Acute ischemic stroke in a patient of Takayasu's arteritis

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ABSTRACT

Takayasu arteritis (TA) is a chronic inflammatory condition usually seen in females mainly affecting aorta and its branches resulting in clinical manifestations such as malaise, claudication, and pulselessness. Raised erythrocyte sedimentation rate and C-reactive protein are important markers of active inflammation other than clinical features associated. Rarely TA may involve coronary and cerebral arteries resulting in myocardial infarctions and cerebrovascular accidents.

KEY WORDS: Takayasu Arteritis; Cerebrovascular Accident; Ischemic Stroke

INTRODUCTION

Takayasu arteritis (TA) is a medium and large vessel vasculitis of unknown etiology characterized by chronic granulomatous inflammation of the vessel wall leading to thickening, stenosis, dilatation, and/or aneurysm of the affected vessel. It primarily affects the aorta and its main branches. It can also affect the coronary and pulmonary arteries. Here, we are reporting a case of acute ischemic stroke in a young female patient of TA who presented to us with weakness of the right half of body with motor aphasia.

CASE REPORT

A 29 years female patient, who was diagnosed as a case of TA about 1 year earlier, presented with complaints of sudden onset weakness of her the right upper limb and right lower limb of 10 days duration. It was associated with inability to speak. The patient had a history of taking oral steroids that were discontinued after 6 months. On examination, the patient was conscious and oriented. Her radial and ulnar pulses on both sides were feeble. The lower limb pulses were normally felt. Blood pressure measurements showed a significant difference between the left and right upper limbs (110/76 mm Hg on the left upper limb and 86/60 mmHg on the right upper limb). A bruit was heard over her right carotid artery. There was motor aphasia with the right sided hemiparesis. Her right plantar reflex showed an extensor response. Cardiac, abdominal, and respiratory system examination revealed no significant abnormality. There was no history of oral contraceptive pill use. There was no history of smoking or recreational drugs. Initial investigations revealed high total leukocyte counts (21,500/cmm, neutrophils - 96%, and lymphocytes - 3%), raised liver enzymes (serum glutamic oxaloacetic transaminase - 90 IU/L, and serum glutamic pyruvic transaminase - 171 IU/L), low serum total protein (5.5 g/dl), and low serum albumin (3 g/dl) levels. Hemoglobin level was 12.4 g/dl and serum urea, serum creatinine, and serum electrolytes were all within normal limits. C-reactive protein (CRP) was positive, and erythrocyte sedimentation

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rate (ESR) was 39 mm after the 1st h. Serum antineutrophil antibodies and rheumatoid factor (rheumatoid arthritis factor) were negative. Routine and microscopic examinations of urine revealed no significant abnormalities. There was no proteinuria. Appropriate antibiotic therapy was started according to blood culture and sensitivity reports. A magnetic resonance imaging (MRI) of the brain revealed an acute to subacute infarct involving the left middle cerebral artery (MCA) and anterior cerebral artery (ACA) territories (Figure 1). Time of flight magnetic resonance angiography showed abrupt cut-off of the left MCA and narrowing of the left ACA (Figure 2). Computed tomography angiography of the aorta and its branches revealed nonspecific aortoarteritis characterized by diffuse wall thickening with significant luminal narrowing involving brachiocephalic artery, bilateral common carotid artery, internal carotid artery, and bilateral subclavian arteries. The aortic arch wall was also mildly thickened. The proximal coeliac artery also showed significant stenosis with post-stenotic dilatation. Echocardiography revealed a structurally normal heart with normal the left ventricular function. During hospitalization, patient received pulse methylprednisolone therapy for 3 days (1 g/day) which was followed by oral steroid therapy at a dose of 1 mg/day. The patient also received daily doses of 75 mg of aspirin and 10 mg of atorvastatin. After 1 month of steroid therapy, markers of inflammation, such as CRP and ESR, were within normal limit. After a month of steroid therapy, methotrexate at a dose of 15 mg weekly was started. She is currently on regular follow up.

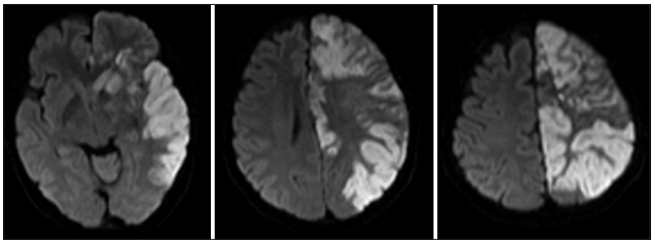

Figure 1: Magnetic resonance imaging (diffusion weighted images) showing acute to subacute infarcts involving the left middle cerebral artery and anterior cerebral artery territory

Figure 2: Magnetic resonance angiography of cerebral vessels showing abrupt cut-off of the left middle cerebral artery (red circle) and narrowing of the left anterior cerebral artery

DISCUSSION

Takayasu's arteritis was first described by a Japanese ophthalmologist, Dr. Mikito Takayasu, In 1905, in a 21-year-old woman who presented with syncope and absent upper extremity pulses. It is a relatively rare vasculitic syndrome and is mostly seen in Asia and Mexico.[1] The patients of TA usually present with non-specific symptoms such as fatigue, malaise, fever, night sweats, weight loss, and arthralgia. Organ ischemia resulting from vascular insufficiency due to stenosis, occlusion, or aneurysm develop months to years later. Neurological involvement includes headache, dizziness, visual disturbances, seizures, transient ischemic attack (TIA), stroke, and posterior reversible encephalopathy syndrome.[2] Ischemic stroke or TIA is rare and is reported to occur in 10-20% of patients suffering from TA.[3] There have also been reports of patients with TA presenting with an acute ischemic stroke as the first clinical presentation.[4] TA patients with stroke are younger and have none of the conventional risk factors associated with stroke due to atherosclerotic mechanisms. Hwang J et al. in their analysis of 207 TA patients reported a relatively lower prevalence of stroke risk factors in TA patients with the exception of hypertension and dyslipidemia. The relatively higher incidence of hypertension was attributed to increase renal artery involvement in TA.[5] There are many possible mechanisms of stroke in TA and includes embolism, hypertension, cardioembolism, and cerebral hypoflow.[6] Intracranial stenosis could also result from vasculitic involvement of the vessel.[7] TA patients have also been shown to have premature atherosclerotic plaques in their arteries which may lead to thromboembolisms through mechanisms quite similar to patients with atherosclerotic diseases.[8] The exact etiology of ischemic stroke in our patient is unknown. Based on the presentation and the MRI images of our patient, it is likely that she suffered an embolic stroke. A transthoracic echocardiography did not reveal any source of the embolus in the heart. Our patient probably had an artery to artery embolism resulting in massive ischemic stroke.

CONCLUSION

In patients with young stroke with acute presentation TA should always be kept under differential diagnosis, other than coagulation disorders and proper imaging should be conducted as a proper diagnosis helps in proper management.

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