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CASE REPORT
Coronary intervention

A rare case report of fatal acute myocardial infarction as a complication of myocardial abscess

Lina Lutsenko [1]*, Maksym Sokolov [1], and Viktoriya Krychkevych [2]

[1]Department of interventional cardiology, State Institution National Scientific Center The M.D. Strazhesko Institute of Cardiology, clinical and regenerative medicine of the National academy of medical sciences of Ukraine, Svyatoslava Horobrogo 5, 03151 Kyiv, Ukraine; and [2]Department of pathology, State Institution National Scientific Center The M.D. Strazhesko Institute of Cardiology, clinical and regenerative medicine of the National academy of medical sciences of Ukraine, Svyatoslava Horobrogo 5, 03151 Kyiv, Ukraine

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Background Myocardial abscess is a very rare life-threatening suppurative infection of the heart. Usually, myocardial abscess is a complication of infective endocarditis, and it is rarely associated with isolated myocardial infection. We present a case of an isolated myocardial abscess presenting with acute myocardial infarction.

Case summary A 61-year-old man with a history of diabetes mellitus and coronary artery disease presented with a 3-h history of chest pain and inferior ST elevation. He had been treated for right-sided pneumonia 1.5 months prior to admission. Coronary angiography revealed acute occlusion of the posterolateral ventricular artery, and he underwent balloon angioplasty, which successfully restored TIMI-3 blood flow. Unfortunately, the patient went into cardiac arrest several hours later from which he could not be resuscitated. A post-mortem revealed a myocardial abscess in the inferior wall of the left ventricle.

Discussion Myocardial abscess is a challenging diagnosis due to the speed of clinical deterioration and rarity. High clinical suspicion and urgent multimodality imaging may aid in the diagnosis.

Keywords Case report • Myocardial abscess • PCI • Myocardial infarction • ST-segment elevation myocardial infarction

ESC Curriculum 3.1 Coronary artery disease • 3.2 Acute coronary syndrome • 3.4 Coronary angiography

Learning points

- Myocardial abscess is very rare and a life-threatening illness.
- Typically, myocardial abscess is a complication of infective endocarditis of native or prosthetic valves.
- Isolated myocardial infection is an extremely rare cause of septic embolism of coronary arteries like in our case.
- A high level of clinical suspicion and multimodality imaging are cornerstones of the correct diagnosis.

Introduction

Myocardial abscess (MA) is a suppurative infection of the myocardium, endocardium, valves, perivalvular structures, or the cardiac conduction system. In most cases, MA is a complication of infective endocarditis of native or prosthetic valves.[1,2] In our case, we admitted a patient with an ST-elevation myocardial infarction (STEMI), which was a complication of the myocardial abscess.

* Corresponding author. Tel: +380666669278, Fax: +380442756622, Email: linalutsenko@gmail.com
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Timeline

4 years before admission	Non-ST-elevation myocardial infarction in 2017, stented 1 drug eluting stent (DES)—circumflex (culprit lesion), 1-DES—left anterior descending
1.5 months before admission	A right-sided pneumonia with negative PCR COVID-19 test
2 weeks before admission	Low-grade fever continued and patient noticed reversion of angina
Day 1–5 p.m.	Hospitalized with an inferior ST-elevation myocardial infarction and primary percutaneous coronary intervention was done
Day 1–8 p.m.	Temporary transvenous pacemaker was inserted due to third-degree atrioventricular block
Day 2–3 a.m.	Cardiac arrest occurred and patient has died.
Day 2	Post-mortem examination showed myocardial abscesses in the inferior wall of the left ventricle.

