Ventricular Septal Rupture Secondary to Late-Presenting Myocardial Infarction

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ABSTRACT

BACKGROUND

Over 800,000 acute myocardial infarction (AMI) events occur annually in the United States. Increased emphasis on primary prevention strategies has decreased the incidence of AMI.[1,2] Treatment of AMI includes reperfusion of the culprit coronary arteries, and expeditious intervention has led to a decrease in the rate of post-AMI complications.[3] However, these complications still occur in approximately 0.3% of patients presenting with AMI; this is estimated to be about 2,400 patients annually.[4,5]

Myocardial tissue necrosis secondary to AMI can lead to several different mechanical complications, including papillary muscle rupture, ventricular septal rupture (VSR), and free-wall rupture.[2,3,6] These complications usually occur within the first seven days after an AMI.[2,3] Mortality from one of these MCs is over 42%, with women and patients older than 75 years of age having an even higher mortality rate.[5] This makes prevention, recognition, and prompt treatment critically important. Here we present a case report of a patient with post-AMI VSR.

KEYWORDS: Ventricular Septal Rupture; Acute Myocardial Infarction; Mechanical Complication

CASE REPORT

An 86-year-old female with a past medical history of coronary artery disease, hypertension, diabetes mellitus, atrial fibrillation without anticoagulation, and previous cerebrovascular accident presented with two days of generalized weakness and nausea. Upon arrival, the patient's vital signs included: temperature 98.6°F; heart rate 137, respiratory rate 21; blood pressure 130/77; pulse oximetry 95% on room air. Physical exam revealed a harsh, holosystolic murmur, heard best along the mid-left sternal border. Electrocardiogram demonstrated anterolateral ST segment elevation without reciprocal depression, and initial laboratory evaluation revealed a high-sensitivity troponin I of 15,857 ng/L. Comprehensive echocardiography was not available at time of the patient's initial presentation; however, a previous echocardiogram from one year prior had shown no significant wall-motion or valvular abnormalities. Due to concern for acute ischemia, the patient was taken to the cardiac catheterization laboratory from the Emergency Department. Cardiac catheterization revealed a total occlusion of the left anterior descending (LAD) artery without evidence of collateralization. A ventriculogram during the cardiac catheterization was suggestive of a ventricular septal rupture with left-to-right shunting. Stenting of the LAD artery occlusion was deferred, a heparin infusion was initiated, and the patient was transferred to the Coronary Care Unit (CCU) for evaluation by cardiothoracic surgery. Placement of an intra-aortic balloon pump (IABP) was deferred prior to transfer, predominantly due to logistic reasons.

The patient had stable vital signs and did not require vasopressor medications. Follow-up laboratory studies revealed a significant increase in high-sensitivity troponin up to 64,876. A comprehensive echocardiogram identified an 8 mm apical septal defect with left-to-right shunting [**Figures 1,2**]. The apex was akinetic and aneurysmal with an estimated left ventricular ejection fracture of 45%.

Within a few hours, and while heart-team discussions were ongoing, the patient suddenly became hypoxic and bradycardic. Advanced cardiovascular life support was immediately initiated, and the patient was intubated. A bedside point-of-care echocardiogram was performed which demonstrated a new moderately-sized pericardial effusion with right ventricular collapse consistent with cardiac tamponade. The patient had a sudden arrest of cardiac activity and cardiopulmonary resuscitation was initiated. The patient was pronounced dead after the family requested termination of resuscitation efforts. The etiology of the patient's acute decompensation was suspected to be progression of the VSR to include free-wall rupture.

DISCUSSION

This case represents the development of a post-AMI VSR, likely further complicated by free-wall rupture. VSR is the most common AMI-related mechanical complication,[5] and it results in a left-to-right shunt which can be appreciated on physical exam by auscultation of a holosystolic murmur.[2,3,6] The left side of the heart will eventually develop volume overload, causing dyspnea and clinical signs of cardiogenic shock.[3] Cardiogenic shock is the most significant cause of mortality following AMI and can be due to either left, right, or biventricular dysfunction.[7,8]

Figure 1. Partial view of the apical four-chamber view defect in the apical portion of the ventricular septum.

