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Subacute Ventricular Septal Rupture Following Elective Percutaneous Coronary Intervention for Chronic Coronary Syndrome

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Patient: Female, 71-year-old

Final Diagnosis: Ventricular septal rupture

Symptoms: Stable ischemic symptoms

Clinical Procedure: —

Specialty: Cardiology

Objective: Unusual clinical course

Background: Ventricular septal rupture (VSR) is a well-documented mechanical complication of acute coronary syndrome (ACS); however, its occurrence following elective percutaneous coronary intervention (PCI) for chronic coronary syndrome is extremely rare, with only isolated cases reported in the literature. This report describes the case of an unexpected subacute VSR after an otherwise uneventful elective revascularization.

Case Report: A 71-year-old woman with chronic coronary syndrome, diabetes, and hypertension underwent successful elective PCI to the left anterior descending artery. Despite an uncomplicated immediate procedural result, she experienced chest pain and ventricular fibrillation 6 hours post-procedure, requiring brief cardiopulmonary resuscitation and immediate defibrillation. Urgent control angiography demonstrated complete stent patency without dissection, thrombosis, or embolization. However, at 34 hours post-PCI, the patient developed sudden-onset dyspnea and rapid hemodynamic collapse. Physical examination revealed a new, harsh pansystolic murmur at the mesocardiac border. Emergency bedside transthoracic echocardiography confirmed an apical VSR with a significant left-to-right shunt. Due to ongoing severe hemodynamic collapse and an extremely high surgical risk, percutaneous transcatheter closure was attempted as a bail-out procedure; however, the patient died during the intervention in the catheterization laboratory. No postmortem tissue exam or direct procedural anatomical inspection of the VSR site was feasible.

Conclusions: This case indicates that life-threatening mechanical complications like VSR can potentially occur outside the traditional paradigm of ACS, extending into the setting of elective revascularization for severe, chronic lesions. Rather than establishing a broad clinical standard, this report highlights the critical importance of post-procedural vigilance and a low threshold for prompt bedside echocardiographic evaluation whenever unexpected hemodynamic instability or a new murmur arises following PCI.

Keywords: Coronary Angiography • Rupture • Ventricular Septal Rupture

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Introduction

Ventricular septal rupture (VSR) remains one of the most catastrophic mechanical complications of myocardial infarction, carrying an extremely high mortality rate that exceeds 40% to 50% even with contemporary surgical or transcatheter interventions (1). While VSR occurs in approximately 0.2% to 0.5% of patients presenting with acute ST-segment elevation myocardial infarction in the modern reperfusion era, it is extraordinarily rare following elective percutaneous coronary intervention (PCI) for stable angina/chronic coronary syndrome [1,2]. Rather than claiming absolute uniqueness, this case serves as evidence of an incremental boundary condition, illustrating that the clinical spectrum of VSR may extend beyond classic acute coronary syndrome (ACS) scenarios to include complex elective interventions for chronic, high-grade lesions.

Case Report

The patient, a 71-year-old woman, presented with stable ischemic symptoms. Her baseline arterial blood pressure was 130/70 mm Hg, and her heart rate was 75 beats per minute. Cardiac auscultation revealed a grade 2/6 systolic murmur at the mitral area, while other physical systems were unremarkable. Electrocardiography (ECG) demonstrated sinus rhythm without acute ischemic changes or arrhythmias. Outpatient transthoracic echocardiography (TTE) prior to the procedure showed a preserved left ventricular ejection fraction of 58% (calculated via the modified Simpson method) and mild mitral regurgitation (Figure 1). Baseline cardiac troponin levels were within normal physiological limits.

Her medical history was significant for diabetes mellitus, hypertension, and non-critical stenosis in the left anterior descending (LAD) artery shown by coronary angiography (CAG) performed 6 years prior. These conditions had been managed conservatively. Her home medications included acetylsalicylic acid 100 mg, nebivolol 5 mg, atorvastatin 20 mg, candesartan 16 mg, metformin 1000 mg, and linagliptin 5 mg. Her current presentation was attributed to progressive coronary atherosclerosis.

