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Case Report

The Silent Duo: Latent Tuberculosis Complicating Takayasu Arteritis in a Young Bangladeshi Woman

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Abstract:

Takayasu disease or pulseless disease is a rare chronic granulomatous inflammatory arteritis of unknown aetiology affecting large vessels, particularly the aorta and its main branches. It mainly affects females more than males with the ratio of 8:1 and in the second and third decade of life. Mechanism may be transmural fibrous thickening of the arterial walls. Takayasu arteritis is characterized by inflammation of the vessel wall, leading to occlusion of the vessel wall. It is represented with claudication, fever, and arthralgia. Clinical features are chest pain, vascular bruits, hypertension. There is indirect evidence signifying a potential link between tuberculosis (TB) and Takayasu's arteritis (TAK); however, the exact mechanism and relationship between TAK and Mycobacterium tuberculosis remain elucidated. This case intends to highlight the association between latent TB and TAK, as early detection can avoid devastating consequences.

Keywords: *Aorta, Claudication, Granulomatous Arteritis, Pulseless Disease, Tuberculosis.*

Background:

Takayasu disease is a chronic granulomatous inflammatory arteritis of unknown aetiology which can affect the aorta and its main branches, as well as the innominate, brachiocephalic, carotid, subclavian, and renal arteries[1]. It was initially described in 1908 by Dr. Mikito Takayasu, a professor of ophthalmology at Kanazawa University, Japan[2]. One in 200,000 people is affected by Takayasu's arteritis (TAK), predominantly affecting females under 40 years with a female to male ratio of 8:1[2,3].

TAK can be present in two phases, a systematic phase followed by an occlusive phase[2]. The first phase shows non-specific constitutional symptoms such as fever, myalgia, fatigue, anorexia, weight loss, tenderness in the affected arteries[1,2]. The acute phase reactant such as erythrocyte sedimentation and C reactive protein is usually raised in this phase[2,4]. The second phase occurs due to chronic inflammation and stenosis of the involved arteries, resulting in claudication of the limb, headache, dizziness, hypertension, chest pain, blood pressure discrepancies between two arms, and diminished or absent peripheral pulses[2,5].

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On the other hand, tuberculosis (TB) is a curable and treatable disease that is distributed worldwide[6]. It affects all age groups, adults being the most targeted population[6]. It is a transmissible bacterial infection caused by *Mycobacterium tuberculosis* chiefly affects lungs[7]. Nonetheless, other tissues and organs may also be involved[7]. Although one-fourth of the world's population is infected with tuberculosis, most of them have latent tuberculosis within their lifetime. The risk of reactivation of latent to active tuberculosis is most significant in people with immune-deficient conditions[8].

It is hypothesized that TAK can be related to genetics, endocrine abnormalities, and infections such as *Mycobacterium tuberculosis* (MTB)[9]. In fact, both latent and active TB infection have been observed in patients with TAK[10]. Here we report a case of a young girl with extensive TAK with concomitant latent TB.

Case report: Mrs X, a 26-year-old Muslim woman from Dhaka Bangladesh presented to us with the complaints of low-grade fever, palpitation and weight loss for 3 months. Fever was not associated with chills and rigors and there was no history of evening rise of temperature or night sweat. For the last 3 months, she had palpitations which persisted even at rest. It was not associated with any chest pain, shortness of breath, dizziness or syncope. On query, she gave history of bilateral upper limb pain for 3 months, especially in both forearm and hands, aggravated by doing work, like combing, dishwashing and relieved by taking rest. She also complained of anorexia, and unintentional weight loss of 10 kg in last 3 months. She had no cough, hemoptysis, joint pain, oral ulcer, color changes in fingers on cold exposure, skin rash, abdominal pain, headache, seizures, pregnancy loss and bleeding from any site. Her bowel and bladder habits were normal. She had no history of exposure to known TB patients. She was BCG vaccinated.

