Left Atrial Appendage Membrane in a Patient Presenting with Stroke

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INTRODUCTION

We present the case of a patient who presented with embolic stroke and was diagnosed with left atrial (LA) myxoma for which he underwent surgical excision. At the time of surgery, intraoperative transesophageal echocardiography (TEE) identified an abnormality of the LA appendage (LAA), which led to a change in the surgical plan.

CASE PRESENTATION

Our patient was a 47-year-old man with no known medical history who presented with acute-onset right-sided hemiparesis, right-sided facial droop, and aphasia. He was diagnosed with embolic stroke on computed tomographic angiography and underwent intravenous tissue plasminogen activator and subsequent percutaneous clot extraction from the left middle cerebral artery with interventional radiology and neurosurgery. On further workup for embolic source, transthoracic echocardiography showed a large LA myxoma (5×3 cm).

Ten days later, the patient underwent removal of the LA myxoma via right anterolateral thoracotomy. TEE was performed after induction of general anesthesia. On TEE, a large mobile myxoma was visualized in the left atrium. The myxoma seemed to be attached to the interatrial septum, was 4.6×3.6 cm, and was associated with peak and mean gradients of 8 and 4 mm Hg, respectively, during left ventricular filling (Figure 1, Video 1). During further interrogation of the left atrium, partial obstruction of the LAA opening into the left atrium was noted by what seemed to be a membrane causing turbulent blood flow between the left atrium and the LAA (Figure 2, Video 2).

After institution of cardiopulmonary bypass, ascending aorta cross-clamping and administration of cardioplegia, the left atrium was opened, and the tumor was seen attached to the interatrial septum. The tumor was excised with its stalk intact, and the interatrial septal defect was closed with a pericardial patch (Figure 3A). The LAA opening was noted to be narrowed to 1.5 cm in length and a few millimeters wide because of a partial fibrotic membrane. Endocardial fibrosis was also noted in the neighboring LA walls. The fibrotic LAA was oversewn with Prolene sutures (Figure 3B). After separation from cardiopulmonary bypass, TEE showed trace mitral regurgitation and transmitral flow with diastolic peak and mean gradients of 2 and 1 mm Hg, respectively. No residual LA tumor was observed, and the LAA orifice appeared closed by two-dimensional and color flow Doppler imaging.

Postoperatively, the patient recovered well and was discharged to an acute rehabilitation center on postoperative day 6 and subsequently discharged home on postoperative day 13. Approximately 4 months after surgery, his neurologic symptoms had improved to a mild right facial droop and mild expressive aphasia, without lateralizing weakness.

DISCUSSION

Left-sided cardiac myxoma is a known cause of cardioembolic stroke, which may be the presenting symptom in 20%–40% of patients.[1,2] Cardiac myxomas are the most common primary cardiac tumor in adults, occurring predominantly in the third to sixth decades of life, with an incidence up to 0.19%.[2,3] Up to 90% of cardiac myxomas are located in the left atrium and present with congestive heart failure or cerebral and/or systemic embolic symptoms.

The LAA is also a major source of cardioembolic stroke, particularly in the setting of atrial fibrillation. In atrial fibrillation, lower flow velocities found in the LAA are associated with thrombus formation and stroke.[4,5] There are four morphologic LAA subtypes (chicken wing, cactus, windsock, and cauliflower), on the basis of three-dimensional reconstruction of the LAA. The orifice size of the LAA is significantly related to thrombus formation, with larger orifices associated with transient ischemic attacks and stroke.[4,5] All morphologies have a different orifice size, with the chicken-wing morphology having the smallest orifice and the least association with stroke.[4] The association with orifice size and morphologic subtype to incidence of thromboembolic events is likely jointly related to the lower flow velocities found in the large orifice sizes and non-chicken-wing subtypes.[4]

LAA membrane has been previously described as a rare abnormality of the LAA in case reports, thought to be congenital in nature.[6-8] These membranes can be obstructive, if at the orifice of the LAA, or nonobstructive, if within the body of the LAA. The clinical significance of LAA membranes is unknown, as clot formation may be increased because of the lower flow velocity within the LAA or, conversely, may be decreased because of the small orifice of the LAA.

