

Traumatic Injury to Both Atrioventricular Valves



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INTRODUCTION

Blunt cardiac injury (BCI) is a well-recognized complication of major trauma; it can involve damage to several cardiac structures, including the myocardium, heart valves, pericardium, and coronary arteries. The prevalence of BCI is not completely known.¹ An especially rare presentation of BCI is simultaneous injury to both atrioventricular valves.

We report a case of mitral and tricuspid valve insufficiency complicating major thoracoabdominal trauma. Our case highlights both the usefulness and limitations of transesophageal echocardiography (TEE) for diagnosing traumatic injury to the atrioventricular valves.

CASE PRESENTATION

A 46-year-old man was transferred to our institution 5 days after an unwitnessed high-speed motor vehicle accident in which he was the solo, unrestrained driver. Initial assessment at a regional hospital identified major thoracoabdominal trauma with multiple fractured ribs, a fractured sternum, bilateral pulmonary contusions with hemothoraces, and a liver laceration with intraperitoneal hemorrhage. The patient was obese but otherwise previously well.

At presentation, the patient was hemodynamically unstable and required aggressive resuscitation with intravenous fluids, blood, and blood products and an infusion of norepinephrine. The patient underwent contrast computed tomographic (CT) imaging of his chest and abdomen that, among other findings, demonstrated a filling defect in his right main pulmonary artery (Figure 1) that was suspicious for pulmonary embolism. A transthoracic echocardiogram (TTE) was attempted, but the windows were suboptimal and nondiagnostic. Esophageal injury was excluded by careful review of the CT scan, so transesophageal echocardiogram (TEE) was performed; TEE demonstrated a posteriorly directed jet of severe mitral regurgitation, mild-to-moderate tricuspid regurgitation, and mild right ventricular impairment. A mobile linear mass was seen, extending from the right ventricle (RV) through to the main pulmonary artery. The patient's

intra-abdominal injuries were managed conservatively. He was commenced on an infusion of unfractionated heparin for presumed pulmonary embolism. The patient was then transferred to our institution for ongoing management.

Following transfer, repeat CT imaging no longer demonstrated a filling defect in the main pulmonary artery. His electrocardiogram (ECG) was unremarkable apart from sinus tachycardia at 146 beats per minute and an elevated serum troponin, which peaked at 257 (normal range, 0-16 ng/L).

The TTE and TEE examinations were repeated. However, TTE imaging was of poor quality, most likely due to the patient's body habitus and the use of mechanical ventilation. The diagnosis of his mitral valve abnormalities by TEE was relatively straightforward. There was flail of the anterior mitral leaflet involving the A2 and A3 segments (Figure 2, Video 1) with a posteriorly directed jet of severe mitral regurgitation (Figure 3, Video 2). There was abnormal motion of the posteromedial papillary muscle, consistent with partial rupture (Figure 4, Videos 3 and 4).

Diagnosis of his tricuspid valve pathology was less clear-cut. There was a complex jet of severe tricuspid regurgitation (Figure 5, Video 5). Standard two-dimensional imaging did not show abnormal leaflet motion (Figure 6, Video 6); however, three-dimensional imaging demonstrated apparent prolapse of the septal leaflet (Figure 7, Video 7). Evaluation of the tricuspid subvalvular apparatus did not show an obvious disruption. A mobile, linear mass was seen in the RV and right ventricular outflow tract (RVOT; Figure 8, Video 8). There was thickening of the RVOT, consistent with contusion (Figure 8). The left ventricle (LV) was hyperdynamic, and RV function was normal.

Valvular heart surgery was delayed for 2 weeks to allow recovery from his other thoracoabdominal injuries due to the concern of precipitating massive hemorrhage with systemic heparinization for cardiopulmonary bypass. At surgery, partial dehiscence of the posteromedial papillary muscle was confirmed. Additionally, there were multiple ruptured chords involving the P2, P3, A2, and A3 segments of the mitral valve. There was a large laceration involving the septal leaflet of the tricuspid valve, but the tricuspid subvalvular apparatus was intact and no thrombus was identified in the right heart. There was a severe contusion involving the RVOT. An attempt to repair the mitral valve was unsuccessful. Therefore, mitral valve replacement with a 27 mm Carpentier-Edwards Preimount MagnaEase Valve (Edwards Lifesciences Corp., Irvine, CA) was performed. The tricuspid valve was successfully repaired by closing the septal leaflet laceration and placing a 30 mm Cosgrove-Edwards Annuloplasty System (Edwards Lifesciences Corp.).

