

## From low back pain to Takayasu arteritis: A rare case report

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### ABSTRACT

Takayasu arteritis (TA) is an uncommon chronic inflammation of blood vessels. It can occur with a range of clinical manifestations. However, low back pain is uncommonly defined as the initial symptom of TA. In this article, we report a 53-year-old woman who presented with low back pain and was diagnosed with TA after a detailed clinical examination and further evaluations. A thorough history and detailed physical examination can help to recognize the underlying cause of low back pain. Takayasu arteritis should be kept in mind as an alternative diagnosis while evaluating low back pain with high inflammation parameters, particularly in women.

**Keywords:** Low back pain, Takayasu arteritis, vasculitis.

Takayasu arteritis (TA) is an infrequently chronic granulomatous large vessel vasculitis with a tendency to involve the aorta and large vessels branching from the aorta.<sup>[1]</sup> It most commonly affects women between the ages of 20 and 30, but there are also reports of TA occurring in patients over the age of 50.<sup>[2]</sup> While the disease has a global distribution, it is widely thought to be much higher prevalence in Asian populations. A recent study in Türkiye has reported the incidence estimates similar to those in Europe (between 0.4 and 1.5 per million).<sup>[3]</sup> Arterial thrombosis, stenosis, dilatation or aneurysm may occur as a result of vascular inflammation and may cause different clinical presentations according to the region of vascular involvement.<sup>[4]</sup> Recognition of TA can be extremely difficult for the clinician, as the early stages of the illness, which follow a mild and stealthy clinical presentation until the onset of vascular ischemic manifestations, are devoid of specific laboratory abnormalities and non-systemic inflammatory manifestations such as malaise, weight loss and fever.<sup>[5]</sup> Musculoskeletal symptoms including myalgia and arthralgia are also common. Although low back pain is a major public health issue worldwide,<sup>[6]</sup> it is rarely described as a presenting feature of TA.

In this article, we describe a case of an adult female case who presented with lower limb claudication and low back pain, and was subsequently diagnosed with TA.

### CASE REPORT

A 53-year-old female patient presented with low back pain that started insidiously for nine months. The pain worsened with standing and walking and improved with sitting and resting. There was no night pain, no morning stiffness, no urine and stool incontinence, and no history of trauma or previous infection. On physical examination, pulse rate was 100/min and blood pressure was 120/80 mmHg in the right upper extremity and 100/60 mmHg in the left upper extremity. Asymmetrical radial pulses and bilateral carotid murmur were noted. Mild tenderness over the lumbar spine and the paravertebral region was found. The straight leg raise (SLR) test result was negative. There was no sensory or motor deficit. Deep tendon reflexes were normoactive. Other system examinations of the patient were normal. She had also hypertension, coronary artery disease, type 2 diabetes mellitus, obstructive sleep apnea syndrome (OSAS), and asthma.

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Her laboratory tests showed normocytic normochromic anemia (hemoglobin of 10.5 g/dL and hematocrit of 33.2%). The total leukocyte count (TLC) was 8,400 cells/ $\mu$ L (79% neutrophils and 21% lymphocytes). Acute phase reactants were in high levels (erythrocyte sedimentation rate 78 mm/h and C-reactive protein 21 mg/dL). Albumin and total serum protein levels were 3.5 and 6.3 g/dL, respectively. Coagulation profile, kidney and liver function tests were within limits of normal. Tests for the autoimmune biomarkers such as anti-nuclear antibody, anti-neutrophilic cytoplasmic antibody, rheumatoid factor and anti-cyclic citrullinated peptide were all negative. Upon imaging, chest radiography and contrast-enhanced magnetic resonance imaging (MRI) of the lumbar spine were non-specific. Carotid-vertebral Doppler ultrasonography (USG) showed uniform and homogeneous circumferential wall thickening and 50 to 70% luminal stenosis in bilateral common carotid arteries, considered secondary to vasculitis. These findings were suggestive of vasculitis, and supra-aortic magnetic resonance angiography (MRA) was requested. The MRA examination showed blurring and irregularity of the walls in arcus aorta, the narrowing of left