On examination, she was mildly anemic, underweight, had

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BMI 17.31 kg/m^2, temperature 100^o F and blood pressure difference in two arms (140/90 mmHg in right arm and 110/70 mm Hg in Left arm). All the peripheral pulses were intact but low volume. Cardiovascular system examination revealed dancing carotid but no tenderness. Apex beat was shifted laterally, 10 cm from the midline and forceful in nature. Heart sounds were normal in all cardiac areas. There was a high pitched, blowing early diastolic murmur in the left lower parasternal area, best heard with the patient sitting and bending forward and breath hold after expiration. Other systemic examination revealed no abnormalities.

On investigation, she had normocytic, normochromic anemia with high acute phase reactant (ESR 126 mm in the first hour and CRP 39 gm/L). Liver function tests, renal function tests and thyroid function tests failed to reveal any abnormalities. ASO titer, LDH, s. uric acid, urine routine examination, ultrasonography of whole abdomen was normal. Vasculitis screening with ANA, Anti CCP, P-ANCA, C- ANCA were negative. TPHA, blood culture was also negative. Chest X-ray PA view showing mediastinal widening (Figure 1). CT chest was normal. Color doppler echocardiography revealed dilated aortic root with moderate to severe aortic regurgitation with no evidence of vegetations or thrombus and no regional wall motion abnormalities with good left ventricular systolic function (LVEF 64%) (Figure 2).

Figure 1: Chest X-ray PA view showing mediastinal widening.

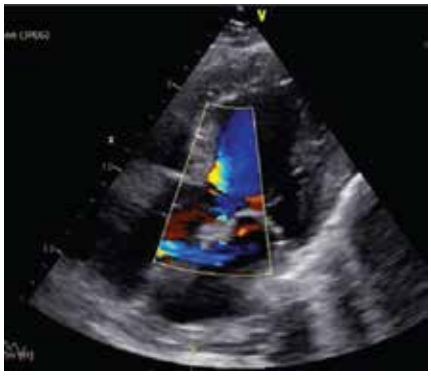

Figure 2: Color doppler echocardiography showing moderate to severe aortic regurgitation.

CT angiogram of both upper limb vessels showed Irregular narrowing and dilatation in both common carotid artery, mild aneurysmal dilatation of the brachiocephalic trunk and right proximal subclavian artery. (Figure 3). CT Aortogram revealed aortic arch aneurysm (diameter 53 mm) having no thrombus (Figure 4).

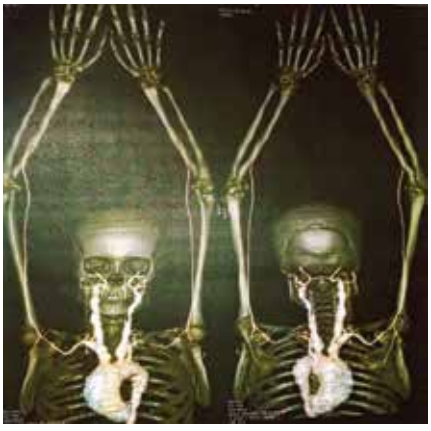

Figure 3: CT angiogram of both upper limb vessels showed Irregular narrowing and dilatation in both common carotid artery, mild aneurysmal dilatation of the brachiocephalic trunk and right proximal subclavian artery.

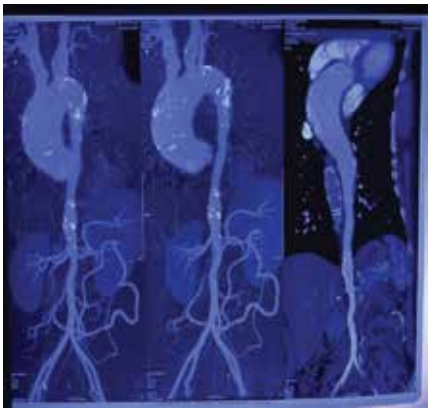

Figure 4: CT Aortogram revealed aortic arch aneurysm (diameter 53 mm) having no thrombus.

