

Intraoperative Inversion of the Left Atrial Appendage Following Ligation



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INTRODUCTION

Intraoperative transesophageal echocardiogram (TEE) is integral to cardiac surgery. This real-time imaging modality allows for assessment of cardiac function and pathology, hemodynamic status, and acute perioperative changes. The intraoperative application of TEE enhances the discovery of unique findings, such as left atrial masses. Left atrial masses identified on echocardiogram have numerous etiologies, including neoplasms, vegetations, and thromboses. Less common etiologies include septal aneurysms, pulmonary vein remnants, diaphragmatic hernias, septal hematomas, and left atrial appendage (LAA) inversions.¹

We present a patient for whom intraoperative TEE allowed for identification of an acute left atrial mass that was absent on preoperative imaging. Left atrial appendage inversion was diagnosed based on the characteristics of the mass. Physician awareness of this entity is crucial to ensure accurate diagnosis and to avoid unnecessary interventions. The limited number of reported cases hinders our full understanding of this rare surgical event. We aim to improve the clinical knowledge surrounding LAA inversion by discussing the diagnosis, risk factors, management, and complications.

CASE PRESENTATION

A 61-year-old man presented with acute-onset dyspnea that was progressively worsening. Emergency medical services transported the patient to the hospital. They recorded a blood pressure up to 240/110 mm Hg and tachycardia up to 120 bpm. On admission to the emergency department, blood pressure remained elevated at 190/100 mm Hg, tachycardia persisted at 110 bpm, and hypoxemia was noted with an oxygen saturation around 80%. In addition to the severe shortness of breath, the patient reported an 8-pound weight gain over the last few days. Their past medical history was significant for atrial fibrillation (AF), hypertension, hyperlipidemia, untreated obstructive sleep apnea, and prior cardiac arrest status post-implantable cardioverter-defibrillator (ICD) placement 7 years prior. Physical examination was significant for cardiovascular and respiratory abnormalities. Cardiac auscultation revealed an irregular rhythm

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VIDEO HIGHLIGHTS

Video 1: Midesophageal two-dimensional TEE 2-chamber view with color flow Doppler showing blood flow through the LAA prior to surgery.

Video 2: Midesophageal TEE three-dimensional rendering of the MV en face. No left atrial masses are visualized prior to the surgery.

Video 3: Midesophageal two-dimensional TEE 4-chamber view revealing a new left atrial mass after ligation.

Video 4: Midesophageal two-dimensional TEE 4-chamber view with color flow Doppler revealing a nonobstructive, inverted LAA after ligation.

Video 5: Midesophageal TEE three-dimensional rendering of the MV en face with 40° of rotation showing an inverted LAA after ligation.

Video 6: Midesophageal two-dimensional TEE mitral commissural view with color flow Doppler showing the LAA beginning to spontaneously evert near the end of the case.

Video 7: Midesophageal two-dimensional TEE mitral commissural view with color flow Doppler revealing spontaneous resolution of the inverted LAA at the end of the case.

Video 8: Midesophageal TEE three-dimensional rendering of the MV en face after spontaneous resolution of the inverted LAA.

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consistent with AF. Respiratory auscultation revealed bilateral coarse crackles, scattered wheezing, increased respiratory effort, tachypnea, and use of accessory muscles. Chest x-ray confirmed bilateral pulmonary edema. Hypoxemia, blood pressure, and dyspnea markedly improved with intravenous furosemide and a nitroglycerin drip. Laboratory workup was significant for brain natriuretic peptide of 1,133 pg/mL (normal, 0-125) and fifth generation troponin that peaked at 143 ng/L (normal <23). After stabilization of acute hypoxic respiratory failure and flash pulmonary edema, the patient was admitted to the cardiac intensive care unit for management of acute decompensated heart failure.

