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IMAGING

CLINICAL CASE

A Common Diagnostic Dilemma

Inflammation or Infarction

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ABSTRACT

Early infarct-associated pericarditis is a rare entity, given the availability of early coronary angiography and intervention. Although the electrocardiogram and the surface echocardiogram are initial studies, definitive imaging with cardiac magnetic resonance is recommended. We present a case of early infarct-associated pericarditis in the setting of a late-presenting silent right coronary artery myocardial infarction. (JACC Case Rep. 2025;30:102722) © 2025 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 60-year-old man presented to the emergency department for evaluation of chest pain and hypoxia noted on a home pulse oximeter. He reported having soreness across his chest 2 days earlier that worsened with deep breaths and lying flat. He noted that the pain improved when he leaned or sat forward and denied any exertional chest pain or radiation down his left arm or to the jaw. He did have a notable sick contact with his wife, who had recently been febrile with a cough. His initial vital signs on presentation were notable only for a pulse of 50 beats/min, and he was otherwise stable. His physical examination was unremarkable, with normal heart sounds and no rubs, murmurs, gallops, friction rub, or knock noted.

LEARNING OBJECTIVES

- To be able to use multimodal imaging for differentiation among causes of acute myopericarditis.
- To recognize early infarct-associated pericarditis and its complications.
- To understand that heart block in the setting of pericarditis raises the question of infarction.

PAST MEDICAL HISTORY

He had a past medical history of hypertension and poorly controlled type 2 diabetes mellitus.

DIFFERENTIAL DIAGNOSIS

Pericarditis, myocarditis, myopericarditis, viral pneumonia, pulmonary embolism or acute coronary syndrome were considered.

INVESTIGATIONS

Initial laboratory test results revealed leukocytosis of 17.5 K/μL and were otherwise unremarkable. The metabolic panel was notable for a potassium level of 5.2 mmol/L, a blood urea nitrogen value of 57 mg/dL, and a creatinine level of of 3.75 mg/dL (with a baseline of 0.8). He had elevated aspartate aminotransferase and alanine transaminase levels of 400 U/L and 500 U/L, respectively, with a normal total bilirubin

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ABBREVIATIONS AND ACRONYMS

AV = atrioventricular

CMR = cardiac magnetic resonance

ECG = electrocardiogram

LGE = late gadolinium enhancement

LV = left ventricular

MI = myocardial infarction

STEMI = ST-segment elevation myocardial infarction

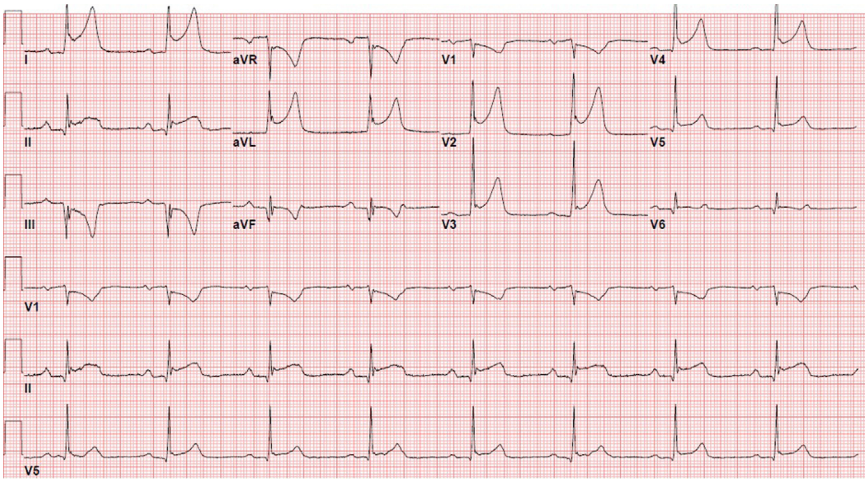
TTE = transthoracic echocardiogram

value. He had an initial high-sensitivity troponin T (fifth-generation cardiac troponin assay) level of 38.7K ng/L, which on repeat 4 hours later was 36.2K ng/L. He also had elevated inflammatory markers, with an erythrocyte sedimentation rate of 102 mm/h and a C-reactive protein level of 29.5 mg/dL. The initial electrocardiogram (ECG) (**Figure 1**), revealed a first-degree atrioventricular (AV) block with sinus bradycardia of 50 beats/min, and diffuse ST-segment elevations, with ST-segment depressions in leads III, aVF, and aVR. A transthoracic echocardiogram (TTE) (Video 1) demonstrated an LV ejection fraction of 55% to 60%, normal LV systolic and diastolic function, and abnormal wall motion in the basal inferolateral and inferior segments.

