Case Report

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Internal Medicine Section

Young-onset Ischaemic Stroke Secondary to Antiphospholipid Syndrome Associated with Mitral Valvular Lesion: A Case Report

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ABSTRACT

Young-onset ischaemic stroke warrants evaluation for uncommon aetiologies, particularly autoimmune and hypercoagulable disorders. Antiphospholipid Syndrome (APS) is an important cause of ischaemic stroke in young adults and is frequently associated with recurrent pregnancy loss and cardiac valvular lesions. A 32-year-old hypertensive female presented with weakness of the left lower limb for three days. Neurological examination revealed monoparesis with exaggerated reflexes and bilateral extensor plantar responses. Magnetic Resonance Imaging (MRI) of the brain demonstrated multiple acute non haemorrhagic infarcts in the right frontoparietal region, along with evidence of previous infarction. Echocardiography revealed a highly mobile echogenic mass attached to the posterior mitral leaflet associated with mitral regurgitation, suggestive of a cardioembolic source. Laboratory investigations demonstrated prolonged activated Partial Thromboplastin Time (aPTT), positive lupus anticoagulant, elevated anticardiolipin Immunoglobulin G (IgG) antibodies, and positive Antinuclear Antibody (ANA) titre. A history of recurrent pregnancy loss further supported the diagnosis of secondary APS with probable cardioembolic stroke. Young stroke associated with adverse obstetric history should raise suspicion for APS. Cardiac valvular lesions in APS may act as embolic sources and contribute to recurrent ischaemic events. Early diagnosis and multidisciplinary management are essential to prevent recurrent thrombotic complications and improve long-term neurological outcomes.

Keywords: Cardioembolic stroke, Libman-Sacks endocarditis, Recurrent pregnancy loss

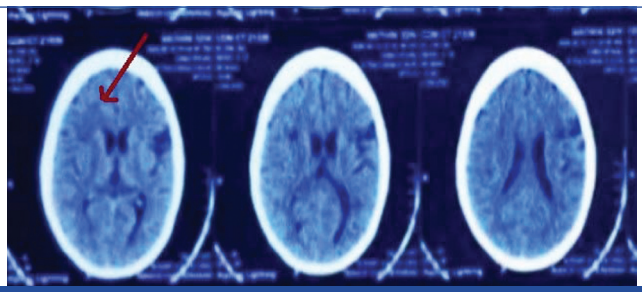
CASE REPORT

A 32-year-old female, a known case of systemic hypertension, presented with weakness of the left lower limb for three days. The weakness was insidious in onset and non progressive. She had been apparently asymptomatic three days prior to presentation, following which she developed difficulty in walking and inability to hold her slippers properly while ambulating.

There was no history suggestive of raised intracranial pressure or cortical involvement, including headache, vomiting, seizures, loss of consciousness, sensory disturbances, slurring of speech, or visual disturbances. She denied bowel or bladder disturbances. Initial evaluation at a peripheral hospital included Computed Tomography (CT) of the brain, which demonstrated a chronic right frontal lobe infarct without evidence of acute intracranial haemorrhage. She was subsequently referred for further evaluation [Table/Fig-1].

Past History and Family History

The medical history of the patient was significant for pregnancy-induced hypertension during her previous pregnancies, approximately 4-5 years prior to the present admission. She had been treated with labetalol during pregnancy and became normotensive after delivery. At the time of the present admission, she was not on regular antihypertensive medication. Her obstetric history revealed a live preterm birth during the first pregnancy at seven months of gestation. The second and third pregnancies were complicated by pre-eclampsia at approximately six months of gestation, followed by pregnancy loss/abortion. There was no history of substance use, known autoimmune disorder, premature cerebrovascular disease in the family, or consanguineous marriage.



[Table/Fig-1]: Initial CT performed at the Outpatient Department, Chidambaram, showed hypodensity noted in right frontal lobe (red arrow), indicating a chronic infarct. No acute infarct or haemorrhage was noted.

