

Left Atrial Dissection Secondary to Retrograde Coronary Sinus Cannulation During a Stanford Acute Type-A Aortic Dissection Repair: A Case Report

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Left atrial (LA) dissections are rare phenomena, often iatrogenic, caused by blood flow into a false lumen, potentially obstructing the pulmonary veins or flow into the left ventricle. Severity can range from an incidental observation or complete circulatory collapse. While LA dissections are often associated with mitral valve repair, there are 2 reported cases of LA dissections associated with retrograde cardioplegia cannula insertion through the coronary sinus. Here, we present a large LA dissection that was directly visualized and tracked to a coronary sinus injury from the retrograde cardioplegia cannula. The clinical presentation and echocardiography findings informed our subsequent management. (A&A Practice. 2022;16:e01568.)

GLOSSARY

CPB = cardiopulmonary bypass; **CS** = coronary sinus; **CSCRI** = coronary sinus catheter-related injury; **CT** = computed tomography; **EQUATOR CARE** = Enhancing the Quality and Transparency Of health Research; **HIPAA** = Health Insurance Portability and Accountability Act; **LA** = left atrial; **POD** = postoperative day; **TEE** = transesophageal echocardiography

Left atrial (LA) dissection is a rare clinical phenomenon in which blood fills a potential space between the endocardium and myocardium, most frequently as a result of endocardial insult along the posterior LA wall.¹ It can range from a benign incidental finding to complete circulatory collapse secondary to obstruction of flow from the pulmonary veins or obstruction of outflow across the mitral valve. The most common etiology of LA dissection is iatrogenic, with more than half of reported cases associated with mitral valve surgery, followed by spontaneous occurrence and secondary to radiofrequency ablation of arrhythmia foci.¹ Given the rarity of this complication, there is no evidence-based approach to managing it.

Less than 5% of all reported cases of LA dissection from 1979 to 2014 were associated with surgery involving the aorta, and only 2 reported cases to date involve retrograde cardioplegia cannulation of the coronary sinus (CS).¹⁻³ Here, we present a case of significant interest due to our direct visualization by transesophageal echocardiography (TEE) of the LA dissection point of entry at the CS, most likely caused by injury during retrograde cardioplegia

catheter cannulation during a type-A aortic dissection repair. Such visualization has only been documented once before during a mitral valve repair.³ Additionally, we describe the clinical presentation of our patient and subsequent intraoperative management decisions and postoperative course.

Written Health Insurance Portability and Accountability Act (HIPAA) authorization was obtained for this report from the patient, which adheres to the applicable Enhancing the Quality and Transparency Of health Research (EQUATOR) CARE guidelines for case reports.

CASE DESCRIPTION

A 62-year-old woman presented to the emergency room with weakness and dizziness, and on transthoracic echocardiography, was found to have pericardial tamponade. After undergoing needle drainage of pericardial sanguineous fluid, the patient had a tonic-clonic seizure and worsening shock; the patient was then intubated and placed on epinephrine and norepinephrine for blood pressure support. Additional workup with computed tomography (CT) revealed type-A aortic dissection with extension to involve the proximal transverse arch, innominate artery, right common carotid artery, right vertebral artery, and left common carotid artery. The patient was taken emergently to the operating room for surgical repair of her ascending aortic dissection.

Surgical repair of the aortic dissection was performed successfully with a hemiarach replacement with a 30-mm Dacron Gelweave branched graft and resuspension of the aortic valve during deep hypothermic circulatory arrest. Cardioplegic solution was administered via antegrade and retrograde cardioplegia technique, and CS pressures were unremarkable throughout.

Immediately before and after separation from cardiopulmonary bypass (CPB), intraoperative TEE demonstrated a

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new dissection flap along the posterior lateral wall of the left atrium, which expanded immediately after CPB. The LA dissection appeared to stabilize after correction of coagulation abnormalities with protamine sulfate (350 mg), fresh frozen plasma, cryoprecipitate, platelets, and prothrombin complex concentrate, with the final LA dissection size measuring 3 cm × 5 cm. The mean and peak pressure gradients across the narrowed LA space were 3 and 6 mm Hg, respectively. Direct communication between the dissection cavity and the CS is demonstrated in TEE images (Figures 1 and 2). The patient required norepinephrine of 7 mcg/min, epinephrine infusion of 4 mcg/min, and vasopressin at 0.1 units/min during the post-bypass period, but had stable blood pressures of 90/60 s on these infusions. The decision was made to defer surgical intervention due to the critical condition of the patient, the presence of normal pulmonary artery pressures, hemodynamic stability, and the challenging location of the CS injury.

