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Clinical Communications: Adults

CHEST PAIN AND DIARRHEA: A CASE OF *CAMPYLOBACTER JEJUNI*-ASSOCIATED MYOCARDITIS

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☐ **Abstract—Background: Diarrhea and chest pain are common symptoms in patients presenting to the emergency department (ED). However, rarely is a relationship between these two symptoms established in a single patient. Objective: Describe a case of *Campylobacter*-associated myocarditis. Case Report: A 43-year-old man with a history of hypertension presented to the ED with angina-like chest pain and a 3-day history of diarrhea. Electrocardiogram revealed ST-segment elevation in the lateral leads. Coronary angiogram revealed no obstructive coronary artery disease. Troponin T rose to 1.75 ng/mL. Cardiac magnetic resonance imaging showed subepicardial and mid-myocardial enhancement, particularly in the anterolateral wall and interventricular septum, consistent with a diagnosis of myocarditis. Stool studies were positive for *Campylobacter jejuni*. Conclusions: *Campylobacter*-associated myocarditis is rare, but performing the appropriate initial diagnostic testing, including stool cultures, is critical to making the diagnosis. Identifying the etiology of myocarditis as bacterial will ensure that appropriate treatment with antibiotics occurs in addition to any cardiology medications needed for supportive care.** © 2014 Elsevier Inc.

☐ **Keywords—*Campylobacter jejuni*; diarrhea; myocarditis; myopericarditis**

INTRODUCTION

Diarrhea and chest pain are common symptoms in patients presenting to the emergency department (ED). However, rarely is a relationship between these two symptoms established in a single patient. We describe a case of myocarditis complicating an episode of traveler's diarrhea. *Campylobacter*-associated myocarditis (CAM) is extremely rare, with only a few cases reported in the literature.

CASE REPORT

A 43-year-old man with a history of hypertension presented to the ED with substernal, pressure-like, nonradiating severe chest pain, which had begun hours earlier. He also reported 3 days of profuse, watery, and occasionally bloody diarrhea. He had no recent travel outside of the United States (US). Cardiac risk factors included hypertension and tobacco use. His only medication was lisinopril 20 mg daily.

On examination, the heart rate was 90 beats/min and blood pressure 124/82 mm Hg. Cardiac auscultation revealed normal S1, S2 with no murmurs, gallops, or pericardial friction rub. Jugular venous pressure was normal, lungs were clear to auscultation, and distal pulses were symmetrical and equal. Palpation of the thorax and abdomen did not aggravate the chest pain. Hyperactive bowel sounds were present.

Complete blood count, electrolytes, creatinine, and liver enzymes were normal. Troponin T was elevated at 0.9 ng/mL (upper limit of normal 0.03 ng/mL).

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Electrocardiogram (ECG) revealed sinus rhythm with mild ST-segment elevations in the lateral leads without T-wave inversions (Figure 1A). An urgent coronary angiogram showed normal coronary arteries without any luminal irregularities or evidence of spasm.

Troponin T continued to rise, peaking at 1.75 ng/mL. The creatine kinase (CK)-MB peaked at 79.5 ng/mL and CK at 630 u/L, giving a relative index of > 10%. High-sensitivity C-reactive protein (hs-CRP) was markedly elevated at 90.7 mg/L. ECG at 48 h demonstrated evolution of the ST segments in the lateral leads (Figure 1B). A cardiac ultrasound showed a left ventricular ejection fraction of 68%, with wall motion abnormalities in the inferior septum. Stool cultures were negative for *Salmonella*, *Shigella*, and *Escherichia coli* H 157, O7. However, enzyme-linked immunosorbent assay of stool was positive for *Campylobacter jejuni.*

With a normal coronary angiogram but symptoms of angina, positive cardiac enzymes, labile ST-segment elevation, and a regional wall motion abnormality, the diagnosis of myocarditis was considered. Cardiac magnetic resonance (MR) imaging with delayed inversion recovery sequences showed subepicardial and mid-myocardial enhancement, particularly in the anterolateral wall and interventricular septum, consistent with a diagnosis of myocarditis (Figure 2). No pericardial enhancement was seen.

