

Case Report

Relapse of Takayasu's arteritis with tuberculosis relapse: a rare presentation

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ABSTRACT

Takayasu's Arteritis (TA) is a disease of unknown etiology with incidence between 1.2 to 2.3 cases per million per year. It is a chronic granulomatous arteritis affecting large elastic arteries, predominantly the aorta, its main branches, pulmonary and coronary arteries characterized histologically by an inflammatory cell infiltrate that affects all the layers of the arterial wall. The etiology of TA is not clear but a causal relationship between TA and tuberculosis (TB) have been suggested. The first case of Takayasu's arteritis was described in 1908 by Japanese ophthalmologist Mikito Takayasu. Despite the association with tuberculosis and the similarity between granulomatous lesions in both the diseases, the exact role of *Mycobacterium tuberculosis* in the pathogenesis of TA is still unknown.

Keywords: Takayasu's arteritis, Tuberculosis, Relapse of Takayasu's arteritis

INTRODUCTION

Takayasu's Arteritis (TA) is a disease of unknown etiology with incidence between 1.2 to 2.3 cases per million per year. It is more common in Asians than in other racial group.¹ It is a chronic granulomatous arteritis affecting large elastic arteries, predominantly the aorta, its main branches, pulmonary and coronary arteries characterized histologically by an inflammatory cell infiltrate that affects all the layers of the arterial wall.² The disease occurs more commonly in young females than males with peak incidence between 15 to 20 years of age. The etiology of TA is not clear but a causal relationship between TA and tuberculosis (TB) had been suggested.³

Here we report a case of relapse of tuberculosis associated with relapse of TA in an adolescent girl which strongly favors the association between both of the diseases.

CASE REPORT

A 17-year-old unimmunized female patient admitted with complaints of cough with expectoration, chest pain, low grade fever and generalized weakness since 3 months. Chest pain, cough and weakness were progressive in nature. Fever was low grade and persistent in nature. History of contact with tuberculosis was absent.

In the past, eight years before this presentation, she was diagnosed as a case of tubercular meningitis and had completed course of antitubercular therapy (AKT) with steroids. Five years back i.e. at the age of 12 years she was again diagnosed as confirmed case of tubercular lymphadenopathy with Takayasu arteritis (as left radial pulse was absent at presentation). That time also she received complete course of AKT as per Recommended-revised National Tuberculosis Control Programme (RNTCP) regimen and was declared cured with recovery of radial pulsation.

On general examination, patient was cachexic with weight and height <3rd centile for age. The left radial and popliteal pulsation was absent. Rest of the pulses were normal. Blood Pressure were 110/70 mmHg and 98/72 in right and left upper limb respectively. Similarly in right lower limb and left lower limb it was 180/100 mmHg and 200/130 mmHg respectively.

On systemic examination patient was having tachypnea (+34/min) with bilateral coarse crepitations. Rest of systemic examination was normal.

Complete hemogram was s/o Mild Anemia (Hb - 10.9 gm/dl), normal leucocyte count (9000/cumm) with lymphocytosis (70%). ESR was elevated (45 mm end of 1hour) and C-reactive protein level were raised >320 mg/dl). Her Complete ANA profile, liver function test, renal function test and urine analysis were normal. HIV ELISA was non-reactive. Three consecutive samples of Sputum were positive for AFB. Mantoux test (2TU) was positive (12 mm). ECG, echocardiography and abdominal sonography were normal.

The X-ray chest was s/o bilaterallower lobe patchy consolidation. The CT Thorax showed bronchogenic spread of TB in both lungs with mediastinal lymphadenopathy (Figure 1). Duplex vascular ultrasound revealed normal right radial and popliteal artery while narrowing with significant stenosis of left radial and popliteal artery (Figure 2).



Figure 1: Color Doppler.



Figure 2: CT thorax showing extensive KOCH'S.

Patient was considered to have relapse of Takayasu arteritis with relapse of Koch's. After sending of sputum for mycobacterial culture, treatment was started for management of relapse of pulmonary Koch's.

DISCUSSION

The first case of Takayasu's arteritis was described in 1908 by Japanese ophthalmologist Mikito Takayasu. Takayasu described a peculiar "wreathlike" appearance of the blood vessels in the back of the eye (retina) with absent wrist pulses. It is now known that the blood vessel malformation occurs due to an angiogenic response to the arterial narrowing and that the absence of pulses noted in some patients is because of narrowing of the blood vessels.

The aetiopathogenesis of TA is largely unknown but most of the available data suggest that it is an autoimmune disease and both cellular³ as well as humoral⁴ immune mechanisms are involved in the pathogenesis of the disease. It is one of the first vasculitides to be associated with a specific infectious agent. Despite the association with tuberculosis and the similarity between granulomatous lesions in both diseases, the exact role of Mycobacterium tuberculosis in the pathogenesis of TA is still unknown. Expression of the heat shock protein (HSP)-65 as well as increased infiltration of T-cells in aortic tissue⁵ and raised levels of circulating antimycobacterial HSP65 (mHSP65) antibodies⁶ in patients indicate that HSP65 whether exogenous or endogenous may be a putative antigen stimulating immune responses in the disease.

A similar study reported two cases in whom a tuberculosis process was documented concomitant with TA, and both of them responded well to prednisolone and anti-TB.⁷ Our patient is unique in the sense that five years back she had developed TA with tubercular lymphadenopathy and had received treatment and got completely cured for both of the conditions by standard anti tubercular regimen without steroids. This time patient presented with relapse of tuberculosis with relapse of pulseless disease. To conclude our case reconfirms that there is strong association between the two conditions and till we don't know the exact etiology of TA. All efforts should be carried out to rule out tubercular infection in such patients. Furthermore a trial of AKT should be given to the patients especially if they belong to developing countries or high risk group.

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