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AN UNSUAL CASE OF APICAL VSD POST ANTERIOR MYOCARDIAL INFARCTION

Dr. Sahithi Sura[1]*, Dr. K. Ravinder Reddy[2], Dr. K. Alekhya[3]

[1]Post Graduate, MD, Internal Medicine, RVMIMS & RC, Telangana, India.
[2]MD, DNB Consultant Cardiologist, Professor & HOD, Department of Internal Medicine, RVMIMS & RC Telangana, India.
[3]Associate Professor, MD Department of Internal Medicine, RVMIMS & RC, Telangana, India.

***Corresponding Author: Dr. Sahithi Sura**

Post Graduate, MD, Internal Medicine, RVMIMS & RC, Telangana, India.

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ABSTRACT

Ventricular septal defect (VSD) is a rare yet devastating mechanical complication of acute myocardial infarction (MI), most often presenting within the first week post-infarction. The condition carries high mortality if left untreated, with surgical or percutaneous closure considered the standard of care. However, in certain anatomically favourable cases, particularly when the defect is apical and the patient remains hemodynamically stable, conservative management may be considered. Long-term survival without surgical repair remains exceedingly uncommon.

KEYWORDS: The condition carries high mortality if left untreated, with surgical or percutaneous closure considered the standard of care.

INTRODUCTION

Post-myocardial infarction (MI) ventricular septal defect (VSD) is an uncommon but serious mechanical complication of acute MI, accounting for less than 1% of cases in the reperfusion era.[1] It typically occurs within the first week following infarction due to transmural necrosis and septal rupture. The condition is associated with high in-hospital mortality, with up to 90% of patients dying within the first few weeks if left untreated.[1] Surgical or percutaneous closure remains the standard of care, as conservative management alone usually results in rapid clinical deterioration due to cardiogenic shock, heart failure, or multi-organ dysfunction.[2] Despite these risks, a small number of patients may survive beyond the acute phase without surgical or interventional closure, often due to favourable anatomical characteristics, hemodynamic adaptation, or compensatory mechanisms.[3] Reports of long-term survival in such patients are exceedingly rare and provide valuable insights into the natural history of post-MI VSD. We present the case of a patient with an apical VSD following acute MI who survived for more than two years without surgical or percutaneous intervention, highlighting the unique clinical course, adaptive physiology, and implications for management.

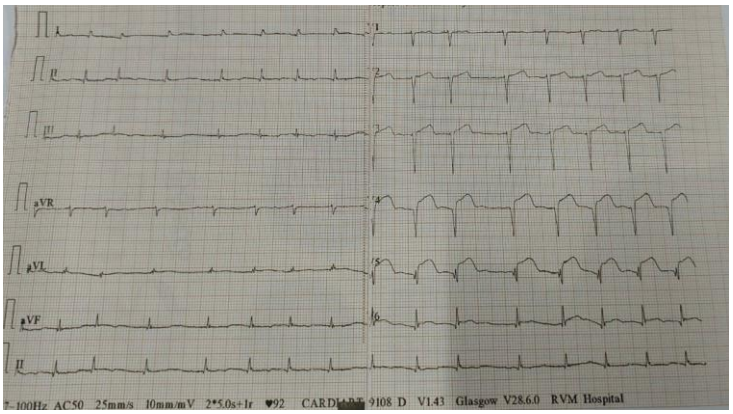
CASE REPORT

A 56-year-old female with a history of non-insulin-dependent diabetes mellitus and coronary artery disease presented with retrosternal chest pain radiating to the left upper limb. She was diagnosed with an acute anterior ST-elevation myocardial infarction (STEMI). Initial laboratory investigations showed.