The patient made a prolonged but uneventful recovery, being discharged from the intensive care unit 10 days after surgery.

DISCUSSION

Traumatic injury to both atrioventricular valves is extremely rare, with only a few reported clinical cases.²⁻⁴ Motor vehicle accidents are the

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VIDEO HIGHLIGHTS

Video 1: TEE, three-dimensional, left atrial view of the mitral valve. Flail involving the A3 segment is clearly seen.

Video 2: TEE, midesophageal long-axis view with color compare. In the left-hand frame, a flail segment of the anterior mitral leaflet is seen. In the right-hand frame, an eccentric, posterior jet of severe mitral regurgitation is evident.

Video 3: TEE, transgastric short-axis view at the midpapillary level. A partial rupture of the posteromedial papillary muscle is seen within the LV.

Video 4: TEE, transgastric two-chamber view. A partial rupture of the posteromedial papillary muscle can be seen within the LV.

Video 5: TEE, modified midesophageal four-chamber view demonstrating severe tricuspid regurgitation.

Video 6: TEE, midesophageal four-chamber view demonstrating a flail segment of the anterior mitral leaflet.

Video 7: TEE, three-dimensional multiplanar reconstruction of the tricuspid valve. Prolapse involving the septal leaflet of the tricuspid valve is evident.

Video 8: TEE, modified midesophageal right ventricular inflow outflow view. A mobile, linear shadow is seen in the RVOT. The free wall of the RVOT appears thickened, consistent with myocardial contusion.

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most common mechanism of traumatic injury to the heart valves, but other causes have been described, including falls and kicks from animals.^{4,5} However, the condition is likely underappreciated given that postmortem and forensic data show that BCI complicates up to 20% of major trauma, with valvular injury contributing to 4% of cases.^{6,7} Postmortem data suggest that the aortic and tricuspid valves are the most commonly injured heart valves.⁶ The reason that clinical cases are rare is that survival from trauma of sufficient severity to cause valvular injury is thought to be unlikely.^{8,9}

The mechanism of traumatic atrioventricular valve injury is most likely compression of the ventricle(s) between the sternum and spine coincident with isovolumetric contraction with a consequent supranormal increase in intraventricular pressure against closed valves.^{6,8} The papillary muscles are the most commonly affected component of the mitral valve, with resultant valvular regurgitation.³ The tricuspid valve is thought to be at increased risk of traumatic injury compared with the mitral valve due to its anterior position and weaker valvular structures. The chordae tendinae are the most commonly injured element of the tricuspid valve, again resulting in valvular regurgitation.¹

Transesophageal echocardiogram is the ideal bedside technique for assessing BCI.¹⁰ In particular, TEE is superior to TTE as it typically affords excellent views of the valvular and subvalvular structures and image quality is less affected by body habitus, mechanical ventilation, and the presence of surgical drains.¹⁰⁻¹² Other methods of assessing cardiac function, such as troponin and ECG, are useful screening tools but are nonspecific, and abnormalities are often transient.¹³ In this case, TEE was useful for characterizing the mitral valve pathology. However, we were uncertain as to the mechanism of the tricuspid valve injury. Furthermore, we were unsure whether the mobile linear

