

CASE REPORT

ADVANCED

CLINICAL CASE

Ventricular Rupture at the Site of a Septic Myocardial Abscess After Acute Myocardial Infarction



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ABSTRACT

Mechanical complications after acute myocardial infarction are well-described yet catastrophic complications of acute coronary syndromes. Uniquely, we describe a rare case of left ventricular free wall rupture at the site of a septic myocardial abscess after an anterior wall myocardial infarction. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2022;4:1484-1489) © 2022 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 72-year-old man presented to the emergency department with a several-day history of progressive shortness of breath and intermittent chest pain. Initial assessment found the patient to have an altered level of consciousness with significant respiratory distress. His vital signs were as follows: heart rate of 122 beats/min, blood pressure of 86/46 mm Hg, respiratory rate of 43 breaths/min, oxygen saturations of 78% on room air, and a temperature of 37.8°C (100°F).

LEARNING OBJECTIVES

- To be aware of septic myocardial abscess formation as a rare complication of ACS.
- To be familiar with the clinical features and the typical clinical course, investigation, and management strategies of patients with myocardial infarction complicated by myocardial abscess formation.

Notable physical examination findings included: sinus tachycardia, thready central and peripheral pulses, elevated jugular venous pressure, S1 and S2 were present in the absence of murmurs or extra heart sounds, extremities were mottled and cool with bilateral peripheral edema, breath sounds were diminished at the lung bases with diffuse crepitations bilaterally, and abdominal examination was unremarkable.

Given the patient's acuity and shock state at the time of presentation requiring emergent mechanical ventilation and an inability to obtain a collateral history because the patient was alone, further detail with respect to his history of presenting illness and specifically infectious symptomatology were unable to be obtained.

PAST MEDICAL HISTORY

The patient's known medical diagnoses were as follows: hypertension, dyslipidemia, type 2 diabetes mellitus on oral anti-hyperglycemic medications, and

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an active 40 pack-year smoking history. The patient had a remote history of atypical chest discomfort with myocardial perfusion imaging demonstrating mild ischemia in the left anterior descending artery (LAD) territory. He was initiated on medical therapy and a coronary angiogram was scheduled. The patient unfortunately did not attend his appointment and thus his coronary anatomy and burden of coronary artery disease was unknown at the time of presentation.

INVESTIGATIONS

The patient's chest X-ray was consistent with severe interstitial pulmonary edema and electrocardiogram demonstrated inferolateral ST-segment elevation (Figures 1 and 2). Pertinent laboratory results were as follows: white blood cell count $8.4 \times 10^9/L$, hemoglobin 104 g/L, whole blood lactate 4.0 mmol/L, creatinine kinase 1,178 μ/L , and high sensitivity troponin T 3,482 ng/L.

The patient was diagnosed with a Killip IV inferolateral ST-segment elevation myocardial infarction, he was intubated for hypoxemic respiratory failure, and he was sent for emergency coronary angiogram. Coronary angiogram demonstrated a culprit lesion in the proximal to mid LAD, which was successfully revascularized with 2 drug-eluting stents (Figure 3, Videos 1 and 2). A chronic total occlusion of the mid right coronary artery with

collaterals from the LAD and left circumflex was noted and left un-revascularized (Video 3). An intra-aortic balloon pump was placed, and the patient was transferred to the cardiac intensive care unit.

A transthoracic echocardiogram demonstrated severe left ventricular dysfunction with an ejection fraction estimated at 20%, and akinesis of the mid to distal anterior, apex, inferior, and lateral walls. There was an absence of left ventricular thrombus, hemodynamically significant valvular disease, pericardial effusion, or abnormalities of the myocardium.