Elective CAG revealed 90% stenosis in both the proximal LAD and the first diagonal branch. A drug-eluting stent (Xience) was successfully deployed across the lesion without immediate angiographic complications. Approximately 6 hours post-procedure, the patient experienced recurrent chest pain rapidly degenerating into ventricular fibrillation (VF). Sinus rhythm and hemodynamic stability were restored following immediate defibrillation and brief (< 1 minute) cardiopulmonary resuscitation. Following resuscitation from VF at hour 6 post-PCI, an immediate cardiac auscultation was performed and



Figure 1. Outpatient baseline transthoracic echocardiogram prior to the procedure showing preserved left ventricular ejection fraction and normal interventricular septum structure.

was completely normal, showing no new or unexpected cardiac murmurs. However, serial physical examinations were not structured or documented over the subsequent hours, and an immediate post-arrest bedside TTE was not obtained. Serial 12-lead ECGs revealed no dynamic ST-segment elevations or new Q waves, though non-specific repolarization abnormalities persisted. Serial cardiac biomarker measurements were initiated; high-sensitivity cardiac troponin I levels demonstrated a progressive rise from normal baseline levels, peaking post-arrest (troponin I: 4.85 ng/mL at hour 12; upper reference limit: 0.05 ng/mL). Urgent control CAG re-evaluation confirmed a fully patent LAD stent and verified the absence of acute stent thrombosis, epicardial coronary artery dissection, distal embolization, side-branch occlusion, or angiographically evident coronary vasospasm. Because initial hemodynamic stability was restored and early post-arrest auscultation remained unremarkable, the VF episode was clinically attributed to transient electrical instability or reperfusion arrhythmia rather than an acute mechanical defect. Consequently, mechanical complications were not actively suspected, and follow-up physical examinations and TTE evaluation were not repeated until the sudden clinical collapse at hour 34.

The patient was transferred to the Coronary Intensive Care Unit. However, 34 hours post-PCI, she developed sudden-onset dyspnea, orthopnea, and rapid hemodynamic collapse. Physical examination revealed a new, harsh pansystolic murmur audible at the mesocardiac border. Emergency bedside TTE confirmed an apical VSR featuring a significant left-to-right shunt (Figure 2). Due to the patient's severe hemodynamic instability, primary surgical repair was anticipated to carry an extremely high mortality risk. Due to the patient's severe hemodynamic

