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Case Report

Takayasu Arteritis: Tuberculosis or Not Tuberculosis?

Shruti Vyas[1], Alpana S Kondekar[2]*, Varun Anand[3], Radha G Ghildiyal[4], Satish Ghatol[5] and Tanmay Khadpe[5]

[1]SMO, Department of Pediatrics, TN Medical College and BYL Nair Hospital, Mumbai Central, Mumbai, India

[2]Associate Professor, Department of Pediatrics, TN Medical College and BYL Nair Hospital, Mumbai Central, Mumbai, India

[3]Assistant Professor, Department of Pediatrics, TN Medical College and BYL Nair Hospital, Mumbai Central, Mumbai, India

[4]Professor, Department of Pediatrics, TN Medical College and BYL Nair Hospital, Mumbai Central, Mumbai, India

[5]PG Student, Department of Pediatrics, TN Medical College and BYL Nair Hospital, Mumbai Central, Mumbai, India

***Corresponding Author**: Alpana S Kondekar, Associate Professor, Department of Pediatrics, TN Medical College and BYL Nair Hospital, Mumbai Central, Mumbai, India

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Abstract

Takayasu arteritis (TA) is a systemic vasculopathy affecting large blood vessels that can progress to cause vital organ ischemia. A possible relationship between TA and tuberculosis has been proposed. Both diseases show similar pathological changes in the form of granulomas in the arterial walls. The diagnosis of childhood TA becomes difficult due to the often non-specific character of symptoms including headaches, fever, dyspnoea, weight loss, vomiting, abdominal pain and musculoskeletal symptoms. Also there may be sometimes symptoms masquerading as that of tuberculosis. The combination of systemic symptoms of inflammation, decreased or absent pulses along with signs of organ ischemia like transient ischemic attacks or renal vascular hypertension should raise the level of suspicion for TA. An 11 year old female presented with history of intermittent fever, abdominal pain and cough since four months. She had feeble radial and brachial pulses with absent dorsalis pedis pulse. CT chest and abdomen revealed circumferentially enhancing mural thickening of aorta and its branches along with necrotic lymphadenopathy suggestive of tuberculous etiology. Aortogram was done to confirm diagnosis which showed luminal narrowing involving descending thoracic aorta in its entire extent consistent with type 5 TA. In conclusion, diagnosis of TA requires high index of clinical suspicion and suspected TA mandates vascular imaging.

Keywords: *Takayasu Arteritis; Tuberculosis; Renovascular Hypertension*

Introduction

Takayasu's arteritis (TA) is a chronic inflammatory disease that involves the aorta, its branches and the pulmonary arteries resulting in varying degree of stenosis, occlusion or dilatation of the involved vessels. Various studies found correlation of TA with hypertension (82.6%), headaches (31%), fever (29%), dyspnoea (23%), weight loss (22%), vomiting (20.1%), abdominal pain (16.6%), and musculoskeletal symptoms (14%) [1]. The incidence of TA in children is unknown. The diagnosis of childhood TA remains challenging due to the often non-specific character of symptoms.

The etiopathogenesis of TA is still scantily understood. An association of TA with tuberculosis has been suggested. Active tuberculosis has been recognized in up to 20% of patients with TA [2]. In spite of the clinical relationship between both conditions, no evident link has been proved until now. Few published data also exist that advocate the connotation of TA with Tuberculosis [3-5]. Here we report a case of co-occurrence of tuberculous symptomatology with type 5 TA which also posed a dilemma in the treatment.

Case Report

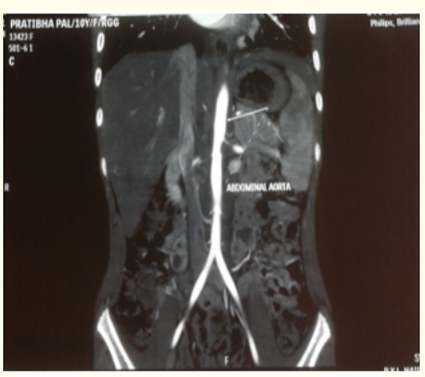
An 11 year old girl presented with a 4 months' history of intermittent fever, breathlessness, abdominal pain and cough. A year ago, she had received Anti-tubercular therapy (ATT) for presumptive clinical diagnosis of Tuberculosis for similar symptoms along with significant abdominal lymphadenopathy. Her current admission to indoor was in view of breathlessness accompanied by mediastinal widening in

