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CASE REPORT
Cardiac imaging

Listeria myopericarditis associated with right atrial mural thrombus: a case report

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Background Pericarditis is a common cardiology presentation, most often due to a viral or idiopathic cause. Listeria as a cause of pericarditis is rare. Listeria is an infection that is readily treatable with antibiotics following accurate identification. Without adequate treatment, Listeria infection has a high mortality rate.

Case summary In this case, a fit and well 59-year-old man complained of headaches and fever to the emergency department (ED). He was provisionally diagnosed with giant cell arteritis (GCA) and commenced on management pathways for GCA. He represented to the ED with chest pain and electrocardiogram (ECG) changes suggestive of a clinical presentation of pericarditis. He received treatment for idiopathic pericarditis with no clinical resolution. Cardiac magnetic resonance imaging (MRI) showed myopericardial inflammation associated with a right atrial mural thrombus. After 2 weeks of poor treatment response, peripheral blood cultures grew Listeria monocytogenes and the patient responded well to antibiotic treatment. Repeat cardiac MRI after an extended course of antibiotics showed resolution of MRI signs.

Discussion This is a case of Listeria myopericarditis. Physicians should consider rarer causes of myopericarditis in treatment resistance cases. Cardiac MRI has utility in atypical or treatment resistant patients to assess myopericardial inflammation and response to treatment.

Keywords Pericarditis • Magnetic resonance imaging • Listeria • InfectionCase report

Learning points

- In atypical presentation or treatment resistant pericarditis, a physician should consider rarer causes of pericarditis.
- Cardiac magnetic resonance imaging can aid in the diagnosis and treatment monitoring of patients with myopericarditis, especially in atypical cases.

Introduction

The most common cause of myopericarditis in an immunocompetent individual is viral or idiopathic, with a specific diagnosis established in only 17%.[1]

Listeria monocytogenes infection is more common in the immunosuppressed, pregnancy and neonates than immunocompetent adults. It occasionally infects previously healthy individuals. Its involvement in cardiac disease has mostly been limited to endocarditis and myocarditis and has a high mortality burden. Where Listeria pericarditis has been described, it has often been associated with an immunosuppressed host and a high mortality.

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Timeline

Day 0	Initial presentation to emergency department (ED). The patient was provisionally diagnosed as giant cell arteritis. Prednisolone 40 mg once daily commenced and patient to await temporal artery biopsy.
Day 6	Second ED presentation. Clinical diagnosis of pericarditis. Ibuprofen 400 mg three times daily commenced. Prednisolone reduced to 20 mg once daily.
Day 12	First cardiac magnetic resonance imaging (MRI) performed. Evidence of myopericardial inflammation and right atrial mural thrombus. Colchicine 500 µg twice daily started. Ibuprofen and Prednisolone continued.
Day 20	Listeria monocytogenes blood cultures were grown. The patient was commenced on IV Amoxicillin 2 g three times daily. Prednisolone stopped.
Day 34	IV Amoxicillin stopped. Oral Linezolid 600 mg twice daily for 2 weeks started.
Day 37	The patient was discharged from hospital with clinical and biochemical improvement.
Day 49	Third cardiac MRI showing resolution of inflammation and extended antibiotic course complete.
5 Months	End of follow-up.

Case presentation

A fit and well 59-year-old man presented with headaches and fevers of 2 weeks duration to hospital. The headaches were a pressure-like sensation, of gradual onset, radiating to the jaw with maximal tenderness in the temporal region. There was no associated photophobia or neck stiffness. He was pyrexial on assessment. A provisional diagnosis of giant cell arteritis (GCA) was made and he was commenced on prednisolone 40 mg daily to await a temporal artery biopsy.

Six days later, he represented with chest pain to hospital. It was of 5 days duration, sharp in character, radiated to the jaw and shoulder and was worse on lying flat. There was no exertional tendency to the pain. There was no associated shortness of breath.

The fever and headaches were ongoing. Recent unintentional weight loss was reported.

A 12 lead ECG showed sinus rhythm, PR depression and ST-segment elevation across all leads (*Figure 1*). A clinical diagnosis of acute pericarditis was made.

