Dynamic Left Ventricular Outflow Tract Obstruction in Acute Myocardial Infarction With Shock

Cause, Effect, and Coincidence

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Case presentation: A 70-year-old white woman with a prior history of tobacco abuse, emphysema, and recent pneumonia presented to an outside emergency room with brief episodes of dull chest pressure recurring over 5 days. Because the current episode was not relieved after 4 hours, and because her ECG showed ST elevation up to 3 mm in V2 through V6, she was given heparin and nitroglycerin infusions and was transferred to the University of Missouri. On admission to our hospital, she was pain-free with stable vital signs. Her examination was remarkable for a grade 2/6 systolic ejection murmur in the left third intercostal space. An ECG showed Q waves in V1 through V3. Echocardiography revealed significant left ventricular (LV) dysfunction, ejection fraction of 35% with systolic anterior motion (SAM) of the anterior mitral leaflet, and moderate mitral regurgitation (MR; Figure 1). LV outflow tract (LVOT) gradients were not quantified owing to MR Doppler contamination. Her maximum troponin was 5 ng/mL, and brain natriuretic peptide was 190 pg/mL. Catheterization showed normal coronaries with anteroapical akinesia and LV dysfunction with an ejection fraction of 30%.

The patient became hypotensive after catheterization, with systolic pressures between 70 and 85 mm Hg. Dopamine infusion did not improve blood pressure, and the murmur increased to grade 3/6 intensity. Atrial fibrillation developed with a ventricular rate of 150 bpm. Dopamine was discontinued, and intravenous amiodarone converted the atrial fibrillation to sinus tachycardia at 115 bpm, but the hypotension and murmur persisted. Under close supervision, intravenous metoprolol was initiated. With reduction of heart rate to below 70 bpm, the murmur disappeared, and her blood pressure improved. Several hours later, a repeat echocardiogram showed no SAM or LVOT obstruction (LVOTO) and only mild MR.

Numerous reports have highlighted the occurrence of dynamic LVOTO as a complication of ST-elevation myocardial infarction (STEMI).1,2 LVOTO has also been detected in ∼20% of transient LV apical ballooning syndrome, also called Takatsubo cardiomyopathy.3 The actual incidence of dynamic LVOTO is unclear, but it may be significantly underdiagnosed and can indeed mimic cardiogenic shock in an acute-care setting.4

Mechanism of LVOTO

Structural and functional factors contribute to the midsystolic development of gradients referred to as dynamic LVOTO. The asymmetrically hypertrophied septum, progressive narrowing of the LVOT during systole, and direction of the bloodstream cause drag forces and a Venturi effect on the anterior mitral leaflet, which results in SAM of the anterior mitral leaflet. This movement results in the anterior mitral leaflet contacting the septum for a period of systole, effectively obstructing the path of ventricular outflow. Failure of the anterior mitral leaflet to coapt with the posterior leaflet in systole results in MR.

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result from various clinical settings such as LV hypertrophy (hypertension or sigmoid septum), reduced LV chamber size (dehydration, bleeding, or diuresis), mitral valve abnormalities (redundant, long anterior leaflet), and hypercontractility (stress, anxiety, or inotropic agents such as dobutamine; Figure 2).

Assessment of LVOTO

Clinical examination consistent with subaortic stenosis should be sought in all subjects who present with hemodynamic instability and possible STEMI. Bisferiens pulse may not be evident if the patient is hypotensive, and the apex may be silent. MR features may predominate, but a sustained apical heave and a separate ejection murmur, best heard in the left third intercostal space, should create suspicion of LVOTO.

Inotropes are known to induce transient SAM and LVOTO in approximately 17% to 21% of otherwise healthy people. Transient LV apical ballooning may account for 1% to 2% of all STEMI cases and up to 12% of anterior STEMI in women. Systolic murmur, SAM, and even gradients across the LVOT could be related to true STEMI or transient LV apical ballooning or could be an inotrope-related benign finding.

Shock in the STEMI setting, therefore, cannot be automatically attributed to LVOTO in all instances in which echocardiography detects SAM. M-mode studies may help estimate the duration and severity of SAM. Color aliasing–guided pulse Doppler interrogation, from the apical 5-chamber view, may help select the LVOT jet from MR contamination. Because these findings can be transient, timing of the echocardiogram is critical. Hypotension should correlate to the severity of LVOTO gradients to attribute instability to SAM. Ideally, an experienced echocardiographer should note the prevailing hemodynamics and murmur at the time of imaging. Basal hypercontractility, regional dysfunction correlating with coronary territory, and apical ballooning should be assessed. MR direction and severity and structural abnormalities of the mitral apparatus also need to be quantified. Echocardiography provides an ideal tool for noninvasive, urgent, and definitive diagnosis of most components at the bedside. Early catheterization remains the method of choice to address the possibility of STEMI. The final proof for the role of SAM in this setting will be in the relief of hypotension by the reduction of LVOTO.

STEMI With Shock, or Is It LVOTO?

Along with acute MR, ventricular septal defect, and free-wall rupture, we propose that dynamic LVOTO be included in the differential diagnosis of STEMI and cardiogenic shock. If a murmur is noted to be suggestive of LVOTO, an echocardiogram should be performed at the earliest possible time. However, catheterization may be the initial study used to avoid delays in early diagnosis and relief of coronary occlusions. If catheterization reveals no significant coronary lesions, assessment of left ventriculography and catheter pullback gradients may help.
sequent hypotension could nevertheless be caused by increasing gradients across the LVOT. Regardless of coronary anatomy and intervention, repeat echocardiography to exclude LVOTO should be considered if an ejection murmur is detected with hemodynamic compromise (Figure 3).

Management of LVOTO
Management depends on early identification of the various issues (Table) and must be individualized on the basis of these factors, prevailing hemodynamics, and coexisting circumstances. Intravenous fluids would benefit by increased intravascular volume and LV volumes, thereby reducing the mitral SAM. Coronary revascularization would improve contractility of apical segments and reduce the basal hypercontractility. If LVOTO is not suspected, inotropes could be easily escalated and further decompensation attributed to STEMI-related LV dysfunction. Even in the absence of significant coronary lesions, this could elevate wall stress in the subendocardium enough to cause leakage of cardiac biomarkers that suggest MI. An intra-aortic balloon pump could induce or worsen LVOTO by reducing afterload. Phenylephrine may selectively improve vascular tone and reduce LVOTO. Use of β-blockers would benefit LVOTO gradients by reducing basal hypercontractility, increasing LV filling and size, and reducing heart rate. Nondihydropyridine calcium channel blockers may be used if β-blockers are contraindicated. Targeting heart rate below 60 to 70 bpm should ensure adequate cardiac inhibition and reduction of LVOTO. MR often improves with reduction of SAM and LVOTO.

Conclusions and Summary
Clinical suspicion, early recognition, and appropriate management of LVOTO, along with the independent addressing of STEMI, would significantly improve the outcome in this critically ill patient subset. In our patient, we showed that withdrawing inotropes and initiating intravenous β-blockers improved hypotension and reduced the LVOTO gradient within minutes. An echocardiogram 3 months after her hospitalization showed normal cardiac function, ejection fraction over 60%, and no mitral valve abnormality.

Disclosures
None.

References


**Key Words:** ventricular outflow obstruction ■ myocardial infarction ■ mitral valve ■ heart failure
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