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Acute Myopericarditis Associated With Campylobacter Gastroenteritis in a Young Immunocompetent Patient

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Abstract

Campylobacter infections are among the most common causes of acute foodborne gastroenteritis. Extraintestinal manifestations, especially cardiac involvement, are considered rare. We present a case of a young immunocompetent female with foodborne acute gastroenteritis who presented with intense pleuritic chest pain that worsened in the supine position, along with diarrhea and vomiting for three days. Laboratory testing revealed elevated hs-troponin levels and high inflammatory markers, while the electrocardiogram showed changes indicative of pericardial inflammation. Cardiac magnetic resonance imaging with intravenous contrast demonstrated increased signal in the pericardium and the epicardial layers of the myocardium in the lateral wall of the left ventricle, findings consistent with myopericarditis. A molecular stool test for pathogen detection identified *Campylobacter* species. Treatment with azithromycin and non-steroidal anti-inflammatory drugs was initiated. The coexistence of chest pain and acute gastroenteritis should raise suspicion for underlying myopericardial inflammation due to enteric pathogens.

Categories: Cardiology, Internal Medicine, Infectious Disease
Keywords: acute myopericarditis, acute pericarditis, bacterial gastroenteritis, campylobacter enteritis myocarditis, cardiac magnetic resonance (cmr)

Introduction

Campylobacter species are among the most common bacterial causes of foodborne gastroenteritis worldwide and are responsible for millions of infections each year [1]. Transmission is usually through contaminated food or water, or direct contact with infected animals. Initial symptoms include diarrhea, abdominal pain, nausea, vomiting, and fever, with the disease being predominantly self-limiting, especially in immunocompetent individuals [2]. Although the majority of infections remain confined to the gastrointestinal tract, the substantial global burden of campylobacteriosis has prompted increasing interest in its extraintestinal manifestations [1].

While cardiac involvement due to *Campylobacter* infection is very uncommon, it can occur as myocarditis, pericarditis, or myopericarditis [3]. These manifestations typically occur a few days after the initiation of the gastrointestinal symptoms and can be accompanied by sudden chest pains, electrocardiographic changes, and cardiac marker elevation, followed by cardiac magnetic resonance (CMR) signs [4]. Even though the mechanism of the disease is unclear, bacterial cytotoxicity, toxin effects, and immunological mechanisms have all been mentioned. The information available now only comprises case reports and series of cases [5,6].

We report a case of a young immunocompetent female who developed acute myopericarditis in the setting of *Campylobacter*-associated gastroenteritis. This case aims to raise awareness of *Campylobacter*-related cardiac complications and to emphasize that myopericardial inflammation should be considered in patients presenting with acute chest pain in the context of recent or ongoing diarrheal illness.

Case Presentation

A 24-year-old Caucasian female presented to the emergency department with acute-onset pleuritic chest pain that initiated a few hours prior to admission. She reported episodes of vomiting and diarrhea in the past three days. Her past medical history consisted of bronchial asthma, allergic rhinitis, gastroesophageal reflux disease, chronic idiopathic urticaria, and dysmenorrhea. Long-term medications included budesonide-formoterol inhaler, levalbuterol, levocetirizine, and montelukast.

Upon admission to the ED, her blood pressure was 110/70 mmHg, and her heart rate was 72 bpm, with no signs of fever (temperature: 36.3°C; Table *1*). Physical examination revealed normal cardiac sounds with no murmurs or friction rub. The respiratory and abdominal examinations were also unremarkable. She reported, however, pleuritic chest pain that worsened in the supine position. Initial laboratory tests revealed elevated

troponin levels (163 pg/mL) and elevated inflammatory markers, including white blood cell count (11.04 x 10^3/μL) and C-reactive protein (4.48 μg/dL; Table 2).

