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CLINICAL VIGNETTE

A Mechanical Complication of Acute Myocardial Infarction: Ventricular Septal Defect in an Inferior Wall ST-Elevation Myocardial Infarction

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Introduction

Mechanical complications of acute myocardial infarction, such as ventricular septal defect, are a significant cause of mortality and should be suspected in patients who present with rapid hemodynamic deterioration and cardiogenic shock. Definitive management is procedural either through surgical or percutaneous repair. While the incidence of ventricular septal defect has decreased in the era of reperfusion, the ideal timing of procedural intervention has been debated. It is a balance between the risk of further deterioration versus higher procedural success by allowing fibrosis and stability of the myocardium. Mortality remains high even with procedural intervention and early recognition is key in initiating a management strategy.

Case Presentation

A 93-year-old male with a past medical history of a 5.5 cm abdominal aortic aneurysm status post endovascular stenting, presented by ambulance with acute onset chest and back pain which began at home. The patient was given aspirin in the field and an inferior wall ST- elevation myocardial infarction (STEMI) was confirmed on ECG upon arrival to the Emergency Department.

Initial vital signs were notable for systolic blood pressures in the 80s, a respiratory rate of 35, and oxygen saturation to 89 percent on room air. The patient was given supplemental oxygen, fluid boluses, and was started on a norepinephrine drip. Laboratory studies were notable for a troponin of 216 ng/ml, BNP of 496 pg/ml, and lactate of 6.5 mmol/L. The patient was taken to the cardiac catheterization lab.

Left heart catheterization was performed and a 100% occlusion of the proximal right coronary artery was found. The patient underwent successful balloon angioplasty with subsequent stenting resulting in TIMI II flow. Persistent hypotension occurred during the catheterization, requiring uptitration of norepinephrine and initiation of dopamine and epinephrine drips. The patient was transferred to the CCU for further care.

The following morning, a pansystolic murmur was heard throughout the precordium on auscultation. The degree of hypotension and hypoxia was thought to be out of proportion to a pure inferior wall STEMI. An urgent transthoracic echocardiogram (TTE) was performed and demonstrated a large 2.5 cm ventricular septal defect (VSD) with a left to right shunt. Left ventricular systolic function was moderately reduced with akinesis of the inferior, inferolateral, and inferoseptal walls. The patient remained critically ill, and after a discussion with the team, a family decision was made to transition the patient to a comfort-based approach. The patient subsequently died after cardiopulmonary arrest later the same morning.

Discussion

VSD is a major complication of acute myocardial infarction (MI) due its high mortality rate. The incidence of post-infarction VSD has declined with the advent of early reperfusion, with an estimated incidence of 0.17-0.34 percent.[1-5] It has a bimodal timing, usually within the first 24 hours or 3-5 days post-MI. The pathogenesis differs depending on the timing. Without reperfusion of the myocardium, coagulation necrosis develops within 3 to 5 days, leading to thinning and disintegration of the tissue. Earlier ruptures tend to occur in infarcts with large intramural hematomas, leading to dissection of the myocardium.[1] Rapid clinical deterioration into refractory cardiogenic shock can occur within hours as in this case and is typically associated with a new systolic murmur. Besides free wall rupture, VSD has been shown to have the highest mortality rate among the complications of acute MI.[6,7] Prompt recognition of a post-infarction VSD is critical, as it may lead to early surgical or percutaneous repair and implementation of measures for hemodynamic support such as an intra-aortic balloon pump (IABP).