MANAGEMENT

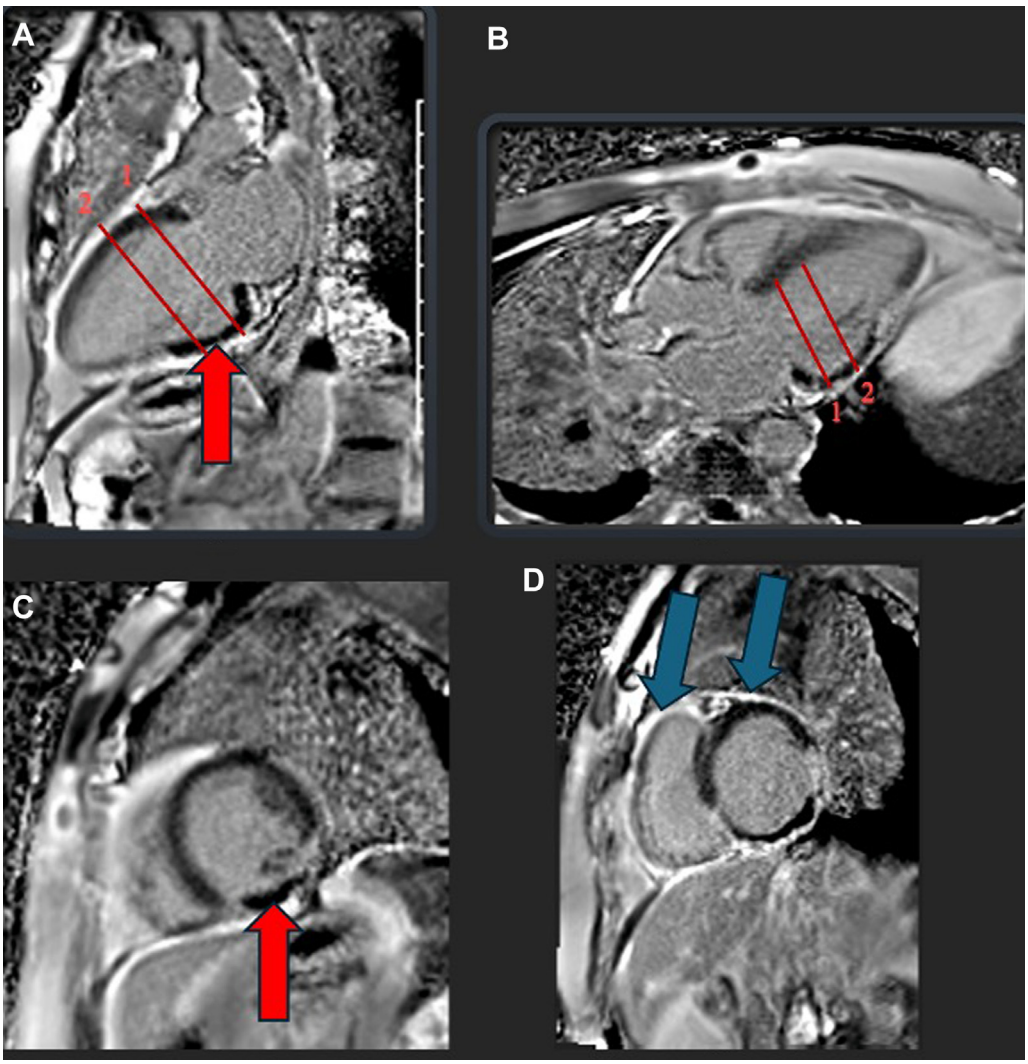
Given the initial diagnostic work-up, pleuritic chest pain and sick contacts, the working diagnosis at the time was myopericarditis. Because of the patient's stage 3acute kidney injury and risk for dialysis, cardiac catheterization was deferred on admission. Notably, his troponin levels peaked on admission at 38.7 K ng/L, and his aspartate aminotransferase and alanine transaminase levels slowly decreased to normal over the following days. His hospital course was first complicated by transient complete heart block, followed by 2:1 heart block on the morning of hospital day 2. Electrophysiology was subsequently consulted and planned for transvenous pacing if the patient maintained high-degree AV block. This pacing fortunately was not required, and the condition later improved to 3:1 conduction with Mobitz 1 block. His hospital course was later complicated by worsening respiratory status requiring bilevel positive airway pressure, in addition to development of bilateral pleural effusions and lower lobe infiltrates. Broad-spectrum antibiotics were initiated at that time. His hospital course was additionally complicated by worsening renal failure. By hospital day 4, he did require a 1-time hemodialysis session, although his renal function and hypoxic respiratory failure continued to improve thereafter with furosemide doses. He ultimately underwent cardiac magnetic resonance (CMR) on hospital day 8 (**Figures 2A to 2D, 3, and 4**, Videos 2 and 3), which demonstrated mildly reduced left ventricular (LV) systolic function with an LV ejection fraction of 40%, a dilated left ventricle, and inferior wall akinesis. In addition, subendocardial late gadolinium enhancement (LGE) involving the inferior wall with a central area of low signal intensity with a surrounding thin line of hyperenhancement, acute edema of the inferior wall, and LGE of the pericardium consistent with acute pericarditis were noted. The imaging was consistent with myocardial infarction (MI) and microvascular obstruction involving the right coronary artery territory with resultant early infarct-associated pericarditis. This finding correlated with

