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When enteritis reaches the heart: acute *Campylobacter jejuni*–associated perimyocarditis in a young adult

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SUMMARY

Campylobacter jejuni is a common cause of bacterial gastroenteritis, while cardiac involvement is a rare extraintestinal complication. We report the case of a 21-year-old immunocompetent man who developed acute perimyocarditis three days after microbiologically confirmed *C. jejuni* enteritis. He presented with pleuritic chest pain, elevated inflammatory markers, and increased high-sensitivity troponin I levels. Echocardiography showed preserved ventricular function, whereas cardiac magnetic resonance imaging (CMR) demonstrated myocardial edema and subepicardial-to-mid-myocardial late gadolinium enhancement, fulfilling the Revised Lake Louise Criteria for acute myocarditis. The patient had an uncomplicated clinical course and was discharged in stable condition. This case highlights the importance of considering perimyocarditis in young patients presenting with chest pain following recent gastroenteritis and underscores the diagnostic value of CMR.

Keywords: Campylobacter jejuni; perimyocarditis; cardiac magnetic resonance imaging; extraintestinal complications; gastroenteritis.

INTRODUCTION

Campylobacter jejuni is one of the most common bacterial causes of acute gastroenteritis worldwide, typically acquired through the consumption of contaminated food or water [1, 2]. Although the infection is usually self-limiting, a variety of extraintestinal manifestations have been described, including reactive arthritis, Guillain–Barré syndrome, bacteremia, and, less frequently, cardiac involvement [3-8].

Acute myocarditis and perimyocarditis associated with *C. jejuni* infection are uncommon but potentially serious complications. Most reported cases have occurred in young, otherwise healthy individuals and typically develop within days of the onset of gastrointestinal symptoms [9-14]. Because the clinical presentation may mimic other causes of acute chest pain, early recognition is essential to ensure appropriate diagnostic evaluation and management. We report the case of a 21-year-old immunocompetent male who developed acute perimyocarditis following a recent *C. jejuni* infection.

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PATIENT HISTORY

The study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent for publication of clinical data was obtained from the patient.

The patient initially presented to the emergency department (ED) with a five-day history of abdominal pain and diarrhea, associated with fever, dehydration and mild hypokalemia. Laboratory investigations showed an elevated C-reactive pro-

tein (CRP) level of 21.79 mg/dL (reference limit <0.5 mg/dL), and stool samples were collected for microbiological investigation. Intravenous rehydration and empirical ceftriaxone were administered, and the patient was discharged after clinical improvement on empirical oral cefixime (400 mg once daily for five days).

Three days later, the patient returned to the ED complaining of acute pleuritic chest pain radiating to the left arm, which worsened with positional changes. Diarrhea had resolved, although stools remained poorly formed, and he was afebrile and hemodynamically stable.

Past medical history was unremarkable, with no previous cardiovascular disease or surgical interventions. No drug allergies were reported. Family history was notable for premature coronary artery disease, as the patient's father had suffered a myocardial infarction at the age of 52 years.

Electrocardiogram (ECG) showed sinus rhythm at 71 bpm and was otherwise within normal limits. Chest X-ray was unremarkable.

Laboratory tests demonstrated significant inflammation and myocardial injury, with a CRP level of 3.28 mg/dL and markedly elevated high-sensitivity troponin I levels (>1000 ng/L on point-of-care testing; 99th percentile upper reference limit: 25.7 ng/L) subsequently confirmed at 7402.4 ng/L by central laboratory analysis (99th percentile upper reference limit: 19.8 ng/L for males). Renal function, complete blood count and serum electrolytes were within reference ranges.

Review of the microbiological investigations requested during the patient's previous ED visit for acute gastroenteritis revealed growth of *Campylobacter jejuni* in stool culture after 48 hours of incubation. Stool cultures were otherwise negative for *Salmonella* spp. and *Shigella* spp., while stool antigen assays for enteric adenovirus and *C. jejuni* and serological tests for *Brucella* spp. and *Salmonella typhi* were negative. In view of the improvement of the gastrointestinal illness, no targeted antimicrobial therapy against *C. jejuni* was initiated after microbiological identification. The patient was subsequently admitted to the coronary care unit (CCU) for further evaluation and monitoring because of the clinical presentation and laboratory findings.