Postoperatively, the patient's hospital course was complicated by intermittent atrial fibrillation with rapid ventricular response and a pericardial effusion requiring mediastinal exploration on postoperative day (POD) 6. TEE during the mediastinal exploration demonstrated the LA dissection size to have decreased 40% to 1.8 cm × 3.8 cm. Four pulmonary veins were visualized with patent, non-turbulent flow into the left atrium (Figure 3). Flow through the LA cavity was normal. As such, surgical correction was deferred, and the patient was discharged home on POD 19

with minimal bilateral leg weakness, believed to be secondary to deconditioning, for which she was receiving physical therapy; she continues to do well.

DISCUSSION

The incidence of coronary sinus catheter insertion-related injury (CSCRI) ranges from 0.095% to 0.6%, with the majority of reported cases referring to midsection CS external rupture or "blowouts."⁴ Similarly, the incidence of LA dissection is exceedingly rare, reported to be between 0.1% and 0.2% during cardiac surgical procedures.⁵ Notably, retrograde cannulation of the CS for administration of cardioplegia solution has been reported as a cause of LA dissection in only 2 cases,^{2,3} with only 1 published set of TEE images documenting this phenomenon.³ For this reason, our case is of interest due to the direct visualization on TEE of the LA dissection point of entry via the CS, as shown in Figures 1 and 2. Having direct visualization and being able to track the LA dissection directly to the CS gave confidence in concluding that the CS was the point of origin, despite the normal CS pressures intraoperatively.

The pathophysiology of LA dissection really depends on its etiology. Generally speaking, any tear into the LA endocardium combined with pressurized flow might be enough to precipitate an LA dissection.¹ It is easy to see how mitral valve surgery would facilitate this, wherein overly aggressive debridement around the valve could create a tear wherein pressurized flow from the left ventricle

