Case report

Reiter's syndrome in association with enteritis due to *Campylobacter fetus* ssp. *jejuni*

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SUMMARY An 18-year-old girl with Reiter's syndrome after febrile diarrhoea was found to be infected with *Campylobacter fetus* ssp. *jejuni*. Excretion of campylobacter into stools was stopped by erythromycin therapy, but active polyarthritis lasted for 6 months. The patient was positive for HLA B27.

The past few years have witnessed increasing interest in the study of campylobacter infection, mostly due to the observation of Skirrow¹ in 1977 that campylobacter is a relatively common cause of human diarrhoea. Two species of campylobacter are known to cause diseases in man: *Campylobacter fetus* ssp. *jejuni*, an intestinal pathogen, and *C. fetus* ssp. *intestinalis*, which causes septic infections and nonintestinal infections, especially in persons with subnormal resistance.²

Like enteritis due to salmonella, shigella or *Yersinia enterocolitica*³⁻⁵ that due to *Campylobacter fetus* ssp. *jejuni* may also be followed by reactive arthritis. Altogether 12 such cases have been described.⁶⁻¹⁰ In addition Urman *et al.*¹¹ have reported an exacerbation of recurrent Reiter's syndrome with *C. fetus* infection. However, they did not give information about the detailed classification of the bacterium.

We report here what we believe to be the first case of Reiter's syndrome due to campylobacter classified as *Campylobacter fetus* ssp. *jejuni*. This patient is one of 8 patients with arthritis briefly reported on recently.¹⁰

Case report

An 18-year-old schoolgirl had an aunt with classical rheumatoid arthritis. Other relatives had no rheumatic diseases. She herself had been very healthy,

and entirely without joint symptoms. In September 1979 she had acute diarrhoea, with abdominal pain and fever. These symptoms lasted for 5 days. She had no known contact with another case of diarrhoea, nor had she been abroad before the illness.

One week after the beginning of the disease she noticed swelling of the first right metatarsophalangeal joint. Five days later swelling and tenderness appeared in the fingers of the right hand, and the patient was admitted to hospital for 2 weeks. At the same time lacrimation and conjunctival redness affected both eyes for 5 days. No urinary symptoms or dermatitis appeared. Ten days after the first joint symptoms she had swelling of the right ankle and parasternal pain. During the following weeks some of these symptoms subsided, but the condition of the joints was worst during November and December. At this time she had swelling of the whole right foot, hydrops of the right knee, severe tenosynovitis of the right hand, and pain in the left sacroiliac joint. Active polyarthritis lasted until March, i.e., for about 6 months. She had sulindac as an analgesic drug and a course of prednisolone, 5 mg daily, from December to February.

The erythrocyte sedimentation rate was 72-95-65-31 mm/h during the polyarthritis, and 12 mm/h when the joint symptoms subsided and treatment was stopped. The white blood cell count on admission was $8.9 \times 10^9/l$ ($8900/mm^3$). At the beginning of the disease, just after the conjunctival symptoms disappeared, transient proteinuria and pyuria were found, but there were no bacteria in the urine. In the synovial fluid the leucocyte count was $19 \times 10^9/l$

Accepted for publication 20 August 1980

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(19 000/mm³); 80% were polymorphs. Synovial fluid protein was 5.1 g/l. Bacterial culture of the synovial fluid was negative, but no specific culture was done for campylobacter. Tests for rheumatoid factor (Waller-Rose and latex) were negative, and no rise was found in antibody titre to streptolysin O, nor in titres for *Yersinia enterocolitica* (agglutination test), *Chlamydia trachomatis* (immunofluorescence test), or *Neisseria gonorrhoeae* (complement fixation test). Stool cultures were negative for *Yersinia enterocolitica*, salmonella, and shigella, but there were several positive findings for *Campylobacter fetus* ssp. *jejuni*. One week after admission the patient was given erythromycin for 10 days, 1 g daily, and the campylobacter disappeared. Agglutinating antibody titres for formalinised suspension of this organism were raised, 1/200, 3 weeks later 1/50. Typing for HLA antigens showed A1, A2; B27, w4, w6; Cw2. Radiographs of the symptomatic joints revealed only soft-tissue swelling; the sacroiliac joints were normal too. The electrocardiogram was normal.

Discussion

The complete clinical triad of Reiter's syndrome includes arthritis, conjunctivitis, and nongonococcal urethritis. The histocompatibility antigen HLA B27 has been found in 63–96% of patients with Reiter's syndrome.^{12–15}

Our patient had polyarthritis, conjunctivitis, and signs of urinary tract infection, and she was positive for HLA B27. The role of *Campylobacter fetus* ssp. *jejuni* in the aetiology of the case was obvious. The patient had diarrhoea preceding arthritis, and the organism was isolated from the stools on several occasions. Agglutinating antibodies were found in the acute sera, diminishing later. Stool cultures for salmonella, shigella, and *Yersinia enterocolitica* were negative. Tests did not show antibodies for *Chlamydia trachomatis* or *Neisseria gonorrhoeae*, nor for rheumatoid factors; and radiographs of the sacroiliac joints were normal.

It is possible that some cases of Reiter's syndrome after enteritis of unknown aetiology have been

caused by *Campylobacter fetus* ssp. *jejuni*. The recent discovery of this pathogen may rather specific isolation techniques necessary may explain the absence of earlier reports. In cases of Reiter's syndrome after intestinal symptoms we suggest that, in addition to serological and bacteriological examinations for salmonella, shigella, and *Yersinia enterocolitica*, tests should also be made for *Campylobacter fetus* ssp. *jejuni*.

This study was supported by grants from the Sigrid Jusélius Foundation, Helsinki.

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