

Intraoperative echocardiographic contrast opacifies the left atrial appendage and assists in surgical exclusion

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ABSTRACT

Surgical exclusion of the left atrial appendage (LAA) for stroke prevention in atrial fibrillation is frequently incomplete and remains to be optimized. We present a man who did not tolerate anticoagulant and failed percutaneous occlusion. Intraoperative echocardiographic contrast was used to assist surgical exclusion. Follow-up showed a persistent occlusion.

Keywords: Atrial fibrillation, contrast agent, left atrial appendage

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Submitted: 31-Jul-2020 **Revised:** 04-Oct-2020 **Accepted:** 02-Dec-2020 **Published:** 21-Jan-2022

INTRODUCTION

Atrial fibrillation (AF) is the most common sustained arrhythmia and is associated with an increased risk of mortality and thromboembolic stroke.^[1] In non-valvular AF, approximately 90% of thromboembolisms originate from the left atrial appendage (LAA) and they are identified by transesophageal echocardiography (TEE).^[2] While stroke prevention strategies include pharmacological and mechanical LAA exclusion approaches^[3]—both percutaneous and surgical—their uses are not evenly distributed. Contraindications to anticoagulation and medication non-adherence make pharmacological approaches challenging. Surgical closure, on the other hand, has been plagued by poor outcomes^[4] and incomplete ligations.^[5,6]

CASE HISTORY

A 62-year-old man with persistent non-valvular AF with one prior cerebrovascular event but no residual neurological deficits, CHA₂DS₂-VASc score of 5, presents for LAA exclusion. The patient could not tolerate warfarin anticoagulation therapy and deploying a Watchman (Boston Scientific, Marlborough, MA) LAAO device after ablation was unsuccessful. While on warfarin therapy, HAS-BLED score of 4, the patient developed significant recurrent epistaxis and lower gastrointestinal bleeds. The international normalized ratios were consistently stable. He was admitted on several occasions for blood transfusions.

The patient's past medical history was otherwise significant for hypertension, four vessel coronary artery bypass grafting in 2014, heart failure with an ejection fraction (EF) of 51%, a permanent pacemaker for Mobitz II bradyarrhythmia, hyperlipidemia, diabetes mellitus type II, and obstructive

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10.4103/aca.ACA_192_20

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How to cite this article: Santana JM, Rosell FM, Dave B, El Manafi A. Intraoperative echocardiographic contrast opacifies the left atrial appendage and assists in surgical exclusion. *Ann Card Anaesth* 2022;25:77-80.

sleep apnea with continuous positive airway pressure therapy at bed time. Medications included metoprolol tartrate 25 mg daily, spironolactone 25 mg daily, aspirin 81 mg daily, sitagliptin 100 mg daily, torsemide 20 mg twice daily and metformin 1,000 mg every night. He was retired and independent with his activities of daily living.

A colonoscopy was performed and revealed non-bleeding colonic arteriovenous malformations. A nasopharyngoscopy was also performed but was unremarkable. Eight weeks prior to surgical occlusion, the patient presented for a Watchman LAAO. The TEE revealed that the left atrium (LA) was moderately to severely dilated and the LAA was devoid of thrombus. Four 27 mm LAAO were deployed at the ostium of the LAA, however, each time it could not be expanded because of its shallow double-lobe morphology [Figure 1a]. The case was thus aborted. Four weeks prior to surgery, a transthoracic echocardiogram revealed normal global left ventricular systolic function with an EF of 61%. The LA was severely enlarged and the estimated pulmonary artery systolic pressure was 60.3 mmHg.

