Case report of *Campylobacter jejuni*-associated myopericarditis: rare case of cardiac involvement by a common gastroenteritis pathogen

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Background	Myocarditis is caused by inflammation affecting the heart muscle. The usual aetiological factor is viral, especially in immunocompetent hosts and developed countries. *Campylobacter jejuni* is a common cause of bacterial gastroenteritis but has rarely been associated with myocarditis.
Case summary	We report a case of a 22-year-old male admitted with pleuritic chest pain and a diarrhoeal illness. Thorough evaluation of the patient history did not reveal any sources for contracting the diarrhoea. Stool cultures confirmed that the patient had *C. jejuni* infection as well as myopericarditis confirmed on cardiac magnetic resonance imaging (cardiac MRI). Treatment with colchicine 0.5 mg BD, ibuprofen 600 mg TDS, and ciprofloxacin 500 mg BD orally for 5 days was started, together with an intravenous infusion of 0.9% normal saline 1 L TDS. The patient showed signs of improvement over a span of three days and the ST changes on electrocardiogram resolved.
Discussion	Although *C. jejuni*-associated myopericarditis is uncommon, it can be potentially life-threatening if not considered in the differential. Its diagnosis involves good history taking, examination, and investigation with electrocardiography, troponins and inflammatory markers, echocardiography, and cardiac MRI. Several mechanisms of infection have been suggested, including direct insult by toxin or bacterium as well as an immune-mediated response. Both supportive and causative treatments are important to ensure recovery while reducing the risk of complications. It is therefore crucial to ensure that the patient receives adequate follow-up to ascertain patient progress and to mitigate any complications that may arise as well as tackle patient concerns. The patient had a rapid recovery.
Keywords	Case report • *Campylobacter jejuni* • Gastroenteritis • Myopericarditis • Magnetic resonance imaging
ESC Curriculum	2.3 Cardiac magnetic resonance • 6.6 Pericardial disease • 2.2 Echocardiography

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Learning points

- As highlighted by this case, even though *Campylobacter jejuni* myopericarditis is not common, it should still be taken into consideration when faced with symptoms of myopericarditis together with diarrhoea.
- The diagnosis of myopericarditis involves good history taking, examination, and appropriate investigation with electrocardiography, troponins and inflammatory markers, echocardiography, and cardiac magnetic resonance imaging (MRI).
- Cardiac MRI can highlight pericardial and/or myocardial involvement as well as the presence of myocardial injury/fibrosis.

Introduction

Myocarditis is caused by inflammation affecting the heart muscle. It is an uncommon condition that can occur as a complication of several infections. However, it is rarely associated with bacterial infections. The usual aetiological factor is viral especially in immunocompetent hosts and in developed countries. Viruses implicated in myocarditis include the Coxsackie virus, Adenovirus, and HIV among others.[1] *Campylobacter jejuni* is a common cause of bacterial gastroenteritis but has rarely been associated with myocarditis.[2]

Timeline

Day 0	Previously healthy 22-year-old gentleman presented to GP with abdominal discomfort, nausea/vomiting together with bouts of diarrhoea.
Day 2	Patient started feeling central compressive chest pain worse on taking deep breaths.
Day 4	Patient was admitted after presenting at casualty with worsening symptoms. Electrocardiogram (ECG) showed significant ST changes associated with elevated cardiac markers.
Day 5	Stool cultures came back positive for *Campylobacter jejuni* and patient was started on oral Ciprofloxacin.
Day 6	Cardiac magnetic resonance imaging confirming myopericarditis
Day 7	Resolution of symptoms and ECG changes together with downtrending cardiac markers. Patient discharged home on colchicine, ibuprofen, and ciprofloxacin.
6-week follow-up	Follow-up after 6 weeks showed no sequalae after this episode. Patient returned to his normal life without any symptoms.

