Pericarditis Associated with *Campylobacter jejuni* Enterocolitis

Maciej Nowakowski, MD
Dariusz Listopadzki, MD
Stephen R. Freeman, MD

Campylobacter jejuni is one of the most common bacterial pathogens causing infectious enterocolitis. Cardiac complications related to this infection, however, including myocarditis and pericarditis, are extremely rare. This article describes the case of a 15-year-old boy who develops pericarditis associated with *C. jejuni* enterocolitis.

CASE PRESENTATION

Initial Presentation and History

A 15-year-old boy presented to a local rural outpatient clinic with 5 days of bloody diarrhea, cramping abdominal pain, dehydration, and malaise. He reported a recent trip to a resort in Florida, where he ate meals that included hamburgers and pork and drank water from water fountains.

There was no medical history of any significant episodes of diarrhea in the past. The patient did have a few episodes of syncope 2 years prior to this presentation. The work-up at that time consisted of an echocardiogram and tilt-table testing, both of which showed normal results.

Initial Examination and Management

Physical examination showed an adolescent boy in no acute distress who was afebrile and had borderline tachycardia. Results of the head and neck examination were normal. The chest was clear to auscultation; heart sounds were normal without gallop, rub, or murmur. The abdominal examination showed diffuse tenderness, most pronounced in the right and left lower quadrants, without any rebound or guarding. Increased bowel sounds were noted. Rectal examination showed brown, watery stool that tested strongly guaiac-positive. The patient's leukocyte count was $7.8 \times 10^3/mm^3$ (normal range, $4.8–10.8 \times 10^3/mm^3$) with a left shift. The hemoglobin level was within normal limits.

The patient was diagnosed with infectious diarrhea and was rehydrated with intravenous normal saline solution. He was sent home with instructions to continue oral hydration and gradually advance his diet. Stool cultures were taken, and no antibiotic therapy was given at that time.

Subsequent Presentation to the Emergency Department

The following day, the patient presented to the hospital complaining of severe retrosternal chest pain radiating to his left shoulder. The pain was significantly worse when lying in a supine position and also was exacerbated by coughing and deep inspiration. The patient did not complain of shortness of breath. His pain was very severe in the upright position, with its intensity rated 6/10 by the patient, increasing to 9/10 during attempts to be supine. He denied having used any illegal stimulants.

On physical examination, patient appeared ill; he was in obvious pain and was very reluctant to lie down. His blood pressure was 95/59 mm Hg. There was no pulsus paradoxus and the Kussmaul's sign was negative. His pulse was 110 bpm. Respirations were regular with a rate of 22 breaths/min. Oxygen saturation on room air by pulse oximetry was 96%. The patient exhibited no pallor or cyanosis, and the mucous membranes in the oropharynx were clear without any swelling or erythema. The examination of the neck showed normal position of the trachea, no lymphadenopathy, and no distention of the jugular veins in either the upright or the supine position. His carotid pulses were equally strong bilaterally without any murmurs.

Auscultation of the lungs showed good air entry bilaterally, without pleural rub or any signs suggesting

Drs. Nowakowski and Listopadzki are physicians at Kimball County Hospital, Kimball, NE. Dr. Freeman is an associate professor of medicine, Division of Gastroenterology, University of Colorado Health Sciences Center, Denver, CO.

