

CASE STUDIES

Presentation of Takayasu's arteritis in a patient with intervertebral disc prolapse

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Abstract

Takayasu's arteritis is an idiopathic, chronic inflammatory disease affecting large and medium sized arteries, especially aorta, its major branches, and the pulmonary and coronary arteries. Though the disease incidence is very rare, it may lead to prolonged morbidity and mortality. The clinical presentations are more often due to ischemic features. In certain cases, the presentation may be atypical to consider the diagnosis of Takayasu. We present here the case of a young woman diagnosed with Takayasu's arteritis who presented with features of intervertebral disc prolapse.

Introduction

Takayasu's arteritis (TA) is an idiopathic chronic granulomatous inflammatory disease of large and medium sized arteries primarily affecting aorta and its branches.¹ The disease is relatively uncommon and majority of the cases are reported from Japan, India, China, Sri Lanka, and Singapore.¹

Case report

A 33-year-old female, working as a mason, presented to the hospital with a history of low backache since past 1 year and pain in both the lower limbs since 3 months. Low backache of moderate intensity was insidious in onset, and it progressively radiated to buttocks during each episode. It got worsened on bending forward, coughing, and sneezing. Lower limbs pain, which was felt in the calves, worsened on walking for about half a kilometer and relieved on rest. However, no history of motor weakness and paresthesia of lower limbs was reported. The patient also experienced loss of appetite and loss of weight of approximately 10 kg since last 6 months. There was no history of cough with expectoration, evening rise of temperature, trauma to lower back, hypertension, diabetes mellitus, ischemic heart disease and menstrual abnormalities.

General physical examination revealed that the patient was moderately built and poorly nourished. Both upper

and lower limbs pulses were very feeble and the blood pressure was not recordable. A thrill was felt in carotid arteries and no clubbing, cyanosis, lymphadenopathy, pedal edema, and goiter was recorded. On cardiovascular examination, there was short early diastolic murmur of grade 2/6 heard in aortic area with loud first and second heart sounds in all areas. No abnormal findings were reported during respiratory, abdominal and nervous system examinations. Chest X-ray was normal. The significant findings of lab investigations are given in table 1.

Table 1: Significant findings of lab investigations

Parameters evaluated	Values
Hb	7.6 gm%
Total leucocyte count	8,910/ μ L
Platelet count	2,20,000/ μ L
ESR	70 mm/hr
C-reactive protein	7.23 mg/dl
RA factor	Negative
ANA, p-ANCA and c-ANCA	Negative
Albumin	2.5g/dl
Globulin	4.9 g/dl

Electrocardiogram revealed sinus tachycardia with heart rate of 130/min. 2D echocardiogram showed mild concentric left ventricular hypertrophy, moderate left ventricular global hypokinesia, grade III left ventricular diastolic dysfunction, and ejection fraction of 52%. Doppler of both carotid arteries revealed homogeneous circumferential thickening of affected vessels with flow velocity elevations beyond stenotic segments. Ultrasound examination of abdomen and pelvis revealed small right kidney with right renal arterial stenosis.

CT aortic angiography demonstrated concentric arterial wall thickening of nearly the whole aorta, its branches, and the pulmonary trunk (Figure 1). Severe stenosis was noted

involving the origins of right brachiocephalic artery, left subclavian and left common carotid arteries (Figure 2). The pulmonary arteries beyond bifurcation were normal with no evidence of wall thickening. Wall calcification was present in the suprarenal aorta and superior mesenteric artery. Significant luminal narrowing of abdominal aorta was noted in the infrarenal segment with total occlusion of aorta up to bifurcation. Reformation of the external and internal iliac arteries from the collaterals arising from mesenteric arteries was noted. The infrarenal aortic branches were not visualized (Figure 3). Aneurysmal dilatation of the supra renal abdominal aorta and left renal artery was noted. No inflammatory infiltrates, granulomas or giant cells were seen in right superficial temporal artery biopsy.