Clinical Examination

On examination, the patient was conscious, oriented, and haemodynamically stable. Her blood pressure was 150/90 mmHg, pulse rate was 88 beats/minute, respiratory rate was 21 cycles/minute, and oxygen saturation was 98% on room air.

Cardiovascular examination revealed a loud pulmonary component of the second heart sound along with systolic murmurs in the pulmonary and mitral areas. Respiratory system examination demonstrated normal vesicular breath sounds bilaterally without added sounds. Abdominal examination was unremarkable.

Neurological examination revealed a Glasgow Coma Scale (GCS) score of 15/15 (E4V5M6), intact higher mental functions, and preserved cranial nerve function. Motor examination demonstrated reduced muscle power (3/5) in the left lower limb with exaggerated knee reflexes and bilateral extensor plantar responses. Sensory examination was normal, and cerebellar signs were absent [Table/Fig-2]. Gait assessment demonstrated circumduction gait.

Laboratory Investigations

Routine laboratory investigations were within normal limits except for a markedly prolonged aPTT of 73.3 seconds, prompting evaluation for an underlying coagulation abnormality [Table/Fig-3].

Neurological examination parameters	Right-side	Left-side
Pupils	2 mm, reacting to light	2 mm, reacting to light
Tone (Upper and Lower Limbs)	Normal	Normal
Power	5/5 (Upper Limb), 5/5 (Lower Limb)	5/5 (Upper Limb), 3/5 (Lower Limb)
Knee flexion	Present	Unable to perform
Knee extension	Present	Unable to perform
Foot plantar flexion	Present	Lost
Foot dorsiflexion	Present	Lost
Hand grip	100%	100%
Toe grip	100%	0%
Biceps reflex	++	++
Triceps reflex	++	++
Knee reflex	+++	Exaggerated
Ankle reflex	+	+
Plantar reflex	Extensor	Extensor

[Table/Fig-2]: Neurological examination on arrival.

differential considerations including healed vegetation, thrombus, or myxomatous degeneration. Although infective endocarditis was initially considered and empirical intravenous ceftriaxone was started after obtaining blood cultures, inflammatory markers including CRP and procalcitonin were negative (within reference range), and repeated blood cultures failed to demonstrate evidence of active infection [Table/Fig-5].

Rheumatology Evaluation and Findings

Further autoimmune investigations showed positive lupus anticoagulants, elevated anticardiolipin IgG antibodies, and positive ANA titre (1:320), strongly suggesting systemic lupus erythematosus with secondary APS [Table/Fig-3]. Taken together, the recurrent pregnancy morbidity, prolonged aPTT, recurrent Ischaemic infarcts, and valvular lesion pointed toward an autoimmune thrombotic aetiology rather than isolated infective pathology.

The patient was managed with dual antiplatelet therapy, anticoagulation with heparin, statins, antihypertensive medications, and supportive care. Multidisciplinary consultation involving neurology, cardiology, cardiothoracic surgery, and rheumatology teams was obtained for further evaluation and long-term management planning. Follow-up details were not available, as the patient was referred to a higher centre of the family's choice for further evaluation, including cardiac MRI and rheumatology consultation. The need for continued anticoagulation, repeat echocardiographic assessment, autoimmune work-up confirmation, and regular neurology, cardiology, and rheumatology follow-up was explained to the patient and her attendants at the time of referral.