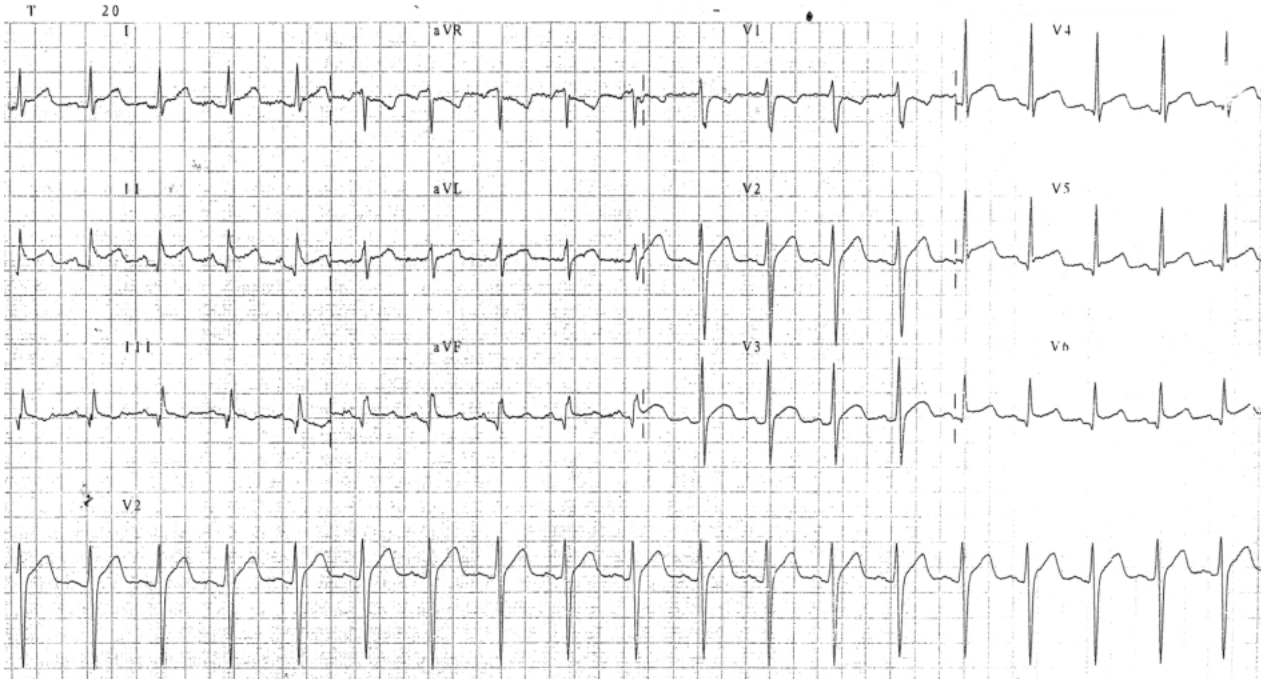

Figure 1. Electrocardiogram of the case patient upon presentation to the emergency department shows changes consistent with acute pericarditis.

lung consolidation or increased secretions in the airways. Palpation of the point of maximal impulse showed a strong impulse in the fifth intercostal space 2 cm medial to the midclavicular line. Cardiac auscultation showed regular first and second heart sounds without any gallop or murmurs. A pericardial rub was not appreciated. Results of the abdominal examination were normal, without tenderness, palpable organomegaly, or pathologic masses. Hepatojugular reflux was not detected. The neurologic examination did not show any focal signs indicating pathology of the cranial nerves or cerebellum or relating to motor power or sensation.

Results of a complete blood count showed 8.1 × 10^3/mm^3 leukocytes with 54% segmented cells, 14% bands, 29% lymphocytes, and 3% eosinophils. Serum levels of creatine phosphokinase, creatinine, lactate dehydrogenase, and amylase were within normal limits, as were serum electrolytes and blood urea nitrogen. Results of liver function testing were normal. Chest radiograph was normal without evidence of consolidation, a widened mediastinum, or pneumothorax. An electrocardiogram was obtained and showed sinus tachycardia with a heart rate of 111 bpm, normal axes, diffuse PQ segment depressions, and horizontal ST-segment elevations in leads I, II, III, and aVF as well as in leads V_2 to V_6 (**Figure 1**).

Diagnosis

The differential diagnosis in this patient included unstable coronary artery disease, pericarditis, pulmonary embolism, pleurisy, and pneumothorax. The presence of coronary artery disease was extremely unlikely in view of the patient's young age, the absence of any risk factors, and his categoric denial of use of any illicit stimulants. A pulmonary embolus was unlikely in view of the good oxygen saturation and the absence of shortness of breath. Pneumothorax was ruled out by a normal chest radiograph. In view of the patient's clinical presentation and electrocardiogram, the patient was diagnosed with pericarditis without clinical evidence of cardiac tamponade.

Stool culture showed extensive growth of *C. jejuni*, and many erythrocytes and leukocytes were noted in the stool. Blood cultures and stool examination for ova and parasites were negative as was screening for *Escherichia coli* O157:H7.