Case presentation

A 61-year-old man with history of diabetes mellitus and coronary artery disease (CAD) presented to our hospital with a 3-h left-sided chest pain and dyspnea. He had lateral non-STEMI in 2017, stented 1 drug eluting stent (DES) circumflex (Cx) (culprit lesion), 1-DES—left anterior descending (LAD). Also, he had persistent atrial fibrillation, ischaemic stroke in 2010 and gout. He had right-sided pneumonia with a negative PCR COVID-19 test 1.5 month before admission. He was hospitalized with pneumonia in the internal medicine department. Nevertheless, he was discharged to his primary care physician the next day due to the pandemic and all hospitals were full of COVID-19 patients. He received amoxicillin/clavulanate 875/125 mg twice daily for 7 days. Symptoms of pneumonia were reduced on the third to fourth day of the disease. After discontinuation of antibiotics, low-grade fever continued, and patient noticed reversion of angina. But he did not seek medical attention until he felt continuous left-sided chest pain.

Medications at the time of admission are acetylsalicylic acid 100 mg once daily, valsartan 160 mg twice daily, amlodipine 10 mg once daily, and atorvastatin 60 mg once daily. At initial physical examination, blood pressure was 110/70 mmHg, bradycardia (48 per minute), SpO2 = 91%, respiratory rate = 21 per minute, temperature 37.3°C.

Electrocardiogram on presentation revealed third-degree atrioventricular (AV) block with 42 rate, complete left bundle branch block, 2-mm ST segment elevation in II, III, and aVF leads (*Figure 1*).

Transthoracic echocardiogram revealed hypokinesis of left ventricle inferior wall with ejection fraction of 58%, no evidence of vegetation and valve dysfunction were found.

Blood tests were unremarkable except leucocytosis—16.52 × 10^9/L (normal range 4.5–10 × 10^9/L), but we thought it was due to myocardial infarction. High sensitive troponin test was positive—527 ng/L (normal range <20 ng/L). C-reactive protein was 120 mg/L (normal range < 5 mg/L).

Considering the fact that patient had chest pain, ST elevation in related leads, high troponin level, previous CAD background, and complete heart block (occlusion of the right coronary artery (RCA) is common cause), coronary angiography was done. Left main, LAD, and Cx were without significant stenosis (*Figure 2*). *Figure 3* shows the second diagonal branch with 60% stenosis, first obtuse marginal with 90% stenosis, diffuse disease of the RCA with acute occlusion of the posterolateral ventricular artery (PLA). Balloon angioplasty of PLA was done, and we received TIMI-3 bloodflow. No-stent strategy was chosen because diameter of PLA was about 2 mm.

Our team was surprised that culprit lesion had so small diameter. However, we thought that in this case, the main reason of third-degree AV block was occlusion of AV node branch, which supplies the AV septal area and the AV node.

