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CASE REPORT

INTERMEDIATE

HEART CARE TEAM/MULTIDISCIPLINARY TEAM LIVE

Left Ventricular Outflow Obstruction From Mechanical Circulatory Support in Critical Aortic Stenosis

A Cautionary Tale

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ABSTRACT

We submit a cautionary tale of a patient with critical aortic stenosis presenting with acute myocardial infarction and cardiogenic shock, who underwent balloon aortic valvuloplasty, insertion of a transvalvular left percutaneous ventricular assist device and high-risk percutaneous coronary intervention, with a post-operative course complicated by outflow obstruction from the device itself. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2023;13:101659) © 2023 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/).

Temporary mechanical circulatory support (MCS) is useful in acute myocardial infarction (AMI) with cardiogenic shock (CS), as it can help unload the left ventricle (LV), provide hemodynamic support, and serve as a bridge to LV recovery. Critical aortic stenosis (AS) may hinder the utility of certain temporary MCS, such as a 14-F transvalvular microaxial left percutaneous ventricular assist device (pVAD), which is relatively contraindicated in severe AS with a valve area (AVA) <0.6 cm^2. We submit a cautionary tale of a patient with critical AS presenting with AMI and CS supported with a transvalvular pVAD whose clinical course was later complicated by further aortic valve outflow obstruction from the pVAD itself, and which resolved after removal of the MCS.

LEARNING OBJECTIVES

- To describe the role of mechanical circulatory support in acute myocardial infarction with cardiogenic shock, as well as device-specific limitations.
- To appreciate the utility of invasive hemodynamics in the management of cardiogenic shock.
- To recognize the potential for mechanical outflow obstruction by a transvalvular percutaneous left ventricular assist device in severe aortic stenosis.

CASE PRESENTATION

An 88-year-old male with known coronary artery disease, severe AS, abdominal aortic aneurysm (AAA), diabetes, atrial fibrillation on apixaban, and hypertension presented with AMI with CS. A 12-lead electrocardiogram showed ST-segment elevation in lead aVR and diffuse ST-segment depression in the precordial and limb leads (**Figure 1**). Emergent transthoracic echocardiography revealed left ventricular ejection fraction (LVEF) of 20% with a mean gradient

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ABBREVIATIONS AND ACRONYMS

AAA = abdominal aortic aneurysm

AMI = acute myocardial infarction

AS = aortic stenosis

AVA = aortic valve area

CS = cardiogenic shock

MAP = mean arterial pressure

MCS = mechanical support

PAD = peripheral arterial disease

pVAD = percutaneous ventricular assist device

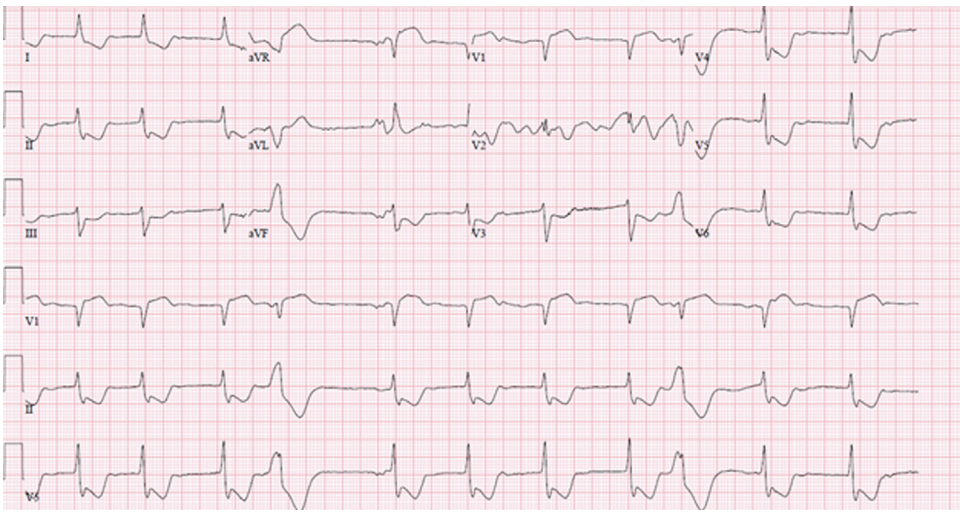
of 35 mm Hg, an estimated AVA of 0.41 cm^2, and an indexed stroke volume index of 17 mL/m^2 per beat, consistent with low flow-low gradient critical AS (most recent transthoracic echocardiography at a different facility 20 months prior showed an LVEF of 60% to 65% with a mean gradient of 41 mm Hg and a calculated AVA of 0.7-0.8 cm^2). Initial labs were significant for severe anemia with a hemoglobin of 4.7 g/dL (compared to 13.2 g/dL 1 year prior, and attributed to upper gastrointestinal bleed), shock liver with elevation of both aspartate aminotransferase and alanine aminotransferase to more than 1,000 U/L, and acute kidney injury with serum creatinine of 1.7 mg/dL. Despite adequate resuscitation with blood products, the patient required phenylephrine and norepinephrine in the emergency room to maintain adequate mean arterial pressures (MAPs), and had evidence of end-organ hypoperfusion. He was diagnosed with Society for Cardiovascular Angiography and Interventions stage C cardiogenic shock due to non-ST-segment elevation myocardial infarction.

