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Case Report

Twenty-Year-Old Female with Takayasu Arteritis and Pulmonary Tuberculosis: A Case Report

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A B S T R A C T

Takayasu arteritis is a rare, large-vessel vasculitis of unknown aetiology, typically affecting young women. It occurs most often in the Asian population. The overall rate is 2.6 per million inhabitants. A relationship between Takayasu arteritis and Mycobacterium tuberculosis has been suggested for a long time, but only a few case report have been done centering on this association. We report a case of Takayasu arteritis in a 20-year-old female revealed by thickness of common carotid artery and then pulmonary tuberculosis appeared after treatment by corticosteroid.

Keywords: Takayasu arteritis; Tuberculosis; Vasculitis

1. Introduction

Takayasu arteritis is a chronic granulomatous vasculitis of large vessels that affects the aorta and its main branches. This granulomatous inflammation may lead to stenosis, occlusion, dilatation, or aneurysm of the involved arteries[1]. Although Takayasu arteritis is frequently seen in young women, it can also affect young infants and adolescents[2]. Takayasu arteritis in childhood is rare, with only 2.6/1 000 000 of childhood-onset Takayasu arteritis reported in literature until now[3]. Features are variable depending on the stage of disease. In fact, the first stage of initial inflammatory process is often unrecognized and characterized by systemic signs[4]. In the second stage, multiple arterial occlusions. and stenosis can occur, and can be revealed by signs of cerebral, visceral, or extremity ischemia[4].

Takayasu arteritis also known as "pulseless disease," is an uncommon, chronic granulomatous vasculitis that mainly affects the large arteries such as the aorta and its primary branches[5]. It was initially described in 1908 by Dr. Mikito Takayasu, a professor of ophthalmology at Kanazawa University, Japan[6]. One in 200,000 person is affected by Takayasu arteritis, it predominantly affects females under 40 years with a female to male ratio of 9:1[5,6]. Takayasu arteritis occurs in every part of the world; however, it is more common in Southeast Asia, India, Japan, China, Korea, Mexico, and Latin America[5,4].

Takayasu arteritis can be present in two phases, a systematic phase followed by an occlusive phase[6]. The first phase shows non-specific constitutional symptoms such as fever, myalgia, fatigue, anorexia, weight loss, tenderness in the affected arteries[6,4]. The acute phase reactant such as erythrocyte sedimentation and C reactive protein is usually raised in this phase[6,7]. 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[5,8]. It is characterized by the infiltration of inflammatory cells in tunica media, hyperplasia of the intima, and thickening of adventitia, histologically[9].

Tuberculosis is a curable and treatable disease that is distributed worldwide[10]. According to World Health Organization (WHO), in 2019, the most significant number of Tuberculosis cases was seen in the WHO Southeast Asian region[11]. Tuberculosis affects all age groups, adults being the most commonly affected

population[10]. It is a transmissible bacterial infection caused by Mycobacterium tuberculosis, it is transmitted via the respiratory route and itI chiefly affects the lungs[12]. Nonetheless, other tissues and organs may also be involved[11]. Although one-fourth of the world's population is infected with tuberculosis, most of them only have latent tuberculosis within their lifetime; the rest of the affected individuals effectively contain their infection[11]. The risk of reactivation of latent to active tuberculosis is most significant in people with immune-deficient conditions[12].

The precise etiology of Takayasu arteritis continues to be unknown[5]. However, autoimmunity is mainly suggested as a cause of Takayasu arteritis[13]. The other causes that might contribute to Takayasu arteritis's etiopathogenesis are genetic and infectious (bacterial, viral) causes[6,13]. Of the bacterial causes, the role of Mycobacterium tuberculosis has been implied[14]. Takayasu arteritis is one of the first vasculitides to be related to a particular infective organism[14]. Initially, this likely co-relation was mentioned due to the morphological resemblance of Langhan's giant-cell granulomas with tuberculous lesions[15]. Another likely finding signifying this correlation is the evidence of tubercular lymph nodes in the arterial lesions, increased agalactosyl IgG level, augmented responses to purified protein derivative of Mycobacterium tuberculosis[13,16,17]. In addition, recent studies suggest the role of mycobacterial heat shock protein (HSP) in linking autoimmune disease and Mycobacterium tuberculosis[18]. The molecular cross-reactivity between host HSPs and mycobacterial HSPs could be the probable trigger for the autoimmune process[18]. Also, in Takayasu arteritis patients' aortic tissues, Soto et al. detected an increased frequency of IS6110 and HupB genes[19]. Takayasu arteritis is commonly seen in East Asia or Southeast Asia, where the prevalence of Tuberculosis is high[20].