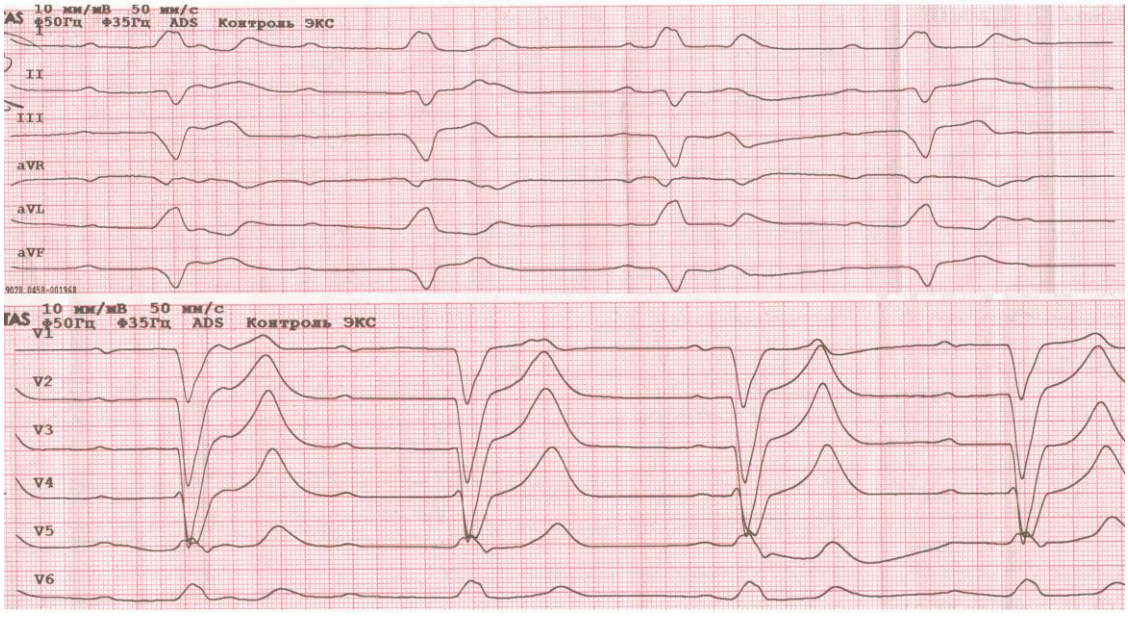

Figure 1 Electrocardiogram on presentation with complete heart block, 2-mm ST segment elevation in II, III, and aVF leads.

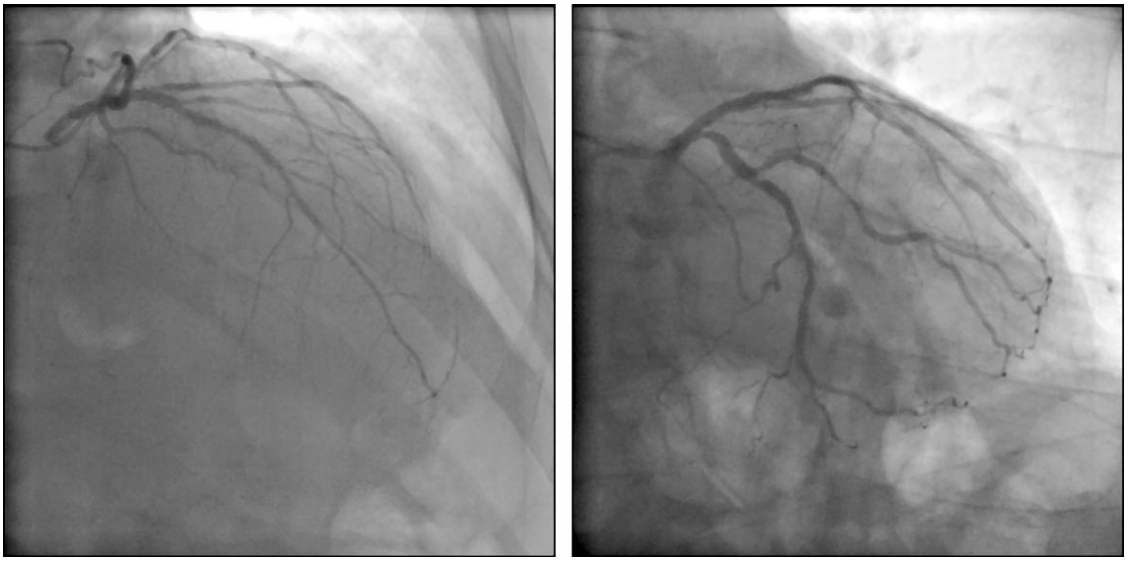

Figure 2 Coronary angiography of left coronary artery with 60% stenosis of first diagonal branch, diffuse disease of circumflex artery, and 90% stenosis of the first obtuse marginal.

