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CASE REPORT

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Left atrial septal pouch thrombus: An unusual cause of an embolic stroke

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Key Clinical Message

Left-atrial septal pouch (LASP) is a newly described anatomical variant caused by incomplete fusion of the septa primum and secundum. This case visualizes LASP as a potential nidus for blood stasis and cardiac embolism and highlights the need for consideration of TEE in the work up of cryptogenic stroke.

KEYWORDS

cerebrovascular disease, cryptogenic stroke, echocardiography, left atrial septal pouch

1 | INTRODUCTION

A large proportion of ischemic strokes have an undetermined cause and are considered "cryptogenic".[1] A left atrial septal pouch (LASP) develops due to incomplete fusion of the septa primum and secundum, forming a pocket at the interatrial septum which can be a potential site of blood stasis with resultant thrombus formation.[2] LASP has a higher prevalence among patients who present with cryptogenic stroke, but it is rare to visualize a thrombus in the LASP.[3] We present a case of stroke with visualization of a LASP with in situ thrombus confirming a pathogenic role.

2 | CASE HISTORY AND EXAMINATION

A 60-year-old female presented to the emergency department with altered mental status. She was found unresponsive at home by her family and became agitated and combative upon interaction with emergency medical services. The patient was sedated with 4 mg of lorazepam and intubated before transport to the hospital.

The patient was intubated and thus was unable to provide a history. Past medical history obtained through the electronic medical record was significant for morbid obesity, type 2 diabetes mellitus, coronary artery disease, hypertension, heart failure with preserved ejection fraction, invasive ductal carcinoma of the breast, and a metastatic neuroendocrine tumor (NET). Past surgical history was notable for a laparoscopic cholecystectomy 14 years prior and a partial left thyroidectomy 6 years prior. The patient was retired and lived at home with her husband and grandchildren. She never smoked or used recreational drugs and had no recorded family history of heart or neurologic disease.

Initial physical examination revealed a heart rate of 62 beats/min, respiratory rate 15 breaths/min, blood pressure (BP) 183/79 mmHg, and oxygen saturation 100% on 100% FiO2. She had no labored respiratory effort and had unhindered air entry with mechanical ventilation. Her heart rate was regular without evidence of murmur. She withdrew to pain and demonstrated intact deep tendon reflexes in upper and lower extremities bilaterally. Given her breast cancer history with metastatic NET, the primary concern at this time was new metastasis to the brain. She

was promptly transferred to the medical intensive care unit for further evaluation and was placed on continuous cardiac monitoring.

A chest X-ray showed mild pulmonary edema. Computed tomography (CT) of the head showed cytotoxic edema in the bilateral middle cerebral artery (MCA) territories prompting a concern for possible acute infarcts. Magnetic resonance imaging of the brain revealed bilateral acute to early subacute MCA territory infarcts involving the cortices of the superior temporal and inferior parietal lobes and posterior insula with distribution suggestive of a central embolic source (Figure 1A). There was no evidence of intracranial metastatic disease. Electrocardiogram demonstrated sinus rhythm with regular rate without evidence of arrhythmia. The patient's serum vitamin B12, folate, thyroid stimulating hormone, and a hemoglobin A1c were within normal limits. Carotid duplex ultrasound demonstrated nonsignificant plaque at the bilateral bifurcations of the carotid arteries with no significant stenosis or dissection of either the carotid or vertebral arteries. Magnetic resonance angiogram revealed no stenosis, occlusion, or aneurysm of the major intracranial arteries. Transthoracic echocardiogram (TTE) displayed a mildly reduced left ventricular ejection fraction (LVEF) of 45%–50% with mild global hypokinesis, unchanged from previous TTE obtained 2 weeks prior. Agitated saline contrast revealed no interatrial or intrapulmonary shunt.

She was treated with aspirin 81 mg daily and atorvastatin 80 mg daily. With subsequent improvement in oxygenation and mental status, she was extubated and transferred to the stroke service for further management. Throughout her admission, she continued to maintain sinus rhythm with normal rate without evidence of arrhythmia on cardiac monitoring. She remained hypertensive with consistent systolic readings above 190 mmHg, but normal diastolic pressures. Permissive hypertension with a goal systolic BP <180 mmHg was allowed as per protocol. She was treated with lisinopril 5 mg daily that was titrated up to 40 mg to optimize BP control.

