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Case Report

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A Rare Trio of Takayasu Artertitis with Extrapulmonary Tuberculosis and Myocarditis

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Introduction

Takayasu arteritis is a rare chronic inflammatory arteritis predominantly affecting the aorta and the large vessels. This progressive wall inflammation leads to wall thickening and stenosis, producing a varied range of ischemic symptoms and can even lead to aneurysms with high morbidity and increased risk of early death. It is more commonly seen in Japan, Southeast Asia, India, and Mexico. **(**2**)** It is most commonly seen in the 2nd and third decades of life. Etiopathogenesis is poorly understood. Evidence implicating Mycobacterium tuberculosis has been provided for more than six decades. **(**3, 4**)** Until now,

few reports of co-occurrence of adult Takayasu arteritis associated with active tuberculosis have been published.

We report here the case of co-occurrence of extrapulmonary tuberculosis & type I Takayasu arteritis in an adult female.

Case Report

A 28-year-old female patient was referred to the hospital with a one-month history of fever, dry cough, and four days history of shortness of breath. The patient also reported a weight loss of 3 kgs. The patient was free from co-morbid conditions and had no history of contact with Tuberculosis patients or any family history of vascular or rheumatological diseases.

On admission, she was febrile with a temperature of 102^0 Fahrenheit and was pale. Physical examination revealed feeble right brachial and right. Radial artery pulses compared to the left side. Peripheral pulses in lower limbs were full, and no bruit was detected over the abdomen or elsewhere. Respiratory examination revealed decreased breath sounds in right lower chest and fine crepitations in the left lower chest. Auscultation showed a heart rate at 108 beats/min with no added sounds. All other systemic examinations were normal.

Laboratory findings were: White blood cells at $8600/mm^3$, hemoglobin at 8.6 g/dl, platelet counts at $522,000/mm^3$.; Serum electrolytes, liver and kidney function tests, and urinary analysis were normal. Viral markers were negative for hepatitis B and C, human immunodeficiency virus. Antinuclear antibody, antineutrophil cytoplasmic antibodies, antiphospholipid and anticardiolipin antibodies were negative, serum BNP was 189 pg/ml. Pleural fluid cell count was $2000/mm^3$ with 40% lymphocyte predominance, pleural fluid glucose was 90mg/dl, and pleural fluid protein was 5.4 g/dl, ADA was 152.2 U/l, and LDH 781 U/l consistent with tuberculous pleural effusion. Pleural fluid gram stain and fungal stain were negative along with cultures. Sputum for gene Xpert- MTB, AFB stain, and pyogenic cultures were negative. USG chest was suggestive of a large single locule pleural effusion on the right side with underlying collapse/consolidation. The patient was started on anti-tubercular drugs. On day 3 of admission patient developed a sudden onset of respiratory distress for which she required non-invasive ventilation. On evaluation Chest X-ray was s/o pulmonary oedema with bilateral pleural effusions and BNP was found to be 1080 pg/ml. 2D echo revealed the presence of global L.V. hypokinesia with eccentric mitral regurgitation. Cardiologist opinion was taken and the patient was started on inotropes and diuretics which could be gradually tapered. CT aortography was done which showed an abrupt cut-off of the Right Subclavian artery beyond the origin of the internal mammary artery. The left subclavian artery showed severe stenosis beyond the origin of the internal mammary artery. Ascending aorta, aortic arch, and descending aorta was normal in course and calibre along with the presence of right upper lobe consolidation.

