Successful Treatment of Reiter's Syndrome in a Patient with AIDS with Methotrexate and Corticosteroids

Sir—Reiter's syndrome (RS) is the most frequent rheumatological disease that occurs in HIV-infected patients. The symptoms may be severe and unresponsive to conventional treatment [1]. Since methotrexate (MTX) is a fairly effective treatment of RS in non-HIV patients, it has been tested in some HIV-positive patients, but it aggravated the underlying immunodeficiency-related diseases. It is therefore generally agreed that this drug should be avoided in such cases. Nevertheless, some patients are so very ill that there is no alternative. We report here the case of a patient with full-blown AIDS and very severe RS who was successfully treated with MTX and corticosteroids, taking stringent precautions.

The patient was a 37-yr-old homosexual man. Skin lesions of Kaposi's sarcoma (KS) led to the discovery of HIV seropositivity in 1989. In 1993, the patient had inflammatory polyarthalgias together with cutaneous lesions suggestive of keratoderma blenorrhagica. He was HLA B27 positive. RS was diagnosed and he was successively treated with salazopyrine, bromocriptine and phenylbutazone. Despite these treatments, the clinical status of the patient worsened. He was referred to our department in September 1994 with a severe inflammatory and exudative polyarthritis. The pain was excruciating, requiring morphine sulphate, and the patient was severely incapacitated. There were three cutaneous lesions of KS, diameter 0.5 cm, on the forearm and the thigh. The CD4 count was 19 cells/mm³. The patient was treated with 10 mg methotrexate once a week and 10 mg prednisone/day. He was also given malocide and disulone as prophylaxis against *Pneumocystis carinii* and toxoplasmosis, zidovudine + lamivudine and bleomycin (15 mg once a week) for the KS. The result was fair, and remained so for 2 months, but a severe flare-up of polyarthritis in December 1994 required two bolus injections of 250 mg methylprednisolone together with intra-articular infiltrations of corticosteroids. Oral corticotherapy was increased to 20 mg prednisone/day and MTX to 20 mg/week: the arthritis completely subsided and the skin lesions were totally cleared. The patient has now been treated for 8 months and remains free of articular symptoms. Prednisone has been tapered to 10 mg/day; the dose of MTX was recently reduced to 10 mg/week. The AIDS treatment has not been altered. The KS was first treated with bleomycin, then with vinblastine and radiotherapy; the lesions remained unchanged. The last CD4 count was 15 cells/mm³.

Since the first report of RS in HIV-positive patients by Winchester *et al.*, in 1987 [2], MTX has been considered to have adverse effects on the immunodepressive disease, favouring the development of KS sarcoma or *P. carinii* infection [3, 4]. On the other hand, Maurer *et al.* [5] recently reported the treatment of three patients with AIDS and severe exacerbations of psoriatic arthritis with high doses of MTX for several months, with a good result on the skin and joint symptoms, and no opportunistic infections in two of them. The patient described here was in severe immunodepression, but he was in dire misery because of his musculoskeletal symptoms, which were insensitive to the usual treatments. His distress prompted us to attempt this controversial therapy, combined with antiviral therapy, chemoprophylaxis of infection and very aggressive therapy against KS. This therapeutic approach was successful, without any exacerbations of the immunodepression-related symptoms. This sort of treatment should be reserved for the most severe cases of RS or psoriatic arthritis, when everything else has failed, but MTX, with or without corticosteroids, should not be systematically rejected for treating RS in patients with AIDS.

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Osteitis Condensans Ilii

Sir—We read with great interest the paper by Clarke and his colleagues dealing with magnetic resonance imaging in osteitis condensans ili [1]. They claim that restriction of bony abnormality to the iliac side of the sacroiliac joint is a characteristic feature of the condition in addition to normality of joint space and articular surfaces.

Recently, in order to demonstrate that patients with 'true' osteitis condensans ili may be differentiated clinically from those with seronegative spondylarthropathy showing sacroiliitis mimicking osteitis condensans ili, our group performed computed tomography of the sacroiliac joints in 15 patients with 'true' osteitis condensans ili [2, 3].
Sacral sclerosis, always wider than 3 mm, was present in 21 (72.4%) of the 29 joints (one patient had unilateral osteitis condensans ilii).

The results of this study confirmed the opinion of those authors who had suggested that sacral sclerosis may occur in osteitis condensans ilii [4–8]. Others had suggested that what sometimes appears to be sacral involvement is no more than sclerosis in the auricular portion of the ilium itself [9–11]. Figures 1 and 2 show the plain radiograph and the CT scan of one patient with osteitis condensans ilii recently seen in our departMENTS.

