

Case Report

Association between Takayasu's arteritis and active tuberculosis in a child: A case report

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Abstract

Takayasu's arteritis (TA) is an autoimmune disease that affects large arteries. A possible relationship between TA and tuberculosis (TB) has been suggested as both diseases have similar chronic inflammatory lesions and occasionally granulomas in the arterial walls. TA commonly presents with cardiac involvement and rarely with sole presentation of ischemic stroke. Response with anti-tubercular drugs and steroid has been well-documented. Only a few studies have been published on TA with TB in children. We present a case of a 9-year-old boy who was presented with low-grade fever, persistent headache, vomiting, and abnormal body movements and on further workup was diagnosed as a case of active TB with TA.

Key words: *Antitubercular therapy, Cardiac presentation, Takayasu's arthritis, Tuberculosis*

Takayasu's arteritis (TA) is a rare systemic, inflammatory large-vessel vasculitis of unknown etiology. If not diagnosed and treated appropriately, TA is potentially life threatening. In acute early phase, patient presents with non-specific symptoms such as hypertension, headache, fever, muscle pain, arthralgia, night sweats, and weight loss. Due to non-specific symptoms and absence of specific laboratory parameters, disease often goes unrecognized in this phase. If left untreated at this stage, it affects aorta, and its main branches leading to concentric wall thickening, fibrosis, and thrombus formation secondary to vessel wall inflammation. Affected vessels may become stenotic or may develop aneurysms. Presenting symptoms at this stage commonly reflect end-organ ischemia such as renal infarction and stroke [1,2].

Diagnosis of TA is based on the clinical criteria and laboratory evidence as large-vessel biopsies are often not possible [3]. Tuberculosis (TB) and TA both are systemic diseases characterized by chronic inflammatory lesions and granuloma suggesting a possible relationship between TA and TB. There have been only a few reported cases of active TB with TA in the pediatric population [4-7]. Therefore, we present such a case of in a 9-year-old child.

CASE REPORT

A 9-year-old boy presented with low-grade, intermittent fever and headache for 1-month, vomiting for 5 days and abnormal body movements followed by unconsciousness for 1-day.

There was no history of convulsions in the past. There was no history of extremities claudication. He had received incomplete treatment for tubercular arthritis of ankle joint 2 years ago. He was developing normally according to his age. There was no family history of hypertension or convulsions; however, family history of chronic cough was present.

On examination, patient was pale, febrile (99.5°F), underweight (body weight was 17 kg, height 110 cm and BMI = 14) with heart rate of 114/min, and respiratory rate of 46/min. Pulses in upper limbs were not palpable and blood pressure (BP) was not recordable while lower limbs pulses were bounding with BP 180/120 mmHg. Neck examination showed a significantly enlarged right cervical lymph node measuring 3.5 cm × 2.8 cm, which was firm, non-tender and not fixed to the underlying structures.

On neurological examination, patient was unconscious with increased tone in all 4 limbs and power of Grade II/V. His deep tendon reflexes were brisk with extensor planter response in both the limbs. On cardiovascular examination, systolic murmur of Grade III/VI was heard over pulmonary area. His respiratory and abdominal examination revealed no abnormal findings. Fundus examination revealed bilateral papilledema. Treatment was started with IV fluids, antibiotics, and IV phenytoin along with supportive measures.

On laboratory workup, his hemoglobin was 8 g/dl with normal total white blood cells and predominant lymphocytosis

and normal platelet count. His sugar and serum electrolytes were normal. His erythrocyte sedimentation rate (120 mm in 1st h) and C-reactive protein (20 mg/dl) were elevated and mantoux test was strongly positive (25 mm). Chest X-ray showed haziness of middle and lower zones of apex of both lung fields, with an enlarged heart shadow. Cervical lymph node biopsy showed caseating granulomatous lesions. Hence, anti-tubercular treatment was started with isoniazid, rifampicin, and pyrazinamide. In view of the difference in pulses and BP, TA was suspected, and relevant investigations were planned.

