A Case of Pericarditis and Pericardial Masses Associated With Mycobacterium Paragordonae

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Clinical Medicine Insights: Cardiology
Volume 17: 1–4

DOI: 10.1177/11795468231189039

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ABSTRACT: Tuberculosis is a common cause of pericarditis worldwide and has been associated with pericardial masses. Non-tuberculous mycobacteria are uncommonly associated with cardiac disease, having primarily been described in cases of endocarditis. Here we describe a case of an immunocompetent patient with Mycobacterium paragordonae infection causing pericarditis with a large effusion containing pericardial masses. The patient presented with chest pain, hypoxia and biochemical evidence of inflammation (CRP 216.1 mg/L). This report illustrates a rare case of pericarditis with pericardial masses associated with non-tuberculous mycobacteria and the first example of pericarditis associated with M. paragordonae.

KEYWORDS: Pericarditis, non-tuberculous mycobacteria, pericardial mass, emerging pathogen

RECEIVED: March 3, 2023. **ACCEPTED:** June 23, 2023.

TYPE: Case Report

FUNDING: The author(s) received no financial support for the research, authorship, and/or publication of this article.

DECLARATION OF CONFLICTING INTERESTS: The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Introduction

Pericardial masses constitute a heterogenous group for which the differential diagnosis includes tumors (primary and secondary/metastatic) and non-neoplastic masses including pericardial cyst, diverticulum, inflammatory pseudotumor, hematoma, thrombus, fibroma, and rarely, infection.[1,2] The clinical presentation varies depending on the etiology of the mass as well as with associated features such as effusion causing tamponade or systemic signs and symptoms of malignancy or infection. Diagnosis requires a combination of medical history, physical exam, imaging, biopsy, and pericardial fluid analyses.

Mycobacterium paragordonae is an emerging pathogen that has been associated with pulmonary disease, vertebral infection and peritonitis.[3-5] It is highly phylogenetically related to the non-tuberculous mycobacterium (NTM) *M. gordonae* and has been identified as a contaminant in hospital water supplies.[3,6,7] Tuberculosis is a common cause of pericarditis in lower middle income countries and has been shown to be associated with pericardial masses[8-10] but NTM only rarely cause cardiac disease. There have been case reports describing NTM in association with pericarditis, however not specifically with *M. paragordonae*.[11,12] They have been described in cases of endocarditis (usually secondary to contaminated procedural equipment)[13-18] and less commonly in cases of pericarditis.[11,19] Furthermore, *M. paragordonae* pericarditis has never been documented. Here we describe the case of a patient with pericarditis and pericardial masses associated with *M. paragordonae*.

Case Presentation

A 60-year-old female with a history of hypothyroidism and dyslipidemia presented to the emergency department at a peripheral hospital complaining of chest pain. The patient was not known to be immunocompromised, HIV testing was negative, and there was no evidence of leukopenia at presentation. Medications on transfer included amiodarone 400 mg po BID, Apixaban 5 mg po BID, Digoxin 1.25 mg po daily, levothyroxine 75 µg po daily (TSH was not assessed in hospital), metoprolol 25 mg po BID, rosuvastatin 10 mg po QHS, amoxicillin/clavulanic acid 875 mg po BID. She had been hospitalized in the intensive care unit at an international facility (Florida, USA) approximately 10 days earlier for epiglottitis secondary to *Haemophilus influenzae* infection for which she was treated with antibiotics and a short course of glucocorticoids. Unfortunately we did not have access to the medical records from that admission. During that index hospitalization she had reported chest pain, subjective fevers and was noted to be in atrial fibrillation for which she was started on oral anticoagulation and amiodarone. She was ultimately discharged on amoxicillin/clavulanic acid. While traveling home, she developed worsening chest pain which prompted her to seek further medical attention.

On assessment in the peripheral hospital, she was afebrile, normotensive with a blood pressure of 133/70 mmHg, tachycardic with a heart rate of 103 beats per minute, and hypoxic requiring 4 L of supplemental oxygen via nasal prongs. The patient described her pain as sharp and positional, worsening with lying flat and improving with sitting upright. Initial investigations included a leukocyte count of 31×10^9/L with a neutrophil count of 27.9×10^9/L, hemoglobin of 145 g/L, platelets of 434×10^9/L, troponin of <0.01 µg/L, creatinine of 54 µmol/L and lactate of 2.8 mmol/L. Echocardiogram revealed a left ventricular ejection fraction of 67%, a large pericardial effusion with a dilated, non-collapsible inferior vena cava and excessive respiratory variation in the tricuspid valve inflow velocities. The effusion was noted to contain multiple echodensities. Given the concern for progression to tamponade and the complex nature

