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Case Series

Tubercular aortitis mimicking Takayasu arteritis: a diagnostic challenge in an endemic region

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ABSTRACT

Takayasu arteritis (TA) is a chronic granulomatous large-vessel vasculitis involving the aorta and its major branches, with a possible pathogenic association with *Mycobacterium tuberculosis*, particularly in endemic regions due to shared immunopathological and epidemiological features. We report three pediatric cases of TA with concurrent or recent tuberculosis infection, all presenting with systemic symptoms, hypertension, and vascular insufficiency; computed tomography (CT) angiography confirmed large-vessel involvement, and inflammatory markers were elevated in all. Tuberculosis screening revealed positive Mantoux tests in each case, with one child having microbiologically confirmed rifampicin-sensitive tuberculosis on cartridge-based nucleic acid amplification test (CBNAAT) from gastric aspirate and two showing granulomatous lymphadenitis suggestive of tubercular etiology. Management included antihypertensives, anti-tubercular therapy, and carefully timed immunosuppression, resulting in clinical improvement and better blood pressure control, although vascular changes persisted. This case series highlights the clinically significant coexistence of TA and tuberculosis in children from endemic regions. The overlapping clinical, radiological, and immunological features pose diagnostic and therapeutic challenges. Screening for tuberculosis should be considered in all patients with suspected or confirmed TA prior to initiating immunosuppressive therapy. Early recognition and a coordinated treatment strategy integrating anti-tubercular therapy with immunosuppression are crucial for optimal patient outcomes.

Keywords: Takayasu arteritis, Vasculitis, *Mycobacterium tuberculosis*, Immune response, Heat-Shock protein

INTRODUCTION

Takayasu arteritis (TA) is a chronic, idiopathic, granulomatous large-vessel vasculitis primarily affecting the aorta and its major branches. It most commonly occurs in young women, particularly in the Asian population, and is characterized by segmental stenosis, occlusion, dilatation, or aneurysm formation in affected vessels.[1] The disease typically progresses through an early systemic inflammatory phase—marked by constitutional symptoms—followed by a chronic occlusive phase resulting in vascular insufficiency, pulselessness, hypertension, and end-organ ischemia. Histologically, TA demonstrates granulomatous inflammation with disruption of the elastic lamina, infiltration of T-cells and macrophages, and varying degrees of fibrosis in the vessel wall.[2,3]

The etiology of TA remains unclear, but infectious triggers, particularly *Mycobacterium tuberculosis*, have been proposed based on epidemiologic, immunologic, and pathological parallels. The prevalence of TA appears higher in regions endemic for tuberculosis, suggesting a potential geographical and epidemiological link. Several studies have detected mycobacterial DNA, heat-shock proteins (HSP65), and immune responses cross-reactive between *M. tuberculosis* antigens and host vascular proteins in patients with TA, supporting a model of molecular mimicry and aberrant immune activation.[4,5] Additionally, latent or prior TB infection is reported with

increased frequency in TA cohorts compared to the general population.[6] We are reporting three cases of TA having evidence of *Mycobacterium tuberculosis* infection.

CASE SERIES

Case 1

A 12-year-old male child presented to our emergency department with complaints of fever for 3 days and left-sided focal seizures. Patient had a heart rate of 88/min, respiratory rate of 22/min, SPO_2 of 98% on room air, blood pressure of 190/104 mmHg (stage 2 hypertension), peripheral pulses absent, and fundus examination showed grade 2 papilledema. Patient was treated in the lines of hypertensive emergency. Lab investigations are mentioned in Table 1. Echocardiogram (ECHO) revealed left ventricular hypertrophy and flow acceleration in the abdominal aorta with spill. Computer tomography (CT) angiography showed circumferential thickening and marked luminal narrowing of distal most descending thoracic aorta and suprarenal abdominal aorta, and non-contrast opacification of the left proximal renal artery, right subclavian artery and proximal right vertebral artery. Patient was continued on anti-hypertensive medications and started on immunosuppressive therapy, keeping a diagnosis of TA. Due to the persistence of fever and association of TA with tuberculosis in endemic regions, Montaux was done, which was positive, and CBNAAT via gastric aspirate showed Rifampicin sensitive tuberculosis. The patient was started on first-line ATT and showed clinical improvement initially, but we lost the patient on follow-up due to hypertensive intracranial bleed.