Chest X-ray showed bilateral blunting of the costophrenic angles. Laboratory results showed elevated white cells (WCC) at 17.1 $\times$ 10^9/L[2–10] and a raised C-reactive protein (CRP) at 149 mg/L (<5). Two separate blood cultures from admission were negative. He had normal renal and liver function tests. High sensitivity troponin T was slightly elevated at 24 ng/L (<14 ng/L).

A magnetic resonance imaging (MRI) of the brain showed no acute abnormality.

The patient was treated with Ibuprofen 400 mg three times a day and the previously commenced prednisolone was reduced to 20 mg daily.

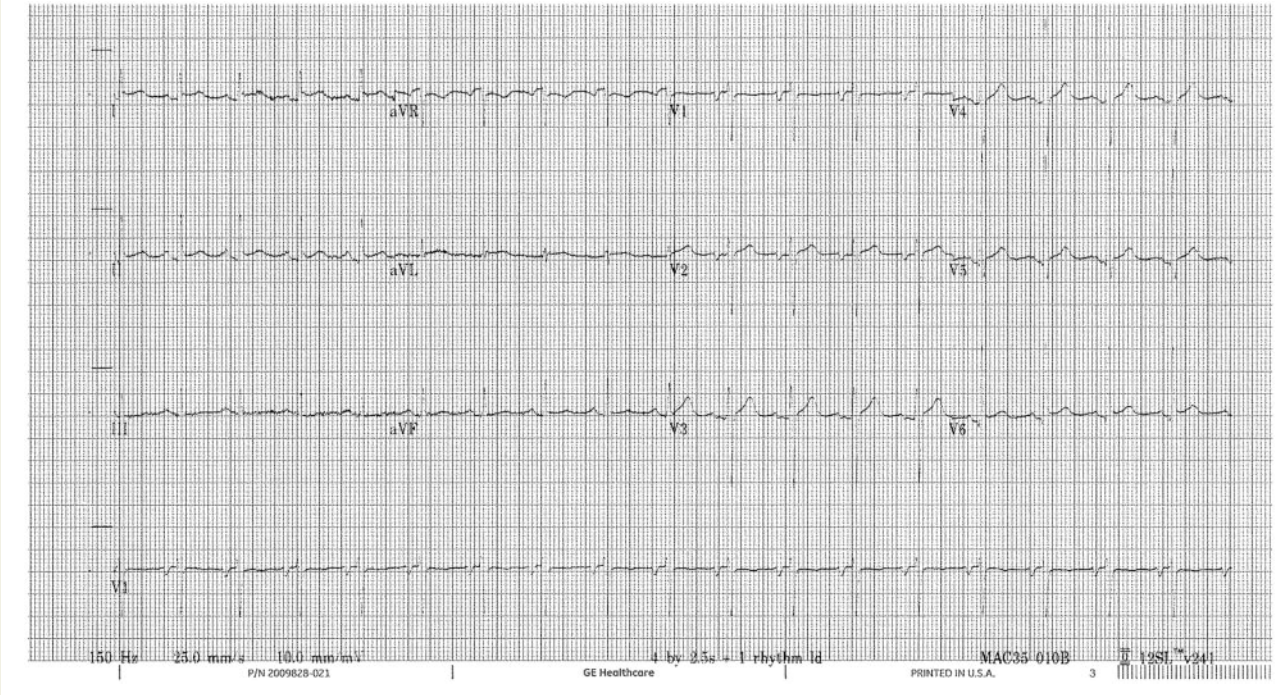

Figure 1 Twelve lead ECG.

