

Category: Case Report

Paper Title: Repair of Type 1 Aortic Dissection in a patient with Human Immunodeficiency Virus Infection: A Case Report

Running Head: Repair of Type 1 Aortic Dissection in a patient with HIV Infection: A Case Report

ABSTRACT

Persons living with Human Immunodeficiency Virus (PLHIV) have become a demographic of concern in cardiovascular health. Aortic pathologies such as aortic aneurysms have been discussed frequently in the literature, but there have been very few reports of Aortic Dissection among PLHIV. We report a case of a 37-year old male with HIV infection and Stanford A, DeBakey 1 Aortic Dissection. He presented with dyspnea, easy fatigability, and bipedal edema. Computed Tomographic Aortogram revealed aortic dissection from the level of the sinus of Valsalva to the aortic arch. His preoperative morbidities included pneumonia, acute kidney injury, and hepatic failure. Once recovered, he underwent Modified Bentall's surgery. Histopathologic report revealed an aneurysmal aortic wall with chronic dissection and moderate medial degeneration. Our patient, similar to the 3 other cases of aortic dissection in PLHIV, was male with hypertension and chronic

kidney disease. The distribution of his dissection was similar in classification to the previous studies. Criteria to consider for surgical intervention include a high CD4 count, absence of AIDS, good nutrition, and good functional status. These were all present in our patient and solidified our decision for surgery.

TEXT

BACKGROUND

The increased life expectancy among people living with Human Immunodeficiency Virus (PLHIV) has brought with it an increased incidence of cardiovascular diseases (CVD), which research has described to be associated with the pathophysiologic mechanisms of HIV. We report a case of a 37-year-old male with HIV infection and Type 1 Aortic Dissection. Reports of this occurrence are very scarce, with no published paper discussing aortic dissection in a Filipino patient with HIV.

CASE PRESENTATION

A 37-year-old Filipino male with HIV infection was admitted complaining of dyspnea and bipedal edema. The patient was diagnosed with HIV infection in 2011 but started HAART in 2016. He had *Pneumocystis jiroveci* infection during the same year which was successfully treated. No other Acquired Immune Deficiency Syndrome (AIDS)-defining illnesses were documented. He was diagnosed with hypertension in 2016, with good

control on Amlodipine. He had a history of 4 pack years of smoking but stopped 10 years prior.

One month prior, he experienced exertional dyspnea slightly relieved by rest. He also had cough, hemoptysis, undocumented fever, bloatedness, and increasing abdominal girth. He was admitted to a primary hospital and treated as a case of pneumonia, but dyspnea persisted and progressed after discharge. Two weeks prior to admission, he developed progressive bipedal edema. There were no symptoms of colds, nausea, vomiting, weight loss, or changes in bowel movement.

Upon admission to our hospital, vital signs were stable with a blood pressure of 110/70 mmHg, heart rate of 70 beats/minute, respiratory rate of 20 cycles/minute, and normal temperature. He had clear breath sounds and symmetrical chest expansion. On cardiac exam, a grade 3/6 diastolic murmur was heard at the 2nd intercostal space on the right parasternal border. He had a protuberant abdomen with normal bowel sounds. There were no bruits or palpable masses noted. Peripheral examination showed grade 3 bipedal pitting edema.

Complete blood count and electrolytes were normal, but his serum creatinine was elevated at 2.0 mg/dL. His CD4 count was 504 cells/mm³ and his viral load was undetectable. Chest X-ray showed bronchovascular crowding at the bibasal region and cardiomegaly with biventricular prominence.

The patient's computed tomography angiogram (CTA) of the aorta showed aortic dissection from the level of the Sinus of Valsalva up to the aortic arch, classified as Stanford A, DeBakey I. Transthoracic echocardiogram (TTE) showed severe aortic

regurgitation secondary to aortic dilatation with effacement of the sinotubular junction and severe tricuspid regurgitation with dilated tricuspid valve annulus. Ejection fraction was normal (62%). There was concentric left ventricular hypertrophy with segmental wall motion abnormality of the inferior interventricular septum and inferior free wall suggesting possible involvement of the right coronary artery. This was ruled out by a CTA of the coronary arteries which showed normal arteries without identifiable plaques and a calcium score of zero.

The patient had a protracted hospital course because of hospital-acquired pneumonia and acute kidney injury (AKI). The pneumonia was treated with antibiotics. The AKI resolved after resolution of infection. However, he subsequently developed liver failure, with an Alanine Transaminase of 383 U/L. This resolved with adjustments of his HAART medications and discontinuation of Statins.

Once stable, the patient finally underwent Modified Bentall's Procedure with tricuspid ring annuloplasty. An adequate portion of the aneurysmal aortic wall was excised just enough for the Dacron tube graft to be placed. The diseased aortic valve was replaced with a mechanical valve. The histopathologic report revealed an aneurysmal aortic wall with chronic dissection and moderate medial degeneration. The patient developed AKI post-operatively for which temporary dialysis was initiated. At the time of writing, he is currently well, off dialysis, and consulting regularly at the out-patient department.

DISCUSSION:

The pathophysiologic correlation between HIV and CVD is endothelial dysfunction, which is caused by chronic inflammation, hypercoagulability, and platelet activation. HIV activates monocytes which stimulate cytokine production which then modulate endothelial function. The HIV proteins tat and gp120 have pathways that result in excessive nitric oxide production (NO). The excess NO reacts with oxygen radicals to produce peroxynitrite which causes oxidative stress and damage to the endothelium [1]. These mechanisms seem to be attenuated by decreased disease activity.

Aortic pathology among patients with HIV has been attributed to three major factors: 1) atherosclerosis, 2) vasculitis, or 3) infections. While there have been several documented cases of aortic aneurysm in patients with HIV, our search of the literature revealed only 3 other studies demonstrating aortic dissection in this particular population [2, 3, 4]. Most of the patients presented with typical symptoms of aortic dissection such as chest or back pain. Our case is notably different as his main symptoms were dyspnea, abdominal bloatedness, and bipedal edema, which we attributed to the severe aortic regurgitation and severe tricuspid regurgitation. Like all of the studies, our patient was male and had type 1 aortic dissection. As in 2 other case reports [3, 4], our patient had other comorbidities such as hypertension and chronic kidney disease.

Factors to be considered in deciding towards surgery in patients with HIV include the absence of AIDS, absence of malnutrition, and CD4 count above 200 cells/mm [5]. Our reasons for proceeding with surgical intervention are similar to those of Baciewicz [4]. Our patient was not terminally ill but had a good functional status prior to his current illness,

and was immunologically well-controlled. The patient also has a higher overall mortality risk due to dissection than from HIV and the surgery performed [4, 6].

The literature suggests that aortic aneurysms in HIV will present histologically with inflammatory infiltrates or atherosclerotic degeneration. However, we did not observe these findings in our case. Specimens of the aortic wall only revealed areas of fibrosis and cystic medial degeneration. This is compatible with the predominant histologic change observed among patients in our institution.

KEYWORDS:

Aortic dissection, Human Immunodeficiency Virus, Aortic pathology

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