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CASE REPORT
Heart failure

Unexpected suicide left ventricle post-surgical aortic valve replacement requiring veno-arterial extracorporeal membrane oxygenation support despite gold-standard therapy: a case report

Peter Andrew Lioufas [1], Diane N. Kelly[1,2,3], Kyle S. Brooks [1,4], and Silvana F. Marasco[5,6,7]*

[1]Department of Intensive Care, Epworth Richmond, Ground Floor, 89 Bridge Road, Richmond, Victoria 3121, Australia; [2]Department of Intensive Care, The Royal Melbourne Hospital, Level 5 Building B, 300 Grattan Street, Parkville, Victoria 3050, Australia; [3]Faculty of Medicine, Nursing and Health Sciences, Monash University, 27 Rainforest Walk, Clayton, Victoria 3800, Australia; [4]Faculty of Medicine, Dentistry and Health Sciences, University of Melbourne, Building 104, Alan Gilbert Building, University of Melbourne, 161 Barry Street, Carlton, Victoria 3010, Australia; [5]Department of Cardiothoracic Surgery, Epworth Richmond, Epworth Centre, Suite 8.6, 32 Erin Street, Richmond, Victoria 3121, Australia; [6]Department of Cardiothoracic Surgery and Transplantation, The Alfred Hospital, Commercial Road, Melbourne, Victoria 3004, Australia; and [7]Department of Surgery, Monash University, The Alfred Hospital, Central Clinical School, Level 6, Alfred Centre, 99 Commercial Road, Melbourne, Victoria 3004, Australia

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Background Suicide left ventricle is a well-documented phenomenon occurring after valve replacement, however, it is most commonly described in the mitral valve replacement (MVR) and transcatheter aortic valve replacement (TAVR) population. Cases within the surgical aortic valve replacement (SAVR) population usually resolve with optimal medical and interventional therapies. We describe a case of left ventricular suicide following SAVR presenting with persistent haemodynamic instability despite currently accepted medical and surgical therapies.

Case summary A 62-year-old male with severe aortic stenosis presented for SAVR and a MAZE procedure. There were no significant signs of ventricular hypertrophy on preoperative transthoracic echocardiogram (TTE). Intraoperatively, there was mild chordal systolic anterior motion of the mitral valve (SAM) which only occurred when underfilled. During recovery in the intensive care unit, the patient's pulmonary arterial pressures were noted to rise with worsening cardiac output. Subsequent TTE showed severe dynamic left ventricular outflow tract (LVOT) obstruction secondary to SAM. Due to refractory medical management, an alcohol septal ablation was performed. Despite resolution of obstruction, the patient exhibited biochemical signs of systemic hypoperfusion, and thus veno-arterial extracorporeal membrane oxygenation (VA-ECMO) support was initiated. Following 72 h of VA-ECMO support, the patient was weaned with complete resolution of biochemical insults. He was subsequently discharged from the hospital without complication.

Discussion Compared to the TAVR population, suicide ventricle post-SAVR is comparatively rare. Patients who exhibit persistent impaired cardiac output postoperatively should be investigated rapidly with echocardiography. Furthermore, resolution of a LVOT obstruction state from procedural intervention may not immediately follow with improved cardiac output, and may require further supportive management.

Keywords Case report • Cardiogenic shock • Acute heart failure • Aortic valve replacement • Extracorporeal membrane oxygenation • Echocardiography

ESC Curriculum 2.2 Echocardiography • 7.1 Haemodynamic instability • 6.4 Acute heart failure • 7.5 Cardiac surgery • 7.3 Critically ill cardiac patient

* Corresponding author. Tel: +61 3 9076 2156, Email: s.marasaco@alfred.org.au
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Learning points

- A low threshold for urgent echocardiography should be considered for cardiac output compromise not resolved by simple interventions (e.g. fluid administration) following surgical aortic valve replacement.
- Although procedural intervention will definitely resolve left ventricular outflow tract (LVOT) obstruction in these cases, there may be considerable delay in haemodynamic improvement post-procedure.
- Short-term extracorporeal membrane oxygenation (ECMO) support may be required post-LVOT obstruction resolution in those without immediate improvement in haemodynamic parameters.
- Monitoring of haemodynamic parameters during weaning of ECMO should be performed with real-time transoesophageal echocardiogram imaging, as standard monitoring via pulmonary arterial thermodilution catheter technique has inherent limitations.

Introduction

Dynamic left ventricular outflow tract (LVOT) obstruction is known to occur following aortic valve replacement with variable outcomes,[1] and particularly in transcatheter aortic valve replacement (TAVR) such an obstruction can lead to the phenomenon known as 'suicide left ventricle'. In the surgical aortic valve replacement (SAVR) population, dynamic LVOT obstruction with systolic anterior motion of the mitral valve (SAM) tends to manifest intraoperatively. In circumstances where it occurs postoperatively, it can often be treated medically. We report a case of suicide left ventricle following SAVR with worsening and persistent cardiac instability despite appropriate medical and surgical therapies.