In our case, the patient presented with embolic stroke, and subsequent imaging found a cardiac myxoma, thought to be the source of emboli. During intraoperative TEE, the LAA was noted to be partially obstructed by a membrane. The membrane occluding the orifice of

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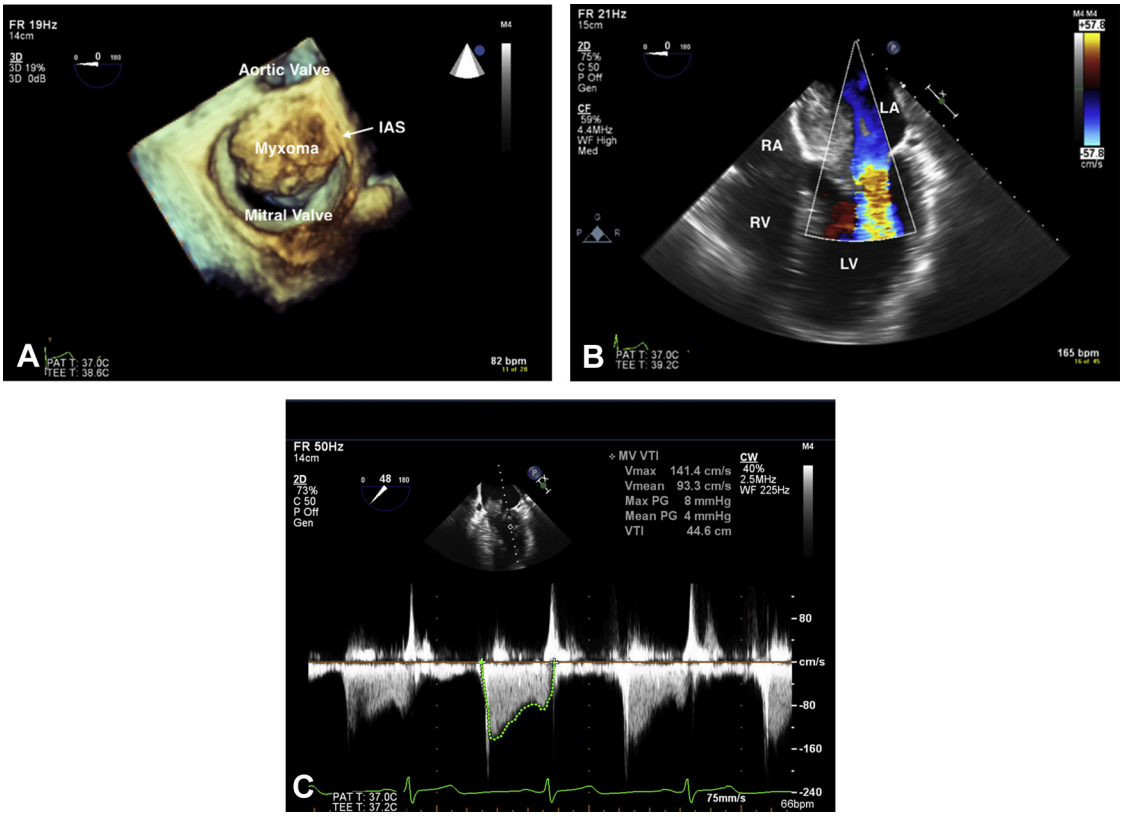

Figure 1 **(A)** Three-dimensional en-face view of the left atrium (LA) myxoma, as seen from the LA. **(B)** Midesophageal four-chamber view with color flow Doppler. The LA myxoma is seen partially obstructing the left ventricular (LV) inflow. **(C)** Continuous-wave Doppler across the mitral valve. *IAS*, Interatrial septum; *RA*, right atrium; *RV*, right ventricle.

