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CASE REPORT

Opposing forces of cardiogenic shock: left ventricular outflow obstruction, severe mitral regurgitation, and left ventricular dysfunction in Takotsubo cardiomyopathy

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Abstract

Rates of stress (Takotsubo) cardiomyopathy have increased during the coronavirus pandemic due to social stressors, even in patients who are not infected with the virus. At times, Takotsubo cardiomyopathy (TC) may present as cardiogenic shock. Herein, we present a case during the pandemic of shock from TC secondary to left ventricular outflow tract obstruction (LVOTO), mitral regurgitation (MR), and left ventricular (LV) dysfunction. The contrasting management strategy of LVOTO, MR, and LV failure was cause for clinical challenge, and we highlight the balance of treating these opposing forces.

Keywords Takotsubo; Cardiogenic; Shock; Mitral regurgitation

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Introduction

The acute phase of Takotsubo cardiomyopathy (TC) is complicated by cardiogenic shock in 12% of patients, and patients with cardiogenic shock have six-fold higher in-hospital mortality.[1,2] The cause of shock in TC is secondary to systolic left ventricular (LV) failure, left ventricular outflow tract obstruction (LVOTO), severe mitral regurgitation (MR), or a combination of these factors.[3,4] The presence of multiple contributors to shock is cause for clinical equipoise because the physiologies of these processes call for disparate management strategies. In contrast to the afterload reduction with or without inotropy for the management of severe MR and LV failure, respectively, cardiogenic shock from LVOTO requires afterload augmentation and is often worsened by inotropy.

We present a case of a patient with TC resulting in cardiogenic shock that is illustrative of this balance of opposing physiologic processes. Her concomitant cocaine use provided an additional layer of complexity to management. Frequent echocardiographic and Swan-Ganz catheter measurements were used to guide dynamic management decisions. Ultimately, shock physiology improved, and the patient was discharged home in stable condition. Her cardiomyopathy resolved on follow-up imaging three months later.

Case Report

A 64-year-old female with history of hypertension and hyperlipidaemia was brought to the emergency room by ambulance as a 'STEMI alert' with acute onset of chest pain about 30 min prior to arrival to the hospital. Social history was remarkable for tobacco use and cocaine abuse. She reported last cocaine use was 1 week prior; however, urine drug screen on arrival was positive for cocaine. Coronavirus testing was negative, and initial troponin-T level was 0.09 mg/mL.

Physical examination was significant for an apical systolic murmur, tachycardia, and an initial mean arterial pressure (MAP) of 84 mmHg. Chest pain persisted and MAP fell to 50 mmHg after the administration of sublingual nitroglycerin. Her initial ECG showed an ectopic atrial rhythm with diffuse

ST elevation without reciprocal depression, and bedside point of care ultrasound revealed apical and mid- LV akinesis (*Figure 1*). The patient was started on norepinephrine and was taken emergently for cardiac catheterization (50 min after the onset of symptoms), which revealed non-obstructive coronary disease. An LV end diastolic pressure (LVEDP) was measured at 30 mmHg, and a left ventriculogram revealed a hyperdynamic base, apical akinesis and severe MR (Supporting Information, *Video S1*), clenching the diagnosis of TC, which was thought to be precipitated by her cocaine use and psychosocial stressors during the coronavirus pandemic. A summary of the angiogram findings is displayed in *Figure 2*.

Due to the concerns for LVOTO, norepinephrine was replaced with vasopressin, and the patient was transferred to the cardiac critical care unit. The inotropy from norepinephrine in addition to the patient's cocaine use likely exacerbated a hyperdynamic base, systolic anterior motion (SAM) of the mitral valve, and LVOTO physiology. Despite usual management of LVOTO with fluid administration, this was avoided given elevated LVEDP. Although TC was elucidated as the primary cause of shock, beta blockade was avoided in the setting of recent cocaine use. An echocardiogram demonstrated a reduced LV ejection fraction with apical and mid-LV akinesis and a hyperdynamic base (Supporting Information, *Video S2A*). SAM of the mitral valve was noted in the setting of LVOTO and a pressure gradient across the LVOT was measured at 64 mmHg (Supporting Information, *Video S2B*). Concomitant severe MR was visualized by color and continuous wave Doppler (Figure 3, Supporting Information, *Video S2C*).

