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Post-infarction ventricular septal rupture: a fatal mechanical complication of late-presenting STEMI - a case report

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Abstract

Introduction: Mechanical complications of myocardial infarction have become rare since the advent of percutaneous coronary intervention, but their occurrence remains associated with a high mortality rate. In the presence of suggestive clinical signs, unexplained hemodynamic instability, or specific risk factors, bedside echocardiography is essential to establish the diagnosis and to guide the optimal management strategy.

Case presentation: We report the case of a 72-year-old man admitted for a late-presenting inferior ST-elevation myocardial infarction in whom coronary recanalization failed. Forty-eight hours after admission, the patient developed recurrent chest pain, ST-segment re-elevation and progressive cardiogenic shock. A second coronary angiography was first performed and was superimposable with the first. The diagnosis was subsequently established on bedside echocardiography, which revealed a massive apical ventricular septal rupture, leading to emergency surgical repair without prior mechanical circulatory support. The patient died from refractory cardiogenic shock and multiple organ failure.

Conclusions: This case highlights the diagnostic pitfalls of post-infarction ventricular septal rupture, the value of repeated bedside echocardiography in patients with suspected mechanical complications, and the controversy regarding emergency surgical repair without prior hemodynamic stabilization by mechanical circulatory support.

Keywords: ventricular septal rupture; acute coronary syndrome; cardiogenic shock; transthoracic echocardiography; mechanical circulatory support; ECMO.

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Introduction

Ventricular septal rupture (VSR) is one of the major mechanical complications of acute myocardial infarction, together with free-wall rupture and acute mitral regurgitation due to papillary muscle necrosis. Post-infarction mechanical complications usually occur in the days following the index event and are more frequent after ST-segment elevation myocardial infarction (STEMI) [1].

Their overall incidence has decreased significantly in the era of percutaneous coronary intervention, with reported annual rates declining by 41.6% over 15 years between 1999 and 2014 [2]. Despite this decline, the overall in-hospital mortality among patients who developed mechanical complications remains high with reported rates at 42.4% in STEMI and 18% in NSTEMI, with no significant change over time [3].

We report the case of a 72-year-old patient who developed a ventricular septal rupture after a late-presenting acute coronary syndrome, together with a brief literature review on the diagnosis and management of this rare but life-threatening complication. This case is illustrative of the complex therapeutic dilemma raised by post-infarction VSR complicated by cardiogenic shock, namely whether to proceed directly to surgical repair or first attempt hemodynamic stabilization with mechanical circulatory support — a question that remains debated in current literature.

Case presentation

A 72-year-old man was admitted to the emergency department for retrosternal chest pain that had been evolving over the previous two weeks and had intensified the day before admission. He had several cardiovascular risk factors, including hypertension, type 2 diabetes mellitus, overweight, and a history of smoking (ceased in 1998). On admission, blood pressure was 140/80 mmHg, heart rate was regular at 110 beats per minute, and oxygen saturation was 96% on room air. The electrocardiogram showed a Q wave and ST-segment elevation in the inferior territory (Figure 1). High-sensitivity troponin I was markedly elevated at

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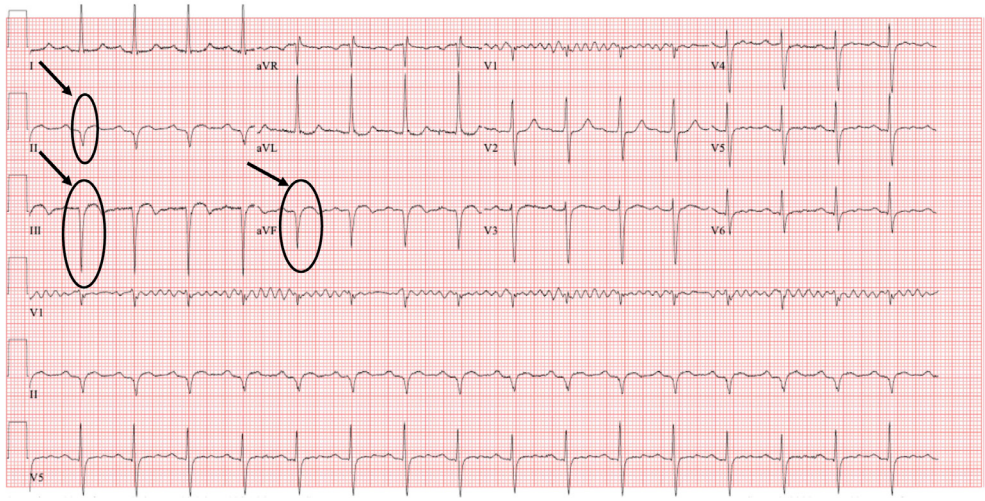


