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Case Report

Inflammatory Cascade in Aortic Wall: Latent Tuberculosis Complicating Takayasu Arteritis in A Young Bangladeshi Lady- the Silent Duo

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Abstract:

Takayasu disease or pulseless disease is a rare chronic granulomatous panarteritis 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 (TB) remain elucidated. This case intends to highlight the association between latent TB and TAK, as early detection can avoid devastating consequences.

Keywords: pulseless disease; granulomatous panarteritis; aorta; claudication; tuberculosis

Introduction

Takayasu disease, also called pulseless disease or aortic arch syndrome, 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 TAK, predominantly affecting females under 40 years with a female to male ratio of 9:1 [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]. Tuberculosis (TB) is a curable and treatable disease that is distributed worldwide [6]. TB affects all age groups, adults being the most targeted population [6]. It is a transmissible bacterial infection caused by *Mycobacterium tuberculosis*, transmitted via the respiratory route that chiefly affects the 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 only 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]. Etiopathogenesis of TAK remains hypothetical, and is hypothesized to be related to genetics, endocrine abnormalities, and infections such as *Mycobacterium tuberculosis* (TB) [9]. In fact, both latent and active TB infection have been observed in patients with TAK [10]. This report describes the case of a young girl with extensive TAK concomitant to latent TB.

Case report

Mrs X, a 26-year-old Muslim lady from Dhaka Bangladesh presented to us with the complaints of fever, palpitation and weight loss for last 3 months. According to the statement of the patient, she was reasonably well 3 months back. Then she developed fever which was low grade, intermittent in nature, and the highest recorded temperature was 100°F. 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 developed palpitations which was gradual in onset, aggravated after exertion and 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 the forearm and hand, which was gradual in onset, aching in nature, non-radiating, present most of the time, and aggravated by doing any kind of work, like combing, dishwashing and relieved by rest. She also

complained about anorexia, and she mentioned that she lost 10 kg of weight in 3 months which was unintentional. She denied any cough, joint pain, oral ulcer, discoloration of fingers on cold exposure, skin rash, abdominal pain, headache, convulsion, miscarriage and bleeding from any site. Her bowel and bladder habits were normal. With these complaints she sought multiple medical consultations and extensive primary investigations. On the basis of a positive MT test (22mm) she was started on anti-TB from outside 9 days back without minimal improvement. She had no significant past history of illness. She was non-smoker and she was nonalcoholic. She had no known exposure to TB patients. She practices safe sexual intercourse and was BCG vaccinated. On examination, she was mildly anemic, BMI was 17.31 kg/m^2, temperature 100^o F, clubbing- absent. Blood pressure 140/90 mmHg in right arm and 110/70 mm Hg in Left arm. All the peripheral pulses were present but of low volume. Visible pulsation is seen in the right carotid area. There was no ulcer, no skin rash but there was induration over the volar aspect of left forearm. Thyroid gland was not enlarged, and no lymph nodes were palpable. On cardiovascular system examination, dancing carotid pulse was seen in right carotid area (Corrigan sign). Apex beat was present in the left 5th intercostal space, shifted laterally, 10 cm from the midline and forceful in nature. There was no palpable P2 or A2. No tenderness was found in the carotid area. First and second heart sounds are normal in all cardiac areas. There is 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, complete blood count revealed normocytic, normochromic anemia with hemoglobin 9.2 gm%. White blood cells and platelet counts were within normal limit. ESR and CRP were raised to 126 mm in the first hour and 39 gm/L respectively. 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 were normal. Vasculitis screening with ANA, Anti CCP, P-ANCA, C- ANCA were nonconclusive. TPHA, blood culture, 2 samples drawn from 2 different sites were negative. Chest X-ray PA view showing mediastinal widening (Figure 1). Ct chest failed to reveal any mediastinal lymphadenopathy or lung parenchymal lesion. Color doppler echocardiography revealed dilated aortic root with moderate to severe aortic regurgitation with no evidence of vegetations or thrombus and no regional motion wall abnormalities with good left ventricular systolic function (LVEF 64%) (Figure 2)