Parameter	Patient value	Normal reference range	Unit
Blood pressure	110/70	90/60–140/90	mmHg
Heart rate	72	60–100	beats/min
Temperature	36.3	36.5–37.5	°C
SpO_2	98	95–99	%

TABLE 1: Vital signs on admission.

SpO_2: peripheral oxygen saturation.

Parameter	Day 1	Day 3	Day 7	Normal reference range	Unit
Troponin	163	126	46	<45	pg/mL
WBC	11.04	7.69	6.32	4.5–10.2	$\times 10^3/\mu L$
CRP	4.48	1.8	0.36	<0.5	mg/dL

TABLE 2: Laboratory findings.

WBC: white blood cell count; CRP: C-reactive protein.

ECG showed diffuse ST-segment elevation, especially in the V4-V6, I, and II leads, and an early repolarization pattern (Figure 1). Autoimmune screening (antinuclear antibodies and rheumatoid factor) and blood cultures for common bacteria were negative. Specific viral serology, including enteric cytopathic human orphan (ECHO) and Coxsackie viruses, was unremarkable. The patient did not report any recent travel abroad or any suspicious contacts that would necessitate further investigations to rule out other infectious causes. A molecular stool test for pathogen detection (film array) was performed, which identified bacteria of the *Campylobacter* genus. No *Campylobacter* species culture was requested, as it is not consistent with regular practice when high-sensitivity molecular tests (PCR) confirm the diagnosis. Echocardiogram did not show any regional wall-motion abnormalities. Due to high clinical suspicion, we decided to further assess potential cardiac involvement using CMR on the third day of her admission. We performed CMR with intravenous administration of paramagnetic contrast, which revealed signal intensity (T2-weighted spectral attenuated inversion recovery sequence) in the pericardium and the epicardial layers of the myocardium at the lateral wall of the left ventricle, findings consistent with inflammation of the myopericardium (Figures 2, 3). The clinical presentation, elevated troponin levels, ECG abnormalities, and CMR findings supported the diagnosis of acute myopericarditis, possibly associated with *Campylobacter* gastroenteritis.

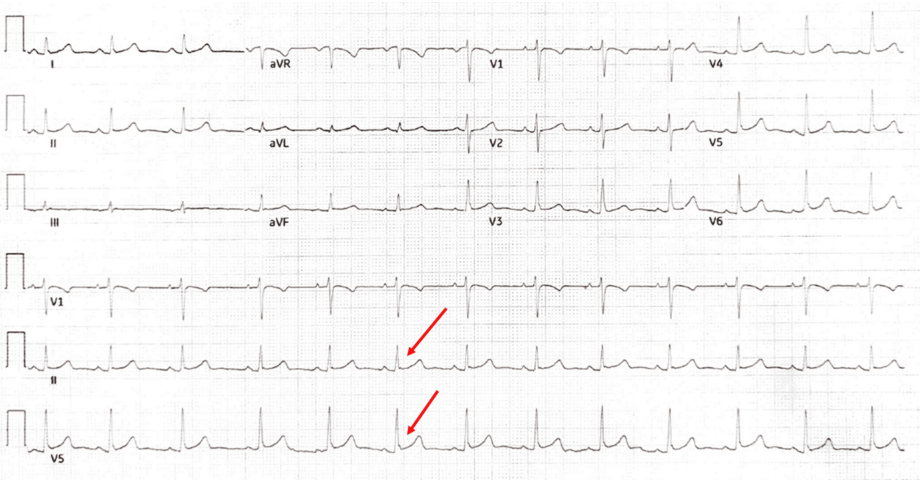


FIGURE 1: Electrocardiographic findings on admission.

Twelve-lead electrocardiogram demonstrating diffuse concave ST-segment elevation (red arrows), most prominent in the lateral leads.

