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CASE REPORT

BEGINNER

CLINICAL CASE

A Case of Post-Myocardial Infarction Ventricular Septal Rupture Complicated by Postoperative Septal Rupture

Kara F. Morton, MD,[a] Usman A. Hasnie, MD,[b] Darryl Prime, MD,[b] Sasha A. Still, MD,[c] Samuel McElwee, MD[b]

ABSTRACT

We present the case of a 60-year-old man who presented with a post-myocardial infarction ventricular septal rupture caused by a delayed presentation of myocardial infarction. Despite revascularization, hemodynamic stability, and a 10-day delay until operative management to allow for tissue healing, the patient experienced a fatal recurrent postoperative ventricular septal rupture. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2023;22:101996)

HISTORY OF PRESENTATION

A 60-year-old man presented to the emergency department with chest discomfort and dyspnea. Symptoms had begun 5 days earlier with poor appetite, nausea, and diaphoresis. The following day, he endorsed dyspnea at rest and was evaluated at an urgent care facility. He was prescribed steroids and antibiotics for bronchitis. His symptoms persisted, and he presented to our institution. Physical examination showed a blood pressure of 109/90 mm Hg and tachypnea. An electrocardiogram on arrival revealed 2-mm ST-segment elevations with T-wave inversions in the inferior leads with reciprocal ST-segment depressions in leads I and aVL (**Figure 1**). Pathologic Q waves were noted in the inferior leads. High-sensitivity troponin was 9,543 ng/L (reference range: 3-20 ng/L), which remained flat on repeat at 9,586 ng/L.

LEARNING OBJECTIVES

- To identify a post-MI VSR with left-to-right shunt physiology infarction and review the expected hemodynamic changes.
- To evaluate approaches to the management and repair of post-MI VSR with subsequent cardiogenic shock.

PAST MEDICAL HISTORY

The patient had no past medical history but had smoked one-half pack of cigarettes daily for the past 30 years and drank 2 to 3 liquor-containing drinks daily.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis included subacute inferior ST-segment elevation myocardial infarction (MI), post-MI mitral regurgitation from chordae tendineae

From the [a]Department of Medicine, Heersink School of Medicine at the University of Alabama at Birmingham, Birmingham, Alabama, USA; [b]Division of Cardiovascular Disease, Heersink School of Medicine at the University of Alabama at Birmingham, Birmingham, Alabama, USA; and the [c]Division of Cardiothoracic Surgery, Department of Surgery, University of Alabama at Birmingham, Birmingham, Alabama, USA.

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ABBREVIATIONS AND ACRONYMS

LV = left ventricle

MI = myocardial infarction

RCA = right coronary artery

RV = right ventricle

VSR = ventricular septal rupture

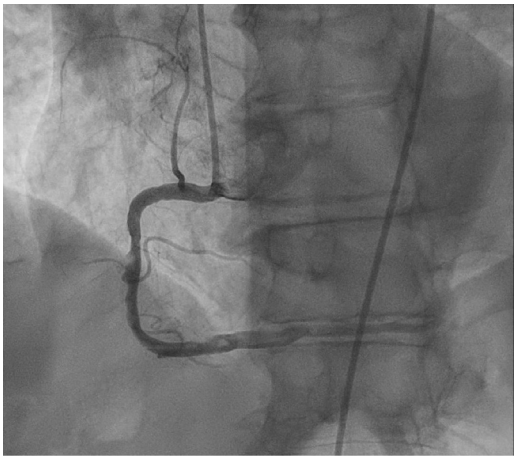
or papillary muscle rupture, ventricular septal rupture (VSR), free wall rupture, or ventricular aneurysm.

INVESTIGATIONS

Coronary angiography revealed thrombus throughout the right coronary artery (RCA) with plaque rupture in the mid-RCA (**Figure 2**, Video 1). Multiple runs of aspiration thrombectomy were performed, and intravascular ultrasound was used to guide the placement of 6 overlapping drug-eluting stents throughout the RCA, which were selected because of institutional availability (**Figure 3**, Video 2).

Because of persistent dyspnea, an intraprocedural echocardiogram was performed and demonstrated a VSR measuring 0.8 cm at the largest diameter, with a serpiginous path in the midinferoseptum with severe right ventricle (RV) dilation. No aneurysm was visualized. The left ventricular (LV) ejection fraction was normal and the basal, midinferior, midinferoseptal, and midinferolateral segments were hypokinetic (**Figure 4**, Videos 3 and 4). Following revascularization, a right heart catheterization revealed elevated filling pressures and a depressed cardiac index (**Table 1**). A shunt run confirmed the presence of a left-to-right interventricular shunt with a step-up in oxygen saturation from the right atrium (50%) to RV (93%). The shunt fraction (Q_p/Q_s) was 4.08.

