with RA (three with corresponding clinical tenosynovitis). On the other hand, one of seven controls and eight of 15 patients with RA with clinical tenosynovitis did not present this special isotopic picture. In the controls a false positive isotopic uptake was noted in 35% and a false negative in 3% of the joints when compared with joint pain.

There was only low agreement between joint pain and the radiological score in patients with nodular osteoarthrosis (42%). For patients with RA the pain and swelling scores were in total agreement in 66% of the joints. These features have been well reported.

When the nanocolloid score was plotted against the pain score the total agreement was 49% for the joints of all subjects (fig 2), 59% for those with nodular osteoarthrosis, and 41-5% for the patients with RA. When the nanocolloid score was plotted against the radiographic score the results were 39% for the joints of all subjects, 29% for those with nodular osteoarthrosis, and 31% for the patients with RA. The correlations between the nanocolloid score and the clinical and radiographic scores were always less than 0·2, which is not significant.

In the patients with RA 72% of the painful joints showed an increased isotopic uptake, but only 36 of the 56 joints (64%) which were simultaneously painful and swollen showed an enhanced nanocolloid uptake. Moreover, of the 201 neither painful nor swollen joints and of the 77 which were not painful, swollen or radiologically abnormal 99 (49%) and 32 (42%) respectively were positive on the scintigram. When the nanocolloid and diphenophophonapendicarboxylic acid scintigrams were compared agreement was 75% (85 joints positive on both scans, 15 negative on both scans, 14 discordant). The association was not specifically in favour of one or other of the techniques (16 joints positive and 16 joints negative with nanocolloid scintigraphy were negative and positive respectively with diphenophonapendicarboxylic acid scintigraphy). The conclusions of this pilot study seem to be first that labelled nanocolloid can effectively accumulate in inflammatory joints and in tenosynovitis. Nevertheless, it is also retained in many normal joints (35%) from controls and in joints from patients with RA (42%) without evidence of progressive deterioration. This might be secondary to an isotopic leakage before clinical expression, which was not investigated by a longitudinal study, but the percentage of these positive scintigrams seems too high to be explained by this hypothesis alone. Why there was no nanocolloid uptake in 28% of patients with RA with painful and swollen joints needs further investigation. Finally, in the few patients with RA for whom both scintigrams were obtained nanocolloid was no better than diphenophophonapendicarboxylic acid. So, despite being an inexpensive, simple, and straightforward method nanocolloid scintigraphy does not supply the rheumatologist with a detector of early inflammation. The results of our study are in agreement with those of Rüther et al.1

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Spondyloarthropathies and IgA deficiency

Sir: Herrera-Beaumont et al recently reported a female patient with IgA deficiency who developed ankylosing spondylitis characterised by a widespread erosive peripheral arthritis and recurrent anaemia of chronic disease.1 They cited three additional reported cases, all with severe disease, and suggested that IgA deficiency is a marker of poor prognosis in the spondyloarthropathies, possibly warranting replacement therapy. We describe four patients with IgA deficiency and a spondyloarthropathy who had variable disease severity.

A 24 year old man presented in 1980 with a three month history of dysuria, early morning stiffness, back pain, and swelling of his knee. Examination showed balanitis and an active arthritis of the left knee. Chlamydia trachomatis was cultured from his urethra and he received tetracycline in addition to indomethacin. The IgA concentration was 52 IU/ml. Symptoms were well controlled with aspirin and exercise. There was radiological evidence of sacroiliitis three years after onset and when last seen in the clinic in 1988 he remained well and active with no deformity.

In 1987 a 26 year old man developed arthritis of the left knee complicated by a ruptured Baker's cyst. This progressed over 18 months to affect his right wrist, both elbows, and lumbar spine. IgA was undetectable in both serum and synovial fluid. His HLA haplotype was A2, B5, B12 (44), DR4, DR5. Diclofenac, sulphasalazine, and repeated intravenous corticosteroid therapy failed to control his arthritis satisfactorily.

The third patient, a 23 year old man, presented in 1989 with arthritis in both knees, bilateral sausage digits, and conjunctivitis following an episode of dysuria. He was HLA-B27 positive with an IgA concentration of 47 IU/ml. Diclofenac and, later, sulphasalazine for six months failed to control persistent arthritis in his right knee.

Recently, a 24 year old man with an IgA concentration of 38 IU/ml and negative HLA-B27 was seen. He presented with a nine year history of inflammatory back pain and early morning stiffness, which responded to non-steroidal drug treatment.

In contrast with the patient reported by Herrera-Beaumont et al,1 our first and last patients had mild disease, whereas the other two patients had intractable peripheral arthritis. IgA concentrations are often raised in ankylosing spondylitis,2 and clinical improvement has been associated with falling levels.3 These patients, however, show that IgA is not necessarily associated with the pathogenesis of the spondyloarthropathies.

IgA deficiency is common, and a true association between IgA deficiency and spondyloarthropathies has not been established. If there is an association this may be, directly, due to lack of IgA on mucosal surfaces4 or, indirectly, by association with disease susceptibility genes.5 Our patients’ data do not support the suggestion that IgA deficiency is a marker of disease severity.

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