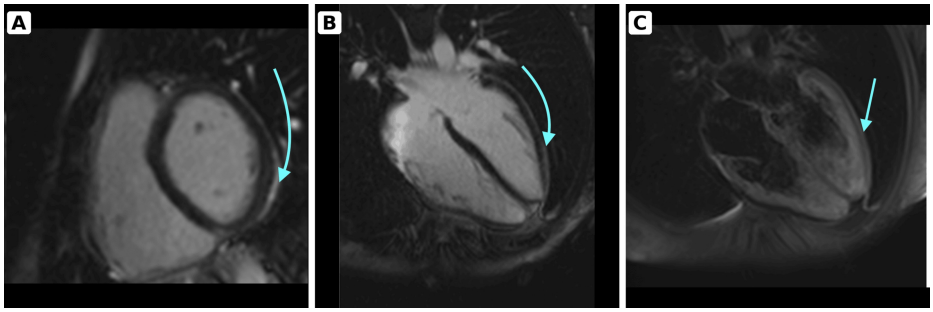


FIGURE 2: Cardiac magnetic resonance findings consistent with acute myopericardial inflammation.

(A) Mid-ventricular short-axis late gadolinium enhancement (LGE) image demonstrating high signal intensity of the lateral wall, indicative of inflammatory damage (arrow). (B) Four-chamber LGE image demonstrating high signal intensity of the epicardial layers of the anterolateral wall, indicative of inflammatory damage (arrow). (C) Four-chamber T2-weighted spectral attenuated inversion recovery (T2W-SPAIR) image showing increased signal intensity within the epicardial layers of the anterolateral wall (arrow), consistent with myocardial edema.

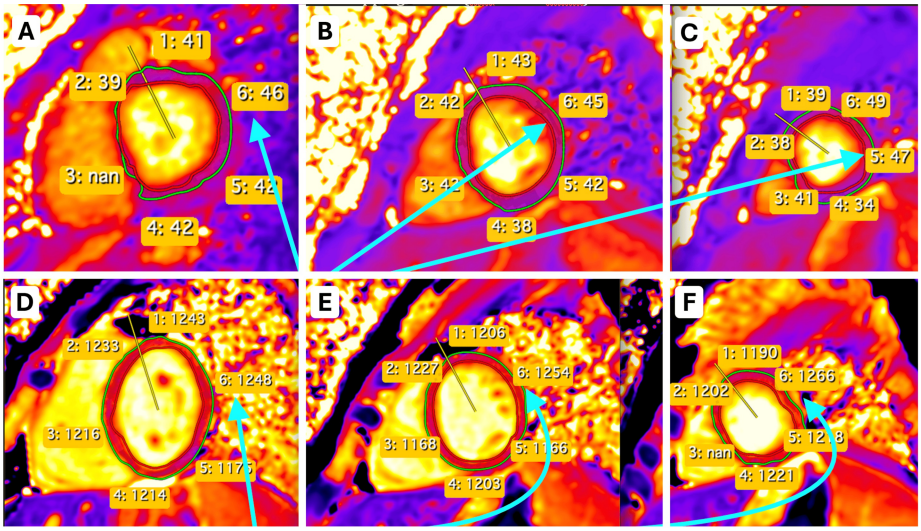


FIGURE 3: Native T2 and T1 mapping demonstrating abnormal myocardial tissue characterization.

(A–C) Native T2 maps obtained at the basal, mid-ventricular, and apical levels, respectively, demonstrating elevated T2 values within the anterolateral left ventricular wall (arrows), consistent with active inflammation. (D–F) Native T1 maps obtained at the corresponding basal, mid-ventricular, and apical levels, respectively, demonstrating elevated native T1 values within the anterolateral wall (arrows), indicative of inflammatory remodeling of the myocardial tissue.

Upon confirmation of diagnosis (day 3), we commenced antibiotic therapy with azithromycin 500 mg once daily for three days, along with the administration of non-steroidal anti-inflammatory drugs (ibuprofen) at 600 mg three times a day for seven days. On day 6, the patient's symptoms had completely resolved and the inflammatory markers returned within the normal range. She was discharged on the seventh day, and a follow-up appointment was offered in a week's time to review biochemical markers and ongoing treatment.