Investigation	On admission	On day 5	Reference Range*	Interpretation
Haemoglobin	12.4 g/dL	9.8 g/dL	12-15 g/dL	Decline in haemoglobin levels
Total leucocyte count	9300 cells/mm^3	8100 cells/mm^3	4000-10000 cells/mm^3	Within normal limits
Platelet count	156000/mm^3	170000/mm^3	150000-450000 cells/mm^3	Normal platelet count
Mean Corpuscular Volume (MCV)	78.7 fL	78.1 fL	83-101 fL	Microcytic indices
Blood urea	17 mg/dL	16 mg/dL	12.8-42.8 mg/dL	Normal renal parameter
Serum creatinine	0.86 mg/dL	0.98 mg/dL	0.6-1.2 mg/dL	Preserved renal function
Sodium	137 mmol/L	137 mmol/L	138-145 mmol/L	Mild hyponatraemia
Potassium	3.8 mmol/L	3.7 mmol/L	3.5-5.1 mmol/L	Normal
Chloride	100 mmol/L	112 mmol/L	102-108 mmol/L	Mild hyperchloraemia on follow-up
Erythrocyte Sedimentation Rate (ESR)	30 mm/h	-	<12 mm/h	Elevated inflammatory marker
C-Reactive Protein (CRP)	Negative	-	Negative	No evidence of active inflammation
Procalcitonin	<0.30 ng/mL	-	<0.30 ng/mL	No evidence of bacterial sepsis
Prothrombin Time/International Normalized Ratio (PT/INR)	13.3 sec / 1.04	-	INR 1-1.5	Normal coagulation profile
aPTT	73.3 sec	-	27.5-38 sec	Markedly prolonged; suggestive of APS
Antinuclear Antibody (ANA)	Positive (1:320)	-	Negative: No Immunofluorescence	Positive autoimmune marker
Lupus anticoagulant	Positive	-	Negative: <100 Positive: ≥100	Supports diagnosis of APS
Anticardiolipin IgG Antibody	Positive (>120)	-	Negative: <12 Positive: ≥12	Supports diagnosis of APS
HIV, HBsAg, HCV	Non reactive	-	Non reactive	Viral markers negative

[Table/Fig-3]: Laboratory investigations during admission.
HIV-Human Immunodeficiency Virus; HBsAg-Hepatitis B Surface Antigen; HCV-Hepatitis C Virus
*The reference ranges mentioned are derived from the Institutional Central Laboratory reporting standards of Mahatma Gandhi Medical College and Research Institute, Pondicherry. These ranges may vary slightly between laboratories depending on analyser methodology, calibration, reagent systems, and population reference intervals.

Neurological Event and MRI Findings

The MRI of the brain demonstrated multiple acute non haemorrhagic infarcts involving the right frontoparietal region and right cingulate gyrus with diffusion restriction. In addition, chronic infarcts with encephalomalacic changes in the frontal lobes were noted, indicating previous silent or inadequately recognised cerebrovascular events [Table/Fig-4].

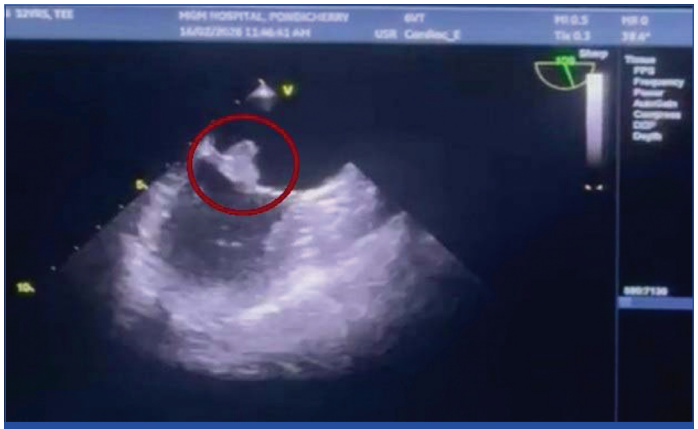
Cardiac Event and Echocardiographic Findings

Cardiac evaluation added another important dimension to the case. Transthoracic echocardiography revealed a highly mobile echogenic mass measuring approximately 10×13 mm attached to the posterior mitral leaflet, associated with mild mitral regurgitation. Subsequent Transoesophageal Echocardiography (TEE) confirmed a globular mobile lesion attached to the posterior mitral leaflet, with

DISCUSSION

Stroke in young adults requires consideration beyond traditional vascular risk factors when the clinical presentation suggests recurrent, embolic, autoimmune or hypercoagulable pathology. APS is an acquired autoimmune thrombophilic disorder characterised by arterial and/or venous thrombosis and/or pregnancy morbidity in the presence of antiphospholipid antibodies [1]. Ischaemic stroke is a

[Table/Fig-4]: MRI Brain (Plain and Contrast)-Plain MRI brain demonstrated acute infarcts involving the right frontoparietal region with chronic encephalomalacic changes in the left frontal lobe. Contrast-enhanced images showed no abnormal intracranial enhancement or evidence of haemorrhage. (blue arrow).



