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CASE REPORT

BEGINNER

CLINICAL CASE

# Post-Myocardial Infarction Free-Wall Rupture

## Rapid Diagnosis and Management

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**ABSTRACT**

We present a case of post-myocardial infarction free-wall rupture in a critically ill patient presenting to the emergency department. Through our case we highlight the prompt evaluation, diagnosis, and management necessary to improve survival in a patient with this life-threatening condition. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2023;18:101915) © 2023 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

### HISTORY OF PRESENTATION

A 69-year-old man was brought in by ambulance after he was found unresponsive at a train station. On arrival, roughly 30 minutes after symptom onset, he was hypotensive to 75/50 mm Hg, with jugular venous distention, muffled heart sounds, and diminished pulses bilaterally. He was initiated on vasopressor agents for shock and was unresponsive to fluids. A prehospital electrocardiogram demonstrated ST-segment elevations in the inferior leads (**Figure 1**), consistent with an inferior ST-segment elevation myocardial infarction (STEMI). An initial high-sensitivity troponin test returned a value >16,000 ng/L. A point-of-care ultrasound examination demonstrated pericardial effusion and heterogenous material causing acute right ventricular compression with possible cardiac tamponade. The patient provided consent for his case to be reported.

**LEARNING OBJECTIVES**

- To understand the risk factors for and presentation of post-MI FWR.
- To understand the role of echocardiography in diagnosing FWR and recognize supportive echocardiographic findings.
- To understand typical management strategies of FWR, including the role of mechanical circulatory support and revascularization before surgical intervention.

### PAST MEDICAL HISTORY

The patient had a history of hypertension.

### DIFFERENTIAL DIAGNOSIS

Given the patient's hypotension, diminished peripheral pulses, inferior STEMI, and pericardial effusion, the initial concern was possible aortic dissection

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**ABBREVIATIONS AND ACRONYMS**

**CRP** = C-reactive protein

**CT** = computed tomography

**FWR** = free-wall rupture

**IABP** = intra-aortic balloon pump

**MI** = myocardial infarction

**PCI** = percutaneous coronary intervention

**PPV** = positive predictive value

**STEMI** = ST-segment elevation myocardial infarction

extending into the right coronary artery vs a mechanical complication such as free-wall rupture (FWR).

## INVESTIGATIONS

The patient underwent chest computed tomography (CT), which had negative findings for aortic dissection, but the imaging did demonstrate a pericardial effusion compatible with hemopericardium (**Figure 2**). He then became obtunded and was intubated for airway protection. He was taken to the cardiac catheterization laboratory for further evaluation and stabilization. Coronary angiography showed left-dominant circulation and total occlusion of the mid-left circumflex artery with involvement of obtuse marginal artery branch 2 (Video 1). An intraprocedural echocardiogram demonstrated a moderate pericardial effusion and a large amount of heterogenous material consistent with a clot causing nearly complete compression of the right ventricle (Video 2).

