*Cardiology in the Young*

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# *Campylobacter coli* Myocarditis: a case report

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**Abstract**

We report the case of a 14-year-old male presented with raised myocardial injury biomarkers, on the workout, *Campylobacter coli* was identified on stool culture, treated with antibiotics with total resolution. Cardiac magnetic resonance showed interventricular septum and lateral wall hypokinesia and subepicardial delayed enhancement, with preserved ventricular systolic function. To our knowledge, this is the first report linking *Campylobacter coli* to myopericarditis in children.

**Brief Reports**

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Myocarditis is a challenging diagnosis due to the heterogeneity of clinical presentation and aetiology.[1,2] Myocarditis has been associated with a variety of infections, metabolic, and immune disorders as well as exposure to drug treatment or radiation.[3]

Viral myocarditis is the most common aetiological cause.[2,4] Although several microorganisms might be associated, *Campylobacter* infection has been infrequently implicated.[2,4]

*Campylobacter jejuni* is the most common species isolated in patients with diarrhoea, followed by *Campylobacter coli*.[5] Complications as bacteriaemia or post-infectious sequelae, as cardiac involvement have been associated with enteritis.[2–4]

To our knowledge, our report describes the first case of myocarditis associated with *campylobacter coli* enteritis in an adolescent.

## Case report

The authors report the case of a healthy 14-year-old male who presented with a 4-day fever, abdominal pain, and profuse diarrhoea, without blood, mucus, or purulent stools. He also reported a 2-day persistent left lateral chest pain, non-radiating, without triggering factors. The patient had no significant medical history or risk factors for cardiac disease. He denied any recent travel, drug use, or sexual activity.

Patient was afebrile, mildly dehydrated. Heart rate and blood pressure were within normal range. On auscultation, there were no additional murmurs or rubs, and the remaining clinical observation was irrelevant.

The chest x-ray disclosed bilateral infiltrate. The electrocardiogram was normal. C-reactive protein was elevated (58.7 mg/dL) with a normal leukocyte count. Serum creatinine kinase (604 U/L) and high sensitivity troponin I (maximal value of 8443.3 pg/mL) were raised. Liver enzymes were mildly elevated, and serum electrolytes were unremarkable.

We assumed a presumptive diagnosis of myocarditis based on clinical presentation and significantly raised myocardial injury biomarkers. The echocardiogram demonstrated normal ventricular dimensions, mild mitral, tricuspid regurgitation, and preserved diastolic function, but with borderline ventricular systolic function (biplane Simpson's ejection fraction 51% (GLS -16.1%). There was no pericardial or pleural effusion. The patient remained on bed rest and started therapy with enalapril and carvedilol.

During etiological investigation, *Campylobacter coli* was isolated on stool culture, while the remaining tests were negative. He started treatment with intravenous amoxicillin/clavulanic acid and gentamicin, based on the local antibiotic resistance. After antimicrobial sensitivity test, gentamicin was discontinued.

Four days after treatment, the patient remained asymptomatic with normalisation of myocardial injury biomarkers on the sixth day.

At discharge, the echocardiogram disclosed improvement of contractility, with systolic function within normal range (biplane Simpson's ejection fraction 53%, GLS −16.3%). The patient completed 14 days of antibiotic therapy and maintained remaining therapy during outpatient follow-up.

Cardiac magnetic resonance showed normal left ventricle volumes with hypokinesia of the basal segments of the interventricular septum and lateral wall. Ventricular function was preserved. There was no pericardial effusion, thickening, or significant myocardial oedema. Subepicardial delayed enhancement (Fig 1) of the inferolateral wall of the left ventricle was observed, consistent with subacute myocarditis (Fig 2).

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**Figure 1.** 2D myocardial late enhancement ventricular short axis.