[Table/Fig-5]: Transoesophageal Echocardiography (TEE)- A highly mobile echogenic globular mass measuring 10×13 mm attached to the posterior mitral leaflet, suggestive of healed vegetation or myxomatous degeneration (Red circle). Mild mitral regurgitation was present with preserved left ventricular function and no evidence of abscess or pericardial effusion.

common arterial manifestation of APS, especially in young women, and may be the first clinical presentation [2]. In the present case, the young age at presentation, recurrent pregnancy loss, prolonged aPTT, lupus anticoagulant positivity, increased anticardiolipin IgG antibody, positive ANA titre and evidence of acute and chronic cerebral infarcts was strongly supportive of an autoimmune thrombotic mechanism rather than isolated hypertensive or atherosclerotic stroke.

The present case is clinically relevant as the patient had neurological as well as cardiac manifestations of APS. Brain MRI demonstrated acute non haemorrhagic infarcts in the right frontoparietal region and right cingulate gyrus and chronic encephalomalacia changes compatible with previous silent or under-recognised ischaemic events. Similar recurrent or multiple cerebral infarctions have been described in APS-associated stroke, in which thrombosis may be caused by endothelial dysfunction, platelet activation, complement-mediated injury and interference with natural anticoagulant pathways [2,3]. The history of adverse pregnancy outcome was also supportive of this diagnosis as recurrent foetal loss and pregnancy morbidity are the classic clinical manifestations of APS [1,4]. Pregnancy-induced hypertension and pre-eclampsia in previous pregnancies could have masked the underlying autoimmune thrombophilia propensity in the present patient and postponed the diagnosis of APS until the development of the cerebrovascular symptoms.

A remarkable finding in the present case was a mobile echogenic mass attached to the posterior mitral leaflet with mitral regurgitation. This raised the possibility of Libman-Sacks Endocarditis (LSE) a sterile non bacterial thrombotic endocarditis associated with systemic lupus erythematosus and APS [5,6] in the setting of APS and positive autoimmune markers. LSE frequently involves the mitral and aortic valves and may manifest as valve thickening, regurgitation or sterile vegetations [6,7]. In the present case, initial consideration was given to infective endocarditis, due to the combination of ischaemic stroke and a mobile mitral valve lesion. However, the lack of fever, negative inflammatory markers, negative repeat blood cultures and positive autoimmune serology favored a non infective thrombotic valvular lesion.

Similar observations are found in the literature. Liang H et al., reported a 26-year-old female with primary APS who developed multiple cerebral infarctions and mitral valve vegetations with mitral regurgitation [8]. The patient was similar to the current case in being young, having had APS-related cerebral ischaemia and mitral valve involvement. However, progression of mitral regurgitation on follow-up was reported by Liang H et al., necessitating mechanical mitral valve replacement, unlike the present patient [8]. This comparison emphasises the importance of serial echocardiographic monitoring in APS-related valvular lesions, as initially moderate lesions may progress despite medical treatment.

Takeuchi K et al., reported a case of persistent primary APS with recurrent stroke from enlarged LSE who underwent mitral valve replacement [9]. The case is similar to the present case in terms of recurrent ischaemic cerebral events and mitral valve vegetation as a probable source of emboli. However, the present patient was treated medically during index admission and referred for further evaluation while the patient reported by Takeuchi K et al. required surgical intervention on account of recurrent stroke and enlargement of valvular lesion [9]. This difference highlights that treatment decisions in APS-related LSE depend on size of vegetations, mobility, recurrent embolic events, severity of regurgitation and response to anticoagulation.

Yazidi MA et al., described a case of ischaemic stroke with LSE in a patient with mitral vegetations and severe mitral regurgitation [10]. The diagnosis was difficult, as in the present case, because infective endocarditis had to be excluded against sterile autoimmune valvular disease. However, their patient presented with fever and severe valvular dysfunction, while the present patient was afebrile and had negative inflammatory markers, which suggest a higher likelihood of non bacterial thrombotic endocarditis. This contrast supports the need to interpret echocardiographic findings in conjunction with clinical, microbiological, inflammatory and autoimmune parameters before arriving at the final diagnosis.