Initial transthoracic echocardiogram (TTE) revealed severe mitral valve (MV) regurgitation based on a flail leaflet and an enlarged left ventricle (LV) with a systolic ejection fraction of 55%. An elevated right ventricular systolic pressure estimated at 67 mm Hg and a 2.7 cm mobile mass on the right ventricular ICD lead were also documented. Transesophageal echocardiography performed on day 4 confirmed posterior mitral leaflet prolapse with a small flail segment of the anterior mitral leaflet. Severe mitral regurgitation with

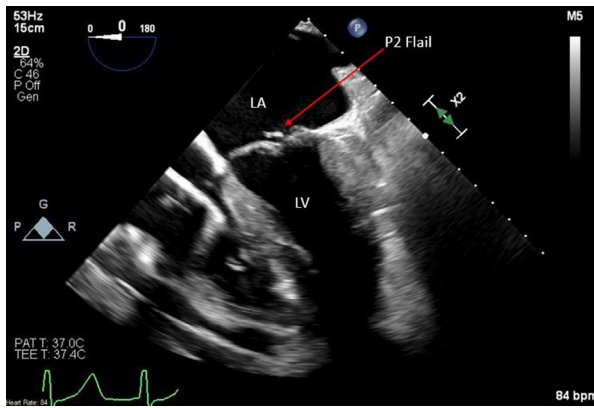


Figure 1 Midesophageal two-dimensional TEE 4-chamber view during systole showing P2 flail of the MV (arrow) prior to the surgery. There are no left atrial masses.

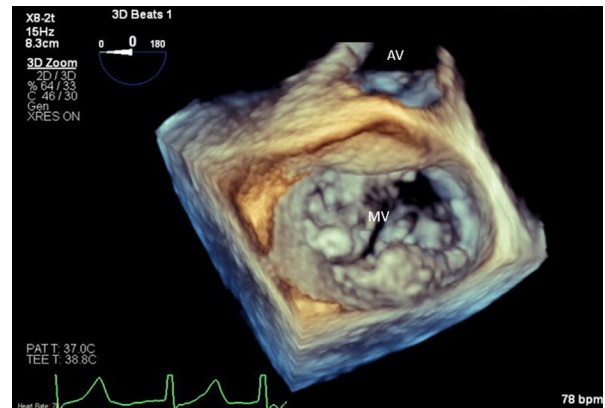


Figure 3 Midesophageal TEE three-dimensional rendering of the MV enface during systole. No left atrial masses are visualized prior to the surgery. AV, Aortic valve.

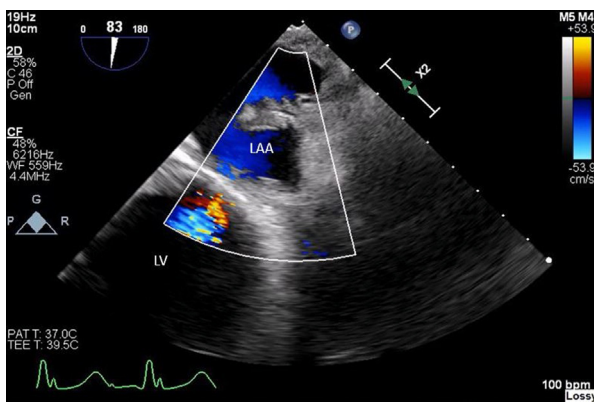


Figure 2 Midesophageal two-dimensional TEE 2-chamber view with color flow Doppler during diastole showing blood flow through the LAA prior to surgery.

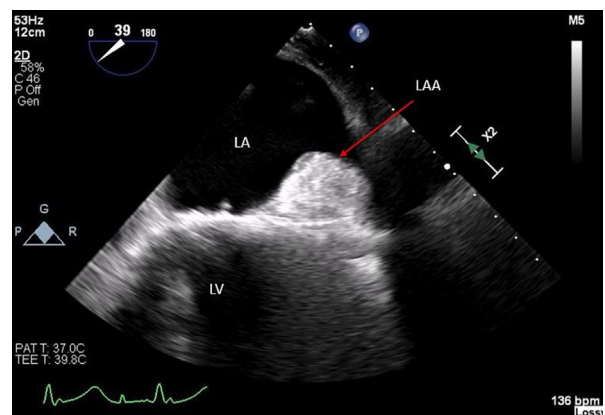


Figure 4 Midesophageal two-dimensional TEE 4-chamber view during systole revealing a new left atrial mass (arrow) after ligation.

Coanda effect was noted. Cardioversion was attempted for the AF but was unsuccessful. The decision was made to proceed with surgical correction of the AF, MV regurgitation, and right ventricular mass. On day 5 of hospitalization, the patient underwent a preoperative cardiac catheterization, which revealed minimal nonobstructive coronary artery disease.

