Case Report

Campylobacter Jejuni Arthritis in Secondary Amyloidosis

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Summary We describe a case of infectious arthritis caused by C. jejuni which developed after C. jejuni enteritis in a patient with rheumatoid arthritis and hypogammaglobulinaemia. Extraintestinal infections of this enteropathogen are rare, but in patients with hypogammaglobulinaemia C. jejuni bacteriaemia is relatively common. The present patient suffered from hypogammaglobulinaemia due to a nephrotic syndrome in secondary amyloidosis. C. jejuni infection must be considered in patients with hypogammaglobulinaemia and arthritis.

Key words Infectious Arthritis, Campylobacter Jejuni, Rheumatoid Arthritis Hypogammaglobulinaemia, Amyloidosis

INTRODUCTION

Infectious arthritis is a well-known complication in rheumatoid arthritis (RA). In adults it is most frequently caused by Staphylococcus aureus (60%) and Neisseria gonorrhoea. Anaerobic pathogens are less commonly implicated, and include gram-negative cocci, Bacteroides fragilis and Fusobacterium (1). Infectious arthritis due to Campylobacter jejuni has been reported only once (2). Extraintestinal Campylobacter jejuni infections are rare and a relationship with hypogammaglobulinaemia has been suggested (3). We describe a Campylobacter jejuni arthritis in a patient with RA, AA amyloid with kidney involvement, nephrotic syndrome and hypogammaglobulinaemia.

CASE REPORT

A 70-year-old man presented with an acute arthritis of his right shoulder. Except for a 12-year history of seropositive, erosive RA for which he was treated with diclofenac and 10 mg methotrexate (MTX) weekly, his previous history was blank. In particular, no proteinuria or intestinal symptoms were ever present. Four days before admission he had eaten boiled chicken, and since the day before admission he had watery diarrhoea. During that evening the pain and swelling of the right shoulder developed within a few hours, and at night he had shaking chills for several hours. The temperature was not measured.

On examination, his temperature was 38.6 °C, blood pressure 130/80 mmHg, pulse 96/min. The right shoulder was red, severely swollen and painful. Many joints showed signs of severe destructive RA without active synovitis. Further physical examination was unremarkable except for peripheral oedema of the hands and the lower legs. X-ray of the right shoulder showed joint effusion, but no new bone or cartilage defects. The chest film was normal.

Laboratory evaluation demonstrated an erythrocyte sedimentation rate (ESR) of 86 mm/h, a white blood cell (WBC) count of 20,800/mm³ with 3% band forms and 55% neutrophils. Serum creatinine was 130 μmol/l, liver function tests were within the normal range. Serum albumin was 22 g/l (34-53 g/l), analysis of the protein spectrum showed a gammaglobulin of 6 g/l (8-18 g/l). Urine analysis demonstrated a proteinuria of 9-20 g/24h. Occult blood loss tests were positive. Arthrocentesis of the right shoulder yielded 750 ml of purulent fluid. WBC count and gram stain appeared to be impossible, due to WBC cloting. Four blood cultures remained sterile. Both cultures of the joint fluid and faeces twice identified C. jejuni type 1. The organism was sensitive to erythromycin and ciprofloxacin.

Treatment with ciprofloxacin 200 mg intravenously twice daily and repeated closed needle aspirations of the shoul-
were no signs of invasive behaviour of C. jejuni or amyloid depositions in the gut. The right shoulder and the joints were affected this time; a recurrent infection could not be demonstrated. Three days later the patient died from a hypovolemic shock. Postmortem examination revealed, besides the healed right shoulder and the joints destroyed by RA, a generalized deposition of amyloid in the kidneys and adrenal glands, dilated heart chambers and congestive lungs and intestine. In particular, there were no signs of invasive behaviour of C. jejuni or amyloid depositions in the gut.

DISCUSSION

The role of C. jejuni as important human enteropathogens is well established. Poultry is an important source of infection, and the typical manifestation of infection with this gram-negative microaerophilic curved motile bacilli is a self-limiting acute enteritis or enterocolitis. Bacteremia is rare and occurs mostly at the extremes of age. Extrainestinal manifestations have been noted in fewer than 1% of patients with C. jejuni infection, and include: cellulitis, cholecystitis, meningitis, peritonitis and retroperitoneal and thoracic abscesses, endovascular infections, urinary tract infections, Reiter's syndrome, reactive arthritis and Guillain-Barré syndrome. According to a recent review of extraintestinal Campylobacter infections, 52% of the patients had a predisposition for such infections, including biliary tract disease, first month of life, immunosuppression, chronic renal failure and hypogammaglobulinaemia. The pathogenesis of C. jejuni infections in hypogammaglobulinaemia is not well known but a failure of serum bactericidal capacity is probably crucial (9,10). Extrainestinal manifestations of C. jejuni are usually treated with erythromycin, quinolones, tetracycline, chloramphenicol or imipenem. The fluoroquinolones, as ciprofloxacin, are being advocated as alternative therapy in adults (11).

Both our patient and the only previous case of Campylobacter arthritis (2), suffered from RA and hypogammaglobulinaemia. RA is known as a risk factor for developing infectious arthritis, but it has been suggested that RA per se leads to an increased susceptibility to infection in general (12). A recent study however found no difference in the frequency or type of infection in RA patients compared to those patients with osteoarthritis (OA) or soft tissue rheumatism (13). Long-term administration of immunosuppressive drugs is said to be a risk factor in the development of infections in RA patients (14). Although the potential development of opportunistic infections, including Pneumocystis carinii, Cryptococcus, Nocardia and Herpes Zoster in RA patients treated with low dose MTX is well recognized even without receiving concomitant immunosuppressive therapy (e.g. corticosteroids), no Campylobacter infection has been described in association with MTX therapy (15). Therefore, it is reasonable to assume that in the present case the hypogammaglobulinaemia was the predisposing factor for the development of C. jejuni arthritis.

REFERENCES