FIGURE 1 Admission Electrocardiogram



The tracing shows first-degree atrioventricular block and diffuse ST-segment elevations with reciprocal changes in leads III and aVF along with Q waves.

FIGURE 2 Cardiac Magnetic Resonance Images



(A) T1-weighted delayed enhancement long-axis 2-chamber cardiac magnetic resonance images with phase sensitive inversion recovery sequences showing microvascular obstruction (red arrow) involving the inferior wall in the right coronary artery RCA territory. The red lines 1 and 2 correlate with the plane for the images in Figures 2D and 2C, respectively. (B)T1-weighted delayed enhancement long-axis cardiac magnetic resonance (phase sensitive inversion recovery sequences) showing pericardial late gadolinium enhancement indicating acute inflammation from pericarditis. The red lines 1 and 2 correlate with the plane for the images in Figures 2D and 2C, respectively, (C) T1-weighted delayed enhancement short-axis cardiac magnetic resonance image with phase sensitive inversion recovery showing microvascular obstruction (red arrow) in the basal inferior wall (dark central area of low signal intensity) surrounded by a thin layer subendocardial late gadolinium enhancement. The image plane correlates with line 2 in Figures 2A and 2B. (D) T1-weighted delayed enhancement short-axis cardiac magnetic resonance images with phase sensitive inversion recovery showing pericardial late gadolinium enhancement (blue arrows) indicating active pericardial inflammation. The image plane correlates with line 1 in Figures 2A and 2B.

his ECGs, which showed an evolution of Q waves inferiorly throughout his stay. He was started on metoprolol tartrate, losartan, furosemide (Lasix), colchicine, apixaban (Eliquis), low-dose aspirin, and atorvastatin and was discharged home, with close follow-up planned.

DISCUSSION

Pericarditis associated with MI is further subclassified into early infarct-associated or late post-MI pericarditis (Dressler syndrome).[1,2] Early infarct-associated typically occurs within 5 days post-MI and is rarely