MANAGEMENT

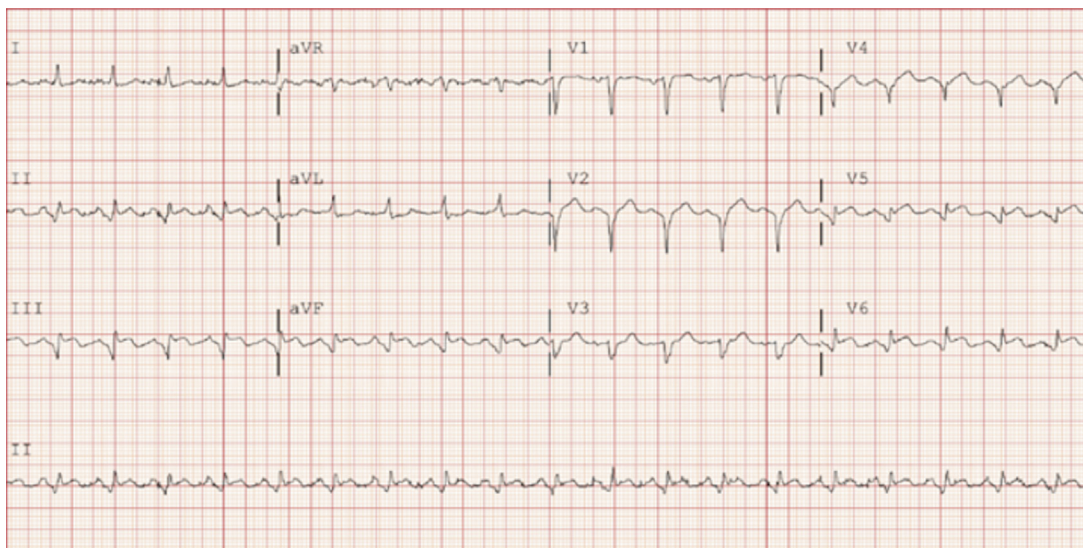
Despite revascularization, the patients shock state persisted with continued requirements of the intra-aortic balloon pump, in addition to multiple vasoactive and inotropic medications (norepinephrine, vasopressin, phenylephrine, and dobutamine). The patients shock state also progressed into oliguric renal failure requiring continuous renal replacement therapy.

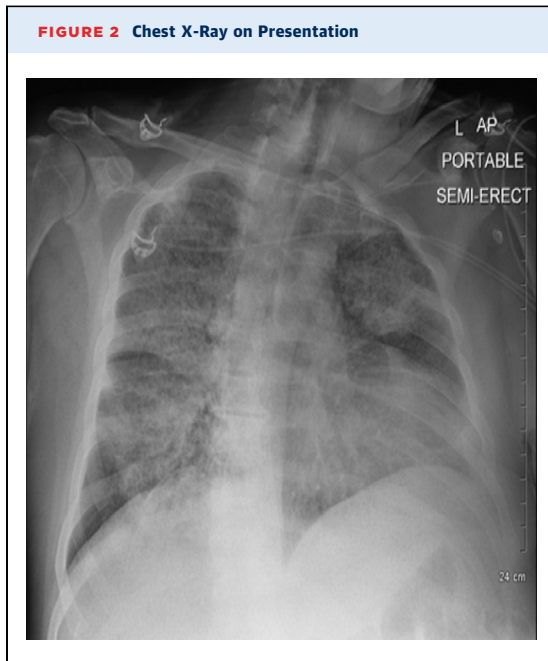
Given the patient's ongoing clinical decline with borderline fevers and thus with a suspected mixed septic and cardiogenic component to the patient's vasoplegia, broad spectrum antimicrobial therapy was initiated with piperacillin/tazobactam and

ABBREVIATIONS AND ACRONYMS

- ACS = acute coronary syndrome
- AMI = acute myocardial infarction
- FWR = free wall rupture
- LAD = left anterior descending artery
- MCS = mechanical circulatory support

FIGURE 1 12-Lead Electrocardiogram on Presentation





vancomycin. Blood cultures returned positive within 12 hours of admission for gram-negative bacilli, ultimately speciating to *Enterobacter cloacae*. Antimicrobial therapy was escalated to meropenem while sensitivities were pending, with the source of bacteremia unknown.

DIFFERENTIAL DIAGNOSIS

The following differential diagnoses for the patients ongoing clinical deterioration and progressive shock state were considered: mixed distributive/septic and cardiogenic shock, recurrent ischemia secondary to stent complication, and an evolving mechanical complication of acute coronary syndrome (ACS) (eg, ventricular free wall rupture [FWR], ventricular septal rupture, papillary muscle rupture, and acute mitral regurgitation).