She was treated for latent tuberculosis with isoniazid and rifampicin according to her weight for 3 months. For TAK, she was given tablet prednisolone 1mg/kg for 2 months to be tapered over the next 6 months and tablet azathioprine 100 mg daily and tablet aspirin 75 mg daily. During outpatient follow up her condition was good. The patient was advised to consult with cardiothoracic surgeon for further management of aortic root dilatation

Discussion:

Takayasu arteritis, also referred to as pulseless disease, is a rare, chronic inflammatory vasculitis that primarily affects aorta, and its branches typically presents in the second and third decade of life in females of Asian descent[11]. Takayasu's arteritis was first described by Dr. Mikito Takayasu in 1908. 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 TAK and tuberculosis, the exact role of *Mycobacterium tuberculosis* in the pathogenesis of TA is still unknown. Most recent reports suggest that cross- reactivity between mycobacteria and a

human heat shock protein (HSP) might have a key role[12,13] or the triggering of superantigens by mycobacteria[14], the suggested role of which is thought to be via the stimulation of autoreactive T cells that induce vascular damage. On the other hand, the second hypothesis is based on the possibility that the arteritis results directly from a latent TB infection which might have a high prevalence of latent TB in TAK patients[9].

Our diagnosis was made on the basis of the criteria of the American College of Rheumatology/ EULAR for the diagnosis of TAK (Table 1), published in 2022[15]. This patient presented with 8, score with 98% specificity.

ABSOLUTE REQUIREMENTS	
Age ≤ 60 years at time of diagnosis	
Evidence of vasculitis on imaging[1]	
ADDITIONAL CLINICAL CRITERIA	
Female sex	+1
Angina or ischemic cardiac pain	+2
Arm or leg claudication	+2
Vascular bruit[2]	+2
Reduced pulse in upper extremity[3]	+2
Carotid artery abnormality[4]	+2
Systolic blood pressure difference in arms ≥ 20 mm Hg	+1
ADDITIONAL IMAGING CRITERIA	
Number of affected arterial territories (select one)[5]	
One arterial territory	+1
Two arterial territories	+2
Three or more arterial territories	+3
Symmetric involvement of paired arteries[6]	+1
Abdominal aorta involvement with renal or mesenteric involvement[7]	+3

Sum the scores for 10 items, if present. A score of ≥ 5 points is needed for the classification of TAKAYASU ARTERITIS.

Table 1: American College of Rheumatology/ EULAR criteria for the diagnosis of Takayasu Arteritis.

Common manifestations at disease onset included loss or asymmetry of pulses (57%), blood pressure discrepancy of limbs (53%), and bruits (53%). Prior to disease diagnosis, about 11% of patients may be asymptomatic. Previous angiographic studies showed aortic abnormalities in 79% of patients and frequent involvement of the subclavian (65%) and carotid (43%) arteries.[16] Ninety-three percent of patients attained disease remission of any duration, but 28% sustained remission of at least 6 months' duration after prednisone was tapped to <10 mg daily.[17] Both angioplasty and vascular surgery were initially successful, but recurrent stenosis occurred in 78% of angioplasty and 36% of bypass or reconstruction procedures.[18]

