Clinical data

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Age/sex</th>
<th>Duration of EMS* before cyclosporin A use (months)</th>
<th>Duration of cyclosporin A use (months)</th>
<th>Significant side effects of cyclosporin A</th>
<th>Reason for dose reduction of cyclosporin A</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>44/F</td>
<td>13</td>
<td>3</td>
<td>‘Stiffness’</td>
<td>Patient’s request</td>
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<tr>
<td>2</td>
<td>61/F</td>
<td>20</td>
<td>4</td>
<td>Abdominal pain, diarrhea</td>
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<tr>
<td>3</td>
<td>35/F</td>
<td>7</td>
<td>19</td>
<td>Renal insufficiency</td>
<td>Renal insufficiency</td>
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<tr>
<td>4</td>
<td>41/F</td>
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<td>8</td>
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<tr>
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<tr>
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<tr>
<td>8</td>
<td>52/F</td>
<td>12</td>
<td>7</td>
<td>Worsening myalgia</td>
<td>Myalgia</td>
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</table>

*EMS = eosinophilia myalgia syndrome.

were included in this study; this group might have derived a substantial benefit from corticosteroid treatment that cyclosporin A would do little to augment. Alternatively, the manifestations unresponsive to cyclosporin A might have been either irreversible or too slow to reverse to be detected in a small, short-term trial.

Our data show limited usefulness of cyclosporin when used relatively early in this disease. Given that is now known about EMS, one would expect that the efficacy of any regimen aimed at reduction of inflammation would diminish further as the syndrome progressed, and that any advances in treatment are unlikely to be found among the immunomodulatory drugs.

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Postural variation in von Willebrand factor antigen

Sîr: von Willebrand factor antigen (vWF antigen) is the antigenic component of von Willebrand factor and is synthesised by endothelial cells and megakaryocytes. Raised concentrations of vWF antigen have been reported in a variety of connective tissue diseases, including vasculitis, systemic lupus erythematosus, and systemic sclerosis. Although it is an acute phase reactant, raised levels in some of these diseases may reflect damage to the endothelial cell. As most inpatients lie quietly on a bed, and most outpatients are subject to movement during their clinic visits we sought to determine whether differences in vWF antigen between these groups might be due to posture and activity.

We tested the effect of different postures on circulating levels of vWF antigen in healthy hospital and laboratory staff. In the first study blood was taken into an EDTA tube from a group of five men and five women (mean age 29 (SD 5) years) immediately after normal kinetic laboratory activity. After resting quietly in the sitting position for 15 minutes, blood was taken from the opposite arm. Plasma was obtained after centrifugation at 3000 rpm for 10 minutes and vWF antigen estimated by enzyme linked immunosorbent assay (ELISA). Mean (SD) concentrations of vWF antigen immediately after activity were 1160 IU/L (150), but after the rest period they had fallen to 1040 (220) (p<0.05), Wilcoxon rank sum test applied. In a separate study six men and five women (aged 22–38) were rested lying down, face up, for 25 minutes, after which blood was taken. They then sat upright in a chair for a further 25 minutes and blood was taken a second time. After 25 minutes lying down plasma vWF antigen concentrations were 950 (270) IU/L, but after 25 minutes sitting up levels increased to 1070 (200) IU/L (p<0.05).

This small study identified changes in vWF antigen concentrations depending upon variations in posture and activity. As vWF antigen has been shown to rise after exercise in this finding is not surprising. It points, however, to a possible artefact in vWF antigen determination in patients. This may be important in the case of patients with, for example, rheumatoid arthritis, who are often admitted to hospital for bed rest. Reductions in vWF antigen in these patients may reflect their posture and not necessarily reduced activation/injury of the endothelium. To avoid the possible problem in outpatient clinics we suggest that patients rest sitting for a minimum of 15 minutes before venepuncture.

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Hypertrophic osteoarthropathy and AIDS

Sir: Rheumatologic manifestations of human immunodeficiency virus (HIV) are diverse. In addition to infections, conditions appear to be unique to HIV infected patients, whereas others, such as Reiter’s syndrome, may be more widespread. Reports of patients with coexistent acquired immune deficiency syndrome (AIDS) and diseases of unknown cause are often interesting as they may help to clarify their pathogenesis. One of these syndromes is hypertrophic osteoarthropathy, an entity occasionally associated with chronic bacterial infections and other conditions, especially bronchogenic carcinoma. Despite the high prevalence of diarrheic infections in patients with AIDS, associated hypertrophic osteoarthropathy has rarely been reported. We present a patient with AIDS who developed severe rapidly progressive hypertrophic osteoarthropathy concurrently with anaerobic necrotising pneumonitis.

A 29 year old man, an intravenous drug and alcohol abuser, known to be HIV positive, was admitted owing to development of severe pain and oedema in his arms and legs two months previously. He also complained of a persistent cough, with foul smelling sputum, occasionally haemoptoeic, fever, and pleuritic chest pain. He had lost 10 kg of weight.

On admission the patient appeared cachectic, was unable to walk, and had a temperature of 38.5°C. His mouth showed oropharyngeal candidiasis. Inspiratory rates were heard in the superior field of the right hemithorax. The heart sounds were normal, and there was a rapid systolic murmur due to mitral insufficiency.