Case presentation

A 22-year-old previously healthy, coronavirus disease-19 (COVID-19) unvaccinated male with no significant cardiac risk factors presented to the A&E Department with a 2-day history of central pleuritic chest pain. The patient denied shortness of breath or syncope but claimed that the pain worsened on taking a deep breath in and improved on sitting up. Systems review revealed a 4-day history of non-bloody loose stools, abdominal discomfort, nausea, and an episode of emesis. The patient denied fever, unwell contacts, or recreational drug use and was not on regular medications. The patient denied any recent change in diet or consumption of food which may precipitate a diarrhoeal illness.

On examination, parameters were stable with a temperature of 37°C, blood pressure of 161/72 mmHg, pulse of 100 beats/min, respiratory rate of 18, and saturations of 100% on room air. Cardiovascular examination revealed normal heart sounds with a three-component pericardial rub at the left sternal edge. His chest was clear on auscultation and the abdomen was soft and non-tender.

Electrocardiogram (ECG) showed ST elevation in leads 1 and aVL, ST depressions in leads II, III and aVF, and T-wave inversions in leads II, III, aVF and V5–V6 (*Figure 1*).

Laboratory investigations showed normal serum electrolytes and blood cell counts but revealed a high C-reactive protein (CRP) of 151 mg/L (0–5 mg/L). Initial troponin-T level was 27 ng/L (3–14 ng/L) with repeat value of 19 ng/L. A viral screen was carried out including Cytomegalovirus IgM and IgG, Epstein–Barr Virus IgM and IgG, Parvovirus IgM and IgG, and Human Immunodeficiency Virus antigen and antibody which were all negative. A routine COVID-19 polymerase chain reaction (PCR) swab was taken on admission which was negative.

Echocardiography showed normal biventricular function, without valvular disease and no pericardial effusion.

Cardiac magnetic resonance imaging (MRI) (*Figures 2–4*) showed pericardial enhancement over basal lateral left ventricular (LV) wall, small pericardial effusion, and global myocardial oedema findings of which were in keeping with ongoing mild acute myopericarditis. It also confirmed a non-dilated left ventricle with preserved systolic function.

Stool cultures and PCR were taken for microscopy, culture, and sensitivity with *C. jejuni* cultured from the stool samples. No growth was detected on blood cultures.

The patient was admitted on monitor for observation and was started on Colchicine 0.5 mg BD and Ibuprofen 600 mg TDS. The patient was also given Ciprofloxacin 500 mg BD orally for 5 days, with an intravenous infusion of 0.9% normal saline 1 L TDS, which was eventually stopped once the patient was tolerating oral intake.

The diagnosis was *C. jejuni*-associated myopericarditis. The patient showed signs of improvement over a span of three days and ST changes on ECG resolved in leads 1, aVL and inferiorly with residual T-wave inversions laterally (V4–V6) (*Figure 5*). On discharge, troponin and CRP levels were 9 ng/L and 22 mg/L, respectively showing considerable clinical improvement.

The patient was visiting the country when he was admitted and was discharged back home asymptomatic. Repeat ECG and echocardiogram 6 weeks later back in his home country were reportedly normal.

Discussion

The commonest aetiological factor in myopericarditis is viral especially in the developed world and in those who are not immunocompromised. Such viruses include the Coxsackie viruses, Adenovirus, HIV, Enteroviruses, and Hepatitis B and C among others.[1] Bacteria such as Salmonella, Shigella, Staphylococci, Streptococci, and Legionella are mentioned in several case reports.[3] *Campylobacter jejuni* is a common cause of bacterial gastroenteritis occurring after consumption of poorly cooked food, but it has rarely been associated with myocarditis.[2]