MANAGEMENT CONTINUED

On the second day of admission, the patient had an abrupt pulseless electrical activity cardiac arrest. Bedside point of care ultrasound demonstrated a large circumferential pericardial effusion. An emergency pericardiocentesis was performed, draining large volumes of arterial blood from the pericardium. The pericardial effusion relentlessly reaccumulated and the patient remained in refractory cardiac tamponade. Mechanical circulatory support (MCS) was considered, however, the patient was deemed not to be a candidate due to uncontrolled septicemia, multiorgan dysfunction, baseline frailty, and, thus, prohibitive surgical risk. Resuscitation efforts were ultimately unsuccessful, with the cause of death suspected to be secondary to left ventricular FWR.

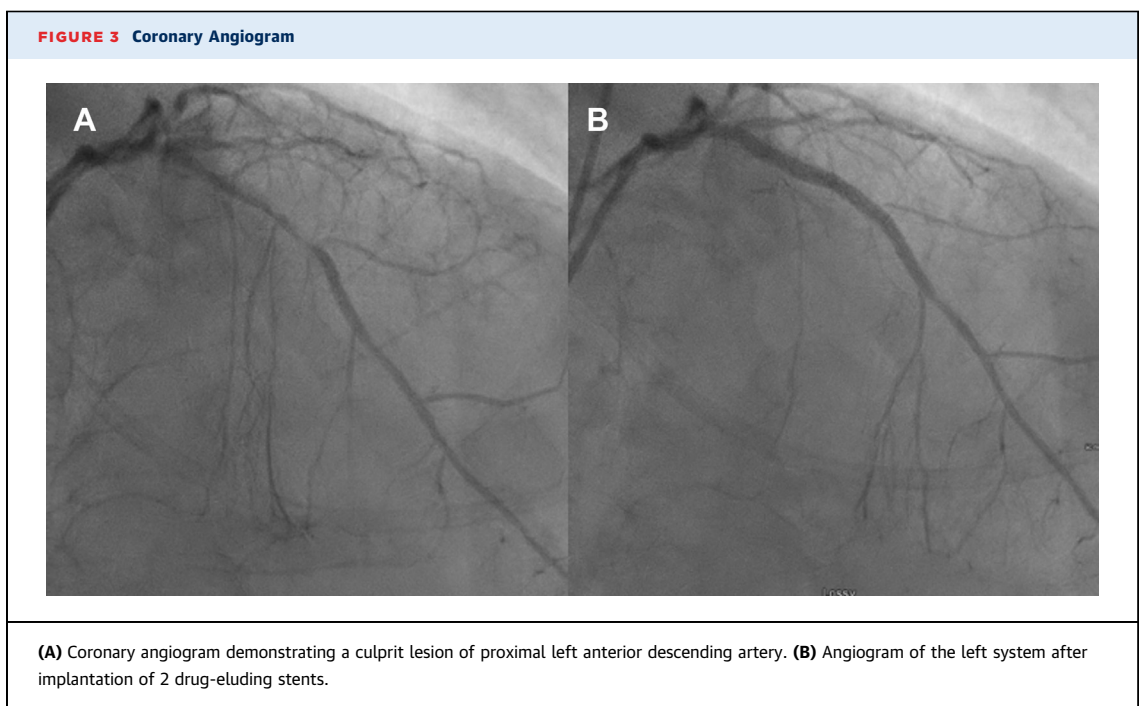
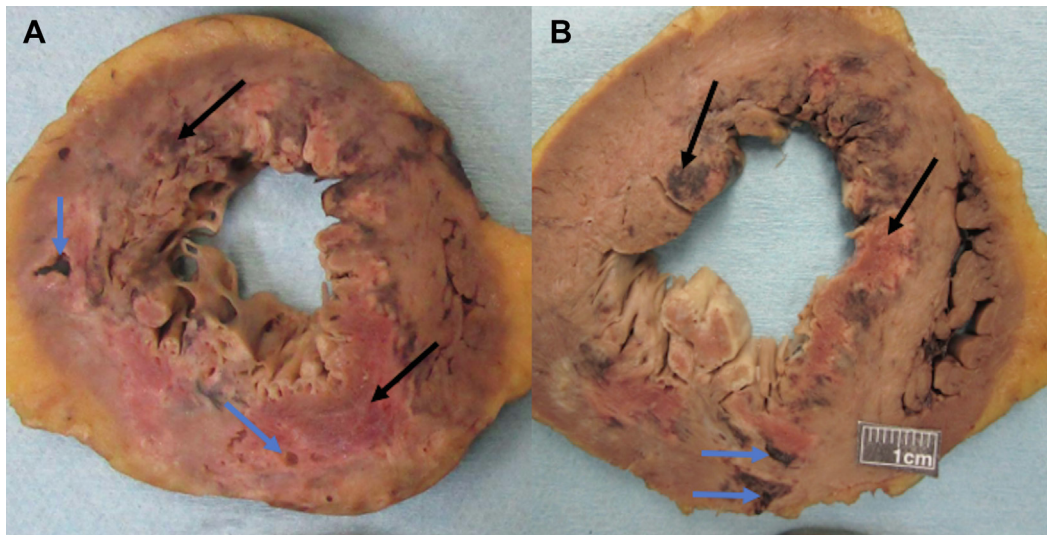


