Case Report

Unraveling Takayasu arteritis and antiphospholipid syndrome in the context of ischemic stroke

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ABSTRACT

This case study explores the complex etiology of bilateral stroke in a 35-year-old female with Takayasu arteritis and antiphospholipid syndrome. The patient presented with altered sensorium and recurrent seizures, with imaging indicating large-vessel involvement. Management included anti-thrombotic and anti-platelet therapy, immunosuppressants, and antibiotics, targeting both vasculitis and thrombotic complications. The inflammatory state posed challenges in proceeding with interventions, highlighting the need for careful risk assessment. This case underscores the interplay of autoimmune disorders in stroke pathogenesis, emphasizing the necessity of a multidisciplinary approach to optimize patient care and improve long-term outcomes.

Key words: Antiphospholipid antibodies, Antiphospholipid syndrome, Autoimmune disorders, Infarct, Stroke, Takayasu arteritis, Vasculitis, Young's stroke

Takayasu arteritis (TA) is a granulomatous large vessel vasculitis affecting the aorta and its branches. The diagnosis of TA relies on clinical features and imaging-based classification systems such as those proposed by De Souza and De Carvalho, which emphasize vascular involvement across multiple arterial territories [1]. Predominantly observed in young women and more common in Asia, TA has a slow progression, with a reported 10-year survival rate of 85%. However, its clinical course is unpredictable, marked by frequent exacerbations that can lead to permanent organ dysfunction and mortality [2]. Antiphospholipid syndrome (APS) is an autoimmune disorder linked to recurrent thrombosis and pregnancy complications due to autoantibodies [3]. It presents with diverse clinical and epidemiological features, often complicating coexisting autoimmune conditions such as vasculitis [4]. The presence of antiphospholipid antibodies (aPL) suggests a potential for increased vascular damage in vascular diseases like TA, given their thrombotic effects. Despite studies suggesting an association between aPL and TA severity, the clinical significance of these antibodies in TA remains debated [5].

The rationale for reporting this case is to highlight the varied clinical presentations of TA and its possible association with APS, thereby emphasizing the importance of screening for aPL in patients with TA presenting with thrombotic or atypical features [6].

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CASE REPORT

A 35-year-old female presented to Shri Mahant Indiresh Hospital emergency with altered sensorium for 9 days, following an acute onset of generalized tonic-clonic seizure episodes. The seizures were associated with abnormal body movements, up-rolling of eyes, frothing from the mouth, and clenching of teeth, predominantly on the left side, as observed by her husband. These episodes were also accompanied by bowel and bladder incontinence. Initially, the patient was drowsy; however, after approximately 3 h, she became responsive but was unable to comprehend or follow commands. The patient was diagnosed with a left-sided middle cerebral artery (MCA) territory infarct 6 months ago, leading to right-sided hemiparesis with residual weakness in the right side of the body for which she was on medications.

General physical examinations were all within normal limits except the patient was not oriented to time, place, and person. The bulk was slightly decreased on the right side. The examination revealed non-recordable blood pressure in the left upper limb, absent pulses in the left upper limb, and hypertonia. Local examination under auscultation revealed an aortic regurgitation

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murmur radiating to the left carotid area. Plantar was a bilateral extensor. Reflexes were bilaterally presented with a little asymmetrical. The patient exhibited a non-responsive state (Glasgow Coma Score E4V2M4) and primitive reflexes.

Investigations of the patient are shown in Table 1. Computed tomography (CT) aortogram demonstrated diffuse circumferential wall thickening of the left common carotid artery. The left subclavian artery showed severe circumferential wall thickening extending approximately 4.7 cm from its origin. Similar diffuse mural thickening was noted in the brachiocephalic artery, along with moderate luminal narrowing. In addition, mild circumferential mural thickening with mild luminal narrowing was observed in the descending thoracic aorta (Fig. 1). CT imaging of the brain revealed a large wedge-shaped hypodensity (HU-19) in the right frontal region with loss of gray-white matter differentiation, suggestive of an acute/subacute infarct involving the right anterior cerebral artery (ACA), MCA, and ACA/MCA watershed territories (Fig. 2). Chronic changes were also noted in the left frontal region, including encephalomalacia and gliosis involving the left ganglio-capsular region, external capsule, left insular cortex, and corona radiata (Fig. 2). Magnetic resonance imaging angiography was planned to further evaluate the vascular involvement; however, it could not be performed due to financial constraints.