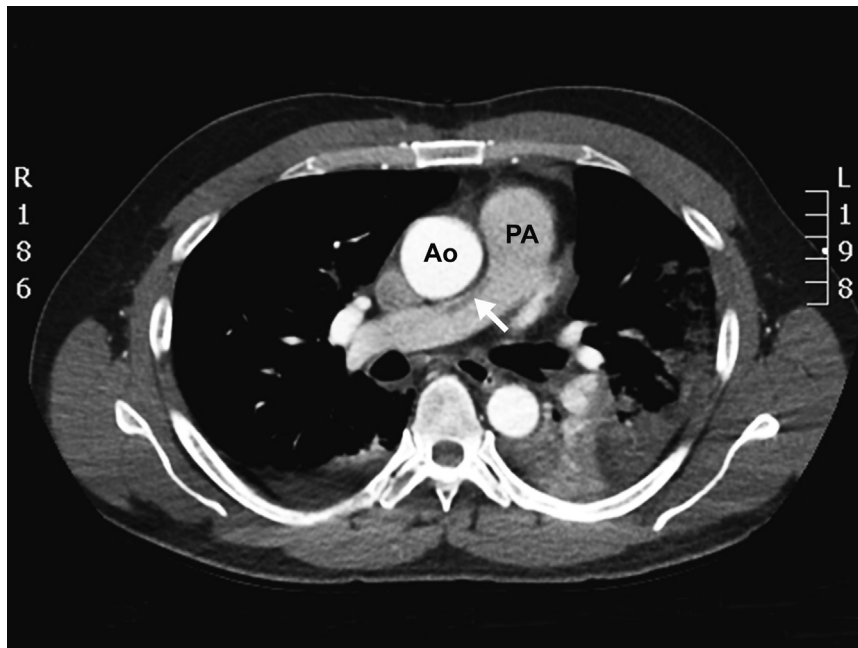


Figure 1 Contrast-enhanced CT scan at the level of the right main pulmonary artery (PA) demonstrating a linear intraluminal hypodensity. Ao, Aorta.

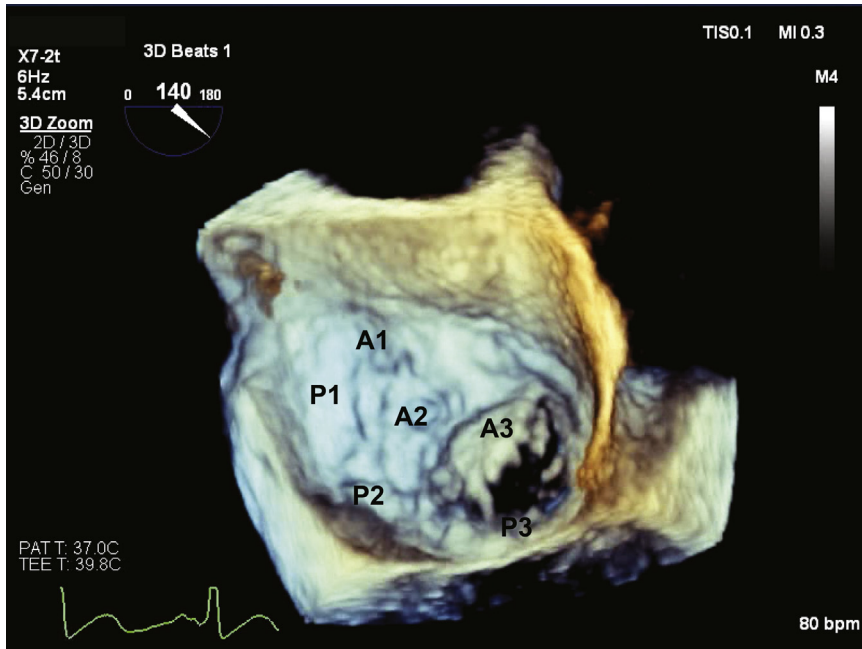


Figure 2 TEE, three-dimensional, left atrial view of the mitral valve in late systole. The leaflet segments are demonstrated. Flail involving the A3 segment is clearly seen.