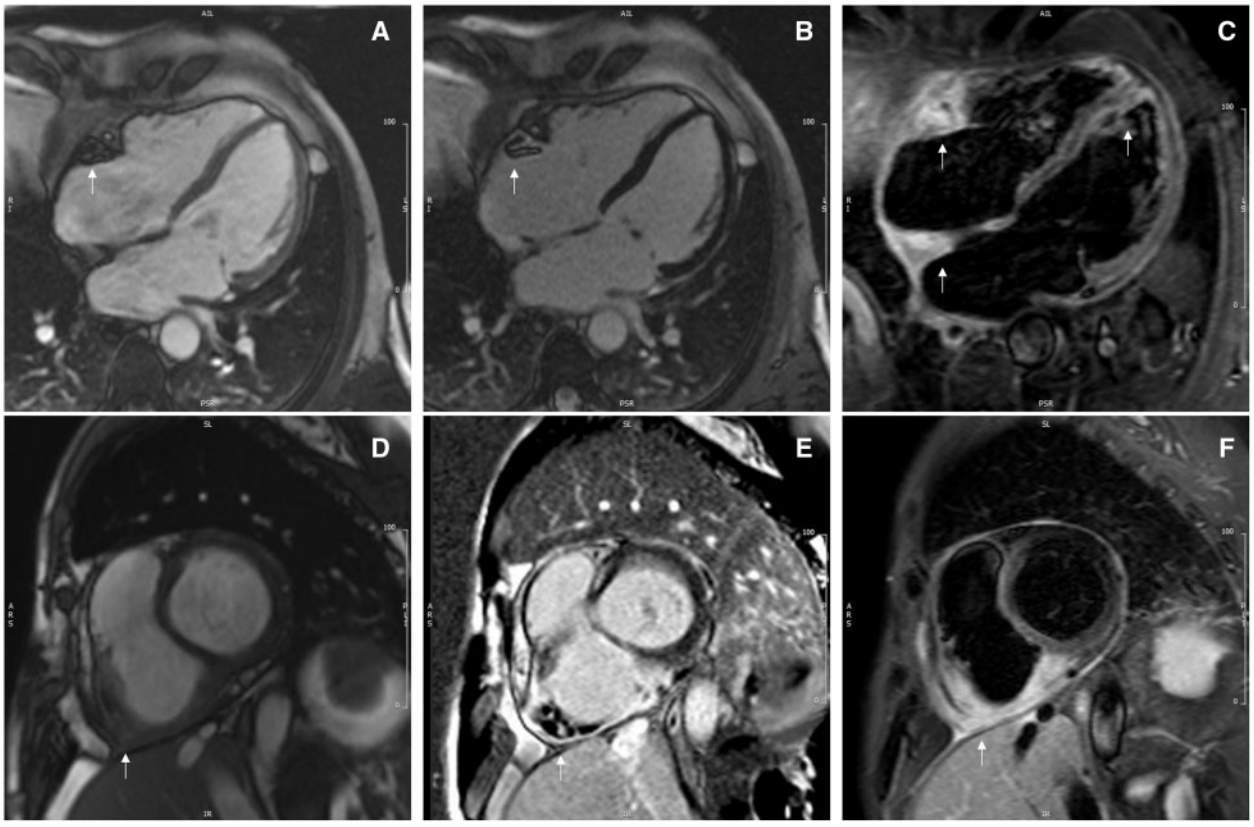

Figure 2 First cardiac MRI scan. (*A*) Four-chamber early gadolinium enhancement (EGE) showing RA thrombus (arrow, images *A*–*E*), (*B*) four-chamber late gadolinium enhancement, (*C*) four-chamber STIR with marker showing myocardial oedema of RA septum and infero-lateral wall (arrows), (*D*) short-axis (SAX) CINE showing RA thickening (arrow), (*E*) SAX late gadolinium enhancement and phase sensitive inversion recovery (PSIR) showing extensive enhancement of RA with mural thrombus (arrow), and (*F*) SAX STIR imaging showing extensive myocardial oedema of inferior wall (arrow).

A transthoracic echocardiogram showed a small pericardial effusion measured at 1.2 cm posterior to the left ventricle (LV) and 0.6 cm behind the right atrium (RA) with no associated haemodynamic compromise. Left ventricular systolic function was normal and ejection fraction measured at >55%. No thrombus was seen.

A computed tomography (CT) Chest–Abdomen–Pelvis was performed to investigate for malignancy due to pyrexia and weight loss. This investigation showed a 30 × 13 × 50 mm lesion in the pericardial space lateral to the RA. On CT this was thought to be a mass of unclear aetiology.

A contrast enhanced cardiac MRI was arranged (*Figure 2A–F*). It showed a thickened pericardium with pericardial enhancement on late gadolinium enhancement. Short-tau inversion recovery (STIR) imaging showed pericardial oedema. There was myocardial oedema in the right atrium and basal inferior wall of the right ventricle on STIR imaging. A soft tissue mass was seen around the posterior wall of the RA. It had higher signal than tissue on T2 and STIR images. No blood supply was seen on perfusion imaging.

