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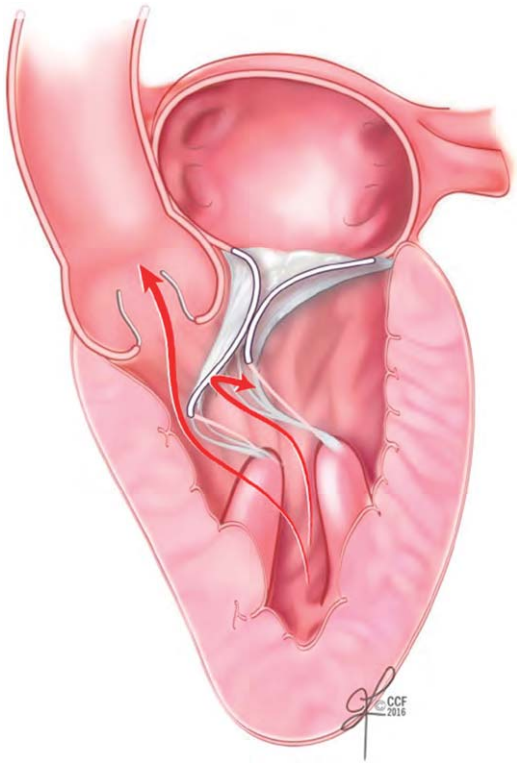

Figure 5. Drag effect: blood flow through left ventricle outflow tract (LVOT) drags the anterior mitral leaflet into the LVOT.

correcting these predisposing factors. Intravascular volume expansion is essential to increase left ventricular end-diastolic volume. Switching from a vasopressor with mixed α- and β-adrenergic effects (i.e., dopamine, norepinephrine, or epinephrine) to a pure α-agonist (phenylephrine) reduces cardiac contractility, heart rate, and LVOT velocity. As in the case presented here, addition of a β-blocker or nondihydropyridine calcium channel blocker can further decrease heart rate and cardiac contractility and resolve LVOT obstruction.

Answers

1. What was the cause of pulseless electrical activity and refractory shock?

The cause of pulseless electrical activity and refractory shock in this patient was development of systolic anterior motion of mitral valve causing left ventricle outflow tract obstruction.

2. What are the precipitating factors and underlying mechanisms for the development of systolic anterior motion of the mitral valve?

Table 1. Predisposing conditions for systolic anterior motion and left ventricle outflow tract obstruction

Reduced LV cavity size during systole
Decreased end-diastolic volume
Hypovolemia
Tachycardia (reduced diastolic filling time)
LV hypertrophy (diffuse or isolated to the septum)
Increased contractility
Increased LV ejection velocity
Increased contractility
Reduced LV afterload

Definition of abbreviation: LV = left ventricular.

A smaller LV cavity from LV hypertrophy, reduced diastolic filling from tachycardia, or hypovolemia decreases the distance between mitral valve and LVOT, allowing the drag effect to trigger SAM and LVOT obstruction. An increase in LV velocity from hype0-contractility or reduced LV afterload precipitates LVOT obstruction by pulling the mitral leaflet into the LVOT via venturi effect.

3. How should clinicians alter their management of septic shock in patients with SAM and LVOT obstruction?

The treatment should focus on correcting the precipitating factors. This includes intravascular volume expansion to increasing LV end diastolic volume, reducing cardiac contractility and heart rate by switching vasopressors with least chronotropic and inotropic effect. Ultra–short-acting β-blockers can be considered to further decrease the heart rate and cardiac contractility. ■

Author disclosures are available with the text of this article at www.atsjournals.org.

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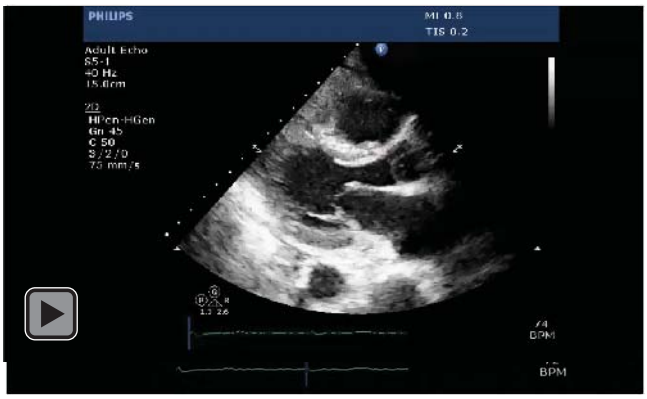

Video 2. Parasternal long-axis view reveals resolution of systolic anterior motion of mitral leaflet and left ventricle outflow tract obstruction.

