

Septal Thrombus in the Left Atrium: Is the Left Atrial Septal Pouch the Culprit?

Swaminatha V. Gurudevan, MD,* Hetan Shah, MD,† Kirsten Tolstrup, MD,* Robert Siegel, MD,* Subramaniam C. Krishnan, MD‡

CARDIOEMBOLIC STROKES ARE RESPONSIBLE for more than 20% of all ischemic strokes. The examination of atrial sources of cardioembolic strokes has focused almost exclusively on the left atrial appendage (LAA) and the pathophysiology of thrombus formation at this site is well understood. However, especially in rheumatic heart disease, thrombi can occur in the left atrium (LA) outside the appendage a significant proportion of the time with poor understanding of the underlying mechanism. We present 3 patients in whom a thrombus was seen adherent to the septal LA. The probable underlying pathophysiology and the relationship to a recently described novel atrial structure, the left atrial septal pouch (LASP) (Fig. 1) that has the potential to be a site of stasis with thromboembolic complications, is discussed (1).

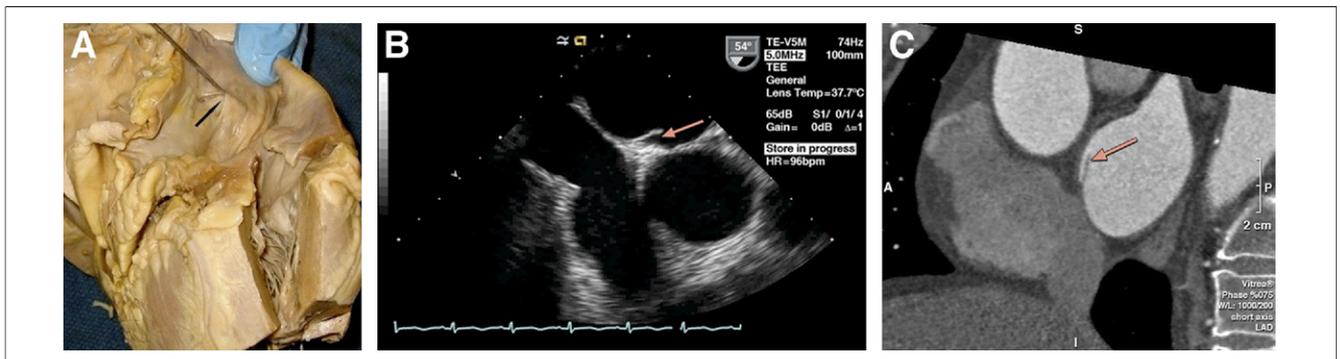


Figure 1. Examples of LASP

(A) Autopsy example of the left atrial septal pouch (LASP). A probing rod is inserted into the blind pouch that opens into the left atrial cavity. In our autopsy study, a LASP was seen in 37 of 94 hearts examined, with an average depth of 8.3 ± 3.5 mm (1). Subsequent studies with transesophageal echocardiograms (B) and computed tomography angiograms (C) revealed the LASP to be present in about 30% to 35% of individuals with a depth similar to the autopsy study. The pouch is felt to represent an incomplete closure of the foramen ovale where fusion between the septum primum and secundum is limited to the caudal portion of the zone of overlap.

From the *Cedars Sinai Hospital, Los Angeles, California; †Lokmanya Tilak Municipal General Hospital and Lokmanya Tilak Municipal Medical College, Mumbai, India; and the ‡University of California Irvine Medical Center, Orange, California. Dr. Krishnan is the owner of intellectual property related to closure of PFOs and to closure of the left atrial septal pouch and has commercialized intellectual property on transseptal puncture technology to St. Jude Medical. All other authors have reported that they have no relationships to disclose.

Address for correspondence: Dr. Subramaniam C. Krishnan, Arrhythmia Services, University of California Irvine Medical Center, 101 The City Drive, Orange, California 92868. *E-mail:* krishnan@uci.edu.



Figure 2. A 62-Year-Old Woman With End-Stage Idiopathic Cardiomyopathy Underwent Orthotopic Heart Transplantation With Cavocaval Anastomosis

The patient subsequently developed a lower extremity deep venous thrombosis that was treated with a 3-month course of warfarin. She presented again for exertional dyspnea and flank discomfort. A transthoracic echocardiogram (TTE) revealed normal ventricular systolic function, mildly enlarged left atrium, and a mass attached to the interatrial septum. A transesophageal echocardiogram confirmed the septal attachment of the mass with normal atrial blood flow. A LASP is seen (arrow) along the zone of overlap, and is possibly the site of origin and attachment of the mass. Due to the concern for a myxoma, the patient was referred for surgery which revealed a globular mass measuring 4 cm in its greatest dimension loosely attached to the septum with a 1.5 × 0.7 cm attachment site. The mass was excised with a part of the septum that was reconstructed surgically. Histopathology confirmed that it was a thrombus with signs of chronic inflammation at the site of the septal attachment. Abbreviation as in Figure 1.