Over many years, studies showed contribution of *Mycobacterium tuberculosis* in the pathogenesis of TAK[19]. One study found 65 cases of latent tuberculosis in patients diagnosed with TAK[20,21]. We discovered that the majority of these observational studies detected latent tuberculosis in a patient diagnosed with TAK. These studies used various methodologies such as the Mantoux tuberculin skin test, Interferon-gamma release assay (IGRA), and QuantiFERON-TB Gold tests to detect latent tuberculosis[20,21,22]. A recent case report by Mangouka et al. in 2020 reported a case of TAK with latent tuberculosis, which was the first-ever case to be reported in Gabon[21], country in Central Africa. Furthermore, Agostinis et al. and Liebscher et al. published a case report of TAK with latent tuberculosis in 2019 and 2017, respectively[23,24]. A 75-year-old male diagnosed with TAK and tuberculosis had bilaterally thickened carotid arteries on ultrasound examination, among other symptoms, which showed significant improvement after two weeks of only isoniazid therapy[23]. A case report by Liebscher et al. presented TAK and infection with *Mycobacterium tuberculosis*, and hepatitis B[20]. Therapy for these infections and methotrexate led to improvement in TAK symptoms[24]. The positive response to TAK symptoms after the treatment suggests the possible role of *Mycobacterium tuberculosis* in TAK development [24]. In 2017, Zhang et al. reported an unusual case of pulmonary tuberculosis diagnosed six months after TAK was diagnosed[25]. Clemente et al. conducted a retrospective observational study to describe TAK's clinical and angiographic features in 71 Brazilian children and adolescents [26]. Their research revealed a higher frequency of tuberculin skin test positivity in their patients than healthy Brazilian children, as reported by the Brazilian Institute of Geography and Statistics[26.] This finding hints at the prevalence of latent tuberculosis in the patient with TAK. Although the exact etiology could not be identified, Clemente et al. highlighted that the immune response in TAK could be a result of cross-reaction between homologous protein present in the vascular wall of the host and the mycobacterial heat shock 65-kD protein[23]. Similarly, a cross-sectional study conducted by Nooshin et al. found the level of purified-protein derivative >10mm in six out of 15 study subjects, stressing the association of latent TB in a patient with TAK [26]. Furthermore, in 2010, Al-Aghbari et al. demonstrated a particular case of TAK who had a strongly positive Mantoux test for TB [27]. This was the first-ever case of TAK associated with TB in Yemen [28]. Lastly, the findings of Muranjan et al. highlighted the correlation between infection and TAK pathogenesis, who detected positive tuberculin skin test or Bacille Calmette-Guerin (BCG) in six (35.2%) out of 17 patients with TAK[29.] Based on data, it is clear that the co-occurrence of TAK and latent tuberculosis can be seen. Nevertheless, careful interpretation is required as positive purified protein derivative (PPD), and IGRA tests could be influenced by the immunosuppressive agents and corticosteroids regularly used in TAK treatment. Additionally, the false-positive reaction to PPD could be due to prior vaccination with BCG, which was not distinctly illustrated in many studies.

The effect of anti-tubercular drugs on TAK symptoms remains debatable among authors[30]. With an antitubercular regimen, some authors portrayed the case of a thorough reduction in TAK symptoms along with the complete return of the affected pulses[30]. Though, it is essential to note that most studies have combined corticosteroid and anti-tubercular drugs to treat co-occurrence of TA and active TB.

Our case was also a young Bangladeshi woman who met the criteria for Takayasu arteritis according to the American College of Rheumatology and had latent tuberculosis

In summary, we analyzed that many studies in our review showed a collection of indirect findings signifying a potential link between tuberculosis and TAK; most have latent tuberculosis in patients with TAK, suggesting that infection with *Mycobacterium tuberculosis* could trigger TAK development. Although the etiopathogenesis of TAK remains

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unclear, tuberculosis was hypothesized to be one of the prompting influences[29]. The direct role of *Mycobacterium tuberculosis* is not entirely suggested as current literature hypothesizes autoimmunity involving both cellular and humoral factors as a chief contributor to TAK 's pathogenesis[29,30]. Still, various other factors such as genetic predisposition, post-infective, and ethnic susceptibility have also been considered[29,30].

Conclusion:

TAK in young remains a challenging diagnosis because of nonspecific features. We insist that TAK must be suspected when regarding systemic signs with hypertension and/or blood pressure differences between extremities. A causal relationship between TB and TAK and evidence of prevention of TAK progression and complications under anti-TB therapy need further investigation. Because of long diagnostic delay, the physician must keep in mind that this pathology can lead to death without immunosuppressive therapy suggesting that this vasculitis is becoming ubiquitous.

Conflict of interest: None declared

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