Clinical Reasoning

The primary pathophysiologic abnormality in septic shock is a loss of vasomotor tone causing decreased left ventricular preload and afterload, low mean arterial pressure, and poor tissue perfusion. In response, increased sympathetic outflow augments the heart rate and myocardial contractility to increase cardiac output and improve tissue perfusion to critical organs. Decreased left ventricular end-diastolic volume, tachycardia, and increased cardiac contractility provide the perfect milieu for the development of SAM, resulting in LVOT obstruction and cardiovascular collapse.

In our patient, the 0.9% saline bolus given during cardiopulmonary resuscitation partially reversed the obstruction and led to a return of spontaneous circulation, but ongoing intrusion of the anterior mitral valve leaflet led to persistent LVOT obstruction as a result of decreased effective circulating blood volume, catecholamine-mediated tachycardia, and augmented cardiac contractility.

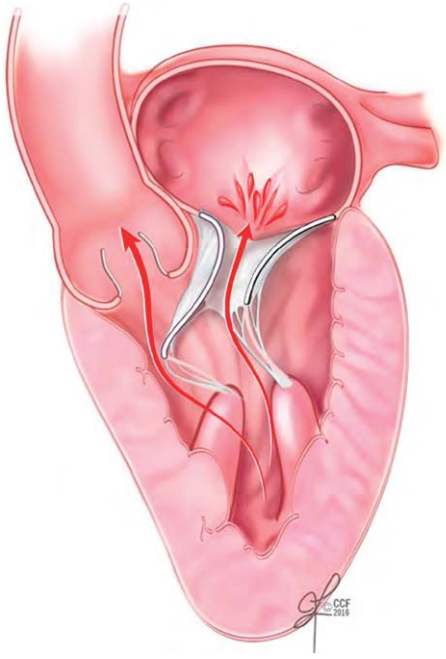

Figure 3. Elongated anterior mitral leaflet obstructing the left ventricle outflow tract. The associated mitral regurgitation is also appreciated in this image.

The Clinical Solution

Therapy was directed at both increasing left ventricular end-diastolic volume by augmenting venous return and diastolic filling time and reducing cardiac contractility. The patient received 2 L of lactated Ringer solution and 5 mg of intravenous metoprolol, and norepinephrine was changed to phenylephrine. This was accompanied by marked hemodynamic improvement.

Another transthoracic echocardiogram (Video 2) showed complete resolution of SAM and LVOT obstruction.

The Science behind the Solution

SAM of mitral valve was first described in the 1960s. It is defined as the anterior excursion of one or both mitral valve leaflets into the LVOT during systole, which leads to narrowing of the LVOT (Figure 3). The hemodynamic consequences of SAM are directly related to the duration and extent of contact between the mitral valve leaflet and the ventricular septum. Over the last decade, there has been growing recognition of the development of SAM in shock states, especially with the use of sympathomimetic medications.

Systolic anterior motion of the mitral valve is believed to result, at least in part, from the Venturi effect, which describes the drop in pressure created when a liquid flows at high velocity through a narrowed orifice (Figure 4). When heart rate and contractility are increased, the high velocity through the LVOT is believed to pull the mitral valve leaflets toward the septum. Alternatively, the drag effect (Figure 5) hypothesizes that some patients are predisposed to the development of SAM because their mitral valve leaflets are positioned in the path of LVOT flow, which drags them anteriorly and superiorly toward the septum. Predisposing factors for SAM are listed in Table 1 and include any condition that reduces LV systolic cavity size or increases blood velocity through the LVOT.

Effective therapy of SAM and LVOT obstruction is directed at reducing or

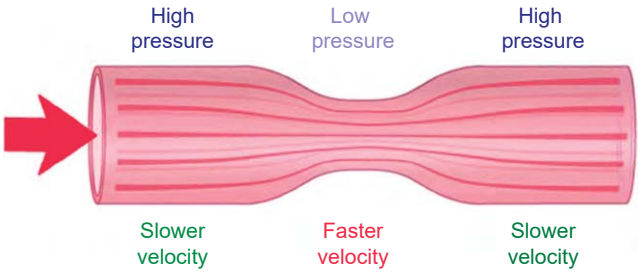

Figure 4. Venturi effect: an increase in fluid velocity along with a decrease in pressure occurs as fluid flows through a reduced cross-sectional area.