3 | METHODS

Due to a nonconclusive initial stroke workup, neurology recommended a transesophageal echocardiogram (TEE)

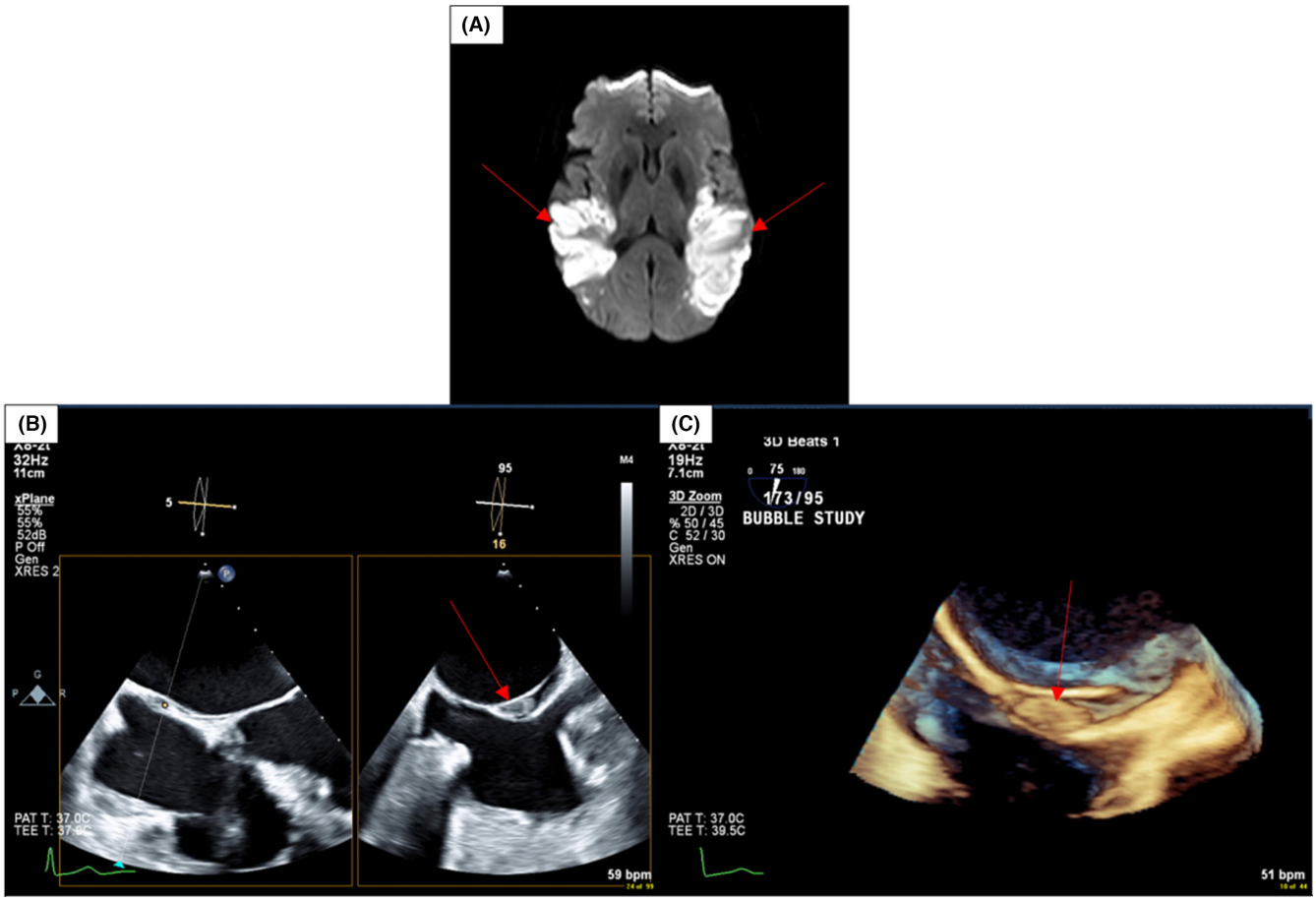

FIGURE 1 (A) DWI sequence of Brain MRI (axial view) showing acute bilateral inferior division MCA infarcts (arrows). (B) TEE Explane image of interatrial septum showing thrombus in atrial septal pouch (arrow). (C) 3D TEE image of atrial septal pouch with thrombus.

for further evaluation. TEE revealed a left atrial septal pouch (LASP) with a visualized thrombus in situ that measured 1.1 × 0.57 cm (Figure 1B,C). Apixaban 5 mg twice daily was given for anticoagulation. Once neurologically, hematologically, and hemodynamically stable, she was discharged to a nursing facility with plans for follow-up at the stroke clinic in 1 week. There are no established guidelines on anticoagulation use nor follow-up management of patients with LASP stroke. Cardiology recommended a repeat TEE in 6 weeks to evaluate the LASP and thrombus. Given that her significant cardiac and oncologic comorbidities along with her LASP thrombus put her at high risk for repeat stroke, the decision was made to maintain her apixaban 5 mg twice daily until her follow-up TEE at which time her anticoagulation regimen would be reassessed depending on her clot burden. Unlike with a left atrial appendage, there are currently no surgical interventions available for management of LASP for the prevention of cardioembolic stroke—therefore, a surgical option was not pursued.