On day 9 of hospitalization, the patient was taken to the operating room. Transesophageal echocardiography at the onset of the case confirmed severe regurgitation with a flail leaflet on the P2-P3 portion of the MV. No masses of the left atrium (LA) were noted at the start of the case as seen in [Figures 1 through 3](#) and [Videos 1 and 2](#). The LAA was identified, and blood flow into the structure was observed.

The distal ascending aorta, superior vena cava, and inferior vena cava were cannulated. A left ventricular vent was placed through the right superior pulmonary vein. Cardiopulmonary bypass (CPB) was initiated, and cardiac arrest was achieved with antegrade cardioplegia. While the patient was on bypass, the MV replacement, biatrial maze procedure, LAA ligation with an exclusion device (AtriClip, AtriCure), and removal of the right ventricular ICD mass were successfully completed. Prior to weaning the patient off CPB, the heart was deaired and the left ventricular vent was pulled back. Subsequent TEE revealed a large echogenic structure in the LA as seen in [Figures 4 through 6](#) and [Videos 3 through 5](#).

The mass was located superior to the MV and inferior to the pulmonary vein but did not obstruct blood flow. It was determined to be an inverted LAA given the acute onset and location within the atrium. The LAA could not be directly visualized by the surgeon. The mass was seen spontaneously everting on TEE, which further supported the diagnosis of LAA inversion ([Video 6](#)). The inverted LAA had spontaneously resolved by the end of the case ([Figures 7 and 8](#), [Videos 7 and 8](#)). Transesophageal echocardiography of the LAA before, during, and after inversion can be seen in [Figures 9 through 11](#).

The case was completed without further complications, and the patient was taken to the cardiac intensive care unit for recovery. There were no significant findings on TTE 4 days later. Atrial fibrillation recurred in the postoperative period after discontinuing temporary pacing and could not be converted to sinus rhythm by cardioversion. The patient's postoperative course was otherwise uneventful. They were discharged home on postoperative day 7.

DISCUSSION

Left atrial appendage inversion remains a rare finding. Intraoperatively, this diagnosis relies on TEE to identify the acute onset and location of the mass. A new left atrial mass in the intraoperative

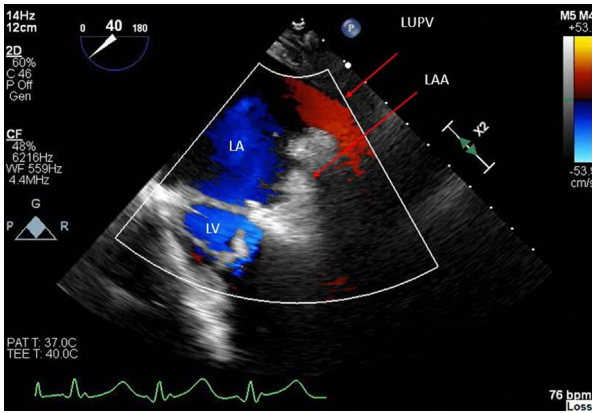


Figure 5 Midesophageal two-dimensional TEE 4-chamber view with color flow Doppler during diastole revealing a nonobstructive, inverted LAA after ligation. Arrows point towards the LAA and LUPV. LUPV, Left upper pulmonary vein.

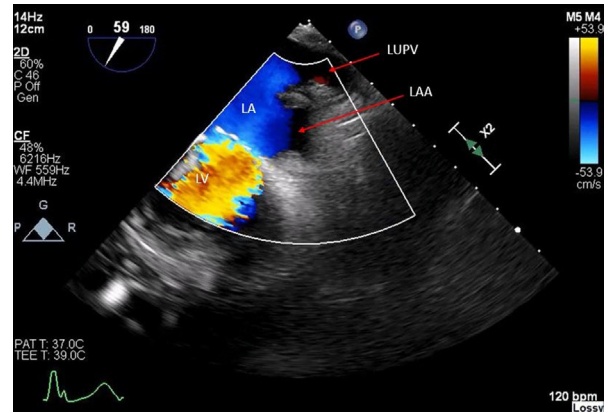


Figure 7 Midesophageal two-dimensional TEE mitral commissural view with color flow Doppler during diastole revealing spontaneous resolution of the inverted LAA at the end of the case. Arrows point towards the LAA and LUPV. LUPV, Left upper pulmonary vein.