FIGURE 4 Cardiac Autopsy: Gross Pathology



(A) Short-axis cut of heart showing widespread necrosis with discoloration from infarct and abscesses (**black arrows**). Lateral and inferior wall cystic spaces representing abscesses (**blue arrows**). **(B)** Short-axis cut of heart showing discoloration from infarct and abscesses (**black arrows**). The inferior wall has a dark serpiginous area representing the area of ventricular rupture through the necrotic inflamed myocardium (**blue arrows**).

POSTMORTEM EXAMINATION. The autopsy showed large transmural myocardial infarction with extensive necrosis. Extensive multiple myocardial abscesses involving both ventricles with gram-negative rods were noted in the myocardium. There was left ventricular FWR of the mid inferior wall, in an area of extensive necrosis and abscess causing hemopericardium (**Figures 4 and 5**). Severe acute pyelonephritis of the left kidney (source of infection) was also noted.

The final diagnosis was a late presenting Killip IV inferolateral ST-segment elevation myocardial infarction complicated by septic shock with *E cloacae* bacteremia secondary to acute pyelonephritis. The cause of death was left ventricular FWR at the site of a septic myocardial abscess. We believe that the patient's acute myocardial ischemia with resultant inflammation, facilitated bacterial seeding into the myocardium and subsequent abscess formation. Ultimately, the patient's FWR was a synergistic complication of both ischemic myocardial necrosis and superimposed abscess with resultant tissue friability.

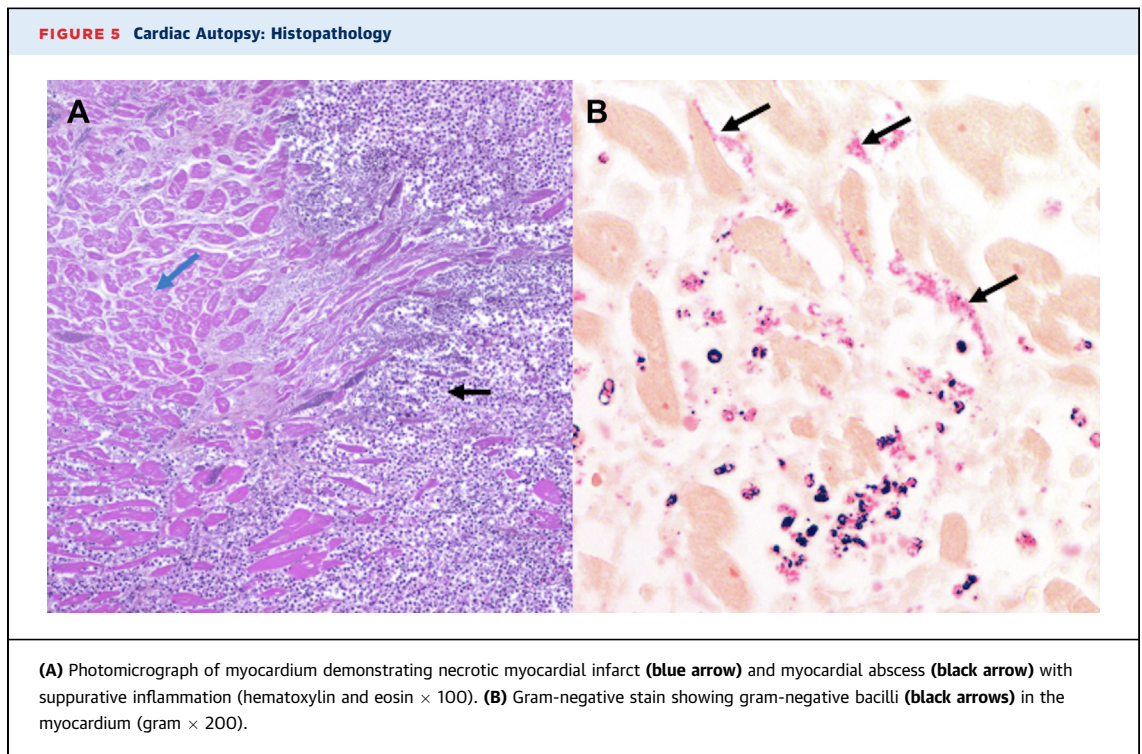
DISCUSSION

Although the incidence of mechanical complications after acute myocardial infarction (AMI) is low, they

