Case Report

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Left Atrial Myxoma Presenting as Acute Cardioembolic Stroke in Young Female: A Rare Case Report

Internal Medicine Section

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ABSTRACT

Left atrial myxoma is the most common primary cardiac tumour and a rare but important cause of cardioembolic stroke. This is a case report of a 22-year-old married female who presented with history of sudden-onset right-sided weakness and difficulty in speech. Brain imaging confirmed acute ischaemic stroke in the left frontoparietal and left gangliocapsular region with haemorrhagic transformation. Further evaluation with echocardiography revealed a large, mobile left atrial mass attached to the interatrial septum, consistent with atrial myxoma. Patient was started on single antiplatelet, statin with anticoagulation. Patient was referred for cardiac Magnetic Resonance Imaging (MRI) and Cardiothoracic and Vascular Surgeon opinion to higher centre. This case highlights the importance of considering cardiac sources such as left atrial myxoma in young or atypical stroke patients without conventional risk factors. Early echocardiographic evaluation can be lifesaving, enabling timely surgical intervention to prevent recurrent embolic events.

Keywords: Echocardiography, Embolisation, Papillary myxoma

CASE REPORT

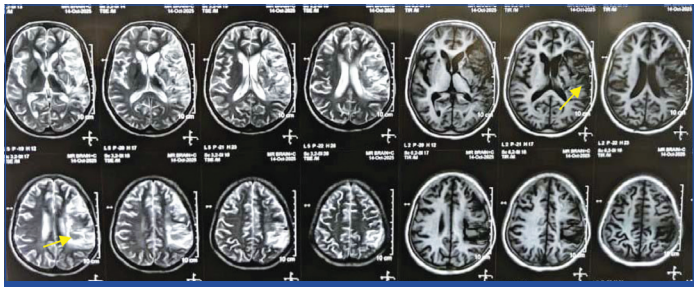
A 22-year-old married previously healthy female presented with complaints of sudden-onset weakness of the right upper and lower limbs and difficulty in speaking. The symptoms began abruptly one month prior to presentation, with no preceding history of trauma, fever, headache, seizures, or loss of consciousness. There was no documented episode of acute worsening after the initial event; however, the neurological deficits persisted without improvement despite supportive care received at a local healthcare facility which included intravenous fluids, multivitamins and limb physiotherapy, and monitoring of vital parameters. There was no history of hypertension, diabetes mellitus, rheumatic heart disease, or prior cerebrovascular events. She had no history of smoking, alcohol intake, oral contraceptive use, or illicit drug use. There was no significant family history of stroke, cardiac disease, or familial cardiac tumours.

On general examination, the patient was conscious and obeying verbal commands. She was afebrile with a pulse rate of 64 beats/min, blood pressure of 124/80 mmHg, and oxygen saturation of 99% on room air. Central nervous system examination revealed a Glasgow Coma Scale (GCS) score of E4V2M6. Pupils were bilaterally equal and reactive to light. Speech was limited to incomprehensible sounds. Motor examination demonstrated hypotonia in the right upper and lower limbs, with muscle power of 0/5 on the right side. Deep tendon reflexes were exaggerated (+3) on the right side, with an extensor plantar response on the right and flexor response on the left, suggestive of an upper motor neuron lesion. Cardiovascular, respiratory, and abdominal examinations were unremarkable.

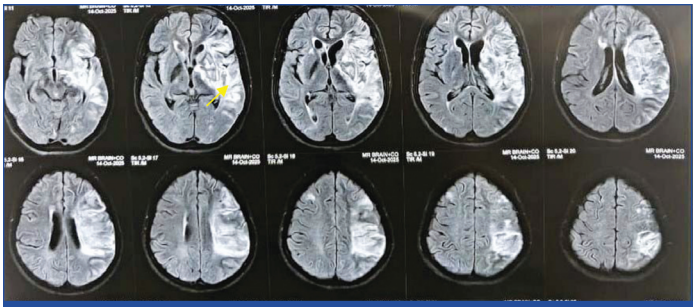
In view of the acute focal neurological deficit in a young patient, a provisional diagnosis of stroke was made. Routine laboratory investigations, including complete blood count, liver function tests, and renal function tests, were within normal limits. The thyroid profile and lipid profile were unremarkable except for LDL which was 150 mg/dL. Inflammatory markers, including Erythrocyte Sedimentation Rate (ESR) and C-Reactive Protein (CRP), were within normal limits, making active vasculitis less likely. Thrombophilia screening- including protein C, protein S, antithrombin III levels, antiphospholipid antibodies, and homocysteine levels- were within normal range. D-dimer levels were not significantly elevated. The Antinuclear Antibody test (ANA) profile was negative. Cardiac biomarkers, including troponin I, were within normal limits. Electrocardiography showed sinus bradycardia.