4 | CONCLUSION AND RESULTS

She missed her neurology follow-up appointment but was seen in the cardiology clinic 6 weeks after discharge. She was appropriately managed for cardiomyopathy. Given her significant cardiac history including coronary artery disease and hypertension, a 2-week cardiac event monitor was ordered to evaluate for atrial fibrillation. She did not follow through with this and did not show for her follow-up TEE.

A brain MRI ordered at an Oncology follow-up appointment about 4 months later due to persistent confusion and aphasia ruled out metastasis of breast cancer to the brain and acute infarcts. It showed evolving, now chronic bilateral middle and posterior cerebral artery watershed territory infarcts.

5 | DISCUSSION

Over the past decade, the anatomic characteristics of LASP, as well as its ability to serve as a nidus for blood stasis and thrombus formation have been described.[2] The partial fusion of the septa primum and secundum that produces a septal pouch can protrude into either the right or left atrium. LASP is the more common anatomical variant—a systematic review that collated two cadaveric studies of 294 total healthy adult hearts reported a prevalence of 40.8%, whereas it was found that 5.1% had a right septal pouch and 3.7% of individuals had both a left and right pouch.[4] The presence of LASP has been documented in several case reports of cryptogenic stroke, but imaging confirming the presence of a thrombus has been exceedingly rare.[5–8] Our case provides rare confirmation demonstrating a LASP thrombus in a patient with bilateral cortical infarcts. To the best of our knowledge, there have only been a few published images of LASP thrombi worldwide.[9,10]

Cryptogenic strokes account for as many as 40% of all ischemic strokes.[1] While some literature has demonstrated LASP to be associated with increased risk of cryptogenic stroke,[3] other work has not.[11] However, a recent systematic review and meta-analysis of 10 retrospective observational studies demonstrated that among patients with a LASP, the aggregate odds ratio for a cryptogenic stroke was 1.618 times greater than those with no cryptogenic stroke ($p < 0.001$).[12] Currently, whether LASP can be considered a risk factor for embolic stroke is still a matter of debate. However, as physicians become aware of the possibility of LASP as an embolic source, more literature may provide insight into the true nature of LASP as a risk factor.

Evaluation of a cryptogenic stroke includes identifying possible atrial fibrillation, paradoxical embolism (atrial septal defect, patent foramen ovale), thrombophilia (malignancy-induced or genetic), atherosclerotic disease, or other vasculopathies.[13] Particularly in patients with coronary artery disease (CAD), emphasis should be placed on ruling out atrial fibrillation. The reciprocal relationship between CAD and atrial fibrillation has been well established. Their pathophysiologies have been found to accentuate one another as a risk factor for adverse cardiac events and a consistent body of evidence has found increased stroke risk among atrial fibrillation patients with CAD compared to those without CAD.[14,15] This was the case in our patient, who had a strong cardiac risk factor profile including CAD. Despite imaging showing a LASP thrombus as the likely cause of her embolic stroke, our patient was continued on continuous cardiac monitoring through the duration of her hospital stay and plans were made to pursue 2 weeks of event monitoring to further rule out atrial fibrillation at her 6 week cardiology follow-up appointment.

We recommend that in patients with a cryptogenic stroke with a negative initial stroke workup, clinicians should consider evaluating for the presence of LASP, especially if the patient has bilateral cortical infarcts, as this suggests an embolic source.[16] Since LASP may not be detected by TTE, TEE is necessary for better identification. Clinicians should also consider LASP for a patient who presents with a cryptogenic stroke and a prior history of atrial septal defect or patent foramen ovale as these atrial conditions have the potential to be misclassified as LASP on echocardiogram.

AUTHOR CONTRIBUTIONS

Aditya Bhave: Conceptualization; investigation; methodology; project administration; writing – original draft. **Aditya Patel:** Conceptualization; investigation; writing – review and editing. **Raag Patel:** Conceptualization; methodology; writing – review and editing. **Walker Barmore:** Conceptualization; investigation; methodology; writing – review and editing. **Mithilesh Siddu:** Conceptualization; methodology; writing – review and editing. **Jeffrey Switzer:** Conceptualization; methodology; writing – review and editing. **Juliet Yirerong:** Conceptualization; investigation; methodology; project administration; writing – original draft; writing – review and editing. **Gyanendra Sharma:** Conceptualization; investigation; methodology; project administration; resources; supervision; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest to disclose.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analyzed during this study.

ETHICS STATEMENT

This is a case report utilizing anonymized patient information and so was classified as exempt from review from the Institutional Review Board.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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