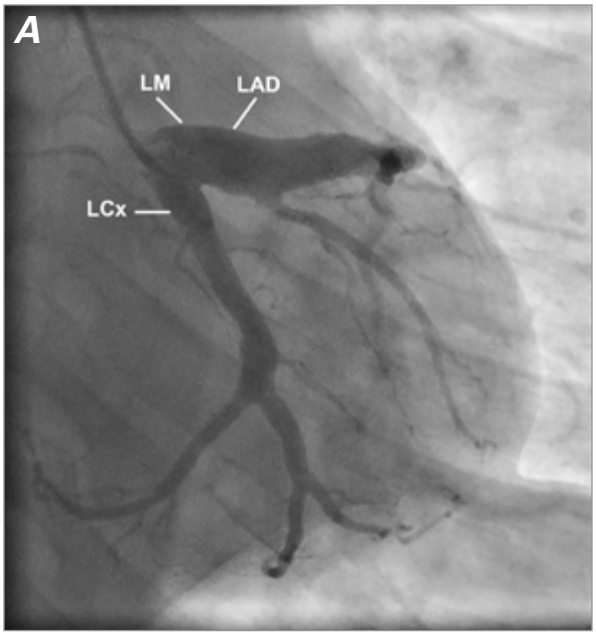

Fig. 1 *Angiograms show coronary artery aneurysms in the **A**) left main (LM), left anterior descending (LAD), left circumflex (LCx), and **B**) right coronary arteries.*

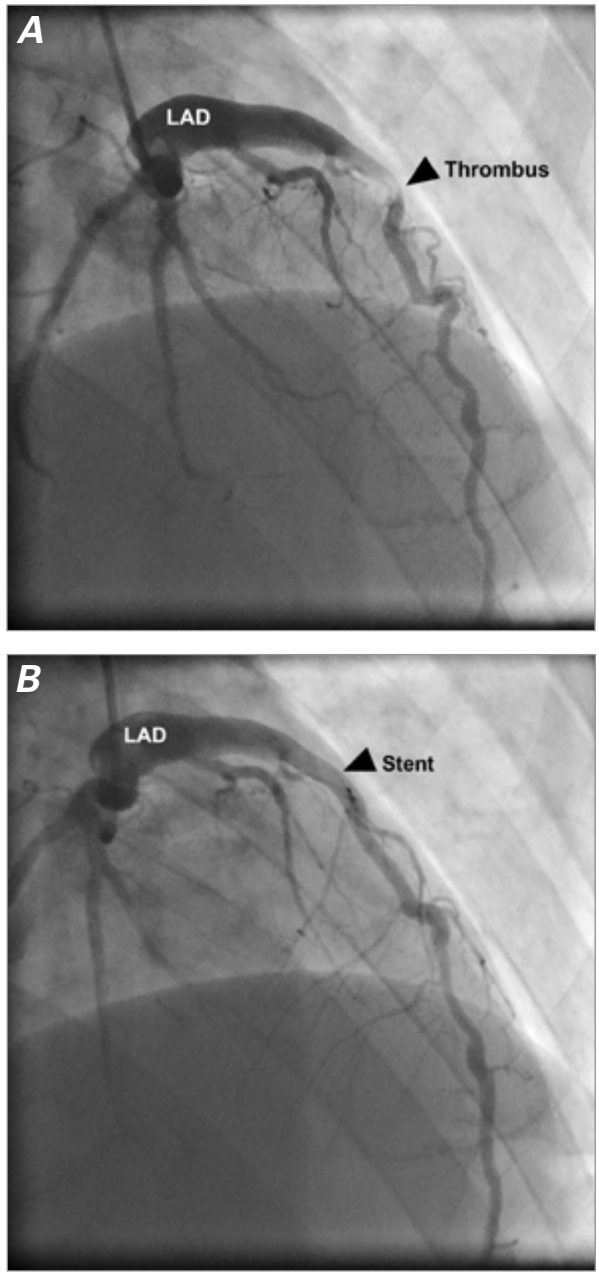

Fig. 2 *Angiograms show an aneurysm in the proximal left anterior descending coronary artery (LAD) with **A**) thrombosis before bare-metal stent insertion and **B**) normal flow thereafter.*

main, left anterior descending (LAD), left circumflex, and right coronary arteries (Fig. 1). A 99% stenotic area with a filling defect that suggested thrombus was seen in the proximal-to-mid LAD immediately distal to the aneurysmal area (Fig. 2A). No areas of atherosclerotic stenosis were noted. Because of the patient's emergent presentation, we did not perform intravascular ultrasonography or optical coherence tomography. A 3.5 × 16-mm VeriFLEX™ bare-metal stent (Boston Scientific Corporation, Inc.; Natick, Mass) was deployed in the LAD, with no residual stenosis (Fig. 2B). No distal embolization was detected. The nonaneurysmal parts of the patient's coronary arteries were of smooth appearance and normal sizes, unusual for premature or diffuse CAD. After intervention, a 2-dimensional echocardiogram showed normal systolic function and no segmental wall-motion abnormalities.

