Takayasu Arteritis: Tuberculosis or Not Tuberculosis?

Shruti Vyas1, Alpana S Kondekar2*, Varun Anand3, Radha G Ghildiyal4, Satish Ghatol5 and Tanmay Khadpe5

1SMO, Department of Pediatrics, TN Medical College and BYL Nair Hospital, Mumbai Central, Mumbai, India
2Associate Professor, Department of Pediatrics, TN Medical College and BYL Nair Hospital, Mumbai Central, Mumbai, India
3Assistant Professor, Department of Pediatrics, TN Medical College and BYL Nair Hospital, Mumbai Central, Mumbai, India
4Professor, Department of Pediatrics, TN Medical College and BYL Nair Hospital, Mumbai Central, Mumbai, India
5PG 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


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
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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].

Discussion

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 inking 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]

<table>
<thead>
<tr>
<th>Type</th>
<th>Vessel involvement</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Branches from the aortic arch</td>
</tr>
<tr>
<td>Ila</td>
<td>Ascending aorta, aortic arch and its branches</td>
</tr>
<tr>
<td>IIb</td>
<td>Ascending aorta, aortic arch and its branches, thoracic descending aorta</td>
</tr>
<tr>
<td>III</td>
<td>Thoracic descending aorta, abdominal aorta, and/or renal arteries</td>
</tr>
<tr>
<td>IV</td>
<td>Abdominal aorta and/or renal arteries</td>
</tr>
<tr>
<td>V</td>
<td>Combined features of types IIb and IV</td>
</tr>
</tbody>
</table>

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.

Acknowledgment

The authors thank Dr. Ramesh Bharmal, Dean- T.N. Medical College and BYL Nair Hospital and Dr Sandeep Bavdekar, Head of the Department, Pediatrics for granting permission to publish this manuscript.

Conflict of Interest

None.

Funding

None.

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.

Bibliography

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Volume 7 Issue 11 November 2018
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