Interventricular Septum Rupture in the Catheterization Laboratory

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INTRODUCTION

This is a case of acute ventricular septal defect (VSD) that was diagnosed while a patient with an acute ST-elevation myocardial infarction (STEMI) was being treated in the cardiac catheterization laboratory. Post-MI VSD is a well-recognized complication of STEMI, and this case was particularly interesting given the circumstances of its diagnosis.

CASE PRESENTATION

A 75 year-old female with a history of well-controlled hypertension presented with 3 days of substernal chest pain radiating down her left arm. The pain was severe, unremitting, associated with shortness of breath, nausea, vomiting, and was not relieved by simethicone or by changes in position. The patient had a blood pressure of 165/92 mmHg, heart rate of 104 beats per minute, and physical examination was unremarkable. The initial electrocardiogram (ECG) showed 1mm ST elevations in the inferior leads (Figure 1). ECGs taken 1 hour later showed an increase in the inferior ST elevations to 2-3mm, and troponin T was elevated to 2.36 mJ (normal range = <0.01 mJ) (Figure 2). Echocardiogram showed inferior and inferoseptal akinesis as well as decreased left ventricular systolic function with an ejection fraction of 30%.

The patient was admitted for STEMI, given a full dose aspirin and atorvastatin, started on eptifibatide infusion, unfractionated heparin infusion, and taken for cardiac catheterization. At this time, her blood pressure remained elevated, so she was started on a nitroglycerin infusion. Subsequently, she became profoundly hypotensive, and the nitroglycerin infusion was discontinued. Coronary angiography showed right dominant coronary circulation with total thrombotic occlusion of the distal right coronary artery (RCA) and severe multivessel disease. Balloon angioplasty of the RCA was performed, with the plan to proceed with definitive coronary artery bypass graft surgery within 24-72 hours. Due to persistent hypotension, an intra-aortic balloon pump was placed with good diastolic augmentation.

DIFFERENTIAL DIAGNOSIS

At this point, the differential diagnosis for the patient's decompensation included cardiogenic shock secondary to left or right heart failure, rhythm disturbances such as complete heart block, and mechanical complications. The three main mechanical complications to consider in the setting of acute hypotension with an acute myocardial infarction include papillary muscle rupture causing acute mitral regurgitation, ventricular free wall rupture, and ventricular septal defect.

During this event, the patient did not demonstrate any new heart block on ECG monitoring. Thus, mechanical causes of her acute hypotension were investigated. Contrast ventriculography has the benefit of evaluating left ventricular function as well as illustrating possible free wall rupture, acute mitral regurgitation, or any new VSD. In this case, contrast ventriculography was performed revealing a large ventriculoseptal defect (VSD) in the inferoseptal wall (**Figure 3**).

Post-procedure transthoracic echocardiogram confirmed inferoseptal VSD and found a contiguous partial myocardial tear of the inferior wall with pseudoaneurysm (Figure 4). Given the high mortality of VSD and impending rupture of the inferior free wall, the patient was taken for operative cardiac repair.

OUTCOME AND FOLLOW-UP

In the case of this patient, the initial plan during her cardiac catheterization was to revascularize her culprit lesion (the RCA) and then ultimately pursue bypass surgery given her multivessel disease. However, after

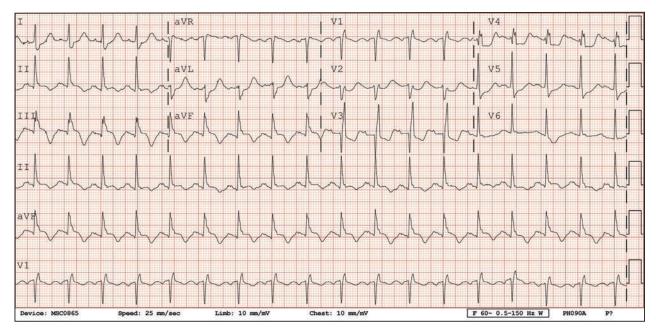


Figure 1: Initial ECG showing 1mm ST elevations in inferior leads

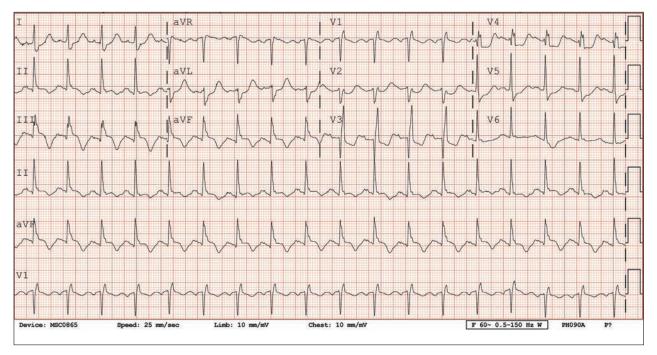


Figure 2: ECG taken 1 hour after presentation showing increase in inferior ST elevations to 2-3mm

