A patient with previously diagnosed ankylosing spondylitis developed a first episode of peripheral synovitis following enteritis with *Salmonella* group D. Ankylosing spondylitis and reactive arthritis are HLA-~B~27-related conditions and flare-ups have been linked to alterations in bowel flora. This case report supports recent suggestions that peripheral synovitis in ankylosing spondylitis is a form of reactive arthritis.

CASE HISTORY

A 44-year-old school teacher presented with acute synovitis of both knees and left wrist shortly after returning from holiday with his family. Two weeks earlier, while in Portugal, he had developed mild watery diarrhoea with minimal systemic upset twelve hours after eating barbecued chicken. There was no recent history of urethritis, conjunctivitis or back pain, but his joint complaints were severe enough to keep him from work. Fourteen years previously, he had sought specialist advice because of pain and morning stiffness in the low back and right hip. X-rays revealed bilateral sacroilitis and symmetric syndesmophyte formation in the upper lumbar spine. A diagnosis of ankylosing spondylitis was made and he was initially treated with phenylbutazone. He has subsequently had intermittent exacerbations of low back symptoms responding to short courses of anti-inflammatory drugs, but denied persistent stiffness.

Physical examination four weeks after the onset of diarrhoea revealed painful bilateral knee effusions, worse on the left, and a tender, swollen left wrist. There was loss of the lumbar lordosis but normal lower lumbar movements, and no peripheral enthesitis. He had no skin rashes, mucocutaneous lesions or ocular inflammation. Investigations showed haemoglobin 14·6 g/dl and white cell count 9·8 x 10^9/l with normal differential white cell count. ESR and C reactive protein were moderately raised at 38 mm/hr and 31 mg/l respectively. Tests for rheumatoid factor were negative, no autoantibodies were detected in the serum, and tissue typing was positive for HLA-~B~27. Antibodies to *Yersinia enterocolitica* and *Campylobacter spp.* were not detected. Inflammatous synovial

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fluid was aspirated from the right knee (WCC 10.6 × 10⁹/1) but no organisms or crystals were detected. Stool culture revealed *Salmonella enteritidis* and antibodies to salmonella somatic antigens were detected in high titre (1280). Radiographs of peripheral joints were normal. There was anterior syndesmophytic bridging in the upper lumbar and lower thoracic regions and considerable narrowing of the sacroiliac joints. The patient was treated with bed rest, wrist splint and indomethacin. One month later his symptoms had lessened although he still reported occasional peripheral joint pain. At four months there were no joint complaints but intermittent loose stools persisted, and stool culture remained positive.

**DISCUSSION**

The pattern of asymmetric, oligoarticular synovitis occurring two weeks after the onset of salmonella enteritis is typical of 'reactive arthritis'. The concept of reactive arthritis is now well established and refers to an aseptic inflammatory peripheral arthritis following infection at a distant site in subjects who usually possess the histocompatibility antigen HLA-B27. Enteric organisms including salmonella, shigella, yersinia and campylobacter are amongst the commonest inciting agents. Reactive arthritis occurs in up to 9.5% of individuals following salmonella outbreaks, most usually with *S. typhimurium* and *S. enteritidis*.

In the present case, reactive arthritis occurred in a patient with pre-existing ankylosing spondylitis. Only two previous reports have documented proven reactive arthritis in such patients, but in neither was the diagnosis of ankylosing spondylitis established (by rheumatological consultation and X-ray) years prior to the reactive episode. Peripheral joint synovitis is a recognised feature of ankylosing spondylitis and may run a clinical course indistinguishable from reactive arthritis. Alterations in bowel flora, histological features of inflammation in the ileocolic region and alteration in bowel permeability have been linked to episodes of peripheral synovitis in patients with ankylosing spondylitis. It has therefore been postulated that it is a form of reactive arthritis usually arising from sub-clinical infection with an enteric organism which alters bowel permeability and possibly allows penetration of exogenous antigen. An analogous situation may occur in patients with inflammatory bowel disease in whom flare-ups of bowel symptoms accompany episodes of inflammatory peripheral joint synovitis.

The finding of peripheral joint synovitis triggered by salmonella infection in this patient with ankylosing spondylitis lends support to the concept that bowel infections may not only lead to the development of reactive arthritis in previously healthy individuals but may also provoke a flare-up of pre-existing spondyloarthropathy. Recent reports of the therapeutic benefit of sulphasalazine in the treatment of HLA-B27-related arthropathies may therefore be due to effects on bowel flora and, if confirmed, may suggest new therapeutic approaches in reactive arthritis.

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