Acute myopericarditis caused by *Salmonella enterica* serovar Enteritidis: a case report

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**Background**

Acute myopericarditis can be caused by a myriad of infectious and non-infectious aetiologies, however, it is often considered to be due to self-limiting viral infection. *Salmonella* spp. myopericarditis is rare and the few cases in the literature suggest significant associated morbidity and mortality.

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**Case summary**

A 44-year-old man presented with fever, dyspnoea, and chest pain. He was found to have a large pericardial effusion with clinical signs of tamponade and sepsis. Therapeutic pericardiocentesis was performed and ceftriaxone and levofloxacin were administered. Fully sensitive *Salmonella enterica* serovar Enteritidis (S. Enteritidis) was isolated in his pericardial fluid and he made a full recovery after a 4-week course of ciprofloxacin. A new diagnosis of type 2 diabetes mellitus was made on admission. A follow-up cardiac magnetic resonance (CMR) scan was suggestive of myocarditis which was unexpected given a normal Troponin T level on presentation.

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**Discussion**

We report a rare case of *S. Enteritidis* myopericarditis. Our case is notable as the patient was immunocompetent apart from newly diagnosed diabetes. This case highlights the value of CMR imaging in assessing for myocarditis and ventricular function.

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**Keywords**

Pericarditis • Myocarditis • Pericardial effusion • *Salmonella* • Case report

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**Learning points**

- Acute bacterial myopericarditis is rare, but can be associated with significant morbidity and mortality
- The role of the CMR can be important in demonstrating myocardial involvement and the presence of late gadolinium enhancement at follow-up can help risk-stratify patients

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**Introduction**

Myopericarditis is inflammation of the pericardium and myocardium, which can be due to infection, toxin-mediated or inflammatory conditions. The infective aetiology of acute pericarditis is poorly understood. However, most cases are thought to be due to viral infection, either due to direct viral cytopathic effect or by post-viral immune-mediated phenomena. Acute bacterial myopericarditis is relatively uncommon in the developed world and can be fatal if not treated aggressively. The most common acute bacterial pathogens include *Staphylococcus aureus* and streptococci including *Streptococcus pneumoniae*. Less frequent causes include *Neisseria meningitidis*, *Mycoplasma pneumoniae*, and *Campylobacter* spp. *Mycobacterium tuberculosis*, while the most common bacterial cause of myopericarditis, often presents with subacute to chronic symptoms.

Non-typhoidal salmonella (NTS) are gram-negative bacteria which generally cause self-limiting gastroenteritis, however, invasive infection...
can occur. Non-typhoidal salmonella are estimated to cause 90 300 deaths per year, and antimicrobial resistance is a growing problem. In Europe, it is the second most common foodborne infection, usually contracted by ingestion of contaminated meat, eggs, or milk.5

**Timeline**

| 6 days prior to presentation | Patient ate undercooked chicken in Lithuania. |
|----------------------------|---------------------------------------------|
| Initial presentation (Day 1) | Sepsis with chest pain and cardiac tamponade. There was widespread ST elevation and serial troponins were normal. |
| Initial management (Day 1) | Therapeutic pericardiocentesis performed. Ceftriaxone and levofoxacin started. |
| Day 2 of admission | Salmonella enterica serovar Enteritidis isolated on pericardial fluid. Ceftriaxone continued. Beta-blocker and angiotensin-converting enzyme-inhibitor initiated. |
| Day 4 of admission | Ceftriaxone changed to ciprofloxacin. Moderate transudative pleural effusion identified, which was culture negative. |
| Day 10 of admission | Discharged with high dose (750 mg BD) oral ciprofloxacin to complete 4 weeks. |
| 4 weeks after discharge | Cardiac magnetic resonance scan demonstrates features of myocarditis with ejection fraction 53% |

**Case presentation**

A 44-year-old Gambian male presented to his local emergency department with a 24-h history of generalized malaise, fever, sharp left-sided chest pain, and breathlessness. On arrival, he was pyrexial (38.4°C), with a sinus tachycardia (heart rate of 110 b.p.m.) and a blood pressure of 105/72 mmHg. On examination, his jugular venous pressure was elevated, heart sounds were normal, and there was no pericardial rub or peripheral oedema. He was previously fit and well, took no regular medication, and reported no significant family history. He did not smoke or drink alcohol. Three days prior to presentation, he had returned from a 4-day work-related trip to Lithuania. He reported having eaten undercooked chicken in Lithuania 6 days prior to presentation; he and a colleague bought the meat at a local supermarket and cooked it in the dark on a hotel barbeque.

An electrocardiogram (ECG) at presentation demonstrated widespread ST elevation (Figure 1). The Troponin T level was 11 (normal range (NR) 0–14 ng/L), however, the white cell count was elevated at 12.7 × 10^9/L (NR 4.0–11.0) with a neutrophil count of 10.0 × 10^9/L (NR 1.5–8.0) and C-reactive protein was 237 mg/L (NR <3). The lactate was 3.6 mmol/L (NR 0–2) and serum glucose was 30 mmol/L (NR 5.6–6.9). A focused ultrasound scan demonstrated a large global pericardial effusion measuring 2.5 cm at its largest point with evidence of right ventricular compromise.

The working diagnosis based on symptoms, ECG findings and clinical signs were pericarditis complicated by cardiac tamponade. His immediate management included intravenous (IV) fluids to improve preload and IV paracetamol. The cause of the pericarditis was initially assumed to be viral infection or potentially secondary to bacterial sepsis originating from another source. His left ventricular function was preserved with a negative troponin T on admission, hence myocardial involvement was not suspected during his admission.

