**Yersinia enterocolitica**

associated myopericarditis: case report and review of the literature

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**ABSTRACT**

We report a case of myopericarditis associated with *Yersinia enterocolitica* infection in an otherwise well 50-year-old man. We discuss the clinical features, microbiology and treatment of this rare cause of myopericarditis.

A 50-year-old man presented with a two-day history of pericardial type pain. The pain was in his left chest and exacerbated by inspiration and lying flat. During the preceding three weeks he had diarrhoea and abdominal pain. Physical examination was normal and he was afebrile. He reported a three-week history of persistent diarrhoea that did not affect any other household members.

Investigations revealed an initial Troponin I (TnI) of 17,566ng/L (reference range 0–34 ng/L), C-reactive protein (CRP) 160mg/L (normal <5mg/L) and a D-dimer of 689ug/L (reference range <500ug/L), but a normal blood count. The ECG showed 2mm ST elevation in leads V2–V6, I and II and no reciprocal changes (Figure 1). An echocardiogram showed normal structure and function of the heart with no pericardial effusion. A computed tomography coronary angiogram showed no obstructive coronary disease.

Stool culture identified *Yersinia enterocolitica*, a notifiable disease in New Zealand, and excluded *Clostridium difficile*, norovirus, astrovirus, rotavirus, adenovirus 40/41 and sapovirus. We diagnosed myopericarditis associated with *Y. enterocolitica*.

**Progress**

The patient remained stable and afebrile during his admission. Monitoring with telemetry showed normal sinus rhythm. His cardiac markers and CRP decreased over his admission while he was not on any antimicrobials due to awaiting stool culture. Following culture results, his infection was managed with a 10-day course of oral ciprofloxacin. Upon follow-up 10 days post initial presentation, the CRP and TnI had resolved (5mg/mL and 10ng/L respectively). He completed the course of antibiotics and had no further gastrointestinal symptoms. A follow-up echocardiogram showed normal ventricular size and systolic function.

**Discussion**

The genus *Yersinia* is a group of gram-negative coccobacilli bacteria from the family Enterobacteriaceae. The *Yersinia* genus are facultative anaerobes, several species of which are motile below 37°C.

The first known member of the *Yersinia* genus, *Y. pestis*, was independently identified in 1894 by both Alexandre Yersin and Kiasato Shibasabuō, Swiss and Japanese bacteriologists respectively.1 *Y. pestis* achieved infamy as the cause of the “Black Death” that swept through Eurasia and North Africa. Since then, several species of the *Yersinia* genus have been identified, including *Y. enterocolitica*, first identified in 1934 by McIver and Pike2 but not comprehensively described until 1968 by Sonnenwirth3.
Y. enterocolitica is a gram-negative, frigophilic, asporogenous rod, able to grow at 4°C and survive freezing. Yersiniosis is an animal-borne disease that can affect humans, most commonly through undercooked pork and contaminated milk or water. Yersinosis typically causes a self-limited enterocolitis, terminal ileitis or adenitis in humans, which can be managed in the community but may present resembling appendicitis. Common symptoms are dependent on age. Children under 5 experience fever, abdominal pain and bloody diarrhoea, and older children and adults experience abdominal pain as the principal symptom. Typically, infection management would involve only hydration and nutritional support if necessary. However, in some instances, it may be advisable to treat the infection directly. Previous studies have shown that Y. enterocolitica is often resistant to penicillins (such as ampicillin and ticarcillin) and the first-generation cephalosporin cefazolin, but it is typically sensitive to the third and fourth generation cephalosporins (cefotaxime, ceftriaxone and cefepime) as well as some fluoroquinolones (ciprofloxacin), aminoglycosides (gentamicin and tobramycin) and sulfonamides (sulphamethoxazole/trimethoprim).

In New Zealand, Yersinosis is a relatively common diagnosis of bacterial gastroenteritis, with 1,202 cases reported in 2018, a rate of 24.6/100,000. Only one death from Yersiniosis was reported in New Zealand between 1999 and 2018.

The most common route of transmission for Y. enterocolitica infection is via food, especially pork, but it has also been associated with untreated water, animal contact and human-to-human transmission. Interestingly, this patient had minimal risk factors for Y. enterocolitica infection. He did not report eating any unusual foods, rarely consumed pork and did not consume raw or undercooked meats. He did not drink unpasteurised milk or live or work rurally. He had no contact with livestock and no sick household contacts. The water supply at his home and work were provided by the local councils and were from secure groundwater that is UV treated or chlorinated water. However, it is worth noting that most Y. enterocolitica infections in New Zealand are sporadic and have no identifiable source.

It is unusual for Y. enterocolitica to be associated with myopericarditis. A literature review showed only one published case in the English literature, one case in German and three in Danish. Several larger case series have alluded to potential cases of Y. enterocolitica myocarditis, although without the necessary clinical detail for comparison to our case.6–15

Figure 1: ECG taken from the patient on the first day of his presentation to hospital. There is 2mm ST elevation in leads V2–V6, I and II and no reciprocal changes.
The mechanism by which *Yersinia* infection results in myopericarditis is uncertain. It has been postulated that this reaction could be an immune sequela due to molecular mimicry.\(^4\) However, there has been evidence of direct *Yersinia* infection of the myocardium in animals, one puppy and one foetal foal.\(^16-17\) As we could not exclude direct infection of the myocardium, we elected to treat with a course of antibiotics and the patient recovered without sequela.
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REFERENCES