Figure 1: CT angiogram showing circumferential wall thickening of descending aorta

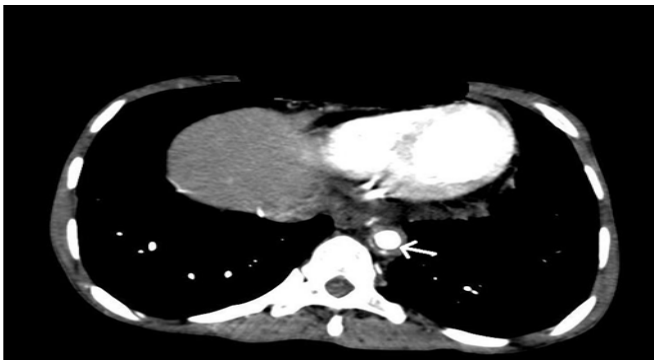
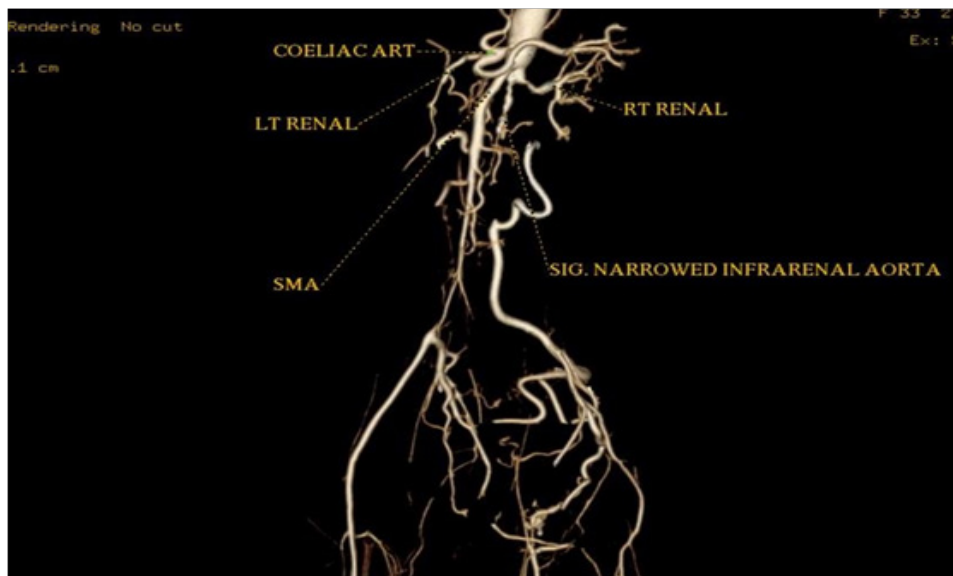


Figure 2: CT angiogram showing narrowed origins of left common carotid and left subclavian artery



Figure 3: Volume-rendered image of CT angiography indicating segmental occlusion of infrarenal aorta with a narrowed right renal artery



MRI scan of LS spine (Figures 4 and 5) revealed disc desiccation with left paracentral disc bulge at L5-S1 level, causing left foraminal stenosis and left exiting nerve root compression.

Based on the clinical evaluation, the diagnosis was confirmed as TA with compressive radiculopathy secondary to L5-S1 disc prolapse and moderate

normocytic normochromic anemia. The patient refused any surgical intervention and she was treated orally with methotrexate 10 mg/week, prednisolone 20 mg twice daily, and NSAIDs. A good symptomatic relief was observed two weeks after the initiation of the treatment. She was discharged and advised to do follow-up once in 3 months to identify any systemic manifestations.

Figure 4: Sagittal T2 weighted MRI image showing disc bulge at L5-S1 level indenting the thecal sac