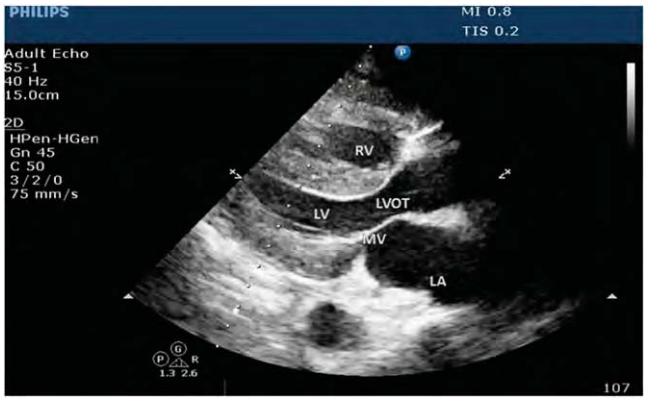

Figure 1. Parasternal long-axis view of the left atrium (LA), left ventricle (LV), mitral valve (MV), and left ventricle outflow tract (LVOT) during ventricular systole. BPM = beats per minute; RV = right ventricle.

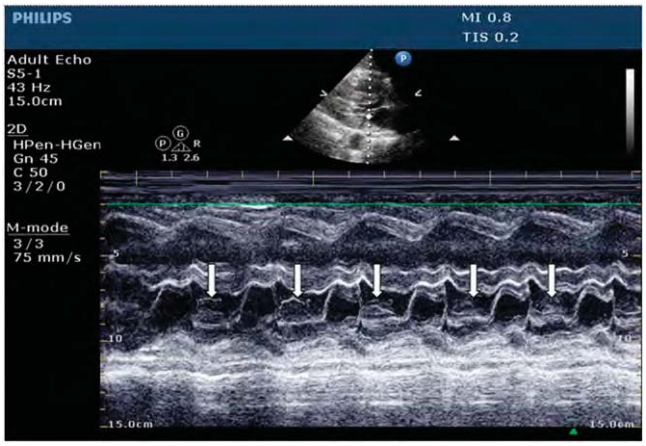

Figure 2. M-mode echocardiography. The *arrows* indicate systolic anterior motion of mitral leaflet during systole, which narrows the left ventricular outflow tract.

The echocardiogram revealed a small, underfilled, hyperdynamic left ventricle with systolic anterior motion (SAM) of the anterior mitral valve leaflet into the left ventricle outflow tract (LVOT) (Figure 1). Outflow tract obstruction was confirmed using M-mode ultrasonography (Figure 2).

Questions

1. What was the cause of pulseless electrical activity and refractory shock?

2. What are the precipitating factors and underlying mechanisms for the development of systolic anterior motion of the mitral valve?

3. How should clinicians alter their management of septic shock in patients with SAM and LVOT obstruction?

[Continue onto next page for answers]

The Clinical Physiologist

Section Editors: John Kreit, M.D., and Erik Swenson, M.D.

All Shock States Are Not the Same

Systolic Anterior Motion of Mitral Valve Causing Left Ventricular Outflow Tract Obstruction in Septic Shock

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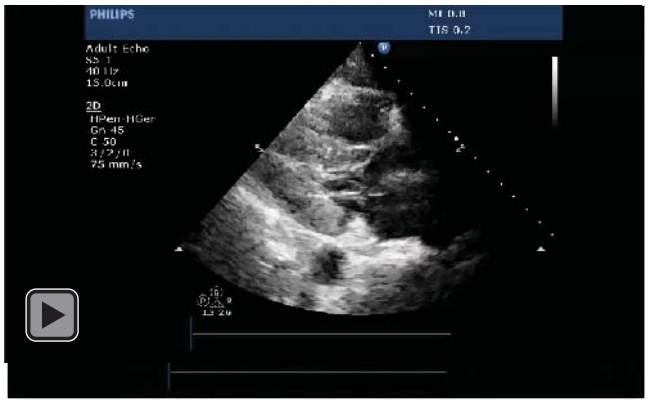
The Clinical Challenge

A 73-year-old man with a history of orthotopic heart transplantation secondary to myocarditis was admitted to the hospital for initiation of chemotherapy to treat leptomeningeal involvement by B-cell lymphoma. His hospital course was complicated by myelosuppression and profound neutropenia.

Eight days into his admission, he was found to be unresponsive on the regular nursing ward, with a heart rate of 122 beats/min and a blood pressure of 64/34 mm Hg. The patient rapidly progressed to pulseless electrical activity. Cardiopulmonary resuscitation was performed, with return of spontaneous circulation.

He was given 1 L of 0.9% saline and was emergently intubated and transferred to the medical intensive care unit. Due to ongoing hemodynamic instability, norepinephrine was started, and the dose was escalated quickly over the next hour due to continuing hypotension.

A bedside transthoracic echocardiogram was performed to determine other possible causes of his persistent shock state (Video 1).



Video 1. Parasternal long-axis view reveals a hyperdynamic left ventricle, with the anterior mitral leaflet moving into the left ventricle outflow tract during systole. A zoom-in view shows the systolic anterior motion of the anterior mitral valve leaflet.

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