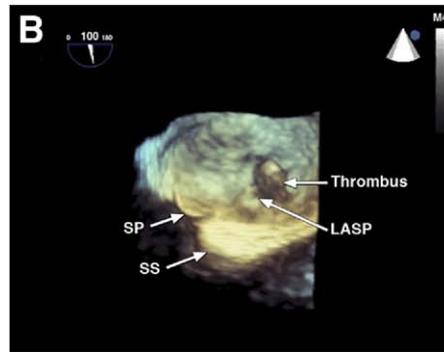
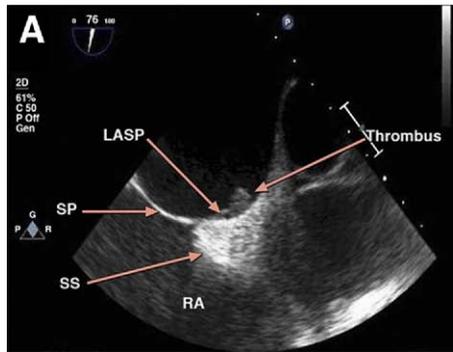


Figure 3. A 71-Year-Old African American Man With a History of Hypertension, Diabetes, and Smoking Presented with Heart Failure

The patient's electrocardiogram showed atrial flutter with rapid ventricular response and an anterior infarction of undetermined age. A TTE showed an ejection fraction of 20% with an anteroapical aneurysm and a pharmacological stress nuclear study revealed a fixed anteroapical defect. A transesophageal echocardiogram (TEE) showed dense spontaneous echo contrast (SEC) in the left atrium (LA) with a thrombus in the LA appendage and a 6-mm by 9-mm thrombus attached to the fossa ovalis (2-dimensional TEE images [A] and 3-dimensional TEE image [B] of the interatrial septum including the septum primum [SP] and septum secundum [SS]). A clear LASP is seen opening into the LA cavity (arrow) and a thrombus is seen, with the site of attachment corresponding to the LASP. A CT scan of the brain showed a large posterior infarct. Multiple medications were needed to control the ventricular rate and the patient was discharged on anticoagulation. RA = right atrium; other abbreviation as in Figure 1.

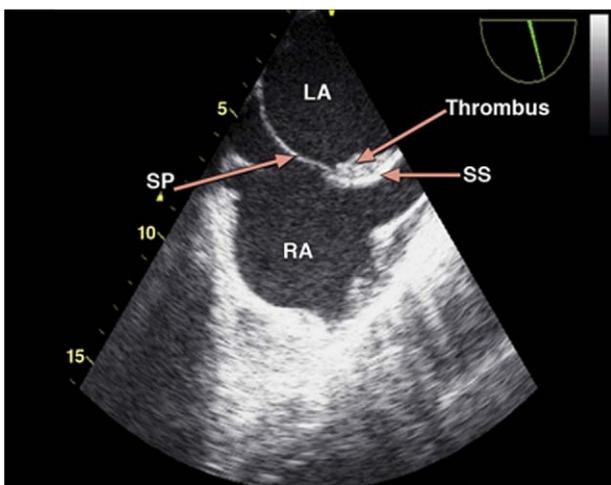


Figure 4. A 43-Year-Old Man Presented With Progressive Dyspnea and Angina

A TTE revealed severe aortic stenosis and mild left ventricular systolic dysfunction (ejection fraction of 45%). Coronary angiography revealed normal sinus. His rhythm was normal sinus. A preoperative TEE revealed a large sessile 7 × 5 × 3 cm LA mass adherent to the septum. Dense SEC indicative of slow LA blood flow was seen perhaps as a consequence of high ventricular filling pressures from the aortic stenosis, ventricular hypertrophy and diastolic dysfunction. Surgery revealed a layered thrombus loosely adherent to the septum. The thrombus was extracted and the calcified aortic valve was replaced. The bicaval view shows a left atrial thrombus adherent to the interatrial septum that appears to arise from the junction between the SP and SS, likely from a septal pouch. A pouch is not well visualized, likely due to its being filled with a thrombus. Abbreviations as in Figures 1 and 2.