On the morning of the surgery, the patient was brought into the operating room and standard American Society of Anesthesiologists monitors were applied with the addition of a pre-induction arterial line. An arterial blood gas revealed a hemoglobin and hematocrit of 7.8 g/dL and 23.5% respectfully. The patient was preoxygenated and anesthesia was induced with intravenous midazolam 2 mg, fentanyl 50 mcg, and lidocaine 2% 80 mg, followed by intravenous etomidate 16 mg. The patient was then paralyzed with intravenous rocuronium 70 mg and intubated with a bronchial blocker. Proper placement of

the bronchial blocker was confirmed with the fiberoptic scope and anesthesia was maintained with isoflurane, MAC 1.0. The surgery proceeded in the right lateral decubitus position with a left thoracotomy after satisfactory position of the bronchial blocker was confirmed.

Prior to surgical manipulation, a TEE exam was performed. The EF was estimated at 40%. There was evidence of trace mitral regurgitation with a moderately to severely enlarged LA. The LA and LAA were closely inspected for the presence of thrombi and spontaneous echocardiographic contrast in various planes from 0 to 120° [Figure 1b and c]. The LAA orifice measured approximately 25 mm [Figure 1b] and was identified as chicken-wing with double lobe morphology [Figure 1c]. Because the LAAO device could not be deployed because of its morphology [Figure 1a], Lumason®, an echocardiographic contrast agent, was administered via a peripheral intravenous access at 34 mcg/kg followed by a 10 mL of agitated normal saline flush [Figure 1d and Video 1]. We used a 50 mm AtriClip (AC) to exclude the LAA [Figure 2a]. Because the AC leaves the LAA in place, flow from the LA can traverse to the LAA if exclusion is incomplete. Fortunately, if flow is seen, the AC may be retrieved and redeployed until completely excluded. Here, using the previously described components for optimizing a TEE contrast examination,^[7] the echocontrast was instrumental in visualizing flow traversing the separation between the LAA and the LA body [Video 1]. Surgical exclusion was further supported by the lack of any anatomical structure between the mitral valve base and pulmonary artery [Figure 2b] and the absence of color Doppler jet traversing the separation between the LAA and the LA body [Figure 2c].

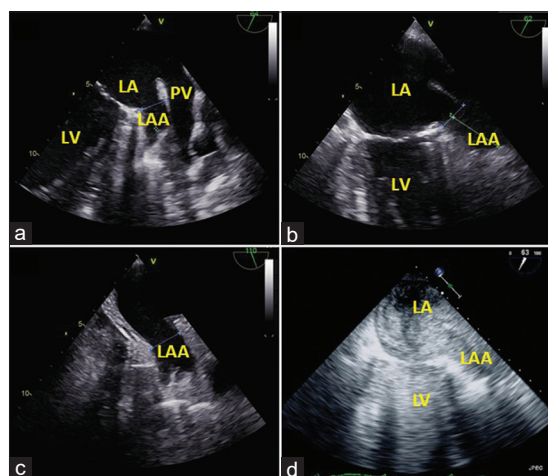


Figure 1: Transesophageal echocardiography. (a) Midesophageal 2-chamber LAA view. (b) Midesophageal mitral commissural view. (c) Midesophageal 2-chamber LAA view. (d) Contrast opacifies the LA and LAA. LA, left atrium; LAA, left atrial appendage; LV, left ventricle

The surgery lasted 4 h and 30 min. The patient received 2 units of packed red blood cells and 800 mL of 0.9% normal saline. Estimated blood loss was 50 mL and the urine output was 450 mL. The patient received intravenous acetaminophen 1,000 mg and ondansetron 4 mg and neuromuscular blockade was reversed. He was extubated in the operating room without complication. He was given oxygen 6 L/min via a simple face mask and transferred to the post-anesthesia care unit for 24-h observation.

At the 1-month follow-up, a repeat TEE was performed. The left ventricular ejection fraction was estimated to be 55–65%. The LA was severely enlarged and the AC remained in satisfactory position [Figure 3a]. Flow to-and-from the LAA was not visualized [Figure 3b]. The patient was otherwise well.