Electrocardiogram showed sinus tachycardia with left axis deviation and signs of left ventricle hypertrophy. Echocardiography showed left ventricular hypertrophy. Renal color Doppler showed narrowing of both renal arteries and magnetic resonance angiography brain showed narrowing of anterior, middle cerebral arteries with hypoplastic intracranial portion of left vertebral artery (Fig. 1). Computed tomography angiography upper thorax showed severe irregular narrowing of bilateral subclavian arteries (right from the origin), axillary arteries, and brachial arteries with collaterals supplying brachial arteries (Fig. 2). A diagnosis of TA was made as per 1990 American College of Rheumatology (ACR) criteria for TA. Prednisolone, amlodipine was also started and atenolol, spironolactone, and prazosin were also added sequentially due to the persistence of hypertension. With treatment, his fever subsided, consciousness regained, and BP stabilized at 132/88 mmHg in next 5 days. He was discharged after 10 days and is doing well on regular follow-up.

DISCUSSION

TA is predominantly a disease of young adults presenting in second and third decades of life, but can be seen earlier in childhood also [8]. The female: male ratio has varied from 9:1 to 1.3:1 in reports from Japan. In India, female preponderance is

less obvious in children. TA initially presents with nonspecific symptoms like fever, malaise, myalgia, weight loss, and anemia. Approximately, 10-30% patients have various cardiac symptoms including hypertension due to renal artery stenosis or aortic narrowing and fibrosis. Hypertension is often severe and may cause encephalopathy or heart failure [8]. Neurological manifestations that may accompany TA include headache, dizziness, visual disturbance or loss of vision. However, stroke and transient ischemic attacks are uncommon symptom and are rarely reported as the first manifestation [9-10].

The diagnosis of TA may be suggested by a missing pulse or a renal or aortic bruit. However, these are not universal. ACR criteria for diagnosis of TA consisted of: (i) age at disease onset <40 years; (ii) claudication of the extremities; (iii) decreased brachial artery pulse; (iv) BP difference >10 mmHg between arms; (v) bruit over subclavian arteries or aorta; and (vi) arteriogram abnormality, which is not related to arteriosclerosis or fibromuscular dysplasia [3]. The presence of 3 or more of these 6 criteria demonstrated a sensitivity of 90.5% and a specificity of 97.8%.

Patients with TA have increased immune response to *Mycobacterium tuberculosis* antigens, especially 65 kDa heat shock protein, suggesting a role of this organism in immunopathogenesis of the disease [11-13]. There have been a few reported cases of active TB with TA in the pediatric population [4-7]. In our case, tuberculin test was strongly positive and caseating granulomatous lesions were found in cervical lymph nodes suggesting an association between TB and TA. Treatment of TA is based on the use of immunosuppressant to decrease inflammatory activity. Antitubercular drugs with prednisolone or/and cyclophosphamide has been reported as an effective treatment for TA. Anti-hypertensive drugs were used in all the reported cases. Our patient received both steroid and anti-tubercular drugs and responded well.

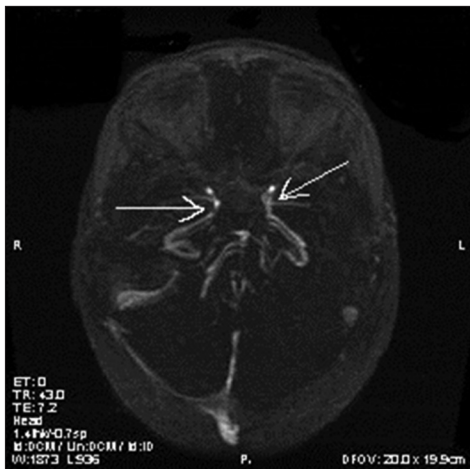


Figure 1: Magnetic resonance angiography showed narrowing of anterior, middle cerebral arteries with hypoplastic intracranial portion of left vertebral artery

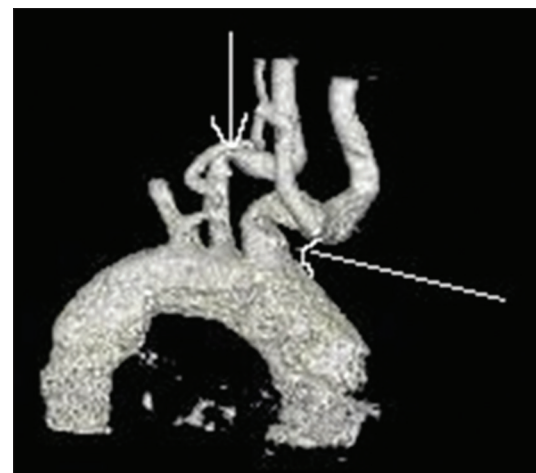


Figure 2: Computed tomography angiography upper thorax revealed severe irregular narrowing of bilateral subclavian arteries (right from the origin), axillary arteries, and brachial arteries

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