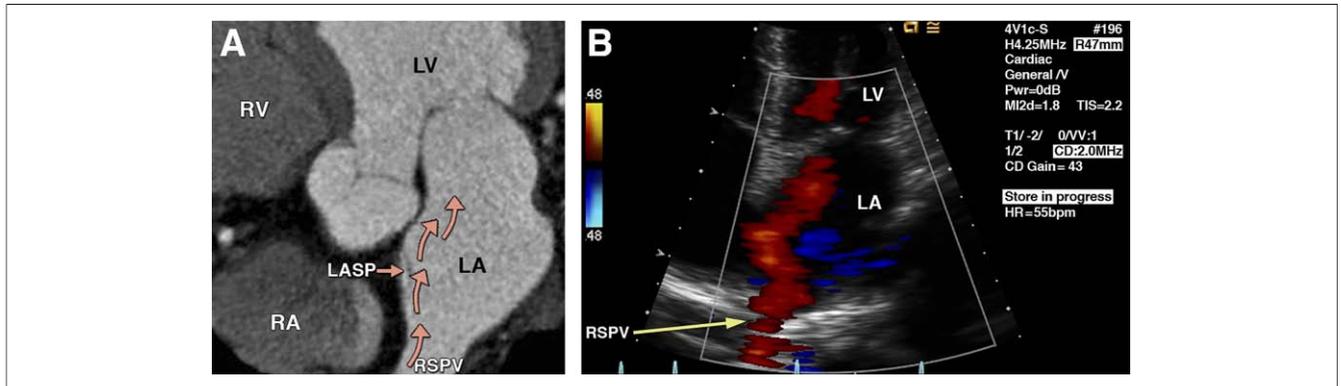


Figure 5. Proposed Underlying Mechanism

Angiographic computed tomography scan showing a modified 4-chamber view and a LASP (A, white arrow) and color flow in a TTE (B) illustrating the relationship of right pulmonary venous blood flow to the septum and septal pouch. Normal left and right pulmonary venous flow have separate paths. Left pulmonary venous blood circulates in a vortex before moving into the left ventricle (LV). In contrast, the right pulmonary venous blood flows in a laminar fashion, sandwiched between the vortex and the interatrial septum (2). Blood from the right pulmonary veins thus, flows briskly immediately adjacent to the septal pouch, and this could possibly protect against clot formation. In the presence of mitral stenosis (where non-left atrial appendage [LAA] clots are common) or high ventricular pressure filling pressures, i.e., in heart failure due to systolic or diastolic dysfunction (as in the patients described in Figures 3 and 4) right pulmonary venous flow is likely to be sluggish and consequently, stasis could occur in the septal pouch with subsequent thrombosis. The spontaneous echo contrast (SEC) seen in the patients illustrated in Figures 3 and 4 could be consistent with this phenomenon. In this situation, even a shallow septal pouch can be a source of thromboembolism. LASP, while not uncommon in imaging and autopsy studies, has not been thought of as a major nidus for a clot. The proposed hypothesis could be one reason for the low rate of occurrence of a septal thrombus. RSPV = right superior pulmonary vein; other abbreviations as in Figures 3 and 4.

Discussion

Seen in one-third of individuals, in addition to the appendage, LASP represents another LA structure that is a potential location for stasis and thromboembolism. We have presented 3 clinical examples, with a thrombus attached to the septal LA. Two patients (Figs. 2 and 3) had clear echocardiographic evidence of a LASP. The patient in Figure 4 had a thrombus with the site of attachment at the junction of the septum primum and septum secundum, where the LASP would normally be present. Due to its anatomical features, the LASP serves as a site of stasis and thrombus formation. The classic Virchow triad necessary for thrombogenesis consists of: 1) circulatory stasis; 2) endothelial injury; and 3) hypercoagulability. Of these, circulatory stasis is considered the most common trigger. In the setting of nonvalvular atrial fibrillation, stasis is felt to occur largely in the LAA and an LA thrombus is present in the LA appendage 90% of the time. However, in the presence of rheumatic mitral stenosis, an LA thrombus occurs in the appendage only one-half of the time. In the remaining, it occurs elsewhere in the LA, and LASP may comprise auxiliary site of thrombus formation especially mitral stenosis (even in sinus rhythm) or with elevated left ventricular filling pressures in heart failure. In the patient presented in Figure 2, while intrapouch stasis cannot be excluded, a thrombus occurred likely due to a combination of a hypercoagulable state and endothelial injury due to inflammation in the region of the LASP. Transesophageal echocardiography in this patient did not reveal spontaneous echo contrast (SEC) in the LA. With normal filling pressures and in the absence of mitral stenosis, right pulmonary venous blood would be expected to flow briskly through the LA (Fig. 5). In the example presented in Figure 3, dense SEC was seen due to a combination of absent atrial contractions from atrial fibrillation and likely high filling pressure evidenced by poor systolic function and elevated B-type natriuretic peptide levels. In the patient presented in Figure 4, the SEC is a consequence of high filling pressure from diastolic dysfunction. We show the complexity of thrombus formation in the LA, the cardiac chamber that is the most frequent source of cardioembolic strokes.

REFERENCES

- Krishnan SC, Salazar M. Septal pouch in the left atrium. A new anatomical entity with potential for embolic complications. *J Am Coll Cardiol Intv* 2010;3:98–104.
- Fyrenius A, Wigstrom L, Ebbers T, Karlsson M, Engwall J, Bolger AF. Three dimensional flow in the human left atrium. *Heart* 2001;86:448–55.