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CASE REPORT

BEGINNER

CLINICAL CASE

Anterior Myocardial Infarction Complicated by Apical Thrombus and Purulent Pericarditis

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ABSTRACT

We describe a man with cardiac tamponade after anterior myocardial infarction (MI) with a mass on echocardiography, complicated by a S. Milleri purulent pericarditis. This case is an example of how 2 cardiac conditions can interfere with the diagnosis and management of each individual disorder, resulting in an extremely complex disease course. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2022;4:101675) © 2022 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 37-year-old man was referred to the emergency department with flu-like symptoms persisting for 2 weeks. At presentation, the patient had a blood pressure of 99/66 mm Hg and a heart rate of 93 beats/min. Body temperature was 100.9 °F (38.3 °C). Physical examination revealed muffled heart sounds and elevated central venous pressure. The electrocardiogram showed an old anterior wall infarction and microvoltages (**Figure 1**). A transthoracic echocardiography showed a dyskinetic left ventricle with a left ventricular (LV) ejection fraction 10% to 20%, pericardial effusion, and >25% mitral valve inflow variation. Moreover, there was a mass present in the apical region (Video 1). The patient was transferred to our tertiary hospital for further treatment and investigation.

LEARNING OBJECTIVES

- To be able to make a differential diagnosis of an apical mass using multiple image modalities.
- To understand the presence of multiple cardiac conditions can interfere with the diagnostic work-up and treatment of each individual diagnosis.
- To know that *Streptococcus Milleri* can be causative of purulent pericarditis.

PAST MEDICAL HISTORY

Besides having a poor dental state, the patient was a healthy adult with no history of medication use.

DIFFERENTIAL DIAGNOSIS

The initial differential diagnosis was cardiac tamponade after recent anterior infarction caused by a (covered) ventricular wall perforation with either an intramyocardial hematoma or an intracardial thrombus present in the apex.

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ABBREVIATIONS AND ACRONYMS

CMR = cardiac magnetic resonance imaging

LV = left ventricular

MI = myocardial infarction

INVESTIGATIONS

The main laboratory investigations showed elevated C-reactive protein of 356 mg/L (<5 mg/L), leukocytes 25.9 × 10^9/L (4.0-10.0 × 10^9/L), and lactate 3.0 mmol/L, whereas cardiac markers were not initially determined. A coronary angiogram was performed, which showed only an occlusion in the mid left anterior descending artery (Video 2). Cardiac magnetic resonance imaging (CMR) and computed tomography were performed but could not differentiate between an intramyocardial hematoma or intracardial thrombus (Videos 3 and 4). Cultures from the pericardial fluid showed a *Streptococcus Milleri*. A fluorodeoxyglucose positron emission tomography computed tomography confirmed the diagnosis of a purulent pericarditis (**Figure 2**).

MANAGEMENT

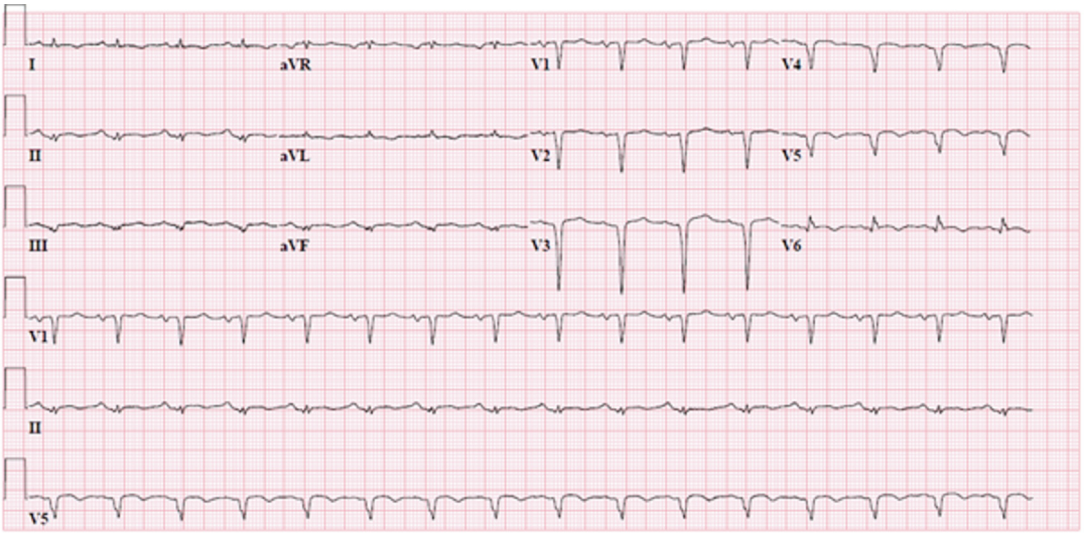
Because of further hemodynamic deterioration after arriving at our hospital, an acute pericardiocentesis was performed after which the patient improved. A coronary angiogram showed an occluded left anterior descending artery for which no intervention was performed. Reversibility of the myocardial damage was deemed unlikely. Given the bleeding risk, the decision was made to withhold anticoagulation until the etiology of the LV mass could be further elucidated. Treatment of the purulent pericarditis consisted of antibiotic therapy (piperacillin/tazobactam). After recurrence of pericardial effusion, percutaneous pericardial lavage was initiated. This was followed by surgical exploration, in accordance to the European Society of Cardiology guidelines on purulent pericarditis.[1] Intrapericardial thrombolysis was deemed unfeasible in the light of a possible intramyocardial hematoma. Pericardial lavage continued until there were multiple negative cultures. The patient received 6 weeks of antibiotic treatment.

