รายงานผู้ป่วย

An Atherosclerotic Thoracic Aortic Aneurysm In a 36-years Old Patient with HIV Infection : A Case Report

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Abstract

A rare case of atherosclerotic thoracic aortic aneurysm was diagnosed in a 36-year old patient with HIV infection. CT scan of both chest and abdomen showed a fusiform aneurysm originating from distal aortic arch and extending to descending aorta. Left thoracotomy with aneurysmectomy and graft interposition was successfully performed and the histopathology demonstrated severe atherosclerosis without evidence of dissection.

Introduction

The vascular pathology has become an increasingly important manifestration of HIV disease.[1] Prior studies of aortic aneurysms in HIV patients have drawn attention to a strong association between aortic aneurysm and a vasculitic etiology,[2-9] caused by direct viral action of the HIV itself or by another infection resulting from immunosuppression. A rare case of an aortic aneurysm secondary to an atherosclerotic process in a young HIV-infected patient on HAART(highly active antiretroviral therapy) has been reported.[1] The author reports a rare case of fusiform thoracic aortic aneurysm secondary to atherosclerotic process in a young HIV-infected patient without HAART. The chest x-ray imaging, computerized tomography and histopathology were illustrated.

Case Report

A 36-year-old HIV positive man presented with recurrent episodes of non-specific thoracic pain for 1 year. He has been HIV-seropositive and treated at the rural hospital for 2 years. Chest X-ray at the time of diagnosis revealed mild dilatation of the aortic arch and

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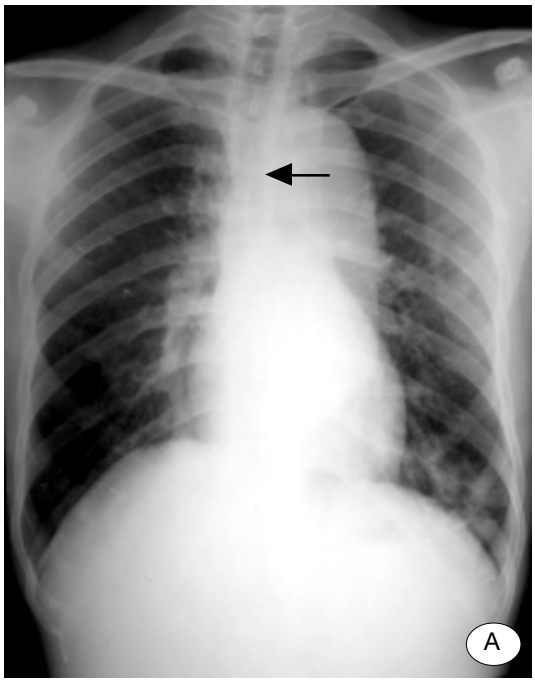
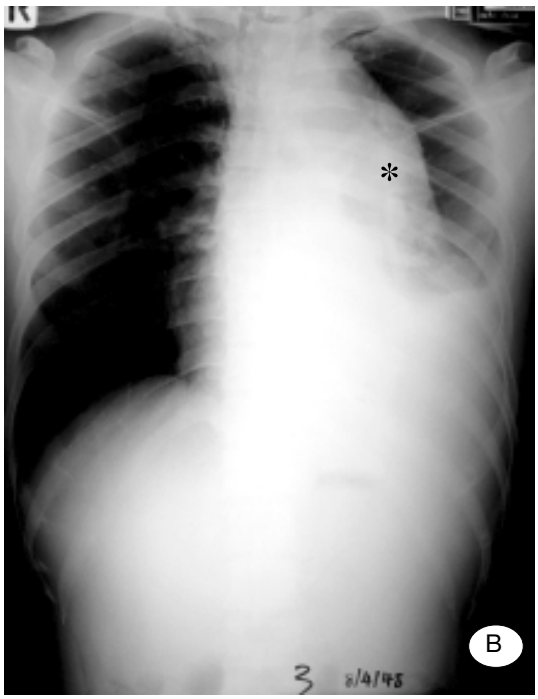
descending aorta with right tracheal deviation (Fig 1A). No further investigation or imaging was done. The patient had not been on highly active antiretroviral therapy(HAART) and no definite opportunistic infections or tumor have developed since the initial diagnosis. Careful questioning revealed no history of trauma or known systemic disease. He had a history of heavy smoking for 15 years but denied history of IV drug use or any familial history of premature coronary artery disease.