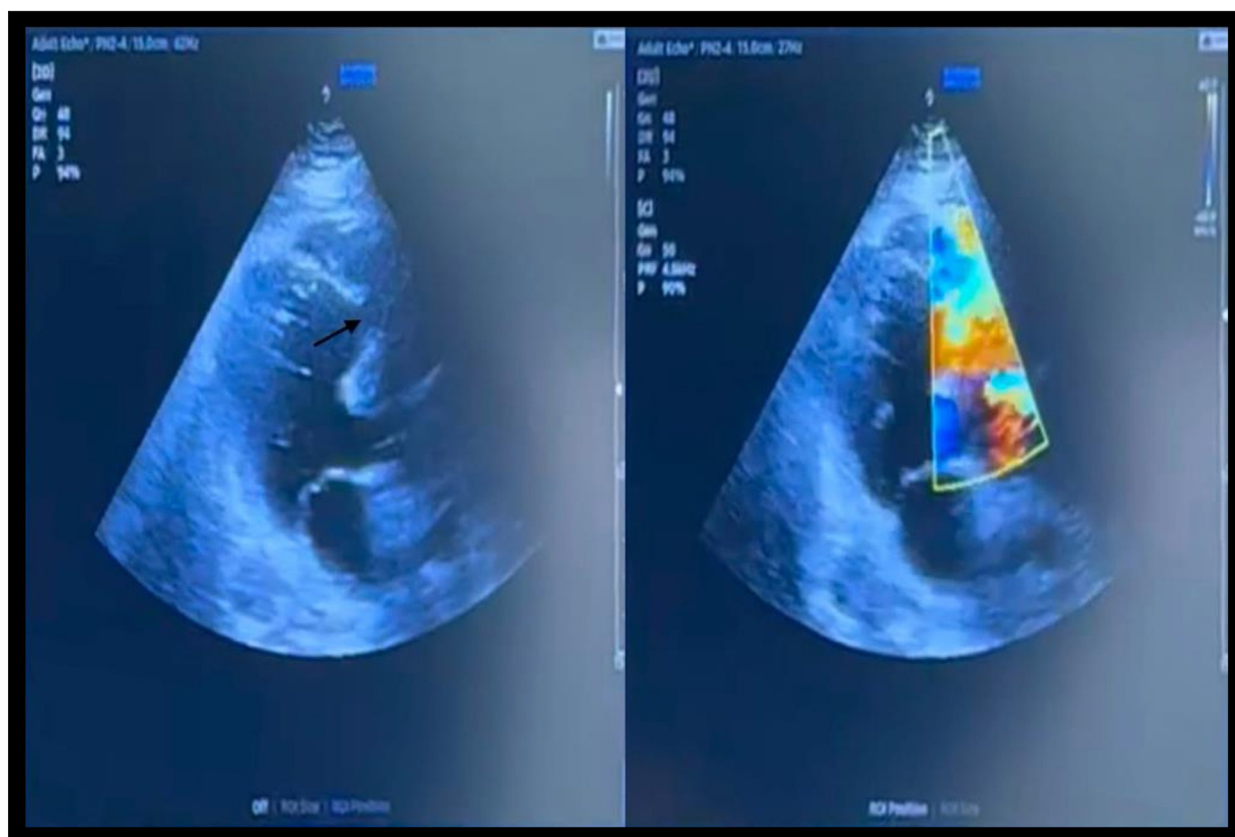


Figure 2. Emergency bedside transthoracic echocardiogram performed 34 hours post-procedure, demonstrating a distinct apical ventricular septal rupture with a significant left-to-right shunt.

collapse, the mortality risk from primary surgical repair was deemed to be prohibitively high. Consequently, urgent percutaneous transcatheter closure was attempted as a bail-out intervention. However, owing to the rapid and fulminant cardiogenic collapse, the patient experienced fatal cardiac arrest during the catheterization procedure before successful device placement could be achieved. No direct procedural or post-mortem anatomical findings of the defect site were available.

No postmortem autopsy or tissue histopathological examination was performed due to lack of family consent / institutional protocol for post-procedural mortality. Consequently, the proposed pathophysiological mechanisms are based on clinical, angiographic, and echocardiographic observations rather than postmortem pathological confirmation.

Discussion

The timeline of VSR development in this patient provides important insights into a plausible, though not definitively proven, underlying pathophysiological mechanism. In the pre-reperfusion era, VSR typically manifested 3 to 5 days post-myocardial infarction due to coagulation necrosis [1-3]. However, in the

contemporary era of rapid reperfusion, the temporal distribution has shifted, exhibiting a bimodal peak where an early cohort presents within the first 24 to 48 hours [4]. Our patient's clinical deterioration at 34 hours fits within this subacute window.

Because the initial PCI was angiographically successful and devoid of procedural dissections or perforations, direct mechanical trauma by wires or balloons can be reasonably excluded. Instead, the sudden onset of VF at the 6th hour and subsequent VSR at the 34th hour allow us to hypothesize a mechanistic account driven by a complex interplay of reperfusion injury, microvascular dysfunction, and tissue degradation.