Simultaneously, heart failure treatment[2] consisting of beta-blocker, angiotensin-converting enzyme inhibitor, and mineralocorticoid receptor antagonist was started and up-titrated. After further stabilization, acetylsalicylic acid was started.

DISCUSSION

Purulent pericarditis is an uncommon condition occurring in <1% of acute pericarditis.[3] Although streptococci are a common causative, involvement of *S. Milleri* is rare. In isolated purulent pericarditis, the recommended treatment is aggressive, because mortality rates are extremely high with conservative treatment.[4] However, the purulent pericarditis in our case was not isolated. Because of the unknown etiology of the apical mass and the recent anterior

FIGURE 1 Electrocardiogram at Presentation



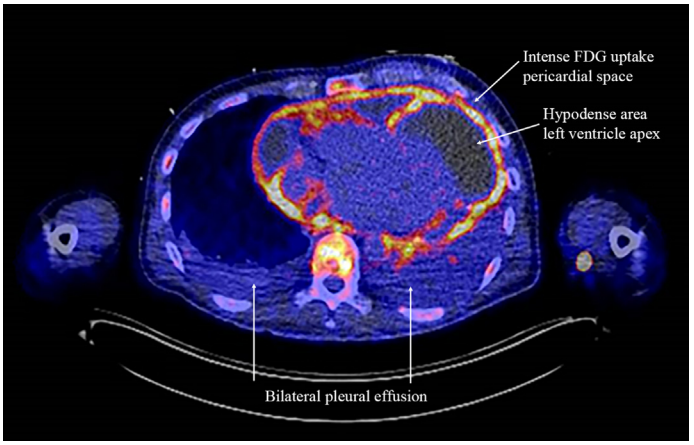
A QS pattern in the frontal leads was suggestive of an anterior wall infarction of earlier date. Microvoltages were suggestive of pericardial effusion.

myocardial infarction (MI), thrombolysis was deemed infeasible. Eventually, with a combination of intravenous antibiotic therapy, surgical drainage, and pericardial lavage, we were able to successfully treat the purulent pericarditis without relapse.

A LV thrombus is a known complication of acute MI with a prevalence of around 4% in the primary percutaneous coronary intervention era. It occurs most often after anterior wall infarction with an LV ejection fraction <40% and apical akinesis.[5] A thrombus is often a homogeneous and smooth mass, but it can also be cavitary and heterogeneous.[6] An intramyocardial hematoma, on the other hand, is a rare complication. It is thought to be the result of hematoma formation between layers of myocardial fibers, and is considered a form of incomplete cardiac rupture. An intramyocardial hematoma starts to develop early during the MI—in 22% of cases it occurs in <24 hours—and is potentially fatal because of the risk of complete rupture.[7] On echocardiography, it presents with nonhomogeneous neocavitations. Flow is often visible between the intracardial and pericardial cavity. In our case, we did not find these "typical" findings. However, a definite diagnosis of an intracardial thrombus was not possible either, because we could not make a distinction between thrombus and layers of pericardium/myocardium, also because of the concomitant presence of pericardial fluid. A delayed enhancement CMR was performed, which is the most accurate modality for detection of LV thrombi.[8] However, the CMR was not conclusive, probably because of the complexity of the anatomy. Moreover, diagnostic accuracy was disturbed by the ongoing purulent pericarditis and surgical interventions for the pericarditis.