Figure 1. 12-lead electrocardiogram on admission showing pathological Q waves and ST-segment elevation in the inferior leads (II, III, aVF), consistent with a late-presenting inferior STEMI.

12 774 ng/L (reference value < 6 ng/L). Chest pain persisted despite analgesics and intravenous nitrates, and a diagnosis of late-presenting inferior STEMI was made.

Although the patient was identified as a late presenter (>48h after onset of complaints), given the persistent pain, in accord with the interventional cardiologist on call, we decided to perform a coronary angiography after administration of 300 mg of aspirin and 5 000 units of heparin. This exam revealed a complete occlusion of the right coronary artery; recanalization attempts using a guidewire and balloon were unsuccessful because the occlusion could not be crossed. Medical management was prioritized, with close monitoring in the intensive care unit. Bedside transthoracic echocardiography confirmed extensive inferior wall akinesia with a globally preserved left ventricular ejection fraction.

During the following hours, the patient experienced recurrent chest pain that quickly resolved with level I analgesics. His clinical condition remained reassuring: hemodynamics were stable, no arrhythmia was detected, and the ST-segment elevation was gradually resolving. Approximately 48 hours after admission, the patient developed a recurrence of severe chest pain with hemodynamic instability and a new electrocardiographic change characterized by reappearance of a Pardee wave in the inferior territory. A second coronary angiography was performed, revealing no new occlusion and no further therapeutic option. A bedside echocardiogram performed in the catheterization laboratory assessed global cardiac function, which appeared relatively preserved.

Back in the intensive care unit, the patient was repositioned and other potential causes of instability, such as bleeding or sepsis, were excluded. A repeat echocardiogram was then performed approximately four hours after the prior study. Echocardiography revealed a large 13-mm ventricular septal defect (VSD) with significant left-to-right shunting, located in the apico-inferior region of the interventricular septum. The pulmonary-to-systemic flow ratio (Qp/Qs) could not be calculated due to poor acoustic windows and inadequate visualization of the right ventricular outflow tract. Right ventricular longitudinal dysfunction was noted, with a tricuspid annular plane systolic excursion (TAPSE) of 12 mm and a tricuspid annular systolic velocity (S') of 8 cm/sec. Significant pulmonary hypertension was also noted, with an estimated pulmonary artery systolic pressure (PAPs) of 55 mmHg. Additionally, a persistent D-shaped left ventricle throughout the cardiac cycle was observed, indicating significant volume overload. (Figure 2). The patient was urgently transferred to a center with cardiovascular surgical capabilities for emergency repair. Following multidisciplinary discussion with the cardiology team, it was decided not to proceed with preoperative mechanical circulatory support (MCS) device implantation.

Intraoperatively, complete infarction of the inferior myocardial wall was confirmed, complicated by a massive ventricular septal defect extending from the area of the posteromedial papillary muscle to the apex. The ventricular septum was reconstructed using a pericardial patch, achieving complete closure of the defect.