The patient was taken urgently for coronary angiography, which revealed calcified bilateral iliac peripheral artery disease (PAD) with a large AAA (**Figure 2**), heavily calcified 90% stenosis of the left main (LM) extending into the left anterior descending (LAD) and left circumflex (LCX) (Medina 1,1,1) (**Figure 3**, Video 1), and 80% stenosis of the proximal right coronary artery. LV end-diastolic pressure was severely elevated at 32 mm Hg.

THE ROLE OF MCS IN AMI COMPLICATED BY CS

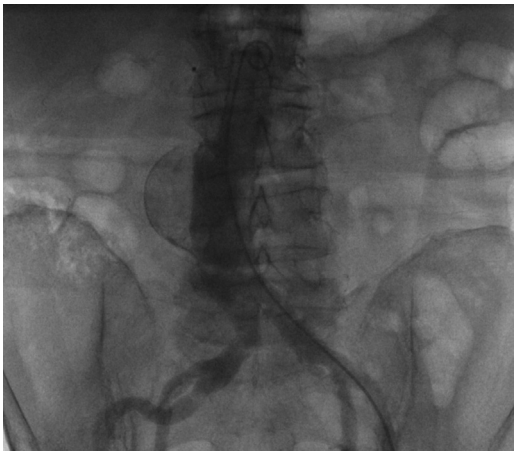
AMI complicated by CS carries mortality approaching 50%, with worse outcomes associated with advanced Society for Cardiovascular Angiography and Interventions stages of cardiogenic shock.[1] Beyond prompt revascularization, other therapeutic interventions including MCS have not shown consistent clinical benefit in randomized control trials and observational studies, possibly constrained by a lack of standardized protocols.[2,3] Our institution uses a multidisciplinary "shock" team in the early identification of cardiogenic shock, initial therapies at stabilization, and decisions regarding escalation and weaning of support. In this patient, due to CS and the need for intervention on high-risk coronary anatomy, we decided to place MCS before revascularization.

FIGURE 1 Peak Left Ventricle-Aorta Pressure Gradients Up to 70 mm Hg



ST-segment elevation in lead aVR and diffuse ST-segment depression in precordial and limb leads.