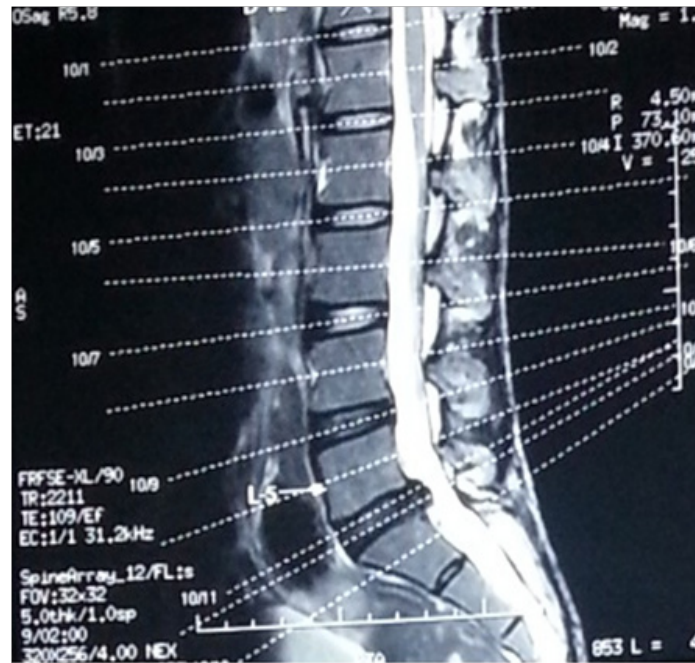
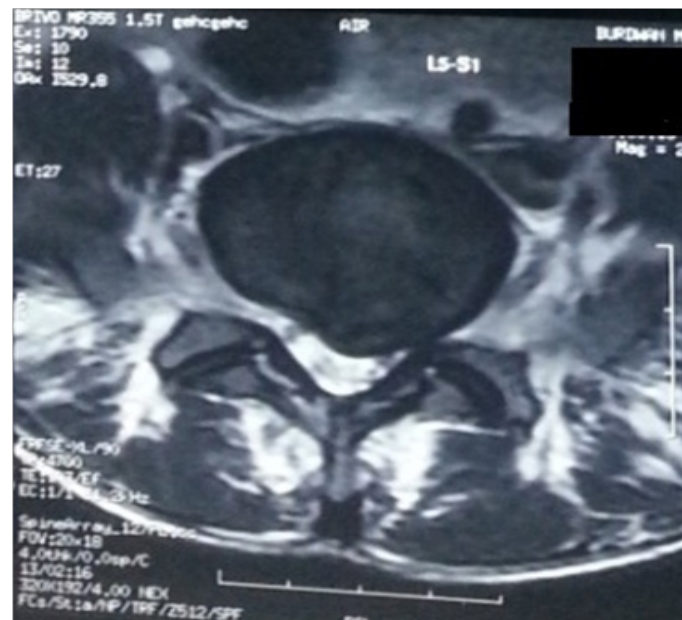


Figure 5: MRI of LS spine showing left paracentral disc bulge at L5- S1 level causing left foraminal stenosis and left exiting nerve root compression



Discussion

The present case, fulfilled most of the American College of Rheumatology criteria for TA including claudication pain in both lower limbs, decreased pulses, angiographic evidence of narrowing or occlusion of aorta and its branches.² The radicular pain noted in the present case was due to nerve root compression developed secondary to disc prolapse. The patient was evaluated further for reduced or absent pulse, in addition to root compression. The common clinical presentation of TA are a) reduced /absent pulses in 84-96% of cases b) bruit in 80-94% c) hypertension in 33-83% d) renal artery stenosis in 28-75% e) aortic regurgitation in 20-24% and f) pulmonary artery involvement in 14-100%.³

The exact etiopathogenesis of TA is unknown and its causal relationship with any of the aforementioned abnormalities has not been well established.⁴ It has been proposed that an increased expression of heat shock protein 60 in combination with a special population of T cells leads to cytotoxic reaction in TA patients and eventual damage of the aortic endothelium.⁵

The disease often begins as an inflammation of tunica adventitia, subsequently involving tunica media and tunica intima leading to thickening, fibrosis, segmental stenosis, aneurysm and thrombus formation.⁶ It progresses in a triphasic pattern. Phase I is the systemic or pre-pulseless period characterized by constitutional symptoms and phase II is the vasculitic stage where constitutional symptoms are accompanied by features of vascular involvement. Phase III is the late, fibrotic, occlusive, phase, wherein characteristic features of TA (pulseless disease) related to arterial stenosis or occlusion appear.⁶ Various criteria proposed for diagnosis of TA include American College of Rheumatology criteria, Ishikawa's criteria, and modified diagnostic criteria for TA by Sharma *et al.*^{2, 7, 8}

In the current case study, ANA was negative, VDRL was nonreactive, and the characteristic features of sarcoidosis like skin lesions, hilar adenopathy on chest X-ray, and Bell's palsy were absent. The presence of giant cell arteritis was ruled out, since the patient was young and the result of superficial temporal artery biopsy was normal. As per the literature evidence, around 25% of the cases of TA were diagnosed at >40 years of age.⁹ The pickup rate of TA is high in patients with suspected

diagnosis. The current case illustrates such an example. Though clinical presentation was backache with radicular pain, unexplainable symptoms led to the diagnosis.¹⁰

Conclusion

TA should be considered in women with constitutional features, hypertension, absence of pulse or low volume pulse, and carotid artery bruit. Early diagnosis and institution of appropriate treatment is crucial to prevent further progression and complications like aortic regurgitations and cerebrovascular accidents.

Competing interests

The authors declare that they have no competing interests.

Citation

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