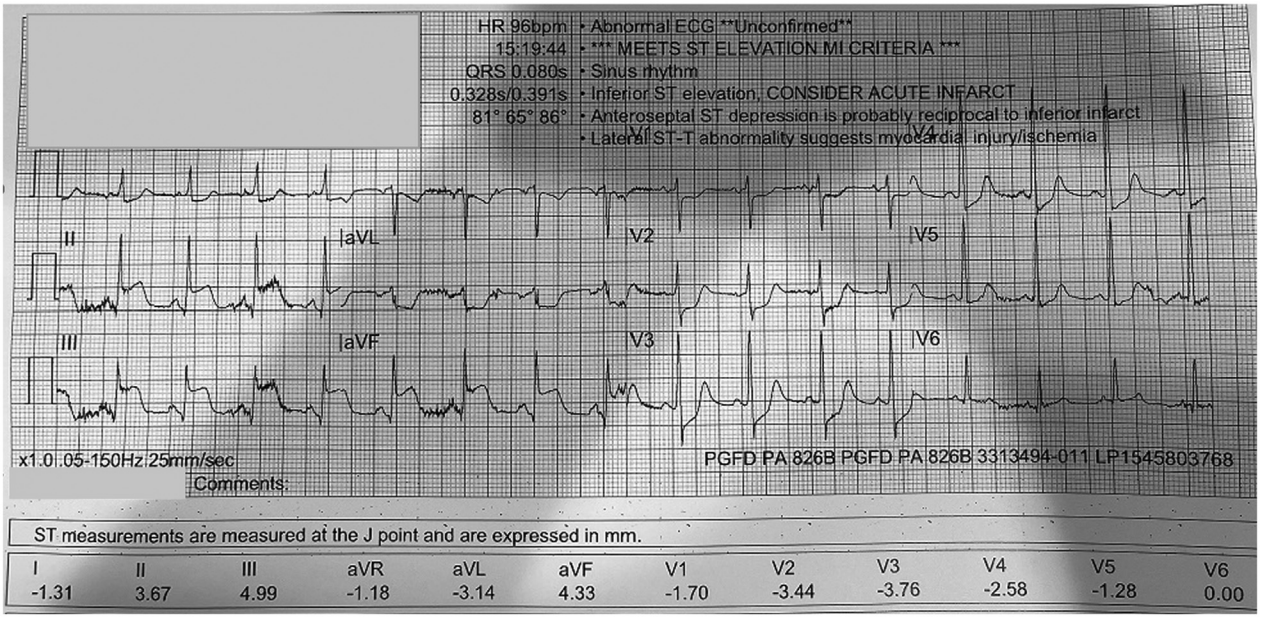
## MANAGEMENT

The patient underwent mechanical aspiration thrombectomy and percutaneous balloon angioplasty (door-to-balloon time of 65 minutes), with restoration of TIMI flow grade 3 (Video 3). Afterward, an intra-aortic balloon pump (IABP) was placed. Despite pharmacologic and mechanical support, the patient continued to be in profound cardiogenic shock. Thus, he underwent emergency pericardiocentesis, with removal of 100 mL of frank blood. After initial improvement of his hemodynamics, he was transferred to a nearby hospital for emergency cardiothoracic surgery. He underwent sternotomy, and his pericardium was opened, with evacuation of 500 mL of blood and large amounts of clot. A large area of infarct was found in the distal circumflex territory, which was repaired using a pericardial patch and BioGlue (CryoLife). The patient was then transferred to the intensive care unit for further monitoring.

## DISCUSSION

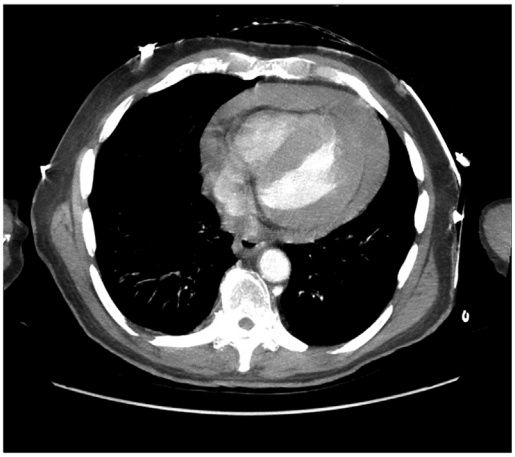
FWR is a frequently fatal complication of acute myocardial infarction (MI), with an in-hospital mortality rate of 80%.[1] Its estimated incidence has decreased to as low as 0.01% since the widespread adoption of reperfusion therapy, although the true incidence is likely higher because a significant portion of patients experience sudden death before hospital evaluation.[2-4] For patients presenting with a

**FIGURE 1 Electrocardiogram**




Prearrival electrocardiogram demonstrating ST-segment elevation in the inferior leads consistent with an inferior ST-segment elevation myocardial infarction.

**FIGURE 2** **Computed Tomography**



Imaging demonstrating moderate complex pericardial effusion compatible with hemopericardium. No aortic dissection was visualized, thus raising concern for free-wall rupture.

window of opportunity for intervention, emergency surgery is key to survival.[2] As a result, it is important for clinicians to diagnose the condition promptly.