Gorantla A et al., also reported embolic phenomena in LSE associated with APS, supporting the fact that sterile valvular vegetations may be a source of emboli and may cause systemic or cerebral thromboembolic events [11]. This mechanism is supported by the present case in which the patient had a mobile lesion of the posterior mitral leaflet and multiple cerebral infarcts in different stages. The presence of old and new infarcts is compatible with recurrent thrombotic activity due to APS, cardioembolism from the mitral valve lesion, or both mechanisms. Thus, in the present case, after exclusion of active infective endocarditis, the case can be regarded as probable APS-associated cardioembolic stroke with suspected LSE.

The TEE is important in the assessment of APS-related valvular disease, since it is more sensitive than transthoracic echocardiography in the detection of vegetations, leaflet thickening and intracardiac thrombi [6,7]. Here, a transthoracic echocardiogram initially demonstrated a mobile echogenic mass, which was further defined by TEE as a globular mobile mass attached to the posterior mitral leaflet. This supports the recommendation that young stroke patients with APS features and a suspected cardioembolic source should be subject to detailed cardiac imaging, preferably including TEE.

Management of APS-related stroke with valvular involvement remains challenging. In general, long-term anticoagulation is recommended for APS patients with arterial thrombosis, especially if there is recurrent thrombosis or valvular lesions [1,4,5]. The patient in the present case was managed with antiplatelets, heparin anticoagulation, statin, antihypertensive therapy, empirical antibiotics initially and multidisciplinary evaluation including neurology, cardiology, cardiothoracic surgery and rheumatology. As reported by Roldan CA et al., combined anti-inflammatory and antithrombotic therapy may improve Libman-Sacks vegetations and associated cerebrovascular disease in selected patients [12]. However, surgical management may be necessary in patients with severe valve dysfunction, large vegetations or recurrent embolic events despite adequate anticoagulation as described by Liang H et al., and Takeuchi K et al., [8,9]. The present case highlights the necessity of an individualised approach depending on neurological recurrences, vegetation features, severity of mitral regurgitation, APS activity and imaging follow-up.

The present case also teaches us another important lesson in respect to the diagnostic value of a prolonged aPTT. Although APS is a prothrombotic disorder, lupus anticoagulant can paradoxically prolong phospholipid-dependent coagulation assays such as aPTT [13]. The markedly prolonged aPTT in the present patient prompted further investigations and contributed to the diagnosis. This finding should not be interpreted as a bleeding diathesis alone but rather should raise suspicion for APS in a young stroke patient with pregnancy morbidity.

This case is another addition to the literature regarding the coexistence of young-onset ischaemic stroke, previous silent cerebral infarction, negative obstetric history, positivity for antiphospholipid antibodies, and a mobile mitral valvular lesion. The clinical presentation in this patient was similar to the typical APS presentation in young females with pregnancy morbidity when compared to previously reported cases where some presented later in life or needed surgical valve replacement. Hence, the present case highlights the importance of early APS screening in young women with stroke, differential diagnosis between infective and non infective valvular vegetations, use of TEE to evaluate cardiac source and multidisciplinary follow-up to prevent recurrent thromboembolic events.

CONCLUSION(S)

Recurrent pregnancy loss and unexplained prolongation of aPTT in the setting of young-onset ischaemic stroke should raise suspicion for APS and prompt appropriate evaluation. Mitral valvular lesions in APS, particularly suspected LSE, can serve as a cardioembolic source and contribute to recurrent infarction. Similar cases in the literature suggest that APS-associated LSE may be managed medically with anticoagulation and anti-inflammatory therapy or surgically with valve intervention if recurrent embolism or severe valvular dysfunction occurs. The present case supports early autoimmune work-up, detailed echocardiographic assessment, exclusion of infective endocarditis and multidisciplinary management in young stroke patients with suspected APS.

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