Soon after presentation, he was transferred to a tertiary cardiac centre where immediate therapeutic pericardiocentesis was performed and he was managed in a coronary care unit facility. Repeat bedside echocardiography post pericardiocentesis showed resolution of the pericardial effusion and subsequent pericarditis treatment included regular Ibuprofen for 2 weeks. He received IV ceftriaxone and levofoxacin on advice from the Clinical Microbiologist. This regimen was chosen because S. pneumoniae was considered most likely the pathogen, and there was concern regarding the risk of penicillin-resistant strains due to his travel history.

Blood cultures on admission were negative, however, Salmonella enterica serovar Enteritidis (S. Enteritidis) was isolated in pericardial fluid. He developed loose stools shortly after admission, however, stool cultures were negative. HIV serology and an autoimmune screen were negative. Furthermore, he had persistently elevated blood glucose levels and was given a new diagnosis of type 2 diabetes.

Following the isolation of S. Enteritidis from pericardial fluid, levofoxacin was stopped, and ceftriaxone was continued. This was changed to ciprofloxacin once the isolate was found to be fully sensitive. His symptoms improved, and after 6 days his fevers resolved. This prolonged time to fever resolution was not considered clinically concerning as it is typical for patients with invasive salmonella infection to defervesce slowly despite appropriate antimicrobials. He was noted to have a moderate left-sided pleural effusion, which was thought to be reactive as pleural fluid was transudative and culture negative. He was discharged after a 10-day hospital admission on oral ciprofloxacin 750 mg twice daily and completed 4 weeks of antibiotics. The longer-term management from a cardiac perspective included a beta-blocker and angiotensin-converting enzyme (ACE)-inhibitor.

When reviewed in cardiology clinic 4 weeks after discharge he reported feeling generally well although he continued to experience occasional pericarditic chest pain. Cardiac magnetic resonance (CMR) imaging 1 month after discharge demonstrated a thickened pericardium (7.5 mm) around the basal to mid left ventricular (LV) anterior and antero-lateral wall with hyperenhancement on late gadolinium images (Figures 2 and 3). There was no residual pericardial effusion or constrictive physiology and the LV ejection fraction was mildly impaired at 53%. Prognostic heart failure medical management (beta-blocker and ACE-inhibitor) was optimized, and plans are in place for longer-term cardiology follow-up.
Discussion

We present a rare case of S. Enteritidis myopericarditis in a patient with new-onset type 2 diabetes. This is likely to have been contracted from the recent ingestion of undercooked chicken rather than more distant acquisition in Gambia because extra-intestinal manifestations of NTS tend to occur as complications of acute infection rather than reactivation of chronic carriage. We identified 10 cases of S. Enteritidis myopericarditis described in the literature, 70% of whom were immunosuppressed, predominantly due to prolonged steroid use. Of these 10 cases, 70% were complicated by tamponade, 30% by constrictive pericarditis, 40% underwent pericardectomy, and 20% died. In four out of five cases where duration of antibiotics was reported, patients received at least 4 weeks of therapy. However, we identified three previous cases of NTS pericarditis in diabetics without other immunosuppression.

Antibiotic therapeutic options for invasive salmonella infection include quinolones, cephalosporins, and macrolides. However, due to increasing rates of antimicrobial resistance, this choice should be guided by susceptibility testing. Early liaison with infection specialists is advised, particularly in the context of recent travel. There is no clear evidence to support decisions regarding the optimum duration of antibiotic therapy, however, 2–4 weeks appear reasonable, guided by clinical and radiological response.

Cardiac management is supportive, although patients may require pericardiocentesis or, at a later stage, pericardectomy if constrictive pericarditis develops. In this case, the CMR added value in demonstrating late gadolinium enhancement (LGE) which was of interest given the admission troponin level was not elevated. Although it is difficult to be certain, this discrepancy may have been due to the measured troponin sample preceding the onset of myocardial injury subsequently demonstrated on CMR. The exact significance of LGE in myocarditis is unclear; however, it typically represents the presence of necrosis or scar, depending on whether performed in the acute or chronic setting, respectively. Grün et al. demonstrated that the presence of LGE at follow-up in myocarditis patients is strongly predictive of adverse cardiovascular events and mortality. Studies have also demonstrated through serial CMR quantification of LGE in myocarditis that the degree of reduction over time correlates positively with favourable LV remodelling. According to the
position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases, CMR may be considered in clinically stable patients prior to endomyocardial biopsy (EMB). Nevertheless, it does not replace the role of the EMB in the diagnosis of myocarditis, especially in life-threatening presentations. The use of CMR as a diagnostic tool in myocarditis should be based on the Lake-Louise criteria. Hence, CMR may play a valuable role in the risk stratification of myocarditis patients and, as in the case of this patient, lead to prolonged medical therapy (ACE-inhibitor and beta-blocker) and extended follow-up.

Conflict of interest: none declared.

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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 author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Figure 3 (A) Four-chamber imaging slices showing thickened pericardium (arrows) around anterior and lateral wall of the left ventricle. (B) Short-axis view showing thickened pericardium (arrows) around anterior and lateral wall of the left ventricle. (C) Short-axis slice at ventricular level with late gadolinium imaging showing sub-epicardial and mid-wall enhancement of the infero-lateral wall (blue arrow). In addition, there is enhancement of the pericardium around anterior and lateral wall (orange arrow).
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