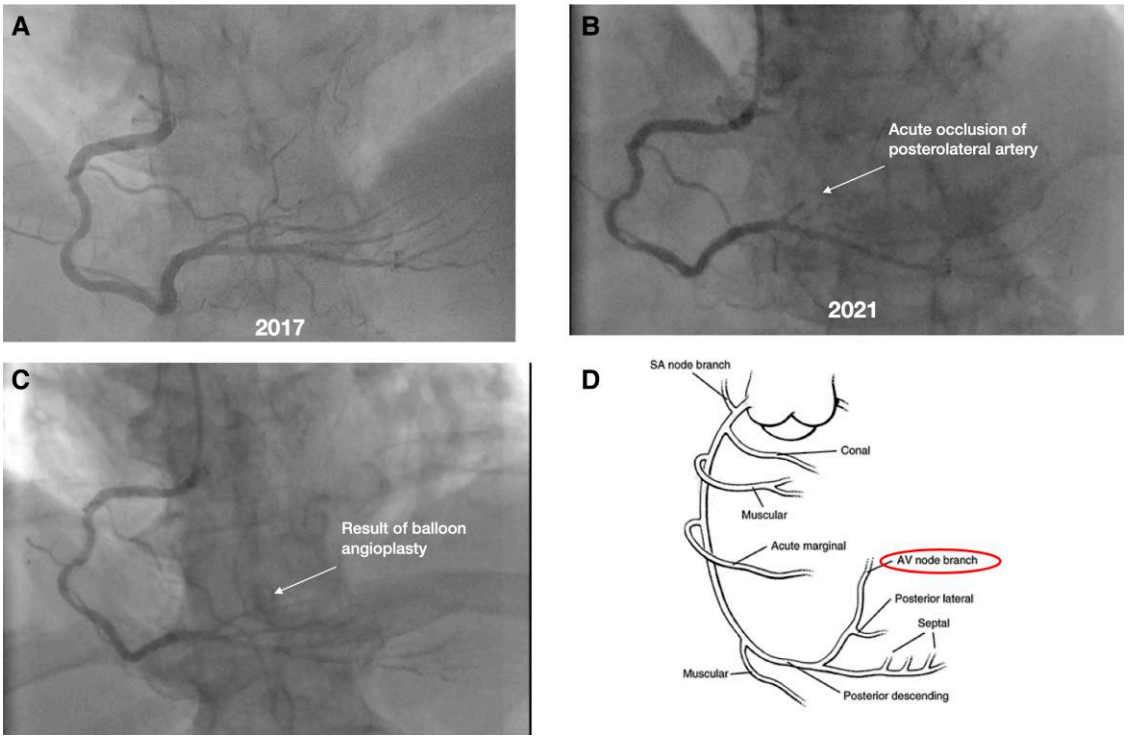

Figure 3 *A*—normal right coronary artery in 2017; *B*—diffuse disease of right coronary artery with acute occlusion of posterolateral artery; *C*—result of balloon angioplasty of posterolateral artery; *D*—anatomy of the right coronary artery.

Nevertheless, third-degree AV block was persisted, and heart rate was 36 per minute. It was decided to insert temporary transvenous pacemaker. In a few hours, monomorphic ventricular tachycardia was started, which then degenerates to ventricular fibrillation. Unfortunately, it was refractory to defibrillate and CPR and patient has died.

Post-mortem examination showed large myocardial abscesses in the inferior wall of the left ventricle (*Figures 4* and *5*).