Timeline

Day 0	
11:00 a.m.	Admitted from cardiac theatre, stable condition and indices.
8:00 p.m.	Increased systolic pulmonary arterial pressure 50 mmHg, cardiac index 1.7.
10:00 p.m.	Transthoracic echocardiogram (TTE), systolic anterior motion of the mitral valve confirmed. Alpha agonists/beta blockade ongoing.
Day 1	
9:00 a.m.	Cardiac index dropped to 1.6, increasing pulmonary arterial pressures.
9:15 a.m.	Repeat TTE showing persistent mid-cavity obstruction. Decision made for alcohol septal ablation.
4:00 p.m.	Transferred for alcohol septal ablation, uncomplicated procedure.
7:00 p.m.	30 min post-procedure, TTE confirmed resolution of mid-cavity obstruction. Cardiac index 1.6.
Day 2	
9:00 a.m.	TTE performed due to persistently low cardiac outputs. Further inotropic and vasopressor agents trialled.
2:30 p.m.	Worsening biochemical parameters, low cardiac output despite inotropy. Decision made for veno-arterial extracorporeal membrane oxygenation (VA-ECMO).
5:00 p.m.	Patient intubated in preparation for VA-ECMO establishment.
11:00 p.m.	VA-ECMO established. Inotropic agents weaned off.
Days 3–4	Slow weaning of extracorporeal membrane oxygenation (ECMO) supports, improving biochemistry.
Day 5	
10:40 a.m.	ECMO weaning transoesophageal echocardiogram study performed, VA-ECMO down to 1.0 L/min.
2:00 p.m.	ECMO ceased and patient decannulated.
Days 6–7	Vasopressor supports weaned off. Complete resolution of biochemical insults.

Continued

Case presentation

A 62-year-old male with known bicuspid aortic valve, and paroxysmal atrial fibrillation (AF) presented for SAVR and Maze procedure following a diagnosis of severe aortic valve stenosis. His past medical history included hypertension treated with oral Telmisartan 80 mg daily and Moxonidine 400 mcg daily. He further had paroxysmal AF following an episode of infective endocarditis eight years prior sustained at the time of a dental extraction. He was anticoagulated with Apixaban 5 mg BD and was treated with oral Amiodarone 200 mg daily which provided some intermittent reversion to sinus rhythm (with a chronic left bundle branch block) in the community. Non-cardiac past medical history includes obstructive sleep apnoea requiring a continuous positive airway pressure (CPAP) machine, and a bladder diverticulum managed conservatively. He was a non-smoker, with a history of social alcohol consumption (with occasionally heavy consumption on weekends). Referral for cardiac surgery was made, whilst the patient was asymptomatic, due to worsening aortic stenosis on routine follow-up transthoracic echocardiograms (TTE), which reported a low-normal left ventricular (LV) systolic function (no ejection fraction was provided) and the presence of concentric LV hypertrophy, with posterior wall thickness 1.2 cm and septal thickness of 1.2 cm. His aortic valve was bicuspid, sclerotic, thickened (*Figure 1A*), and had

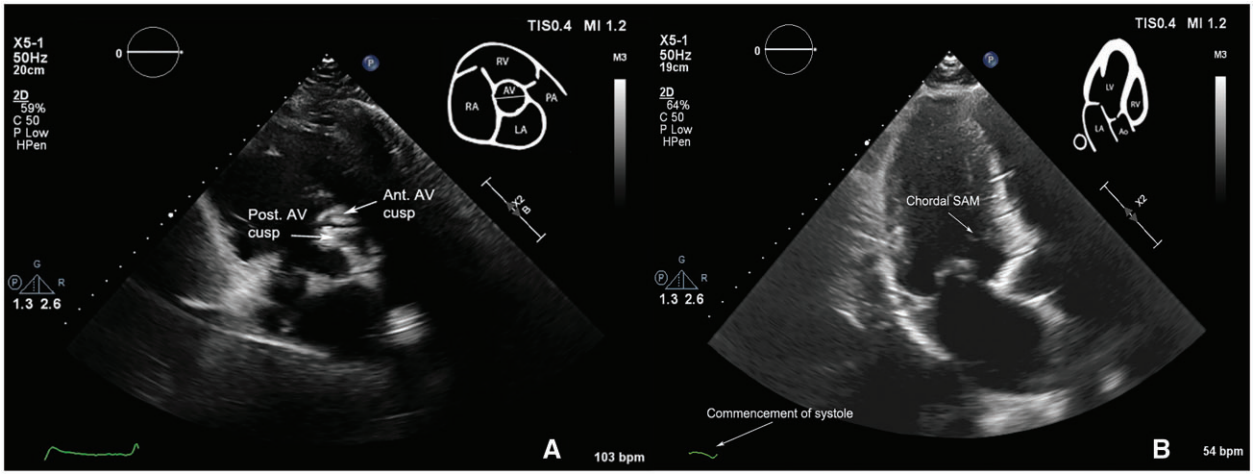


Figure 1 Preoperative transthoracic echocardiogram. (*A*) Parasternal short-axis view showing thickened, sclerotic, true bicuspid aortic valve. (*B*) Evidence of chordal SAM on apical three-chamber view, with increased gain.

severely restricted aortic valve leaflets, a flow velocity of 5.1 m/s, a mean gradient of 63 mmHg (maximum 104 mmHg), and an LVOT diameter of 2.8 cm. Unfortunately, the preoperative TTE was performed at an external site to the operating hospital, with only the report and no images provided. Notably, the septal thickness measurement was significantly lower than was recorded on the TTE studies from our institution. Review of the images shows that the septal thickness was undermeasured and the septal hypertrophy was underappreciated on preoperative imaging when compared with current guidelines[2] (*Figure 2*). Chordal SAM was further noted during this review and was not commented on by the reporting cardiologist (*Figure 1B* and *Video 1*).

