CASE REPORTS

Campylobacter jejuni Infection with Acute Self Limiting Colitis and Polyarthritis

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Abstract

Acute self limiting colitis may mimic the first episode of ulcerative colitis on clinical, sigmoidoscopic and histological grounds. We present a case with Campylobacter jejuni colitis with self limiting polyarthritis treated initially for idiopathic ulcerative colitis and later successfully with chloramphenicol. Polyarthritis has not been reported before in association with Campylobacter colitis.

Key words: Campylobacter colitis, idiopathic ulcerative colitis, polyarthritis, acute self limiting colitis.

Introduction

Campylobacter fetus subsp. jejuni (C. jejuni) has recently been recognized as a human enteric pathogen. This organism can be isolated by selective culture of feces of over 7% of unselected patients with diarrhea and from less than 1% of asymptomatic persons. An incidence of 7%—16% among diarrhoeal stool samples has been reported from India. C. jejuni colitis has been known to mimic ulcerative colitis on clinical, sigmoidoscopic and histological grounds. However, no such Indian case has yet been reported. Further, acute reactive polyarthritis similar to that associated with other invasive bacterial diarrheaes (Shigella, Salmonella, Yersinia) may follow C. jejuni enteritis, 7 though this has not been reported with Campylobacter colitis. We report a case with Campylobacter colitis with polyarthritis.

Case Report

KJ, a 48 years old married male, was transferred to our hospital with a history of bloody diarrhea, irritable bowel pain and weight loss (4 kg) of 1 month's duration. Initially the patient had 12-15 motions per day, but the frequency decreased to 7 per day at the end of 15 days and it was 3-4 per day at the time of transfer to our hospital. He continued to pass frank blood per rectum. Three weeks after the onset of bloody diarrhea, he developed pain in all joints of the upper and lower limbs, except the shoulder joints. There was no history of concomitant arthritis, conjunctivitis or mucocutaneous lesions. He was treated initially with antiinflammatory drugs and then with oral steroids. As there was no significant response to treatment, he was transferred to our hospital.

On examination, he was pale and had clubbing. Per abdomen, tenderness in both iliac fossae was noted. All joints of the upper and lower limbs except the shoulder joints were clinically inflamed and had restricted movements. The spine was not involved clinically.

Investigations

Hemoglobin 15.3 g/dl, packed cell volume 41%, ESR 35 mm/1h, total WBC count 8,500/cmm (6,000 lymphocytes); stool routine showed RBCs and pus cells but no ova or parasites. Blood biochemistry was within normal limits. RPR was negative, antinuclear antibody negative. X-ray skeletal joints normal, HLA-B27 negative. Sigmoidoscopy on admission revealed friable mucosa, multiple superficial ulcerations and mucopus. A provisional diagnosis of ulcerative colitis with polyarthritis was made. He was started initially on steroid retention enema, oral steroids and salazopyrine, and phenylbutazone for arthritis. Biopsy showed focal transgressions of the mucosa, normal population of goblet cells, occasional crypts showing branching, mixed cellular infiltration of the lamina propria, and hyper trophy of the muscular mucosa (Fig 1). At this time did not suggest idiopathic ulcerative colitis, salazopyrine and steroids were withdrawn.

Fig 1: Rectal biopsy showing mucosal erosion, minimal crypt distortion and mixed cellular infiltration in lamina propria (H & E × 100).

A repeat sigmoidoscopy was performed a week later and biopsy taken, which showed similar changes as in the previous one. However, on this occasion, the patient developed sigmoid colon perforation for which an emergency exploratory laparotomy with primary suturing of the perforation and transverse colostomy was done. In the meantime, stool showed growth of C jejuni, sensitive to chloramphenicol, tetracycline, kanamycin, gentamicin, nalidixic acid and erythromycin. (The medium used was Ebro's medium with the following constituents—octyl BA base No 2 with 5-7% lysed horse blood and vancomycin 10 mg/l, polymyxin B sulphate 2500 IUI and trimethoprim lactate 5 mg/l). The patient was treated with parenteral chloramphenicol. Colostomy was closed three weeks later. By this time the polyarthritis resolved. Three weeks after colostomy, a repeat sigmoidoscopy showed normal mucosal biopsy taken at this time was also normal (Fig 2). Stool culture repeated for C jejuni was negative.
Discussion

The prominent clinical feature in previous reports of Campylobacter infections has been small bowel type of diarrhoea; this was supported by finding ileitis on laparotomy or autopsy. An appreciable proportion of patients (14%) have been reported to have colitis.

A proportion of cases who in the past were diagnosed as ulcerative colitis but had no further episodes of colitis could possibly have been due to infections. Campylobacter fetus being one of them. Our patient had clinical and sigmoidoscopic features resembling ulcerative colitis. The presence of joint involvement strengthened the suspicion. The presence of large watery motions as early symptoms however needed explanation. The first sigmoidoscopic biopsy did not rule out ulcerative colitis since the first biopsy in this condition may be fallacious and demonstration of persistent features such as crypt inflammation, crypt branching, mucosal gaps and muscularis mucosai gaps during a follow up may be necessary to make a firm diagnosis.

When the second biopsy showed no such features a diagnosis of acute self limiting colitis (ASLC) was considered. The growth of C jejuni on stool culture explained why he had clinical features of involvement of both small and large bowel. Though such a presentation is well documented with C jejuni infection, no such case has been reported from our country before. With adequate antibiotic treatment the patient improved clinically and sigmoidoscopically and the rectal biopsy returned to normal.

Another important feature which has not been commonly reported with C jejuni infection is polyarthritis. Our patient presented early with polyarthritis, which lasted for 4 weeks. Investigations ruled out other causes of this condition.

References