There are no observational of randomized studies available that describe a safe initiation of anticoagulation therapy in an intramyocardial hematoma. Therefore, also considering the extensive surgical drainage and our inability to establish a definite diagnosis, we opted for a conservative treatment with only single antiplatelet therapy.

FIGURE 2 **Positron Emission Tomography Computed Tomography With Low-Carb Preparation**

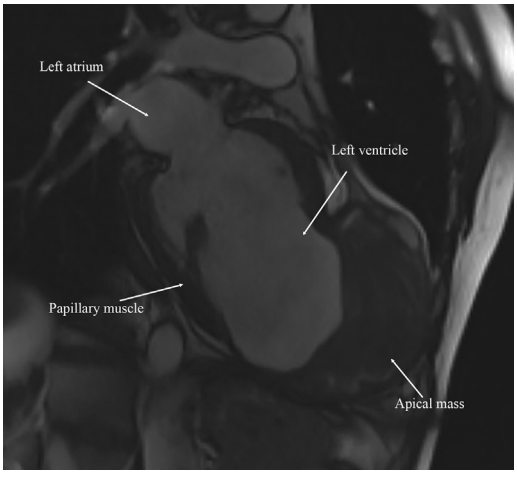


An intense fluorodeoxyglucose (FDG) uptake in pericardial space is visible, compatible with the diagnosis of purulent pericarditis. Also, a hypodense area in the left ventricle apex is shown without fluorodeoxyglucose uptake.

FOLLOW-UP

On day 98, the patient was discharged in reasonable condition. In the outpatient clinic, CMR was repeated and showed a persisting, denser, and organized left ventricle mass that was suspect for intracardial thrombus (**Figure 3**). Acetylsalicylic acid was converted to vitamin K antagonist (acenocoumarol). A bleeding occurred from a fistula (retrosternal to subcutaneous cavity below xiphoid) that developed during pericardial drainage, which was managed conservatively. During follow-up, the intraluminal mass slowly decreased in size with flow visible on the edges, proving an intracardial LV thrombus, that eventually almost entirely disappeared (Video 5). After 2 years, the patient is in good cardiac condition, New York Heart Association functional class I-II,

FIGURE 3 **CMR 4 Months After Discharge**



An apical 2-chamber view showing a mass in left ventricle apex with a more organized aspect.

while the LV ejection fraction remains poor and with an aneurysmatic left ventricle.

CONCLUSIONS

We present this case as an example of the unique combination of rare cardiac diagnoses and how the presence of both interferes with the diagnostic work-up and treatment of each individual condition. Resulting in an extremely complex disease.

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REFERENCES

1. Adler Y, Charron P, Imazio M, et al. 2015 ESC guidelines for the diagnosis and management of pericardial diseases: the Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC). Endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J*. 2015;36(42):2921–2964.

2. Yancy CW, Jessup M, Bozkurt B, et al. 2017 ACC/AHA/HFSA focused update of the 2013 ACCF/AHA Guideline for the management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Failure Society of America. *J Am Coll Cardiol*. 2017;70(6):776–803.

3. Imazio M, Brucato A, Mayosi BM, et al. Medical therapy of pericardial diseases: part I: idiopathic and infectious pericarditis. *J Cardiovasc Med (Hagerstown)*. 2010;11(10):712–722.

4. Pankuweit S, Ristic AD, Seferovic PM, Maisch B. Bacterial pericarditis: diagnosis and management. *Am J Cardiovasc Drugs*. 2005;5(2):103–112.

5. Gianstefani S, Douiri A, Delithanasis I, et al. Incidence and predictors of early left ventricular thrombus after ST-elevation myocardial infarction in the contemporary era of primary percutaneous coronary intervention. *Am J Cardiol*. 2014;113(7):1111–1116.

6. Mathew JP, Swaminathan M, Ayoub CM. *Clinical Manual and Review of Transoesophageal Echocardiography*. 2nd ed. McGraw-Hill Education; 2010.

7. Lewis AJ, Burchell HB, Titus JL. Clinical and pathological features of postinfarction cardiac rupture. *Am J Cardiol*. 1969;23(1):43–56.

8. Roifman I, Connelly KA, Wright GA, Wijeysundera HC. Echocardiography vs. cardiac magnetic resonance imaging for the diagnosis of left ventricular thrombus: a systematic review. *Can J Cardiol*. 2015;31(6):785–791.

KEY WORDS case report, intramyocardial hematoma, left ventricular thrombus, purulent pericarditis

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