Hybrid open and endovascular treatment of an aortic arch pseudoaneurysm in a patient with human immunodeficiency virus infection

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ABSTRACT

We describe an hybrid approach for aortic pseudoaneurysm with open and endovascular treatment as an alternative treatment for a high-risk patient infected with human immunodeficiency virus. A 42-year-old man, serum positive for human immunodeficiency virus, presented, with a large pseudoaneurysm of the arch aorta measuring 61 × 70 mm. An aortic arch debranching was performed, completed by thoracic endovascular aneurysm repair. A control computed tomography scan performed 3 months later showed a complete thrombosis of the pseudoaneurysm. The outcome of this treatment, particularly regarding the rate of infection, is yet to be determined, Longer follow-up is needed with a greater of patients. (J Vasc Surg Cases and Innovative Techniques 2020;6:516-9.)

Keywords: Aortic arch pseudoaneurysm; Hybrid treatment; Human immunodeficiency virus

Arterial pseudoaneurysm has been reported as a possible complication of human immunodeficiency virus (HIV) infection, which is known to affect the vascular system resulting in vasculitis, perivasculitis, fibroproliferative diseases and aneurysms.[1] Thoracic aortic arch (TAA) pseudoaneurysm is an uncommon, but surgically challenging problem, particularly in patients infected with HIV.

Open surgical management of vasculopathy in HIV-infected patients is characterized by high morbidity and mortality.[2] The literature contains only isolated case reports and small case series on the role of endovascular therapy in patients with HIV with vasculopathy and aneurysmal disease.[3,4]

We describe a case of a hybrid approach with open and endovascular treatment of a TAA pseudoaneurysm as an alternative treatment for a high-risk patient infected with HIV.

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The patient consented to publication of his case details and images with a signed consent form.

CASE

A 42-year-old man was admitted for the evaluation of a recurrent, nonspecific thoracic pain and dyspnea. The patient had no history of trauma, had chronic hepatitis hepatis B and V infection, and has been HIV-seropositive for 7 years. The infection was transmitted by sexual intercourse. The patient's clinical history was positive for opportunistic infections and had an atypical mycobacteria pneumonia 6 months ago, treated with rifampicin, ethambutol, and pyrazinamide. Prophylactic treatment has been started with sulfamethoxazole and trimethoprim (Bactrim simple).

On examination, he was afebrile and normotensive. Hematologic data indicated both anemia and hypoalbuminemia: hemoglobin, 9.2 g/dL; white blood cell count, 4600/mm^3; CD4 count, 180/mm^3; and albuminemia, 24 g/L. Renal function was within normal limits. Serologic nonspecific and specific tests for syphilis were negative.

The patient's HIV infection has been well-controlled with highly active antiretroviral therapy (HAART), including two nucleoside analogue reverse transcriptase inhibitors and one protease inhibitor. Viral load was 250 RNA copies/mL. HAART was started to decrease the viral load, increase the number of CD4$^+$ lymphocytes, and prevent opportunistic infections. Biological markers were performed to rule out bacterial infections, and his procalcitonin was less than 0.05 ng/mL and C-reactive protein was 32.4 mg/dL. Blood culture results were negative.

The diagnosis was confirmed by a computed tomography (CT) scan of the chest that revealed the presence of a large pseudoaneurysm (Fig 1, *A*) arising on the inferior