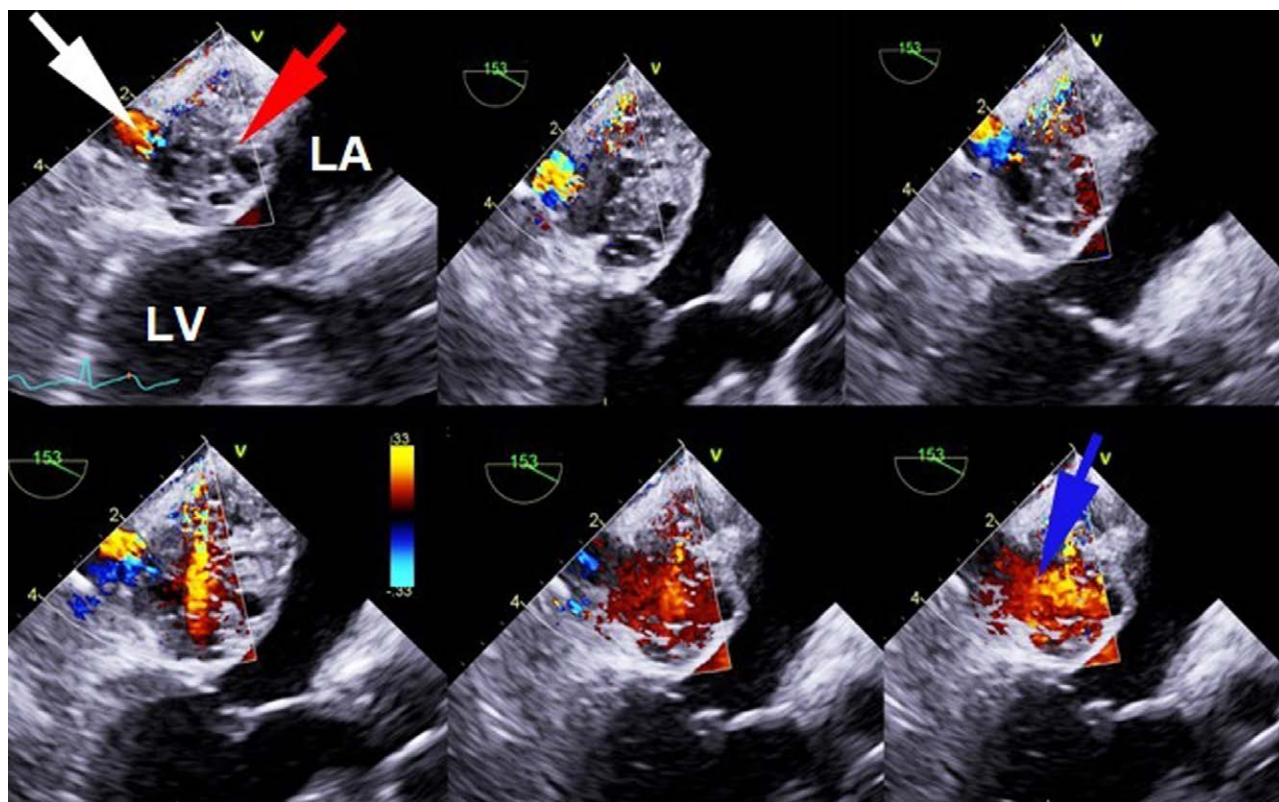


Figure 1. Midesophageal long axis view. CS (white arrow) is demonstrated to be near the LA dissection (red arrow) after the patient was weaned from CPB. LA dissection was shown to be hyperechoic, indicating that it is likely beginning to clot off. CS can be seen communicating directly with the left LA dissection cavity (blue arrow). CPB indicates cardiopulmonary bypass; CS, coronary sinus; LA, left atrial; LV, left ventricle.