Two months before admission, he was refered to Prapokklao Hospital for evaluation of left pleural effusion and posterior mediastinal mass(Fig 1B). On physical examination, the patient





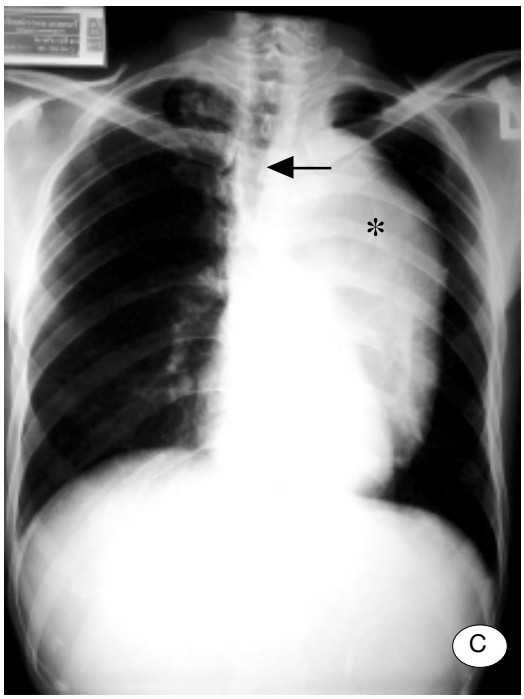


Fig 1. Chest PA radiograph (A), 2 years before surgery; mild dilatation of the aortic arch and descending aorta with right tracheal deviation(arrow).
(B) Two months before surgery shows markedly increased dilatation of the aorta(*) with left pleural effusion.
(C) In this admission, left pleural effusion is disappeared.

was febrile with blood pressure of 110/80 mmHg, a regular pulse of 80 beats/min, and a respiration rate of 18 /min. Left side breath sound decreased on auscultation and no murmur or sign of cardiovascular disease was detected. The other systemic examination was unremarkable. Laboratory findings showed a CD4 count of 258 cells/mm3. A complete blood count showed mild leukopenia and VDRL was non reactive. Sputum were negative for acid fast bacilli and culture for tuberculosis for 3 successive days. The blood culture was negative. Fasting blood sugar was within normal limits but a lipid profile was not performed.

He subsequently underwent a tranesophageal echocardiogram (TEE) and spiral CT scan. The TEE showed large aneurysm with internal thrombus. Aortic valve, aortic root and heart were normal. CT scan of both chest and abdomen revealed a fusiform aneurysm measuring 8.6 x 9 cms (diameter x length) originating from distal aortic arch and extending to descending aorta until the level just above the left diaphragm (Figure 2). Internal thrombus, more on the posterolateral wall and some calcifcation deposited in the aortic wall with intimal irregularity were detected without periaortic soft tissue mass. Minimal fibroreticular infiltration at right lung apex and left pleural effusion were seen. No evidence of rupture or dissection was detected. The abdominal aorta appeared normal. Left pleural aspiration and biopsy were done but no malignancy cells or organisms was detected. He was suspected and treated as TB pleura. The follow up CT scan, 2 months after antituberculous therapy showed disappeared left pleural effusion. Slightly increased in aneurysmal diameter (from 7.8 to 8.7 cm) with minimal pleural thickening adjacent to posterior wall of the aneurysm were also detected(Fig 3). The patient was refered to cardiac surgery unit for aortic aneurysm repair. No other imaging studies were performed in this admission.

