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CASE REPORT
Heart failure

Dynamic left ventricular outflow tract gradient resulting from Takotsubo cardiomyopathy ameliorated by intra-aortic balloon pump counterpulsation: a case report

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Background Takotsubo cardiomyopathy is a variant of acute coronary syndrome with characteristic acute left ventricular apical ballooning. Uncommonly, there can be associated left ventricular outflow tract (LVOT) obstruction causing cardiogenic shock refractory to inotropic support. The use of afterload-reducing mechanical support such as intra-aortic balloon pump (IABP) counterpulsation is not routinely employed in instances of this kind.

Case summary In our case report, we describe a 66-year-old female with acute Takotsubo cardiomyopathy and associated LVOT obstruction which failed to respond to high-dose dobutamine and whose clinical trajectory was worsened by fast atrial fibrillation with rapid ventricular response. Within 24 h of admission, the patient had an IABP placed which rapidly improved her haemodynamics. Two days later, IABP was removed and within 6 days of admission, apical ballooning and LVOT obstruction had fully recovered.

Conclusion We recommend early use of mechanical support with IABP counterpulsation to expedite recovery in patients with acute Takotsubo cardiomyopathy with associated LVOT obstruction.

Keywords Takotsubo • Cardiomyopathy • Outflow • Tract • Obstruction • Balloon • Pump • Recovery • Case report

Learning points

- Takotsubo cardiomyopathy can rarely associate with left ventricular outflow tract (LVOT) obstruction and cardiogenic shock.
- Inotropes are usually avoided in the setting of cardiogenic shock related to acute LVOT obstruction. However, indicated therapies such as β-blockers, phenylephrine, and fluids can also prove difficult to administer in the setting of profound hypotension and pulmonary oedema.
- Intra-aortic balloon pump placement can reduce afterload, thereby ameliorating LVOT obstruction, and facilitate the administration of other recommended medical treatments to help alleviate cardiogenic shock.

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Introduction

Takotsubo cardiomyopathy or apical ballooning syndrome is a well-recognized presenting variant of acute coronary syndrome.[1] Typically, left ventricular (LV) apex and mid-ventricular segments become profoundly hypokinetic with hyperdynamic basal segments. Although generally associated with a good long-term prognosis with reported inpatient mortality rates of <5%,[2] initial presentation can involve severe cardiogenic shock requiring complex cardiac supportive measures. Infrequently, the clinical course is complicated by dynamic left ventricular outflow tract (LVOT)[3,4] obstruction. In this scenario, flow acceleration in the LVOT due to differential wall-motion abnormalities (WMAs) leads to a Venturi effect causing systolic anterior motion (SAM) of the mitral valve (MV) exacerbating LVOT obstruction and haemodynamic compromise. We present a case report of a significant LVOT obstruction in the setting of acute Takotsubo cardiomyopathy with cardiogenic shock in which the use of intra-aortic balloon pump (IABP) counterpulsation was associated with rapid clinical stabilization.

Timeline

Presentation	Acute chest pain, troponin rise, and electrocardiogram changes suspicious for acute coronary syndrome
4 h	Ongoing cardiogenic shock, commenced on dobutamine and referred for coronary angiography
5 h	Normal epicardial coronary arteries and severe left ventricular outflow tract (LVOT) gradient 60 mmHg, echocardiogram shows left ventricular ejection fraction (LVEF) 30% with LVOT gradient 40 mmHg
24 h	Atrial fibrillation with rapid ventricular response requiring electrical cardioversion and amiodarone infusion, dobutamine continued, ongoing worsening cardiogenic shock
26 h	Intra-aortic balloon pump placement and dobutamine discontinued
27 h	Improved haemodynamics
72 h	Intra-aortic balloon pump removal
6 days	Left ventricular outflow tract gradient recovered and LVEF completely normalized on echocardiography, patient discharged

Case presentation

A 66-year-old lady with a history of hypertension presented to the hospital following an acute episode of central chest discomfort and dyspnoea following strenuous garden work. She reported recent emotional stress following the recent death of a family member approximately 2 weeks earlier. Initial electrocardiogram (ECG) showed sinus rhythm with widespread ST depression, deep T-wave inversion, absence of significant Q-waves, and a prolonged corrected QT-interval (QTc) of 520 ms (*Figure 1*). High-sensitivity cardiac troponin I (hsTnI) was 670 ng/L and N-terminal pro-brain natriuretic peptide was 1437 ng/L. There was no clinical or biochemical evidence of sepsis, acute anaemia, pulmonary embolism, or raised intracranial pressure. Polymerase chain reaction for SARS-CoV-2 was negative at admission and again at 48 h. She was commenced on intravenous dobutamine due to evolving cardiogenic shock and referred to our institution for urgent coronary angiography.

