and tests for rheumatoid factor, antinuclear factor, and antibodies to DNA were negative. Liver function tests were normal. After a short course of systemic and intra-articular steroids the skin rash resolved and joint pain improved.

She was readmitted in April 1990 with a recurrence of arthropathy, rash, and fever with a leucocytosis (white cell count 13.9 × 10⁹/l), raised CRP (226 mg/l), and plasma viscosity of 2.36 mPa.s. She complained of lower abdominal pain and developed peritonitis. A limited laparotomy showed sterile peritonitis. Systemic lupus erythematosus was considered unlikely, and an ultrasound of the kidneys and a diagnosis of adult onset Still's disease was made. She responded to high dose corticosteroids, and sulphasalazine was introduced. By discharge the CRP had fallen to 1 mg/l, and when taking sulphasalazine 500 mg twice daily, she was not to be jaundiced. Investigations showed an acute hepatitis reaction (aspartate 559 IU/l, bilirubin 57 µmol/l, alkaline phosphatase 793 IU/l, γ-glutamyltransferase 163 IU/l, CRP 62 mg/l), which resolved within two weeks of discontinuing sulphasalazine. No other cause for hepatitis was found. The patient has remained well on a reduced dose of corticosteroid.

This case fulfils the criteria for adult Still's disease proposed by Medger and Christy. The hepatitis which developed in our patient was a true sulphasalazine, and to the best of our knowledge this is the first reported case of sulphasalazine induced hepatitis in adult Still's disease, though from experience with children sulphasalazine sensitivity in Still's disease may be more common than expected. Acetylator status is a major determinant of sulphasalazine pharmacokinetics, but sulphasalazine also undergoes hydroxylation metabolism. The reduction in hepatic cytochrome P450 induction may occur as a consequence of inflammatory arthritis. Prolongation of sleep times induced by barbiturates (metabolised by P450 pathways) occurs in rats with adjuvant arthritis, and a role for interleukin 1 in depression of drug metabolism has been proposed. This cytokine is implicated in the pathogenesis of erosive arthropathies. Furthermore, fever induced by the administration of etiocholanolone or other pyrogens is associated with a decrease in drug metabolism. Thus these studies support the hypothesis that interleukin 1 released during adult onset Still's disease might be capable of reducing hepatic cytochrome P450. Such a mechanism might account for an alteration in drug metabolism and an increased potential for adverse drug reactions.

The Achilles tendinitis subsided after eight months from onset. The bilateral Achilles tendinitis shown by our patient is consistent with a B27 associated disease process, and the clinical and echo- graphic aspects are shown to be typical. This case supports our hypotheses about B27 associated peripheral enthesitis—namely, that (a) peripheral enthesitis may for a long time be the only clinical manifestation, and as is true for other clinical features of the B27 associated disease process—that is, peripheral asymmetric oligoarthritis, dactylitis, acute anterior non-granulomatous uveitis, and aortic regurgitation and conductive cardiac murmurs; (b) it may occur either in childhood or in adulthood; (c) Achilles tendinitis may be the only isolated peripheral enthesitis of spondyloarthropathy.

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Longstanding HLA-B27 associated Achilles tendinitis

Sir: In 1987 we published the case of a B27 positive male who had presented for about four years with bilateral Achilles tendinitis as the only clinical manifestation of a B27 associated disease. We have followed this up since his first visit in December 1986. The Achilles tendinitis went into remission in 1988. No other clinical manifestation of spondyloarthropathy had developed so far. We have recently seen a similar case, which we report here. A 36 year old man was evaluated at our rheumatic disease unit in November 1990 for bilateral Achilles tendinitis which had persisted for four months. His medical history showed that he had had a similar episode in 1983, lasting for one month, which was attributed to 10 days' skiing. He denied any inflammatory spinal pain, peripheral arthritis, diarhoea, urticaria, conjunctivitis, uveitis, psoriasis, cardiac symptoms, or physical injury. His family history was negative for spondyloarthropathy and other B27 associated syndromes.

Physical examination showed warmth, tenderness, and soft tissue swelling along both Achilles tendons and their calcaneal insertions. There was no limitation of spine movement or chest expansion. Laboratory evaluation showed a C reactive protein of 12 mg/l (normal <5). HLA typing showed the B27 antigen.

Ultrasoundography by the technique of Fornage showed a diffuse thickening of both Achilles tendons, which was more severe on the right side (9 mm v 8 mm (figure)). Sacrificio joint, lumbar spine, and foot radiographs were normal.
Longstanding HLA-B27 associated Achilles tendinitis.

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