Preoperative examination revealed a well-looking, 98 kg Caucasian male with an ejection systolic murmur heard maximally in the aortic region consistent with aortic stenosis. Lung fields were clear. His vital signs preoperatively showed a regular heart rate of 59 b.p.m. with electrocardiogram showing a left bundle branch block, blood pressure of 152/87 mmHg, respiratory rate of 18 breaths per minute with an oxygen saturation of 97%. He was afebrile with a body temperature of 35.8°C. A preoperative coronary angiogram performed 1 week prior to surgery revealed normal coronary arterial flow with no plaque disease, and a concomitant right heart catheterization revealed normal pulmonary arterial and pulmonary capillary wedge pressures with no intracardiac shunt. The pre-bypass transoesophageal echocardiogram (TOE) confirmed marked concentric LV hypertrophy, severe aortic stenosis, and both trace aortic and trivial mitral regurgitation (*Figure 3A* and *B*). The left and right ventricles demonstrated normal systolic function. No SAM was evident.

The procedure was completed uneventfully. Radiofrequency clamp was used to perform a left-sided maze procedure. An *Atriclip* (Atricure, OH, USA) was then deployed across the base of the atrial appendage. A true bicuspid aortic valve was identified. Surgical aortic valve replacement was completed with antegrade and retrograde cardioplegia. Following intraoperative sizing of the annulus, a 27 mm bioprosthetic, bovine, sutured *Inspiris Resilia* pericardial valve (Edwards LifeSciences, CA, USA) was sited. This type of valve was selected to facilitate a future in-valve TAVR if required. As there was no sign of dynamic LVOT obstruction preoperatively or intraoperatively no surgical myectomy was performed. Total bypass time was 87 min, with a total cross-clamp time of 70 min. Intraoperative photography was not available.

The post-bypass TOE showed a marked improvement of the aortic gradient at 7 mmHg over the valve, with a normally functioning bioprosthetic valve *in situ* (*Figure 3C* and *D*). No signs of patient–prosthesis mismatch were encountered, and no paravalvular leak was identified.

Mild chordal SAM was evident when the left ventricle was underfilled and was seen to resolve with fluid bolus and 0.5 mg of metaraminol. A cardiac index (CI) of 2.5 (L/min/m^2) was measured following the weaning of bypass, with pulmonary arterial pressures of 30/17 mmHg.

The patient remained stable in the intensive care unit (ICU) with good cardiac indices for the first few hours postoperatively, requiring only 1 L of 4% albumin fluid resuscitation, until he was extubated. Subsequently, his initial CI of 2.4 L/min/m^2 continued to fall steadily to 1.7 (equivalent to a cardiac output of 3.7 L/min), with pulmonary arterial pressures increasing up to 50/30 mmHg. The patient developed a first-degree atrioventricular block alongside his previously present left bundle branch block and was switched to a dual-chamber pacing mode (DDD). No ST changes or new T-wave inversions developed during this time. Given the LV hypertrophy noted intraoperatively, a metoprolol infusion was commenced to aim for a heart rate <65 b.p.m., and further fluid was administered.

An urgent TTE was performed showing marked SAM, resulting in severe dynamic LVOT obstruction (*Figure 4* and *Video 2*). On this study, the septal wall thickness was recorded at 1.8 cm (parasternal long-axis view). The measured peak gradient was 170 mmHg, with a max velocity of 6.5 m/s. Left ventricular systolic function was preserved.