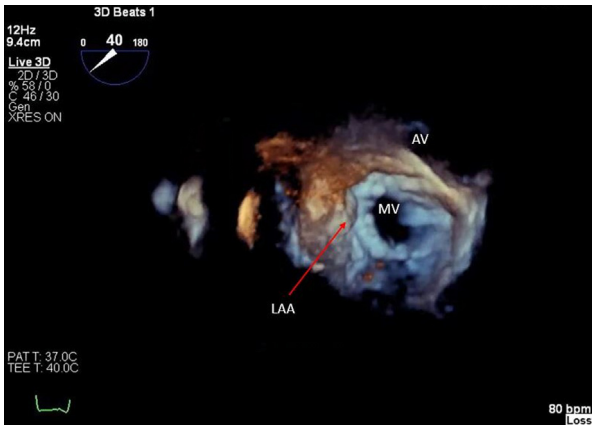


Figure 6 Midesophageal TEE three-dimensional rendering of the MV en face during diastole showing an inverted LAA after ligation (arrow). AV, Aortic valve.

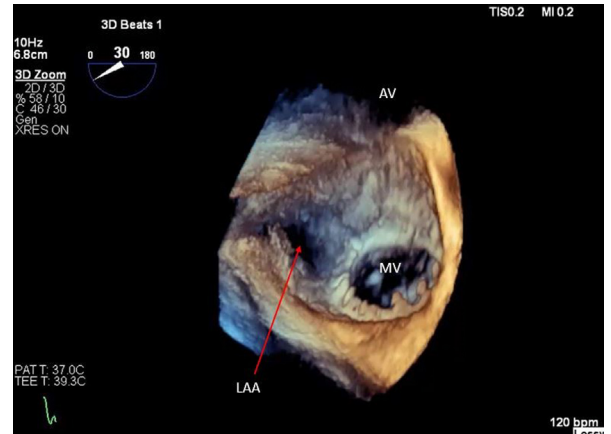


Figure 8 Midesophageal TEE three-dimensional rendering of the MV en face during systole after spontaneous resolution of the inverted LAA (arrow). AV, Aortic valve.

period eliminates common differentials such as neoplasms and vegetations assuming prior echocardiograms were obtained. Thrombosis is unlikely in patients undergoing CPB since significant anticoagulation is required. The location of the mass can also help determine the etiology. The LAA is located superior to the MV and inferior to the pulmonary vein. Inverted LAAs will appear as a mass attached to the lateral or anterolateral wall of the LA in this region.¹⁻⁵ The midesophageal 2-chamber and mitral commissural views on TEE allow for sufficient investigation of the LAA. A midesophageal 4-chamber view and long-axis view can also be utilized to investigate left atrial masses. Lack of color flow Doppler through the mass with an inability to identify the normal LAA supports the diagnosis of inversion. The spontaneous resolution of the left atrial mass is another supporting feature of LAA inversion, which occurs when the LAA independently everts. If these characteristics cannot be obtained from TEE, direct visualization of the inverted LAA from the surgical field is adequate for diagnosis.

Inversion occurs when excessive negative pressure is exerted on the left side of the heart. Cardiac surgeries requiring CPB create optimal conditions for inversion at predictable points. Specifically, retraction of the left ventricular vent or deairing maneuvers can

produce enough negative pressure to invert the LAA. Less frequently, inversion can arise from excessive external pressure such as a pericardial effusion or a hyperinflated lung.^{3,6} Direct traction on LAA tissue with devices during cardiac procedures can also produce inversion.⁷ While inversion is most common in the intraoperative period with CPB, any source of negative pressure can theoretically invert the LAA. For instance, multiple cases have been reported after left ventricular assist device implantation.^{5,8,9}