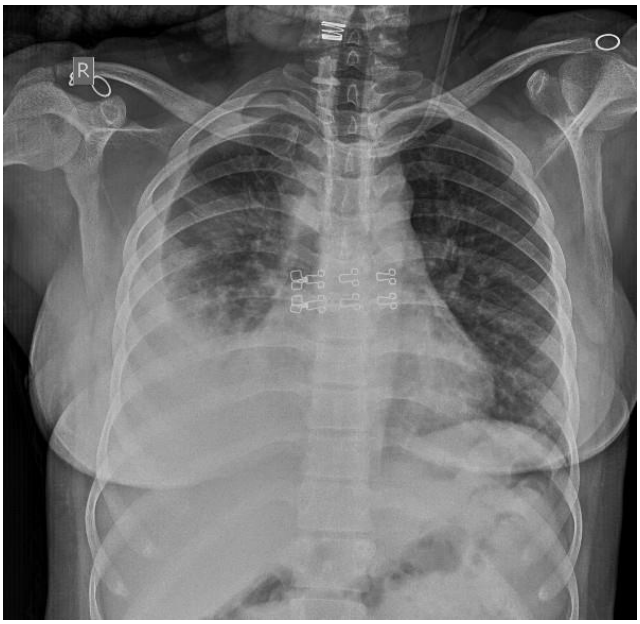

Figure 1

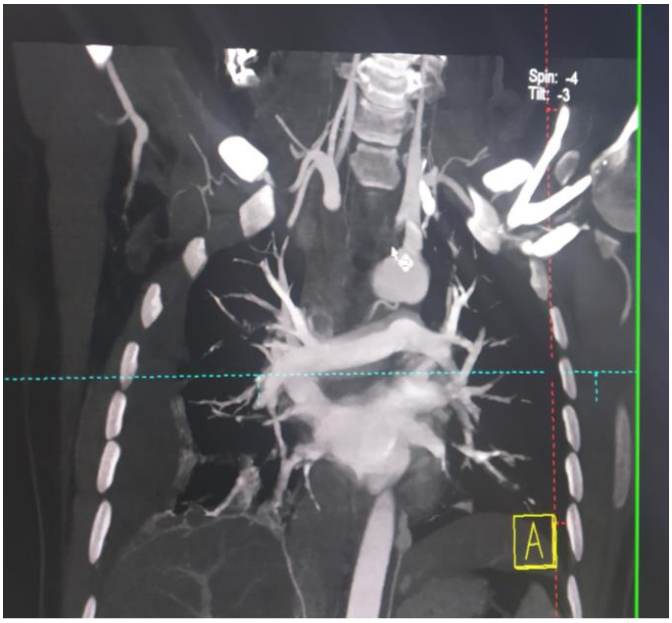

Figure 2

All these data were compatible with the diagnosis of TA according to the final EULAR/PRINTO/PRES TA criteria

Table 1. 1990 ACR criteria for the classification of Takayasu arteritis **(**5**)**

Criterion	Definition
Age at disease onset ≤40 years	Development of symptoms or findings related to Takayasu arteritis at age ≤40 years
Claudication of extremities	Development and worsening of fatigue and discomfort in muscles of 1 or more extremity while in use, especially the upper extremities
Decreased brachial artery pulse	Decreased pulsation of 1 or both brachial arteries
Blood pressure difference >10 mm Hg	Difference of >10 mm Hg in systolic blood pressure between arms
Bruit over subclavian arteries or aorta	Bruit audible on auscultation over 1 or both subclavian arteries or abdominal aorta
Arteriogram abnormality	Arteriographic narrowing or occlusion of the entire aorta, its primary branches, or large arteries in the proximal upper or lower extremities, not caused by arteriosclerosis, fibromuscular dysplasia, or similar causes; changes usually focal or segmental

Table 2. Angiographic classification of Takayasu arteritis **(**6**)**

Type	Vessel involvement
Type I	Branches from the aortic arch
Type IIa	Ascending aorta, aortic arch and its branches
Type IIb	Ascending aorta, aortic arch and its branches, thoracic descending aorta
Type III	Thoracic descending aorta, abdominal aorta, and/or renal arteries
Type IV	Abdominal aorta and/or renal arteries
Type V	Combined features of types IIb and IV

The patient was placed on anti-tubercular therapy and received 500mg of methylprednisolone for 3 days following which it was tapered to 45 mg/day. Within six months of therapy, the patient showed clinical recovery of fever, dry cough, and night sweats. The steroid dose was gradually decreased. Repeat 2D echo after 2 months showed improved LV function and the presence of only mild mitral regurgitation.

Discussion

We report the clinical findings of the co-occurrence of extrapulmonary tuberculosis and type I TA with severe mitral regurgitation and myocarditis in an adult patient.

An exhaustive electronic search found only 76 cases of TA published over 25 years. Among them, very few adults presented with extrapulmonary tuberculosis. The early diagnosis of Takayasu arteritis remains challenging. The diagnosis is often established in the advanced stages of the disease when presenting with complications. Fever, weight loss are common symptoms of infectious diseases, especially tuberculosis in India. In our report, the diagnosis of tuberculous pleural effusion was made based on high ADA levels along with a lymphocyte-predominant pleural effusion. Chronic cough has not been cited as a symptom of Takayasu arteritis. Pulmonary involvement usually consists of luminal stenosis or occlusion of the pulmonary artery on CT angiography scans. Cardiac lesions are seen in up to 8.6% of patients and can involve valve, myocardium and coronary and pulmonary arteries. **(**7**)**

The main causes of heart failure in Takayasu arteritis patients include increased afterload due to renovascular hypertension and aortic regurgitation. Myocardial ischaemia induced by myocarditis or coronary artery involvement & severe pulmonary hypertension due to pulmonary artery involvement was also found to be the causes. **(**8**)** Talwar et al. performed endomyocardial biopsies in Takayasu arteritis patients and found that 8 of 18 and 24 of 54 patients had myocarditis. (8) For the treatment, early use of immunosuppressive therapy seems to be useful. Takeda used steroid therapy in a 15-year-old patient with myocarditis secondary to Takayasu arteritis and found marked alleviation of the patient's symptoms along with improved cardiac morphology and function. **(**9**)** An aggressive immunosuppressive regimen could not be used in our patient due to the presence of extrapulmonary tuberculosis.

Previous studies have shown that Takayasu arteritis tends to concur with tuberculosis. The pathophysiology possibly involves the cross-reactivity against vascular peptides that mimic the antigens of mycobacterium tuberculosis. Soto et al. used IS 6110 and HupB gene sequencing (IS6110 sequence identifies the Mycobacterium tuberculosis complex and the HupB establishes the differences between M. tuberculosis and M. Bovis) in aortic tissues of Takayasu arteritis and found a higher frequency of IS6110 and HupB gene sequences and suggested that arterial damage could be due to mycobacterium tuberculosis patients. **(**10**)**

Conclusions

Various presentations of Takayasu arteritis have been reported including cardiac valvular involvement, pulmonary arterial involvement and pulmonary tuberculosis. However, the combination of myocarditis with severe mitral regurgitation and extrapulmonary tuberculosis is extremely rare and has not been reported so far. The clinical scenario poses a great challenge for rapid diagnosis and an appropriately tailored immunosuppressive regimen because of the presence of co-existing extrapulmonary tuberculosis.

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