FIGURE 2 Preintervention Coronary Angiography

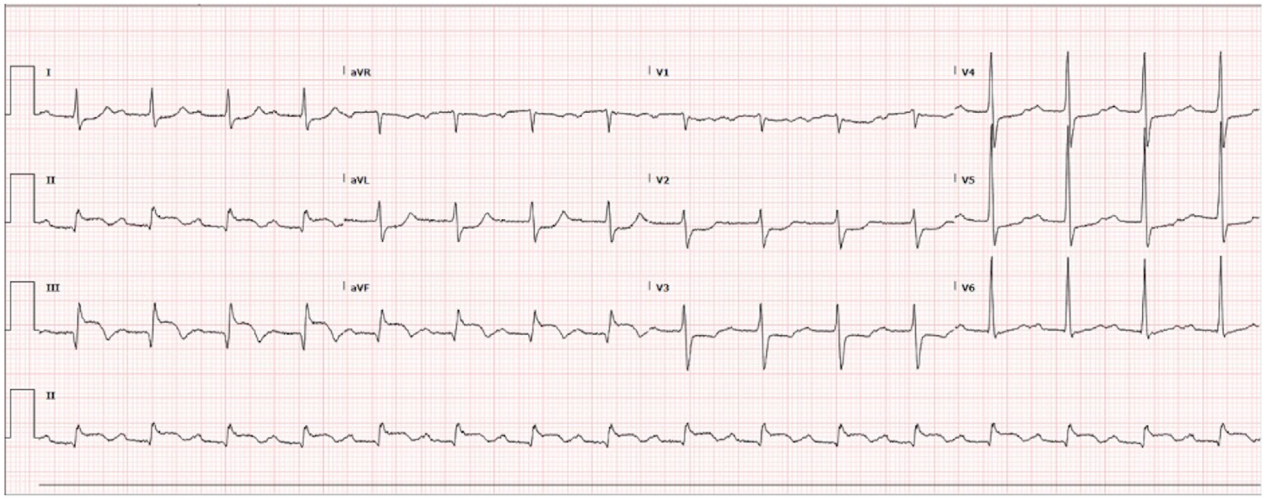


Angiography showing significant thrombus throughout the right coronary artery.

MANAGEMENT

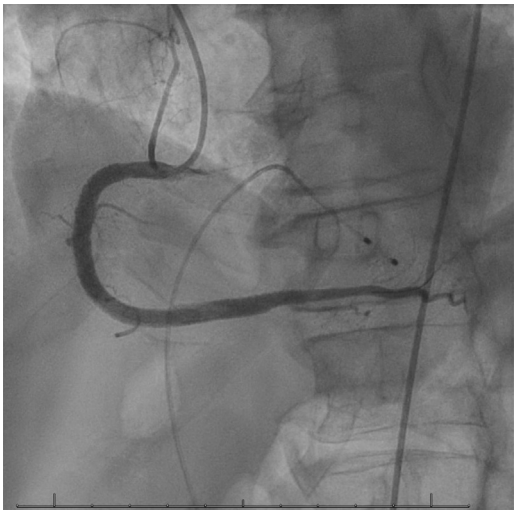
An intra-aortic balloon pump was placed to temporize the shunt physiology. The serum lactic acid improved from 3.4 mmol/L to 1.2 mmol/L (reference range: 0.5-2.2 mmol/L), and blood pressure stabilized within 24 hours after placement. A heart team approach was taken, and surgical repair of the VSR was planned.

FIGURE 1 Initial Electrocardiogram



Electrocardiogram consistent with subacute inferior ST-segment elevation myocardial infarction.