Coronary angiography demonstrated patent epicardial coronary vessels, elevated left ventricular end-diastolic pressure of 35–40 mmHg and a significant pullback gradient of 60 mmHg with a focal 35–40 mmHg gradient localized within the sub-aortic LVOT (*Figure 2*). Transthoracic echocardiography on dobutamine showed akinesis of the LV apex, anteroapical, and inferoapical segments with hyperdynamic basal segments, left ventricular hypertrophy with an interventricular septal thickness of 1.5 cm, a left ventricular ejection fraction (LVEF) of 30%, SAM of the MV, and an LVOT gradient of 40 mmHg (*Figure 3*).

The patient was transferred to the intensive care unit where she initially remained on dobutamine 5 cmg/kg/min. Twelve hours later, she developed paroxysmal atrial fibrillation with rapid ventricular response, which failed to cardiovert following direct current cardioversion, but eventually spontaneously cardioverted with the administration of intravenous amiodarone using a 300 mg bolus and 900 mg/24 h infusion. Due to further clinical deterioration and worsening cardiogenic shock with mean arterial pressure of 50 mmHg and central venous pressure 30–40 mmHg, dobutamine was discontinued, a noradrenaline infusion was commenced, and an IABP was placed (*Figure 4*). This resulted in rapid improvement in the patient's clinical condition with blood pressure now 110/65 mmHg, mean arterial pressure 75 mmHg, and central venous pressure 10–15 mmHg. Within 48 h, IABP was weaned and removed with preserved haemodynamics. Repeat transthoracic echocardiography 6 days after admission showed complete normalization of LV function (LVEF 55–60%) and no residual LVOT obstruction (*Figure 5*). The patient was discharged 1 week after presentation on rivaroxaban 20 mg o.d. for paroxysmal atrial fibrillation with a CHA_2DS_2VASc score of 4 in addition to ramipril 2.5 mg b.i.d. and bisoprolol 2.5 mg o.d. as anti-remodelling agents. When last reviewed 6 weeks following hospital discharge the patient remained well, ECG had completely normalized (*Figure 5*), and she had resumed normal daily activities.

Discussion

The management of dynamic LVOT obstruction in the setting of Takotsubo cardiomyopathy remains a challenge in real-world settings. The presence of LVOT obstruction should prompt considerable changes in management such as avoidance of typical inotropes like dobutamine, dopamine, and nor-adrenaline[5] and employment of less commonly used acute measures such as the use of up-front cardioselective β-blockers, α-agonists such as phenylephrine and fluid resuscitation.[6] However, these strategies may prove impractical in the setting of cardiogenic shock and pulmonary congestion, which