For acute cerebrovascular accident, low-molecular-weight heparin (LMWH) is administered along with dual antiplatelet therapy. To treat the root cause of large vessel arteritis, 40 mg of methylprednisolone was administered once daily for 5 days. The associated sepsis was treated with culture-sensitive antibiotics. Due to the patient's ongoing sepsis and raised erythrocyte sedimentation rate and C-reactive protein, which indicate an active inflammatory process, the initial scheduled peripheral angiography and stent placement, which was supposed to deal with the large vessel presence, had to be postponed.

During the course of hospitalization, the patient developed a hospital-acquired infection, which progressed to severe sepsis irrespective of ongoing management. Unfortunately, the patient did not survive. This unexpected and rapid impairment prohibited any further clinical follow-up or long-term outcome assessment.

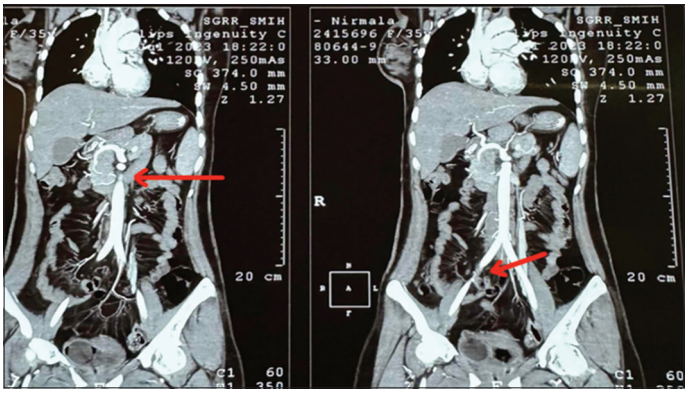

Figure 1: Computed tomography aortogram showing mural thickening and luminal narrowing involving the thoracic aorta and major branches (red arrows)

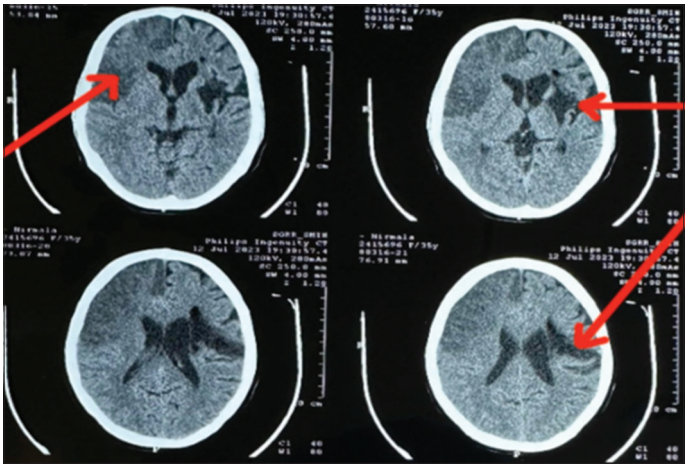

Figure 2: Axial computed tomography brain images showing (right): Wedge-shaped hypodense area in the right frontal lobe indicating acute/subacute infarction; (left): Encephalomalacic changes in the left frontal region consistent with chronic ischemic injury and gliosis

DISCUSSION

The aorta and its major branches are the primary targets of TA, a rare, chronic large-vessel vasculitis that progresses over time and causes ischemia, fibrosis, and stenosis. Its clinical course is unpredictable, alongside phases of inflammation in addition to vascular occlusion, and it primarily affects young women. An early inflammatory phase causes vascular damage in TA, which subsequently develops into fibrosis and narrowing, resulting in decreased perfusion and ischemic complications such as myocardial infarction, stroke, and limb ischemia. Direct vascular obstruction, artery-to-artery embolism, and compromised collateral circulation can all result in stroke in TA. Conversely, APS is an autoimmune disease marked by hypercoagulability and recurrent thrombosis brought on by aPL, such as anticardiolipin and lupus anticoagulant antibodies. When paired with TA, the

Table 1: The investigations done with their results

Test	Result
Hb/TLC/PL COUNT	11.4 g/dL/21900/cc/200 thou/cc
Urea/Creatinine	44/0.5 mg/dL
Sodium/Potassium/Calcium	143/4.3/8.3 mmol/L
T.BIL/D.BIL/I.BIL	1.1/0.7/0.40 mg/dL
Albumin	3.40 g/dL
SGOT/SGPT/ALP/GGT	41/22/85/141 unit/L
Homocysteine	2.00 umol/L
ESR/CRP	89 mm/1st h/224 mg/dL
Lipoprotein (A)	81.3 mg/dL (H)
Lupus anticoagulant dRVVT ratio	77 s (H)/2.40
ANA/ANCA PROFILE	Negative
BETA-2 GP/cardiolipin-IgG/IgM	Negative