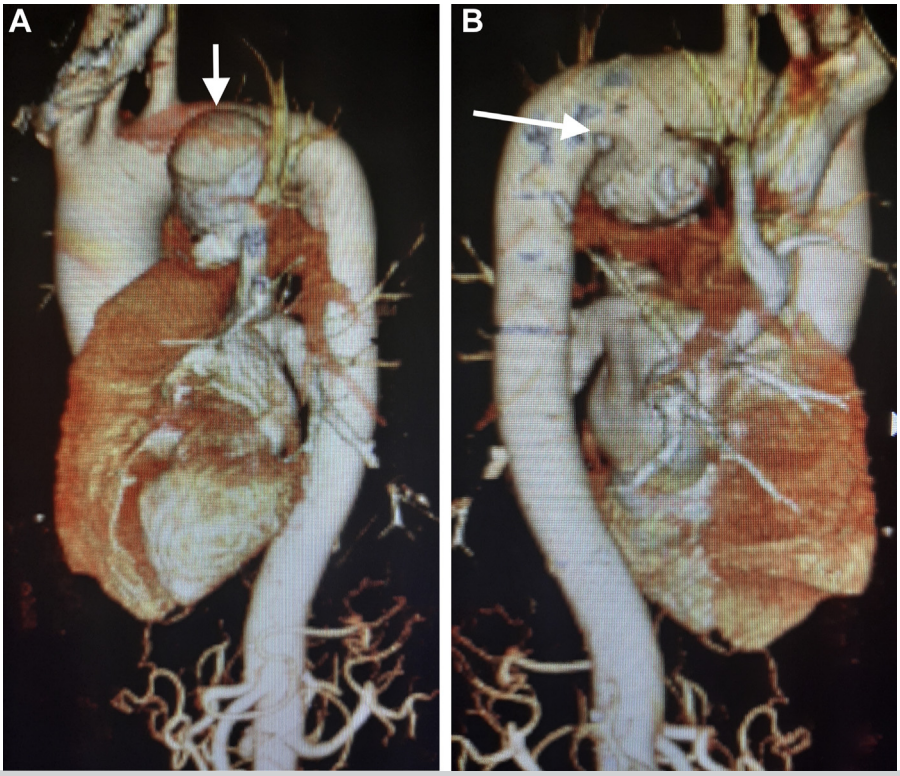

Fig 1. A, Three-dimensional computed tomographic angiography showing a large pseudoaneurysm (*arrow*) within the thoracic Aorta. **B,** Posterior view demonstrating the pseudoaneurysm arising on the inferior (*arrow*) aspect of the arch aorta measuring 61 × 70 mm.

aspect of the arch aorta measuring 61 × 70 mm (Fig 1, *B*). We identified some high-risk features in this case, namely, (1) immune status with a low CD4 count, (2) opportunistic infection in the past and, (3) hypoalbuminemia, that justified hybrid approach instead of a definitive open approach in a young patient.

An aortic arch debranching was performed first, a right to left common carotid artery bypass with a 8-mm Dacron graft, using retroesophageal tunneling was performed, and reimplantation of the left subclavian artery. Second, thoracic endovascular aneurysm repair was performed under general anesthesia via a femoral artery approach. Catheterization of the aortic arch was done with a hydrophilic guidewire and angiography was performed through the pigtail catheter. After replacing the hydrophilic guide wire with a Lunderquist wire, a 40 × 36 × 167 mm Zenith Alpha endograft (Cook Medical. Bloomington, Ind) was implanted through the left femoral axis with a landing close to the innominate artery (Fig 2).

Postoperatively, the patient received dual antiplatelet therapy of clopidogrel 75 mg plus aspirin 75 mg/d for 3 months. Thereafter, aspirin 160 mg/d was prescribed indefinitely.

A control CT scan performed 3 months later showed no endoleaks, and complete thrombosis of the pseudoaneurysm. Carotid bypass with reimplantation of the left subclavian artery was patent (Fig 3, *A-C*). However, the diagnosis of a mycotic aneurysm has not been excluded, and we have planned long-term

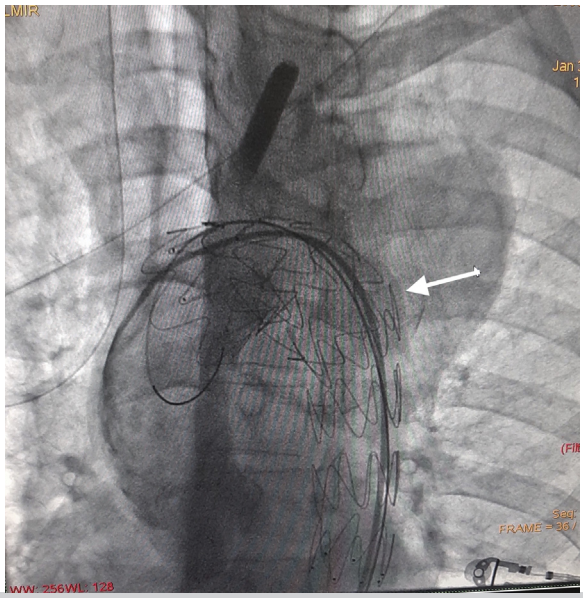

Fig 2. Catheter-directed angiogram after debranching demonstrating final angiographic control after thoracic endovascular aneurysm repair revealing pseudoaneurysm exclusion (*arrow*).