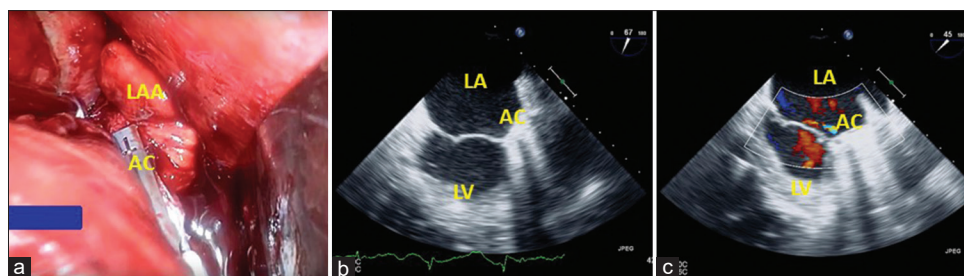


Figure 2: Peri-exclusion assessment. (a) AtriClip grappling the LAA. (b) Transesophageal view of the LAA after surgical clipping. (c) Color Doppler blood flow does not traverse the communication between the LAA and the LA body. LA, left atrium; LAA, left atrial appendage; LV, left ventricle; AC, AtriClip

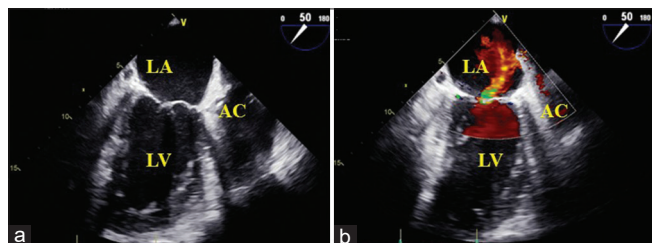


Figure 3: One-month follow-up transesophageal echocardiography. (a) Midesophageal mitral commissural view. (b) Midesophageal mitral commissural view with Doppler. LA, left atrium; LAA, left atrial appendage; LV, left ventricle; AC, AtriClip

DISCUSSION

We present the case of a patient with persistent AF who did not tolerate anticoagulation, and, after an ablation procedure, could not have a LAAO device deployed. The patient was subsequently referred for surgical exclusion, the least recommended strategy for stroke prevention. Because of the technical difficulty encountered during the LAAO procedure coupled with the unfavorable successful rates of surgical closure, the LAA was opacified and identified with an echocardiographic contrast agent to assist surgical exclusion.

Surgical exclusion of the LAA is frequently incomplete as determined by TEE^[5,6] and the incidence of incomplete ligation detected immediately postoperatively and months thereafter is similar.^[5] This information suggests that incomplete ligation results from an intraoperative phenomenon. Surgery can either excise or exclude the LAA. While excision is performed by removal of the LAA, exclusion is performed by closing the orifice into the LAA cavity with the appendage remaining attached.^[6] Success rates can be variable and have been found to be more successful in excisions than exclusions.^[6] When the appendage is incompletely ligated, the appendage cavity, stump or pouch remaining in the LAA is visualized as is the flow to-and-from the surgical site.^[5,6] Intraoperative use of echocardiographic contrast enabled us to identify the LA and the LAA to successfully exclude the LAA.

Echocardiographic contrast agents have been approved by the United States Food and Drug Administration since 1990 but remain underutilized because of its concern about possible adverse reactions^[8] which have been well refuted.^[9] They are composed of microbubbles with an outer protein or phospholipid shell that encapsulates a fluorocarbon gas which in circulation produce non-linear echos.^[10] Their indications range from suboptimal echocardiography to identifying breast, liver and urinary tract abnormalities.^[10] They have not been specifically indicated for LAA opacification in surgical scenarios and their use in this capacity is lacking in the current literature.

CONCLUSION

Echocardiographic contrast agents may be safely and confidently employed in conjunction with surgical techniques where LAA closure success rates may be low.

Acknowledgement

The authors would like to thank the assistance of Dr. Michael Youseff and the cardiothoracic perioperative staff at Staten Island University Hospital for their unwavering support in the writing and submission of the manuscript.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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