A diarrhoeal illness complicated by heart failure

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INTRODUCTION

Myocarditis, defined as myocardial inflammation associated with tissue necrosis, has been reported secondary to infection, systemic disease, toxins and drugs. *Campylobacter jejuni* has a broad spectrum of disease manifestations. A rare association is with myocarditis and acute hepatitis. This article describes the first documented case of *C. jejuni*-associated myocarditis presenting as a myocardial infarction complicated by heart failure, hepatitis and renal failure.

CASE REPORT

A 34-year-old man presented to casualty with a history of anginal chest pain, nausea and sweating. He had a 2-week history of diarrhoea and night sweats after ingestion of poorly cooked poultry. On examination the patient was pyrexial (39°C) with a blood pressure of 110/80 mmHg. His heart rate was 120 per minute with a gallop rhythm. Jugular venous pulse was elevated at 8 cm. There was marked pitting ankle oedema. Oxygen saturations were 93% on air and there were coarse crackles at both lung bases. Abdominal and neurological examinations were normal.

Admission electrocardiogram (ECG) showed sinus tachycardia with ST segment elevation in leads V2–V4 (*Figure 1*). Anterior myocardial infarction was diagnosed and the patient was treated with aspirin and diamorphine, before thrombolysis with reteplase. Following thrombolysis the chest pain subsided but there was no resolution of ST segment elevation. The patient was transferred to the authors' hospital for further assessment. Blood results on admission showed a leucocytosis (33×10^9/litre, predominantly neutrophils), with mild renal impairment (urea = 9.3 mmol/litre, creatinine = 123 μmol/litre). Creatine kinase and alanine transferase were elevated (692 iu/litre and 4503 iu/litre respectively), as was C-reactive protein (410 mg/dl). Troponin T was positive at 0.59 ng/litre, consistent with myocardial injury. The remaining liver function tests and electrolytes were normal. A chest radiograph showed a globular heart with upper lobe blood diversion. A transthoracic echocardiogram demonstrated significant left ventricular impairment (left ventricular ejection fraction was estimated at 30%) with antero-apical hypokinesia.

The patient remained pyrexial and breathless on minimal exertion. He became oliguric with increasing leucocytosis (white cell count = 45×10^9/litre) and deteriorating renal and liver function tests (urea = 10 mmol/litre, creatinine = 257 μmol/litre, bilirubin = 39 μmol/litre, alanine transferase = 4503 iu/litre, alkaline phosphatase = 86iu/litre). Urine dipstick was positive for protein, but microscopy was negative for cellular casts. Abdominal ultrasound was normal. The results of stool culture demonstrated *Campylobacter jejuni*. Serology was strongly positive for *Campylobacter* (immunoglobulin (Ig)M > 10240 iu, IgG = 2560 iu, IgA = 1280 iu), but was negative for coxsackie, adenoviruses, Mycoplasma and Legionella. A diagnosis of acute hepatitis, myocarditis and heart failure secondary to *Campylobacter* infection was made. The patient was treated with erythromycin, furosemide and ramipril.

Following commencement of antibiotics, dyspnoea improved and pyrexia settled. This coincided with an improvement in liver function tests, inflammatory markers and renal biochemistry which all had normalized by the 10th day of admission. Coronary angiography demonstrated normal coronary arteries with moderate left ventricular function (estimated ejection fraction 50%). The patient was discharged with a further 2-week course of erythromycin, furosemide and ramipril. At 3-month follow up he was well with a normal electrocardiogram (*Figure 2*). Repeat echocardiography demonstrated good left ventricular function (ejection fraction 80%).

DISCUSSION

This patient presented with clinical and electrocardiographic (ECG) features of a myocardial infarction associated with left ventricular failure, acute hepatitis and renal failure. Although the patient was initially treated as having a myocardial infarction a correct diagnosis of myocarditis was made in retrospect.

C. jejuni has several disease manifestations, the most frequent of which is enteritis. Other manifestations include arthritis (septic and reactive), cholecystitis and Guillain–Barré syndrome. Myocarditis is a rare complication of *C. jejuni* enteritis. There are five described cases of myocarditis associated with *C. jejuni* and only in one of these cases was congestive cardiac failure a complication (Ponka et al, 1980; Florkowski et al, 1984; Cox et al, 2001; Wanby and Olsen, 2001). In a series of 342 patients with *C. jejuni* enteritis, myocarditis occurred in only two patients (Ponka et al, 1980).