Intraoperatively, thoracic aortic aneurym from distal aortic arch to 2 cm above diaphragm

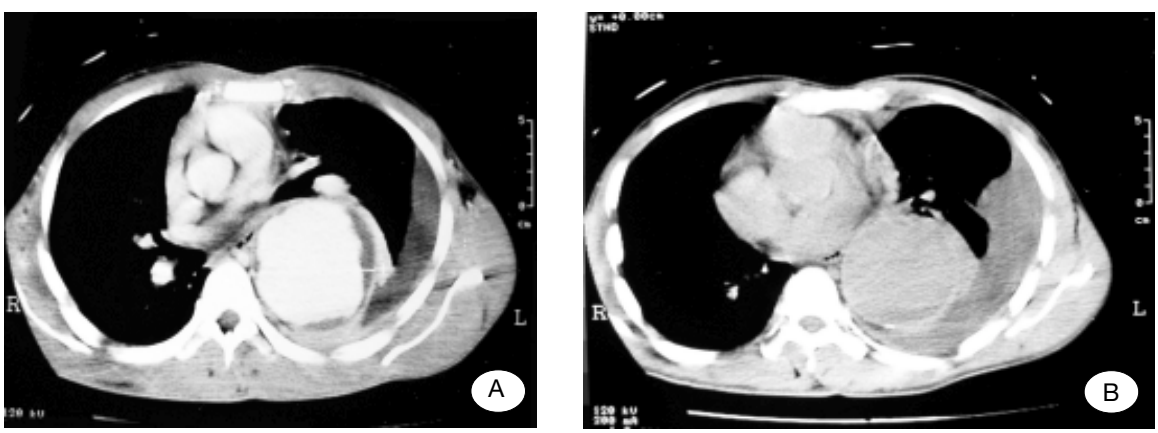


Fig 2. CT scan without (A) and with contrast enhancement(B), 2 months before surgery shows a 7.8 cm in diameter, fusiform aneurysm at level distal aortic arch and descending aorta with mural thrombus, more on the posterolateral wall. Rim calcification with intimal irregularity are seen. Left pleural effsuion is also detected.