Blood studies showed haemoglobin 90 g/l, leucocytes 7·3×10⁹/l (lymphocytes 1·965×10⁹/l (CD4+ of 0·55×10¹⁰/l), CD4+/CD8+ ratio 0·5), platelets 367×10⁹/l, erythrocyte sedimentation rate 105 mm/h. Serum alkaline phosphatase was 425 U/l (normal <275). Results of other routine tests were normal, including calcium, phosphorus, creatinine, glucose, aspartate transaminase, alanine transaminase, and lactate dehydrogenase. Urine analysis results were also normal.


A protein profile disclosed no abnormalities except for mild hypalbuminemia (30 g/l). Antibodies against hepatitis B surface antigens and hepatitis B core antigens were positive. A test for hepatitis surface B antigen was negative. Complement C3 and C4 were normal. Immunoglobulin A was 5 g/l (normal <3.75). Tests for IgG and IgM, rheumatoid factor, antinuclear antibodies, the Veneral Disease Research Laboratory test, tests for thyroid hormones and somatotropin were all normal or negative, as were HLA-B27 and serological assays for Brucella, Salmonella, Yersinia enterocolitica, and Chlamydia spp.

Roentgenograms of the chest showed images of consolidation and multiple cavitations in the superior lobe of the right lung, compatible with necrotising pneumonitis. Intra-dermoinjection with tuberculin 2U was positive. Microbiological examination of samples of sputum showed Gram positive cocci. Sputum and blood cultures were negative, and repeated searches found no mycobacteria in the sputum. Thereafter, percutaneous needle aspiration of the lung was performed. Cultures of the samples in anaerobic media were positive for microaerophilic Gram positive cocci and Bacteroides melaninogenicus.

Cultures of stools were positive for Candida albicans. Upper gastrointestinal fibre endoscopy confirmed the presence of intense candida oesophagitis and hastral hernia.

Radiographs of the hands, feet, forearms, and legs showed typical signs of hypertrophic osteoarthropathy (figure). Synovial fluid from one knee was mildly inflammatory, it contained 3.5 x 10^6 cells/l, with more than 90% lymphocytes. The glucose concentration was 3.9 mmol/l, C3 was 500 mg/l, C4 80 mg/l. A search for microorganisms and crystals was negative.

The patient was diagnosed as having AIDS, anaerobic necrotising pneumonitis, and secondary hypertrophic osteoarthropathy. He was treated with clindamycin, indometacin, and i.v. zidovudine (AZT) and both his general condition and lung lesions improved rapidly. Five months later he was asymptomatic in his joints, and bone radiographic changes had almost disappeared.

To the best of our knowledge the case of only one patient with hypertrophic osteoarthropathy and AIDS has been published, being a young male abuser of parenteral drugs with Pneumocystis carinii pneumonia.1 The severe osteoarticular manifestations of the patient that we report reinforce both the possibility of this association and the typical painful character common to many of the rheumatic syndromes that have been described in HIV infected patients.2

The excellent response of our patient to antibiotics suggests that hypertrophic osteoarthropathy was due to lung infection. As the patient had severe manifestations we do not exclude a possible role of HIV or its effects on immunity as modulator of the inflammatory mechanisms that led to hypertrophic osteoarthropathy. Some authors have proposed immune mechanisms in the pathogenesis of hypertrophic osteoarthropathy.3 Further studies are needed before this possibility can be confirmed.

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Disseminated gonococcal infection in an elderly patient

Sir: We present the case of an elderly patient with disseminated gonococcal infection and congenital C2 deficiency.

A 72 year old married woman was admitted to our hospital because of arthritis of her right wrist and knee. She had been well until 10 days before admission, when she developed chills with painful swelling of the wrist and knee. She denied any rash, diarrhoea, vaginal discharge, recent sexual contacts, ocular or genitourinary symptoms. Her husband also denied sexual activity.

On examination she had a fever (38°C) and painful swelling of the right wrist and knee. No cutaneous lesions or tenosynovitis were seen.

A diagnostic arthrocentesis of the right knee showed 20 ml of purulent synovial fluid containing 80 x 10^6 cells/l. Gram staining showed no microorganisms, but Neisseria gonorrhoeae was cultured. Blood cultures remained sterile. The C-reactive protein and sedimentation rate was 75 mm/h, results of a routine biochemical test and urine analysis were normal and serological tests for syphilis and brucellosis were negative. The hands, knees, and pelvis radiographs were normal. Total haemo-lytic complement (CH50) was 0 U/ml. No complement C2 was detected in two consecutive determinations.

She was treated with intravenous benzyl-penicillin 1.2 g four times a day for seven days, followed by oral amoxycillin 1.5 g daily for 10 days. She required closed drainage for three consecutive days.

Patients with deficiencies in the terminal components of the complement system may develop episodes of disseminated infection with N gonorrhoeae and N meningitidis.1 C2 deficiency is the most common complement deficiency, often associated with immune complex disease and recurrent sepsis.2 To our knowledge, only one case of disseminated gonococcal infection associated with C2 deficiency has been reported.3 Our patient had not had any previous neisserial or recurrent infections.

Disseminated gonococcal infection has been infrequently reported in the elderly4; patients with disseminated gonococcal infection are usually younger than 40.5,7 The importance of suspecting this diagnosis, even in elderly patients without an appropriate history, is that the organism may be missed if incorrect cultures are taken.10 N gonorrhoeae is recovered from 25% of purulent effusions, and positive blood cultures may occur in only 30% of patients; therefore, in suspected cases, cervical, urethral rectal, and pharyngeal cultures should be obtained. This report highlights the need to consider a broad range of microorganisms in elderly patients with septic arthritis.

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