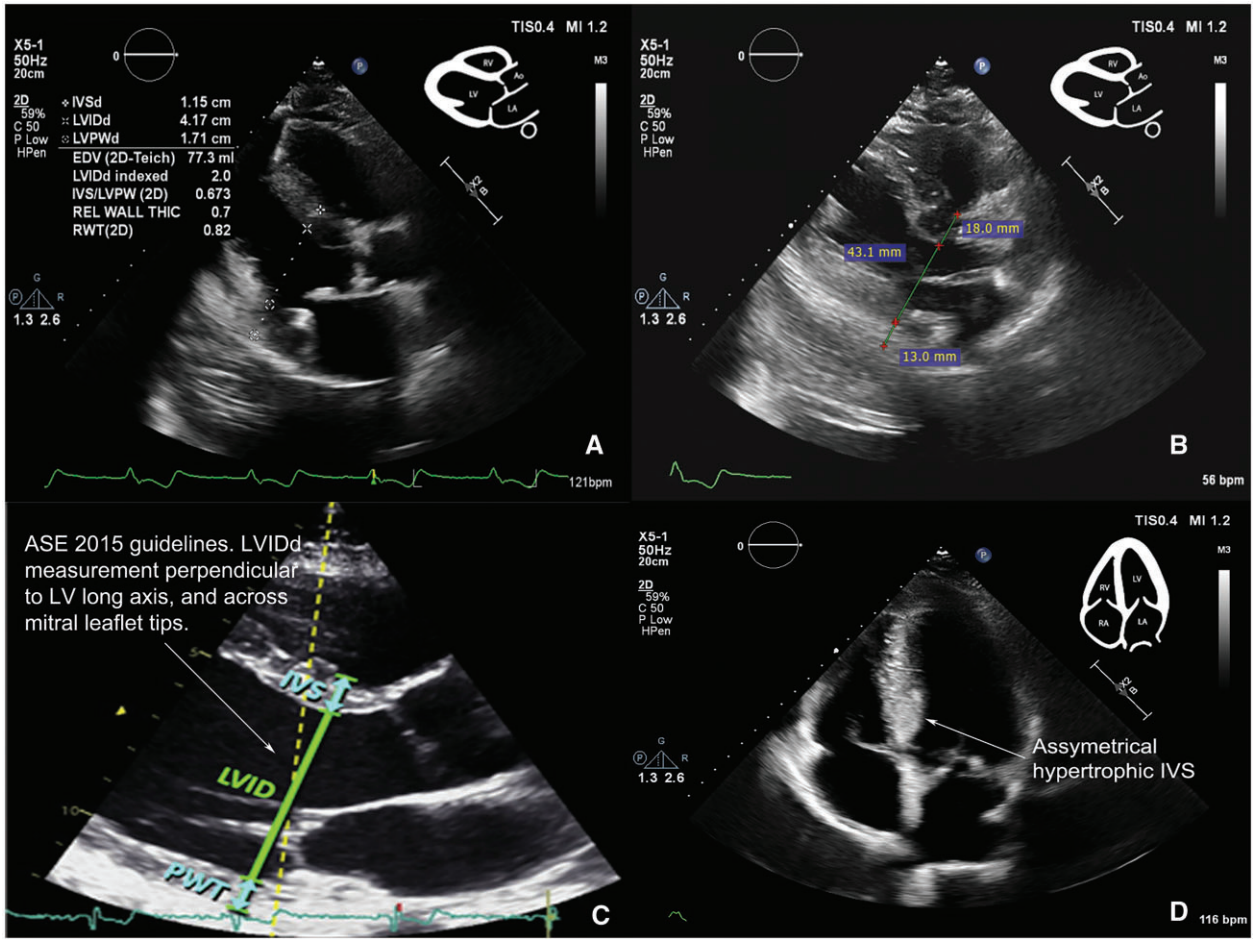
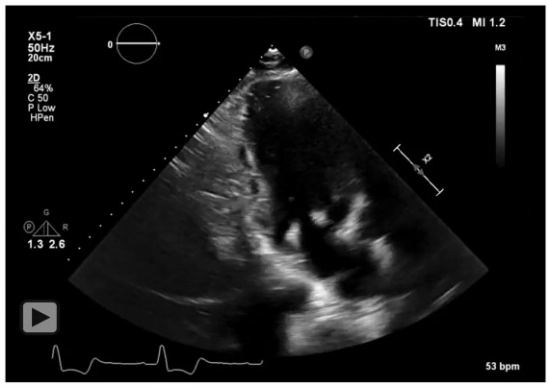


Figure 2 Preoperative transthoracic echocardiogram. (*A*) External institution left ventricular dimension measurements. (*B*) Retrospective analysis and re-measurement of left ventricular measurements, with increased interventricular septum size. (*C*) American Society of Echocardiography 2015 guidelines example for left ventricular dimension measurement.[2] (*D*) Interventricular hypertrophy evident on apical four-chamber view.



Video 1 Preoperative transthoracic echocardiogram. Loop in apical three-chamber view showing chordal SAM.

Medical therapy was continued in the form of a phenylephrine, metoprolol, and a prophylactic amiodarone infusion to prevent AF. Unfortunately, his CI worsened to 1.6 L/min/m^2 with a repeat echocardiogram revealing persistent, severe SAM with a moderately dilated left atrium (28 cm^2) and a peak LVOT gradient of 86 mmHg. Given the absence of any new segmental hypokinesis on TTE or acute ischaemic electrocardiogram changes, and with pre-operative coronary angiogram showing plaque-free coronary arteries, an acute ischaemic event was not considered to be the likely causative pathology. As a result, cardiac enzymes were not sent at this time as it was felt that they would not aid in overall diagnosis.