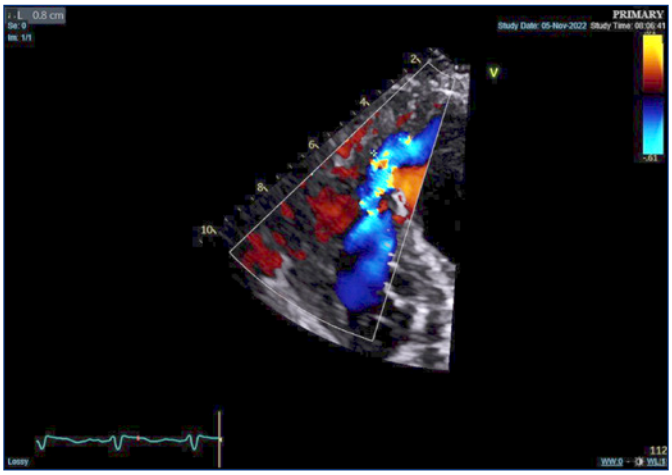
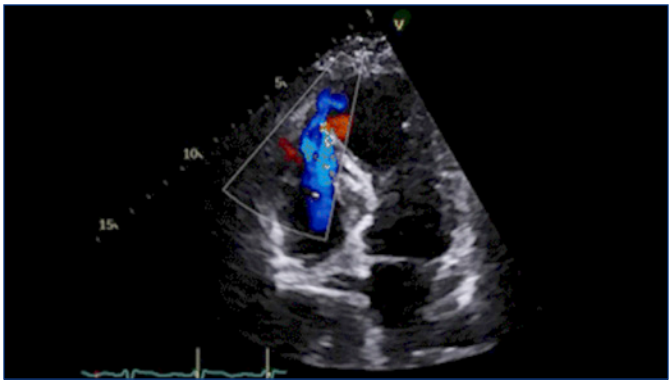


Figure 2. Clipped image of an apical four-chamber view showing a defect in the apical portion of the ventricular septum which shows left-to-right shunting.



Before the introduction of thrombolytic and percutaneous coronary intervention (PCI) therapies, VSR occurred in 1–3% of AMI cases. After these therapies were adopted, the incidence of VSR dropped to 0.2–0.5%.[7] The risk of developing a VSR after an AMI occurs in a bimodal fashion, with highest risk in the first 24 hours and again three to five days later.[9] The median time from AMI symptom onset to VSR has been reported to be between 16 hours and one day.[10,11] Longer time to PCI or thrombolytic administration increases the risk for development of a VSR.[12]

Diagnosing post-AMI mechanical complications require suspicion based on history and exam followed by emergent imaging, the latter including bedside echocardiography and ventriculography during cardiac catheterization. Mortality is related to management of cardiogenic shock prior to and after repair of the VSR, and thus immediate treatment of a post-AMI VSR involves management of cardiogenic shock. While ultimately closure of the defect, either with open or percutaneous surgical repair, is necessary, optimal timing of the repair is in question.[3,6,13] Based on case series and retrospective analyses, a delayed repair is associated with improved repair success and outcome.[14,15] The delay allows time for tissue remodeling, a reduced chance of defect progression, an opportunity to manage cardiogenic shock, and an opportunity to better define the defect and associated dysfunction with more advanced imaging.[14-17] Stabilization of patients awaiting surgical closure of a VSR often require vasopressors and inotropes, which may increase myocardial stress and oxygen consumption, potentially leading to increased defect progression.[14]

Alternatively, mechanical circulatory support (MCS) could be employed and has shown to reduce stress on the infarct and per-infarct zone while potentially limiting extent of cardiac injury.[5,17] Reduction of cardiac stress is crucial for reducing the risk for progression to free wall rupture or ventricular pseudoaneurysm.[18] An intra-aortic balloon pump reduces cardiac loading conditions and the VSR-induced left to right shunt; however, its role in increasing cardiac output is minimal.[17] Extracorporeal Membrane Oxygenation (ECMO) effectively increases systemic blood flow but, when placed peripherally, there may be an increase in afterload due to retrograde perfusion from the circuit, causing added strain on the left ventricle.[17] More recently, the role of temporary ventricular assist devices (tVAD) have been investigated in the management of cardiogenic shock in patients with a post-AMI VSR.[17] Clinical studies on tVAD are ongoing, and the available clinical data is still limited. Mortality was not found to be significantly improved with the use of IABP or ECMO. [5] In most studies, the placement of MCS in elderly patients typically refers to those over 65 or 70 years old. However, the use of MCS in octogenarians, including our patient, is less well documented and has been considered a relative contraindication.[19-21] Additionally, due to the logistical and procedural complexity of MCS, there are limitations on when and where it can be implemented, as was the case for our patient, who initially presented to a small hospital and required transfer for further care.

CONCLUSIONS

Our patient presented with a post-AMI VSR. Although the diagnosis was made in a timely manner, progression of the defect to include a free-wall rupture likely occurred, causing a fatal outcome. Immediate suspicion and emergency imaging are critical toward implementing immediate therapy, which is directed at preventing defect expansion and pression while managing cardiogenic shock. It is possible that immediate percutaneous revascularization and implementation of a MCS device might have reduced the likelihood of progression.

Despite available therapies, mortality associated with a VSR remains high. Current opinion supports a delay in definitive repair and early implementation of MCS to reduce cardiac load and stress, to permit time for per-infarct tissue

remodeling, to manage cardiogenic shock, and allow the opportunity to accurately define the defect with advanced imaging. Multidisciplinary teams involving emergency medicine physicians, cardiologists, interventional cardiologists, and intensivists play a vital role in optimizing outcomes for patients with a post-AMI VSR.

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