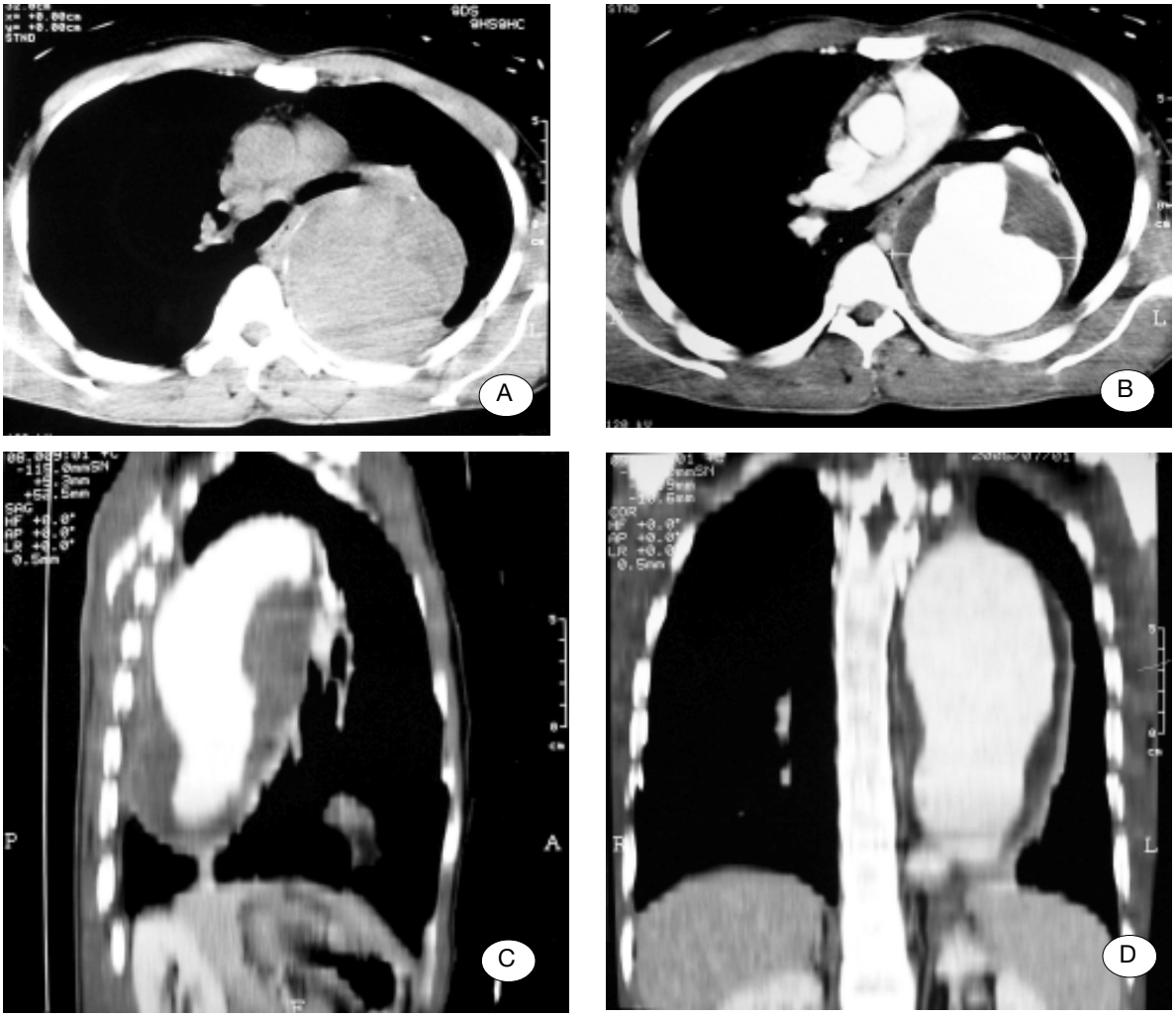


FIG 3. A, B, C and D. Follow up CT scan with contrast enhancement (in this admission) left pleural effusion is disappeared. Minimal pleural thickening adjacent aneurym is observed without evidence of rupture. Slight increased aneurysmal diameter.

was seen and left thoracotomy with aneurysmectomy and graft interposition was performed.

Pathological specimens were obtained from aortic arch and descending aorta. These demontrated evidence of severe aterosclerotic change of the descending aorta. Mural thrombus culture was negative for organisms. The conclusion of the pathologist was atherosclerosis, not an aortitis. The patient had an remarkable postoperative course.

Discussion

The association between HIV infection and aneurysm formation has been recently described. The mechanism of aneurysm formation is unclear.[7] Prior studies of aortic aneurysm in HIV patients have drawn attention to a strong association between aortic aneurysm and vasculitis etiology