Upon admission to the CCU, the patient was alert, oriented, and cooperative. He was without ongoing chest pain and dyspnea at rest and was breathing comfortably on room air. Physical examination revealed normal vesicular breath sounds bilaterally, without crackles or other adventitious sounds. No peripheral edema was observed.

The ECG showed sinus rhythm at 71 bpm, with a normal PR interval and nonspecific repolarization abnormalities.

Transthoracic echocardiography performed on the same day demonstrated a non-dilated left ventricle (end-diastolic diameter 47 mm; end-diastolic volume 102 mL) with preserved global systolic function (LVEF 55%) and circumscribed hypokinesia of the mid-inferior wall. Global longitudinal strain was normal (-21%). Mild mitral regurgitation was present. The left atrium was of normal size (indexed left atrial volume 23 mL/m^2). The aortic root was not dilated (25 mm). Diastolic function was normal, with an E/e' ratio of 5. Right heart chambers were within normal limits, with preserved right ventricular systolic function (TAPSE 26 mm). Mild tricuspid regurgitation was noted, with an estimated systolic pulmonary artery pressure of 25 mmHg. No pericardial effusion was detected.

During CCU admission, continuous cardiac monitoring revealed no arrhythmias or hemodynamic instability. Ramipril (1.25 mg twice daily) was initially administered during the early phase of cardiological management. Serial troponin measurements showed a progressive decline, and after three days, following clinical stabilization, the patient was transferred to the cardiology ward, where cardiac magnetic resonance (CMR) imaging was performed. Following completion of the diagnostic work-up and confirmation of preserved left ventricular systolic function, ramipril was progressively reduced and discontinued before discharge.

CMR revealed normal left ventricular size and preserved biventricular systolic function (LVEF 56%; RVEF 54%), with mild hypokinesia of the basal inferolateral wall. T2-weighted sequences demonstrated myocardial edema in the basal inferolateral wall, with increased native T1 and T2 values and corresponding subepicardial-to-mid-myocardial late gadolinium enhancement. These findings fulfilled the Revised Lake Louise Criteria [15]. A small circumferential pericardial effusion, not detected on the initial echocardiography, was also present, without pathological pericardial thickening or contrast enhancement. Representative CMR images are shown in *Figure 1*.