Case 2

A 10-year-old female child presented with complaints of progressive fatigue, intermittent low-grade fever for one month, and pain in the left upper limb on exertion for two weeks. There was no history of seizures or visual disturbances. On examination, the child had a heart rate of 92/min, respiratory rate of 20/min, and oxygen saturation of 99% on room air. Blood pressure recordings showed a significant discrepancy between limbs, with right upper limb BP of 164/96 mmHg and left upper limb BP of 118/70 mmHg. Left radial and brachial pulses were feeble, while lower limb pulses were palpable. A bruit was audible over the left supraclavicular region. Laboratory investigations revealed anemia, elevated inflammatory markers, and mildly deranged renal function (Table 1). CT angiography revealed long-segment concentric wall thickening with luminal narrowing of the aortic arch extending into the left common carotid and left subclavian arteries, with mild narrowing of the descending thoracic aorta. A provisional diagnosis of TA was made, and the child was started on oral corticosteroids along with antihypertensive therapy. Given the endemic background and persistent low-grade fever with additional findings of bilateral cervical lymphadenopathy, tuberculosis evaluation was undertaken. Mantoux test was strongly positive (18 mm induration), and FNAC performed on lymph node aspirate showed granulomatous inflammation; however, CBNAAT was negative for tuberculosis. Immunosuppressive therapy was withheld temporarily, and the patient was initiated on first-line anti-tubercular therapy. Over the next six weeks, the child showed clinical improvement with resolution of fever, reduction in inflammatory markers, and better blood pressure control, after which low-dose steroids were cautiously reintroduced.

Table 1: Laboratory parameters.

Parameters	Patient 1	Patient 2	Patient 3
Haemoglobin (g/dl)	8.3	9.2	8.8
TLC ($\times10^3$/mm^3)	12.5	8.5	9.6
Platelet ($\times10^3$/mm^3)	125	156	98
ALP (U/l)	<20	<20	<20
AST (U/l)	13	10	12
ALT (U/l)	18	19	22
Total bilirubin (mg/dl)	1.3	1.2	0.9
Blood urea nitrogen (mg/dl)	118	88	104
Serum creatinine (mg/dl)	1.9	1.2	2.1
ESR (mm/hour)	60	56	68
CRP (mg/dl)	14	26	32
Montaux test (mm)	Positive (15)	Positive (22)	Positive (18)
CBNAAT	Positive	Negative	Negative

Case 3

A 14-year-old female presented with complaints of persistent headache, easy fatigability and episodic dizziness for two months. There was a history of weight loss and night sweats during the preceding six weeks. The patient also reported painless swelling in the neck for three weeks. On examination, the patient had a heart rate of 86/min and respiratory rate of 18/min. Blood pressure was markedly elevated in all four limbs, with a maximum of 176/110 mmHg in the right upper limb. Peripheral pulses were asymmetrically reduced, with diminished femoral pulses bilaterally. Fundoscopic examination revealed grade 1 hypertensive retinopathy. Baseline investigations showed elevated ESR and CRP with normal total leukocyte count and mild thrombocytopenia (Table 1). Renal function tests were deranged, suggestive of renovascular involvement. Echocardiography revealed left ventricular hypertrophy with mild diastolic dysfunction. CT angiography demonstrated diffuse circumferential thickening of the abdominal aorta with critical narrowing at the level of the renal arteries, along with ostial stenosis of both renal arteries and focal involvement of the superior mesenteric artery (Figures 1 and 2). Based on clinical, laboratory, and imaging findings, Takayasu arteritis was diagnosed, and the patient was initiated on oral steroids.

However, due to persistent constitutional symptoms and poor response to initial immunosuppression, further evaluation for infectious etiology was pursued. The Mantoux test was positive, and FNAC of the cervical lymph node showed granulomatous inflammation. CBNAAT was negative for tuberculosis. The patient was started on standard ATT along with antihypertensive therapy. Over three months of follow-up, the patient demonstrated improvement in systemic symptoms and stabilization of blood pressure, although vascular lesions persisted on imaging, necessitating long-term follow-up.

