swelling around the third and fourth finger on both sides. There was a slight joint space narrowing of the proximal interphalangeal joint of the right middle finger and rather extensive degenerative changes of the interphalangeal joint of both thumbs, including irregular joint space narrowing, osteophytes, and cysts (fig 1). A synovial biopsy showed an aspecific chronic inflammation. No amyloid or iron was present. Without specific treatment the complaints gradually lessened.

In 1989 she attended the outpatient clinic of the department of rheumatology again, with increasing pains in her hands. At that time she was treated with azathioprine (100 mg daily), prednisone (alternating 7.5 and 10 mg daily), and atenolol (50 mg daily). On examination there was a bony swelling of the interphalangeal joint of both thumbs and a telescopic shortening of the left ring finger and the right little finger. Furthermore, there was a tenosynovitis of musculus extensor digiti proprii on the right side. Laboratory investigation showed an erythrocyte sedimentation rate of 10 mm in one hour, normal peripheral blood counts, serum calcium, and parathyroid hormone concentrations. Rheumatoid factors were negative. HLA typing: A10, A26, A29, B8, Bw72, Cw6, DR4, and DR7. Radiographs of her hands showed extensive erosive changes, predominantly in the proximal interphalangeal joint of the third and fifth digit of the left hand and the fourth digit of the right hand (fig 2).

Our patient developed a destructive arthropathy after a successful renal transplantation. The clinical and radiological picture closely resembled the changes observed in patients with psoriatic arthritis, but she did not have any skin lesions. As azathioprine and prednisone were used in this treatment she wished to cancel the study. Furthermore, she may develop skin lesions in the future as some patients with psoriatic arthritis show the first skin lesions up to 15 years after the joint complaints.1 Psoriatic arthritis is associated with (among others) HLA-Cw6 and DR7.4,5 HLA-DR4 has been shown to be associated with the development of erosions.3 All three histocompatibility antigens were present in our patient. In their article, Choi et al did not discuss the radiological differential diagnostic problems of erosive osteoarthritis and psoriatic arthritis. Both are erosive polyarthritides with prominent interphalangeal joint involvement, often resulting in bony ankylosis.6,7 Psoriatic arthritis characteristically gives asymmetrical ill defined erosions, unaccompanied by significant osteoporosis and associated with extensional subluxation on X-ray. Erosive osteoarthritis is characterised by symmetrical joint involvement, subchondral erosions, and more subtle linear periosteal bone apposition.

Our patient developed extensive erosions during a clinically quiescent period. These findings are similar to those of the long term studies on the effect of second line treatment in rheumatoid arthritis: the patients showed a clinical and haemato logical response, but no changes in radiological progression.4 In conclusion, the patient presented has an erosive arthropathy after successful renal transplanta tion, both clinically and radiologically almost indistinguishable from psoriatic arthritis.

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Figure 1: Hand radiograph.

Figure 2: Hand radiograph.

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Lack of serum antibodies to native type II collagen in leprosy
Sir: We were most interested in the finding of antibodies to native type II collagen in lepromatous leprosy reported by Choi et al.1 They specifically looked at lepromatous leprosy because of the high titre antibody to Mycobacterium leprae and the apparent lack of joint involvement in this disease. This last presumption is incorrect as a rheumatoid-arthritis-like syndrome with nodules has been described in this disease.2 We wished to confirm these findings by looking at patients with leprosy who had definite arthritis. If verified, we wondered whether there might be a cross-reactivity between epitopes on Mycobacterium leprae and native type II collagen. This would have important implications for the pathogenesis of rheumatoid arthritis (RA).

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Serum antibodies to native type II collagen and denatured type II collagen, and enzyme linked immunosorbent assay (ELISA)

<table>
<thead>
<tr>
<th>Antibody to (units/ml)</th>
<th>Nat II*</th>
<th>Denat II*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper limit of normal</td>
<td>&lt;198</td>
<td>&lt;716</td>
</tr>
<tr>
<td>0-50</td>
<td>500</td>
<td>1000</td>
</tr>
<tr>
<td>RA* (positive control)</td>
<td>1434</td>
<td>1616</td>
</tr>
</tbody>
</table>

*Nat II = native type II collagen; Denat II = denatured type II collagen; RA = rheumatoid arthritis.

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We obtained serum samples, from 10 patients (nine male, one female, aged 23-60 years) with leprosy (seven lepromatous leprosy, two tuberculoid leprosy, and one borderline), all with joint involvement (both large and small joints in five, small joints only in three, and large joints only in two). Five were receiving regular treatment, three had taken intermittent treatment, and two were untreated.

Serum antibodies to native type II and denatured type II collagen were measured by enzyme linked immunosorbent assay (ELISA).3 Serum samples from 22 healthy controls were used to determine an upper limit of normal as three standard deviations above the mean. A known high positive control (RA) was included. The results are shown in the table.

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None of our patients had raised serum antibodies to native type II collagen, though three patients had slightly raised levels to denatured type II collagen (757, 814, and 1001 units/ml). Our results for native type II collagen therefore disagree with those of Choi et al, who found 11 out of 20 sera to be positive. The reason for this difference is not apparent. We do very much agree that native and denatured type II collagen occur, though in low titre and less commonly than the 13 out of 20 sera previously reported positive. This probably reflects an antibody response to connective tissue inflammation because of cross reactivity with other denatured collagens or the binding of immune complexes. Further studies in larger series of patients with lepromatous leprosy are needed, but our negative results suggest that antibodies to native type II collagen are largely restricted to RA.

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