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**Fig. 1.—** Anteroposterior view of the pelvis showing typical findings of osteitis condensans ilii.

**Fig. 2.—** CT of the synovial part of the sacroiliac joints showing normal joint space and surfaces, and extension of the sclerosis into the depth of the bone on both the iliac and the sacral sides.
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Chronic Polyarthritis and Graft-Versus-Host Disease

Sir—In previous studies, the presence of arthralgias and transitory arthritis in graft-versus-host disease (GVHD) has been reported, but not chronic arthritis. We will consider a case of non-erosive polyarthritis after a bone marrow transplantation in the context of chronic GVHD.

A 32-yr-old male was diagnosed at the age of 27 as having acute myeloblastic leukemia. One year after the diagnosis, he received an allogenic bone marrow transplant for acute myeloblastic leukemia. One year after the transplantation, he was admitted for cough and greenish expectoration, with radiological pulmonary infiltration, diagnosed as interstitial bilateral pneumonitis. During the time he was in hospital, acute renal insufficiency developed, brought on by nephrotoxic drugs. The cyclosporin A was then stopped. An increased level of bilirubin and alkaline phosphatase was detected, with negative markers of hepatitis B and C, and the hepatic biopsy showed changes compatible with GVHD and moderate siderosis. A generalized pruritus began without evidence of skin lesions. The patient reported slight xerophthalmia. Schirmer's test indicated a lacrimal secretion of 9 and 12 mm. Chronic GVHD was diagnosed with hepatic, pulmonary and ocular involvement, and treatment with prednisone 30 mg and thalidomide 300 mg was prescribed, with good response.

Seven months later, generalized oedema began, with nephrotic type proteinuria becoming evident with conservative renal function. ANA and anti-DNA antibodies were negative. The renal biopsy showed membranous glomerulonephritis, leading to treatment with prednisone 60 mg, by which nephrotic syndrome was resolved.

Two years after the transplant, through treatment with prednisone 20 mg, pain in the right elbow and left ankle began, at first subacute. General examination was normal; no skin lesions were observed. Synovitis was detected in the right elbow. Laboratory studies revealed a haemoglobin of 149 g/l, white blood count 7700/mm³, platelet count 210 000/mm³, ESR 60 mm/h, alkaline phosphatase 1201 IU/l, GGT 670 IU/l, ALT 71 IU/l, total bilirubin 11 μmol/l, creatinine 69 μmol/l and a proteinuria of 0.15 mg/dl. Rheumatoid factor, smooth muscle and antimitochondrial antibodies were negative. The serology and antigenaemia for cytomegalovirus (CMV) were negative. The synovial fluid of the elbow revealed 30 000 leucocytes/mm³ and a neutrophil tendency. The crystals study, cytology and the synovial fluid cultures in habitual media and in Lowenstein-Jensen medium, and for CMV, were negative. In the simple X-rays, signs of avascular necrosis of the right external humeral epicondyle were observed and minimal compact periotic reaction in tibial metaphysis. X-ray images of the thorax, lumbar spine and pelvis were normal. The bone scan with technetium-99m showed increased uptake in the elbows and ankles. The synovial biopsy on the right elbow showed up as unspecific, with degenerative signs, slight chronic inflammation, and centres of haemosiderosis and microcalcifications. The presence of granulomas or amyloid was ruled out, and the culture of biopsy material in Lowenstein-Jensen medium was negative.

Seven months after the initial elbow pain, polyarthritis was observed affecting the wrists, MCPs, PIPs, knees and ankles with prolonged morning stiffness; a similar picture to that of rheumatoid arthritis. There was no family history of relevance to the development of the arthritis. The synovial fluid had inflammatory characteristics and absence of crystals. In the X-rays, there were signs of avascular necrosis of the external condyle of the right knee. X-rays of the hands were normal. The patient was treated with indomethacin and small doses of deflazacort with partial improvement. Two years from the start, the polyarthritis showed a chronic course involving the elbows, wrists, PIPs and right knee. A bone marrow study showed complete remission of the leukaemia.

GVHD is the most common non-infectious complication in transplant patients [1]. It can be acute (<100 days post-bone marrow transplant) or chronic. The acute form occurs in between 42 and 75% of the cases. It is characterized by the presence of dermatitis, diarrhoea and hepatitis, and the skin is affected in almost 100%. The skin involvement brings to mind that which occurs in scleroderma. In the joints, articular contractures, arthralgias and arthritis occur [2–4]. In one study, arthralgias of the large joints were observed in eight of 20 patients and transitory arthritis in four of 18 [2]. Other frequent findings are hepatic [3] and oesophageal involvement, diarrhoea, malabsorption, sicca syndrome and interstitial pneumonitis. The