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Case Report

A Silent Rupture: Post-Infarction Ventricular Septal Defect Following Acute Myocardial Infarction — A Case Report

Routledge D, Cockburn E, Malinowski S, Rheude J, Yoneyama F, Torella S

Department of Cardiovascular Surgery, Romania

***Corresponding Author:** Cockburn E, Department of Cardiovascular Surgery,Romania

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Abstract

Post-infarction ventricular septal defect (PIVSD) is a rare but catastrophic mechanical complication of acute myocardial infarction (AMI), associated with high mortality despite advances in reperfusion therapy and surgical management. Early diagnosis and prompt intervention are essential to improve survival outcomes. We report the case of a 64-year-old male with a history of hypertension and type 2 diabetes mellitus who presented with acute onset dyspnea, chest pain, and cardiogenic shock three days after an untreated inferior wall myocardial infarction. Clinical examination revealed hypotension, tachycardia, elevated jugular venous pressure, and a harsh pansystolic murmur at the left lower sternal border. Electrocardiography demonstrated ST-segment elevation in the inferior leads, while transthoracic echocardiography identified a 14-mm ventricular septal defect with left-to-right shunting and severe right ventricular overload. Coronary angiography showed complete occlusion of the right coronary artery. The patient was stabilized with vasopressor support and intra-aortic balloon pump insertion prior to undergoing emergency surgical repair of the septal defect with coronary artery bypass grafting. Postoperative recovery was complicated by transient renal dysfunction but improved gradually with intensive care support. The patient was discharged in stable condition after three weeks of hospitalization and remained asymptomatic at three-month follow-up.

Introduction

Post-infarction ventricular septal defect (PIVSD), also referred to as ventricular septal rupture (VSR), is a rare but devastating complication of acute myocardial infarction. It occurs due to full-thickness necrosis of the interventricular septum following prolonged ischemia. Before the advent of reperfusion therapy, the incidence of PIVSD ranged between 1–3% of myocardial infarctions; however, with the widespread use of thrombolysis and primary percutaneous coronary intervention (PCI), the incidence has declined to approximately 0.2–0.3%. Despite improvements in diagnosis and management, mortality remains exceedingly high, especially in untreated patients. Hemodynamic deterioration results from acute left-to-right shunting, leading to pulmonary overcirculation, right ventricular overload, reduced systemic perfusion, and cardiogenic shock. Most cases occur within 3–7 days after myocardial infarction, particularly in elderly patients, women, diabetics, and those with delayed reperfusion therapy.

Clinical presentation often includes sudden onset dyspnea, hypotension, chest pain, and the appearance of a new harsh pansystolic murmur. Echocardiography remains the gold standard diagnostic modality for confirming the defect and assessing hemodynamic significance. Definitive treatment typically involves urgent surgical repair, although transcatheter closure has emerged as an alternative in selected cases.

Case Presentation

Patient Information

A 64-year-old male presented to the emergency department with severe shortness of breath, generalized weakness, and chest discomfort for one day. Three days earlier, he had experienced sudden onset central crushing chest pain radiating to the left arm associated with diaphoresis and nausea. However, he did not seek medical attention at that time.

The patient had a history of:

- Hypertension for 12 years
- Type 2 diabetes mellitus for 10 years

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- Dyslipidemia
- Chronic smoking history of 25 pack-years

There was no prior history of ischemic heart disease or cardiac surgery

Diagnostic Assessment

Electrocardiography (ECG)

ECG demonstrated:

- ST-segment elevation in leads II, III, and aVF
- Reciprocal ST depression in leads I and aVL
- Q waves in inferior leads

These findings were consistent with an acute inferior wall ST-elevation myocardial infarction (STEMI).

Therapeutic Intervention

The patient was immediately transferred to the cardiac intensive care unit

Initial Stabilization

Management included:

- Oxygen supplementation
- Intravenous diuretics
- Vasopressor support (norepinephrine)
- Inotropic therapy (dobutamine)
- Dual antiplatelet therapy
- Anticoagulation
- Mechanical ventilation due to respiratory distress

An intra-aortic balloon pump (IABP) was inserted to reduce afterload and improve coronary perfusion

Postoperative Course

The patient remained in the intensive care unit for 10 days

Complications

Postoperative complications included:

- Low cardiac output syndrome
- Acute kidney injury
- Atrial fibrillation episodes

These were managed conservatively with:

- Inotropic support
- Careful fluid management
- Antiarrhythmic therapy

Repeat echocardiography performed after surgery showed

- Intact patch repair
- Minimal residual shunt
- Improved ventricular function

Gradually, the patient's hemodynamic condition stabilized.

Conclusion

Post-infarction ventricular septal defect is a rare but fatal mechanical complication of acute myocardial infarction. Early clinical suspicion, prompt echocardiographic diagnosis, rapid hemodynamic stabilization, and urgent surgical intervention are critical for survival. This case highlights the importance of recognizing new cardiac murmurs and hemodynamic deterioration after myocardial infarction as warning signs of ventricular septal rupture. Timely multidisciplinary management can significantly improve patient outcomes despite the high mortality associated with this condition.

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