Septal myectomy was considered. Due to the risks of a second cardiopulmonary bypass run and aortic cross-clamp, in addition to explantation of the aortic prosthesis to perform the resection, an alcohol septal ablation was decided to be a more appropriate intervention. It was completed without complication approximately 30 h post-initial operation, with accompanying coronary angiography

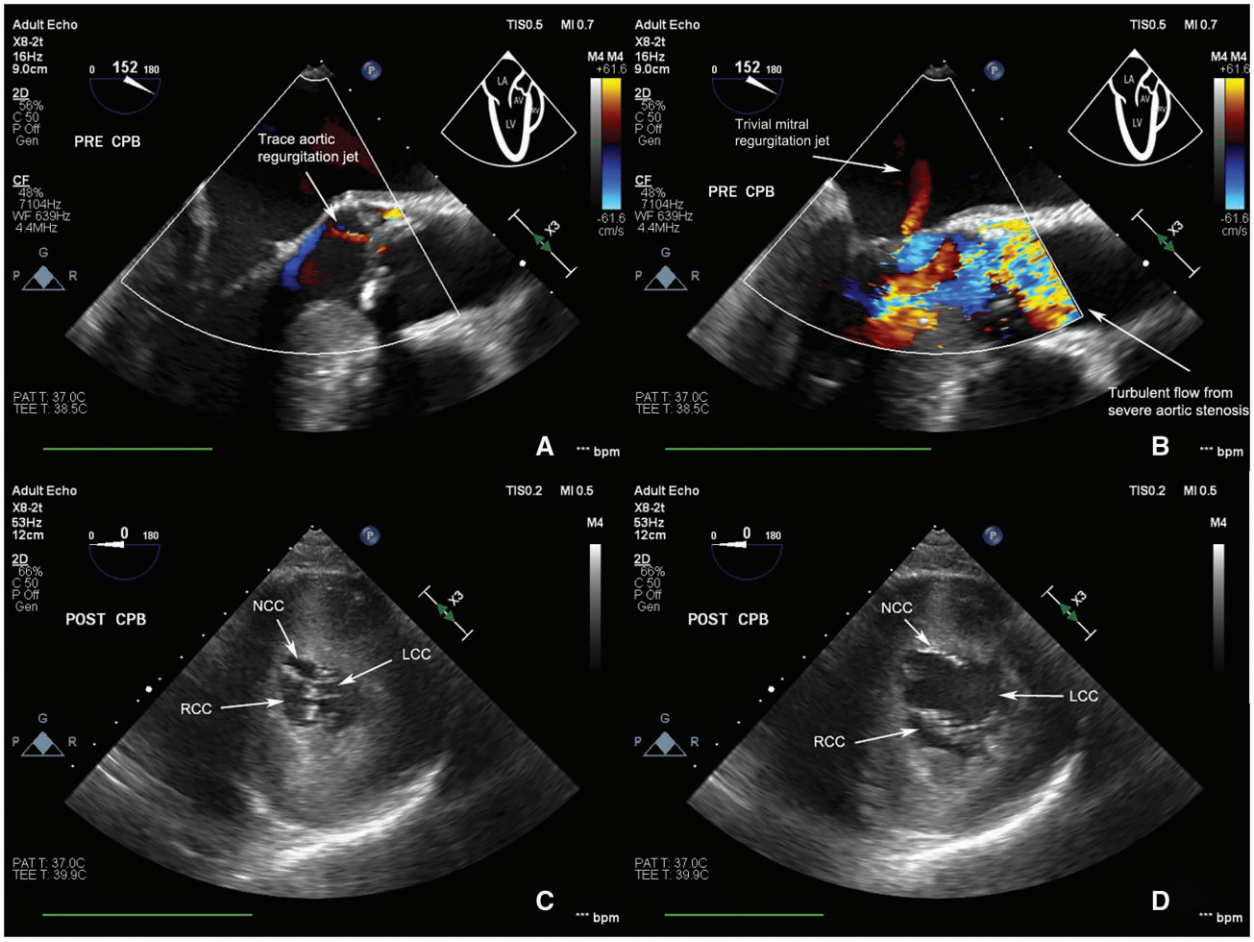


Figure 3 Intraoperative transoesophegeal echocardiogram. (*A* and *B*) Pre-CPB mid-oeseophageal views showing colour Doppler of aortic and mitral valve function. (*C* and *D*) Post-CPB mid-oesophageal short-axis view of *Inspiris Resilia* aortic valve in both closed and opened conformations.

