

# Subacute pericarditis following calcified ischemic apical aneurysm rupture and pseudoaneurysm formation

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We present a case of a 78-year-old diabetic male with multivessel coronary artery disease and a history of non-revascularized anterior myocardial infarction (15 years earlier), with severe left ventricular systolic dysfunction and apical aneurysm developed at that time. He had a single-chamber, apical cardioverter-defibrillator implanted soon after as a primary prevention strategy.

He presented with a two-week history of fatigue, weight loss, and fever. On admission, he was hemodynamically stable, febrile, with elevated inflammatory markers. Chest X-ray showed mild left pleural effusion (Supplementary material, *Figure S1*) and an electrocardiogram revealed sinus rhythm, PR segment depression, and anterior QS complexes (Supplementary material, *Figure S1, S2*).

Considering the systemic inflammatory syndrome, the patient underwent a thoraco-abdominopelvic computed tomography scan showing an enlarged left ventricle with a large intracavitary thrombus and an aneurysmatic and calcified apical wall, with two regions of apparent discontinuity, communicating with the saccular cavities also filled with thrombus, suggesting pseudoaneurysms (*Figure 1*). Echocardiography also showed mild pericardial effusion (Supplementary material, *Videos S1* and *S2*). Further investigation excluded infectious, autoimmune, and neoplastic causes. Antigranulocyte scintigraphy demonstrated isolated high uptake in the pericardium, apical wall, and pseudoaneurysms.

Although speculative, the subacute (two-week) presentation of a systemic inflammatory syndrome, with the exclusion of infectious, neoplastic, or autoimmune causes,

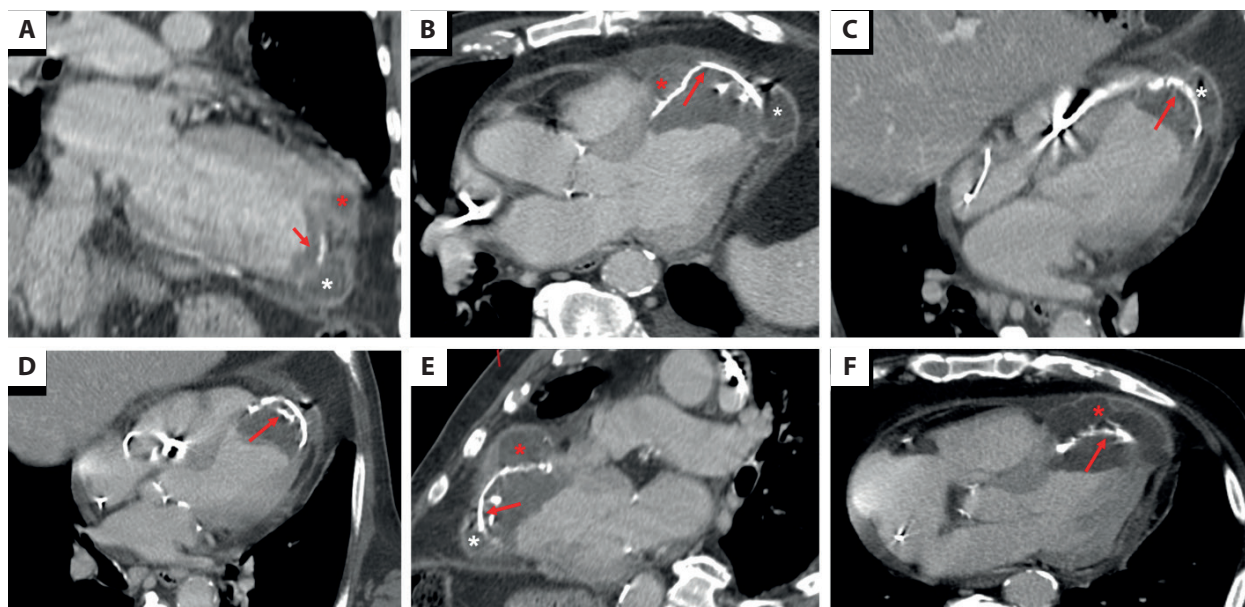
allied with the cardiac and nuclear imaging findings, supported a working diagnosis of pericardium — contained myocardial apical wall rupture with pseudoaneurysm formation and probable hemopericardium.

Therapeutic decisions were made by the Heart Team. The patient was considered inoperable due to unacceptable surgical risk. Pericardiocentesis was not pursued due to the risk of increased bleeding into the pericardial space (which precluded a definite etiological diagnosis of the effusion). Anticoagulation was not initiated because, despite a large apical thrombus, the contained free ventricular wall rupture posed a significant bleeding risk.

Considering this, we followed a conservative treatment of the pericarditis, with high-dose acetylsalicylic acid and colchicine. This strategy was successful in reducing inflammatory markers and bringing down fever, which made the working diagnosis more likely. Magnetic resonance imaging (MRI) was not performed since it would not have impacted management decisions and the device was not MRI conditional.

The patient was eventually discharged, under an anti-inflammatory tapering scheme and palliative care, when clinical status and pericardial effusion stability were evidenced. We later found that he suffered sudden death three weeks after discharge. Its cause is unknown but probably related to pseudoaneurysm rupture and cardiac tamponade.

We presented a patient with a late mechanical complication of myocardial infarction and its rare presentation as subacute hemopericarditis. The thin calcified wall of the left ventricular apex was a ticking time bomb,



**Figure 1.** Contrast-enhanced computed tomography: multiplanar reconstruction images, projecting 2-chamber (A), 3-chamber (B), 4-chamber (C), and 5-chamber (D) views, and a modified plane (E). The left ventricular (LV) apex is aneurysmatic, with a very thin and calcified wall (red arrow) and contains a large thrombus (27 × 49 mm). There are 2 regions of apparent discontinuity of the calcified LV wall in relationship with 2 globular sacs that protrude within the pericardium, with a hypodense lumen (red and white asterisks). There is minimal pericardial effusion. These images suggest a contained rupture of the LV apex (or pseudoaneurysms) filled with thrombus, a probable late sequel of myocardial infarction

and it could have presented itself earlier as sudden cardiac death. However, contained ruptures can be clinically silent, or they can present as mild unspecific clinical scenarios, including pericarditis [1]. Overall, contained ventricular wall with pseudoaneurysm formation has a poor prognosis if not subject to surgical repair. Patient management is often challenging and requires multidisciplinary and case-by-case analysis [2, 3]. In this case, due to anatomical and clinical conditions, the patient was not suitable for surgery, thus conservative management was pursued with knowledge of its probable unfavorable outcome.

### Supplementary material

Supplementary material is available at [https://journals.viamedica.pl/kardiologia\\_polska](https://journals.viamedica.pl/kardiologia_polska).

### Article information

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