Neuroimaging with Computed Tomography (CT) brain and MRI brain with neck angiography revealed features consistent with an acute ischaemic infarct. In CT brain, a very large area of hypoattenuation was seen involving the cortical and subcortical aspect of the left parietal-temporal lobe with contiguous involvement of the gangliocapsular and thalamocapsular structures. Significant mass effect was seen in terms of effacement of the lateral ventricle with midline shift of at least 10 mm toward the right. MRI brain plain and contrast showed altered signal intensity area appearing hyperintense on T2-weighted Fluid-Attenuated Inversion Recovery (T2/FLAIR) was noted in the left frontotemporoparietal region and left gangliocapsular region, with few areas showing diffusion restriction (in peri-rolandic area and frontoparietal region) and few areas (gangliocapsular region central gyrus, and frontal regions) showing blooming on SWI, s/o subacute infarct with haemorrhagic transformation [Table/Fig-1,2]. In MRI brain head and neck angiography, common carotid arteries, both internal carotid arteries, middle cerebral arteries, posterior cerebral arteries, the basilar artery and both posterior cerebral arteries were normal. No evidence of aneurysm was noted.

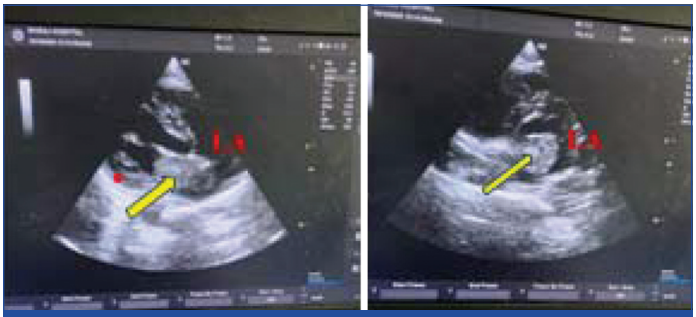
Two-dimensional echocardiography [Table/Fig-3] was performed as part of the aetiological workup for young stroke. Transthoracic echocardiography was suggestive of a well-defined, mobile echogenic mass measuring 3.1×2.1 cm is noted within the left atrium. The mass was attached by a stalk to the interatrial septum, likely arising from the region of the fossa ovalis. The lesion is



[Table/Fig-1]: MRI Brain plain and with contrast. Left part: Axial T2-weighted image demonstrating hyperintense signal in the left frontotemporoparietal region and left gangliocapsular region, consistent with oedema and ischaemic injury. Right part: Axial T1-weighted image showing hypointensity in the left frontotemporoparietal and gangliocapsular region corresponding to the infarcted territory, with focal areas of intrinsic T1 hyperintensity in the gangliocapsular region, central gyrus, and frontal regions representing methaemoglobin in subacute haemorrhagic transformation.



[Table/Fig-2]: MRI Brain Axial FLAIR image showing persistent hyperintensity in the left frontotemporoparietal and gangliocapsular regions, confirming the subacute nature of the infarct with suppression of normal CSF signal.



[Table/Fig-3]: Transthoracic echocardiography- Parasternal long-axis view showing a well-defined echogenic mass (3.1×2.1 cm) in the left atrium attached to the interatrial septum at the fossa ovalis.

protruding into the left atrial cavity, suggestive of atrial myxoma. Left ventricular size and systolic function are preserved. Left Ventricular Ejection Fraction (LVEF) is 55%, which is at the lower end of the normal range. No regional wall motion abnormalities are observed. The interatrial and interventricular septa appeared structurally intact on transthoracic imaging. No obvious Patent Foramen Ovale (PFO) was detected on colour doppler assessment. No intracardiac thrombus or vegetations were seen. No pericardial effusion was present. The left atrial appendage was visualised without evidence of thrombus; however, Transesophageal Echocardiography (TEE) was recommended for more detailed evaluation, if clinically indicated. Valvular assessment revealed mild mitral regurgitation. No evidence of aortic regurgitation, tricuspid regurgitation, or pulmonary artery hypertension was noted.