Management of these patients can be complex though ultimately surgical intervention must always be the first consideration. An acute VSD initially leads to a left to right shunt with subsequent volume overload of the right ventricle, left atrium, and left ventricle.[8] Supportive devices such as an intra-aortic balloon pump or percutaneous heart pump (Impella) can be used to augment cardiac output.[9] Surgical techniques developed over the years include the Daggett technique of infarctectomy with direct patch placement of prosthetic

material between the RV and LV, or the David technique of infarct exclusion of patch placement sewn into the LV endocardium to exclude the VSD and infarct from the left ventricular cavity.[9-11] Percutaneous closure has also been found to be a viable alternative in the acute setting to definitive treatment or bridge to surgery.[12]

Surgical repair is the definitive treatment for a post-MI VSD, however optimal timing remains uncertain. The 2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction states emergent surgical repair is necessary, even in hemodynamically stable patients due to the risk of sudden hemodynamic collapse from abrupt rupture site expansion.[13] However, outcomes from the Society of Thoracic Surgeons National Database show lower operative mortality if the repair is greater than seven days from the index MI and the European Society of Cardiology advocates for a delayed surgical strategy so long as conservative measures are tolerated.[14,15] Improved mortality with delayed repair has also been seen in percutaneous repair of VSD.[16] The benefits of delayed repair are thought to be from allowing time for myocardial fibrosis and thereby avoiding closure of highly friable, actively necrotizing tissue. Survivor bias confounds the results since sicker patients whose hemodynamics do not tolerate conservative measures are more often taken to surgery early, and overall mortality may be higher in the medically managed group.[5] Even with surgical repair, the high mortality rate of post-MI VSD should prompt establishing realistic expectations and early discussion of goals of care especially if high-risk clinical features of advanced age, inotrope dependent cardiogenic shock, renal failure, unrevascularizable coronary artery disease, and large defect size are present.[12,17]

Our patient had multiple risk factors for a poor outcome if post-infarction septal defect closure was offered, and it was valuable to determining the management approach when the family endorsed his wishes of no heroic measures.

Learning Points

Although the incidence of post-infarction ventricular septal defect has declined with reperfusion, it remains a significant cause of rapid hemodynamic deterioration and mortality.

Early recognition of mechanical complications such as a VSD, is crucial and the diagnosis should be suspected in patients in cardiogenic shock manifested with hypotension and/or hypoxia or a new systolic murmur on auscultation.

Despite being a long-recognized complication of acute myocardial infarction, optimal timing and approach to repair remains challenging and expectations should be conservative.

