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Case report

Campylobacter jejuni enteritis and reactive arthritis

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SUMMARY A further case of reactive arthritis following *Campylobacter jejuni* enteritis is reported. The interim results of a small prospective study are discussed. It may be desirable to do serological studies for campylobacter infection in the investigation of mono- or polyarthritis of acute onset.

The original report of a reactive arthritis following enteric infection with *Campylobacter jejuni* was in 1979 from Berden *et al.*[1] in the Netherlands. Subsequently Weir *et al.*[2] described 2 patients in whom an arthritis developed following campylobacter enteritis, although *C. jejuni* was specified in only one. Recently, limited information has been published from Kosunen *et al.*[3] in Finland describing this condition in a further 8 patients, although there could well be doubt about the diagnosis in some of the Finnish patients, 2 of whom had concurrent infection with species of salmonella. If the diagnosis is accepted for these 10 cases, 6 have been HLA B27 positive, 3 negative and one not tested.

We should like to report a further patient with this condition, who also showed some features well recognised in the reactive arthropathies but as yet unreported in this specific form.

Case report

In June 1979 a 31-year-old woman, a secretary, presented with a 24-hour history of pleuritic-type pain in the left upper chest and shoulder, and diarrhoea with frequent, loose, and watery stools, containing no blood or mucus. Examination was normal except for a temperature of 38·5°C, which settled in 24 hours. The diarrhoea resolved in 48 hours with the administration of dihydrocodeine tartrate. Haematological, biochemical, radiological, virological, and bacteriological investigations at this time were all normal apart from the isolation of *Campylobacter jejuni* in the stools. No cause for the chest pain was found.

Three weeks later she was readmitted with a 2-week history of pain and swelling in the right knee. There was no history of trauma or previous arthritis. The knee was warm with a marked effusion, but the patient was afebrile. Clear, straw coloured fluid was easily aspirated from the affected joint. The following investigations were performed: haemoglobin 13 g/dl; white cell count 7×10^9/l; ESR, 66 mm in the first hour, and this remained raised for many weeks; liver function tests normal; anti-DNA antibody levels normal; SCAT, latex, and ANF tests negative on three separate occasions over the ensuing 8 weeks; LE cells not seen; serum uric acid 0·18 mmol/l (normal less than 0·39 mmol/l). Wasserman reaction and gonnococcal complement fixation tests were negative both at the time of the effusion and when repeated 4 months later. Stool culture was negative; hepatitis B surface antigen negative; serum rubella antibodies no rise. Microscopy of the joint fluid revealed no crystals and only a few polymorphonuclear leucocytes, lymphocytes, and histiocytes, and on culture there was no bacterial growth. The latex test on the fluid was negative, but the SCAT gave a result of greater than 256 international units. Slit-lamp examination of the eyes revealed no uveitis. Radiographs of the knees and feet showed possible erosions of the affected joints; radiographs of the pelvic bones showed sclerosis of the left sacroiliac joint. The patient was HLA B27 antigen positive. Agglutination tests on sera from the acute enteritic, acute arthritic and convalescent

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phases against *C. jejuni* serotype 6 (Public Health Laboratory Service, Withington, Manchester) gave results of less than 1 in 10, rising to 1 in 320, and falling again to 1 in 20 in the respective samples; complement fixation tests on the same samples showed no significant change.

The acute phase of the illness led a protracted course over 7–8 weeks, during which time our patient developed symptoms and signs of involvement of the left second metatarsophalangeal and left tibiofibula joints, and at the end of the second month the ESR had only just started falling to 30 mm/hour, although the effusion had resolved. She had been treated with a one-week course of oxytetracycline during the initial stages of the illness but otherwise only with non-steroidal anti-inflammatory drugs. By the fourth month she was symptomatically no better, complaining of widespread intermittent arthralgias, but with no real objective signs to be found, and by now the ESR was quite stable at around 5 mm/hour. It was not until about one year from the onset of her symptoms that she felt she had almost fully recovered.

FURTHER STUDIES

For an 8-month period from June 1979 all patients and staff of this hospital having diarrhoea and *C. jejuni* isolated from stool culture were contacted, and 15 of the 18 were seen and examined 3 times during the 6 weeks following isolation of the organism. Ten were hospital employees, but time and space distribution did not suggest a common source. None of the 15 developed arthritis, urethritis, or uveitis. All were HLA B27 negative.

Discussion

The evidence that *C. jejuni* was responsible for the reactive arthritis in our patient is substantial. The other 2 patients who have been reported in detail[1 2] presented with polyarthritis which recovered fully after 8 and 2 weeks respectively, and had no reported radiological abnormalities. Our patient presented with a monoarthritis, had radiological evidence of sacroiliitis, and had a more prolonged illness. Keat *et al.*[4] suggested that a protracted course in the reactive arthropathies is commoner in HLA B27 positive patients, although this has been disputed by Calin and Marks.[5] Of the 2 patients referred to above, one was HLA B27 positive and the other negative.

In our subsequent study over 50% of the patients were hospital personnel. In many cases the symptoms were mild and of short duration, and it is probable that stool cultures were performed only because they worked in hospital. This observation, taken in conjunction with the report by Skirrow[6] that *C. jejuni* is the commonest bacterial cause of diarrhoeal illness, suggests that enteritis due to this organism may be common in the community and yet not specifically diagnosed.

We suggest therefore that *C. jejuni* be considered in the aetiology of an arthritis following even a mild enteric infection, and, although the serological diagnosis of campylobacter infection may be difficult,[7] nonetheless this test may be justified in the investigation of acute mono- or polyarthritis.

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References

1 Berden J H M, Muytjens H L, van de Putte L B A. Reactive arthritis associated with *Campylobacter jejuni* enteritis. *Br Med J* 1979; **i:** 380–1.

2 Weir W, Keat A C, Welsby P D, Brear G. Reactive arthritis associated with campylobacter infection of the bowel. *J Infection* 1979; **1:** 281–4.

3 Kosunen T U, Kauranen O, Martio J, *et al.* Reactive arthritis after *Campylobacter jejuni* enteritis in patients with HLA B27. *Lancet* 1980; **i:** 1312–3.

4 Keat A C, Maini R N, Pegrum G D, Scott J T. The clinical features and HLA associations of reactive arthritis associated with non-gonococcal urethritis. *Q J Med* 1979; **48:** 323–42.

5 Calin A, Marks S H. Reactive arthritis. *Br Med J* 1980; **281:** 311–2.

6 Skirrow M B. Campylobacter enteritis: a 'new' disease. *Br Med J* 1977; **ii:** 9–11.

7 Watson K C, Kerr E J C, McFadzean S M. Serology of human campylobacter infections. *J Infection* 1979; **1:** 151–8.