FIGURE 2 Abdominal Aortogram



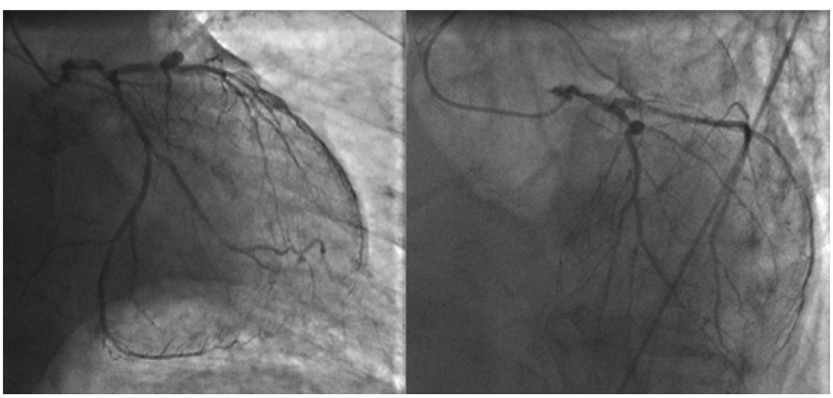
A large infrarenal abdominal aortic aneurysm with mural thrombus, and severe calcific aorto-iliac disease.

A transseptal pVAD was not available at our institution. Intra-aortic balloon pump was thought to be inadequate for support and contraindicated due to the patient's severe PAD and AAA. Peripheral veno-arterial extracorporeal membrane oxygenation was also relatively contraindicated due to the patient's PAD and severe anemia restricting systemic anti-coagulation. Thus, a 14-F transvalvular pVAD was chosen, which was successfully inserted following a balloon aortic valvuloplasty (BAV) with an 18-mm balloon (**Figure 4**), with a post-BAV reduction in mean gradient to 30 mm Hg as assessed by simultaneous aortic and LV catheter measurements. Coronary revascularization was then performed with rotational atherectomy from the LM into the proximal LAD, followed by intravascular ultrasound-guided percutaneous coronary intervention with bifurcation stenting of the LM/LAD/LCX (2-stent, mini-crush strategy) (**Figure 5**, Video 2). The transvalvular pVAD was left in place post-intervention due to the patient's ongoing hemodynamic instability, and heparinization was done through the purge solution alone.

THE UTILITY OF RIGHT HEART CATHETERIZATION IN THE MANAGEMENT OF CS

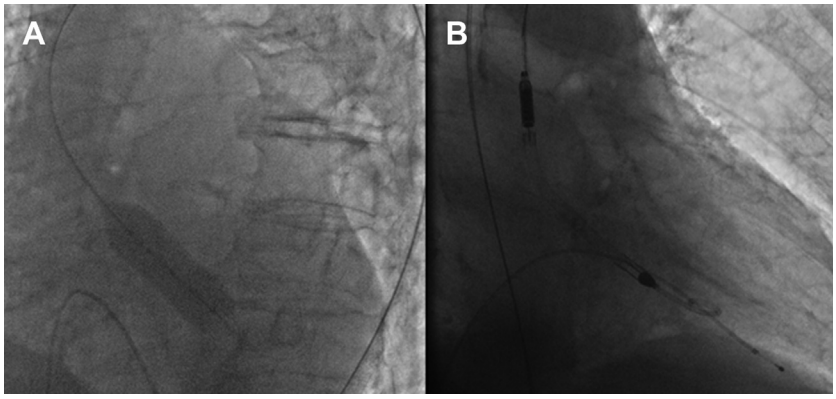
Right heart catheterization allows for the early identification of CS and severity of shock, and also the tracking of responses to therapeutic interventions. Invasive hemodynamics also identifies right ventricular or biventricular involvement in CS, aids decision-making in the type of MCS needed, and helps guide weaning or escalation of support.[4] In our patient, immediate postoperative right heart catheterization (while supported with dobutamine, norepinephrine, phenylephrine, and MCS with pVAD at maximum power) showed decreased cardiac output and index by thermodilution (thermodilution cardiac output [TDCO] and thermodilution cardiac index [TDCI]) to 3.2 L/min and l.57 L/min/m^2, respectively. Systemic arterial pressure was 67/60 with an MAP of 62 mm Hg, and a cardiac power output of 0.44 W. At that time, the pVAD power level was 8 with a calculated flow of

FIGURE 3 Coronary Angiogram



A heavily calcified 90% stenosis of the left main coronary artery extending into the left anterior descending and left circumflex arteries (Medina 1,1,1).