Due to the competing haemodynamics, a Swan-Ganz catheter was placed at the bedside for tailored therapy. Initial filling pressures revealed a pulmonary wedge pressure of 20 mmHg, pulmonary artery (PA) saturation of 36%, cardiac index of 2 L/min/m^2 by Fick and a systemic vascular resistance (SVR) of 1623 dynes/seconds/cm^{-5}. Despite high filling pressure, fluid removal was avoided given concerns for the potential unwanted effect of worsening LVOTO. Inotropy for dysfunctional LV was also deferred due to the possibility of increasing basal contractility and exacerbating LVOTO. MAP declined further. Since LVOTO was thought in part to contribute to her cardiogenic shock, phenylephrine was considered, however fear of worsening MR and LV dysfunction with pure afterload and given recent cocaine use prompted optimization with vasopressin, which was cautiously increased. Despite her complicated haemodynamics, the patient was resting comfortably on room air.

Several hours later, however, the patient began to develop respiratory distress. Her filling pressures were noted to increase further; careful diuresis was initiated with good effect and without hypotension. The patient's condition stabilized. Over the next day, pressors were slowly weaned off and beta blockade was introduced. The addition of a beta blocker with an overlay of recent cocaine use resulted in an asymptomatic increase in SVR to 2300 dynes/seconds/cm^{-5} which

Figure 1 Initial electrocardiogram showing ectopic atrial rhythm and diffuse ST segment elevation without reciprocal depression.