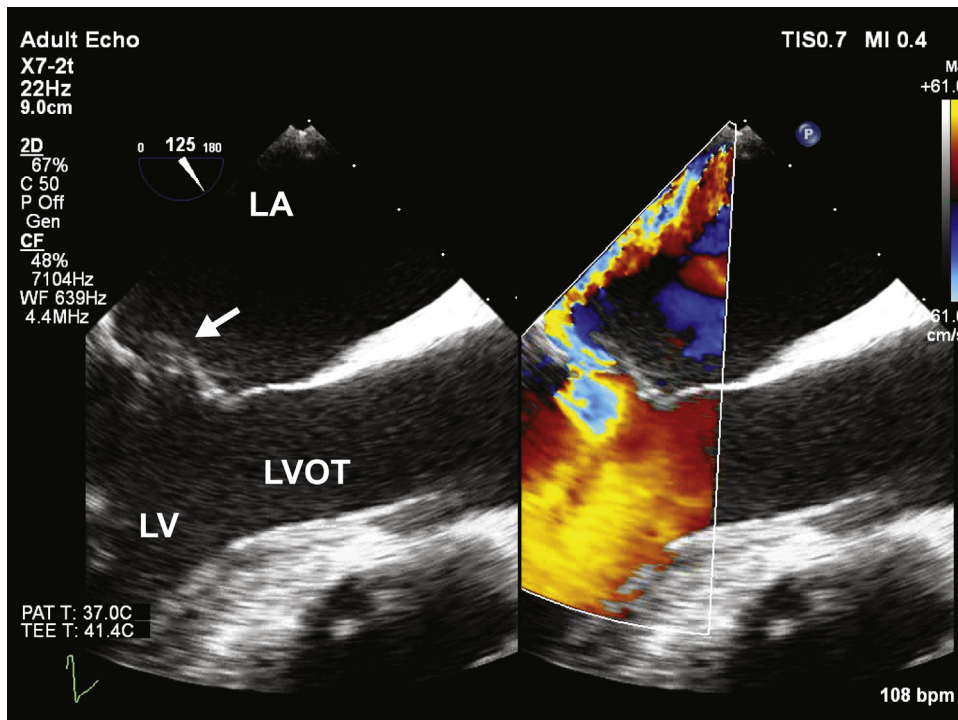


Figure 3 TEE, midesophageal long-axis view with color compare. In the left-hand frame, a flail segment of the anterior mitral leaflet is seen (arrow). In the right-hand frame, an eccentric, posterior jet of severe mitral regurgitation is evident. LA, Left atrium; LVOT, left ventricular outflow tract.

mass in the RV represented thrombus, chordal tissue, or a combination of both. The surgical findings indicate that the tricuspid injury was related to traumatic laceration of the septal leaflet rather than disruption to the subvalvular apparatus. The linear mass in the

RVOT probably represented thrombus, which had dissipated or embolized by the time of surgery.

Injury to the cardiac valves following major trauma is commonly missed or the diagnosis is delayed.⁹ Because traumatic injury to the

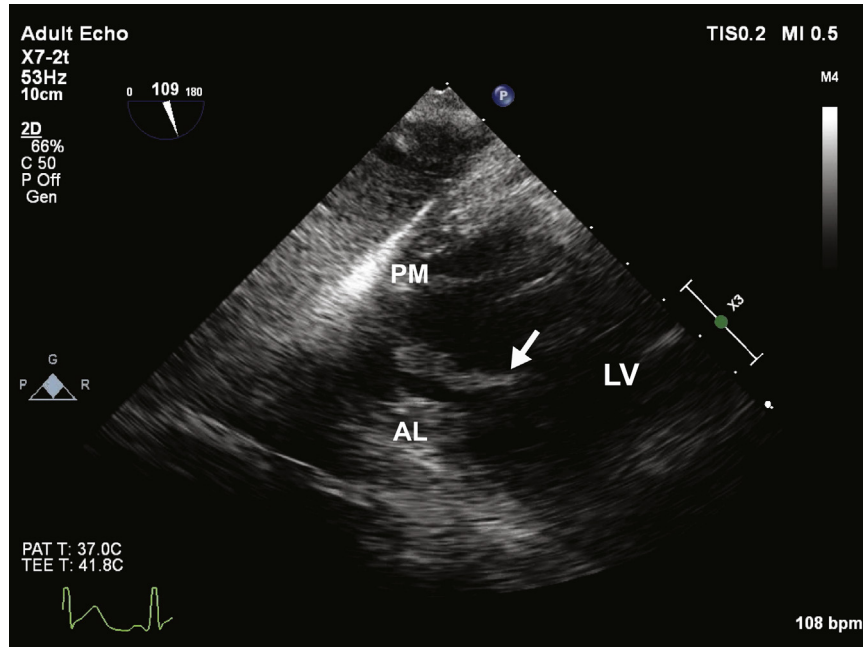


Figure 4 TEE, transgastric two-chamber view. The posteromedial (PM) and anterolateral (AL) papillary muscles are demonstrated. Partial rupture of the PM papillary muscle is shown (arrow). In the associated clip, the ruptured head of the papillary muscle can be seen flicking freely in the LV.

heart valves is rare, and the patient may have other more obvious injuries, clinicians may fail to consider the diagnosis. Abnormalities on the ECG, an elevated troponin, and unexpected hemodynamic instability should raise the possibility of BCI. An echocardiogram for BCI, where the primary suspected lesion is typically myocardial contusion, should also include detailed evaluation of the heart valves. However, failure to be able to fully appreciate the extent of BCI with early TTE has been described in another recent case

report involving injury to the aortic and tricuspid valves.¹² A second reason for delayed diagnosis is that valvular function may deteriorate over time following the initial injury due to damaged tissue subsequently becoming necrotic or forming a weak scar with subsequent rupture.^{8,9,11-13} Late presentations of valvular dysfunction, weeks and even several months after initial injury, have been reported.^{7,12} In this patient, the presence of severe thoracic trauma in association with hemodynamic instability