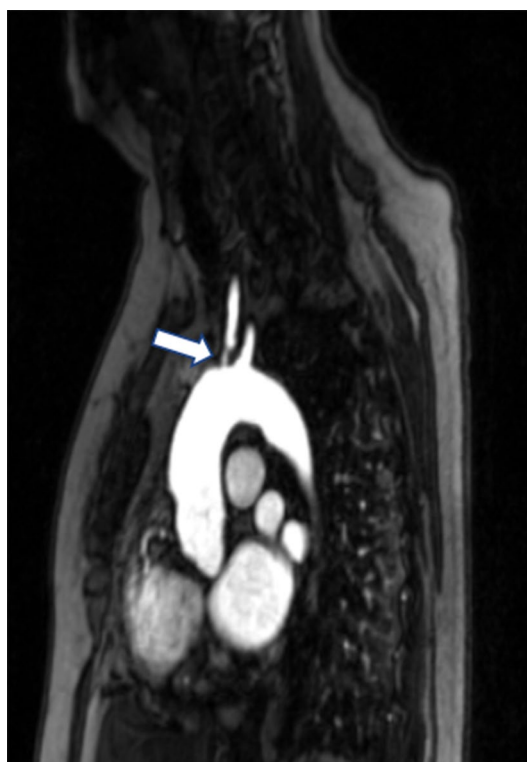
common carotid artery (Figure 1), and widespread contour irregularities in the proximal portion of the left internal carotid artery (Figure 2).

Based on these laboratory and imaging results, the patient was diagnosed with TA. Azathioprine was added to 30 mg daily oral corticosteroid therapy to maintain remission. With this treatment, the patient's low back pain disappeared within a few months and a decrease in inflammatory parameters was observed.

## DISCUSSION

Takayasu arteritis is a chronic vasculitis mainly involving the aorta and its main branches.<sup>[4]</sup> The for TA are the American College of Rheumatology (ACR) criteria.<sup>[7]</sup> The presence of three of the six ACR criteria is required to differentiate TA from another systemic vasculitis. Although the ACR defines age below 40 years as the criteria for TA, it has been shown that 13.0 to 17.5% of TA patients have onset of the disease after the age of 40 years, as in our patient.<sup>[2]</sup>

Vascular inflammation may cause different clinical manifestations based on the site of vascular



**Figure 1.** Blurring and irregularity of the walls in arcus aorta, the narrowing of left common carotid artery in magnetic resonance angiography (arrow).



**Figure 2.** Widespread contour irregularities in the proximal part of the left internal carotid artery (arrow).

involvement. The most frequent vascular presentations include decreased or absent pulse, claudication, carotid bruit, hypertension, stroke, transient ischemic attack, sudden blindness and asymmetric arm blood pressure.<sup>[1]</sup> In our case, there was low extremity claudication accompanying low back pain. In the peripheral artery examination which was carried out for the differential diagnosis of claudication (neurological or vascular), a decrease in radial and brachial artery pulse on one side, 20 mmHg systolic blood pressure difference and carotid bruit were detected. The symptoms of decrease or disappearance in pulses, claudication, systolic blood pressure difference between the arms, and the vascular murmur, which are included in the ACR criteria, were present in our case. Low back pain is seldom identified as a presenting symptom of TA in the medical literature. Melamed et al.,<sup>[8]</sup> Shikino et al.<sup>[9]</sup> and Azhar et al.<sup>[10]</sup> reported cases presenting with symptoms of back pain that were later diagnosed as TA cases. Takayasu arteritis was reported as a case by Fanning and Hickey<sup>[11]</sup> in whom the patient was admitted with persistent low back pain, lower extremity claudication, weight loss and episodic fever. Slobodin et al.<sup>[12]</sup> reported a similar case of TA who was presented with nonspecific pain in the thoracolumbar spine and whose diagnosis and treatment were delayed due to the lack of a comprehensive physical examination and differential diagnosis.

Low back pain in TA may appear as an underestimated and underreported symptom. It is thought that this chronic pain pattern may occur due to the affectation of the aorta and its branches, with both mechanical and chemical nociceptors playing a role. As TA leads to damage to the aorta and its branches, although less recognized, back pain may be the first complaint.<sup>[10]</sup> It has been suggested that back pain may develop following inflammation of the adventitia because of destruction of the nerves at the adventitia-media junction.<sup>[13]</sup> Chemical nociceptors and mechanoreceptors and may play a part in pain associated with a wall-thickened, inflamed aorta due to reduced compliance and cytokine release.<sup>[14]</sup> In addition, mechanosensory transduction, the consequence of chemicals released from endothelial cells under mechanical stress, may serve as an important mechanism of nociceptive activation.<sup>[15]</sup>

In conclusion, TA can lead to various clinical signs and symptoms depending on the vessels it affects. Low back pain may also be one of these symptoms, but it is not well-defined as a clinical manifestation of the disease. The most common problem in the diagnosis of

TA is not considering TA in the differential diagnosis. Therefore, we believe that TA should be kept in mind in the differential diagnosis in patients presenting with complaints of low back pain and lower extremity claudication and high inflammatory parameters, particularly in women.

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