- Haemoglobin: 12 g/dL
- Serum creatinine: 0.9 mg/dL
- Troponin I: 14 ng/mL
- AST: 43 U/L
- ALT: 25 U/L

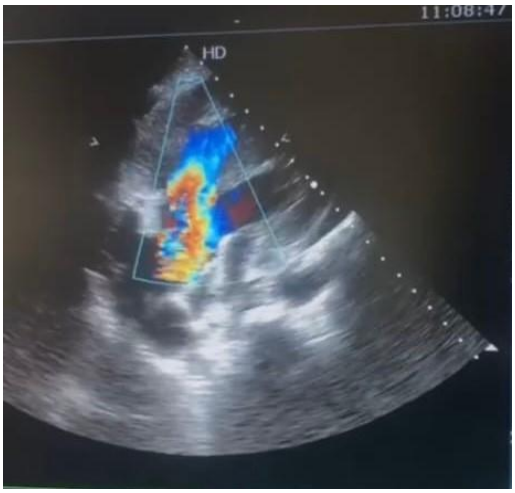
A 12-lead ECG showed ST-segment elevations in anterior leads, along with Q waves. Transthoracic echocardiography revealed a normal left ventricular size with apical akinesia and moderate left ventricular dysfunction (EF: 40%). Coronary angiography demonstrated diffuse disease in the left anterior descending artery with 99% occlusion. The patient underwent percutaneous coronary intervention (PCI) and was discharged in a stable condition on antiplatelets, statins, cardioselective beta-blockers, and oral

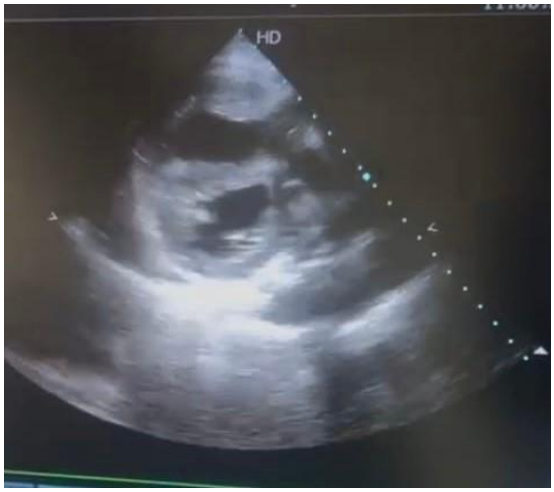
hypoglycemics.



Two weeks later, she presented with dyspnea, orthopnea, and paroxysmal nocturnal dyspnea, initially attributed to left ventricular dysfunction. On auscultation, a murmur was best heard along the left sternal border. Repeat laboratory parameters were within normal range.

- Hemoglobin: 12.2 g/dL
- Serum creatinine: 0.9 mg/dL
- Troponin I: Negative
- AST: 45 U/L





- ALT: 27 U/L
- NT-proBNP: Elevated

Repeat echocardiogram showed a large apical VSD measuring 1.5 cm with a left-to-right shunt. Cardiothoracic surgery was consulted, and surgical repair was advised. However, the patient declined surgical intervention and opted for conservative medical management.

DISCUSSION

Ventricular septal defect is a rare but serious complication of acute MI, with an incidence of less than 1% in the reperfusion era.[1] Although rare, it remains one of the most fatal complications, with in-hospital mortality rates exceeding 90% if untreated.[2]

Surgical or percutaneous closure is recommended, particularly in the setting of cardiogenic shock or hemodynamic compromise.[3] This case is notable for the patient's survival for over 24 months post-infarction without surgical or percutaneous repair. Although she experienced moderate heart failure symptoms (NYHA Class II–III), her clinical status remained relatively stable without right ventricular dysfunction or pulmonary hypertension. Several factors likely contributed to the stable course, including the apical location of the defect, which typically results in a smaller shunt volume compared to basal or mid-septal defects.[4] The absence of significant right ventricular dysfunction and pulmonary hypertension likely mitigated the usual.

progression toward heart failure. Similar outcomes have been described in a few case reports where anatomical characteristics and early adaptation permitted conservative management.[5]

Current guidelines by the American Heart Association and American College of Cardiology recommend early

surgical repair, especially in unstable patients.[6] However, individualised management is crucial, taking into account patient preference,anatomical considerations, surgical risk, and symptom trajectory. In this case, the patient's refusal of surgery, combined with her initial hemodynamic stability, prompted a conservative approach with close follow-up and guideline-directed medical therapy (GDMT).

Despite the lack of major complications, the patient remained symptomatic with moderate heart failure, reflecting the chronic volume overload and raising concerns for future complications like ventricular remodelling or arrhythmias. This case underscores the importance of personalised decision-making in managing post-MI VSD and illustrates a rare instance of long-term survival without surgical intervention.

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