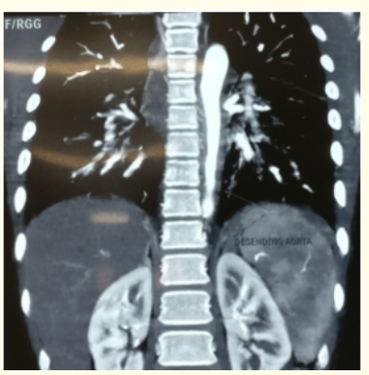
chest X-ray. On clinical examination all peripheral pulses were found to be feeble and blood pressure (BP) was not recordable in bilateral upper limbs. Complete blood count was suggestive of anemia (hemoglobin- 8.6gm%), total leucocyte counts of 7900 cells/mm and total platelet counts of 3.8 lakhs. Her ESR (82 mm/hour), CRP (21 mg/L) were raised and ANA, ds-DNA were negative. CT chest and abdomen revealed multiple necrotic mediastinal and abdominal lymphadenopathy suggestive of tuberculous etiology. There was circumferentially enhancing mural thickening of aorta and its branches. Aortogram showed luminal narrowing involving descending thoracic aorta in its entire extent, its left subclavian branch and abdominal aorta till origin of renal arteries suggestive of type 5 TA. Echocardiography showed bicuspid aortic valve and left ventricular hypokinesia with ejection fraction of 35%. She was restarted on category II ATT in view of CT guided biopsy of mediastinal lymph node showing caseous granulomatous necrosis. Prednisolone was started at 2 mg/kg/day after 2 weeks. Later, intravenous pulse cyclophosphamide every 2 weekly was planned after paediatric rheumatologist opinion. She was discharged after receiving uneventful first dose of cyclophosphamide but again presented after 10 days of discharge with multiple episodes of seizures and altered sensorium along with hypertensive emergency. Hypertension was refractory to multiple anti-hypertensive drugs thus renal artery stenting was done. Post procedure BP was within normal levels on three anti-hypertensive drugs. She required long term prednisolone and methotrexate for control of disease activity.

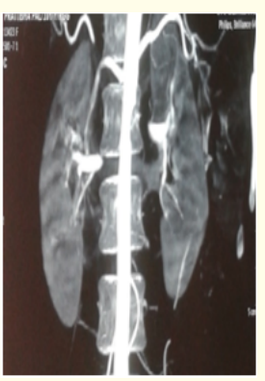
Discussion

Both TA and tuberculosis are chronic granulomatous diseases. The role of tuberculosis in the pathogenesis of TA is unclear. Aggarwal., *et al*. showed that patients (36 cases) with TA have increased immune response to *Mycobacterium tuberculosis* antigens, in particular to its 65 kDa, a heat shock protein that has been also found to be expressed in the arterial wall of TA patients [6]. In a Mexican case control study Soto., et al. identified in a higher frequency of IS6110 and hupB gene sequences of *Mycobacterium tuberculosis* and *bovis* in the aortic tissue of TA patients and in tuberculosis compared to patients with atherosclerosis with important statistical differences suggesting that arterial damage could occur due to the previous infection with *Mycobacterium tuberculosis* [7]. There have been a few reported cases of active tuberculosis with TA in the pediatric populace. In two of the cases, patients responded to treatment with ATT and prednisolone [4,5]. On the other hand, Mukherjee., *et al*. reported a case who also required cyclophosphamide and azathioprine [8]. Recently, Khemiri., *et al*. reported a case where methotrexate was added for controlling relapse [3].

Constitutional symptoms being 2 times more widespread in pediatric age group as contrast to adults if presents with weaken pulses, high BP and localised bruits inkling the diagnosis and imaging should be warranted for confirmation [9]. It is a systemic vasculopathy that can progress to cause vital organ ischemia thus protracted follow up is recommended. An understanding of TA, and appropriate suspicion, is the first move for early clinical diagnosis for the paramount outcome.







New angiographic classification of TA [10]

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

Conclusion

This case emphasizes the importance of clinical examination in early diagnosis of TA. Treatment should be initiated as almost immediately when diagnosis is made because disease may be hastily progressive as in described case. Patient should be narrowly monitored to intervene, previous to the initiation of complications.

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Conflict of Interest

None.

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Contribution of Authors

- Preparation of first draft: SV, AK.
- Literature Search: VA, AK, RGG, TA, KJ.
- Conceptualization: RGG, AK, SG, TK, VA.
- Intellectual inputs for improvement of Manuscript: RGG, AK, SG, TK.
- Approval of Final Draft: AK, RGG.

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