Myocarditis Associated With Campylobacter Enteritis: Report of Three Cases

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Myocarditis connected with bacterial infection is rare in immunocompetent hosts.1 Campylobacter jejuni infection is a commonly recognized cause of bacterial gastroenteritis. Several case reports have suggested an association between Campylobacter enteritis and the development of myocarditis and pericarditis.2,3

Case Reports

Case 1. Six days after the onset of acute watery diarrhea, a 42-year-old previously healthy man was admitted with abnormal tiredness, dyspnea, and persistent watery diarrhea. Biochemical analysis showed elevated plasma creatine kinase (178 U/L; normal value 170 U/L), troponin I (3.67 μg/L; normal value <0.13), and NT-proBNP levels (1722 ng/L; normal value <115). The ECG was normal on admission. A moderately decreased systolic left ventricular (LV) function with diffuse hypokinesia was seen on echocardiography. Cardiac magnetic resonance imaging (cMR) confirmed a reduced LV systolic function (ejection fraction [EF] 61%; Supplemental Video 1 and 2; see online-only supplement) and showed patchy areas of increased signal intensity on T2-weighted images, suggesting myocardial edema (Figure 1, panel A). After administration of intravenous gadolinium, diffuse and persisting enhancement of the subepicardium and the midwall was seen (Figure 2, panel A). The next day troponin I reached a peak level of 15.6 ng/L, and new repolarization disturbances in the inferolateral leads were noticed. C jejuni, resistant to ofloxacin, was recovered from the stool culture. We made the diagnosis of acute myocarditis associated with C jejuni enteritis. Azithromycin was started, together with an angiotensin converting-enzyme inhibitor. Four weeks after admission the patient feels better, but still notices a subnormal exercise capacity. Troponin I levels remain slightly elevated (0.25 μg/L). His NT-proBNP level has normalized (36 ng/L), as was the left ventricular ejection fraction on cMR (EF 61%; Supplemental Video 3 and 4; see online-only supplement). The pathological late wall enhancement on cMR is still visible, but less pronounced (Figure 2, panel B).

Case 2. Two days after the onset of acute watery diarrhea and vomiting, a 34-year-old previously healthy man was admitted because of dehydration. He was started on intravenous fluid therapy. Two days after admission, he developed chest pain. ECG revealed elevated ST-segments in leads V4 through V6, and troponin I levels were elevated (8.9 μg/L). Cardiac catheterization showed a normal coronary angiogram. The ventriculogram revealed diffuse hypokinesia and a moderately decreased LV function (EF 40%). Chest pain resolved within 24 hours, as did the diarrhea. cMR revealed persisting late enhancement after administration of gadolinium, confined to the subepicardium of the inferolateral wall, as well as a small pericardial effusion. Global systolic LV function on cMR was mildly decreased (EF 50%), with hypokinesia in the affected segments. C jejuni, resistant to ofloxacin, was detected in the stool culture. The diagnosis was acute myopericarditis associated with Campylobacter infection. No antibiotic treatment was started because diarrheal and cardiac symptoms had subsided. Angiotensin converting-enzyme inhibitors and a low dose of β-blockers were initiated on discharge. Four weeks later, the patient is doing well with normal exercise capacity reported. On repeat cMR, LV systolic function has recovered to normal (EF 62%), but focal areas of pathological late enhancement remain visible.

Case 3. A 21-year-old previously healthy man developed chest pain, 3 days after the onset of acute febrile diarrhea. ECG on admission revealed elevated ST-segments in leads V4 through V6. An echocardiogram showed a moderately decreased systolic LV function (EF 40%). Creatine kinase and troponin I levels were elevated (641 U/L and 11.6 μg/L, respectively), as was his NT-proBNP level (318 ng/L). Mild enlargement of both left and right ventricles and a small pericardial effusion was seen on cMR. Myocardial edema was demonstrated in the lateral wall of the LV (Figure 1,
panel B), where, after administration of intravenous gadolinium, pathological delayed enhancement of the subepicardium also was present. A stool culture, taken before admission, revealed *Campylobacter* species, not identified to the species level. We made the diagnosis of *Campylobacter*-associated myopericarditis. Ciprofloxacin, started before hospitalization, was continued during hospitalization, and he was put on angiotensin converting-enzyme inhibitors. One month later, the patient has recovered completely. cMR shows normal dimensions of both ventricles and a normal systolic LV

**Figure 1.** Focal areas of increased signal intensity (arrows) are visualized by cardiac magnetic resonance imaging on short-axis T2-weighted images, indicating the presence of myocardial edema, secondary to myocytolysis and leukocyte infiltration. In case 1, there is a diffuse, patchy pattern of myocardial edema visible (panel A), whereas in case 3, the increased signal intensity is confined to the lateral wall of the left ventricle (panel B).

**Figure 2.** Cardiac magnetic resonance imaging shows patchy areas of pathological late enhancement (arrows) on T1-weighted images after administration of gadolinium intravenously (panel A). Follow-up examination after 4 weeks shows a decrease in the pathological late enhancement, suggesting resolving inflammation or evolution toward fibrosis (panel B).
function (EF 56%). The late wall enhancement is still visible, though less pronounced.

**Discussion**

We report 3 cases of myocarditis associated with *Campylobacter* enteritis in young, immunocompetent male patients. The diagnosis of myocarditis was established by a combination of clinical, laboratory, and cardiac imaging criteria. The diagnosis of *Campylobacter* enteritis was confirmed by positive stool cultures.

On cMR, increased signal intensity in the myocardium, suggesting myocardial edema, was observed on the T2-weighted images, as well as pathological late enhancement after administration of gadolinium on the T1-weighted images. Hyperintense areas on T2-weighted images are the result of an increase in the myocardial free water content, due to lymphocyte infiltration and myocytolysis. Increased left ventricular wall thickness sometimes can be seen in association with myocardial edema. However, regional wall thickness measurements were within normal ranges on cMR in all 3 cases, without any significant difference during the acute onset scan and the follow-up examination. Increased delayed wall enhancement is caused by cell damage and extravasation of fluid in the interstitial space, whereby gadolinium accumulates in the area of inflammation. On repeat cMR after 4 weeks, abnormal late wall enhancement was still visible, but less pronounced, suggesting resolving inflammation or evolution toward fibrosis.4

The underlying pathophysiology of myocarditis secondary to *Campylobacter* infection is unclear. The short time interval between the onset of enteritis and the onset of myocarditis makes an immunologic mechanism unlikely. We assume damage to the myocardium by direct bacterial invasion or through circulating toxins.

**Disclosures**

None.

**References**


**Key Words:** Campylobacter enteritis ■ magnetic resonance imaging ■ myocarditis
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*Circ Heart Fail.* 2012;5:e19-e21
doi: 10.1161/CIRCHEARTFAILURE.111.964882
*Circulation: Heart Failure* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-3289. Online ISSN: 1941-3297

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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Supplemental Material

Video Legends

Video 1: Four-camber view on initial cine MRI in case patient 1 demonstrates diffuse hypokinesia and a reduced global left ventricular systolic function.

Video 2: Short-axis view on initial cine MRI in case patient 1 shows diffuse hypokinesia and a reduced global left ventricular systolic function.

Video 3: Four-camber view on follow-up cine MRI in case patient 1 shows normal regional contractility and restoration of global left ventricular systolic function.

Video 4: Short-axis view on follow-up cine MRI in case patient 1 reveals normal regional contractility and a normal global left ventricular systolic function.