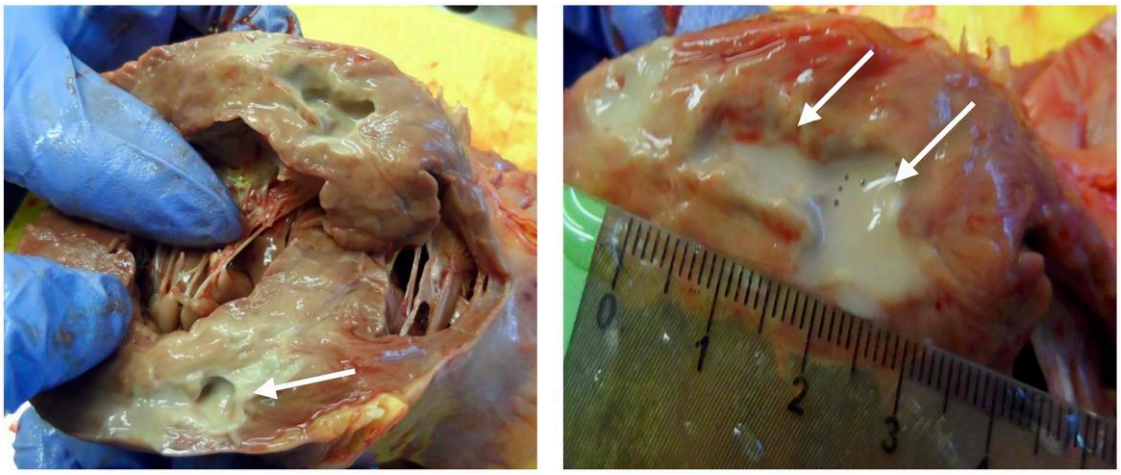

Figure 4 Macroscopic view of the myocardial abscess. Diffuse myocardial abscess is localized near right coronary artery in the basal inferior segment of the left ventricle. Myocardial abscess was represented by a cavity that was 30 × 15 × 20 mm and filled with purulent masses of grey and yellow color, which have viscous consistency and volume of 4 mL.

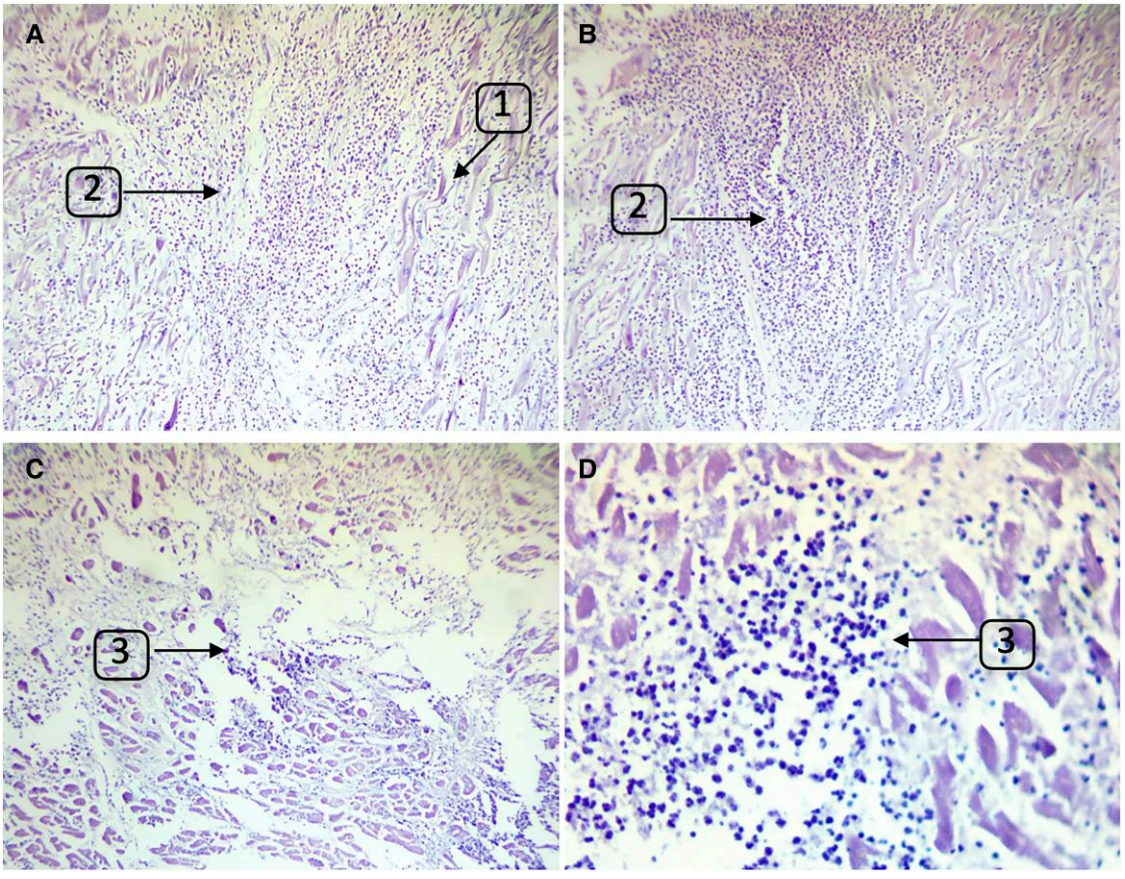

Figure 5 Microscopic view. Haematoxylin-eosin (*A*, *B*—10×, *C*—20×, *D*—40×) stained microscopic images showing disorganization of cardiomyocytes (arrow-1), their destruction of various degrees of severity with their necrosis and the formation of a cavity filled with leukocytes (arrow-2). Interstitial edema, diffuse fibrotic changes, and lympho-leukocyte infiltration (arrow-3) were observed in undamaged areas. It is also important to note the hyperaemia of the microvasculatory, erythrocyte stasis, and edema of the stroma.

