

Tubercular mycotic aortic aneurysm: A case report

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ABSTRACT

Tubercular aneurysms of larger vessels, particularly the aorta is very rare. The first case of tubercular involvement of the aorta in the form of aortitis was reported in 1882 by Weigert and the first case of tubercular mycotic aneurysm of the aorta was reported in 1895. The preoperative diagnosis of tubercular aortic aneurysm is difficult. Even at surgery, determining the tubercular nature of the lesion is problematic. The gross appearance may not be distinctive, and acid-fast stains are unlikely to be performed. We report the case of a young female patient who was started on antitubercular treatment for pleural effusion and was found to have aortic aneurysm, which later on proved to be tubercular in origin.

KEY WORDS: Anti-tubercular treatment, mycotic aneurysm, saccular, tubercle *Bacilli*

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INTRODUCTION

The term mycotic aneurysm was first used by Osler in 1885 when he described a patient with multiple aortic aneurysms secondary to what he termed malignant mycotic endocarditis.^[1] Because the appearance of the lesion resembled a fungal growth, he used the term mycotic aneurysm. Although extension of infection to the walls of the small pulmonary and meningeal arteries from neighboring or contiguous inflammatory foci often caused aneurysms in tubercular cavities and the meninges, tubercular aneurysms of larger vessels, particularly the aorta, whether from within or without, are very rare. The first case of tubercular involvement of the aorta (aortitis) was reported in 1882 by Weigert and the first case of tubercular mycotic aneurysm of the aorta was reported in 1895.^[2]

We report the case of a young female patient who was started on anti-tubercular treatment for pleural effusion and sent home. She was re-admitted when she developed antitubercular therapy (ATT)-induced hepatitis. This time, she was found to have aortic aneurysm which later

on proved to be tubercular in origin. Though the patient was operated upon, despite aggressive management, the patient could not survive.

CASE REPORT

A female patient aged 21 years was admitted with complaints of fever and dyspnea from 20 days. She also had significant weight loss and constitutional symptoms. On examination, vitals were normal; there were clinical findings suggestive of pleural effusion, splenomegaly, and ascites. Chest radiograph revealed bilateral pleural effusion. Pleural tap was done, and serosanguineous colored fluid was tapped. Investigations revealed pleural fluid: Adenosine deaminase - 73.9 U/L, cytology - 162 white blood cell (N-10%, L-90%), proteins - 3.2 g/dl, culture - sterile, Gram stain-negative, and acid fast *Bacilli* (AFB)-negative. Hemogram: Total leukocyte count (TLC) - 6800/cmm, polymorphs 67%, lymphocytes 27%, hemoglobin - 10.2 g%, platelets - 209,000/cmm, and erythrocyte sedimentation rate (ESR) - 30 mm 1st h. Biochemistry: Random blood sugar - 76 mg%,

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urea - 32 mg/dl, creatinine - 0.8 mg/dl, sodium - 137 mEq, potassium - 4.0 mEq, and chloride - 102 mEq. Liver function tests were normal. Blood culture was sterile. No organisms were detected on Gram staining of sputum, sputum culture was sterile and it was negative for AFB. The patient was started on anti-tubercular treatment and discharged.

After 45 days, she presented with a history of yellowish discoloration of eyes from 20 days and palpitations from 15 days. There was no dyspnea or chest pain. Chest radiograph revealed globular mass adjacent to left heart border as shown in Figure 1. On detailed work up with computed tomography (CT) thorax followed by angiography, there was a saccular pseudoaneurysm in descending thoracic aorta with impending rupture [Figures 2-4] with left pleural effusion and minimal pericardial effusion. Hemogram: TLC - 5800/cmm, polymorphs 89%, lymphocytes 8%, hemoglobin - 11.7g%, platelets - 209,000/cmm, ESR - 75 mm 1st h. HIV ELISA was nonreactive. Biochemistry: Random blood sugar - 80 mg%, urea - 17 mg/dl, creatinine - 0.8 mg/dl, sodium - 131 mEq,

potassium - 4.4 mEq, and chloride - 98 mEq. Liver function tests: Bilirubin (total - 7.2%, conjugated - 5.8 mg%), serum glutamic oxaloacetic transaminase - 550 IU, serum glutamic pyruvic transaminase - 251 IU, and alkaline phosphatase - 156 IU. Finally, the patient was diagnosed to have disseminated tuberculosis (TB) with ATT-induced hepatitis. She was started on modified ATT and planned for surgery. In the meantime, she developed upper gastrointestinal (GI) bleed and immediately was taken up for surgical intervention by the department of cardiothoracic surgery. Aorto-aorto bypass grafting with excision of aneurysm with polytetrafluoroethylene graft with feeding jejunostomy was performed along with repair of esophagus as she had aorto-esophageal fistula. Again after 2 days, second surgery was performed in which diversion cervical esophagostomy and gastro-esophageal junction transection with exclusion with tube gastrostomy was done. However, later, patient developed septicemia and finally expired on the 17th day of admission. Aortic aneurysm histopathology report, which was received later on, revealed epithelioid granulomas and was positive for AFB.