It is not possible to predict which patients will have LAA inversion; however, the risk of inversion may be influenced by the structural characteristics of the LAA. It has been suggested that shorter and wider appendages with a larger-diameter orifice increase the susceptibility for inversion.¹ Conversely, it has also been suggested that longer and thinner appendages with a smaller-diameter orifice increase the risk for sustained inversion.^{10,11} This case supports the theory that a shorter LAA has a higher likelihood of inverting. Inversion likely occurred from the negative pressure created when the left ventricular vent was pulled back after ligation. The placement of the clip may have been distal, which increased susceptibility for inversion by artificially shortening the LAA. This particular exclusion device clips the

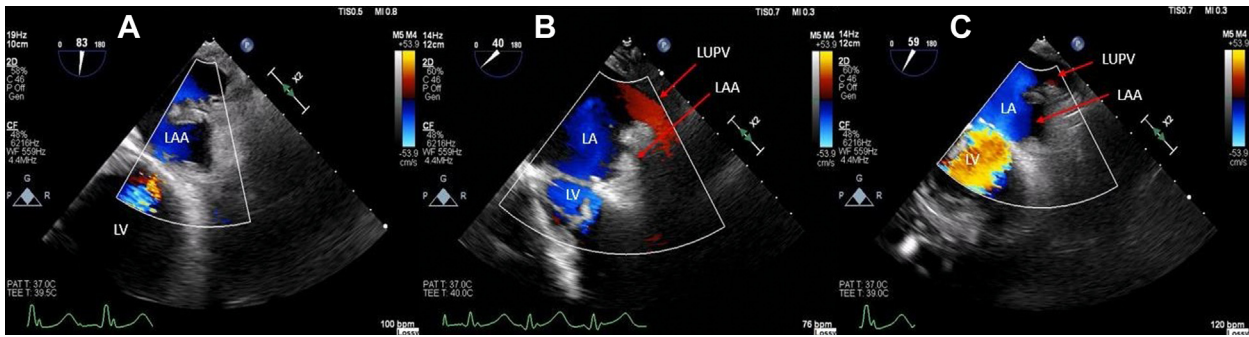


Figure 9 A series of images from a mid-esophageal two-dimensional TEE with color flow Doppler showing the LAA throughout the case. **(A)** Two-chamber view of the LAA prior to ligation and inversion. **(B)** Four-chamber view of the LAA after ligation and inversion. **(C)** Mitral commissural view of the ligated LAA after spontaneous resolution of the inversion. *Arrows* point towards the LAA and LUPV. LUPV, Left upper pulmonary vein.

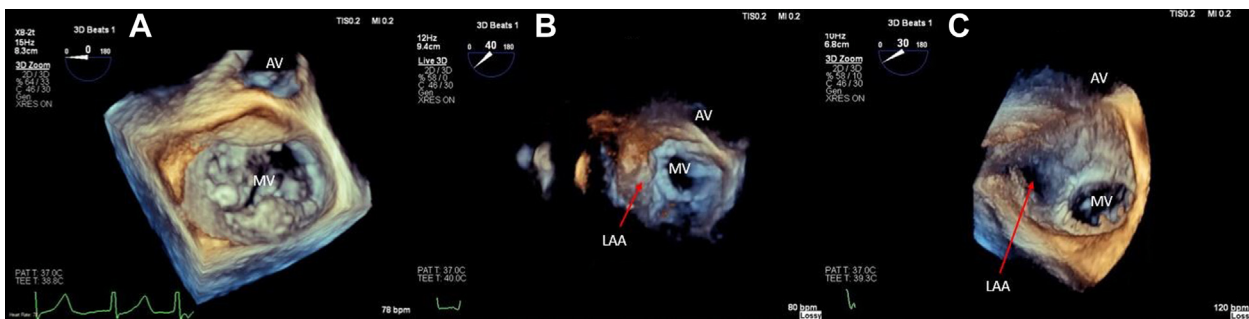


Figure 10 A series of images from a mid-esophageal TEE three-dimensional rendering of the MV en face showing the LA throughout the case. **(A)** Prior to the ligation no left atrial masses are present. **(B)** A new left atrial mass after ligation of the LAA. **(C)** The ligated LAA after spontaneous resolution of inversion. *Arrows* point towards the LAA. AV, Aortic valve.