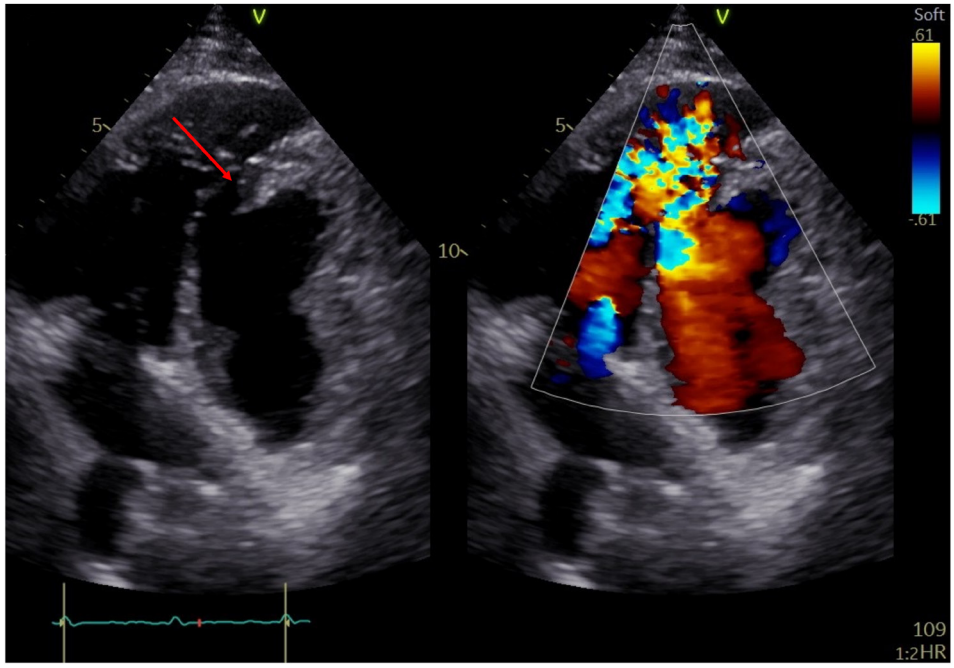


Figure 2. Transthoracic echocardiography (apical four-chamber view) with color Doppler performed approximately 52 hours after admission, revealing a large apical ventricular septal defect with a significant left-to-right shunt.

Table 1. Timeline of clinical events from admission to death. STEMI: ST-elevation myocardial infarction, RCA: right coronary artery, TTE: transthoracic echocardiography, LVEF: left ventricular ejection fraction, VSR: ventricular septal rupture, MCS: mechanical circulatory support, VA-ECMO: veno-arterial extracorporeal membrane oxygenation, VSD: ventricular septal defect

Phase	Timeframe	Key events and decisions
1 — Late presentation	D−14 → D0	Two-week history of chest pain culminating in admission. Diagnosis of late inferior STEMI.
2 — Failed recanalization	D0 (H+1)	Coronary angiography: total RCA occlusion. Recanalization unsuccessful. ICU admission with extensive inferior akinesia on TTE; LVEF preserved.
3 — Apparent stabilization	D0 → D2 (H+48)	Stable hemodynamics, recurrent but self-limiting pain.
4 — Clinical deterioration	D2 (H+48 → H+52)	Recurrent severe pain + reappearance of Pardee wave. 2nd angiography unchanged; cath-lab TTE limited to LVEF. VSR was finally diagnosed on repeat TTE 4 h later, after onset of cardiogenic shock.
5 — Surgery and unfavorable outcome	D2 (H+56) → D4	Emergency surgery repair without prior MCS. VA-ECMO initiated post-op. Recurrence of residual VSD, multiple organ failure, withdrawal of care, death.

Due to persistent cardiogenic shock, veno-arterial extracorporeal membrane oxygenation (VA-ECMO) was initiated at the end of the procedure. On the following day, a control echocardiogram revealed recurrence of a large residual ventricular septal defect. The patient's clinical condition deteriorated progressively, leading to multiple organ failure and refractory vasoplegia. After multidisciplinary discussion and in the absence of any further therapeutic option, a decision was made to withdraw life-sustaining therapy. The patient died shortly thereafter.