Figure 1: Chest radiograph posteroanterior view showing a rounded opacity along the left heart border

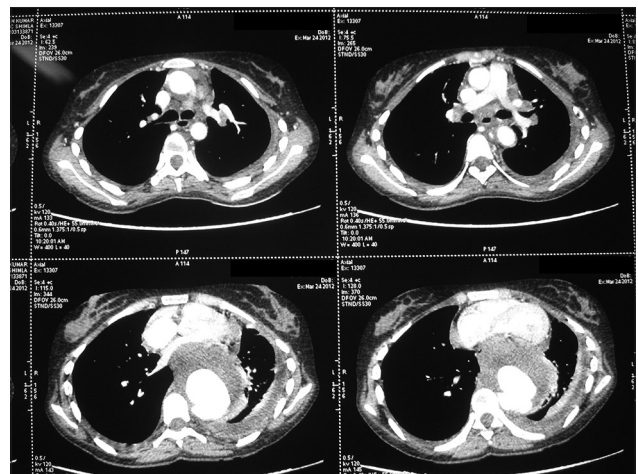


Figure 2: Computed tomography chest (axial sections) showing aortic aneurysm

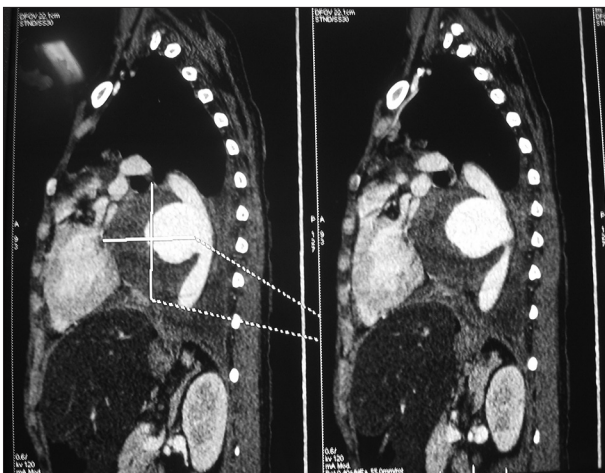


Figure 3: Computed tomography chest showing aortic aneurysm with impending rupture

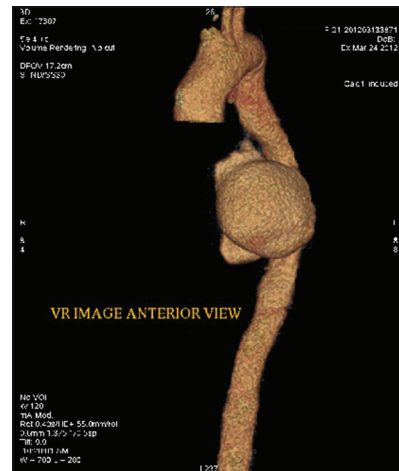


Figure 4: VR image anterior view of the aortic aneurysm

DISCUSSION

A tubercular aneurysm of the aorta is exceedingly rare. Reviews by Haythorn^[3] in 1913 and by Silbergleit *et al.*^[4] in 1965, in both the pre- and post-antibiotic era, suggest that this lesion has been rare. In a recent population-based series of patients with disseminated TB (a group that is at increased risk of tubercular aortic aneurysm), not a single case of tubercular aortic aneurysm was identified over a 15 years period.^[5] Moreover, most of the tubercular aortic aneurysm case reports are from the industrialized world, where the prevalence of TB has declined over the past half-century.