FIGURE 3 Postintervention Coronary Angiography



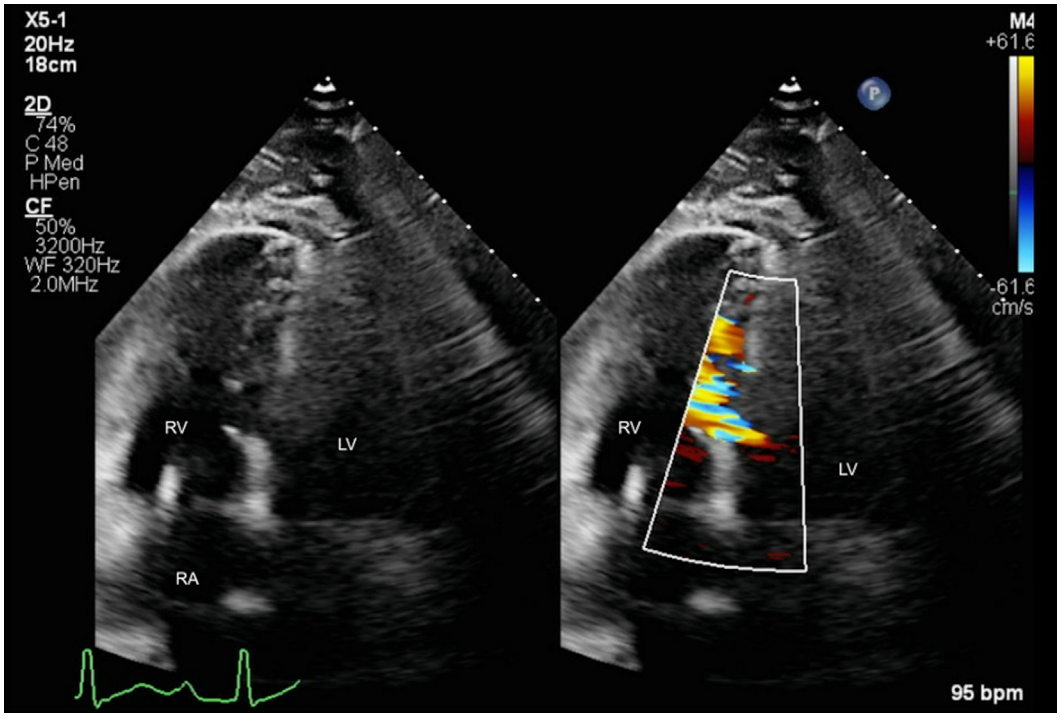
Revascularization of the right coronary artery with 6 overlapping drug-eluting stents.

The patient was upgraded to an axillary LV assist device to allow greater mobility. ST-segment elevations resolved on electrocardiogram within 48 hours of revascularization, and the patient remained free of chest pain and electrically stable and did not require vasopressor support. However, he progressively developed pulmonary edema and RV dysfunction despite diuresis, so surgical repair was undertaken 10 days later (Video 5).

Intraoperatively, a 3-cm defect from the RV to LV was noted posterior to the septal leaflet of the tricuspid valve with a large, circumferential partial thickness tear around the VSR extending nearly 3 cm beyond the distal margin of the defect itself (**Figure 5A**). A patch was sewn deeply into the endocardium of the LV, spanning the entire partial thickness injury (**Figure 5B**). The tricuspid valve was replaced given the proximity to the septal leaflet. Postoperatively, there was no evidence of shunt on or off bypass.

On postoperative day 1, the patient had a mucus-plugging event with hypoxia, triggering ventricular tachycardia arrest. Emergent bedside sternotomy and

FIGURE 4 Transthoracic Echocardiogram



Transthoracic echocardiogram demonstrating posterior ventricular septal rupture with left-to-right shunting. LV = left ventricle; RA = right atrium; RV = right ventricle.

TABLE 1 Hemodynamics on Right Heart Catheterization

Right atrial pressure	22 mm Hg
Right ventricular pressure	51/16 (mean 22) mm Hg
Pulmonary artery pressure	48/25 (mean 35) mm Hg
Pulmonary capillary wedge pressure	27/35 (mean 24) mm Hg
Cardiac output (Fick)	2.82 L/min
Cardiac index (Fick)	1.28 L/min/m^2

cardiac massage were performed, and the patient was cannulated for extracorporeal membrane oxygenation. Repeat transesophageal echocardiogram revealed a new VSR with bidirectional flow located near the commissure between the right coronary cusp and noncoronary cusp of the aortic valve with severe depression of RV systolic function (Figure 6).

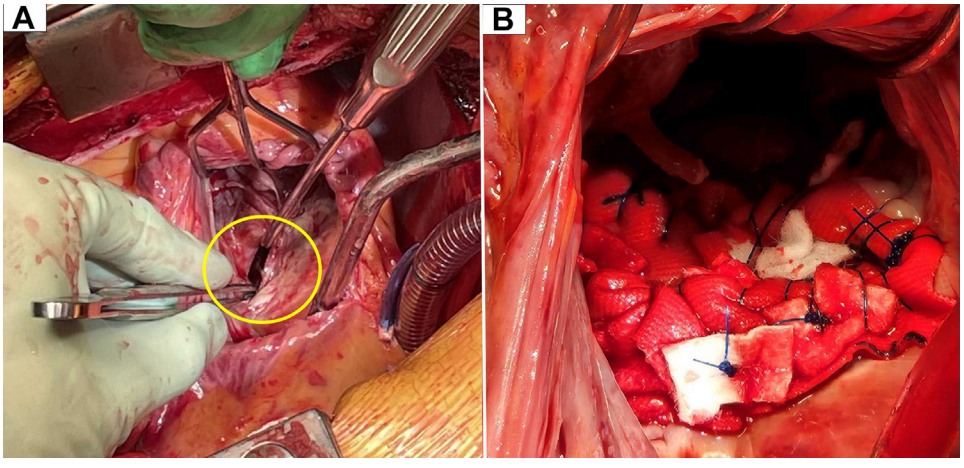
His hospital course was further complicated by renal failure, atrial fibrillation with rapid ventricular response, and incessant polymorphic ventricular tachycardia. Ultimately, because of clinical decline, ongoing arrhythmia, and positioning of the new VSR near the aortic valve, he was not a candidate for repeat surgical repair. The family decided to pursue a comfort-based approach, and the patient died 23 days from the index percutaneous coronary intervention.