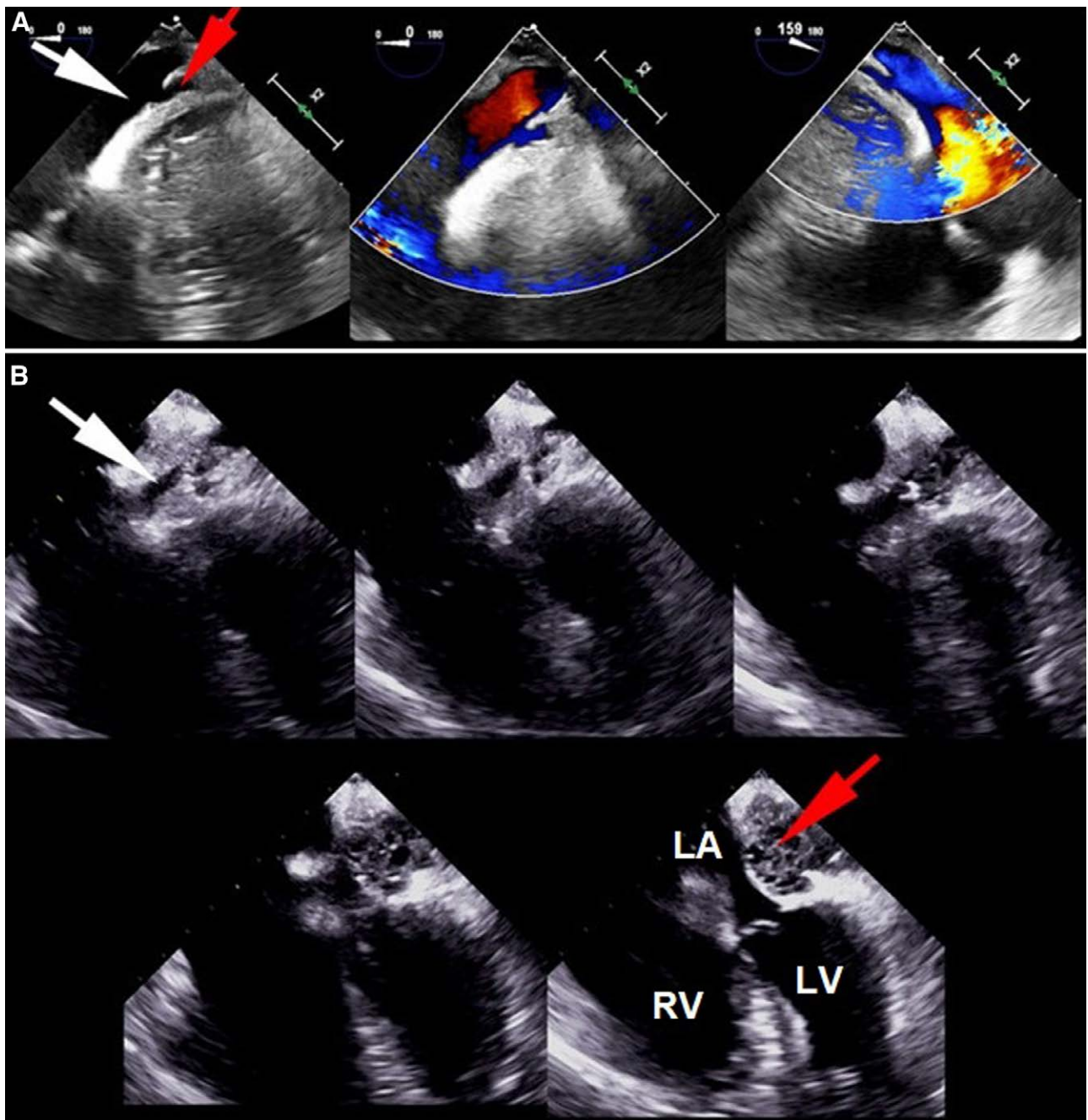


Figure 2. A, The first 2 images show lower esophageal 0° coronary sinus, and the third image is lower esophageal coronary sinus 120° with color Doppler. A tear and false lumen (red arrow) in the CS (white arrow) were directly observed. B, Top row: lower esophageal 0° CS view; bottom row: midesophageal 4-chamber view. CS (white arrow) is tracked into the LA dissection (red arrow shown to be close to the mitral valve). CS indicates coronary sinus; LA, left atrial; LV, left ventricle; RV, right ventricle.

would expand into the tear, causing a false lumen and eventual dissection. Likewise, for less invasive procedures involving catheters, an overly stiff wire could easily penetrate the LA endocardium, causing a tear vulnerable to pressurized flow and potential LA dissection. However, LA dissections need not necessarily originate from the LA endocardium. If the insult is done while catheterizing a coronary artery or during retrograde cardioplegia such as in this case, the insult would then be in the LA myocardium; and if near any source of pressurized flow, it could create a false lumen and eventual LA dissection.

The differential diagnosis of an LA dissection with hematoma should include atrial tumors such as myxoma as well as common anatomic variants of the interatrial septum, including Chiari network, eustachian valve, atrial septal aneurysm, and lipomatous hypertrophy; however, these findings are less likely since there are documented normal pre-CPB images ruling out any anatomic abnormalities. The LA dissection has a filamentous epicardium surrounding the thrombosis that can be followed to its attachment site to the heart. This is unlike a pericardiac effusion or clot, in which there is no epicardium surrounding the