Although myopericarditis in the context of *C. jejuni* infection is rare, it can be potentially life-threatening. Therefore, thorough assessment utilizing clinical examination, laboratory investigations, and imaging modalities are crucial to obtain the diagnosis. Such a diagnosis
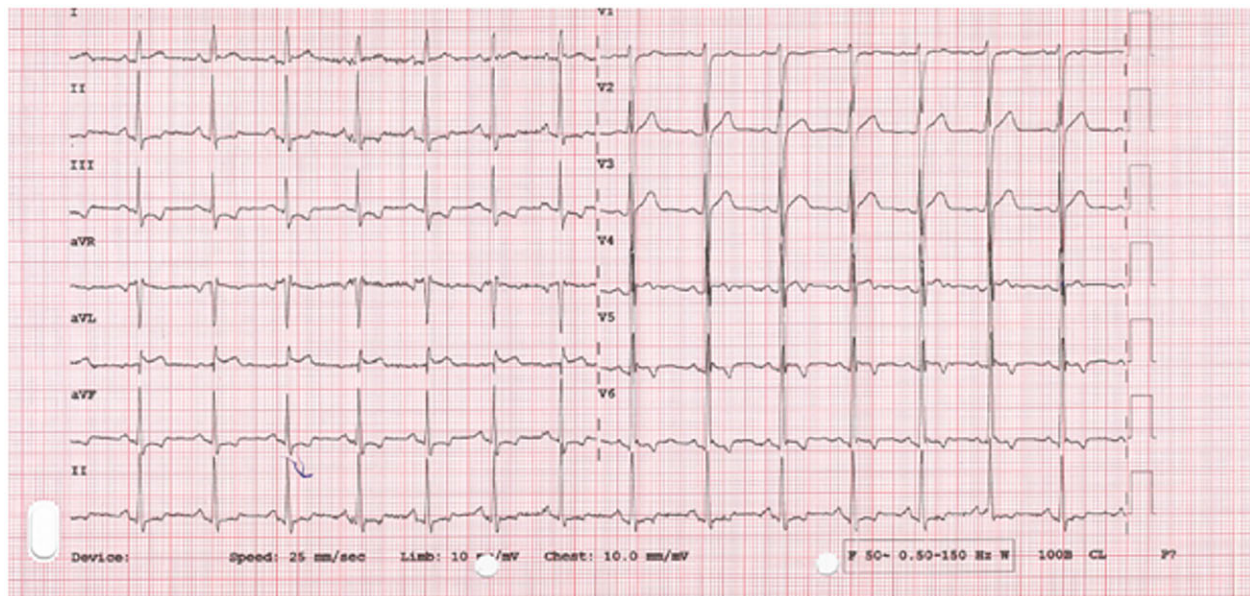
Figure 1 Electrocardiogram on admission showing ST-segment elevation in leads 1 and aVL, ST-segment depressions in leads II, III and aVF, and T-wave inversions in leads II, III, avF and V5–V6.
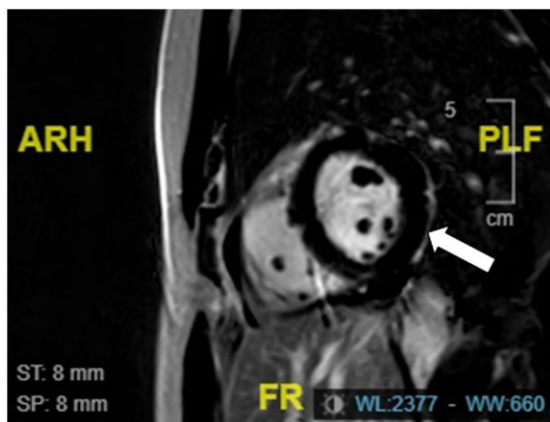

Figure 2 Cardiac magnetic resonance imaging showing pericardial late gadolinium enhancement (white arrow) signifying pericardial inflammation over basal lateral left ventricular wall.
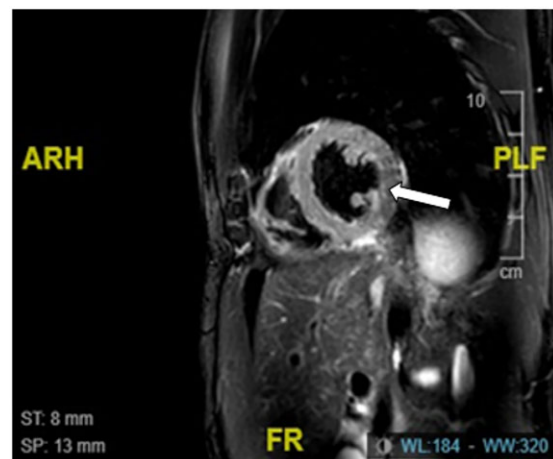