FIGURE 4 Fluoroscopy

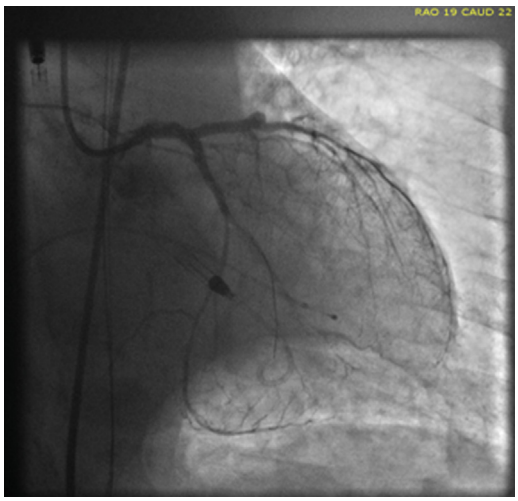


(A) Balloon aortic valvuloplasty. **(B)** Placement of the transvalvular percutaneous ventricular assist device.

3.4 to 3.8 L/min and minimal arterial pulsatility, implying hemodynamic dependence on the pVAD and poor native LV contribution to the cardiac output (CO) (Table 1).

The patient was diuresed in the cardiac care unit, and optimized on pharmacologic support with the weaning of phenylephrine and the addition of dopamine for added inotropy. Twenty-four hours after revascularization, TDCO increased to 4.2 L/min with a CI of 2.1 L/min/m^2. However, attempts to wean the pVAD were unsuccessful because of a concomitant decrease in blood pressure and CO. Although arterial pulsatility had improved, the waveforms on the pVAD console suggested significant valvular obstruction with a peak LV-aorta gradient of 60 to 70 mm Hg, likely affecting the ability to wean the pVAD (Figure 6). Because of concerns for outflow obstruction and significant hemolysis (evident by elevated lactate dehydrogenase level of 3,280 U/L and plasma hemoglobin of 47 mg/dL), the pVAD was rapidly weaned and removed in the cardiac catheterization laboratory 36 hours after device placement. TDCO and TDCI improved to 5.7 L/min and 2.8 L/min/m^2, respectively, promptly after pVAD removal (Table 1). Echocardiogram after pVAD explant showed an LVEF of 20% with a mean gradient of 31 mm Hg and an estimated AVA of 0.6 cm^2. He was subsequently weaned off inotropic support within 2 days, and had full recovery of hepatic and renal function upon discharge. Ultimately, the patient elected to forego transcatheter aortic valve replacement and was bridged to palliative care, surviving over 1 year from his initial presentation.

FIGURE 5 Coronary Angiogram



Coronary angiogram after percutaneous coronary intervention with percutaneous ventricular assist device in place postintervention.

A TRANSVALVULAR pVAD IS CONTRAINDICATED IN SEVERE AS

The use of a pVAD to unload and support the LV in patients presenting with AMI and CS may be beneficial. However, use of a transvalvular pVAD is contraindicated in patients with severe AS and an AVA of <0.6 cm^2 due to concerns that the 14F device through a severely stenotic atrioventricular orifice could worsen the valvular outflow obstruction and further compromise native cardiac hemodynamics.

