Case history

Campylobacter myocarditis; loose bowels and a baggy heart

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Abstract

We report an unusual case of acute myocarditis associated with \textit{Campylobacter jejuni} enterocolitis leading to severe impairment of left ventricular systolic function. Contrast-enhanced cardiac magnetic resonance imaging was used to confirm the presence of acute myocardial inflammation and its resolution. © 2001 European Society of Cardiology. All rights reserved.

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1. Case report

A previously fit 32-year-old man presented with a 2-week history of diarrhoea, increasing breathlessness and chest pain. He had suffered an acute onset of profuse watery motions associated with fever and malaise during a business trip to South America. He also described worsening exertional dyspnoea and a band-like chest pain which was unrelated to exertion, respiration or change in posture. He was a non-smoker, consumed approximately 35 units of alcohol per week and was receiving no regular medication.

On initial examination, he was pyrexial (37.3°C) and appeared pale and sweaty. He had a regular resting pulse rate of 90 beats/min and the systemic blood pressure was 110/90 mmHg. Auscultation revealed normal heart sounds with a mid-systolic click but no additional heart sounds or murmurs. The jugular venous pressure was not elevated and there was no peripheral oedema. Respiratory examination was unremarkable. The abdomen was soft with mild tenderness in the right upper quadrant and active bowel sounds on auscultation.

Chest X ray revealed an increased cardio-thoracic ratio (17.5/32 cm) with fluid in the transverse fissure and upper lobe blood diversion. The initial electrocardiogram indicated a low atrial rhythm with a mean frontal QRS axis of $-30^\circ$ and symmetrically inverted T waves in leads V4-6, SI and aVL. Two dimensional echocardiography revealed a dilated left ventricle (end diastolic internal diameter [LVIDd] 6.8 cm; end systolic internal diameter [LVIDs] 6.0 cm) with globally impaired systolic function. There was mild mitral regurgitation on Doppler echocardiography but no other abnormalities. A full blood count, urea and electrolytes and liver function tests were all within normal limits. The serum creatinine kinase (399 units/l) and lactate dehydrogenase (677 unite/l) were elevated, as were the erythrocyte sedimentation rate (126 mm/h) and the C reactive protein (123 mg/l).

A diagnosis of acute infective diarrhoea and myocarditis was made. The patient was treated with frusemide and captopril with good symptomatic effect. Intravenous gentamycin was added following isolation of \textit{Campylobacter jejuni} from the stool cul-

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Fig. 1. (a) CMR during the acute phase of the illness revealed diffuse enhancement of the myocardial signal intensity following the intravenous administration of gadolinium-DTPA, particularly in the septum and lateral wall; these findings were compatible with active inflammation in these areas. (b) Repeat CMR 3 months after initial presentation showed a significant reduction in the enhancement of the myocardial signal intensity following intravenous gadolinium-DTPA suggesting resolving myocardial inflammation.

ture. Serological tests indicated high antibody titres to
*C. jejuni* in all classes (IgM 5120, IgG 2560, IgA 1280).
Routine stool cultures for *Salmonella* and *Shigella*
species were negative as were all other serological
tests (including cytomegalovirus, adenovirus, enteroviruses, hepatitis A, B and C, *Mycoplasma pneu-
moniae, Trypanosoma cruzi, Chlamydia psittaci, Coxiella burnetti and Entamoeba histolytica). Cardiac magnetic resonance (CMR) was performed and revealed diffuse enhancement of the myocardial signal intensity following the intravenous administration of gadolinium-DTPA (Fig. 1a); this was particularly evident in the septum and lateral wall and was compatible with active inflammation in these areas [1].

During follow-up, the patient reported a gradual improvement in his exercise tolerance and repeat echocardiography indicated a reduction in the left ventricular dimensions LVIDd 6.5 cm, LVIDs 4.4 cm at 6 months follow-up). Repeat CMR 3 months after initial presentation showed a significant reduction in the enhancement of the myocardial signal intensity following intravenous gadolinium-DTPA (Fig. 1b), suggesting resolving myocardial inflammation.

2. Discussion

We have identified only four previous case reports of acute myocarditis associated with C. jejuni enterocolitis in the world literature [2–4]. In all cases, the myocarditis followed a benign course and was not associated with heart failure. In the present case, left ventricular systolic function was severely impaired and did not return to normal over a period of several months. It seems possible that our patient had an undiagnosed cardiomyopathy prior to the episode of enterocolitis. Nevertheless, the clinical history and CMR findings support the hypothesis that the heart was involved in an acute inflammatory process associated with C. jejuni infection. The nature of any myocardial involvement in C. jejuni infection remains speculative but seems more likely to involve bacterial invasion of the myocardium than immunologically mediated inflammation since the myocarditis appears to occur at the same time as the enteric infection [3,5]. In any event, it is well to be aware that C. jejuni enterocolitis may be associated with an acute myocarditis.

The value of performing endomyocardial biopsy in patients with acute myocarditis has been questioned due to the low rate of positive histological findings in patients with clinical signs of acute myocarditis and the absence of proven benefit of immunosuppressive therapy in patients with positive biopsy findings [6]. Such procedures are also associated with a significant incidence of complications and, in this respect, contrast media-enhanced CMR offers a useful non-invasive diagnostic alternative. In our patient, the contrast-enhanced CMR demonstrated diffuse enhancement of the myocardial signal intensity during the acute phase of his illness although this was particularly evident in the septum and lateral wall. The inflammatory changes in acute myocarditis can be focal or multifocal and this may partly explain the high incidence of negative biopsy findings in patients with clinical evidence of acute myocarditis. Other studies using contrast-enhanced CMR have reported similar regional variations in myocardial signal intensity in patients with myocarditis and such variations are thought to reflect the distribution, activity and extent of myocardial inflammation [1].

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References