The overall appearance was of myopericarditis and a right atrial mural thrombus. Anticoagulation was not commenced. This decision was based on the MRI appearances of the thrombus; that it did not appear mobile and appeared attached to the atrial free wall. Colchicine 500 µg twice daily was added to Ibuprofen and Prednisolone after the cardiac MRI findings.

The cardiac MRI was repeated after 1 week to assess for change. The patient continued to be intermittently pyrexial and complain of chest pains and headache. There was no improvement in the myopericardial inflammation in comparison to the previous MRI.

Two weeks after admission, inflammatory markers remained high; WCC 21.9 × 10^9/L and CRP 183 mg/L and five fevers had been observed. At this time, two sets of peripheral blood cultures grew Listeria monocytogenes. It was a fully sensitive organism. On review by the infectious diseases team, a lumbar puncture was not performed as there were no signs of meningism. After consultation with microbiology, he was treated with IV amoxicillin 2 g three times daily for 2 weeks and then 2 weeks of oral linezolid 600 mg twice daily. Ibuprofen and colchicine were continued. Investigations for immunosuppression (HIV, Epstein-Barr Virus, hepatic cirrhosis, immunoglobulins, blood film, cancer) were negative.

The patient improved clinically and biochemically when started on antibiotics. WCC was 13.9 × 109/L and CRP 3 mg/L on completion of IV antibiotics and no further fevers were recorded. He was discharged home to complete the 2 weeks of linezolid.