The remainder of the hospital course was une-

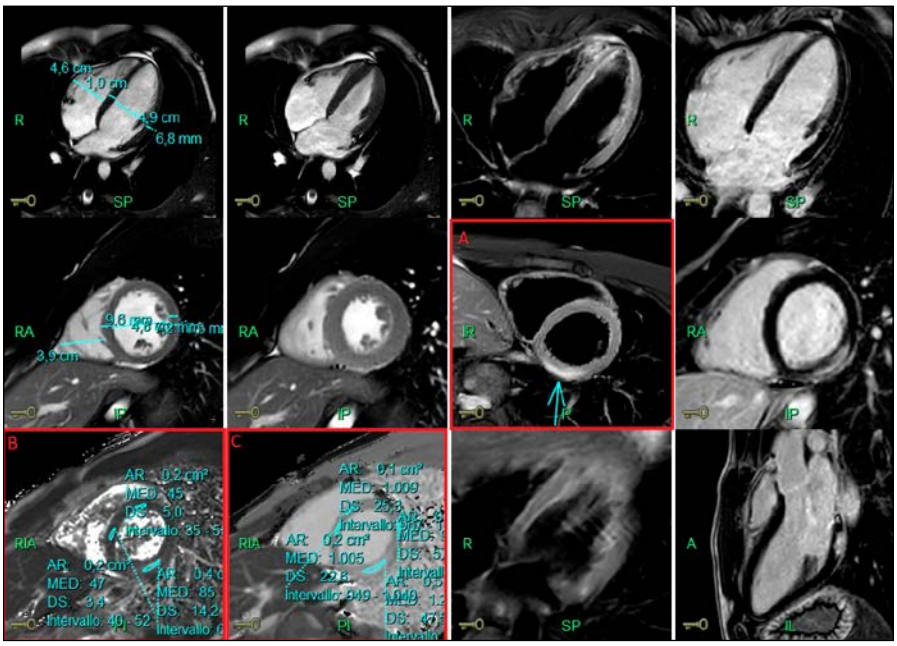

Figure 1
Representative cardiac magnetic resonance images obtained during the acute phase. The red-highlighted panels show the main tissue-characterization findings: (A) short-axis late gadolinium enhancement image demonstrating focal subepicardial-to-mid-myocardial enhancement of the basal inferolateral wall (arrow); (B) T2 mapping showing an increased T2 relaxation time in the same region (85 ms; reference value 48 ± 3 ms), consistent with myocardial edema; and (C) native T1 mapping showing an increased T1 relaxation time in the basal inferolateral wall (1200 ms; reference value 983 ± 26 ms).

Table 1 - Clinical course and serial laboratory findings. Timeline of the main clinical events and serial high-sensitivity troponin I (hs-TnI) and C-reactive protein (CRP) values during the acute phase and follow-up. Laboratory values shown were obtained by central laboratory analysis. The 99th percentile upper reference limit for hs-TnI was 19.8 ng/L in males, and the CRP reference limit was <0.5 mg/dL. CCU, coronary care unit; CMR, cardiac magnetic resonance; CRP, C-reactive protein; ED, Emergency Department; hs-TnI, high-sensitivity troponin I.

Time point	*Clinical course and main events*	*hs-TnI (ng/L)*	*CRP (mg/dL)*
Day 1	First ED presentation for febrile gastroenteritis, dehydration, and persistent diarrhea for 5 days. Stool microbiological investigations were performed. Intravenous rehydration and empirical ceftriaxone were administered. The patient was discharged after clinical improvement on oral cefixime.	–	*21.79*
Day 4	Second ED presentation for recurrent prolonged pleuritic and positional chest pain radiating to the left arm over the previous 2 days, with a third episode occurring during ED evaluation. The patient was afebrile and hemodynamically stable. ECG was within normal limits. Cardiology assessment and echocardiography raised suspicion of myocarditis/pericardial involvement, and the patient was transferred to the CCU. Diarrhea was no longer present, although stools remained poorly formed.	*7402.4*	*3.28*
Day 5	Clinically stable and asymptomatic; serial ECG showed no significant abnormalities. Cardiac and inflammatory biomarkers progressively declined.	*3389.3*	*1.99*
Day 6	Continued clinical stability with further decline in hs-TnI.	*790.9*	–
Day 7	Asymptomatic, with further decline in cardiac and inflammatory biomarkers.	*235.7*	*0.75*
Day 10	CMR findings consistent with acute myocarditis, with preserved biventricular systolic function and a small circumferential pericardial effusion. The patient was discharged in stable clinical condition.	–	–
~4-month follow-up	Clinically asymptomatic, with normalization of cardiac and inflammatory biomarkers.	*<2.3*	*0.31*
~12-month follow-up	Clinically asymptomatic, with hs-TnI within the reference limit.	*2.8*	*0.66*

ventful, with no recurrence of chest pain, no signs or symptoms of heart failure, and no arrhythmias detected on continuous telemetry monitoring.

At discharge, the patient was free of chest pain and dyspnea at rest, afebrile, eupneic on room air, and hemodynamically stable, with a blood pressure of 110/60 mmHg. Physical examination revealed normal vesicular breath sounds bilaterally, without crackles. Cardiac auscultation showed a regular rhythm with normal heart sounds and a grade 2/6 systolic murmur. No peripheral edema was present. He was advised to refrain from strenuous physical activity for 6 months and was scheduled for outpatient cardiology follow-up. At approximately 4 months, the patient remained clinically asymptomatic, with normalization of hs-TnI (<2.3 ng/L) and CRP (0.31 mg/dL). At approximately 12 months, he remained asymptomatic and hs-TnI was still within the reference limit (2.8 ng/L). *Table 1* summarizes the clinical course and serial laboratory findings.