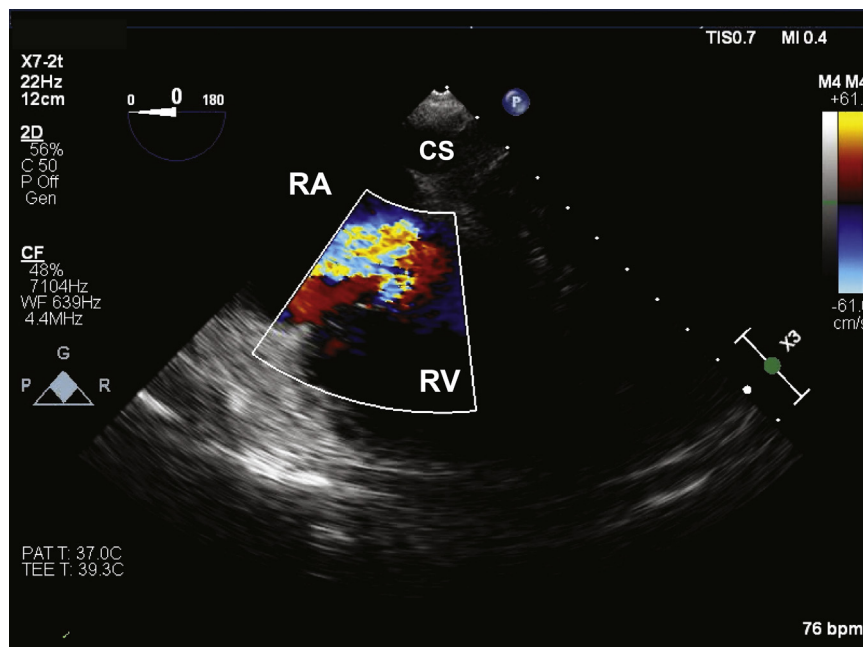


Figure 5 TEE, modified midesophageal four-chamber view in late systole demonstrating severe tricuspid regurgitation. CS, Coronary sinus; RA, right atrium.

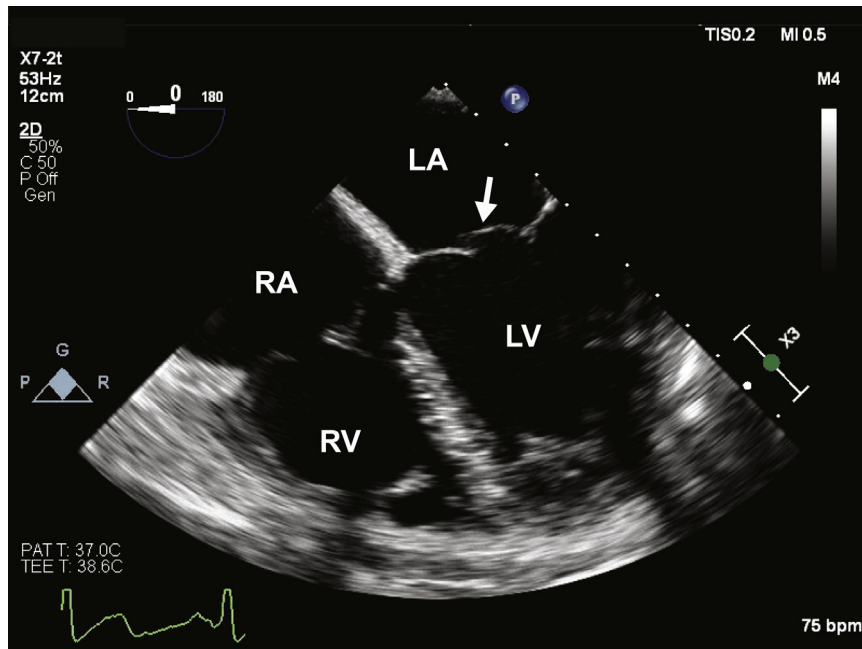


Figure 6 TEE, midesophageal four-chamber view in late systole. A flail segment of the anterior mitral leaflet is seen (*arrow*). However, there is no obvious abnormality of the tricuspid valve, despite the presence of severe tricuspid regurgitation (see [Figure 5](#)). LA, Left atrium; RA, right atrium.