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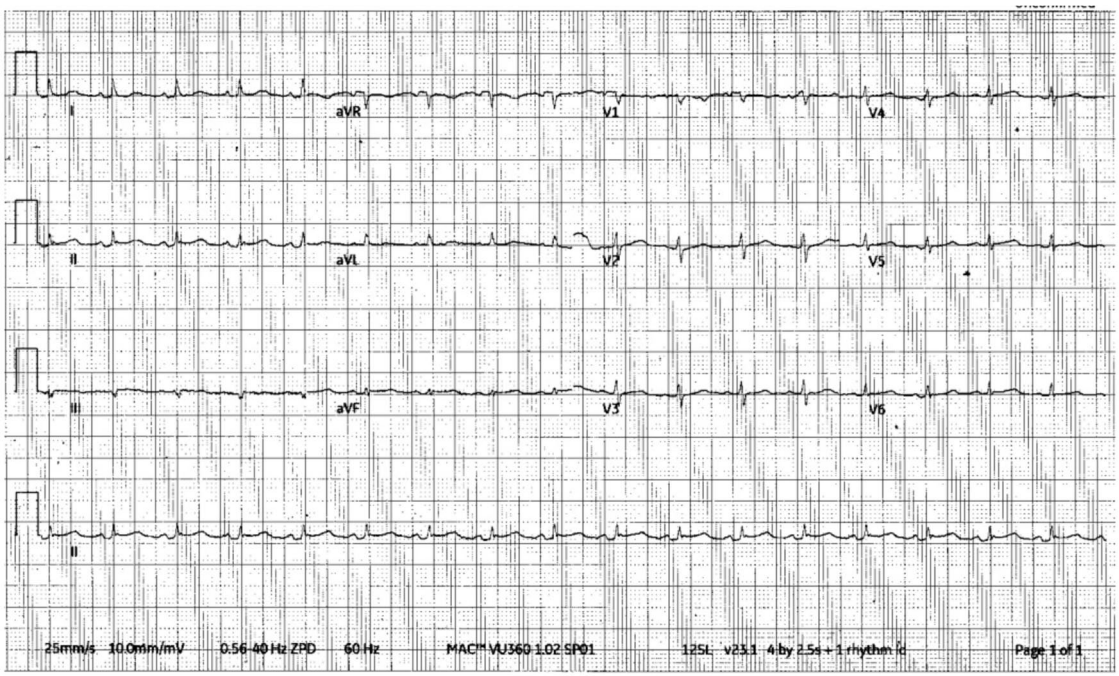

Figure 1. Twelve lead electrocardiogram obtained at the time of admission shows low voltages and ST segment elevation in the inferior leads.

of the effusion, the patient was transferred to the Cardiac Care Unit and our center for further management. Prior to transfer, treatment for pericarditis was initiated with aspirin 650 mg po bid and colchicine 0.6 mg po bid.

On our initial assessment, the patient remained afebrile, tachycardic (heart rate 102 beats per minute) and hypoxic (oxygen saturation 93% on 5 L supplemental oxygen). Physical exam was otherwise remarkable for mild pitting edema to the lower limbs. The heart rate was regular with no murmurs and no evidence of a pericardial friction rub. We completed an initial diagnostic workup in keeping with current guidelines.[20] Results of laboratory investigations were similar to those performed in the peripheral hospital. C-reactive protein (CRP) was 216.1 mg/L, lactate was 2.8 mmol/L and the leukocyte count was 31×10^9 cells/L. The ECG showed sinus tachycardia with low voltages and ST segment elevation in the inferior leads (Figure 1). The echocardiogram was repeated and confirmed the prior findings. With respect to the echodensities, there was a free-floating mass measuring 6.1 cm × 1.5 cm with 2 smaller densities measuring 1.8 cm × 1.2 cm and 1.1 cm × 2.3 cm respectively These were thought to be extensions of the larger mass. A further 2 echodensities measuring 1.1 cm × 0.5 cm and 1.1 cm × 1.6 cm were identified and noted to be adherent to the visceral pericardium (Figure 2A and B). The differential diagnosis for the masses as listed in the echocardiogram report included thrombi, inflammatory, and neoplasm.