DISCUSSION

This case highlights the high mortality from mechanical complications conferred by delayed-presentation MI. A post-MI VSR can often be diagnosed clinically with the presence of a new systolic murmur, a palpable thrill, and shock.[1,2] Since the inception of reperfusion techniques, the incidence of post-MI VSR has declined from 1% to 2% to < 0.2%.[2-4] Post-MI VSR is also diagnosed sooner, which may be attributable to greater use of echocardiography or altered pathophysiology of viable reperfused tissue.[2-4] Even with prompt revascularization and surgical repair, post-MI VSR still carries a mortality risk of between 20% and 87%.[1] A greater delay between symptom onset and presentation for lytics or percutaneous coronary intervention also increases the likelihood of post-MI septal rupture, highlighting the importance of prompt door-to-balloon time.[5]

Guidelines recommend surgical repair when a post-MI VSR is suspected regardless of hemodynamic stability, but this often differs from clinical practice.[1] Clinical instability often deters surgical teams from urgent operative intervention given the higher mortality risk. Some studies also show that delayed time to surgical repair for 3 to 4 weeks if hemodynamics allow may improve outcomes because of scar tissue formation and less tissue fragility around the site of interest.[6-8] This may contribute to some degree of surgical bias, with the more stable patients being selected more often for surgery to reduce negative surgical outcomes. However, more than half of patients with post-MI VSR experience cardiogenic shock requiring invasive support devices and early surgical repair, and mortality remains high.[7] Inferior-basilar septal ruptures also carry a 1.73-times higher mortality than an anterior-apical defect because of the more difficult intraoperative access.[7] Consequently, a

FIGURE 5 Intraoperative Photos



Ventricular septal rupture **(A)** pre- and **(B)** post-surgical closure with patch.

challenge exists in the decision to pursue early vs delayed surgery to improve mortality in these cases; a heart team approach involving surgical teams and cardiac intensivists is therefore imperative to consider each patient's overarching clinical trajectory.

FOLLOW-UP

After our patient's death, his family respectfully declined autopsy.

CONCLUSIONS

Prompt identification and reperfusion of acute MI remains the most effective method of preventing post-MI structural complications. The incidence of postinfarction VSR has declined with reperfusion, though mortality remains high despite surgical repair. If hemodynamics can be stabilized, delayed surgical repair of VSR may reduce mortality. However, a heart team approach is essential when weighing the risk of mortality from cardiogenic shock against the risk of operative complications to reduce potential surgical bias.

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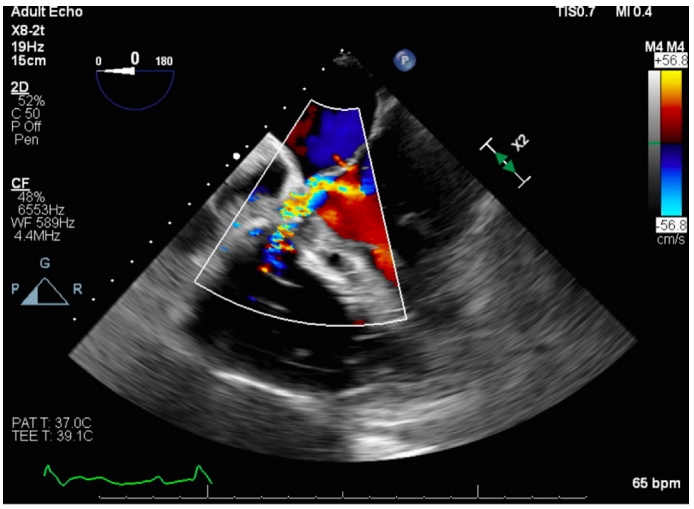
The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

FIGURE 6 Transesophageal Echocardiogram



Transesophageal echocardiogram postarrest showing new ventricular septal rupture near the aortic valve.

ADDRESS FOR CORRESPONDENCE: Dr Kara Morton, Tinsley Harrison Internal Medicine Residency Program BDB 327, 1720 2nd Avenue South, Birmingham, Alabama 35294-0012, USA. E-mail: kfmorton@uabmc.edu. @KaraMortonMD.

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KEY WORDS cardiogenic shock, coronary interventions, mechanical complications, myocardial infarction, noncoronary interventions, ventricular septal rupture

APPENDIX For supplemental videos, please see the online version of this paper.