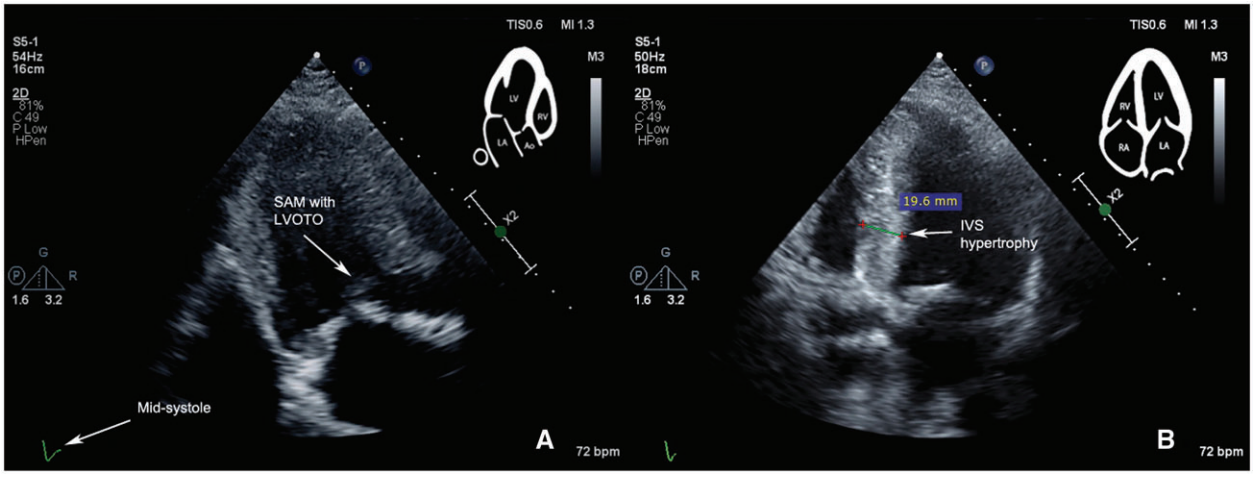
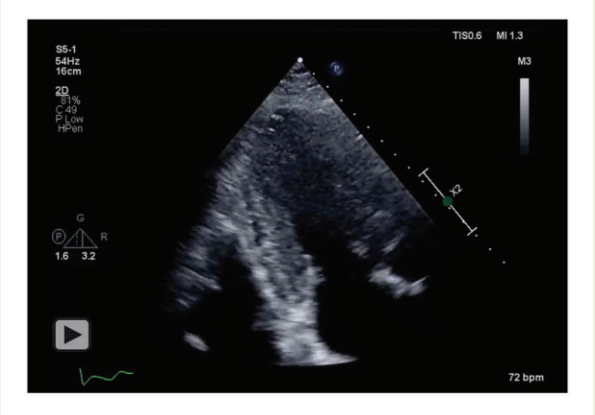


Figure 4 Postoperative urgent transthoracic echocardiogram. (*A*) Apical three-chamber view confirming presence of SAM. (*B*) Apical four-chamber view confirming magnitude of interventricular septum size (overestimated in A4c window).



Video 2 Postoperative urgent transthoracic echocardiogram. Loop in apical three-chamber view showing worsening SAM causing left ventricular outflow obstruction.

confirming a large calibre left anterior descending artery, free of disease. A subsequent TTE showing an akinetic basal septum, mild persistent SAM, a mild residual outflow tract gradient of 23 mmHg, and aortic valve mean gradient of 7 mmHg (*Figure 5* and *Video 3*).

Despite the improvement in LVOT obstruction, the patient had progressive end-organ deterioration with ischaemic changes on liver function tests an acute kidney injury and a rising lactate level. Monitoring of troponin levels was also commenced following alcohol septal ablation until improvement was observed (Supplementary material online, *Table S1*).

Vasopressin was commenced to maintain an appropriate mean arterial pressure (MAP), and a Milrinone infusion was trialled to assist with inotropy. Given the ongoing poor cardiac indices peripheral veno-arterial extracorporeal membrane oxygenation (VA-ECMO) was instituted to optimize systemic perfusion whilst awaiting improvement of the patient's cardiac function. After 72 h, end-organ dysfunction had improved, and a TOE weaning study was performed

