Concurrent subacute myocardial infarction and bilateral pneumonia presenting as acute heart failure: a case report

Mario Špoljarić[1]*,
Katica Cvitkušić Lukenda[1],
Kristina Vorkapić[1],
Marko Galić[1],
Matea Kirchbauer Špoljarić[1],
Dijana Bešić[2],
Anđela Jurišić[2]

[1]General Hospital "Dr Josip Benčević", Slavonski Brod, Croatia
[2]University Hospital Dubrava, Zagreb, Croatia

KEYWORDS: acute heart failure, bilateral pneumonia, myocardial infarction.
CITATION: Cardiol Croat. 2026;21(1-2):48-9. | https://doi.org/10.15836/ccar2026.48
***ADDRESS FOR CORRESPONDENCE:** Mario Špoljarić, Opća bolnica "Dr. Josip Benčević", Andrije Štampara 42, HR-35000 Slavonski Brod, Croatia. / Phone: +385-99-1949-302 / E-mail: mariospoljaric@hotmail.com
ORCID: Mario Špoljarić, https://orcid.org/0000-0001-5770-3012 • Katica Cvitkušić Lukenda, https://orcid.org/0000-0001-6188-0708 Kristina Vorkapić, https://orcid.org/0009-0002-8636-7331 • Marko Galić, https://orcid.org/0009-0003-0437-6750 Matea Kirchbauer Špoljarić, https://orcid.org/0009-0000-9709-6413 • Dijana Bešić, https://orcid.org/0000-0001-9701-0253 Anđela Jurišić, https://orcid.org/0000-0001-8316-4294

Introduction: Systemic infections, particularly acute respiratory infections, are recognized triggers of acute coronary syndromes (ACS). They may precipitate plaque destabilization through systemic inflammation, endothelial dysfunction, and platelet activation, thereby lowering the threshold for plaque rupture or erosion.[1,2]

Case report: 59-year-old woman with no prior comorbidities was admitted to the Coronary Care Unit with acute heart failure following five days of persistent chest pain. During the same period, she experienced sustained fever accompanied by chills, rigors, and a dry cough. On admission, the patient was dyspneic and orthopneic and reported mild pressure in the left hemithorax. Laboratory tests showed elevated high-sensitivity troponin I (3546 ng/L), N-terminal pro-B-type natriuretic peptide (6225 pg/mL), and inflammatory markers (C-reactive protein 125 mg/L, leukocytes 11×10^9/L). Serial measurements demonstrated a gradual decline in hsTnI levels. Electrocardiography revealed ST-segment elevation in leads V1–V4 with Q-wave formation, while echocardiography confirmed akinesia of the apical, interventricular septal, and anterior walls, reduced left ventricular ejection fraction, and an apical thrombus (**Figure 1**). Given the concurrent fever and chest pain, acute myocarditis was initially included in the differential diagnosis. Chest X-ray showed extensive bilateral pneumonia, confirmed by MSCT pulmonary angiography (**Figure 2**). Coronary angiography performed the following day revealed occlusion of the ostial left anterior descending artery with heterocollateralization from other epicardial vessels, consistent with a subacute anterior ST-elevation myocardial infarction (**Figure 3**).

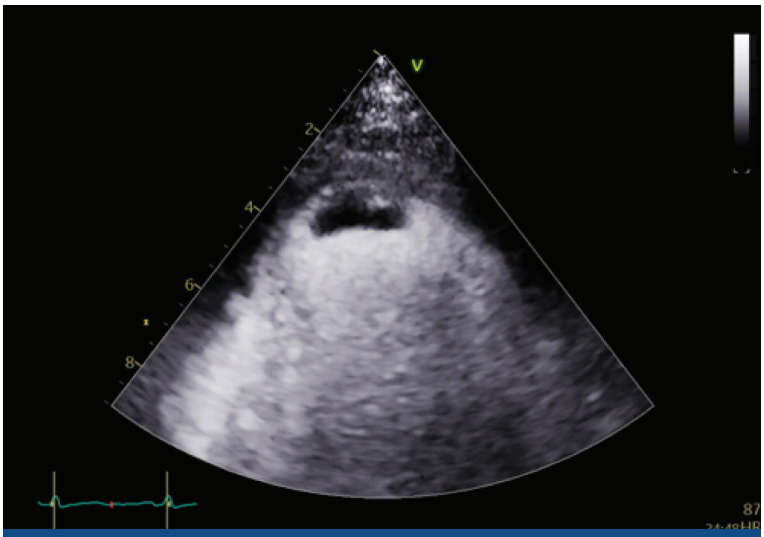

FIGURE 1. Contrast-enhanced echocardiogram demonstrates a left ventricular apical thrombus.