We proceeded with pericardiocentesis and removed a total of approximately 300 mL of serous, non-purulent fluid. A CT scan of the chest, abdomen and pelvis 3 days later showed a small pericardial effusion with no evidence of enhancing lesions within the pericardium. Repeat echocardiogram after

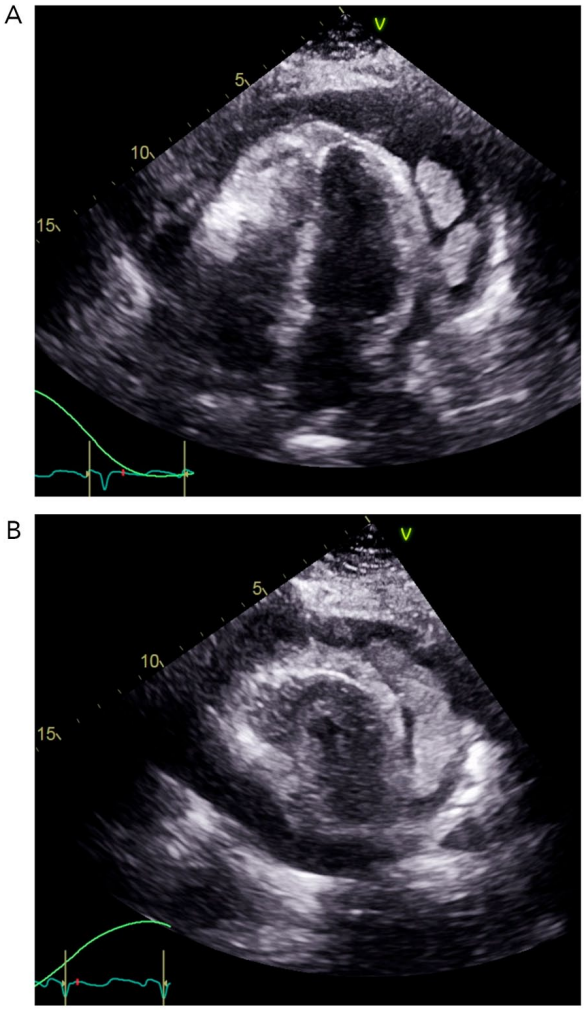

Figure 2. Apical 4 chamber (A) and parasternal short axis (B) echocardiogram images show multiple pericardial masses.

pericardiocentesis showed a trivial pericardial effusion with a persistent echodensity anterior to the left ventricle, albeit less prominent than on the previous imaging. We did not perform a cardiac MRI.

Bacterial culture and PCR of the pericardial fluid were both negative, as was the stain for acid fast bacilli and the fungal culture. Mycobacterial culture however was positive for *M. paragordonae*. Given the delay in obtaining this result, the patient had since been discharged with a plan to complete a course of amoxicillin/clavulanic acid for a total of 4 weeks. She was clinically stable on colchicine for ongoing pericarditis treatment and repeat echocardiogram showed only a small circumferential pericardial effusion with no evidence of pericardial masses. The CRP had decreased to 45.1 mg/L and the leukocyte count had normalized. The patient was seen in follow up by the Infectious Disease Team and the decision was made to treat with ethambutol 1600 mg po daily, ciprofloxacin 500 mg po bid, and doxycycline 100 mg po bid for a duration of 12 months.

Discussion

The differential diagnosis for pericardial masses is broad with pericardial cysts, lipomas, mesothelioma and metastatic breast cancer amongst the most common etiologies.[1] There have been case reports of tuberculous pericarditis associated with pericardial masses[8-10] but NTM have never been associated with this rare finding. In all of these cases, the masses were surgically excised and described as soft, yellow and disc-shaped. The pathological description varied between granulomatous tissue with or without necrosis and amorphous threads with numerous acid-fast bacilli. In our case, we proceeded directly to pericardiocentesis and the masses were no longer visible on CT scan, however, the echocardiographic appearance of the largest mass, prior to drainage of the effusion, was consistent with these previous case studies.

Both *M. gordonae* and *M. paragordonae* are opportunistic pathogens. Interestingly, it has previously been shown that HIV infection is associated with reduced granuloma formation in tuberculous pericarditis, particularly in patients with CD4 cell counts of less than 200 cells/μL, suggesting that an intact immune response is important for this process.[21] Our patient developed an infection with *M. paragordonae* in the setting of recent epiglottitis requiring treatment with antibiotics and a short course of steroids. While we cannot say with certainty that these pericardial masses were granulomatous in nature, the similarity in appearance is suggestive and NTM have been associated with granuloma formation.[22]

Unfortunately we did not have access to the patient's medical records from the initial presentation in Florida. The source of the *M. paragordonae* remains unclear and may have been acquired during the index admission. Another possibility is that the *M. paragordonae* is a contaminant and the pericardial masses have another etiology. This seems less likely as the remainder of the pericardial fluid analyses as well as imaging studies were negative.

Conclusions

M. paragordonae is an emerging pathogen with a disease profile that remains to be fully elucidated. Our case highlights several novel and important clinical features including the association of this organism with pericarditis and pericardial masses reminiscent of tuberculous disease.

Author Contributions

RJ and TR drafted the manuscript which was reviewed by MS.

Consent Statement

We have obtained written informed consent from the patient to share the details of this case.

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