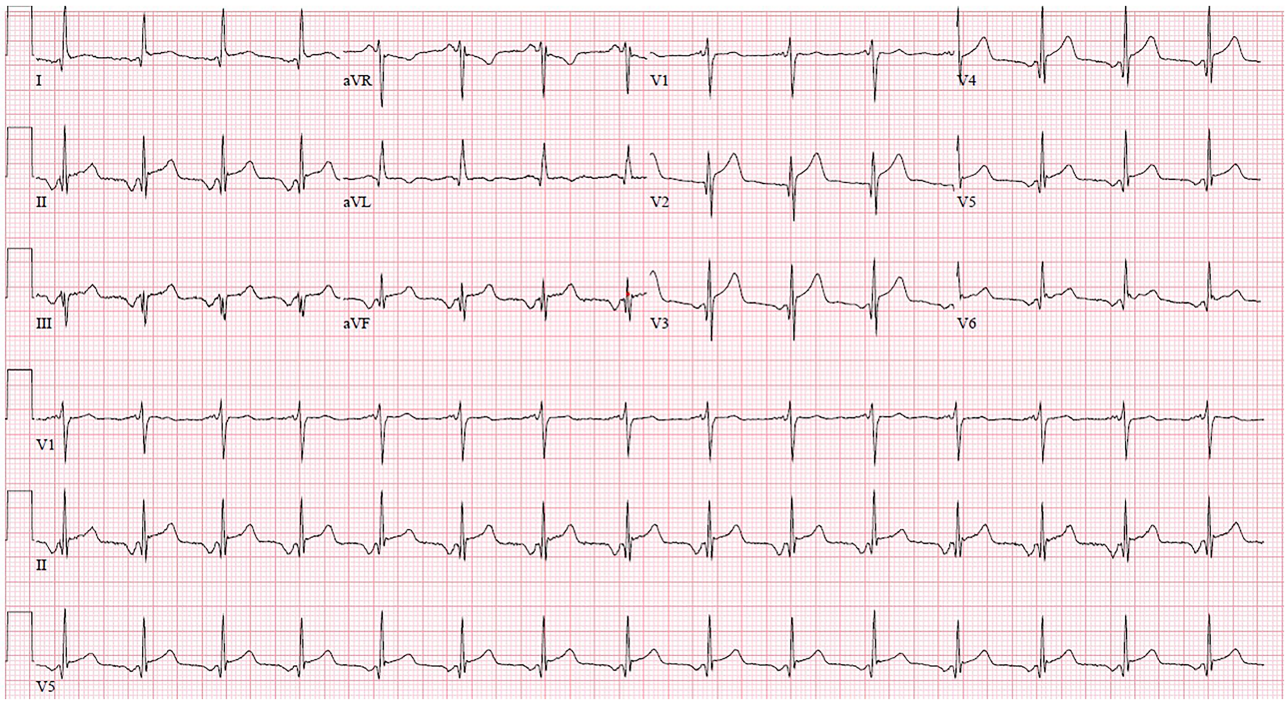

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Figure 2 Coronary angiogram and left ventriculogram findings in Takotsubo cardiomyopathy. (A) Select injection of the right coronary artery in the RAO view demonstrating mild non-obstructive disease. (B) Select injection of the left coronary artery in the LAO-CAU view demonstrating mild non-obstructive disease of the LAD. (C) Left ventriculogram illustrating apical ballooning, suggestive of Takotsubo cardiomyopathy in the setting of non-obstructive coronary disease. RAO, right anterior oblique; LAO, left anterior oblique; CAU, caudal; LAD, left anterior descending.

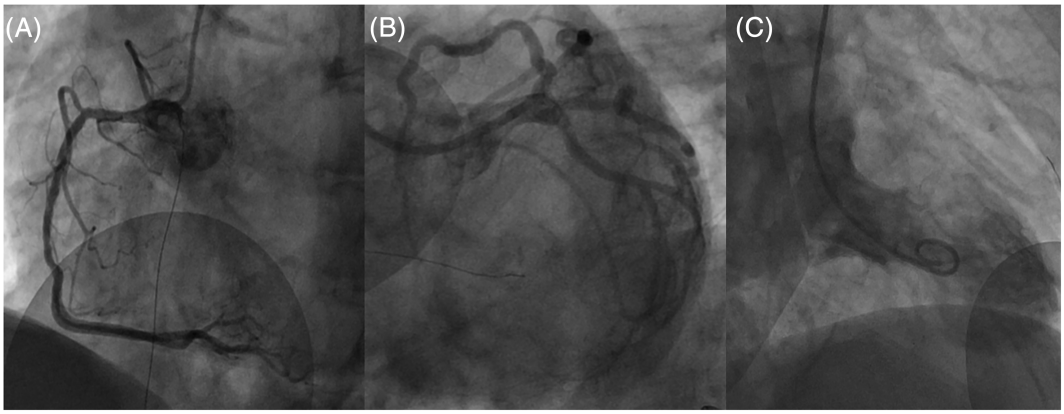
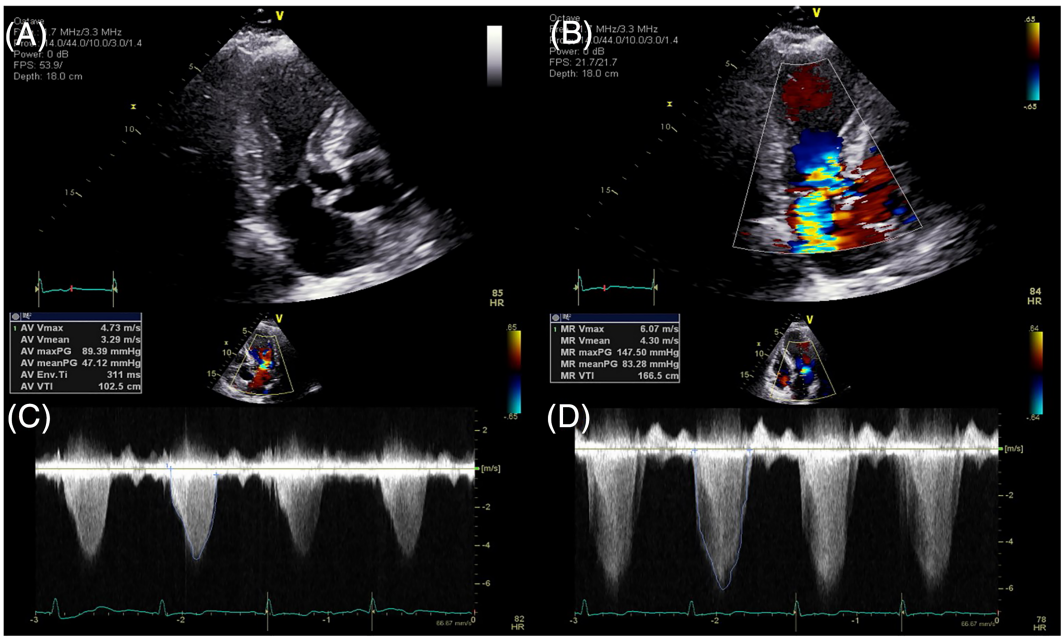