FWR usually occurs within 7 days of MI.[2] Observational studies have frequently reported age >70 years, female sex, and lower rates of percutaneous coronary intervention (PCI) as risk factors for rupture. Conditions thought to promote the development of collateral circulation, such as hypertension, diabetes, and previous MI, have been negatively correlated with rupture, although this correlation has been inconsistent across studies.[1-5]

Patients with FWR may present with chest pain, restlessness, hemodynamic compromise, or cardiogenic shock. Further, more than 80% of patients present with associated cardiac tamponade. Thus, physical examination findings may be significant for a raised jugular venous pulse, muffled heart sounds, or pulsus paradoxus.[6]

Elevated cardiac biomarkers and C-reactive protein (CRP) have also been associated with rupture. In a single-center retrospective study, patients with FWR had an average troponin level of 8.6 ng/mL and CRP value of 50 mg/L, compared with 0.5 ng/mL and 5.3 mg/L, respectively, in post-MI patients without rupture.[1-5] Clinically, FWR manifests either acutely or subacutely. Acute rupture generally manifests with electromechanical dissociation and cardiogenic shock secondary to tamponade and often does not allow for any possible treatment. Subacute rupture can manifest with cardiogenic shock and tamponade and allows time for surgical intervention. The rupture itself can be divided into "blowout" or "oozing" subtypes. Blowout ruptures are often associated with hemodynamic instability or cardiac arrest, whereas oozing ruptures vary in terms of severity.[5,6]

Echocardiography is the most important test for prompt diagnosis of FWR. In 1 study, the presence of a pericardial effusion (>5 mm) was 100% sensitive and 93% specific, although the positive predictive value (PPV) was only 27%. Findings associated with a PPV ≥70% included pericardial effusion >15 mm, cardiac tamponade, right ventricular compression, electromechanical dissociation, and hemopericardium. The combination of a pericardial effusion >5 mm, tamponade, and intrapericardial echocardiograms had a PPV >90%.[7] In our case, the presence of a pericardial effusion, tamponade, and findings consistent with a pericardial clot on echocardiography was strongly supportive of FWR. CT evidence of hemopericardium without aortic dissection was further supportive. Emergency CT is thus a valuable adjunct to echocardiography because it can differentiate between FWR and intrapericardial extension of an ascending aortic dissection, both of which can manifest with hemopericardium and hemodynamic collapse.[8]

Initial treatment of FWR involves volume resuscitation and hemodynamic support. Mechanical ventilation is generally avoided in the setting of tamponade because of hemodynamic collapse, but it was required in this case and was successfully implemented with minimal ventilatory settings and low positive end-expiratory pressure. In our patient, an IABP was inserted for hemodynamic support, and PCI was performed before surgical intervention. Preoperative or intraoperative insertion of mechanical support is warranted to provide temporary hemodynamic stabilization and reduce wall stress, which can prevent oozing ruptures from transforming to blowout ruptures or reduce the risk of repeat rupture postoperatively. However, studies have not consistently demonstrated survival benefits. Cardiac catheterization before surgery is also controversial. Some investigators note that revascularization has a positive impact on survival, whereas others recommend avoiding coronary angiography in the interest of time. Overall, studies have not demonstrated survival benefits of concomitant revascularization.[6,9,10] Definitive treatment of FWR almost always requires surgical intervention. Surgical techniques used to

treat FWR are divided into sutured and sutureless categories. Sutured techniques use sutures to close the myocardial tear or secure a patch to the infarcted myocardium, whereas sutureless techniques use tissue adhesives to secure a patch to the infarcted myocardium. The choice of surgical technique is controversial, although postoperative mortality rates are similar between the 2 techniques.[9,10]

## FOLLOW-UP

The patient had a prolonged hospital course but eventually stabilized and was discharged to a long-term acute care hospital.

## CONCLUSIONS

FWR is a rare but devastating complication of acute MI with a high in-hospital mortality rate. Risk factors include age >70 years, female sex, and lack of PCI post-MI. Patients with FWR may also present with elevated troponin and CRP levels. Prompt diagnosis using echocardiography and emergency surgical intervention are key to survival.

## FUNDING SUPPORT AND AUTHOR DISCLOSURES

Dr Waksman has served on the advisory boards of Abbott Vascular, Boston Scientific, Medtronic, Philips IGT, and Pi-Cardia Ltd; has served as a consultant for Abbott Vascular, Biotronik, Boston Scientific, Cordis, Medtronic, Philips IGT, Pi-Cardia Ltd, Swiss Interventional Systems/SIS Medical AG, Transmural Systems Inc, and Venous MedTech; has received institutional grant support from Amgen, Biotronik, Boston Scientific, Chiesi, Medtronic, and Philips IGT; and has invested in MedAlliance and Transmural Systems Inc. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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**KEY WORDS** free-wall rupture, myocardial infarction

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