Treatment and Outcome

Because the patient did not have signs of hemodynamic instability or impending cardiac tamponade, it was considered safe to admit him to the small rural hospital, continuously monitor his vital signs, and perform echocardiography at a later available date. The patient was

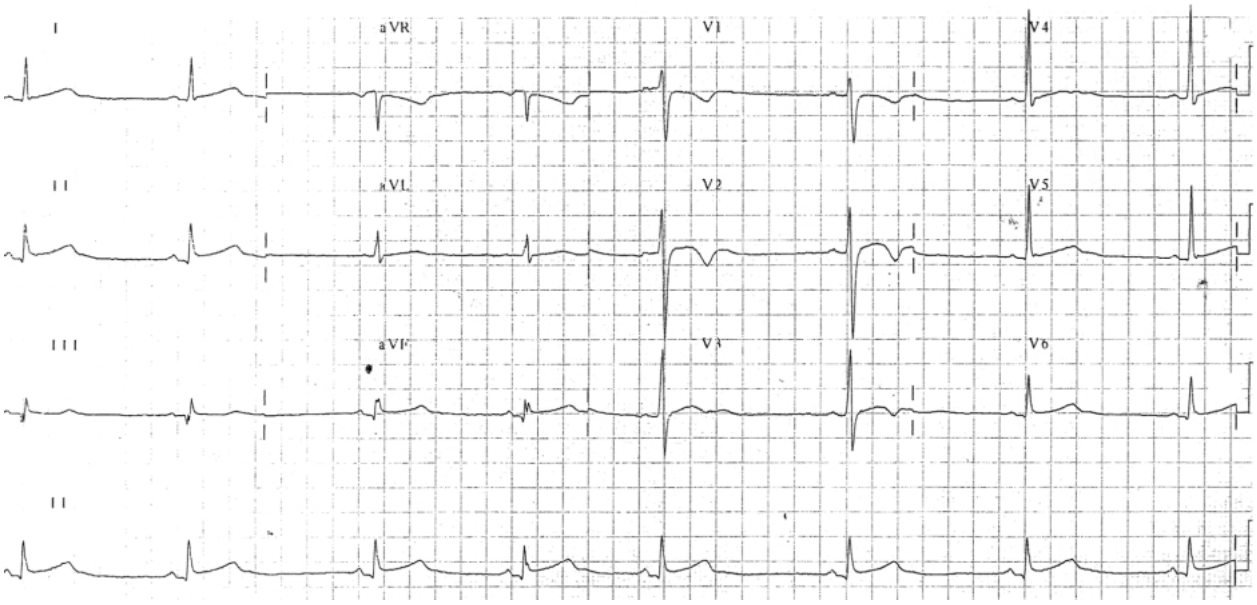

Figure 2. Follow-up electrocardiogram of the case patient performed 3 days after discharge shows changes consistent with resolving pericarditis.

admitted to the hospital, rehydrated intravenously, and started on intravenous erythromycin. His pain was successfully managed with intravenous morphine and oral hydrocodone/ibuprofen. He showed dramatic improvement on the next day and was discharged home on oral ibuprofen and erythromycin. His subsequent recovery was uneventful. An electrocardiogram performed 3 days after discharge showed changes consistent with resolving pericarditis (**Figure 2**). An echocardiogram performed 5 days after discharge showed neither evidence of pericardial effusion nor any other abnormalities.

DISCUSSION

Campylobacter Enterocolitis

Infections of gastrointestinal tract cause of significant mortality and morbidity worldwide, especially among children. People from highly industrialized countries who visit underdeveloped parts of the world are frequently affected. Travelers within industrialized countries often are affected as well.[1] Children become infected more often because of their high fecal-oral contamination rate. Juvenile and adolescent tourists have an increased risk of gastrointestinal infection owing to their often free and unpredictable style of traveling.

Campylobacter is one of the most frequent bacterial causes of infectious enterocolitis. The *Campylobacter* genus consists of several species, of which *C. jejuni* and *Campylobacter coli* are associated with clinically significant enterocolitis in humans. In most cases, the culprit is *C. jejuni*, a gram-negative bacteria that usually is discovered on routine stool culture. The infection is transmitted via the fecal-oral route, most frequently through contaminated milk or undercooked poultry, as well as through contact with pets. The pathogenic mechanism of the enterocolitis relies on the production of the enterotoxins and cytotoxins as well as direct invasion of the intestinal mucosa.[2]