Discussion

Myocardial abscess is a suppurative infection of the myocardium, endocardium, valves, perivalvular structures, or the cardiac conduction system. In most cases, MA is a complication of infective endocarditis of native or prosthetic valves. Even more rarely, MA originates at the site of a myocardial infarction due to bacteremia without a known cardiac source.[3,4]

The most common pathogen is Staphylococcus aureus, but other Gram-negative infections can also be present. Kim T.Y. *et al.*[5] in their pictorial review have reported about cardiac complications in patients with hematologic diseases such as thrombotic complications, granulocytic sarcoma, hemochromatosis, amyloidosis, and MA. Immunocompromised status is considered as the main factor of MA. Uncontrolled diabetes is also a known risk factor of immunosuppression like in our case.[6] Fungi, particularly Candida and Aspergillus, are commonly found in immunocompromised patients either.

In our case, patient had a bacteremia due to bad response to antibiotics regarding pneumonia. Bacteremia had led to septic embolism and as a complication—myocardial abscess. After all, we admitted patient when he had another septic embolism, which caused occlusion of the PLA and acute coronary syndrome. According to the literature, most cases of septic coronary embolism are associated with infective endocarditis.[7–11] Unfortunately, in our case, prior source of the septic embolism remained unclear.

Myocardial abscess is a life-threatening illness. It is quite challenging to make a life-time diagnosis of myocardial abscess. First of all, diagnostic search starts from transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE), in which a myocardial abscess appears as a hypoechoic mass lesion. Nevertheless, the sensitivities of TTE and TEE are low (28.3% and 48%, accordingly).[12] Certainly, cardiac computed tomography (CCT) and cardiac magnetic resonance imaging (CMRI) are the most helpful and accurate methods to identify MA.[14]

For surgical intervention, CCT and CMRI provide an opportunity to accurate anatomical location and relationship to surrounding structures. The MRI is more sensitive to abscess detection than other imaging techniques due to its superior temporal and spatial resolutions.[5]

It is extremely challenging to make a right diagnosis on time due to fast worsening condition of the patient and low clinical suspicion of doctors about myocardial abscess. COVID-19 pandemic made more complex medical service at all levels—from visit to family a doctor to the correct management of patients with chest pain.

Most patients require immediate treatment, and it is difficult to perform randomized controlled studies. Therefore, evidence-based guidelines are not yet available. Most of the recommendations have been made indirectly from the care of patients with infective endocarditis. Aggressive antibiotic treatment and prompt surgical intervention are the cornerstones of treatment this life-threatening illness.[13] Urgent surgery is recommended in most cases of MA since the delay to surgical intervention raises the perioperative risk and the risk of rupture.[1]

Beyond any doubt, an early diagnosis, aggressive medical therapy, multidisciplinary care, and timely surgical intervention may save the patient's life in this otherwise fatal condition.[4]

Conclusions

Our case describes impressively rare diagnosis of MA without infective endocarditis and obvious source of embolism. Multiple coronary septic embolism, which caused MA and STEMI, represents situation when a routine diagnosis of acute myocardial infarction could bring surprise in clinical practice.

Lead author biography

Dr Lina Lutsenko works at interventional cardiology department at National scientific center, M.D. Strazhesko institute of cardiology in Kyiv, Ukraine. She is currently focusing on primary PCI for patients with STEMI.

Supplementary material

Supplementary material is available at *European Heart Journal—Case Reports* online.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the relatives of the patient in line with COPE guidance.

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Data availability

The data underlying this article are available in the article and in its online supplementary material.

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