Subacute (hematic) pericarditis following calcified ischemic apical aneurysm rupture and pseudoaneurysm formation

Authors: David Sá Couto, Mariana Santos, Maria Trêpa, Severo Torres, Patrícia Rodrigues
Article type: Clinical vignette
Received: July 1, 2023
Accepted: August 24, 2023
Early publication date: October 2, 2023
Subacute (hematic) pericarditis following calcified ischemic apical aneurysm rupture and pseudoaneurysm formation

Short title: Pericarditis due to calcified aneurysm rupture

David Sá Couto¹, ²*, Mariana Santos¹, ²*, Maria Trêpa¹, ³, ⁴, Severo Torres¹, ², Patrícia Rodrigues¹-⁴

Affiliations:
¹Department of Cardiology, Centro Hospitalar Universitário de Santo António (CHUdSA), Porto, Portugal
²ICBAS — School of Medicine and Biomedical Sciences, University of Porto, Porto, Portugal
³UMIB — Unit for Multidisciplinary Research in Biomedicine, ICBAS — School of Medicine and Biomedical Sciences, University of Porto, Porto, Portugal
⁴ITR— Laboratory for Integrative and Translational Research in Population Health, Porto, Portugal
*Both authors equally contributed to the study

Correspondence to:
David Sá Couto da Maia Romão, MD,
Department of Cardiology,
Centro Hospitalar Universitário de Santo António,
Address: Largo do Prof. Abel Salazar,
4099–001, Porto, Portugal,
phone: +351 222 077 500,
e-mail: david.sa.couto@gmail.com

A 78-year-old diabetic male with multivessel coronary artery disease and history of non-revascularized anterior myocardial infarction (15 years prior), evolved with severe left ventricle systolic dysfunction and apical aneurysm at the 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. At admission he was hemodynamically stable, febrile, with elevated inflammatory markers. Chest X-ray showed mild left pleural effusion (Supplementary Figure S1) and the electrocardiogram revealed sinus
rhythm, PR segment depression and anterior QS complexes (Supplementary material, Figure S1, S2).

Considering the systemic inflammatory syndrome, the patient underwent thoraco-abdominopelvic computed tomography scan revealing an enlarged left ventricle with a large intracavitary thrombus and an aneurysmatic and calcified apical wall, with two regions of apparent discontinuity, communicating with saccular cavities also filled with thrombus, suggesting pseudoaneurysms (Figure 1). Echocardiogram also showed a 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 exclusion of infectious, neoplastic, or autoimmune causes, allied with the cardiac and nuclear imaging findings, supports a probable diagnosis of contained myocardial apical wall rupture with pseudoaneurysm formation and probable hematic pericarditis.

Therapeutic decisions were made by a 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 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 fever resolution, making the presumed diagnosis more likely. Magnetic resonance imaging was not done since it would not impact management decisions and the device was not MRI conditional.

The patient was eventually discharged, under an anti-inflammatory tapering scheme and palliative care, after evidence of clinical and pericardial effusion stability. It was later known that he suffered sudden death three weeks after discharge, although unknown, probably related to wall rupture progression and cardiac tamponade.

We presented a patient with a late mechanical complication of a myocardial infarction and its rare presentation as subacute hematic pericarditis. The thin calcified wall of the left ventricular apex was a ticking time bomb, and it could have presented itself earlier as sudden cardiac death. However, contained ruptures can be clinically silent, or present themselves as mild unspecific clinical scenarios, including pericarditis [1]. Overall, contained myocardial rupture 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, the patient was not considered to have anatomical or clinical conditions 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.

**Article information**

**Conflict of interest:** None declared.

**Funding:** The publication of this article was funded by UMIB — Unit for Multidisciplinary Research in Biomedicine, ICBAS — School of Medicine and Biomedical Sciences, University of Porto, Porto, Portugal. Reference: UIDB/00215/2020; UIDP/00215/2020; LA/P/0064/2020.

**Open access:** This article is available in open access under Creative Common Attribution-Non-Commercial-No Derivatives 4.0 International (CC BY-NC-ND 4.0) license, which allows downloading and sharing articles with others as long as they credit the authors and the publisher, but without permission to change them in any way or use them commercially. For commercial use, please contact the journal office at kardiologiapolska@ptkardio.pl.

**References**


Figure 1. Computed tomography (with contrast): 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 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 an apparent discontinuity of the calcified apical LV wall in relationship with 2 globular sacs that protrude within the pericardium, with 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.