The initial differential diagnoses considered included cardioembolic stroke secondary to atrial myxoma, hypercoagulable states, and vasculitis. Thrombophilia screening, including protein C, protein S, antithrombin III levels, antiphospholipid antibody panel (anticardiolipin antibodies and lupus anticoagulant), and serum homocysteine levels, were all within the normal range. D-dimer levels were not significantly elevated. Antinuclear Antibody (ANA) profile was negative, effectively excluding an underlying autoimmune or connective tissue disorder as a contributory aetiology. Inflammatory markers including ESR and CRP were within normal limits, making active vasculitis unlikely. In the context of echocardiographic evidence of a mobile intracardiac mass with stalk attachment to the interatrial septum, and in the absence of any laboratory evidence supporting a primary hypercoagulable state, autoimmune vasculitis, or systemic inflammatory process, a cardioembolic aetiology secondary to left atrial myxoma was considered the most probable underlying cause of the ischaemic stroke with haemorrhagic transformation

The investigations confirmed a diagnosis of left atrial myxoma as the source of cardioembolic ischaemic stroke with haemorrhagic transformation in a young female patient with no prior co-morbid illness. The patient was managed with single antiplatelet therapy and statins, along with supportive neurorehabilitation. She was referred for cardiothoracic surgical evaluation for definitive excision of the cardiac mass.

An ophthalmology consultation was obtained to assess for ocular embolic manifestations and raised intracranial pressure. Fundoscopic examination revealed features suggestive of early papilloedema, consistent with increased intracranial pressure secondary to the acute cerebral infarction. The patient was started on intravenous mannitol (20% solution, 100 mL; 0.5 g/kg) over 20 minutes thrice daily and intravenous dexamethasone 8 mg thrice daily for cerebral oedema management. As per cardiology advice, intravenous unfractionated heparin 5,000 units four times daily was initiated to prevent further embolic events, in addition to single antiplatelet therapy, statins, and a single antiepileptic drug for seizure prophylaxis. She was managed in the critical care unit and, after clinical stabilisation, was referred to the higher centre for Cardiac MRI and Cardiothoracic and Vascular Surgery (CTVS) team for surgical excision of the left atrial myxoma. On follow-up, gradual neurological improvement was observed with physiotherapy, and definitive surgical management was planned to prevent recurrent embolic complications.

DISCUSSION

Cardiac myxoma is a rare primary cardiac tumour with a marked female preponderance, showing a female-to-male ratio of approximately 3:1. Most cases are sporadic, while familial forms are relatively uncommon [1,2]. Myxomas may arise in any cardiac chamber; however, the left atrium is the most frequently involved site, typically originating from the interatrial septum near the fossa ovalis [3]. Morphologically, cardiac myxomas are classified into polypoid and papillary types. Polypoid myxomas are more often associated with obstructive cardiac symptoms, whereas papillary myxomas are friable and carry a higher risk of systemic embolisation [4]. Clinically, cardiac myxomas classically present with a triad of constitutional symptoms, obstructive cardiac manifestations, and embolic events. Although histologically benign, if left untreated they are inexorably progressive and potentially fatal [5].

Approximately, 30-40% of patients with left atrial myxoma present with cerebral embolism, though clinical manifestations vary widely. Presentations may include transient ischaemic attacks, large territorial infarctions, retinal artery occlusion, peripheral arterial embolism, or recurrent embolic events. Constitutional symptoms such as fever, weight loss, or elevated inflammatory markers may occur due to cytokine release, particularly interleukin-6. Some patients present with features mimicking mitral stenosis due to obstruction of the mitral valve orifice. In the present case, the clinical presentation was predominantly neurological, without constitutional or obstructive cardiac symptoms [6].

According to the American Heart Association and American Stroke Association (AHA/ASA) stroke guidelines, young patients presenting with ischaemic stroke should undergo comprehensive evaluation to identify uncommon but treatable causes. This includes detailed vascular imaging, cardiac evaluation with echocardiography, prolonged cardiac rhythm monitoring when indicated, and selective thrombophilia screening. Early echocardiographic assessment-preferably TEE when a cardioembolic source is suspected-is recommended to detect structural cardiac abnormalities such as atrial myxoma, left atrial appendage thrombus, or PFO. Prompt recognition of a cardiac source is crucial, as definitive surgical excision significantly reduces the risk of recurrent embolic events and improves long-term prognosis [7].