Figure 3 Echocardiographic findings demonstrating severe mitral regurgitation in the setting of LVOTO. (A) Apical 3 chamber view with mid- left ventricular akinesis, apical ballooning, hyperdynamic base and evidence of systolic anterior motion of the mitral valve. (B) Colour Doppler showing posteriorly directed mitral regurgitant jet and flow acceleration through the LVOT. (C) Continuous wave Doppler through LVOT demonstrating classical 'dagger shaped' envelope seen in LVOTO. (D) Continuous wave Doppler across the mitral valve showing early systolic flow suggesting regurgitation as opposed to LVOTO. LVOT, left ventricular outflow tract; LVOTO, left ventricular outflow tract obstruction.



improved with the addition of oral afterload reduction. Repeat echocardiogram the next day demonstrated persistent mid-apical akinesis but a reduced LVOT gradient, mild MR, and absence of SAM (Supporting Information, *Video S3A,B*). An outpatient follow-up echocardiogram three months later shows normalized LVEF and only persistent mild MR (Supporting Information, *Video S4A,B*).

Discussion

Management of cardiogenic shock in the setting of competing physiological processes requires a complete understanding of haemodynamics in order to appropriately manage this complicated presentation. In this example, despite the presence of severe MR, afterload was haemodynamically

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tolerated because of the haemodynamic contribution from concomitant severe LVOTO, and the initiation of afterload augmentation with vasopressin improved LVOT gradients. Usual treatment of individual pathologic states of TC and LVOTO as Conradi *et al*. highlight was limited because of simultaneous processes: treatment of TC with beta blockade was initially avoided because of recent cocaine use, and treatment of shock from LVOTO with fluid administration and pure afterload was avoided because of the systolic dysfunction and already high LVEDP.[5] In a case of isolated LVOTO, preload augmentation might aid in shock resolution to reduce LVOT gradient and obstruction, but the additional haemodynamic data pointed to an already overloaded ventricle. Additionally, afterload augmentation with phenylephrine was a consideration because of the possibility to help 'tent open' the LVOT to reduce obstruction; this, however, may have exacerbated the concomitant processes of MR, LV failure, and perhaps might have little benefit because of the saturated alpha receptor recruitment from recent cocaine use. Although Rangel *et al*. and Lopez *et al*. highlight the safety of beta-blocker use in acute chest pain related to cocaine and systolic heart failure with active cocaine use in their retrospective reviews, the risk in acute decompensation/cardiogenic shock gave additional pause to use up front.[6,7] Although a temporary LV assist device was a viable bailout option, Swan-Ganz data allowed for tailored medical therapy. Ultimately after resolution of shock, subsequent diuresis and beta blockade improved both the MR and systolic dysfunction. The information provided by the Swan-Ganz catheter was helpful in the timing and initiation of treatment.

This vignette highlights the clinical equipoise necessary for management of cardiogenic shock in TC. Recognizing the delicate balance in this rare cause of cardiogenic shock is critical for successful management.

Conflict of interest

None declared.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Video S1. Left ventriculogram illustrating apical ballooning and severe mitral regurgitation.
Video S2. Transthoracic echocardiogram demonstrating Takotsubo cardiomyopathy, severe mitral regurgitation, and left ventricular outflow tract obstruction.
Video S2A. Apical 4-chamber view showing hyper dynamic base with mid- ventricular and apical akinesias.
Video S2B. Apical 3-chamber view demonstrating systolic anterior motion of the mitral valve.
Video S2C. Apical 3-chamber view with color Doppler revealing severe mitral regurgitation and accelerated flow through the left ventricular outflow tract.
Video S3. Repeat transthoracic echocardiogram after treatment and resolution of shock.
Video S3A. Apical 4-chamber view showing resolution of systolic anterior motion of the mitral valve.
Video S3B. Apical 5-chamber view revealing improvement in mitral regurgitation.
Video S4. Repeat transthoracic echocardiogram 3 months after discharge.
Video S4A. Apical 4-chamber view demonstrating recovered left ventricular function and resolution of wall motion abnormalities.
Video S4B. Apical 4-chamber view revealing only minimal mitral regurgitation.

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