One possible mechanism is that severe reperfusion injury manifested as intramyocardial hemorrhage and hemorrhagic infarction [5]. While the patient carried a diagnosis of chronic coronary syndrome, the 90% proximal LAD and first diagonal branch stenosis subtended a large territory of the anterior wall and interventricular septum. Long-standing, high-grade stenosis often leads to chronic adaptations in the downstream microvasculature [6]. The sudden, high-pressure restoration of epicardial blood flow into this vulnerable, chronically underperfused vascular bed may have induced profound endothelial disruption, capillary rupture, and subsequent intramyocardial

hemorrhage [7]. It is critical to note, however, that intramyocardial hemorrhage was not directly confirmed via advanced imaging or histopathological analysis in this case; it remains a proposed explanation consistent with the clinical course rather than an established finding.

In this conceptual framework, the cardiac arrest episode at hour 6 post-PCI represents a critical variable in the pathophysiological timeline. It is entirely plausible that the primary insult leading to cardiac arrest, whether profound localized ischemia, intense microvascular dysfunction, or acute intramyocardial hemorrhage, served as the initial trigger for septal structural disruption. Because TTE was not performed immediately after resuscitation and explicit serial cardiac auscultations documenting the absence of a murmur were lacking between hours 6 and 34, the possibility that a subclinical micro-rupture or intramyocardial dissection originated during or immediately following the arrest cannot be excluded. Over the subsequent hours, ongoing hemodynamic wall stress, enzymatic degradation, and tissue softening likely accelerated the propagation of this lesion, culminating in a full-thickness defect and clinical collapse at hour 34. Furthermore, while the cardiopulmonary resuscitation performed during the VF episode was brief (< 1 minute), a potential contributing role of external chest compressions in generating acute mechanical trauma or stress on an already vulnerable and ischemia-weakened septum remains a plausible factor that cannot be entirely ruled out [5-7].

This case also highlights how traditional cardiovascular risk factors might synergistically predispose a patient to such tissue-level complications. The patient's history of long-standing hypertension and diabetes mellitus likely altered myocardial resilience. Chronic hypertension induces hypertrophic changes and structural remodeling, which can modify shear stress vectors across the septum [8,9]. Concurrently, diabetes mellitus impairs collagen cross-linking and microvascular endothelial function, potentially rendering the myocardial tissue more susceptible to accelerated necrosis and less resilient to acute volumetric shifts following reperfusion [10].

Crucially, the control CAG performed immediately after the VF episode revealed a completely patent stent and no macrovascular flow abnormalities. This provides a vital clinical lesson: a normal control angiogram does not exclude the evolution of tissue-level or mechanical complications. When our patient experienced sudden dyspnea and hemodynamic collapse at 34 hours, the prompt auscultation of a new pansystolic murmur at the mesocardiac area immediately redirected clinical suspicion toward a mechanical defect, which was confirmed via emergency bedside TTE.

Additionally, in patients presenting with acute post-PCI VSR and profound hemodynamic collapse, emergency surgical intervention carries exceedingly high operative mortality rates. Although percutaneous transcatheter closure represents a potential bail-out option or bridge to surgery in high-risk candidates, rapidly progressive cardiogenic shock can prevent successful device deployment during the procedure, leading to fatal outcomes as observed in this case. Because neither direct intraoperative visualization nor postmortem histopathological analysis was available, the detailed tissue-level mechanisms described remain a proposed clinical interpretation.

Conclusions

This case underscores that life-threatening mechanical complications such as VSR, though rare, can potentially occur following elective percutaneous revascularization of severe, chronic lesions. Rather than establishing a new broad standard of care or implying a paradigm shift from a single observation, this report serves as a focused reminder of the critical importance of post-procedural vigilance. Clinicians should maintain an awareness of these rare sequelae and have a low threshold for prompt clinical reassessment specifically utilizing early bedside echocardiography whenever a patient develops unexpected hemodynamic instability or a new cardiac murmur in the post-PCI period.

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Department and Institution Where Work Was Done

Karaman Training and Research Hospital, Karaman, Türkiye.

Patient Consent

Written informed consent was obtained from the patient's next of kin for the publication of this case report and any accompanying clinical images.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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