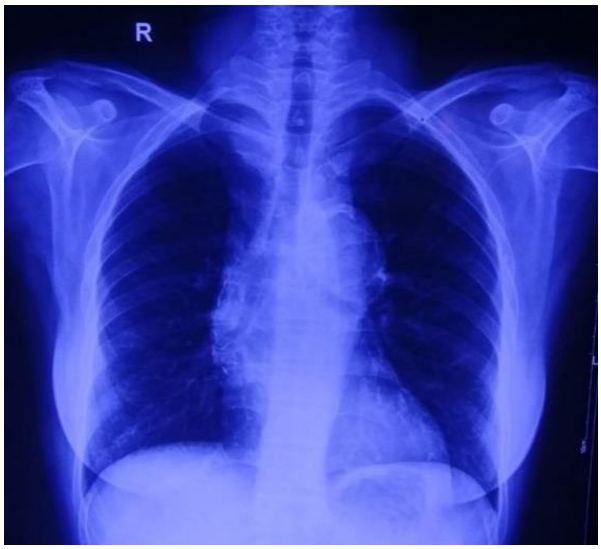


Figure 1: Chest X-ray PA view showing mediastinal widening.

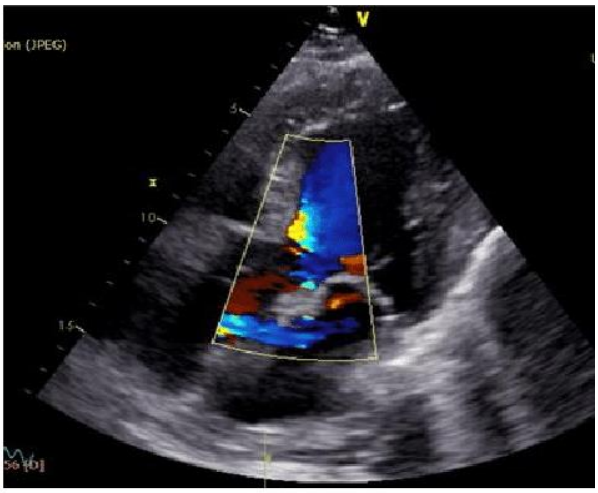


Figure 2: Coloe doppler echocardiography showing moderate to severe aortic regurgitation.

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).

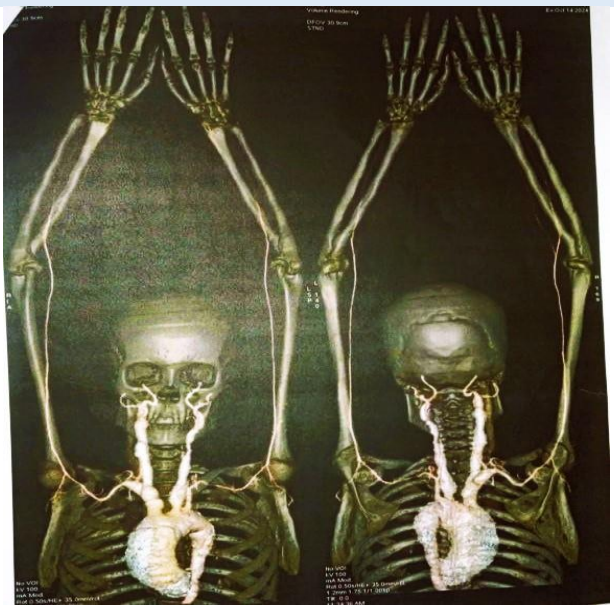


Figure 3: CT angiogram of both upper limb vessels showed Irregular narrowing and dilatation in both common carotid artery, mild aneurysmal dilatation of the brachiocephalic trunk and right proximal subclavian artery.