In this case report aimed to examine and consolidate the relevant information on this connection. And, it intends to highlight the association between Mycobacterium tuberculosis and Takayasu's arteritis and the possible cause for this link from the studies done previously.

2. Case Presentation

A 20-year-old woman presented with abdominal and neck pain. Her medical history was unremarkable. Physical examination showed pulseless left brachial and radial arteries, decreased right brachial and radial artery pulses. There was a blood pressure difference between arms (Right arm: 101/59 mmHg; Left arm 90/51 mmHg). The presence of bruits over left subclavian and left common carotid artery. High sensivity CRP was elevated 206.7 mg/L (≤ 3 mg/L), erythrocyte sedimentation rate was elevated 124 mm (first hour) and 136 mm (second hour). ANA test was negative, anti dsDNA was negative, ANCA screen was negative, TSH, free T4 was normal, mild hypochromic microcytic anemia with Hb: 10 g/dL, high white blood cell with 14.63 x109/L, platelet 593 x 109/L. Electrocardiography showed sinus tachycardia 120 bpm (**Figure 1**). Chest X-ray showed normal findings (**Figure 2**). Carotid ultrasound revealed concentric thickening of the left common carotid artery with 70% stenosis. Chest and abdomen aortic CT scan with contrast showed stenosis of the right brachiocephalic artery, left common carotid artery, 50% stenosis of the left subclavian artery and 60% stenosis of the right renal artery (**Figure 3**).

After physical and systemic examination, we suspect That this patient has Takayasu Arteritis because of having 4 over 6 criteria: onset age ≤ 40 years; decreased brachial artery pulse; > 10mmHg difference in systolic blood pressure between arms; a bruit over subclavian or aorta arteries according to The American College of Rheumatology 1990 Criteria[21] for the Classification of Takayasu Arteritis with a sensitivity of 90.5% and a specificity of 97.8%. The chest and abdominal CT angiography showed Moderate renal artery stenosis (60%). 50% stenosis of the left common carotid artery. Patient was prescribed prednisone 1mg/kg daily. Unfortunately, after taking corticosteroids, the patient coughed a lot and had a fever in the afternoon. The patient had a chest x-ray with suspicion of pulmonary tuberculosis. The patient was tested for Xpert gene; the result was positive. The patient was treated with a 9-month anti-Tuberculosis regimen rifampicin, isoniazid, pyrazinamide, and ethambutol with prednisone 5mg/day. The patient continued taking prednisone for 2 years. Follow-up was favorable clinically, biologically, and radiologically.

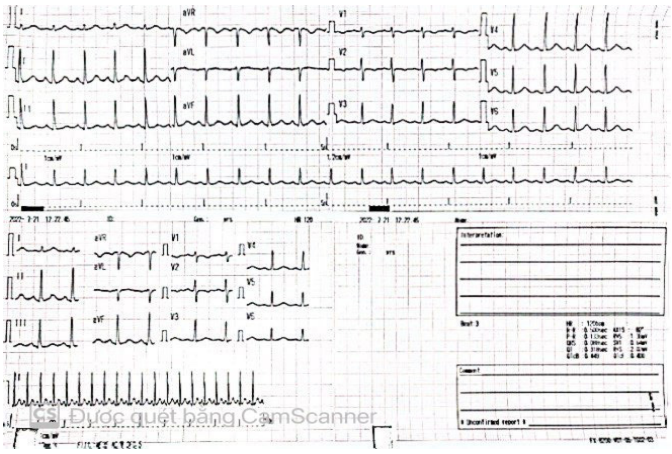

Figure 1: Electrocardiography showed sinus tachycardia 120 bpm.