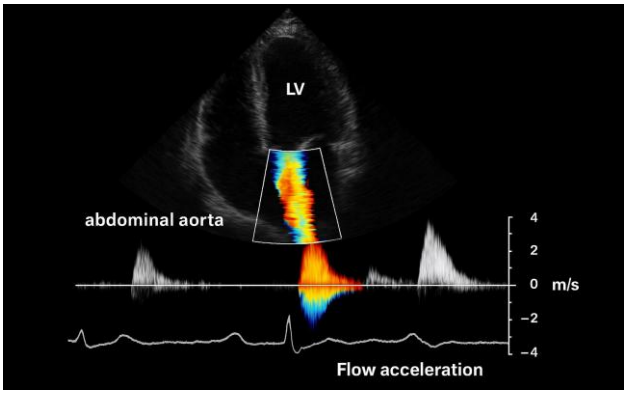

Figure 1: Echo showing flow acceleration in abdominal aorta.

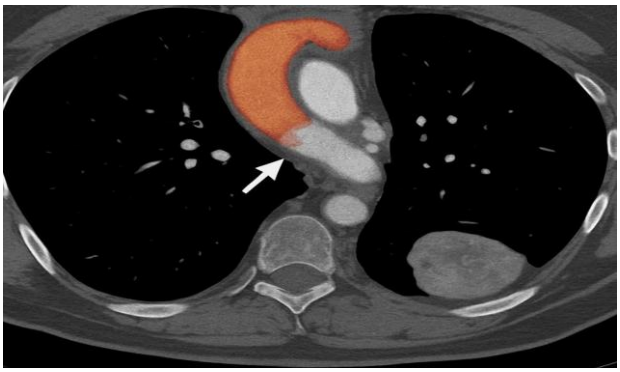

Figure 2: CT angiography showing circumferential thickening and luminal narrowing of abdominal aorta.

DISCUSSION

The proposed association between TB and TA is supported by several immunopathological similarities. Both diseases demonstrate a granulomatous inflammatory response with infiltration by activated macrophages and CD4+ T cells. Studies have shown increased expression of heat-shock protein 65 (HSP-65) of M. tuberculosis in patients with TA, which shares homology with human HSP-60, suggesting a mechanism of molecular mimicry leading to vascular inflammation. Additionally, mycobacterial DNA has been detected in the aortic tissue of some patients with TA, further reinforcing a possible causal role.[7]

Epidemiological studies have revealed a striking association between tubercular aortitis and TA. A 2023 meta-analysis reported that up to 31.27% of patients with TA also had latent or active tuberculosis, with regional variation as high as 63.6% in Africa and 43.3% in the Americas.[8] Clinical studies have shown that TA with coexisting TB tend to present with more severe vascular damage, including higher rates of aortic dilation and aneurysm formation, especially in the abdominal aorta.[9]

This overlap presents significant diagnostic and therapeutic challenges. For instance, differentiating tubercular aortitis from TA based solely on imaging may be impossible, and initiating immunosuppressive therapy in undiagnosed TB can lead to worsening infection. Conversely, misdiagnosing TA as TB may delay critical immunosuppressive treatment.

CONCLUSION

The coexistence of TA and tuberculosis in this case series highlights an important and increasingly recognised clinical association, particularly in regions where TB is endemic. Shared granulomatous pathology, immunological overlap, and the presence of mycobacterial components in vascular tissue suggest that *Mycobacterium tuberculosis* may act as a trigger for TA in genetically susceptible individuals. However, a definitive causal relationship has yet to be established, and the association may reflect both pathogenetic links and regional epidemiology.

This case series underscores the need for clinicians to maintain a high index of suspicion for TA in patients with tuberculosis who present with vascular symptoms, unexplained hypertension, or limb claudication. Early diagnosis and an integrated approach to treatment—combining appropriate anti-tubercular therapy with carefully monitored immunosuppression—are essential to optimizing patient outcomes.

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