Figure 3 Short tau inversion recovery images showing global oedema by black blood oedema (white arrow).

should be considered in a patient who presents with symptoms of chest pain with no background coronary artery disease accompanied by a concurrent or preceding history of diarrhoeal illness with detection of the bacterium in the stool.[4]

Young men are more likely to be affected by campylobacter-associated myopericarditis[5] in keeping with the male preponderance for cardiovascular disease.[6] The precise pathological process for this is not known but speculation on the role of hormones, genetics, and the immune system together with the degree of manifestation of the disease and response to treatment has been made.[7]

Troponin and inflammatory markers are often elevated and can be utilized as markers of improvement and prognosis. Echocardiography is essential to elucidate any pericardial effusions or myocardial dysfunction. Although gold standard for definitive diagnosis is endocardial myocardial biopsy, cardiac MRI is useful because it further supports the diagnosis of probable myopericarditis, a condition with

no pathognomonic signs or symptoms.[8] Since myopericarditis was highly probable, endocardial myocardial biopsy was not considered, as risks outweighed the benefits.[2]

Cardiac MRI confirmed global oedema by black blood technique on Short tau inversion recovery images. Myocardial oedema was assessed based on the signal intensity ratio of the myocardium over skeletal muscle, which was 2.3–2.8 with values more than 2.0 being
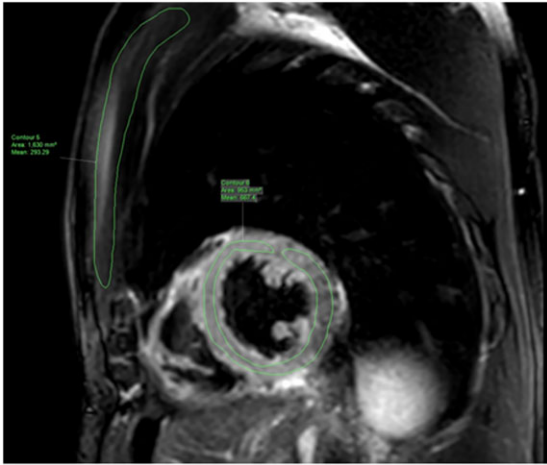

Figure 4 Myocardial oedema assessment was based on signal intensity ratio of myocardium over skeletal muscle (green areas highlighted). The signal intensity ration is 2.3–2.8 with values more than 2 being significant.

significant. It also showed pericardial late gadolinium enhancement (LGE) signifying pericardial inflammation over basal lateral LV wall associated with a localized mild (6 mm) pericardial effusion over the posterior aspect of the heart. Despite that there was no subepicardial myocardial fibrosis on LGE, one main criterion—myocardial oedema and one supportive criterion—pericarditis, were found thus establishing the diagnosis of myopericarditis utilizing the updated 2018 Lake Louise Criteria.[9]

The exact pathophysiological mechanism of *C. jejuni*-associated myopericarditis is not fully understood. However, the short time frame between the onset of gastroenteritis and the onset of cardiac symptoms may indicate that the likely mechanism involves direct myocardial insult by a toxin or a direct effect by the bacterium as opposed to an immunological aetiology.[4] This contrasts with the immune-mediated response which is seen in conditions such as Guillain-Barre syndrome where *C. jejuni* causes neurological symptoms after diarrhoeal illness.[3] Although blood cultures were negative, this does not exclude the possibility of a transient bacteraemia or the effect of an invasive toxin. Another mechanism involving circulating immune complexes or cytotoxic T-lymphocytes has also been postulated which may cause a more delayed form of the symptomatology.[10] Viral co-infection was excluded since Cytomegalovirus IgM and IgG, Epstein–Barr Virus IgM and IgG, Parvovirus IgM and IgG, and Human Immunodeficiency Virus antigen and antibody were all negative.