With a diagnosis of myocarditis, the patient was treated with high-dose aspirin (3000 mg by mouth [p.o.] daily in divided doses) and metoprolol tartrate (25 mg p.o. daily), and azithromycin (500 mg p.o. daily) for his *Campylobacter jejuni* diarrhea. His chest pain improved and he was discharged on the third hospital day with a final diagnosis of myocarditis induced by *Campylobacter jejuni*.

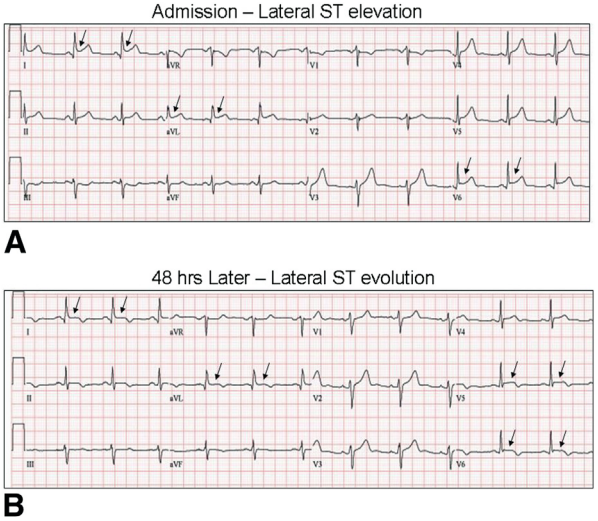

Figure 1. Electrocardiograms at admission and at 48 h. (A) ST-segment elevation in the lateral leads (arrows) at admission. (B) ST-segment evolution in the lateral leads (arrows) after 48 h.

DISCUSSION

Acute myocarditis is an inflammatory disease of the heart muscle commonly due to a viral illness or deemed idiopathic (1). Viruses most commonly associated with myocarditis include Echo, Coxsackie, and enterovirus, but may also involve influenza, Epstein-Barr, cytomegalovirus, adenovirus, enterovirus, parvovirus B19, herpes simplex, hepatitis A, B, and C, and human immunodeficiency viruses (2). Uncommonly, it may be due to a bacterial infection. Enteric infections like *Salmonella* and *Shigella* can rarely cause myocarditis (3). Myocarditis can also been caused by *Mycobacterium tuberculosis* and other bacteria, including *Streptococci*, *Staphylococci*, *Pneumococci*, *Neisseria*, *Legionella*, *Coxiella*, and *Haemophilus* (2).

The *Campylobacter* genus is one of the most common causes of infectious enterocolitis in the developed world. The estimated annual incidence of *Campylobacter* infections in the US is as high as 1% (1000 per 100,000 population) (4). However, CAM is extremely rare, with only very few cases reported in the world literature, the majority from Europe (5,6). One reported fatal case of CAM from the US involved an individual who had returned from Thailand after a tsunami relief operation (7).

There are two recognized patterns of heart disease caused by *Campylobacter* species: 1) *Campylobacter fetus* (*C. fetus*), causing bacteremia and pericarditis, and 2) *Campylobacter jejuni* (*C. jejuni*), causing myocarditis without bacteremia. Patterns of involvement are attributed to the presence of a complement-resistant surface layer in *C. fetus* and absence of the same in *C. jejuni*. This gives *C. fetus* the ability to evade the host immune system and use the bloodstream as a transport system with a capability to cause systemic campylobacteriosis, especially in immunocompromised hosts. For this reason, direct invasion is the likely pathogenesis of pericarditis by *C. fetus*. In *C. fetus*-associated pericardial involvement, bacteria have been reportedly isolated, commonly from the blood or pericardial fluid. However, systemic campylobacteriosis due to *C. jejuni* is extremely rare and usually affects immunocompromised patients. This is due to the fact that *C. jejuni* lacks the surface layer and is susceptible to the bactericidal activity of human serum (8). *C. jejuni* is also not known to have predilection for the cardiovascular system or capability of directly invading the vascular endothelium, unlike *C. fetus* (9,10).

The exact nature of myocardial involvement by *C. jejuni* is not known for CAM. It could be due to direct bacterial invasion, due to toxins, cytotoxic T-cell-mediated