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CASE REPORT

INTERMEDIATE

CLINICAL CASE

Impella Use in Cardiogenic Shock Due to Takotsubo Cardiomyopathy With Left Ventricular Outflow Tract Obstruction

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ABSTRACT

Cardiogenic shock (CS) due to Takotsubo cardiomyopathy (TCM) is often managed with cautious fluid administration and inotropic support; however, the co-existence of a left ventricular outflow tract obstruction (LVOTO) can complicate this management approach. This report describes a case of CS due to TCM and LVOTO. It was successfully managed with the Impella 2.5. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2019;1:161–5) © 2019 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 90-year-old woman presented with a syncopal episode while visiting a relative in the hospital; it lasted a few seconds with a quick recovery. The episode was preceded by dizziness, but she denied chest pain or palpitations. Her initial blood pressure was 110/54 mm Hg, her heart rate was 72 beats/min, her respiratory rate was 17 breath/min, and she had saturation of 94% on room air. On cardiovascular physical examination, a loud systolic murmur was audible at the right sternal border. The rest of the physical examination, including the central nervous system examination, was unremarkable. From her past medical history, the patient had essential hypertension and took amlodipine 10 mg/day.

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LEARNING OBJECTIVES

- To reflect on the pathophysiology of LVOTO in TCM.
- To recognize the challenges of managing cardiogenic shock in the setting of TCM with LVOTO.
- To reflect on the uses of an Impella device as a temporary LVAD in certain settings.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of syncope is extensive; sudden episodes like this with rapid and full recovery could be due to cardiac arrythmias, severe aortic stenosis, or a massive pulmonary embolism with persistent hypotension. Postural hypotension is possible in the correct setting, whereas a vasovagal event is less likely in the absence of typical symptoms.

INVESTIGATIONS

Her laboratory workup, including complete blood count, electrolytes, and renal function, were normal. The electrocardiogram showed nonspecific ST-T changes, and troponin I was elevated at 2.62 ng/ml. The chest x-ray and a computed tomography of the head demonstrated no acute changes.

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ABBREVIATIONS AND ACRONYMS

EF = ejection fraction

IABP = intra-aortic balloon pump

LV = left ventricle

LVAD = left ventricular assist device

LVOT = left ventricular outflow tract

LVOTO = left ventricular outflow tract obstruction

TCM = Takotsubo cardiomyopathy

TTE = transthoracic echocardiogram

SAM = systolic anterior motion

MANAGEMENT

She was treated as having a non–ST-segment elevation myocardial infarction; an urgent coronary angiogram was performed, which showed diffuse moderate nonobstructive coronary artery disease. Her left ventriculogram revealed impaired function and severe distal dyskinesia with a hyperdynamic base suggestive of Takotsubo cardiomyopathy (TCM) (Figure 1).

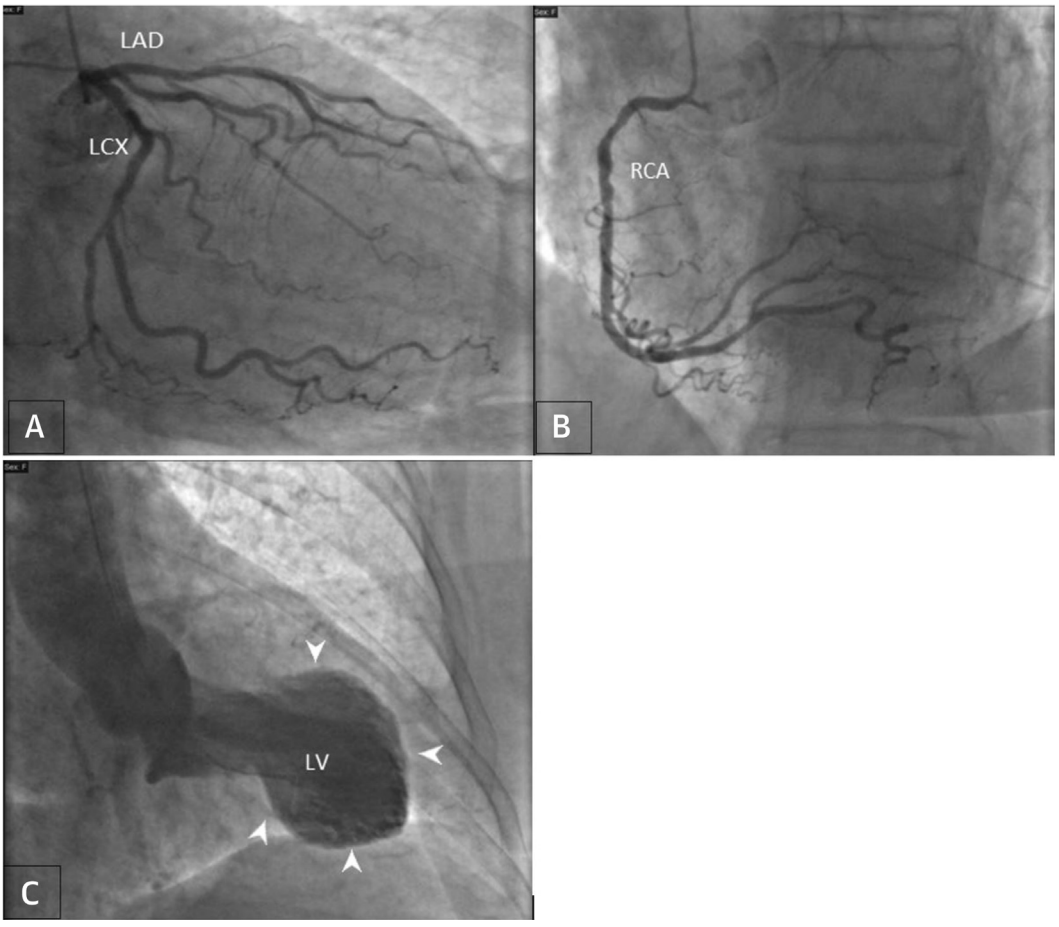
A transthoracic echocardiogram (TTE) showed an ejection fraction (EF) of 20% to 25% and a left ventricular outflow tract (LVOT) gradient of 100 mm Hg with systolic anterior motion (SAM) of the mitral valve (Figure 2).