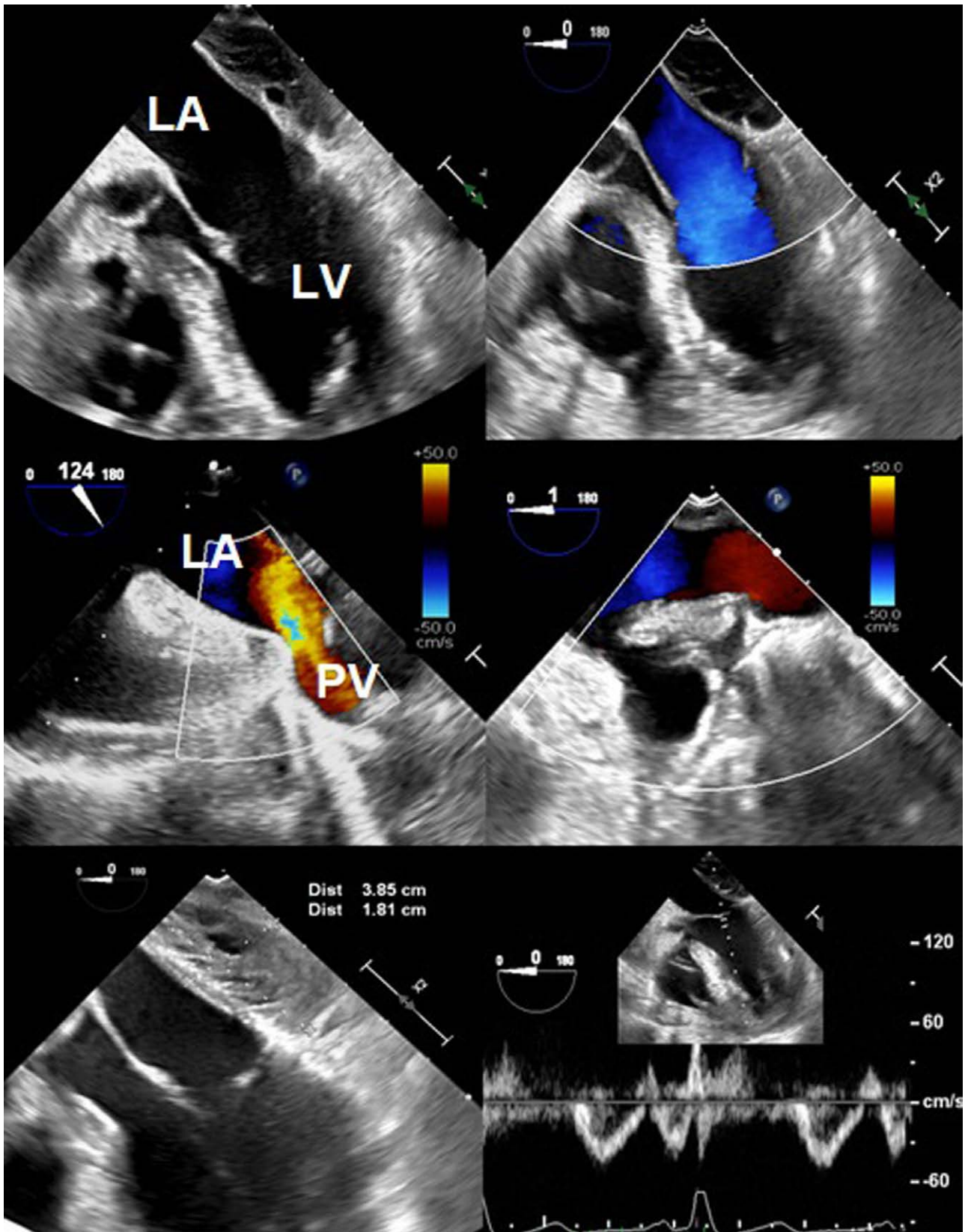


Figure 3. Top and bottom rows display the midesophageal 5-chamber view. The image on the left (middle row) is the right upper PV obtained from a right clockwise position starting at the midesophageal bicaval view. The image on the right (middle row) is a nonstandard midesophageal image of the LA appendage and the left upper PV. TEE images obtained during the second operation for mediastinal exploration on POD 6 demonstrate no obstruction of pulmonary venous flow. LA indicates left atrial; LV, left ventricle; POD, postoperative day; PV, pulmonary vein, TEE, transesophageal echocardiography.

hyperechoic mass, and it is clearly outside the walls of the heart but within the pericardium.

After LA dissection has been identified, immediate TEE imaging goals are to determine whether it might lead to hemodynamic instability. Evaluating the mitral valve area to ensure that both flow and the pulmonary veins are unobstructed should be the highest priority, as compromise of flow in either of these regions could cause low-output cardiac state or congestive heart failure and prevent weaning from CPB.¹

The management of LA dissection regardless of etiology depends on the patient's hemodynamic profile and clinical picture. Obstructive shock would clearly favor intervention, such as closing the LA dissection entry point or internal drainage.^{1,6} In the case of LA dissection secondary to CSCRI, such an approach has been described.^{3,7} However, conservative management has also been documented in a case of nonobstructive LA dissection with a hemodynamically stable patient, wherein the LA dissection ultimately resolved on serial TEEs.² As of 2015, the review of LA dissections by Fukuhara et al¹ determined that roughly 24% of LA dissections were managed with conservative measures, and 72.7% were managed with surgical repair. Due to the limited number of documented LA dissections, there is no evidenced-based approach for managing them beyond these individual reports.

The patient maintained a mean mitral gradient of 3 mm Hg and a left ventricular ejection fraction of 55% to 60% on TEE throughout the post-CPB period while on pharmacologic inotrope and blood pressure support. As such, the benefits of what would be a complex primary repair of the CSCRI and LA dissection were balanced with the critical nature of the patient, who already had suspicion of neurological injury, evidenced by tonic-clonic seizure and clinical encephalopathy during preoperative examination.

This case highlights the value of TEE imaging by an adept anesthesiologist in identifying the underlying source of LA dissection, as this may guide surgical decision-making if LA

dissection arises during cardiac surgery. As more cases are reported, we expect that a more evidence-based picture will emerge regarding when to intervene and by what means. Until then, we believe that it is prudent to document the various LA dissection presentations, etiologies, and chosen management. ■■

DISCLOSURES

Name: Christopher Halline, BA.

Contribution: This author helped draft the manuscript and assisted with edits.

Name: Andrew Winegarner, MD.

Contribution: This author helped draft the original abstract, created the figures, conducted a literature search, and edited the manuscript.

Name: Andrew Maslow, MD.

Contribution: This author helped make critical edits to the manuscript and transesophageal echocardiography images.

Name: Michelle Gorgone, MD.

Contribution: This author helped make critical revisions to the manuscript and directed discussions.

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