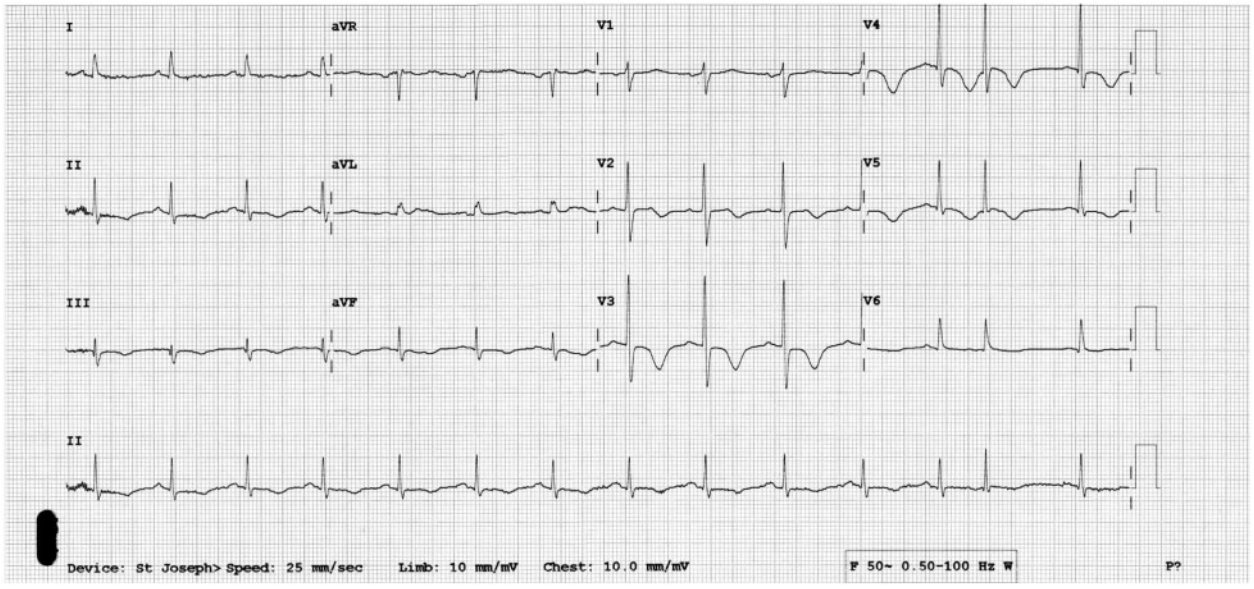

Figure 1 Twelve-lead electrocardiogram showing diffuse ST-segment depression, T-wave inversion, and QTc prolongation.

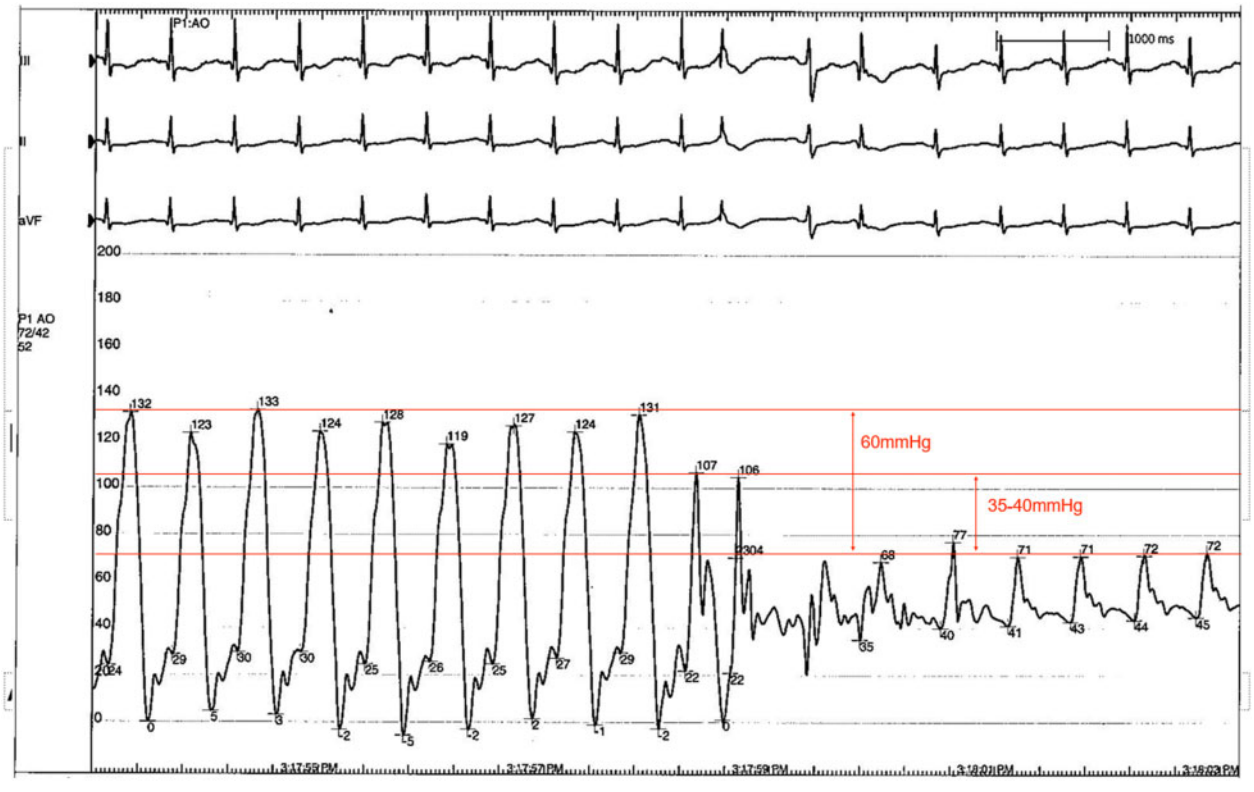

Figure 2 Haemodynamic waveform during catheter pullback across aortic valve showing significant left ventricle to aorta gradient of 60 mmHg with focal gradient of 35–40 mmHg within the sub-aortic left ventricular outflow tract.