Discussion

In a young patient presenting with acute pleuritic chest pain, elevated cardiac troponin levels, and ECG abnormalities, several diagnoses should be considered. It is important to rule out an acute coronary syndrome (ACS), particularly coronary vasospasm or spontaneous coronary artery dissection (SCAD), which can occur in young individuals. However, in our case, the diffuse ECG changes without a clear coronary distribution, in addition to the absence of a regional wall-motion abnormality in the initial echocardiogram, made ACS unlikely. Pulmonary embolism can also manifest with pleuritic chest pain and nonspecific ECG changes, yet the absence of hypoxia made this an unlikely diagnosis. Additionally, CMR showed subepicardial late gadolinium enhancement and myocardial edema in a non-ischemic pattern. SCAD should also be considered in young women presenting with chest pain and elevated troponin levels. However, given the clinical presentation and imaging findings, an ischemic cause was considered less likely, and coronary angiography was not performed.

Campylobacter species represent major causes of bacterial gastroenteritis worldwide. In most cases, the infection is self-limiting and restricted solely to the gastrointestinal tract. However, recent reports have described growing evidence regarding possible extraintestinal manifestations such as Guillain-Barré syndrome, reactive arthritis, and cardiac complications [2]. A recent systematic review reported seven instances of myocarditis and/or pericarditis related to *Campylobacter jejuni* infection, highlighting the rarity of cardiac involvement despite the large global burden of *Campylobacter* infections [6].

In a systematic review by Sanicola et al., most of the included patients were young, with ages ranging from 13 to 25, and predominantly male. They developed cardiovascular symptoms within the first two to five days after the onset of gastroenteritis. Their symptoms were accompanied by elevated cardiac biomarkers, in addition to ECG and imaging abnormalities [6]. Similarly, individual case reports have also linked species such as *C. jejuni* and *C. coli* with myocarditis and myopericarditis, highlighting comparable demographic and clinical patterns [7,8]. In our case, a young immunocompetent woman presented with pleuritic chest pain three days after the onset of the gastrointestinal symptoms. The elevated cardiac biomarkers and the CMR imaging abnormalities closely mirror the clinical profile described in the current literature. The severity of *Campylobacter*-associated cardiac complications can vary. In most cases, patients experience full recovery within the first week with supportive care and the addition of antibiotic and anti-inflammatory therapy [4,5]. However, rare instances of fatal *Campylobacter*-associated myocarditis have been described [9].

The underlying pathophysiological mechanisms by which gastrointestinal *Campylobacter* infection leads to myocardial and pericardial inflammation remain unclear. Several theories have been proposed, including direct bacterial invasion, toxin-mediated injury, and molecular mimicry-related autoimmune myocarditis. However, the available evidence is mainly based on case reports and small case series, and the exact mechanism remains uncertain. Although cardiac symptoms often develop shortly after gastrointestinal symptoms, this timing alone cannot determine the underlying pathophysiology [5,6,10].

Our case also underlines the importance of multimodality imaging in the diagnosis of suspected myocarditis. According to the European Society of Cardiology (ESC) guidelines, the diagnosis of suspected cases of myocarditis includes clinical presentation, positive levels of cardiac biomarkers, and/or ECG and imaging changes consistent with the diagnosis, after excluding coronary artery disease and other differential diagnoses [11]. Cardiac magnetic resonance has replaced other imaging modalities as the gold standard for tissue characterization in suspected cases of myocarditis. According to the updated Lake Louise Criteria on the diagnosis of myocarditis, published in 2018, the diagnosis of suspected cases of acute myocarditis requires both a T2-based marker of myocardial edema and at least one T1-based marker of myocardial injury, which could be late gadolinium enhancement or abnormal T1 mapping [12]. In our case, CMR showed subepicardial late gadolinium enhancement in the region of the pericardium, as well as in the epicardial portion of the lateral wall of the left ventricle, which is indicative of a non-ischemic myocardial injury pattern. This is related to localized myocardial as well as pericardial inflammation, as evidenced in acute myopericarditis, according to modern CMR criteria.