RECEIVED:
October 19, 2025
ACCEPTED:
November 14, 2025

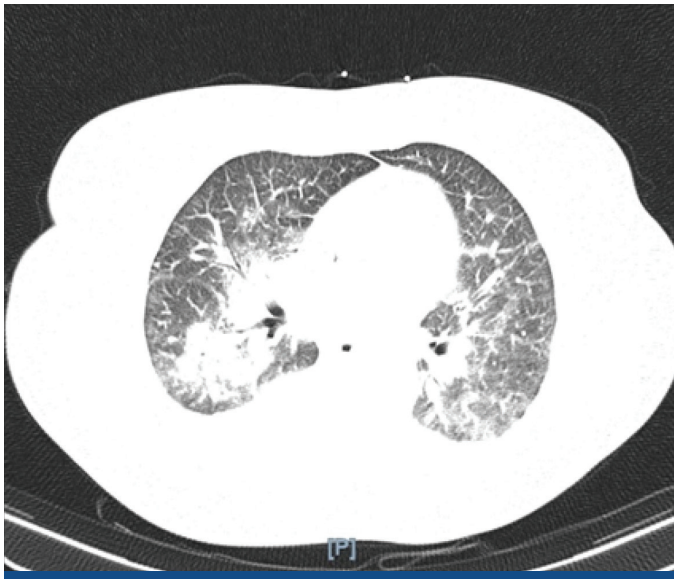

FIGURE 2. The scan shows bilateral, predominantly peripheral ground-glass opacities with patchy consolidation, consistent with pneumonia.

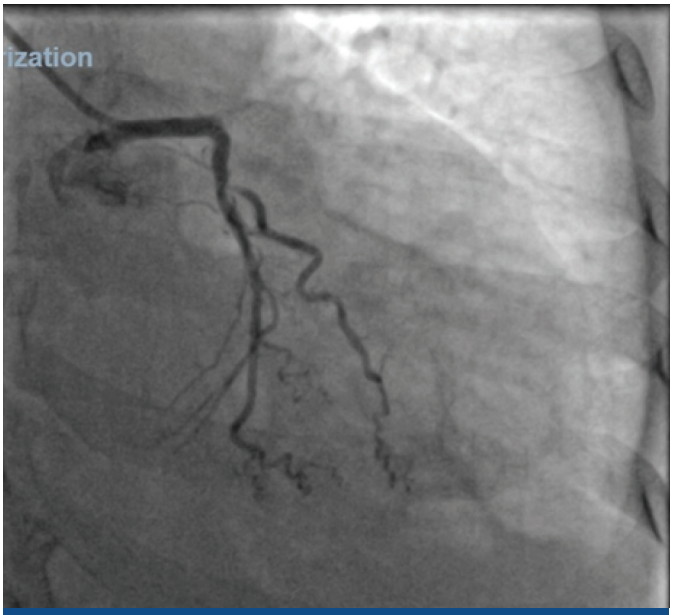

FIGURE 3. Ostial left anterior descending artery (LAD) occlusion with collateral flow from the circumflex artery.

The patient received broad-spectrum intravenous antibiotics and optimal pharmacological therapy for heart failure. Gradual cardiac recompensation was observed, with resolution of chest pain and a marked decline in inflammatory markers. She was discharged on the seventh day of hospitalization on optimized pharmacotherapy for coronary artery disease and heart failure, including anticoagulation with warfarin. Myocardial viability assessment is planned three months post-discharge to guide further interventional management.

Conclusion: This case highlights how acute infection and systemic inflammation can mask or mimic acute coronary syndromes, complicating the diagnosis and delaying definitive treatment. Early coronary evaluation should be considered in febrile patients presenting with chest pain and elevated cardiac biomarkers to ensure timely management and improve clinical outcomes.

LITERATURE

1. Kwong JC, Schwartz KL, Campitelli MA, Chung H, Crowcroft NS, Karnauchow T, et al. Acute Myocardial Infarction after Laboratory-Confirmed Influenza Infection. N Engl J Med. 2018 Jan 25;378(4):345-353. https://doi.org/10.1056/NEJMoa1702090
2. Corrales-Medina VF, Madjid M, Musher DM. Role of acute infection in triggering acute coronary syndromes. Lancet Infect Dis. 2010 Feb;10(2):83-92. https://doi.org/10.1016/S1473-3099(09)70331-7