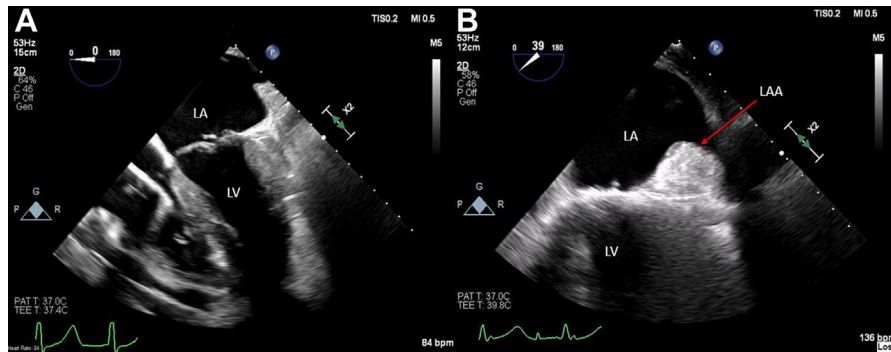


Figure 11 A comparison of the LA from a mid-esophageal two-dimensional TEE 4-chamber view during systole. **(A)** Prior to the surgery, there are no left atrial masses. **(B)** After ligation, there is onset of a new left atrial mass (*arrow*).

LAA, resulting in ischemia with eventual resorption of tissue distal to the clip. The inversion seen on TEE consisted of tissue proximal to the clip. One case documents inversion after ligation more than a year postoperatively.¹² There is no definitive evidence to suggest that ligation of the LAA prevents inversion. If ligation is performed for this reason, the clip should be placed as proximally as possible to the base of the LAA.

The long-term consequences of LAA inversion are poorly described as most cases resolve spontaneously or intraoperatively. Spontaneous resolution occurs as the left atrial pressure increases. In the context of CPB, this occurs when the cardiac volume is replen-

ished.^{2,3,5} In the absence of spontaneous resolution intraoperatively, interventions such as the Valsalva maneuver, manual eversion, or ligation have been used to resolve the inversion.^{1,3-5,10,11,13} Ligation of the LAA has been performed to prevent recurrent inversion and thrombosis.^{5,9} The intraoperative inversion after ligation in this case challenges the efficacy of ligation as a treatment. In the few cases in which LAA inversion persisted postoperatively, there was spontaneous resolution within 6 to 12 months.^{1,14} If inversion occurs in the postoperative period, identifying potential causes and reducing the negative pressure may result in eversion.⁶ Potential complications of sustained LAA inversion include MV obstruction, tissue necrosis, or

rupture.^{3,4,10,11} Right ventricular failure from MV obstruction secondary to inversion is one of the most severe sequela documented.^{5,9} Resorption of the LAA has not been documented from sustained inversion, but simulations suggest that intentional inversion may promote resorption of the LAA and effectively manage AF.¹⁵ Unnecessary corrective surgeries or medical interventions contribute to additional complications if LAA inversion cannot be distinguished from a thrombus or vegetation.^{9,11,14}

The potentially severe complications of sustained LAA inversion and risks associated with corrective surgeries make intraoperative diagnosis and management ideal. Enhanced physician knowledge and regular TEE assessment of the LAA after cardiac surgeries with increased risk for inversion can improve intraoperative diagnosis and promote proper management. Additional research is needed to elucidate the specific structural risk factors for LAA inversion, prevalence of inversion during cardiac surgeries versus the postoperative period, and the rate of spontaneous resolution.

CONCLUSION

Intraoperative utilization of TEE in cardiac cases enables early diagnosis and management of LAA inversion, obviating the need for excessive interventions and treatments. This case provides insight into the structural characteristics that influence inversion and suggests that a shorter LAA may be more likely to invert. Inversion of the LAA in this case suggests that ligation may increase the susceptibility for inversion, calling into question the efficacy of ligation as an intervention. The immediate inversion of the LAA after ligation is a rare finding but should be considered by physicians when a new left atrial mass is discovered.

ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans.

CONSENT STATEMENT

Complete written informed consent was obtained from the patient (or appropriate parent, guardian, or power of attorney) for the publication of this study and accompanying images.

FUNDING STATEMENT

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DISCLOSURE STATEMENT

B.L.C. receives speaker honoraria and is a consultant for Edwards LifeSciences. The remaining authors report no conflicts of interest.

SUPPLEMENTARY DATA

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.case.2023.02.003>.

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