Campylobacter Pericarditis

Pericarditis is a very unusual complication of campylobacter enterocolitis. Acute pericarditis is most commonly idiopathic or viral, which combined, account for up to 75% to 80% of cases.[3] Coxsackieviruses A and B, echovirus, adenovirus, and HIV are the predominant causes of viral pericardial infection. Other notable agents of infectious pericarditis include streptococci, staphylococci, *Haemophilus* sp., and *Mycobacterium tuberculosis*. Occasional culprits include mycoplasmata, rickettsiae, chlamydiae, fungi, and parasites. Meningococcal infection can cause an immune complex–mediated (reactive) pericarditis as well as a true pericardial infection.[4,5] Other causes of immune complex–mediated pericarditis include autoimmune rheumatic diseases (systemic lupus erythematosus, systemic sclerosis, mixed connective tissue disease, rheumatoid arthritis), inflammatory gastrointestinal diseases (Crohn's disease, ulcerative colitis), drugs

(procainamide, phenytoin, hydralazine), and Dressler's syndrome (ie, post–myocardial infarction syndrome).

Cardiac manifestations of campylobacter infection are highly unusual and may be caused by direct invasion of cardiac tissue by the bacteria, bacterial toxins, circulating immune complexes, or cytotoxic T cells.[6] The most commonly described pericarditis related to *Campylobacter* species is purulent pericarditis, which is associated with pericardial infection in a septic patient. In the case presented, the nature of pericardial injury is not clear, but it probably was caused by bacterial toxins or an immunologic mechanism. The association of acute pericarditis with campylobacter enterocolitis is so rare that there are no pathologic studies explaining the mechanism of cardiac injury.[7] In the Western medical literature, we found only a few cases of campylobacter-induced myocarditis,[7–10] 1 case of both myocarditis and pericarditis,[11] and 2 cases of pericarditis without involvement of the myocardium.[12,13] One of the two reported patients with campylobacter-induced pericarditis had an underlying pericardial effusion caused by pre-existing hypothyroidism.[12] The other patient described, as in the current case, did not have any other heart disease; he was a young traveler and made an uneventful recovery.[13]

CONCLUSION

The patient described, a young US traveler, presented with acute infectious diarrhea due to *C. jejuni*, complicated by acute pericarditis. Treatment of the underlying infection resulted in rapid resolution of both the enterocolitis and the pericarditis. Acute pericarditis is a very rare complication of *C. jejuni* enterocolitis. **HP**

REFERENCES

1. Ericsson CD, DuPont HL. Traveler's diarrhea: approaches to prevention and treatment. Clin Infect Dis 1993,16: 616–24.
2. Beltinger J. [A case from practice {326}. Dysentery, inflammatory diarrhea, cause *Campylobacter jejuni*.] [Article in German.] Schweiz Rundsch Med Prax 1995;84:708–9.
3. Shabetai R, Soler-Soler J, Corey GR. Etiology of pericardial disease. In: UpToDate. Wellesley, MA, 2003. Available at www.uptodate.com. Accessed 1 Dec 2003.
4. Beggs S, Marks M. Meningococcal pericarditis in a 2-year-old child: reactive or infectious? J Paediatr Child Health 2000;36:606–8.
5. Finkelstein Y, Adler Y, Nussinovitch M, et al. A new classification for pericarditis associated with meningococcal infection. Eur J Pediatr 1997;156:585–8.
6. Mason JW. Myocarditis. Adv Intern Med 1999;44:293–310.
7. Florkowski CM, Ikram RB, Crozier IM, et al. *Campylobacter jejuni* myocarditis. Clin Cardiol 1984;7:558–9.
8. Westling K, Evengard B. Myocarditis associated with campylobacter infection. Scand J Infect Dis 2001;33:877–8.
9. Wanby P, Olsen B. Myocarditis in a patient with salmonella and campylobacter enteritis. Scand J Infect Dis 2001;33:860–2.
10. Cox ID, Fluck DS, Joy MD. Campylobacter myocarditis; loose bowels and a baggy heart. Eur J Heart Fail 2001;3: 105–7.
11. Ponka A, Pitkanen T, Pettersson T, et al. Carditis and arthritis associated with *Campylobacter jejuni* infection. Acta Med Scand 1980;208:495–6.
12. Lieber IH, Rensimer ER, Ericsson CD. Campylobacter pericarditis in hypothyroidism. Am Heart J 1981;102(3 Pt 1):462–3.
13. Rahman M. Bacteraemia and pericarditis from campylobacter infection. Br J Clin Pract 1979;33:331, 4.

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