Reactive arthritis associated with campylobacter enteritis

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SUMMARY Campylobacter enteritis is a known aetiological factor in reactive arthritis. We surveyed patients in the Harrow Health District known to have had campylobacter enteritis for manifestations of arthritis. Acute attacks of arthritis occurred in 8 of 33 adults admitted with enteritis. None were identified in patients under 16 or in those diagnosed in the community.

Reactive arthritis is well known to occur after enteritis caused by salmonella, certain shigella species and Yersinia enterocolitica, and after generalised infections such as meningococcaemia. Exacerbations of Reiter's syndrome have recently been linked to Campylobacter fetus infections,\textsuperscript{1} and subsequently reactive arthritis with \textit{C. jejuni} enteritis.\textsuperscript{8} In reactive arthritis an acute arthritis is associated with infection elsewhere in the body without evidence of infection in the joints. To estimate the frequency with which reactive arthritis is associated with diarrhoea caused by campylobacter species we used the computerised records of the Department of Microbiology at Northwick Park Hospital as an unbiased source of patients.

Materials and methods

The computer identified 77 patients in whom campylobacter species were isolated from their stools in 1978; 47 were hospital inpatients, and 30 had specimens sent in by their general practitioners. The hospital notes of all 33 identified adult inpatients (aged over 16 years) as well as those of 9 younger inpatients (aged 5–16) were searched for evidence of joint or spinal complaint. The general practitioners of 28 community patients were identified by the computer, and they were asked whether any joint or spinal problems had occurred at or about the time of the campylobacter infection. Nineteen replies were received.

Results

Possible episodes of reactive arthritis had occurred in 8 of the 33 adult inpatients. In 2 patients the episode had been sufficiently severe to require a rheumatological consultation; in the remaining 6 the episodes had been symptomatic only, involving peripheral joints in 4 and lower back pain in 2. No arthritis was noted in the 19 community patients.

CASE 1
A week after a conference in Norway a 36-year-old female research worker developed profuse watery diarrhoea. A week later her neck, wrists, toes, and hands were affected by arthritis. A rheumatologist noted bilateral knee effusions, a swollen wrist, an erythematous and tender interphalangeal toe joint, and tenderness over both sacroiliac joints. A diagnosis of reactive arthritis was made. The symptoms gradually settled over 3 weeks, but one knee was still swollen 1 month later. The patient was virtually symptom-free 6 months later. Histocompatibility typing did not show HLA B27.

CASE 2
A 55-year-old man was admitted with an acutely painful swollen knee, having had intermittent watery diarrhoea for 6 weeks associated with lower abdominal cramping pain. The knee was hot and sufficiently swollen to be held in flexion. In addition a rheumatologist noted a definite popliteal cyst, which may have ruptured into the calf. Diagnoses entertained were reactive arthritis or a calcium pyrophosphate crystal synovitis. Extensive chondrocalcinosis was seen on the radiograph, but no crystals were seen in the synovial fluid, which was sterile. One month later the patient was free of symptoms.

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Discussion

Estimates of the frequency with which certain infections are complicated by reactive arthritis are fraught with difficulty, and the subject was recently reviewed by Masi. In brief, the frequency after salmonella enteritis varied from 1.6% to 9.5% and after yersinia infection 33%. A more recent estimate by Larsen was of 188 cases of acute arthritis in 13,000 patients referred for antibody tests to Yersinia enterocolitica. In a different context, of reactive arthritis following meningococcal infection, Greenwood and Whittle found a higher proportion of patients with reactive arthritis amongst the adults. From our survey reactive arthritis may complicate campylobacter enteritis in a frequency comparable to salmonella infections. To identify these cases stool cultures should be so performed that campylobacter species may be isolated.

The histocompatibility antigen HLA B27 is known to increase the likelihood of reactive arthritis or Reiter’s syndrome occurring, and also the severity and chronicity of the episode. Calin and Fries estimate that in a shigella epidemic between 16 and 37% of HLA B27 positive people affected will develop Reiter’s syndrome. In this context it is of interest that our first patient did not have HLA B27, contrasting with the case reported by Berden et al.

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References