Traditionally, stool culture is considered to be the gold standard for detecting the presence of *Campylobacter* species, but modern molecular assays are being increasingly implemented and may improve sensitivity and turnaround times [1]. As described in the literature, most diagnoses are confirmed through stool culture, with PCR-based methods applied occasionally to supplement them. In our case, the offending bacteria were detected via a multiplex molecular stool panel; this provided quick proof of the *Campylobacter* infection temporally related to the cardiac presentation [6]. Multiplex molecular assays allow the detection of *Campylobacter* DNA directly from stool samples without requiring bacterial isolation. However, an important limitation is that these assays may not differentiate individual *Campylobacter* species and do not provide antimicrobial susceptibility testing. Consequently, conventional culture remains valuable, particularly when species identification or antimicrobial resistance assessment is required. In our case, although *Campylobacter* was detected in the stool sample, this does not confirm that the infection directly caused the myocardial and pericardial inflammation. In our case, the association was supported by the close timing between gastrointestinal and cardiac symptoms, along with the characteristic clinical and CMR findings, and in the absence of an alternative underlying pathology. A direct causal relationship would not be possible to establish with absolute certainty due to the natural process of myopericarditis in general. However, based on the current clinical context, a *Campylobacter*-induced myopericardial inflammation was the most likely diagnosis, particularly in the absence of other risk factors (negative anti-viral serology, bacterial blood cultures, negative autoimmune screen, and epidemiological criteria).

The management of *Campylobacter*-associated myopericarditis is not standardized. The 2025 ESC guidelines on the management of myocarditis and pericarditis emphasize supportive care, which includes arrhythmia and heart failure management when these complications occur [11]. Etiological therapy is also recommended if the cause is due to infection. However, routine immunosuppressive therapy is not recommended in cases of uncomplicated viral and bacterial myocarditis. In the case of pericardial complications, the ESC guidelines state non-steroidal anti-inflammatory drugs as the first line in the management of pericarditis, with or without colchicine, in the management of acute pericarditis [11].

Due to the self-limiting nature of *Campylobacter* enteritis, antimicrobial therapy is generally not indicated. Antibiotics are recommended when patients experience severe disease (bloody diarrhea, extraintestinal involvement) or when high-risk individuals, including pregnancy, older age, or immunocompromised patients, are infected [13]. In the case of *Campylobacter*-associated myopericarditis, macrolides like azithromycin have been predominantly used. In most cases treated with macrolides like azithromycin, the symptoms have shown complete remission. In addition, fluoroquinolones like ciprofloxacin have also been used, although they are not the preferred choice given the rising rates of resistance [5]. In our case, we decided to commence treatment due to the prolonged duration of acute gastroenteritis presentation (three days) and the extraintestinal involvement (acute myopericarditis). Our patient received azithromycin 500 mg once daily for three days and ibuprofen 600 mg three times a day for seven days and made a complete recovery with symptom remission and normal cardiac biomarkers within three days after the antibiotic and anti-inflammatory treatment were initiated.

Conclusions

Extraintestinal manifestations in *Campylobacter* infections are rare but clinically significant. Clinicians should maintain a high level of suspicion for myocardium and/or pericardium involvement in patients presenting with diarrhea accompanied by chest pain or elevated cardiac biomarkers. Prompt recognition and rapid management are crucial to ensure favorable outcomes. Despite the self-limiting profile of *Campylobacter* infections, the selective use of antimicrobial therapy, particularly macrolides, may be beneficial in the setting of systemic disease. Further reporting of such cases is warranted to explore the

underlying pathophysiological mechanisms and optimal treatment strategies.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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