remain a well-recognized but catastrophic complication of ACS.¹ Of the mechanical complications, ventricular FWR is the most commonly reported.¹ The classical mechanism of ventricular FWR after AMI is secondary to myocardial necrosis and tissue friability.¹ Uniquely, we report a case of ventricular FWR at the site of a septic myocardial abscess after AMI.

Myocardial abscess formation at the site of ischemic myocardial necrosis, due to metastatic spread into the necrotic tissue, is exceptionally rare.² Since its original description in 1933, there has been a very low frequency of reports within the literature.^{2,3} A retrospective review of 16,000 autopsies over 10 years found the incidence of myocardial abscess post-AMI to be 0.125 per thousand cardiac autopsies.⁴

The most common primary sources of infection for myocardial abscesses in the setting of AMI include respiratory, urinary tract, and skin and soft tissue infections.⁵ It is also not uncommon that a source of infection is never identified.⁶ The most common causative organisms are *Staphylococcus* and *Streptococcal* species, although a variety of other organisms have also been described.^{2,7} Contributing to the rarity of this case is abscess formation by an anaerobe. Although anaerobic myocardial abscesses are exceedingly rare, it is hypothesized these organisms are supported by the relatively hypoxic postinfarction environment.⁵



The identification of myocardial abscesses post-AMI is challenging and rarely diagnosed antemortem.^{5–8} They are characteristically silent and often obscured by the superimposed septic process and sequelae of the presenting ACS.⁸ Useful features to raise clinical suspicion include bacteremia, persistent fever despite appropriate antimicrobials, and progressive hemodynamic deterioration.⁸ To facilitate early diagnosis suggested strategies within the literature include having a high index of suspicion in all patients with bacteremia after ACS and the focused application of echocardiography.⁵ Relevant echocardiographic features associated with myocardial abscesses include wall thickening, honeycomb appearance to the myocardium, and fistulous tracts that may drain into the pericardium.⁵ We were unable to identify any abnormalities of the myocardium in our patient after a focused review of his transthoracic echocardiogram guided by the gross pathology. This further highlights how challenging this clinical entity can be to diagnose antemortem. It also exemplifies how detection of this particular pathology on echocardiography is limited by the spatial resolution of the imaging modality, thus suggesting a role for advanced cardiac imaging in these circumstances.

The clinical course of a myocardial abscess after AMI is variable; however, it is typically fatal with death often occurring within days to weeks.⁹

Ventricular rupture secondary to a myocardial abscess is a catastrophic complication; we were only able to find a single case report of a patient who survived a contained rupture of an abscess after AMI.⁵

Early identification of myocardial abscesses and FWR is essential for a chance of survival. Although there is sparse literature specifically on the management of FWR secondary to myocardial abscesses, the immediate placement on extracorporeal membrane oxygenation at the time of circulatory collapse as a bridge to immediate surgical repair has been suggested for ischemic FWR.¹ However, this is typically confounded by the patient's candidacy for MCS and the absence of a prohibitive surgical risk.¹ Furthermore, effective extracorporeal membrane oxygenation is often limited by poor venous return due to cardiac tamponade.¹ Despite the use of MCS and modern surgical techniques ventricular FWR after AMI is frequently fatal with a reported mortality rate of $>50\%$.¹

CONCLUSIONS

Our case highlights an exceptionally rare yet catastrophic complication of AMI, ventricular FWR at the site of septic myocardial abscess. Despite its rarity, awareness of this potential complication in a septic patient after ACS is essential to prompt diagnosis and

thus early intervention in hopes to circumvent an otherwise inevitably fatal outcome.

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KEY WORDS acute myocardial infarction, mechanical complications, myocardial rupture, myocardial abscess

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