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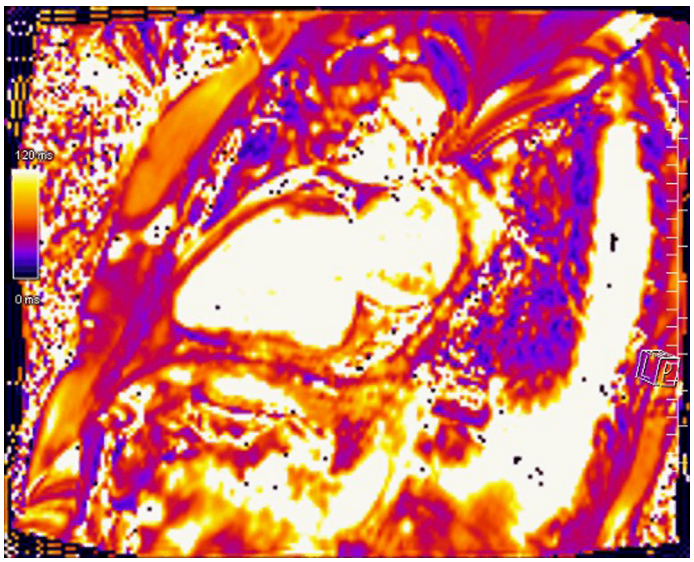
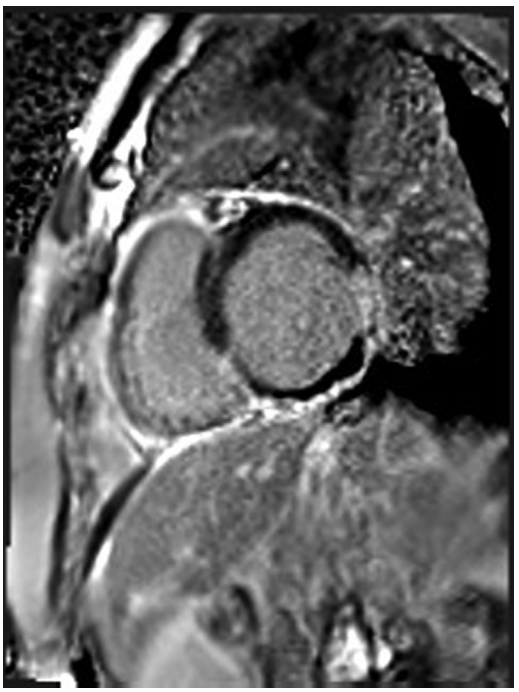
FIGURE 3 Long-Axis 2-Chamber T2 MyoMap (Siemens Healthineers)



The free-breathing with motion correction image shows prolonged T2 in the basal inferior wall consistent with edema (black arrow).

FIGURE 4 Short-Axis T1-Weighted Delayed Enhancement Cardiac Magnetic Resonance Images With an Inversion Recovery Sequence



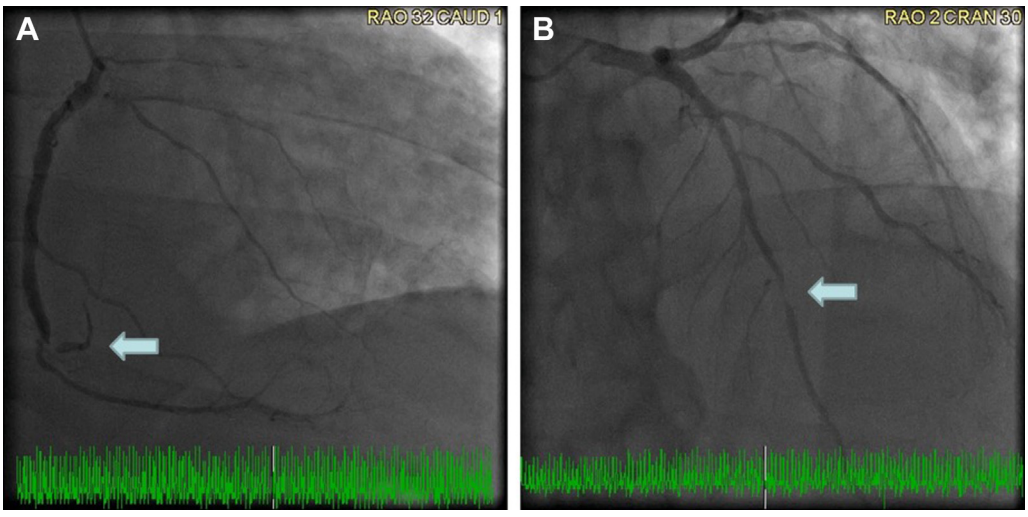
The images show pericardial late gadolinium enhancement of the inferior wall and redemonstrating signs of microvascular obstruction of the right coronary artery.