Treatment usually involves supportive therapy with causal treatment including macrolide or fluoroquinolone antibiotics.[5] Supportive treatment plays an important role as it not only prevents dehydration but also reduces the risk of electrolyte imbalances which can lead to further complications such as cardiac arrhythmias. Full recovery is
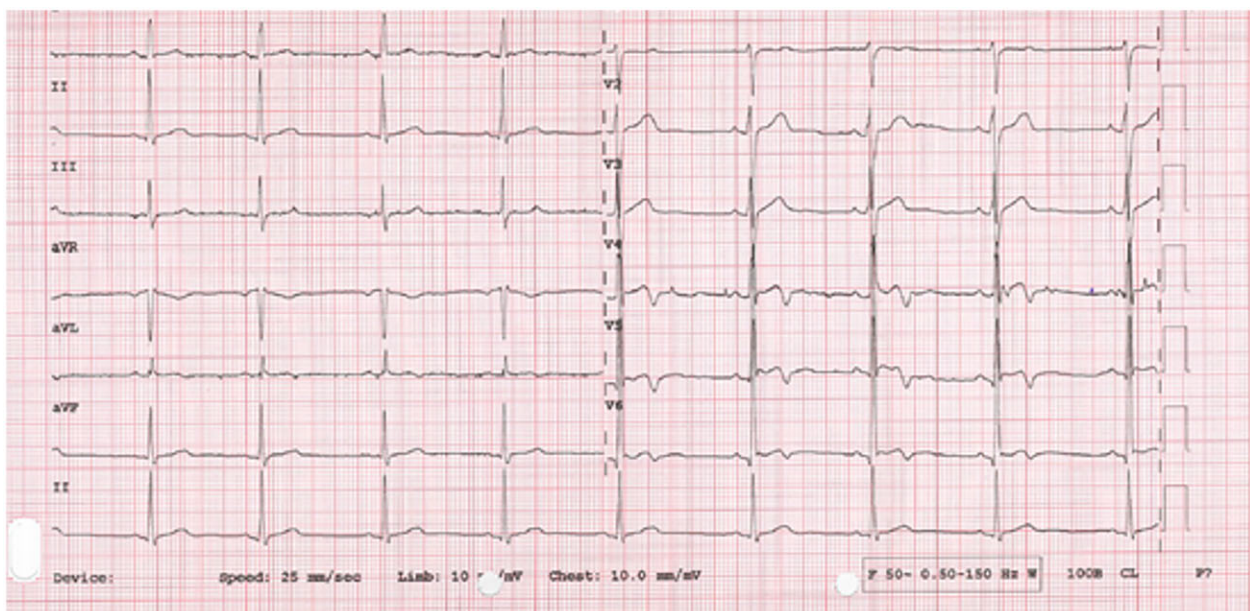

Figure 5 Electrocardiogram on discharge showing ST-segment changes resolved in leads 1, aVL and inferiorly in leads II, III and aVF with residual T-wave inversions laterally (V4–V6).

expected in most patients although certain complications such as cardiac failure[11] or arrhythmias[12] can persist if there is significant myocardial necrosis and scarring. Although the myopericarditis may be severe,[12] in this particular patient the symptoms were mild and the patient had a rapid recovery.

Lead author biography

Lara Miruzzi is a first year Basic Specialist Trainee in General/Internal Medicine at Mater Dei Hospital, Malta. She received her Doctor of Medicine and Surgery Degree from the University of Malta in 2019 and is currently furthering her training and studies to achieve Membership of the Royal College of Physicians. She has an interest in a variety of medical fields including Cardiology, Gastroenterology, Endocrinology, and Neurology. During her Foundation Training, she participated in several clinical audits and case reports as she believes that these are an important supplement to clinical practice.

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.

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