Egyptian Journal of Chest Diseases and Tuberculosis (2014) **63**, 523–527

The Egyptian Society of Chest Diseases and Tuberculosis

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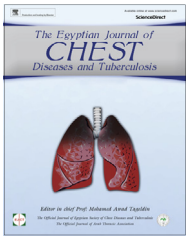

CASE REPORT

Dissecting aortic root aneurysm and severe aortic regurgitation following pulmonary tuberculosis

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Received 21 November 2013; accepted 2 December 2013
Available online 22 December 2013

KEYWORDS

Aneurysm;
Aortitis;
Tuberculosis;
Aortic regurgitation;
Aortic root

Abstract Aneurysms of the aortic root and ascending aorta are often due to degenerative disease of media but tuberculosis is an important but extremely rare cause of aortic root dilatation especially in tropical countries like India where tuberculosis is endemic. Tubercular aneurysmal dilatation of aorta with dissection leading to aortic regurgitation is a rare but important complication of tuberculosis. With worldwide resurgence of tuberculosis due to increasing incidence of drug-resistant tuberculosis and its association with acquired immunodeficiency syndrome, the tubercular aneurysm has become a real clinical entity. Although tubercular aortitis is fairly common, tuberculous mycotic aneurysm of the aorta is rare, with involvement of the aortic root being exceedingly uncommon.

Here we describe a case of 18 year old male presenting with severe breathlessness and was found to have dissecting aortic root aneurysm with aortic regurgitation with active pulmonary tuberculosis and spinal deformity in the form of kyphoscoliosis. He underwent a Bentall procedure, and excised aortic root tissue showed epithelioid cell granulomas with panarteritis.

Case report

A 18-year-old male was admitted with complaints of recurrent chest pain, progressive dyspnea with exertion, and inability to walk for more than 100 m. He complained of fatigue, weight loss, night sweats, and adenopathy of his left axilla and neck for past 3 months. Physical examination revealed diastolic murmur in right second intercostal space with visible pulsation in the suprasternal region and neck, apex beat was displaced downwards into the 6th intercostal space. His admission chest radiograph demonstrated cardiomegaly, left sided lung was markedly destroyed due to active pulmonary tuberculosis,

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Peer review under responsibility of The Egyptian Society of Chest Diseases and Tuberculosis.

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chest radiograph also suggestive of marked spinal deformity in the form of kyphoscoliosis probably due to extensive pulmonary fibrosis or as a sequel of tubercular infection of spine. The patient underwent cardiac evaluation with 2-dimensional echocardiography which revealed a severely dilated aortic root measuring about 55 mm with severe aortic regurgitation, left ventricular ejection fraction of 55%. Patient underwent cardiac computed tomography which demonstrated dissection and dilation of aortic root and ascending aorta rest of the aorta is normal in caliber also the coronary arteries were normal. Patient was a known case of primary pulmonary tuberculosis for which he received antitubercular 4-medication regimen for 3 months.

After his full workup patient and attendants were fully explained about the whole scenario and clinical condition and about the high risk involved in surgery. After taking informed consent the patient was operated and Bentall procedure was done with replacement of incompetent aortic valve and dilated ascending aorta with valved conduit of 25 mm mechanical valve size. The excised aortic tissue was sent for histopathological examination which revealed panarteritis with epithelioid cell granulomas in background of chronic inflammatory cells.

In postoperative period the patient was ventilated for 36 h and was then extubated. In immediate postoperative period the patient maintained his hemodynamic status and was on minimal inotropic support which was soon weaned off and also patient drained only about 300 ml of blood through chest and mediastinal drains which was removed on the second postoperative day and the patient was started treatment with injectable streptomycin. Patient maintained normal blood gases for almost 48 h after that patient stated developing tachypnoea and started retaining carbonmonoxide in blood and became drowsy following this the patient was immediately reventillated electively, the patient improved and was again extubated and aggressive chest physiotherapy was started but the patient was not able to maintain normal blood gases and was again ventilated. Apart from respiratory dysfunction patient was maintaining his hemodynamic status very well and without any inotropic supports, his renal and hepatic functions were also within normal range. Later the patient developed acute respiratory distress syndrome as evident by fluffy opacities found in his chest X ray. Despite the aggressive antibiotic cover and ventilatory support patient succumb to his illness after 10 days of surgery due to respiratory failure (Figs. 1–5).

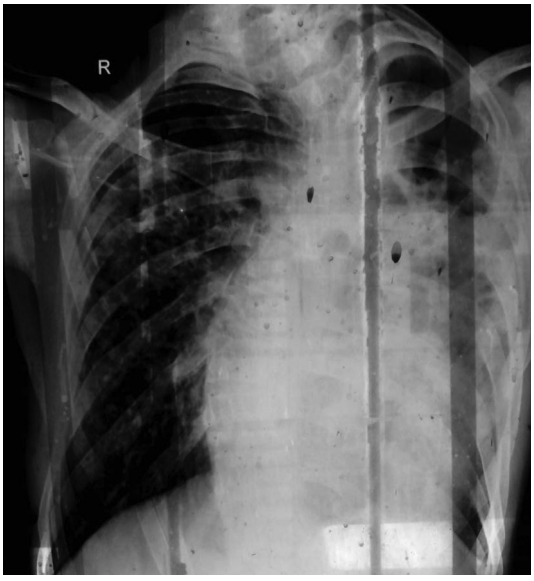

Figure 1 X ray chest showing destroyed left lung with cardiomegaly with spinal deformity in the form of kyphoscoliosis.

Figure 2 Angiographic film showing dilated aortic root with dissection.

Discussion

Dissecting aortic aneurysms is most often caused by atherosclerosis, other causes includes:

- Marfan syndrome (a genetic connective tissue disorder)
- Other non-specific connective tissue disorders (characterized by a family history of aneurysms)
- Presence of a bicuspid aortic valve
- Syphilis
- Tuberculosis and trauma are rare causes

Tuberculous dissecting aneurysms of the aorta is exceedingly rare, but the lesion is uniformly fatal if not diagnosed promptly. Typical clinical scenarios include evidence of tuberculous lymph nodes in 70% of cases, with 1 or more of 3 presentations: (1) fever and persistent pain related to the location of the aneurysm, (2) hypovolemic shock or other evidence of massive bleeding, or (3) pulsatile, rapidly expanding paraaortic mass [1]. The perforation of the aortic wall is generally surrounded by thrombotic debris and inflammatory tissue [2]. Out of these presentations our patient has enlarged cervical lymph nodes with complains of fever and weight loss and dyspnoea and chest pain.

Establishing the diagnosis of infectious aortitis or mycotic aortic aneurysm early is critical because this condition is associated with a high rate of rupture and subsequent mortality if left untreated [9–13]. The diagnosis of tuberculous aortitis is very difficult to establish because this disorder is exceedingly rare and can mimic Takayasu arteritis [14]. Delay in diagnosis