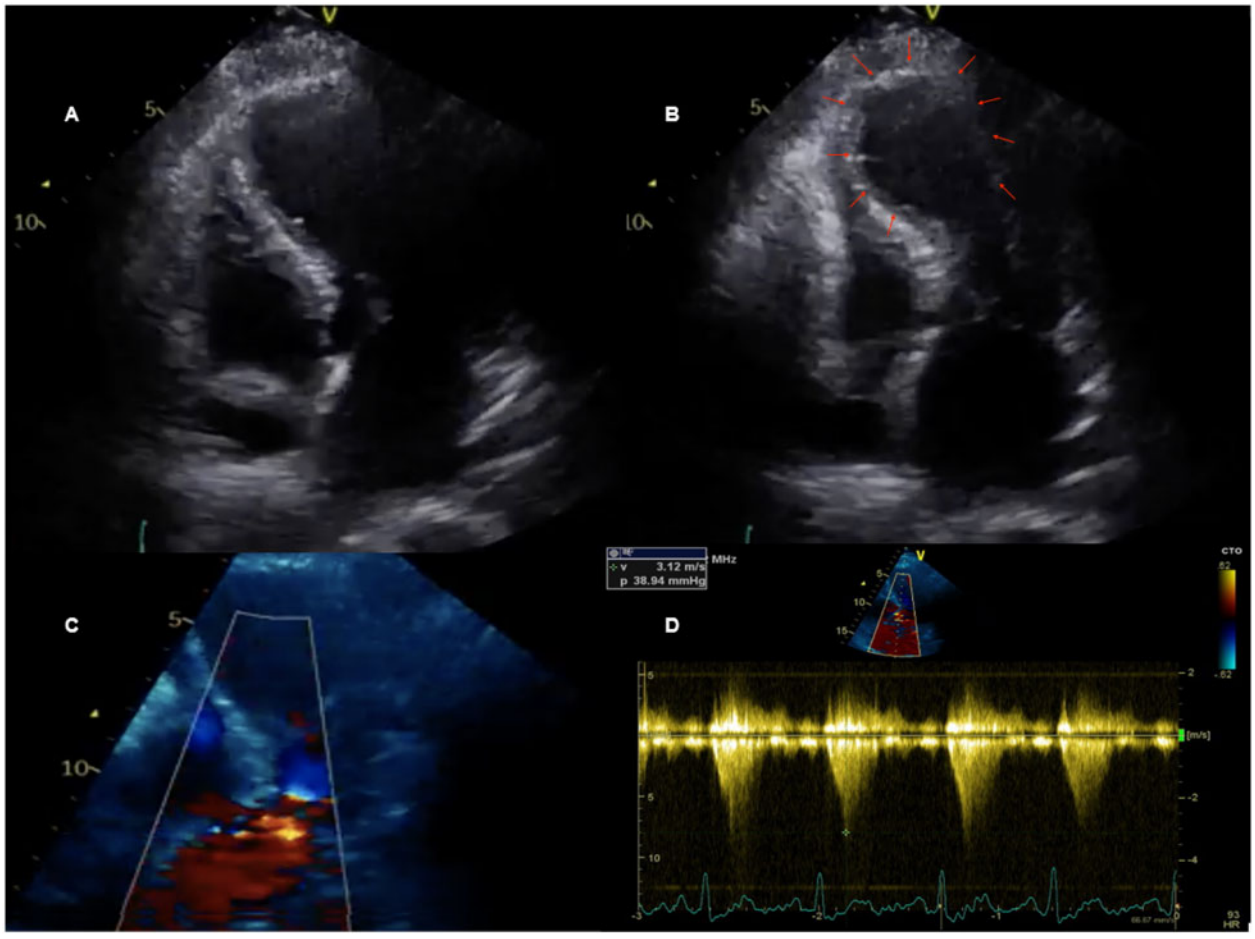

Figure 3 Transthoracic echocardiography during dobutamine infusion; (*A*) apical four-chamber view during left ventricular diastole; (*B*) apical four-chamber view during left ventricular systole showing severe apical ballooning (red arrows) with hyper contractile basal segments; (*C*) colour-flow Doppler showing significant left ventricular outflow tract turbulence; (*D*) continuous wave Doppler in the left ventricular outflow tract showing peak pressure gradient of 40 mmHg.