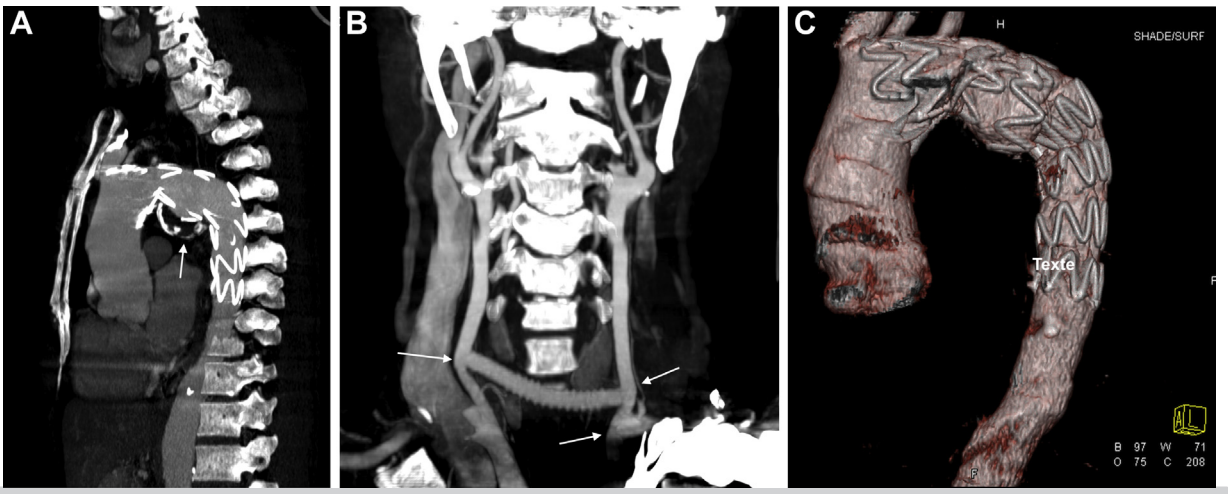

Fig 3. A, Sagittal image of a 3-month postoperative control computed tomography angiography (CTA) revealing a complete pseudoaneurysm exclusion (*arrow*). **B,** Frontal image of CTA showing a good patency of carotid bypass (*arrow*) and left subclavian artery transposition. **C,** Three-dimensional CTA showing a complete pseudoaneurysm exclusion.

clinical and imaging surveillance with control CT scans at 6, 12, 18, and 24 months postoperative. Biologic markers were performed monthly to rule out bacterial infections.

DISCUSSION

The last decades have been marked by an increase of a heterogeneous range of HIV-associated vascular pathologies under the rubric of HIV-associated vasculopathy.[3] The pathogenesis of
aneurysmal disease is multifactorial. Four pathophysiologic processes have been implicated, including immune complex deposition causing inflammation and endothelial injury,
direct HIV infection of the artery, bacterial infection, and side effects of HAART therapy.[5] Rupture is common at an early stage with the development of pseudoaneurysms.[6]

Atypical locations of aneurysms in HIV-positive patients poses some questions concerning its etiology, optimal treatment, and postoperative disease management.

This vascular disease has a predilection for affecting young males and an association with advanced stages of HIV disease, as manifested by immune status with low CD4 counts and opportunistic infections.[3] The present case confirms these clinical and hematologic data. Syphilis is a problem in the HIV community and can cause luetic aneurysms. Serologic tests for syphilis were negative in our patient. However, we should be aware of the potential for false-negative serology in both primary and secondary infection in the HIV-positive patient.[7] False-positive nontreponemal antibody tests are encountered more frequently in HIV-positive individuals and may be seen in up to 11% of cases.[8] The potential causative role of syphilis is not excluded in this case.

The rare association between infection with HIV and the development of vascular aneurysm has been reported by Marks and Kuskov[9] in a review of 12 young HIV-positive patients with focal areas of vascular disease. There were specific clinical features in this group of patients: they were young, they were all indigenous Africans with no history of atherosclerosis, there was rapid development of focal necrotizing vasculitis with aneurysm formation, and there was a slow, progressive development of granulomatous vasculitis.[9]

The use of HAART has resulted directly in dramatic decreases in morbidity and mortality among HIV-infected patients by decreasing the viral load and increasing the number of $CD4^+$ lymphocytes, thus allowing prevention of opportunistic infections and extending the life expectancy of persons living with AIDS.[10,11] HIV-positive patients on HAART should be considered good candidates for surgery.[12]

The results of conventional surgery are associated with a relatively increased incidence of operative morbidity and mortality owing to a high incidence of late graft infection, but they are dependent on several factors such as low CD4 lymphocyte counts and hypoalbuminemia.[13]

EVAR is a less invasive procedure, and is now often used as an alternative to open surgery, for management of abdominal and thoracic aortic aneurysm. Machado et al[14] reported two cases of HIV-infected patients in whom an AAA was diagnosed. He emphasized the importance of HAART associated with endovascular treatment in ameliorating the inflammatory process, which lead to aneurysm regression.[14] Thoracic endovascular aneurysm repair was an adequate approach to exclude aortic pseudoaneurysm in our case. Hybrid approach options may further expand the number of patients who can benefit from this treatment modality.[15] Cases of hybrid open and endovascular treatment have also been reported in this population.[16]

Other benefits of the endovascular approach include a decrease in the risk of transmission of blood-borne pathogens, including HIV, to healthcare workers, avoidance of thoracotomy or sternotomy incision, avoidance of aortic cross-clamping, decreased blood loss, decreased end-organ ischemia, and shorter operative time and hospitalization.[14,17]

In conclusion, we report a successful exclusion of a TAA pseudoaneurysm with an hybrid approach in a high-risk patient also for conventional surgery. The outcome of this treatment, particularly regarding the rate of infection, is yet to be
determined. Longer follow-up is needed with a greater number of patients.

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