seen as a result of early reperfusion, with a reported incidence of <5%.[1,2] In the acute phase however, differentiating among infarct-associated pericarditis, myopericarditis, and late-presenting ST-segment elevation myocardial infarction (STEMI) can be difficult, as seen with our patient. Delineating these entities relies predominantly on ECG and TTE because inflammatory markers may be elevated but nonspecific, and cardiac biomarkers are significantly elevated in both entities.[1,2] Compared with STEMI, ST-segment elevations are not widespread and lack the PR-segment depressions seen in pericarditis. Notably, although the first stage of pericarditis and STEMI both can have ST-segment elevations on a ECG, pericarditis rarely has ST-segment elevations in lead V_6 or PR-segment elevation in lead aVR.[2]

TTE can demonstrate wall motion abnormalities in MI that are typically not seen in pericarditis or myopericarditis. However, TTE is limited in that it does not allow for detailed visualization of the pericardium.[1] CMR, however, provides anatomic information on the pericardium and cardiac hemodynamics[1,3] and as such can best demonstate findings highly specific to the diagnosis in question. Findings specific to pericarditis include pericardial thickening on the T1-weighted black-blood sequence, LGE suggesting active inflammation, and pericardial edema on T2-weighted images.[3] Our patient's CMR results demonstrated subendocardial LGE involving the inferior wall, as well as acute edema of the inferior wall and LGE of the pericardium consistent with acute pericarditis. Thus, the findings suggested an inferior wall MI complicated by early infarct-associated pericarditis.

Our patient's initial TTE demonstrated inferior and inferolateral wall motion abnormalities that did suggest ischemic changes. Although the European Society of Cardiology guidelines recommend consideration for left-sided heart catheterization in the acute phase to rule out acute coronary syndrome, the evidence is low grade (Grade C), and the need or timing for revascularization has variable study results. Given our patient's acute kidney injury on admission and the need for hemodialysis during hospitalization, heart catheterization was deferred. The Occluded Artery Trial in 2011 noted that for STEMI manifesting at 3 to 28 days, there was no difference in outcomes of death, reinfarction, or NYHA

FIGURE 5 Fluoroscopy Showing Coronary Artery Occlusion and Stenosis



(A) Right anterior oblique fluoroscopy illustrating a chronic total occlusion of the distal right coronary artery. (B) Anteroposterior cranial fluoroscopy illustrating a 95% stenotic lesion in the mid-left anterior descending coronary artery. CAUD = caudal; RAO = right anterior oblique.

functional class IV heart failure for patients treated with revascularization or percutaneous coronary intervention vs medical therapy for most patients.[4] More recently, in 2021, Bouisset et al[5] studied 12- to 48-hour postpresentation percutaneous coronary intervention and showed lower 30-day mortality and reinfarction. If these findings had manifested earlier in the disease course, our patient likely would have not developed infarct-associated pericarditis, but there are no data to suggest that revascularization at the time of our patient's presentation would have changed this outcome.

FOLLOW-UP

The patient tolerated medical therapy well after discharge. However he had 2 episodes of exertional chest pain in the months after his hospitalization. He underwent left-sided heart catheterization in the outpatient setting 2 months after his original hospitalization (**Figures 5A and 5B**). Findings demonstrated a chronic total occlusion to the mid-distal right coronary artery, with right-to-right collateral vessels, as well as moderate (60%) disease of the mid-left anterior descending coronary artery and distal D2, a severe (95%) stenotic lesion of the distal left anterior descending coronary artery for which a 2.75 mm × 28 mm Xience drug-eluting stent (Abbott) was placed for revascularization. It is suspected that the infarction was complete by the time of the coronary angiogram 2 months later, hence the total occlusion noted on the angiogram.

CONCLUSIONS

Early infarct-associated pericarditis is a rare entity, given the current prevalence of early revascularization therapy. Our patient presented with a late inferior MI, a complication infrequently encountered. The utility of CMR in pericarditis, when differentiating pericarditis from myocarditis, myopericarditis, and infarct-associated pericarditis, is high because it delineates pericardium anatomy and can assist in determining the cause of pericarditis and guide treatment.

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KEY WORDS **CMR, infarct-associated pericarditis**

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