often accompany acute Takotsubo cardiomyopathy. In our case, dobutamine likely exacerbated the degree of LVOT obstruction due to worsening SAM of the MV and narrowing of the LVOT through heightened catecholaminergic basal hypercontractility.[7] It may have also contributed to the development of atrial fibrillation which can have a dual insult of worsening adrenergically-mediated microvascular ischaemia of the LV apex[8] and rate-related loss of cardiac myocyte contractility. Amiodarone administration can also be hazardous in acute Takotsubo cardiomyopathy due to its effects on QTc prolongation and heightened risk of polymorphic ventricular tachycardia.[9]

The optimal management of Takotsubo cardiomyopathy complicated by LVOT obstruction is a matter of some debate. On one hand, α-adrenergic agents such as phenylephrine are recommended as they diffusely increase afterload, incentivizing stronger myocyte contraction.[10] On the other hand, an increase in afterload results directly from the LVOT obstruction,[11] an effect which exaggerates the longitudinal dyssynchrony between basal and apical segments, thus worsening systolic wall stress and LV stroke volume. From this perspective, IABP afterload *reduction* becomes more desirable, and the negative aortic pressure generated during systolic deflation may in fact encourage a greater fraction of blood in the LV to exit the aorta in a more laminar fashion, especially in hypotensive patients where LVOT geometry can be further distorted by functional hypovolaemia. Additionally, IABP afterload reduction reduces the need for acute diuresis and allows for fluid resuscitation and the use of phenylephrine.

The role of alternative strategies for mechanical support is unclear,[12] though case reports support improvement with microaxial left ventricular assist device.[13] In our case, IABP support showed an immediate clear benefit and may represent the treatment of choice in patients with Takotsubo cardiomyopathy-induced LVOT obstruction where fluids, β-blockers, and phenylephrine have proven either ineffectual or unusable.

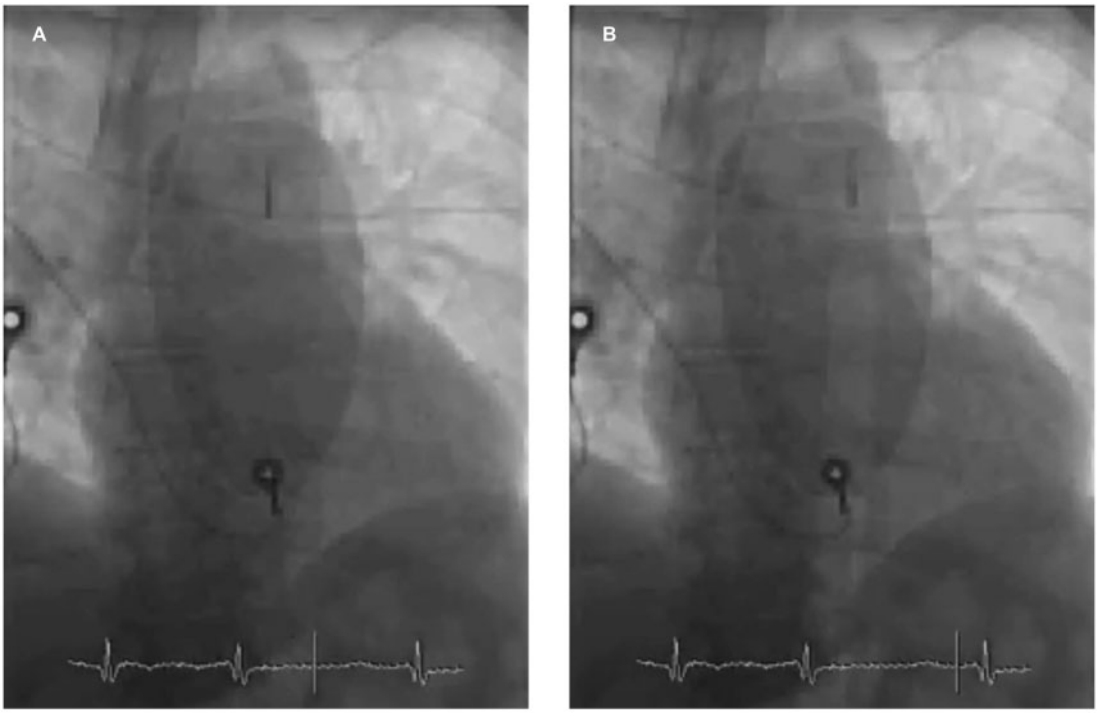

Figure 4 Intra-aortic balloon pump placement; (*A*) intra-aortic balloon pump positioning in the proximal descending aorta just distal to the left subclavian artery; (*B*) inflated intra-aortic balloon pump counterpulsation with electrocardiogram-triggering.