her VSD was diagnosed, she had a balloon pump placed and she was taken emergently to the OR with successful repair of her defect. Unfortunately, after her procedure, she required significant vasopressor and inotropic support. She also developed and remained in complete heart block. After a week of aggressive medical therapy, her family ultimately elected to withdraw care.

DISCUSSION

While ventricular septal rupture is a well described complication of acute myocardial infarction, this case is relatively unique in that the diagnosis was made in the cardiac catheterization laboratory with the aid of contrast ventriculography. The incidence of VSD has decreased significantly after the advent of reperfusion therapy, from 2% of cases of infarction to as low as 0.2% described in the GUSTO trial.¹ However, it remains a problem with very high morbidity and mortality, especially in the setting of cardiogenic shock, as in this patient. It remains uniformly fatal without surgical intervention. This case helps highlight the importance of early and immediate recognition of VSD in facilitating emergency surgical repair. It also illustrates the importance of keeping VSD in the differential of acute hypotension during management of an acute myocardial infarction in the cardiac catheterization laboratory and the use of contrast ventriculography in making the diagnosis.

Ventricular septal rupture tends to occur 3 to 5 days after a myocardial infarction; however, cases have been described as recent as within 24 hours of an MI. Notably, this patient's initial MI was most likely 3 days prior to presentation to the ER. Risk factors include single vessel disease of the left anterior descending artery, advanced age, female sex, myocardial damage, massive MI, and poor septal collateral circulation.² In particular, patients with a "wrap-around" left anterior descending (LAD) appear to have an elevated risk. In the majority of individuals, the inferior third of the interventricular septum is supplied by the RCA; however, in some individuals the LAD extends beyond the apex, wrapping around to supply the inferior septum. Thus, these patients tend to have a higher risk of septal rupture in the setting of a STEMI, and ECG changes often show both anterior and inferior ST elevations.³

Patients present with hemodynamic compromise caused by biventricular failure, as well as a new harsh, holosystolic murmur heard best at the lower left and right sternal borders.⁴ Patients may also have a palpable thrill and a hyperdynamic precordium. Among patients who are not reperfused, such as this patient, septal rupture is often associated with persistent ST elevation for greater than 72 hours.⁵

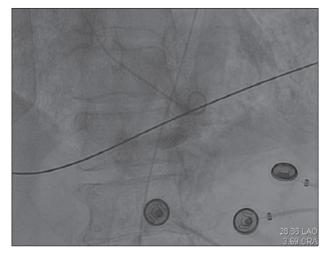


Figure 3: Contrast ventriculography showing a defect in the inferior wall of the left ventricle.



Figure 4: Parasternal short axis view with Doppler showing flow through the inferoseptal wall consistent with ventricular septal rupture

Diagnosis can be made in several ways. The gold standard remains insertion of a pulmonary artery balloon catheter which demonstrates a significant left to right shunt. Giant pulmonary capillary pressure V-waves may also occur secondary to volume overload and reduced atrial and ventricular compliance.⁶ Two dimensional transthoracic echocardiogram can also visualize the defect directly. This ability is significantly enhanced with the use of color Doppler imaging, and one study

demonstrated that adding color Doppler improved the visualization of the VSD from 40% to 100%.⁷ While left ventriculography can also be used to document the presence of the shunt, as in our patient, it tends to be unnecessary. However, with our patient presenting with acute cardiogenic shock while undergoing cardiac catheterization, it proved invaluable.

In patients presenting with frank cardiogenic shock, emergent surgery is required to prevent imminent death. Acute medical management involves afterload reduction with vasodilators and intraaortic balloon pump, which decreases the left to right shunt. Inotropic agents may also be used to increase cardiac output. Notably, several studies have also demonstrated that full revascularization of patients during operative repair of their VSD with bypass surgery also improves long term mortality.^{8,9} Novel approaches are also being used, such as percutaneous closure in patients with contraindications to bypass surgery. However, long-term study outcomes of this procedure are currently unavailable.

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