Case Report:

A rare case of Takayasu’s arteritis presenting with stroke

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ABSTRACT

Takayasu’s arteritis is a chronic inflammatory large vessel vasculitis of unknown aetiology that commonly affects women of child bearing age. It appears to have an acute early phase, with non-specific symptoms such as hypertension, headache, fever, muscle pain, arthralgia, night sweats and weight loss. Stroke, as presenting feature of Takayasu’s arteritis is rare. Our patient presented with brocas aphasia, right hemiplegia and right-sided facial palsy. Physical examination revealed left-sided carotid bruit and absent peripheral pulses. CT angiography showed to have right subclavian, left internal carotid and middle cerebral arteritis. She was treated with immunosuppressive treatment and showed significant improvement. Though subclavian artery involvement is not rare, presentation with stroke is rare. Takayasu’s arteritis must be considered as a cause of stroke in young women.

Key words: Takayasu arteritis, Ischaemic stroke, Vasculitis, Central nervous system


INTRODUCTION

Takayasu’s arteritis, is an inflammatory large vessel vasculitis of unknown aetiology that commonly affects women of child bearing age.1,2 The most common presenting vascular symptoms are claudication (35%), reduced or absent pulse (25%), carotid bruit (20%), hypertension (20%), carotidynia (20%), light headedness (20%) and asymmetrical blood pressure in arms (15%). Stroke, aortic regurgitation and visual abnormalities are present at onset in less than 10% of patients.3 The most common sites of lesions in Takayasu’s arteritis are aorta (65%) and the left subclavian (93%) arteries.3 We present a rare case of Takayasu’s arteritis involving right subclavian artery, presenting with stroke.

CASE REPORT

A 19-years-old, unmarried female patient presented with sudden onset of weakness of right upper and lower limbs, deviation of angle of mouth to left and inability to speak but preserved comprehension. There was no history of loss of consciousness, seizures, head injury, fever during the episode. She had no past history of diabetes mellitus, hypertension, tuberculosis, heart disease or seizures. She had no family history of similar illness. She was a non-smoker and did not consume alcohol. Her bowel and bladder habits were normal.

On examination, she was conscious and coherent. She was moderately built and nourished. There was no pallor, icterus, cyanosis, clubbing, pedal oedema or generalised lymphadenopathy. Pulse was 82/ min, regular. Her right radial, brachial pulses were not palpable, right carotid was feeble. All other peripheral pulses were felt. A bruit could be heard over the left carotid. Her blood pressure was 130/90 mm Hg in left upper limb.
in supine position and 140/90 mm Hg in both lower limbs over popliteal artery. It was not recordable over right brachial artery. On nervous system examination, brocas aphasia, right upper motor neuron type facial palsy and right hemiplegia were present. Vision was 6/9 in both eyes and fundus showed diffuse venous dilatation and mild arteriolar narrowing (Figure 1). Cardiovascular system was normal on examination. Laboratory evaluation revealed haemoglobin 11.4 g/dL, total leucocyte count 10,800/mm$^3$ with normal differential count, platelet count 220,000/mm$^3$. Erythrocyte sedimentation rate was 85 mm at the end of the first hour, C-reactive protein tested positive. Sickling test was negative. Random blood glucose was 94 mg/dL, serum creatinine 0.9 mg/dL. Serum total cholesterol was 173 mg/dL, low density lipoprotein 121 mg/dL, high density lipoprotein 32 mg/dL and triglycerides 99 mg/dL. Magnetic resonance imaging (MRI) of the brain revealed acute non haemorrhagic infarct in left frontotemporal and basal ganglia regions extending to corona radiata (left middle cerebral artery territory) (Figure 2). Computed tomography (CT) aortography revealed irregular, circumferential luminal narrowing in right brachiocephalic artery at its origin, gross uniform narrowing of the right subclavian artery and right common carotid artery suggestive of aorto-arteritis, (Takayasu’s disease) (Figure 3). CT cerebral angiography revealed occlusion of distal portion of ICA in the neck and in petrous and cavernous portion and reduced caliber of A1, M1 and M2 segments on left side suggestive of arteritis (Figure 4).

**DISCUSSION**

Takayasu’s arteritis, a potentially life threatening illness is a granulomatous inflammation of large arteries. It appears to have an acute early phase, with non-specific symptoms such as hypertension, headache, fever, muscle pain, arthralgia, night sweats and weight loss. Due to non-specific symptoms and absence of specific laboratory parameters, the disease is often unrecognized at this phase. The most common manifestations of Takayasu’s arteritis are limb claudication and ischemia due to peripheral vascular involvement, hypertension from renal artery stenosis, ophthalmologic disease as manifested by retinopathy or amaurosis fugax, aortic regurgitation resulting from dilatation of the ascending aorta, cardiac ischaemia or congestive heart failure as a result of hypertensive and aortic disease, pulmonary hypertension from pulmonary arterial involvement, and neurologic disease (seizures and stroke) as a result of intra and extra-cranial arterial inflammation or thrombosis. Stroke is a common complication of Takayasu’s arteritis with an estimated incidence of 10%-20%. Stroke as the first manifestation, however, is rare. Since large-vessel biopsies are most often not possible, the diagnosis of TA is based on clinical criteria. Laboratory investigations should support the diagnosis of TA and imaging results must be confirmatory.

The presence of 3 or more of the six criteria of the American College of Rheumatology is sensitive (91%) and specific (98%) for the diagnosis of Takayasu’s arteritis.
Stroke, as presenting feature of Takayasu’s arteritis is rare. Involvement of intracranial vasculature is rather unusual. Approximately 10%-20% of patients with Takayasu’s arteritis are likely to have cerebrovascular accidents. Occlusion of vertebral or carotid arteries may cause ischemic stroke. Patients with Takayasu’s arteritis may also develop intracranial aneurysms. Embolism of stenotic or occlusive lesions of the aortic arch and its branches, hypertension, cardio embolism and cerebral hypoflow have been postulated as the mechanisms for occurrence of stroke in Takayasu’s arteritis.

Our patient presented with stroke as the initial manifestation and she fulfilled American college of Rheumatology Criteria for Takayasu’s arteritis. She has type 1 disease (as per angiographic classification proposed by International Cooperative study on Takayasu’s arteritis) showing severe involvement of aortic arch and its branches. Though subclavian artery...
involvement is not that uncommon, presentation with stroke is rare.1

Patient was treated with prednisolone 40 mg/day, mycophenolate 500 mg/day, aspirin and physiotherapy. She responded well and improved symptomatically over 3-4 weeks.

Though stroke at presentation is seen in only 5% of cases of Takayasu’s arteritis,7 the disease should be suspected in young female patients of less than 40 years. All peripheral pulses should be examined for any bruit. Immunosuppressive therapy can reduce the morbidity.

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