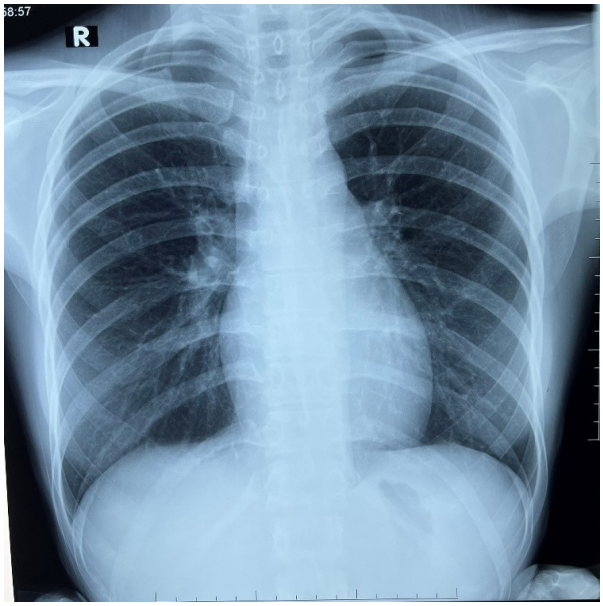

Figure 2: Chest X-ray appears unremarkable.

3. Discussion

In 2017, Zhang et al. reported an unusual case of pulmonary tuberculosis diagnosed six months after Takayasu arteritis was diagnosed[22]. Clemente et al. conducted a retrospective observational study to describe Takayasu arteritis clinical and angiographic features in 71 Brazilian children and adolescents[23]. 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[23]. This finding hints at the prevalence of latent tuberculosis in a

patient with Takayasu arteritis. Although the exact etiology could not be identified, Clemente et al. highlighted that the immune response in Takayasu arteritis 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].

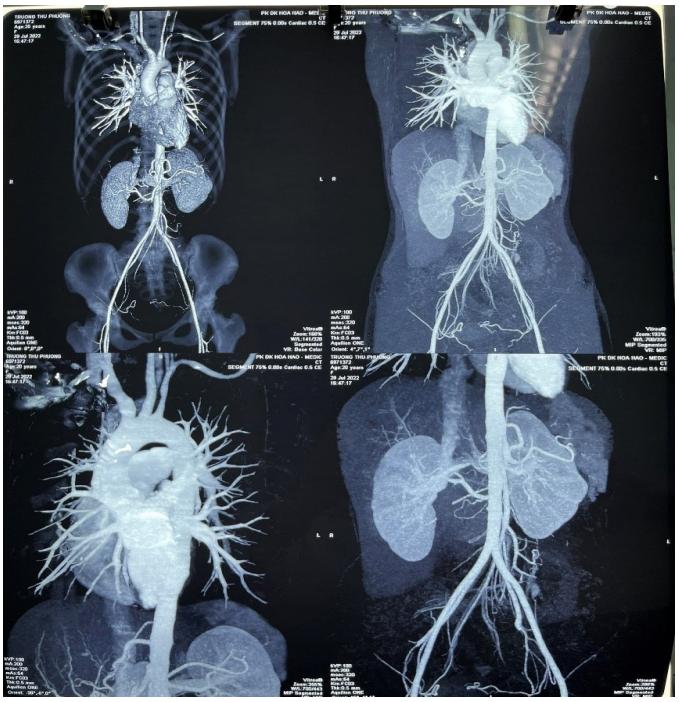

Figure 3: Chest and abdomen aortic CT scan with contrast showed stenosis of the right brachiocephalic artery, left common carotid artery, 50% stenosis of the left subclavian artery and 60% stenosis of the right renal artery.

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 Tuberculosis in a patient with Takayasu arteritis[25]. Furthermore, in 2010, Al-Aghbari et al. demonstrated a particular case of Takayasu arteritis who had a strongly positive Mantoux test for TB[18]. This was the first-ever case of Takayasu arteritis associated with Tuberculosis in Yemen[19]. Lastly, the findings of Muranjan et al. highlighted the correlation between infection with Mycobacteria tuberculosis and Takayasu arteritis pathogenesis, who detected positive tuberculin skin test or Bacille Calmette-Guerin (BCG) in six (35.2%) out of 17 patients with Takayasu arteritis[25-27].

Although there is no evidence that anti-Tuberculosis therapy prevents Takayasu arteritis progression or its complications, the combination of corticosteroids and anti- Tuberculosis drugs was efficient to control the disease activity observed in our patient.

4. Conclusion

We conclude that Takayasu arteritis must be suspected when there were blood pressure differences between extremities. A causal relationship between tuberculosis and Takayasu arteritis and evidence of prevention of Takayasu arteritis progression and complications under anti-tuberculosis therapy need further investigation. In Takayasu arteritis, a close monitoring of the clinical disease activity and damage associated with inflammatory markers and imaging are necessary to better identify the onset of disease and adapt therapy to prevent morbidity and mortality.

5. Conflict of Interest

None declared.

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