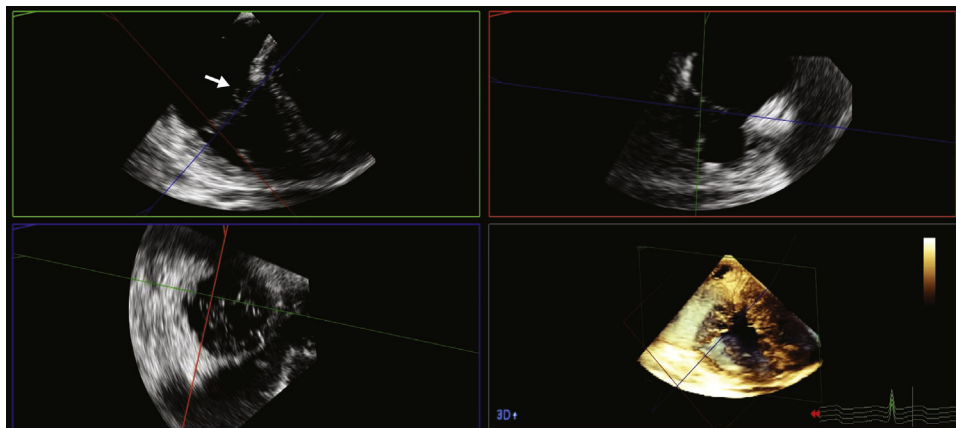


Figure 7 TEE, three-dimensional multiplanar reconstruction of the tricuspid valve in late systole. Prolapse involving the septal leaflet of the tricuspid valve is evident (*arrow*), which was not apparent on standard two-dimensional imaging (see [Figure 6](#)).

raised the possibility of BCI. The initial TEE examination was performed shortly after the patient presented to the referring hospital. While severe mitral valve regurgitation was identified at this time, the tricuspid regurgitation was graded as only mild to moderate. It is uncertain whether severe tricuspid regurgitation was missed at the first TEE examination or developed subsequently; however, given the surgical findings of a lacerated septal leaflet, the former is more likely than the latter.

Ideally, valvular repair or replacement should occur within the first few days following injury.⁸ However, in the presence of other injuries surgery may need to be delayed, which was the case here.

CONCLUSION

Our case highlights the potential for injury to the heart valves following major thoracoabdominal trauma and adds to the small number of reports of survival following simultaneous traumatic injury to both atrioventricular valves. The mechanism of injury is likely due to the compressive force leading to excessive intraventricular pressure and damage to valvular structures. Transesophageal echocardiogram is an essential tool for evaluating the mechanism of valve injury and the severity of valve dysfunction. Early diagnosis allows for medical and surgical treatment, which can be lifesaving.

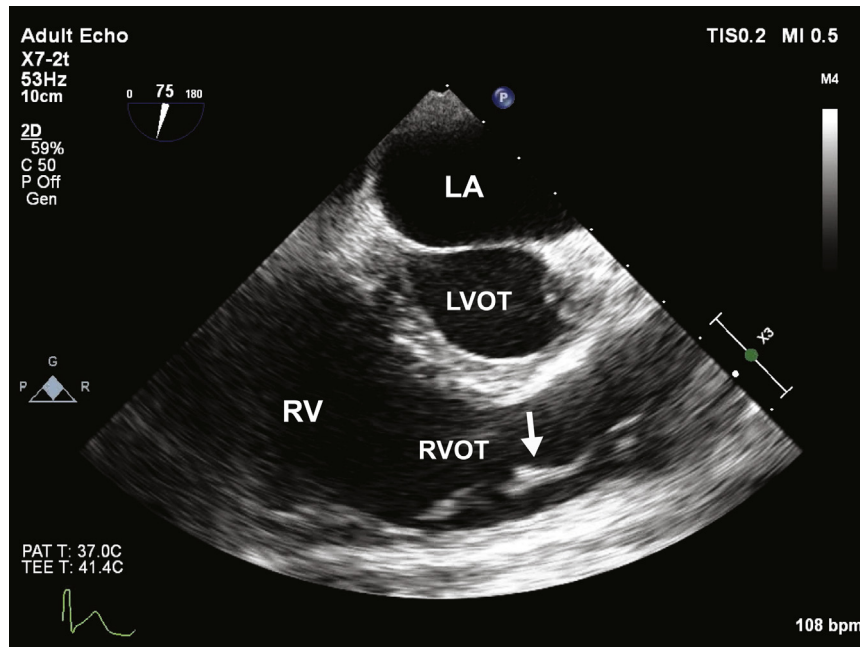


Figure 8 TEE, modified midesophageal right ventricular inflow outflow view. A mobile, linear shadow is seen in the RVOT (arrow). In addition, the free wall of the RVOT appears thickened, consistent with myocardial contusion. LA, Left atrium; LVOT, left ventricular outflow tract.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.case.2021.07.001>.

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