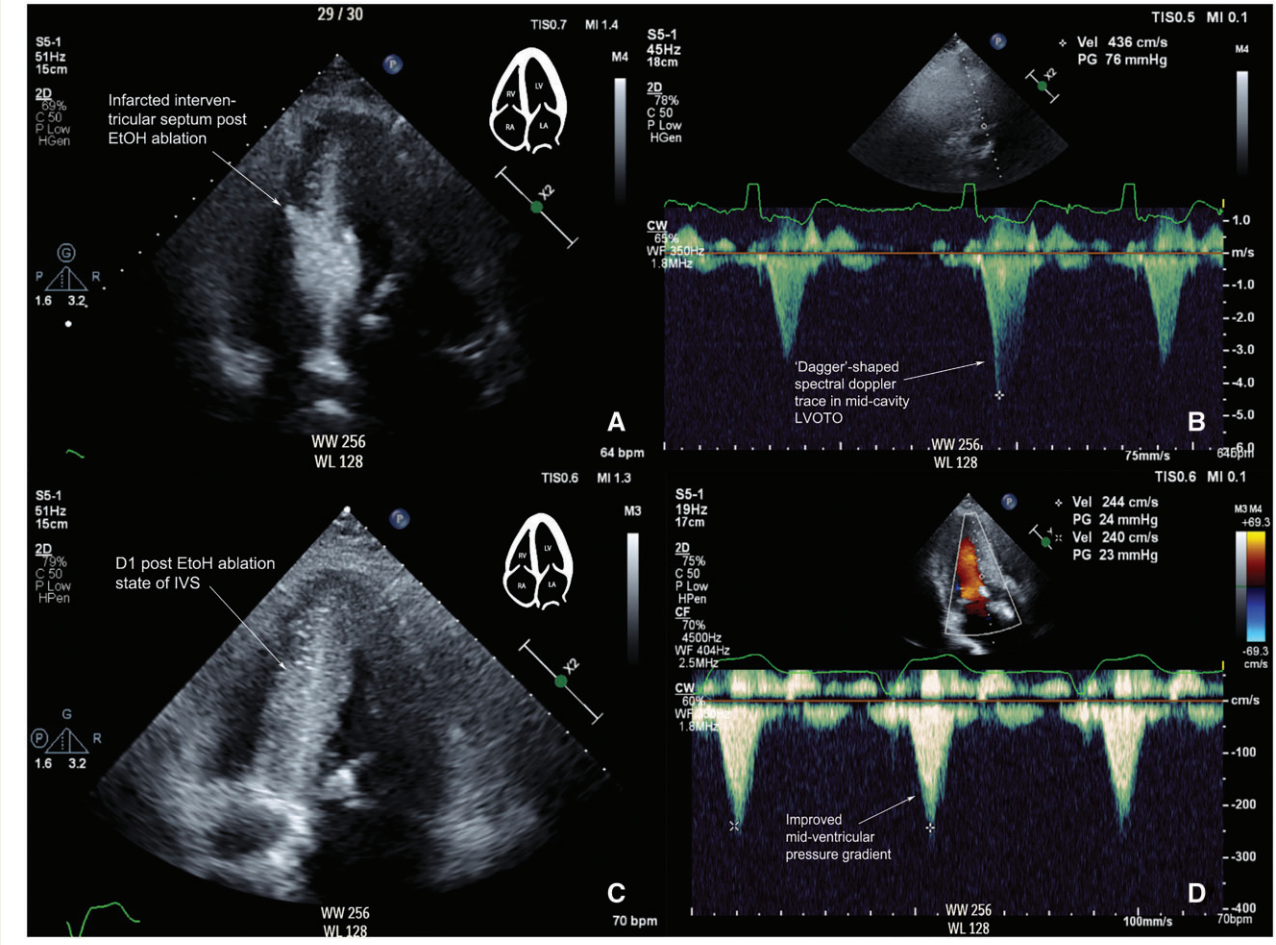
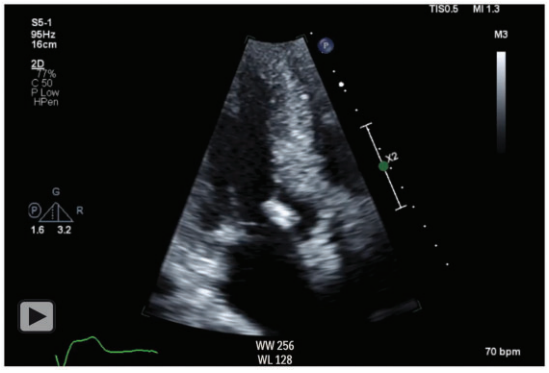


Figure 5 Comparison transthoracic echocardiograms intra- and post-alcohol septal ablation therapy. (*A*) Immediate post-ablation view of interventricular septum. (*B*) Spectral Doppler view with marked peak gradient at time of ablation therapy. (*C*) D1 post-ablation view of interventricular septum. (*D*) Spectral Doppler view with improved pressure gradient D1 post-ablation therapy.



Video 3 D1 post-alcohol septal ablation transthoracic echocardiogram. Loop in apical three-chamber view showing akinetic basal septum, and mild persistent SAM.

with measurement of LVOT gradient at decreasing VA-ECMO flows down to 1.0 L/min, with no SAM or mitral regurgitation observed.

No formal, regular TTE monitoring was performed, whilst the patient was on VA-ECMO, with a single scan performed D1 post-commencement, and otherwise, a subsequent scan performed just prior to a formal TOE weaning study (Supplementary material online, *Video S1*). Haemodynamic monitoring in this setting was achieved with cardiac output readings from the pulmonary arterial thermodilution catheter, and regular arterial and mixed venous blood sampling.

The formal weaning study was commenced after VA-ECMO flow had demonstrably been weaned to approximately half of initial flow (2.81 L/min) with no need for vasopressors/inotropes. At this time a TOE probe was inserted and extracorporeal membrane oxygenation (ECMO) flow was decreased steadily over a 40 min period to 1.0 L/min. Throughout this time haemodynamic parameters such as MAP, central venous pressure, end-tidal CO_2, were monitored, with cardiac output measured by both thermodilution pulmonary artery catheter technique, and calculated via TOE measurements (with documented LVOT VTI, LVOT gradient, and stroke volume). Once ECMO flow had been decreased to 1.0 L/min, only minor chordal SAM was noted and no need for vasopressors/inotropy was confirmed, then ECMO flow was brought back to ~2.8 L/min and the patient's case was discussed with the Cardiothoracic Surgeon. A formal decision was then made to decannulate from ECMO in the operating theatre.

The patient was decannulated and subsequently achieved a CI of 2.5 L/min/m^2, requiring only a further 24 h of vasopressor support (Supplementary material online, *Table S2*).

A final inpatient TTE was performed 3 days post-wean of ECMO, showing a cardiac output of 8.0 L/min, persistent mild SAM with a residual LV gradient of 18 mmHg, dyskinetic basal septum consistent with prior alcohol septal ablation, and aortic valve gradient mean of 9 mmHg with no paravalvular leak identified (Supplementary material online, *Figure S1*).

