Myocarditis associated with *Campylobacter jejuni*

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**Abstract**

Myocarditis is frequently associated with a multitude of different viral infections but much less commonly a bacterial source. We present the case of a 33-year-old male with *Campylobacter jejuni* enteritis who subsequently developed myocarditis confirmed on cardiac MRI.

*Campylobacter jejuni* gastroenteritis is the most common notifiable disease in New Zealand. It comprises 41% of all 2011 notifications with a total rate of 151.9 per 100,000 population, with a regional bias towards Canterbury.¹

Myocarditis and pericarditis are frequently attributed to a multitude of different viruses, fungi and protozoa and very occasionally bacteria. A review of the literature however reveals only a few reported cases of myocarditis as a result of *Campylobacter jejuni*, this being the third such reported case in New Zealand.²³

**Case report**

A 33-year-old normally fit and healthy male was admitted to Christchurch Hospital (Christchurch, Canterbury, New Zealand) with a 2-day history of new onset chest pain.

He awoke on the day of admission with a dull central chest pain with no radiation graded 6/10 in severity. He had no associated shortness of breath, diaphoresis, palpitations, nausea, dizziness or pre-syncopal elements. There was no pleuritic component and his pain was not exacerbated or relieved by movement. The pain lasted about 1 hour before gradually subsiding. This was his second such episode in 2 days.

On admission he was pain-free. Review of systems revealed a 10-day history of diarrhoea. Smoking was his only cardiac risk factor.

On examination he appeared comfortable had a regular pulse of 70 bpm, blood pressure 110/70 mmHg and a respiratory rate of 12 saturating at 98% on room air. He was apyrexial. Cardiovascular examination was normal with normal heart sounds and no audible murmur or rubs.

His complete blood count, creatinine, urea and serum electrolytes were all within normal limits. His C-reactive protein (CRP) was elevated at 28 mg/L (n<5mg/l) as was his Troponin I (TnI) at 8.9 mcg/L (n<0.03 mcg/L).

Electrocardiogram demonstrated 1-2 mm ST elevation in leads V2-V6, II, III and aVF and hyperacute T-waves in leads V2-V4.
Due to the initial concern in the Emergency Department that he may be suffering from acute coronary syndrome he was commenced on 180 mg of ticagrelor, 300 mg of aspirin and given a therapeutic dose of enoxaparin.

Further history revealed that 10 days prior to this admission he described feeling unwell suffering from fevers and rigors associated with one episode of vomiting. In addition to this he developed multiple episodes of watery diarrhoea. Initially this was non-bloody but then progressed to three episodes of bloody diarrhoea after 5 days. There was no associated abdominal pain.

He attended his GP and subsequent faecal testing confirmed *Campylobacter jejuni*. He was commenced on roxithromycin 300 mg daily. There was no coccidian protozoa seen. No giardia or cryptosporidium antigens were detected.

The provisional diagnosis was therefore changed to myocarditis and/or pericarditis secondary to *Campylobacter jejuni* and he was commenced on analgesia and continued with the oral antibiotics.

A transthoracic echo reported a low normal systolic function with a LVEF of 56% but otherwise normal with no evidence of pericarditis.

He underwent a cardiac MRI (cardiomyopathy protocol) which demonstrated a dilated left and right ventricle with subepicardial high signal intensity consistent with myocarditis.

There was no evidence of subendocardial enhancement that would be consistent with acute coronary syndrome. Selective coronary angiography was not performed.
Over the next few days his ST elevation settled. His TnI rose to a peak of 18.6 mcg/L (tested on day 2) and then subsequently decreased to 4.3 mcg/L on the day of discharge.

His diarrhoea lasted a total of 15 days. He remained in hospital for 7 days suffering only 1 further episode of chest pain. He was discharged on metoprolol CR 47.5 mg with outpatient follow up.

**Discussion**

Myocarditis as defined by the WHO is an inflammatory heart muscle disease associated with cardiac dysfunction. It is a well-recognised possible complication of many infectious diseases with a variable presentation. Most commonly fatigue, dyspnoea on exertion, palpitations and chest pain at rest.

Our patient’s chest pain occurred 10 days following the onset of diarrhoea. A review by Hanu et al in 1995 reported a maximum delay of about 2 weeks. De novo heart failure may also occur. Myocarditis has also been implicated in between 8.6 and 12% of cases of the sudden death of young people.

Clinical findings consistent with myocarditis, markers of myocyte necrosis (CK-MB and Troponin I or T) and increased wall stress (BNP) are all supportive of a diagnosis. ECG findings may include arrhythmias (ventricular or supraventricular), atrioventricular block, a pattern of acute injury or pericarditis and nonspecific repolarisation abnormalities. ECG can also be normal. Echocardiographic findings...
may include segmental or global LV dysfunction, RV dysfunction or pericardial effusion.\(^5\)

Increasingly cardiac MRI has become much more useful as a non-invasive diagnostic test. The International Consensus Group on Cardiovascular MR recently published the Lake Louise consensus criteria which among other things looked for evidence of new or recent myocardial damage, increased T2 signal or delayed enhancement on CMR.\(^9\)

Given the prevalence of *Campylobacter jejuni* enteritis we recommend that physicians be aware of all of the potential sequelae of this bacterium.

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