Figures

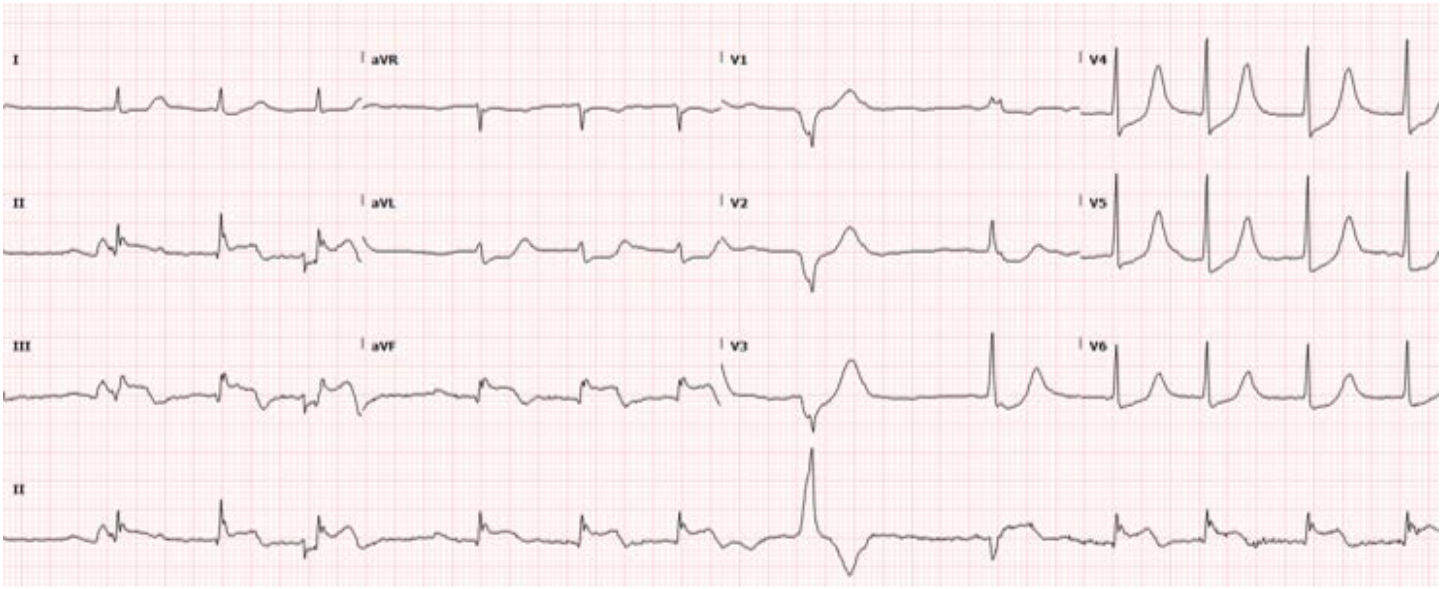

Figure 1 – ECG on arrival to the Emergency Department demonstrating atrial fibrillation with acute inferior myocardial infarction (leads II, III, aVF) and posterior wall involvement (leads V1-V3).

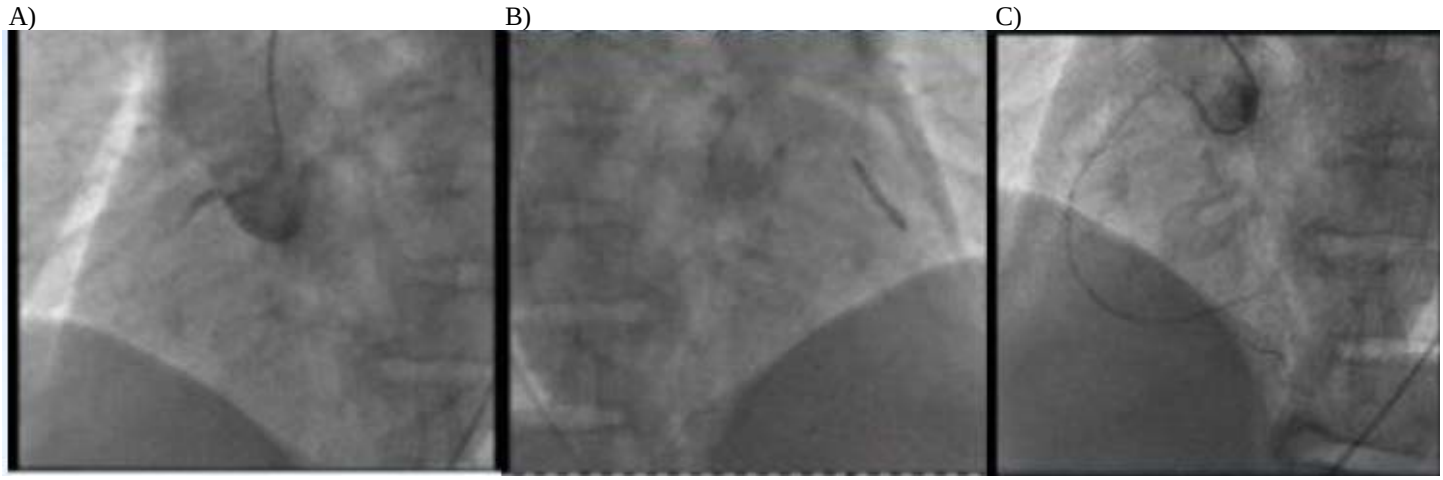

Figure 2 – Cardiac catheterization. A - 100% occlusion of the proximal right coronary artery. B – Drug eluting stent inflated in the proximal-to-middle segment. C – TIMI II flow after stent placement.

Figure 3 – Transthoracic Echocardiogram demonstrating a large VSD at the basal septal wall with left-to-right shunt during systole by color Doppler. A – Short axis view. B – Apical four chamber view. C – Subcostal view.

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