TABLE 1 Hemodynamics by Hospital Day and Level of Support

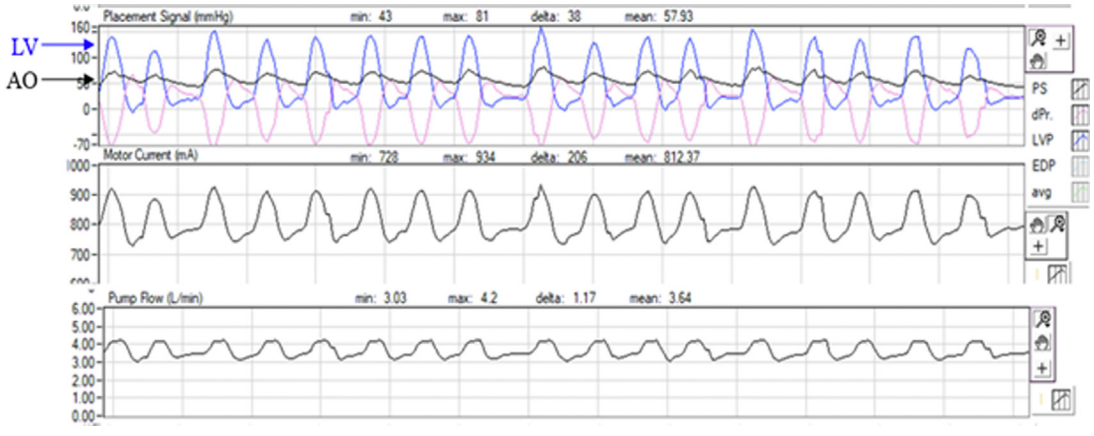
Pharmacological and Mechanical Support	PHE 50 Day 0	BAV, PCI, pVAD, at P-8, DB 3, PHE 90, NE 26 Day 1	pVAD at P-8, DA 3, NE 26, PHE 40 Day 2	pVAD at P-8, DB 3, DA 3, NE 14 Day 3	DB, NE 10 Day 4
RA		18	14	7	5
PCWP		18	20	16	18
CO		3.20	2.91	4.22	5.74
MAP	58	62	79	66	70
CPO		0.44	0.51	0.62	0.89

DA = dopamine (μg/kg/min); DB = dobutamine (μg/kg/min); RA = right atrium; PCWP = pulmonary capillary wedge pressure; CO = cardiac output; MAP = mean arterial pressure; CPO = cardiac power output; PHE = phenylephrine (μg/min); BAV = balloon aortic valvuloplasty; PCI = percutaneous coronary intervention; pVAD = percutaneous ventricular assist device; P-8 = power level 8; DB = dobutamine (μg/kg/min); NE = norepinephrine (μg/min);

Indeed, Blasé Carabello described the eponymous "Carabello sign" as the decrease in aortic pressure when a catheter is placed in the LV in critical AS, caused by the catheter itself enhancing the obstruction across the aortic valve. The cross-sectional area of a 14-F pVAD is 0.167 cm^2, and the transvalvular device may further restrict leaflet excursion. The valvular obstruction caused by the pVAD in a severely stenotic aortic valve can result in higher afterload faced by the LV, increased wall stress only partially relieved by the unloading from the pVAD, and highly turbulent flow around the pVAD resulting in greater hemolysis.

Case reports show that the use of a transvalvular pVAD may be safe as temporary support in patients with severe AS in various clinical settings.[5] However, the pVAD is usually removed after the intervention, even in patients presenting in CS. Our case is unique in that the transvalvular pVAD was kept in place for 36 hours to allow for continued hemodynamic support, LV unloading, and bridge to recovery. In this patient, the pVAD likely further deformed the stenotic aortic valve, worsened outflow obstruction and afterload, contributed to significant hemolysis, and impeded LV recovery. This is supported by the significant and rapid improvement in hemodynamics after removal of the pVAD. Nonetheless, the pVAD was beneficial in this patient's initial recovery and prevented further clinical deterioration. Early identification of device-related outflow obstruction and timing of device removal were also critical contributors to this patient's recovery. To our knowledge, this is the first report of hemodynamically significant LV outflow obstruction caused by a transvalvular pVAD, which resolved after device removal.

FIGURE 6 Waveforms on the Percutaneous Ventricular Assist Device Console



Peak left ventricular aorta gradients up to 70 mm Hg.

CONCLUSIONS

We present a case of AMI with CS requiring a multipdisciplinary shock team approach to management. Prolonged use of a transvalvular pVAD for hemodynamic support and as a bridge to myocardial recovery in patients with severe AS should be performed with caution, with careful monitoring of hemodynamics to assist with early device weaning and removal.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS aortic stenosis, cardiogenic shock, mechanical support, myocardial infarction, percutaneous ventricular assist device

APPENDIX For supplemental videos, please see the online version of this paper.