Hb: Hemoglobin, TLC: Total leukocyte count, PL: Platelet, T.BIL: Total bilirubin test, D.BIL: Direct bilirubin test, I.BIL: Indirect bilirubin test, SGOT: Serum glutamic-oxaloacetic transaminase, SGPT: Serum glutamic pyruvic transaminase, ALP: Alkaline phosphatase, GGT: Gamma-glutamyl transferase, ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein, ANA: Antinuclear antibody, ANCA: Antineutrophil cytoplasmic antibodies

probability of stroke is greatly increased because of a combination of inflammatory vascular stenosis as well as hypercoagulability. APS is one of the leading causes of arterial thrombosis.

In this case, a 35-year-old woman presents with absent pulses in her left upper limb, seizures, and recurrent strokes. Large-vessel arteritis was confirmed by a CT aortogram that showed circumferential mural thickening in addition to narrowing of the luminal in the left brachiocephalic, left subclavian, and left common carotid arteries. Multiple infarcts on brain imaging suggested a combination of embolic stroke and ischemia related to vascular stenosis. The diagnosis of APS was further supported by elevated levels of lipoprotein (a) and lupus anticoagulant. This patient's co-occurrence of TA and APS resulted in a dual cause for stroke, with APS increasing the possibility of thrombotic events and TA contributing to large-vessel occlusion.

In a published case by Memon *et al*., the patient also presented with cerebral infarction as the first manifestation of TA; however, that case lacked an APS overlap and primarily focused on diagnostic delays and vascular imaging [7]. Our patient, on the other hand, had a dual stroke mechanism: Vasculitic occlusion due to TA and a hypercoagulable state due to APS, which posed unique therapeutic challenges. This makes our case more complex in terms of both diagnosis and management. Hall *et al*. conducted a study of 32 North American patients with TA, identifying variable systemic presentations but emphasizing that neurological symptoms were not always the first sign [8]. Similarly, Khealani and Baig and Silver highlighted TA-induced ischemic strokes but did not address autoimmune thrombophilia as a cofactor [9,10]. Our case adds to this literature by demonstrating how the co-existence of APS can significantly increase morbidity in TA and demand a different treatment algorithm. Although rare, overlap syndromes involving TA, systemic lupus erythematosus (SLE), and APS have been reported, with stroke being a major clinical manifestation in such cases [11].

Differential diagnoses for bilateral infarcts in young females include SLE, other vasculitis, infective endocarditis, hyperhomocysteinemia, giant cell arteritis, sepsis, and human immunodeficiency virus. These were ruled out in our patient through negative serologies and clinical workups.

Treating TA with APS poses special difficulties since anticoagulation to avoid thrombosis and immunosuppression to regulate vascular inflammation must be balanced. The patient received maintenance corticosteroids to reduce inflammation after starting pulse methylprednisolone therapy (1g IV for 5 days). LMWH and dual antiplatelet therapy (aspirin and clopidogrel) were initiated, with the goal of switching to long-term oral anticoagulation. However, because anticoagulation increases the risk of restenosis, hemorrhage, and vessel fragility in patients with active vasculitis, it must be closely monitored. Due to constantly elevated inflammatory markers, which indicate current disease progression and a higher chance of procedural complications, revascularization through angioplasty and stenting was suggested but ultimately postponed.

Lifelong care is necessary for both TA and APS, including routine vascular imaging, anticoagulation adjustments, and monitoring of inflammatory markers. This case emphasizes how crucial early detection, interdisciplinary teamwork, and tailored treatment plans are to the management of complicated autoimmune-related strokes. A customized strategy is necessary to reduce the risk of complications, improve long-term results, and strike a balance between thrombosis prevention and inflammation control.

CONCLUSION

This instance highlights the intricate relationship between thrombophilia in APS and inflammatory vasculitis in TA, demonstrating the multifactorial character of stroke in young individuals with autoimmune diseases. Cerebrovascular risk is greatly increased when these conditions coexist, necessitating a customized treatment strategy. Immunosuppression is necessary to manage vascular inflammation in TA, whereas anticoagulation is required for APS to avoid thrombotic complications. Because active inflammation raises procedural risks, the duration of revascularization needs to be carefully considered. This case highlights how crucial early detection, interdisciplinary teamwork, and tailored treatment are to improving patient outcomes and averting repeat ischemic episodes.

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