The patient's ICU stay was prolonged by intermittent episodes of AF and delirium. He was discharged from the ICU at D13 postoperatively, and from the hospital at D19 postoperatively. At 2 months postoperatively, the patient had essentially returned to baseline function. Echocardiographic follow-up post-discharge was delayed due to the COVID-19 pandemic, and at the time of writing is not available.

Discussion

The demonstration of high intracavity gradients after aortic valve replacement has been identified in the surgical literature for decades, and in severe cases can resemble hypertrophic obstructive cardiomyopathy.[3] The expansion of TAVR in late-stage aortic stenosis and particularly in small elderly women with small ventricular cavities has highlighted this postoperative complication. The term 'suicide ventricle' has been popularized and is an apt description of this potentially fatal physiology.

The cause of this intracavitary gradient is essentially two-fold. Dynamic LVOT obstruction can occur post-aortic valve replacement due to the development of SAM which, if eventuates following the weaning of bypass, can mimic the presentation of cardiac tamponade,[4] necessitating definitive imaging to diagnose. Alternatively, following relief of aortic stenosis, the contraction of the hypertrophic LV obstructs forward flow manifesting clinically as a low cardiac output state with severe haemodynamic instability.[5] In our case, the cause of dynamic LVOT obstruction was a mix of the above pathologies, with a profound SAM observed on echocardiography.

In cases of dynamic LVOT obstruction due to the above pathologies, medical management aims to optimize physiology, targeting specific risk factors which perpetuate the problem—namely hypovolaemia, vasodilatation, and increased blood flow velocity in the LVOT.[6] Medical approaches include sufficient volume loading, titrated doses of beta-blockers to promote negative chronotropy and inotropy, and the administration of alpha-1 agonists to decrease the LVOT pressure gradient through increasing systemic vascular resistance.[7] Where medical therapy is unable to adequately relieve the LVOT obstruction, procedural management can be undertaken to optimize anatomy. SAM post-SAVR leading to such severe haemodynamic instability remains a rare occurrence, more frequently being described in the post-MVR and TAVR population, and as such there is currently no consensus about definitive management should medical therapy fail.[8] Surgical myectomy or alcohol septal ablation therapy remain the primary strategies, with the latter reserved for patients at a higher risk of negative outcomes from open heart surgery.[6] Results following these procedures usually show immediate reduction in LVOT gradient with associated improvement in symptoms over the short- and long-term.[9–11] Ongoing cardiogenic shock following the relief of LVOT obstruction is not well described in the literature and there is a paucity of data regarding its management.

Extracorporeal membrane oxygenation therapy has been sparsely documented as a rescue strategy for severe, refractory cardiogenic shock in post-TAVR patients.[12,13] Extrapolating from these cases, we initiated supportive management with ECMO to ameliorate perfusion whilst awaiting improvement of cardiac function following resolution of the patient's LVOT obstruction.

Although our hospital institution did not have a formal protocol for ECMO weaning, the process was similar to that described by Cavarocchi *et al.*,[14] with the notable differences that the trial only began once flow had been steadily weaned to half of initial flow, and

ECMO flow was kept steady at 15 min intervals whilst weaning to 1.0 L/min. No further fluid bolus was required during the study, as haemodynamic parameters remained overall stable. Utilization of TOE real-time monitoring whilst weaning VA-ECMO remains a valuable instrument for accurate determination of cardiac output, as pulmonary arterial catheter measurement alone can be rendered inaccurate due to the inherent assumptions that blood flow remains constant throughout the entirety of the circuit.[15]

It remains unclear why our patient's cardiac output did not improve more quickly following definitive resolution of the LVOT obstruction. It is likely that the preceding period of low cardiac output state had resulted in reversible ischaemic damage and myocardial stunning. The initiation of ECMO and immediate optimization of the cardiac output state therefore may have assisted to improve the intrinsic cardiac function, preventing further end-organ damage.

Conclusion

We have described an unusual case of LV suicide post-SAVR, persisting following targeted therapy, necessitating ECMO support. It is important to have a low threshold to investigate for LVOT obstruction in the haemodynamically unstable postoperative SAVR who has not undergone perioperative myectomy. Timely echocardiography, targeted medical therapies, and escalation to definitive procedural interventions are imperative to the successful management of this complication. Ongoing cardiogenic shock and subsequent end-organ dysfunction can occur following definite therapy. Extracorporeal membrane oxygenation should therefore be considered as a rescue therapy in the appropriate patient population to allow for improvement in cardiac function following the above treatment strategies.

Lead author biography

Dr Peter Andrew Lioufas graduated from Monash University in 2015. He is an Intensive Care Registrar at the Epworth Richmond Hospital, with an interest in cardiac critical care and trauma medicine. He was accepted as an accredited trainee to the College of Intensive Care Medicine of Australia and New Zealand in 2020 and is in the process of completing a Certificate in Clinician Performed Ultrasound in Rapid Cardiac Echocardiography through the Australasian Society for Ultrasound Medicine.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for the submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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