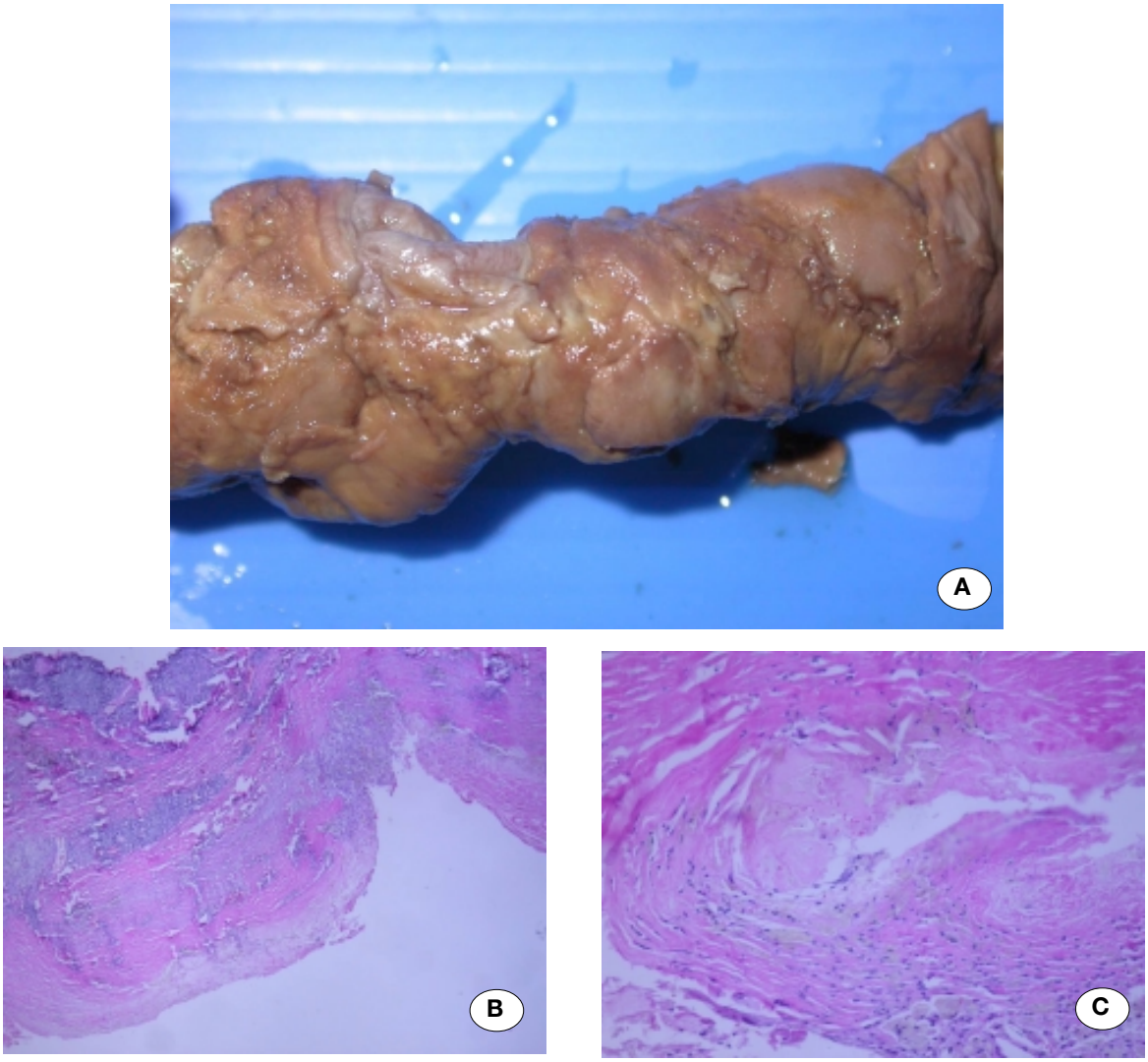


Fig 4. (A) Macroscopic finding shows severe atherosclerotic aorta (B) microscopic finding (low power) demontrates intimal thickening with atheromatous plaque (C) (high power) showing aortic wall with atheromatous plaque consisting of fibrous cap and a central lipid core.

as well as direct action of the HIV itself or by another infection resulting from immunosup-pression.[2-9]

These aneurysms mainly develop in young HIV patients with no sign of atherosclerotic disease, traumatic injury or any other known pathogenic factor. Radiologic examination (CT, MRI or ateriography) usually characterizes the aneurysms of these patients as being of saccular type (pseudoaneurysm or mycotic aneurysm). In addition to vascultis, accelerated atherosclerosis has observed in HIV- infected individual without traditional coronary risk factor (age, LDL, cholesteral, and smoking were strong predictors of atherosclerosis).[1,10,11]

An important study by cardiologists,

endocrinologists and HIV physicians found more atherosclerosis in persons with HIV, and much faster progression than in the general population.[10,11]

Mirza H. et al, report a rare case of an HIV infected patient on HAART (highly active antiretroviral therapy) who presented with a large ascending aortic aneurysm. A noteworthy finding on pathological analysis of the aorta was an etiology of accelerated atherosclerosis rather than the more expected vasculitis.1

To my knowledge, this is the first report of an atherosclerotic aortic aneurysm in an HIV postitive without HAART.

CT scan is the preferred imaging modality to evaluated aortic aneurysm espectially in the emergecy condition because it is widely aviable, fast and able to depict associated findings.[12] Features evaluated both on CT and MR studies included aneurysm location, size and shape (saccular or fusiform; branch involvement ;adjacent soft ttssue mass stranding, and/or fluid and additional findings such as evidence of dissection or rupture but calcification in the aotic wall and presence of gas are evaluated on CT scan only.[6,13]

CT scan of the patient in this report shows evidence of atherosclerotic aneurysm rather than infected (mycotic)aneurysm due to 1) no periaortic soft tissue mass or stranding 2) evidence of atherosclerotic change from foci of rim calcification with intimal irregularity and mural thrombus 3) presence in the descending aorta with fusiform shape and 4) history of heavy smoking more than 10 years that is strong predictor of aterosclerosis. Finally ,it may be expected that with the increase in the number of HIV patients in the population and their life expectancy, the incidence of aneurysms and a development of atherosclerotic vascular disease could become an important complication

Acknowledgement

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