Discussion

Epidemiology and risk factors

Ventricular septal rupture is one of the major mechanical complications of acute myocardial infarction, along with free-wall rupture and acute mitral regurgitation due to papillary muscle necrosis. Their overall incidence has decreased significantly in the era of percutaneous coronary intervention, but the prognosis remains poor [1]. Data from a large observational analysis of approximately 9 million admissions for acute

myocardial infarction in the United States from 2003 to 2015 reported a rate of mechanical complications of 0.27% in the STEMI cohort and 0.06% in the non-STEMI (NSTEMI) cohort. VSR was the most common mechanical complication. In-hospital mortality was higher in patients older than 75 years in both cohorts, and in women compared with men in the STEMI cohort. Other predictors of mortality in patients with STEMI and cardiogenic shock were valvular heart disease, obesity and peripheral artery disease [3].

Delayed or unsuccessful revascularization is a well-established risk factor for post-infarction cardiac rupture, including VSR. In a large 30-year cohort of 6678 STEMI patients, admission delay among patients who subsequently developed cardiac rupture remained persistently prolonged (24–30 hours) throughout the study period, in contrast to a progressive shortening to under 10 hours among STEMI patients without rupture, and the decline in rupture incidence (6.2% to 3.2%) paralleled an increasing use of reperfusion therapy (0% to 75.1%) [4]. Prolonged ischemia time is thought to favor rupture by increasing infarct transmurality and wall tension, whereas effective and timely reperfusion limits infarct extent and thereby reduces this risk [4]. Septal rupture has also been associated with single-vessel disease, total occlusion of the culprit vessel and the absence of collateral flow to the infarct territory [5].

Diagnostic challenges

VSR usually occurs between the third and seventh day after acute myocardial infarction. Clinical manifestations may include new acute chest pain, cardiogenic shock, pulmonary congestion, and right heart failure symptoms [1]. Transthoracic echocardiography plays an essential role in rapid bedside assessment. Because the defect may not be visible in standard imaging planes, careful interrogation of all myocardial segments is required; comprehensive evaluation of intracardiac shunting includes two-dimensional imaging, color Doppler and continuous-wave Doppler [6].

The present case describes an acute coronary syndrome with delayed admission and an unsuccessful attempt at recanalization, both of which increased the risk of mechanical complications. Despite close monitoring in the intensive care unit, the diagnosis of VSR was delayed. The recurrence of severe chest pain together with new electrocardiographic changes 48 hours after admission led clinicians to suspect worsening coronary pathology and justified a second coronary angiography. On reviewing the sequence of events, this delay can be attributed to several converging factors. The bedside echocardiogram performed in the catheterization laboratory was limited to a global assessment of cardiac function, which appeared relatively preserved, and did not specifically screen for mechanical complications; the interventricular septum was not thoroughly interrogated, a limitation compounded by poor echogenicity. The delay before a more targeted re-examination was further compounded by logistical constraints, including difficulty repositioning the patient and staff shortages during the on-call shift. Left ventriculography, which might have revealed a left-to-right shunt, was not performed and is, in any case, no longer routinely used for this purpose.

This case highlights the importance of considering mechanical complications systematically in any post-infarction patient with unexplained clinical deterioration. In such situations, repeat transthoracic echocardiography should be performed promptly and, if doubt persists or in case of low echogenicity, completed by transoesophageal echocardiography (TOE). TOE can also accurately characterize the size and location of the septal defect but requires specific expertise.

Management strategies

The management strategy proposed by the European Society of Cardiology includes initial hemodynamic stabilization (diuretics, intra-aortic balloon pump, inotropes). Surgical repair is ideally delayed beyond seven days after the diagnosis to allow scar tissue formation and clear demarcation of viable myocardium; however, current guidelines recommend prompt surgery in case of persistent refractory shock [1]. Surgical treatment consists of the apposition of a synthetic or pericardial patch over the septal defect. Mortality associated with a conservative approach is extremely high, reaching 95-100% [7,8]. Although surgical repair improves survival compared with medical therapy alone, the mortality rate remains high, ranging from 19% to 60% in operated patients [9].