To the authors' knowledge this is the first documented case with simultaneous evidence of myocarditis, hepatitis and renal failure, and ECG findings consistent with myocardial infarction. Despite marked ECG changes and moderate impairment in ventricular function, creatinine kinase (CK) did not rise greater than 692 iu/litre. While the time of onset of cardiac symptoms and the magnitude of CK elevation are consistent with other documented cases of *C. jejuni* myocarditis (Ponka et al, 1980; Florkowski et al, 1984; Cox et al, 2001; Wanby and Olsen, 2001), the marked acute inflammatory response (C-reactive protein (CRP) = 410 mg/dl) is inconsistent with other cases where CRP did not exceed 75 mg/dl (Cox et al, 2001; Wanby and Olsen, 2001). The 2-week delay between diarrhoeal symptoms and cardiac manifestations suggests an immune-mediated mechanism, although direct organism invasion or the presence of a myocardial toxin is possible.

Acute hepatitis is a rare complication of *C. jejuni*. There are four docu-

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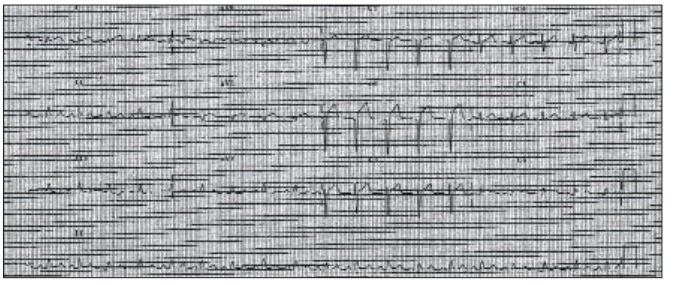

Figure 1. Admission electrocardiograph, showing ST elevation in chest leads V2 and V4.

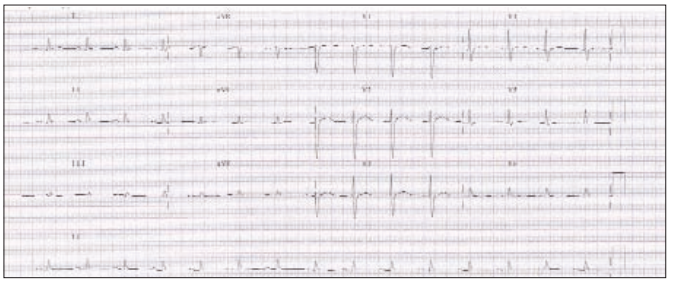

Figure 2. Electrocardiograph at follow up 3 months later. Admission changes resolved.

mented cases of acute hepatitis in this context. While it is possible that hepatitis in this patient was ischaemic in origin, two features suggest an infectious origin. First, individuals with ischaemic hepatitis tend to normalize transaminases within 5 days of correction of the hypotensive event. In this patient transaminase levels remained elevated for more than 8 days with an adequate blood pressure throughout his hospital stay. Second, patients with acute viral hepatitis tend to have a normal lactate dehydrogenase level, which contrasts with ischaemic hepatitis where levels are markedly elevated. Although the mechanisms for acute hepatitis are unknown, animal models of *C. jejuni*-induced hepatitis suggest the production of a hepatotoxin (Korman et al, 1997).

This patient's renal dysfunction cannot be directly ascribed to *C. jejuni* as renal investigations failed to suggest glomerular injury. Renal dysfunction was more likely to be secondary to cardiac failure.

It is difficult to comment on the long-term prognosis of *Campylobacter* myocarditis because of the limited number of cases, although all documented cases survived the acute phase of the illness with antibiotic therapy.

CONCLUSIONS

Myocarditis, hepatitis and renal disease are rare complications of *Campylobacter* infection. *Campylobacter* myocarditis may present with ECG changes similar to those seen in myocardial infarction. Response to antibiotics appears to be good although long-term prognosis is unknown. HM

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