Eight hours later, she became hypoxic and hypotensive. Despite beta-blocker and phenylephrine therapy combined with fluid resuscitation, she continued to be in severe refractory shock. She was transferred urgently to the cardiac catheterization laboratory, where a coaxial left ventricular assist device (LVAD), the Impella 2.5 (Abiomed, Abbott, Danvers, Massachusetts), was placed via the right femoral artery, delivering 2.5 l/min of cardiac output. The device inlet sits in the left ventricle (LV) cavity and the outlet sits in the ascending aorta. Afterward, her symptoms and hemodynamic status significantly improved, and the Impella device was removed on the eighth day.

DISCUSSION

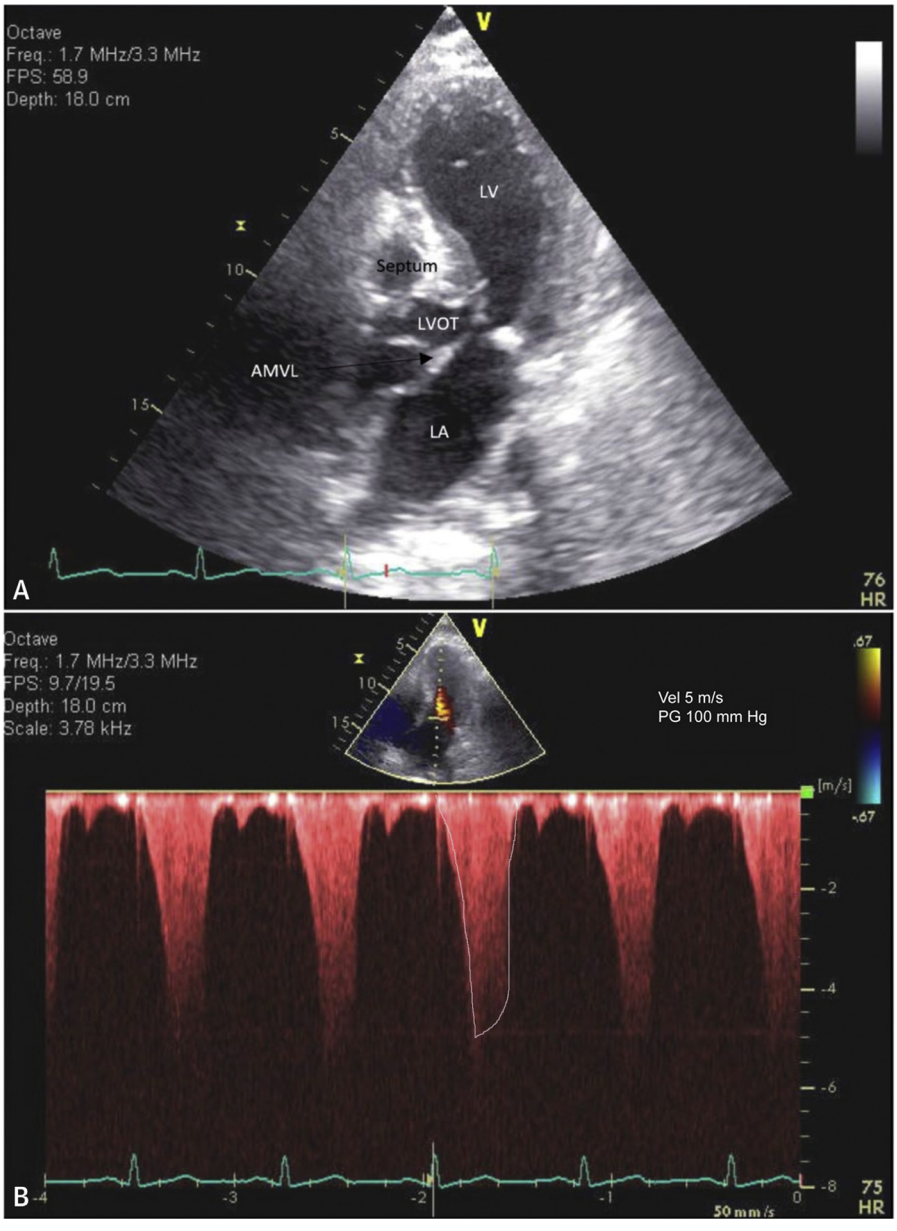
TCM is an uncommon reversible acute myocardial dysfunction reported in 0.02% of all-cause hospitalizations in the United States (1). The diagnosis of TCM