DISCUSSION

Cardiac involvement is an uncommon extraintestinal manifestation of *C. jejuni* infection. Although *Campylobacter* spp. are among the leading causes of bacterial gastroenteritis worldwide, myocarditis and perimyocarditis have been only occasionally reported. Available evidence suggests that affected patients are predominantly young, otherwise healthy males who develop cardiac symptoms within a few days of the onset of gastrointestinal illness. Clinical presentation is typically characterized by acute chest pain, elevated inflammatory markers, and increased cardiac troponin levels [9-14].

The pathophysiological mechanisms underlying *Campylobacter*-associated perimyocarditis remain incompletely understood [16]. Because bacteremia occurs in fewer than 1% of *Campylobacter* infections, direct myocardial invasion is considered unlikely in most cases. Current evidence favors an immune-mediated process, potentially driven by molecular mimicry and systemic inflammatory activation, similar to the mechanisms implicated in other post-infectious complications of *Campylobacter* infection [17]. The close temporal relationship between enteritis and cardiac involvement observed in our patient is consistent with this hypothesis.

In young patients presenting with chest pain and elevated cardiac biomarkers, the differential diagnosis includes acute coronary syndrome, viral myocarditis, Takotsubo cardiomyopathy, and isolated acute pericarditis [3, 15, 18-19]. In our case, an ischemic etiology was considered unlikely because electrocardiography showed no ischemic changes, and CMR demonstrated no resting myocardial perfusion defects and a non-ischemic pattern of myocardial injury, characterized by subepicardial-to-mid-myocardial involvement of the basal inferolateral wall [15]. Echocardiography showed preserved global left ventricular systolic function despite limited regional hypokinesia. Takotsubo cardiomyopathy was considered unlikely because the pattern of regional wall motion abnormalities was not typical and CMR demonstrated focal inflammatory myocardial tissue abnormalities. Although viral myocarditis could not be completely excluded, the close temporal association with microbiologically confirmed *C. jejuni* enteritis, together with the absence of a more likely alternative etiology, was considered suggestive of *Campylobacter*-associated perimyocarditis.

CMR played a pivotal role in the diagnostic work-up. As the reference non-invasive imaging modality for suspected myocarditis, it is recommended by the latest ESC Guidelines on Myocarditis and Pericarditis for myocardial tissue characterization and enables the detection of myocardial edema and non-ischemic patterns of myocardial injury [18]. In our patient, the presence of myocardial edema and subepicardial-to-mid-myocardial late gadolinium enhancement fulfilled the Revised Lake Louise Criteria for acute myocarditis, supporting the diagnosis [15]. Similar imaging findings have been reported in previous cases of *Campylobacter*-associated myocarditis and perimyocarditis, comprehensively reviewed by Hessulf et al. and more recently updated by Zouganeli et al. [9-10]. These reports describe a remarkably consistent clinical presentation, with young, otherwise healthy patients developing acute chest pain within a few days of *Campylobacter* enteritis, associated with elevated cardiac biomarkers and non-ischemic CMR findings. Our findings are in keeping with this previously described clinical pattern.

CONCLUSIONS

C. jejuni–associated perimyocarditis is an uncommon but clinically relevant complication of bacterial gastroenteritis. Cardiac involvement should be considered in patients presenting with chest pain shortly after enteric infection, particularly when *Campylobacter* infection has been microbiologically confirmed. Early recognition and timely CMR evaluation are essential for establishing the diagnosis, excluding alternative etiologies, and guiding appropriate management.

Conflicts of interest
The authors declare no conflict of interest.

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Nothing to declare.

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