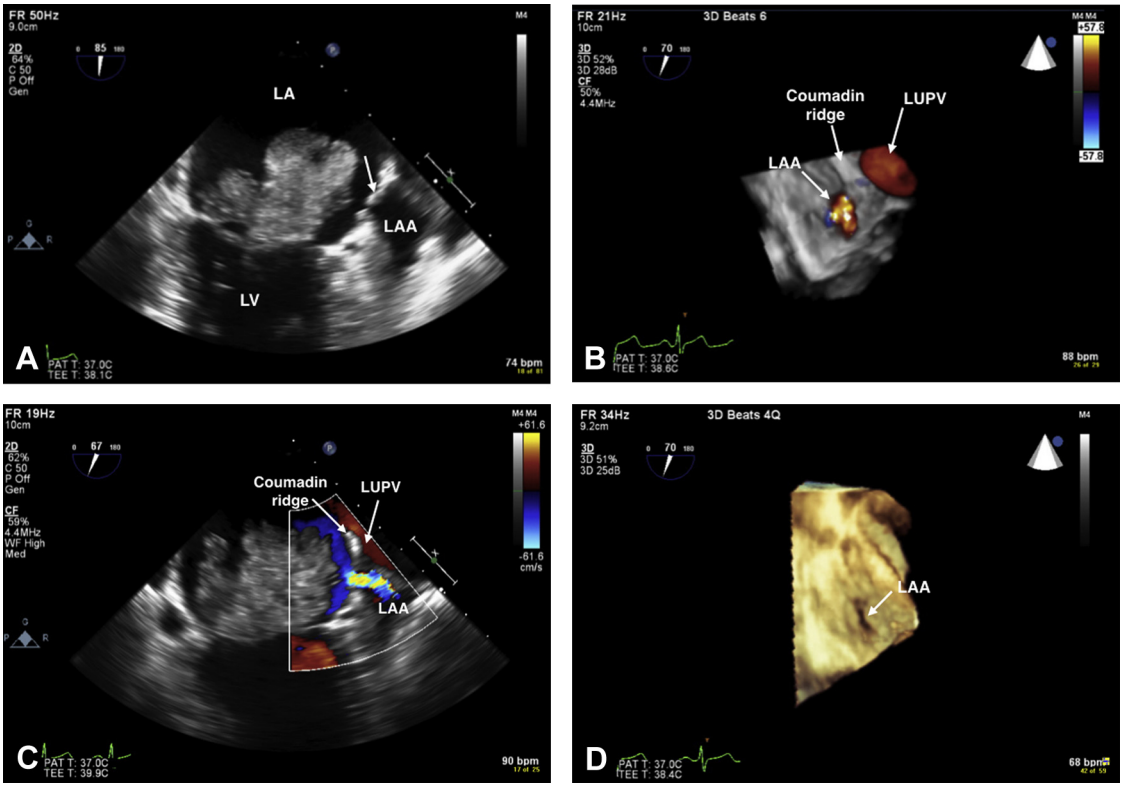

Figure 2 **(A)** Midesophageal two-chamber view focused on the left atrial appendage (LAA) and mitral valve. The myxoma is seen in close proximity with the LAA. A partially occluding membrane (*arrow*) is seen at the opening of the LAA in the left atrium (LA). **(B)** Three-dimensional data set with color flow Doppler showing en-face the opening of the LAA with turbulent blood flow. **(C)** Midesophageal mitral commissural view focused on the LAA and mitral valve. **(D)** Three-dimensional en-face view showing a very narrow opening of the LAA. *LUPV*, Left upper pulmonary vein; *LV*, left ventricle.

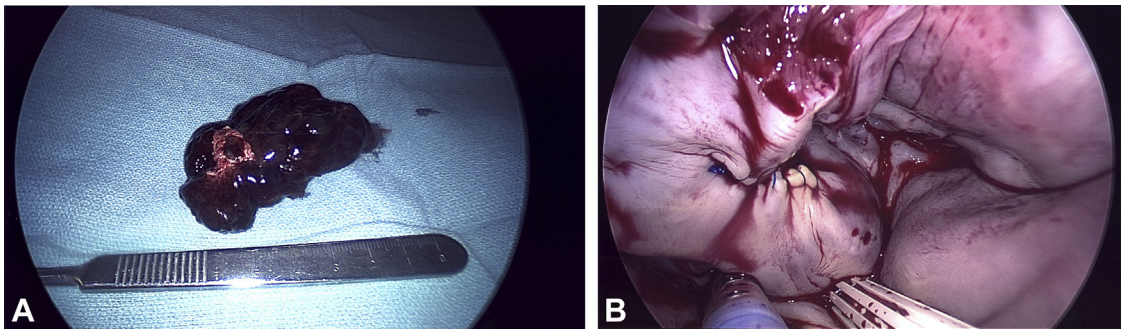

Figure 3 **(A)** Surgical specimen. **(B)** View of the opened left atrium showing the surgical closure of the left atrial appendage opening.

the LAA in our patient was thought to be acquired from fibrotic changes caused by the prolonged friction between the myxoma and the LA walls, which induced endocardial fibrosis. The clinical significance of this nonoccluding membrane and propensity for thrombus formation of the LAA in the presence of this membrane was unknown. The more likely source of embolus in our case was the myxoma, but the possibility of thrombus formation in an LAA that does not empty efficiently cannot be excluded. Surgical exclusion of the LAA did not add significant procedural time, as the left atrium had already been opened for the excision of the LA myxoma.

Our case also highlights the importance of performing complete intraoperative TEE, on the basis of an imaging protocol, which ensures consistent imaging of all structures, whether involved in the current disease process or not. Comprehensive intraoperative TEE, which included the diligent interrogation of the LAA by two-dimensional, three-dimensional, and color flow Doppler imaging, allowed us to diagnose the partial occlusion of the LAA and to direct the surgeon into performing surgical exclusion, which could be easily performed during LA myxoma resection. The addition of this procedure eliminated the partially occluded LAA as a potential source of emboli.

CONCLUSION

LAA membranes are a rare abnormality of the LAA and may be a potential source of cardioembolic stroke. Comprehensive intraoperative TEE is paramount in excluding additional abnormalities that may influence surgical decision making and the outcome of the patient.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.case.2017.07.004.

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