FIGURE 1 Left Heart Catheterization



(A) Left coronary artery and **(B)** right coronary artery with minimal disease. **(C)** Left ventriculogram showing akinetic apex **(arrowheads)** during systole. LAD = left anterior descending; LCX = left circumflex; LV = left ventricle; RCA = right coronary artery.

FIGURE 2 **Transthoracic Echocardiogram**



(A) Apical 3-chamber view showing sigmoid septum with systolic anterior movement of the anterior mitral valve leaflet (AMVL) causing left ventricular outflow tract (LVOT) obstruction. **(B)** Continuous Doppler at the LVOT showing flow velocity of approximately 5 m/s equivalent to a pressure of 100 mm Hg. LA = left atrium; other abbreviations as in Figure 1.

should be considered in adults (particularly postmenopausal women) who present with suspected acute coronary syndrome, especially when the clinical manifestations and electrocardiographic abnormalities are out of proportion to the degree of cardiac biomarker elevation. A physical or emotional trigger is present in two-thirds of the cases, and chest pain is absent in 25% of the patients, as was true in our patient (2). Compared with those without left ventricular outflow tract obstruction (LVOTO), patients with TCM and LVOTO have higher incidences of congestive heart failure and mitral regurgitation (3). In addition,

LVOT with gradients >40 mm Hg often predispose patients to hemodynamic instability.

The pathophysiology of the LVOTO is not well understood; however, it is believed to be caused by ventricular septal hypertrophy, a hyperkinetic base, and anterior displacement of the mitral leaflets in systole. The part of the anterior mitral leaflet distal to the coaptation point is subjected to venturi and/or drag forces, resulting in SAM and subsequent mitral leaflet-septal wall contact, which causes the subaortic obstruction (4). Because LVOTO can occur later in the course of the disease, close monitoring of these patients for signs of deterioration is important. Like hypertrophic obstructive cardiomyopathy, dynamic LVOTO usually resolves with maneuvers that reduce the gradient, such as pre-load augmentation with intravenous fluid, beta-blocker therapy, and phenylephrine (5).

Managing cardiogenic shock due to TCM with severe LVOTO can be challenging. Our patient was in acute heart failure; therefore, intravenous fluids had to be administered cautiously. Inotropic agents are generally contraindicated in TCM due to their negative effect on myocardial function; by augmenting myocardial contractility, they increase the LVOT gradient, hence, worsening the LVOTO. Although intra-aortic balloon pump (IABP) counter-pulsation provides some LV support, it can also worsen the LVOTO by reducing LV afterload and increasing the gradient across the LVOT.

The Impella device was studied in the settings of complex coronary intervention and cardiogenic shock post-myocardial infarction or cardiac surgery. Compared with IABP, the Impella 2.5 did not provide mortality benefit. However, there was significant improvement in the cardiac index and mean arterial pressure in the Impella group (6,7). Prospective studies for cardiogenic shock in TCM with LVOTO are lacking, but coaxial LVADs, such as the Impella, have shown promising results, particularly when the LVOTO is significant. Three cases in the literature described the use of LVADs as a bridge to recovery in TCM with profound LVOTO (8–10); the degree of LVOTO was variable, ranging between 24 and 110 mm Hg, and all patients had favorable outcomes with eventual recovery of LV function.

This case highlights the promising outcome of using the LVAD Impella 2.5 in such a setting as a bridge to recovery. It also prevented excessive fluid resuscitation and inotropic use, which can be detrimental in these patients.

FOLLOW-UP

A repeat TTE 1 week after admission showed improvement in EF from 30% to 35% and LVOT gradient decrease to 65 mm Hg. Her hospital course was complicated by atrial fibrillation, thrombocytopenia, and anemia, which required blood transfusion. Guideline-directed heart failure therapy was initiated, and a complete recovery of LV function was achieved over 3 months.

CONCLUSIONS

Due to the different treatment approaches, it is important to promptly identify subsets of TCM patients who have LVOTO. Managing cardiogenic shock due to TCM within the context of severe LVOTO can be challenging because inotropic agents and IABP counter-pulsation could exacerbate the condition. In contrast, early use of coaxial LVADs might provide sufficient cardiac output as a bridge to recovery.

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KEY WORDS **left ventricular apical ballooning syndrome, left ventricular outflow obstruction, ventricle assist device**