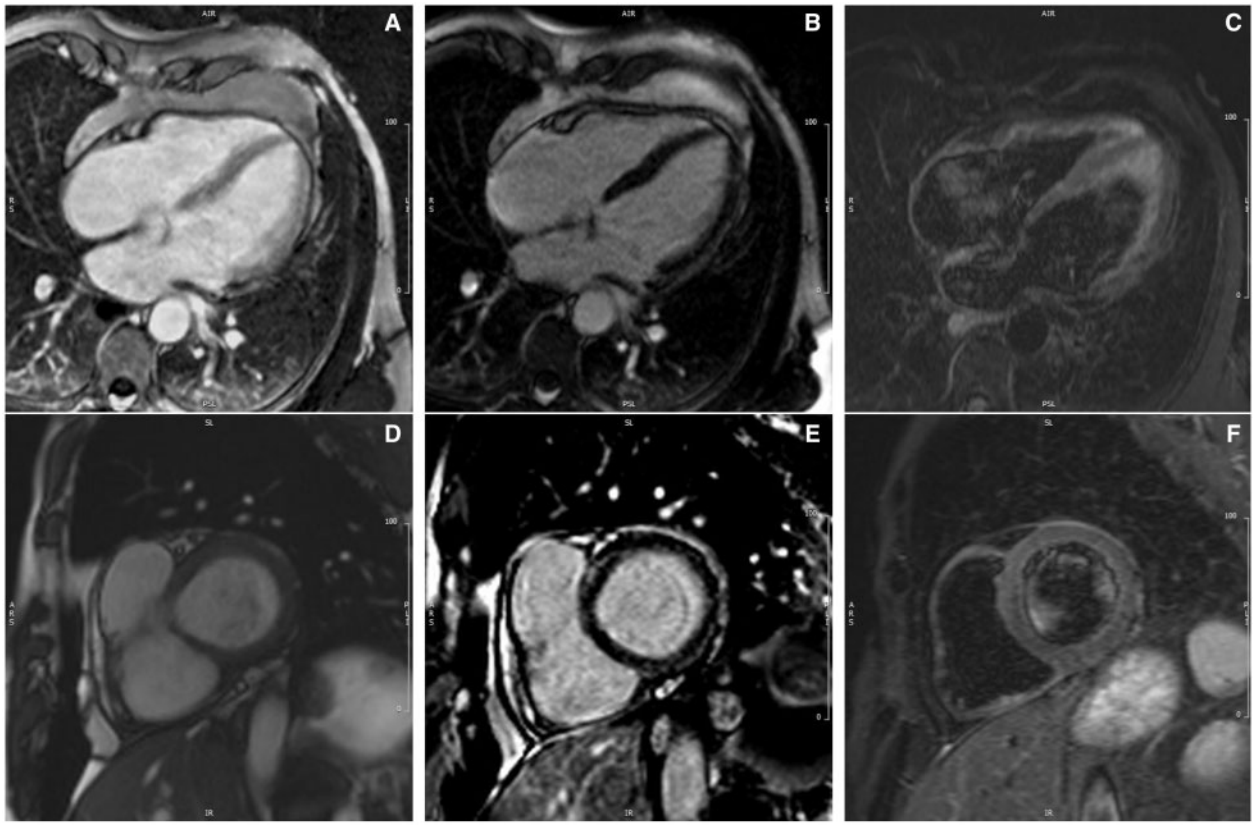

Figure 3 Third cardiac magnetic resonance imaging scan showing resolution of abnormalities seen in *Figure 2*. (*A*) Four-chamber EGE, (*B*) four-chamber late gadolinium enhancement, (*C*) four-chamber STIR, (*D*) SAX CINE, (*E*) SAX post-contrast late gadolinium enhancement, and (*F*) SAX STIR imaging.

A follow-up cardiac MRI (*Figure 3A–F*) was performed after completing antibiotics (6 weeks after admission). This showed improvement of myopericardial inflammation and right atrial mural thrombus had entirely resolved.

The patient recovered well from myopericarditis and has reached end of follow-up.

Discussion

Cardiovascular involvement of Listeria monocytogenes infection is rare and usually presents as myocarditis with endocarditis. A Medline search shows 10 previously reported cases of Listeria pericarditis. There is a high mortality rate reported; 60%. The majority (8/10) of cases had immunosuppression (HIV infection, hepatic cirrhosis, cancer or immunosuppressive medication, haemodialysis) predisposing to Listeria infection.[2–8,11] The described case contrasts with previously reported literature of Listeria pericarditis, occurring in an immunocompetent host with excellent resolution following antibiotic treatment.

Right atrial mural thrombus associated with pericarditis is very rare; the association limited to a case report.[9] In one case series, right atrial mural thrombus was most commonly associated with central venous catheter insertion, 96%, with 25% associated with endo/myocarditis.[10]

Right heart thrombi can be categorized as Type A or Type B based on its characteristic appearance with Type A appearing serpiginous and highly mobile resulting in excessively high mortality and Type B arising from within the heart and having thrombus related mortality of 4%.[12,13]

In this case, the appearance of the lesion on cardiac MRI was a thrombus with Type B features; appearing attached to the atrial wall and not highly mobile. It did not have the appearance of a vegetation based on tissue characterization. It was presumed to have arisen in association with the cardiac abnormality. Consequently, the patient was managed conservatively with the treatment of the underlying cause, listeria myopericarditis. This treatment resulted in complete resolution of the thrombus and no adverse event for the patient.

Listeria monocytogenes is gram-positive facultatively anaerobic, rod-shaped bacterium. It commonly affects immunosuppressed, the elderly, women during pregnancy and neonates. It occasionally affects previously healthy individuals. Common clinical forms of listeria infection include neurolisteriosis and bacteraemia. Infection is associated with high mortality; the MONALISA trial, a prospective observational cohort study in France found 3-month mortality of 45% in Listeria bacteraemia.[14] There was no immunodeficiency disorder identified

to make the patient at higher risk of Listeria monocytogenes infection. Prednisolone was initially commenced for GCA treatment and continued at a low dose once the diagnosis of myopericarditis was made. This is contrary to ESC guidelines whereby Ibuprofen, and Colchicine as adjunct therapy, has Class I recommendation. Corticosteroids have a Class IIa recommendation in cases of treatment failure and when an infectious cause has been excluded. The patient received a cumulative dose of 520 mg prednisolone prior to the positive blood cultures, and diagnosis, of Listeria. This dose of prednisolone is lower than that associated with an increased risk of infection from corticosteroid use.[15]

This is a case of Listeria myopericarditis. Physicians should consider rarer causes of myopericarditis in treatment resistance cases. Magnetic resonance imaging was of utility to characterize and monitor myopericarditis and thrombus in this case.

Lead author biography

Andrew Brown is a clinical fellow in cardiology at Northumbria Healthcare NHS Foundation Trust. He graduated in 2016 from Newcastle University and completed UK foundation training at Northumbria. He has been a clinical fellow for the past 12 months gaining further experience in the speciality of cardiology.

Supplementary material

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

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 patient in line with COPE guidance.

Conflict of interest: none declared.

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