Paradoxical Hypotension in the setting of IABP: A Case Report

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Background

Left ventricular outflow tract obstruction (LVOTO) has classically been observed in patients with hypertrophic obstructive cardiomyopathy (HOCM) where it occurs secondary to asymmetric septal hypertrophy and systolic anterior motion (SAM) of the mitral valve [1]. However, there are some instances that lead to hypercontractility of myocardium, and with a combination of other physiologic conditions, result in systolic anterior motion of mitral valve leaflet [2, 3]. We present such a case of an acute inferolateral wall myocardial infarction that was complicated by cardiogenic shock, requiring intra-aortic balloon pump and inotropic support which paradoxically provoked left ventricular outflow tract obstruction.

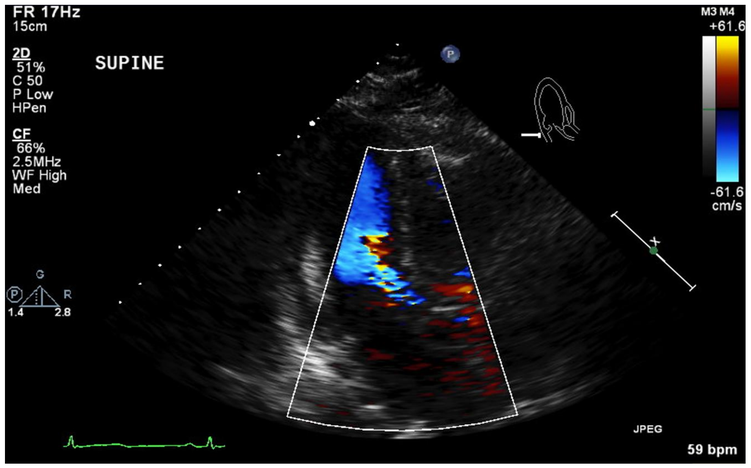

Fig. 1 Apical long axis view with color flow imaging showing sever turbulence at LVOT

Clinical Case

A 79-year-old Hispanic female with a past medical history of hypertension presented following an episode of severe substernal chest pain after which she collapsed and was resuscitated in the field appropriately. The initial rhythm on the field was asystole, followed by two shockable rhythms. In the emergency department, electrocardiogram (ECG) was performed which showed ST-segment elevation myocardial infarction (STEMI) in the inferior leads with reciprocal changes in the anterior leads. Code STEMI was activated, and she was taken immediately to cardiac catheterization lab. A left ventriculogram was performed which was significant for left ventricular ejection fraction (LVEF) of 30-35% with severe hypokinesis of the mid-to-apical anterior wall, infero-apical wall, and apex in a pattern of Takotsubo cardiomyopathy. Cardiac catheterization revealed 100% occlusion of the left circumflex (LCX) artery; a Xience drug-eluting stent was placed with restoration of TIMI 3 flow.

Norepinephrine and dopamine infusions were begun as the patient was in cardiogenic shock with an elevated Left Ventricular End Diastolic Pressure (LVEDP) of 32 mmHg. An IABP with one-to-one augmentation/counterpulsation was also initiated for a goal systolic pressure of 90 mmHg. Despite this, the patient's extremities remained cold with worsening hypotension.

The following day, an echocardiogram was performed which revealed hyperdynamic anterior, anteroseptal wall and SAM of the anterior mitral valve leaflet with severe flow turbulence across the LVOT (Fig. 1). Patient was supine and hence imaging quality was limited. Clinical examination revealed harsh 4/6 systolic over the left lower sternal border. Double tapping of carotid impulse was also noted. We switched IABP to 2:1 mode and the arterial wave form demonstrated a spike and dome appearance in the augmented beat.

At this time, the strategy was reversed. Pt was given IV fluids and the balloon pump was reduced to 1:3, which resulted in improvement in blood pressure with rapid tapering of pressors. In addition, a B-blocker was initiated, which resulted in further improvement of blood pressure along with urine output and peripheral warmth. The balloon pump was weaned off and removed over the next 24 hours. The patient remained hemodynamically stable off the pressors and was discharged to the nursing home on aspirin, ticagrelor, high-intensity statin, ACE Inhibitor, and beta-blocker for coronary artery disease.

Discussion

The left ventricular outflow tract (LVOT) is a region of the left ventricle that lies between the anterior leaflet of mitral valve and ventricular septum. Dynamic LVOTO is sensitive to changes in preload and afterload. Several cases have been reported in literature showing association of LVOTO with STEMI [4]. In inferolateral wall MI, anteroseptal wall frequently becomes hypercontractile in compensation. In females, who generally have small LV, increased contractility of anteroseptal wall can increase the flow acceleration in LVOT. If these patients are subjected to physiologic changes that increase contractility and dramatically reduce the afterload, it can produce LVOTO even in the absence of asymmetric septal hypertrophy. Moreover, it can cause clinically significant mitral regurgitation that decreases the afterload, further worsening the cardiac output.

IABP is one of the most widely used circulatory assist device. It dramatically decreases afterload, reduces cardiac work, and myocardial oxygen demand while increasing diastolic coronary blood flow [5]. However, by doing so, IABP has the potential of compromising hemodynamics by inducing or worsening the LVOTO in the appropriate clinical scenario.

Our patient had an inferolateral wall MI with hypercontractile anterior and anteroseptal wall. As she was hypotensive, multiple pressors were started and IABP was placed in Cath. Lab. This paradoxically worsened her blood pressure by producing a physiology of obstructive cardiomyopathy. Recognizing the complete pathophysiology was paramount in improving hemodynamic stability.

The management of dynamic LVOTO includes increasing left ventricular volume with fluid administration, increasing afterload with vasoconstrictor or by removal of IABP, and decreasing heart rate and inotropy with beta blockers. Inotropes are believed to cause or worsen LVOTO in 7-21% of patients, by promoting hypercontractility, accelerating blood in LVOT and worsening of SAM. In our case, patient's hemodynamics were further improved with B-blockers and tapering of inotropes.

Conclusion

Post inferolateral wall MI, compensatory hypercontractility of anterior wall in the setting of IABP which dramatically drops afterload, can cause hypotension secondary to SAM. If not recognized early, use of pressors will further decrease blood pressure and hence it demands the need for more meticulous evaluation of patient's hemodynamics. In our patient, the dramatic bisfriens carotid pulse with a harsh LV outflow systolic murmur guided us to further evaluate her cardiac physiology. The timeliness of the appropriate changes in the management resulted in stability of the patient. .

References

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