Tubercle *Bacilli* may reach the aortic wall in one of three ways. The *Bacilli* may get implanted directly on the internal surface of the vessel wall, the *Bacilli* may be carried to the adventitia or media by the vasa vasorum, or the involvement of the vessel wall may occur by direct extension or indirectly via the lymphatics from a contiguous focus such as a lymph node or paraspinal abscess. In the majority of the reported cases of tubercular aortic aneurysm (75%), a contiguous focus of TB was described, mainly lymph nodes (63%); the other sites included paraspinal abscess, lung, pericardium, vertebrae, and prostate (37%). In the remaining 10 reported tubercular aortic aneurysm cases (25%), no contiguous focus of disease was described, thus implicating either of the first two mechanism described above.^[2] The patients in whom a primary contiguous focus could not be identified, they may be termed as having primary mycotic aneurysm, a term coined by Crane.^[6] In our case also, there was an adjacent focus of infection in the form of a lymph node that probably eroded into the aorta.

In our patient, ATT was started on the basis of findings of pleural effusion. The diagnosis of aortic aneurysm was made only after CT thorax was done for a suspected mass adjacent to left cardiac border, which was followed by angiography to confirm the diagnosis. In the series by Long *et al.*,^[2] aneurysms of the thoracic and abdominal aorta occurred with equal frequency. Most of the aneurysms were saccular (98%) and false (88%). Our patient also had a saccular aneurysm in the descending thoracic aorta.

As was found in the series by Long *et al.*,^[2] one or more of the three clinical scenarios suggested tubercular aortic aneurysm in 39 of 40 patients (98%). Each scenario suggested a complication of tubercular aortic aneurysm that may be an indication for surgical intervention: (1) Persistent chest, abdominal, or back pain, (2) hypovolemic shock or other evidence of major bleeding, particularly into the lung or GI tract, but also into the pleural space, peritoneal cavity, retro-peritoneum, or pericardial space, or (3) palpable or radiographically visible para-aortic mass, especially if expanding or pulsatile. Our patient had palpitations as the only symptom and radiological para-cardiac opacity on chest radiograph.

The patient developed major upper GI bleeding through the aorto-esophageal fistula and required urgent surgery. Rupture of tubercular aortic aneurysm into the digestive tract is even rarer, with only a few cases being reported in literature. Amonkar *et al.*^[7] reported a 60-year-old male patient who presented with massive hematemesis and died suddenly. Autopsy revealed the cause to be a ruptured aortic aneurysm.

In India, Choudhary *et al.*^[8] in their study over 3 years reported 5 young patients (22–40 years) with tubercular pseudoaneurysm. Site of involvement included ascending aorta, distal aortic arch, proximal descending thoracic aorta, distal descending thoracic aorta, and infrarenal abdominal aorta. All patients either had received ATT previously or were receiving it at the time of presentation. Rapid deterioration in the clinical status was the most marked clinical feature.

In addition to clinical suspicion and suggestive findings on chest radiographs, contrast-enhanced CT scan is the most reliable means of demonstrating the aneurysm, and it often provides useful information on the status of the para-aortic tissue as well. In our case also, it was the saccular appearance on CT chest that was suggestive of an aortic aneurysm. Because the patient had disseminated TB, a remote possibility of tubercular aortic aneurysm was kept, which was confirmed later on.

In a patient when there is disseminated TB, persistent chest pain, abdominal pain or back pain, evidence of major bleed with hypovolemic shock or a palpable or radiologically visible mass, tubercular aortic aneurysm should be considered. The treatment for symptomatic tubercular aortic aneurysm is clearly both medical and surgical. No patients survived without medical therapy. Both the patients who underwent surgery but did not receive anti-tubercular drugs died, and no patient survived without surgery in the series by Long *et al.*^[2] They also found that despite 1 or more months of anti-TB drugs, progression of the lesion resulting in death or surgery occurred in 17 of the total of 41 cases. It is conceivable that access to the aneurysm by what is otherwise an effective anti-tubercular drug regimen is limited to the aortic blood (the small vessels within the vicinity of the aneurysm were invariably thrombosed) which may not easily penetrate the laminated thrombus that forms the wall of the aneurysm. Our patient also received anti-tubercular drugs for 45 days, but she had progression of her aneurysm despite the treatment, though the drugs were modified after admission to the hospital.

It must be recognized, however, that although the presence of an aneurysm and its mycotic nature might be predicted preoperatively, the likelihood of it being tubercular might not be anticipated. In over one-third of the patients in the above-mentioned series,^[2] TB was not diagnosed at presentation. Even at surgery, determining the tubercular nature of the lesion is problematic. The gross appearance

may not be distinctive, and acid-fast stains are unlikely to be performed.

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Conflicts of interest

There are no conflicts of interest.

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