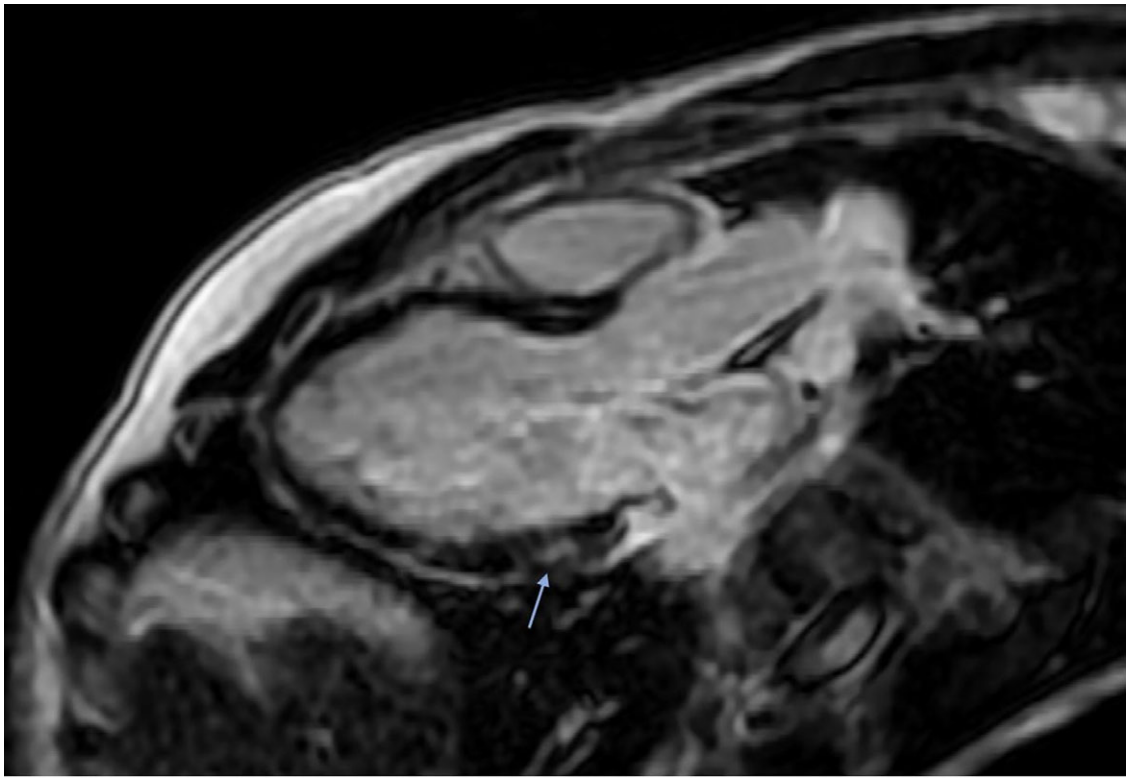


**Figure 2.** 2D myocardial late enhancement three chambers left view.

## Discussion

Myocarditis is an inflammatory condition of the myocardium.[3] It presents from mild symptoms to life-threatening cardiogenic shock and ventricular arrhythmia. Dilated cardiomyopathy might be a late consequence, reported in 6–30% of patients with worsening cardiac function.[1,3]

The incidence of myocarditis might be underdetermined due to variability of clinical presentation.[6] In the paediatric population, it can be a cause of sudden death or evolved to dilated

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cardiomyopathy, with the risk of death and heart transplantation persisting up to 12 years after diagnosis.[3,6]

Although a rare cause, most common bacterial infections associated with myocarditis are *Legionella*, mycobacteria, streptococci, and staphylococci with *Salmonella* and *Shigella* mentioned in some case reports.[2,6]

Myocarditis associated with bacterial enteritis has only rarely been described, and the pathogenesis is unclear, raising questions whether the myocardial inflammation is caused by direct action of the bacteria or by toxic or immunological mechanisms.[7]

*Campylobacter jejuni* is the most common cause of human bacterial enteritis in developed countries.[2,4] Infections often are asymptomatic and self-limited with rare extraintestinal complications, including arthritis, meningitis, or Guillain-Barré syndrome.[8] *Campylobacter,* although uncommon, are increasingly recognised to be associated with myopericarditis,[4] with almost all cases reporting *Campylobacter jejuni* or *fetus* enteritis as the etiological pathogen is associated with cardiac injury.[2] Much effort has been invested to elucidate its pathogenic mechanism, although it remains unclear.[4,9]

To our knowledge, this is the first report associating *Campylobacter coli* with myocarditis in a paediatric patient.

The diagnosis was supported by clinical presentation, raised myocardial injury biomarkers, and changes on cardiac magnetic resonance. We emphasise that a normal electrocardiogram does not exclude myocarditis; once its reported sensitivity is 47%.[6]

Broad-spectrum antibiotic therapy was initiated due to the systemic involvement of the infection,[4] either by direct infection of the myocardium, an immune hypersensitivity reaction, or the effect of bacterial toxins.[4]

Myocardial biopsy and analysis of pericardial fluid, demonstrating bacteria infiltration, support a direct infection pathophysiologic mechanism. Moffatt et al. reported two immunocompromised patients with myopericarditis and *Campylobacter* isolated in pericardial fluid,[4] while other reports did not identify *C. jejuni* on blood cultures.[9]

As described by other authors, immune-mediated aetiology was excluded, because of the short mediated time between the onset of gastrointestinal symptoms and those of myocardial injury.[9] This short period favours a toxin-mediated mechanism, although there is no evidence of a specific *Campylobacter*-associated toxicity to the myocardium.[10]

Myocardial biopsy remains the gold standard for the diagnosis of myocarditis; however, it is not performed routinely, due to known complications.[1]

Cardiac magnetic resonance has become an important non-invasive alternative for assessment of patients with suspected myocarditis.[3,6] Although myocardial biopsy can demonstrate the inflammatory infiltrate associated with myocyte necrosis or damage required to the diagnosis of myocarditis,[3] cardiac magnetic resonance diagnostic targets include functional and morphological abnormalities, as well as tissue pathology characteristic of myocardial injury.[6]

Reported clinical outcomes for *C. jejuni* and *C. coli*-associated myopericarditis in adult patients appear favourable.[4]

## Conclusion

To our knowledge, this is the first report of myocarditis associated with *Campylobacter coli* infection in a paediatric patient. Although a rare cause of myocarditis remains, it should be considered in paediatric patients with enteric symptoms.

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**Conflicts of interest.** None.

**Ethical standards.** The authors declare that the procedures followed were in accordance with the regulations of the relevant clinical research ethics committee and with those of the Code of Ethics of the World Medical Association (Declaration of Helsinki).

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