Cardiac myxoma remains an important though rare cause of embolic stroke, particularly in young patients without conventional vascular risk factors [8]. Left atrial myxoma may result in cerebral ischaemic stroke, cerebral aneurysm formation with secondary haemorrhage, seizures, retinal embolic phenomena, and recurrent systemic embolisation [9]. Given that cerebral embolism is the most frequent presenting feature in up to 30-40% of cases, early cardiac evaluation with echocardiography should be strongly considered in all young patients with cryptogenic stroke to facilitate timely diagnosis and potentially curative surgical intervention [6,10].

In a case reported by Sabri M et al., a 30-year-old woman with hypertension and prior i.v. drug use presented with dizziness and limb numbness in both upper limb and lower limbs. Brain MRI revealed multiple embolic ischaemic strokes in left cerebellum, left occipital lobe, right temporal lobe, and bilateral frontal lobes, without evidence of haemorrhage. Although endocarditis was initially suspected, blood cultures were negative. Transthoracic echocardiography showed a large mobile left atrial mass attached to the interatrial septum. Surgical resection confirmed the diagnosis of a cardiac myxoma [1]. In a case report by Misra N and Prasad T, a 62-year-old woman presented with acute right arm and leg weakness of one-day duration. Initial CT brain and CT angiography were unremarkable, but brain MRI revealed multiple subacute infarcts in different vascular territories consistent with an embolic aetiology. Transthoracic echocardiography identified a 2×5 cm left atrial myxoma prolapsing into the left ventricle. She underwent successful surgical resection with left atrial appendage clipping and closure of an incidental PFO. Her postoperative course was complicated by transient atrial flutter, after which she recovered fully and remains asymptomatic at one-year follow-up. The patient was started on anticoagulation with apixaban and subsequently recovered completely. At discharge, she was advised to continue daily amiodarone and apixaban 2.5 mg twice daily for three months, as recommended by cardiology for thrombo prophylaxis given the presence of a left atrial appendage clip. She continues to follow with cardiology and remains clinically stable one year post-surgery [4].

In this case report, a 22-year-old female presented with subacute ischaemic stroke with haemorrhagic transformation in the absence of pre-existing co-morbidities. Transthoracic echocardiography revealed an echogenic mass within the left atrium, attached by a stalk to the interatrial septum, consistent with left atrial myxoma, thereby identifying the source of cerebral embolism. Haemorrhagic transformation was most likely secondary to a large cardioembolic infarct in the left middle cerebral artery territory as such infarcts are predisposed to reperfusion injury and vascular fragility, particularly in tumour-related emboli. Contributing risk factors included large cortical involvement and delayed presentation. Anticoagulation with heparin was initiated after haemorrhagic transformation had already been documented on neuroimaging, making it unlikely to be the primary cause, although a potential risk of haemorrhagic expansion was carefully considered. The patient was managed with single antiplatelet therapy, statin, anticoagulation to prevent recurrent embolic events, antiepileptic for seizure prophylaxis and mannitol with dexamethasone to reduce cerebral oedema. Following clinical stabilisation in the critical care setting, she was referred to the higher centre for cardiac MRI and CTVS team for definitive surgical excision of the left atrial myxoma. Cardiac MRI is useful for detailed characterisation of the tumour, including precise assessment of its size, site of attachment, mobility, tissue composition, and differentiation from thrombus or other intracardiac masses [11]. In case report by Wan Y et al., emphasises the need for surgical resection of cardiac myxoma, particularly in patients who have already experienced cerebral infarction, to prevent recurrent embolic events from tumour fragmentation. Although mechanical thrombectomy achieves high recanalisation rates, overall prognosis may remain guarded; therefore, early definitive tumour excision combined with appropriate endovascular management is crucial for optimal outcomes [12]. Similarly, this case underscores the importance of early recognition of cardiac myxoma as a reversible and treatable cause of stroke in young patients and highlights the crucial role of timely surgical intervention in preventing recurrent embolic complications and long-term morbidity.

CONCLUSION(S)

Evaluation of young patient with acute stroke is important from diagnostic and therapeutic point of view. Atrial myxoma, may manifest with obstructive, embolic, or constitutional symptoms. Cerebral embolisation occurs due to tumour fragmentation or thrombus detachment. Early diagnosis via echocardiography and prompt surgical intervention is crucial for preventing recurrence and improving outcomes.

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