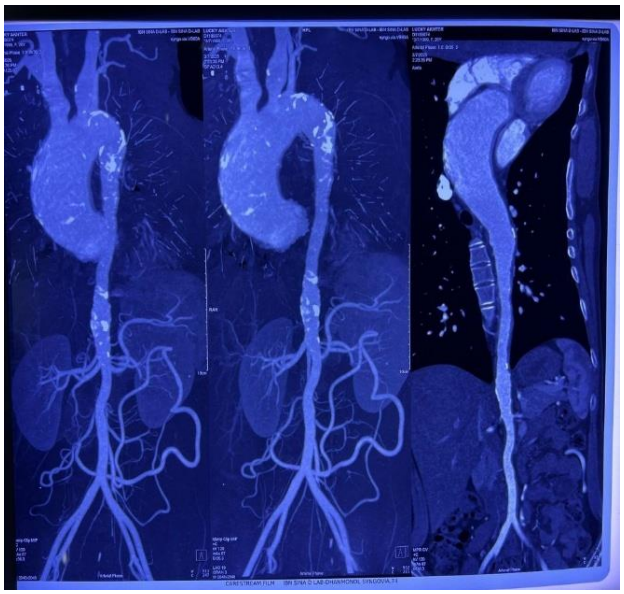
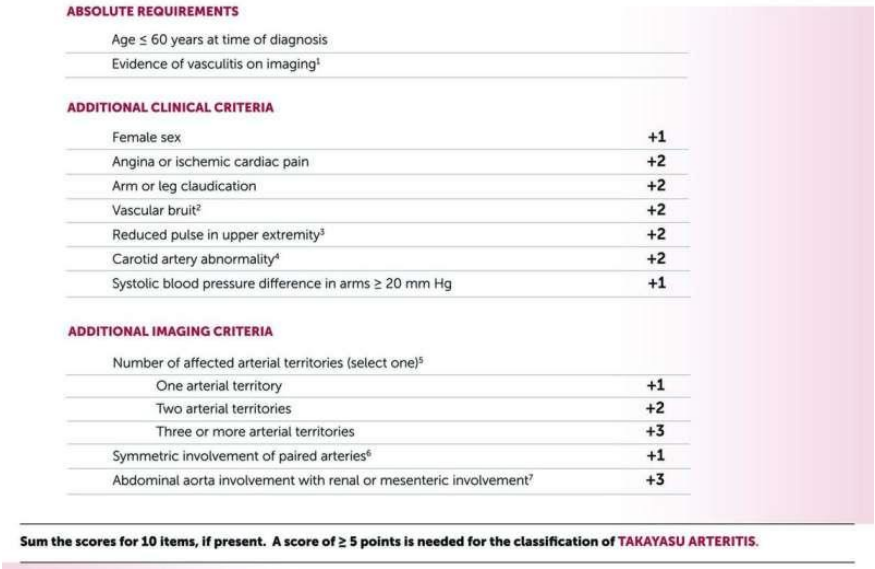


Figure 4: CT Aortogram revealed aortic arch aneurysm (diameter 53 mm) having no thrombus.

She was started treatment for latent tuberculosis with isoniazid and rifampicin according to her weight, to be continued 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. Along with the medical treatment, the patient was advised to consult with cardiothoracic surgeon for the management of aortic root dilatation. She is under regular outpatient follow up and is doing well.

Discussion

Takayasu arteritis (TAK), also referred to as pulseless disease, is a rare, chronic inflammatory vasculitis that primarily affects large- and medium-sized vessels and 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 points, 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%), limb blood pressure discrepancy (53%), and bruits (53%). 11% of patients were asymptomatic prior to disease diagnosis. Initial 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/reconstruction procedures.[18].

Latent Tuberculosis in TAK Patients

Over many years, many studies show shreds of evidence implicating the contribution of *Mycobacterium tuberculosis* in the pathogenesis of TAK [19]. Our studies 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 tests to detect latent tuberculosis in the study subjects [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]. 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 reduction 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 a 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 [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 this data from these observational studies, the co-occurrence between 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.

Role of Anti-Tubercular Drug on TAK Prognosis

In the study of Khemiri et al., anti-tubercular drugs did not affect TA vasculitis and did not aid in the prevention of new relapses [30]. On the contrary, a case reported by Agostinis et al. showed complete remission of TAK symptoms after treatment with only isoniazid for two weeks [23]. Additionally, the case report by Moura et al. showed substantial improvement in symptoms after therapy with anti-tubercular drugs and steroids [31]. Correspondingly, Liebscher et al. reported a case of TAK with TB, which revealed positive results after treatment with anti-tubercular medications [32]. 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. In summary, we analyzed that many studies in our review showed a collection of indirect findings signifying a potential link between tuberculosis and TAK; however, only one failed to detect the aforementioned link. In these observational studies, most have latent tuberculosis in patients with TAK, suggesting that infection with *Mycobacterium tuberculosis* could trigger TAK development. Although the etiopathogenesis of TAK remains unclear, tuberculosis was hypothesized to be one of the prompting influences [29]. Karadag et al. suggested that the previously reported studies that addressed the connection between the prior exposure to *Mycobacterium tuberculosis* and TAK patients may be considered incidental in countries where TB is endemic [33]. 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

Our final diagnosis was Takayasu arteritis in a young Bangladeshi woman who met the criteria for Takayasu arteritis according to the American College of Rheumatology and criteria for latent tuberculosis. 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.

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