Mortality is particularly high when urgent surgery is performed in hemodynamically unstable patients [8]. For this reason, several authors have emphasized the importance of achieving hemodynamic stabilization with MCS prior to surgical repair. MCS is considered to be a "bridge device" used to restore the function of the failing heart, stabilize hemodynamics and allow the patient to reach the next therapeutic step. A vari-

ety of technologies are available, including the intra-aortic balloon pump, percutaneous cardiopulmonary support, VA-ECMO, percutaneous ventricular assist devices, and extracorporeal ventricular assist devices [7]. Rob et al. suggested that early VA-ECMO for post-infarction VSR complicated by cardiogenic shock, instead of emergency surgery, may represent a paradigm shift in patient management [8]. More recently, Shibasaki et al. proposed that ECpella therapy, a combination of Impella and VA-ECMO, could offer a superior bridging strategy in patients with VSR and cardiogenic shock [7].

In this case, the patient presented with a ventricular septal rupture (VSR) complicated by cardiogenic shock and hemodynamic instability. The medical team opted for immediate surgical intervention rather than a bridging strategy with mechanical circulatory support (MCS).

Reasons for not initiating VA-ECMO included the patient's advanced age, unavailability of this device at our center and subsequent delayed access to surgical intervention. Alternative devices, such as an intra-aortic balloon pump (IABP), might have been considered.

The unfavorable outcome (marked by persistent shock, progressive multiple organ failure, and recurrence of the residual shunt after surgical repair) suggests that urgent surgery without prior hemodynamic stabilization may not be the most effective management strategy, as highlighted by several recent publications.

Surgery is not the only option for closing a post-infarction septal defect. Percutaneous transcatheter closure techniques — which may avoid the morbidity and mortality of open surgery — have been described since 1988 and have been used increasingly in recent years, with encouraging results [10]. Nonetheless, percutaneous closure is not always feasible. Anatomical features of the defect — its size (< 24 mm or > 35 mm), single or multiple nature, complexity, quality of the surrounding rims, and possible involvement of the valvular apparatus — significantly influence the choice of the optimal strategy [11]. Multidisciplinary evaluation by a "Heart Team" appears central to the management of this condition and to the provision of personalized, comprehensive patient care [10].

According to data from the UK national registry (2010–2021), percutaneous and surgical repair are two viable options for the management of post-infarction VSR, with no significant difference in long-term post-discharge mortality between the two groups [10]. However, direct comparison remains difficult because of the small annual volume of percutaneous interventions, the paucity of head-to-head data, and the cross-over that occurs between modalities [11]. There is also substantial selection bias, since surgery is often proposed to younger patients while many centres reserve percutaneous closure for those deemed unsuitable for surgery [11]. A 2024 meta-analysis based on seven retrospective studies showed a statistically significant lower short-term mortality and fewer residual shunts or re-interventions with surgical closure, while no significant difference was observed in long-term mortality [12].

Conclusions

This case highlights the importance of maintaining a high index of suspicion for mechanical complications after myocardial infarction, particularly when the clinical course is atypical or unexpectedly deteriorates. Strengthening echocardiographic expertise within on-call intensive care teams may help reduce diagnostic delays in such critical scenarios. The optimal timing and modality of intervention remain subjects of debate and should be discussed within a multidisciplinary Heart Team whenever possible. However, urgent surgical repair in the setting of cardiogenic shock and hemodynamic instability does not appear to offer the best outcomes; in such cases, mechanical circulatory support should be considered as an early bridging strategy.

Declarations

Patient consent

The patient described in this case report is deceased. Written informed consent for publication of this case report and any accompanying images was obtained from the patient's next of kin. The case has been anonymized so that the patient cannot be identified from the manuscript or figures.

Authors' contributions

AB - Writing original draft, Writing review and editing, Conceptualization, Data curation.

SR - Supervision, Writing review and editing, Conceptualization.

All authors read and approved the final version of the manuscript.

Conflicts of interest

None declared.

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