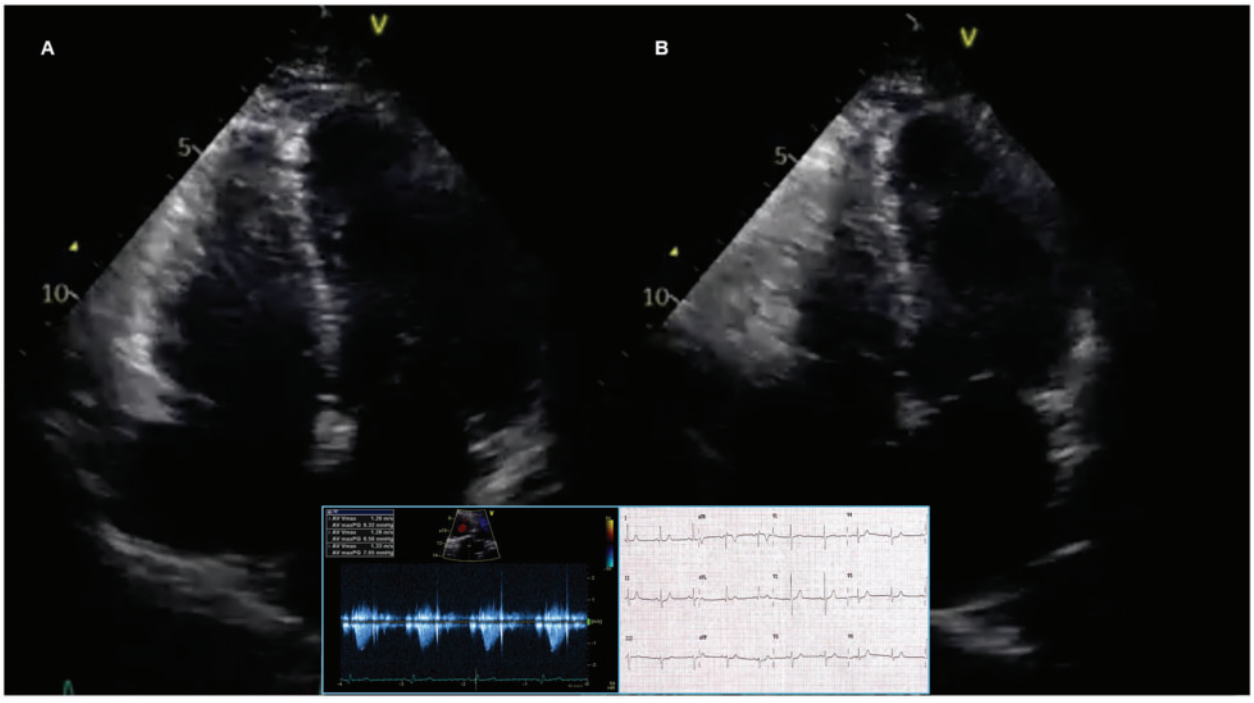

Figure 5 Repeat transthoracic echocardiography on Day 6 of admission; complete left ventricular systolic function recovery shown in diastole (*A*) and systole (*B*); left ventricular outflow tract gradient has dropped to 7 mmHg on continuous wave Doppler (inset left); 12-lead electrocardiogram at 6-week follow-up showing recovered T-wave changes and QTc of 470 ms (inset right).

Conclusion

We report a case of a patient with severe cardiogenic shock in the setting of acute Takotsubo cardiomyopathy with associated LVOT obstruction refractory to medical therapy and worsened by inotropic support who benefitted from transient intra-aortic balloon pump counterpulsation and who has since made a full recovery. Intra-aortic balloon pump should be considered in Takotsubo cardiomyopathy and dynamic LVOT obstruction as a temporizing means of allowing cardiac recovery and may even prove ameliorative of outflow tract turbulence in some instances.

Lead author biography

Dr Jim O'Brien is a Cardiology Clinical Fellow.

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 these cases and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for 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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