Immediately after the coronary intervention, the patient was started on simvastatin therapy (40 mg once daily), with a possible change to pravastatin if ART were to become necessary. He was discharged from the hospital after 6 days, in stable condition.

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In view of the thrombus in the patient's LAD and the multiple aneurysmal areas in his coronary arteries, we recommended adding warfarin anticoagulation and antiplatelet therapy of aspirin and clopidogrel. The patient chose dabigatran for anticoagulation, despite its off-label use for that purpose. He stopped taking the dabigatran after 2 months but continued taking aspirin and clopidogrel. At his 2-month examination, a screening profile for thrombophilia was positive for lupus antibody and negative for prothrombin and Factor V Leiden gene mutation; the protein C and S levels were normal.

The patient was re-examined 9 months after the MI. He underwent treadmill stress testing and exhibited excellent exercise capacity with no evidence of myocardial ischemia. He was advised to continue long-term dual antiplatelet therapy as secondary prevention of coronary thrombosis. As of March 2013, he was asymptomatic.

Discussion

We report the highly unusual case of an HIV patient, naïve to ART, who presented with acute MI, coronary thrombosis, multiple large CAAs, and no evidence of atherosclerotic CAD. The cause of his CAAs might have been related to HIV infection, autoimmune inflammation causing intimal disease or disruption, or HIV-negative vasculitides.[6] Naidoo and Beningfield[7] classified HIV vasculopathy as HIV-related coronary vasculitis, HIV-ART–related atherosclerotic vascular disease, and mixed type. Our patient had no clinical signs of some HIV-related cardiac manifestations, such as myocarditis, CAD, cardiomyopathy, or pericardial effusion. Some specific vasculitis, including polyarteritis nodosa, systemic lupus erythematosus, Behçet disease, and Takayasu arteritis, might contribute to the development of CAAs in the general population[8,9]; however, this has not been reported in HIV patients. Barbaro[5] reported autopsy findings in a 32-year-old HIV patient who died of MI one week after the onset of flu-like symptoms. There was evidence of HIV-1 in the arterial wall; and immunoglobulin A plasma cells, a feature of Kawasaki disease, were within the vascular lesion. Stankovic and colleagues[10] reviewed the cases of 20 HIV patients who had severe immunosuppression and a high viral load. Eighteen presented with Kawasaki-like syndrome while on ART and responded to aspirin and intravenous immunoglobulin therapy. Stankovic and colleagues did not report the presence of CAAs; however, cerebral artery aneurysms have formed in young patients infected with HIV.[11] Chetty and colleagues[12] observed black African patients who had large-artery vasculopathy and aneurysms involving the common carotid artery, the abdominal aorta, and the common iliac, femoral, and popliteal arteries; prominent leukocytoclastic vasculitis of the vasa vasorum and periadventitial vessels was present. It is unclear whether racial background and genetic makeup predisposes some HIV-positive patients (including ours) to develop various aneurysms, including CAAs. The risk of thrombosis in our patient's CAAs might have been further increased by the presence of positive lupus antiphospholipids—the coagulation abnormality most frequently found in HIV patients.[5] In a cohort of 562 patients with Kawasaki disease, Tsuda and colleagues[13] reported that all new aneurysms formed at the sites of stenosis, after follow-up periods as long as 17 years. Our patient had a different type of coronary dilation and had aneurysms without focal stenosis, except for the lone segment of acute LAD thrombosis.

Because our patient had no further coronary thrombotic events, the quandary arose of how long to continue aspirin and clopidogrel therapy in a 22-year-old man with a very active lifestyle. On the basis of current knowledge of adult patients with Kawasaki disease and coronary aneurysms,[14] we considered it prudent that our patient continue long-term antiplatelet therapy, given the ongoing risks of coronary thrombosis in the setting of previous acute MI, coronary stent implantation, multiple and large CAAs, positive lupus antibody, and background HIV infection. In the absence of bleeding complications, he could at least benefit from lifelong aspirin intake beyond the first year after coronary stenting.

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