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 what is now known about EMS, one would expect that the efficacy of any regimen aimed at reducing inflammation would diminish further as the syndrome progressed, and that any advances in treatment are unlikely to be found among the immunomodulatory drugs.

SIR: Rheumatic manifestations of human immunodeficiency virus (HIV) are diverse. Some 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 opportunistic 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 haemoptysis, fever, and pleuritic chest pain. He had lost 10 kg of weight.

A chest X-ray showed cackectasis, was unable to walk, and had a temperature of 38.6°C. His mouth showed oropharyngeal candidiasis. Inspiratory rates were heard in the superior field of the right hemithorax. Heart sounds were normal, and there was no systolic or diastolic tachycardia. The liver was felt 5 cm under the costal margin. There was no splenomegaly and diseased lymph nodes were not palpable. Both forearms, hands, legs, and feet showed prominent oedema, localised to his arms and legs and tenderness to pressure. Osteoarthritic examination disclosed prominent clubbing of the fingers and toes, as well as synovitis, with moderate effusion of the knees, ankles, elbows, wrists, and proximal interphalangeal joints of the hands.

Blood studies showed haemoglobin 90 g/l, leucocytes 7.3 x 10^9/l (lymphocytes 1.965 x 10^9/l, neutrophils 5.5 x 10^9/l), platelets 367 x 10^9/l, erythrocyte sedimentation rate 105 mm/h. Serum alkaline phosphatase was 425 U/l (normal <279). Results of other routine tests were normal, except for hypoalbuminaemia, hypocalcaemia, hypophosphataemia, hyperglycaemia, and uric acid. Liver function tests were normal. After reviewing the patient's medical history and excluding other causes of his clinical features, we diagnosed hypertrophic osteoarthropathy.
A protein profile disclosed no abnormalities except for mild hypoaalbuminaemia (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-dermoection 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 hiatal 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 $3.5 \times 10^6$ 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, indomethacin, and azidovudine (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 complications 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 for fungi or its effects on immunity as modulator or amplifier of the 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.

LOURDES GIL-GARCIA J MANUEL MARTIN-SANTOS MANUEL BLANCO-CABERO ANGEL TAPIAS DEPARMENbytero y Hospitai Rigor, de Hugardia, Valladolid, Spain

Correspondence to: Dr J M Martin-Santos, Unit of Rheumatology, Hospital 'Del Pintorio Horta', Carornal Torquemada s/n, 47010 Valladolid, Spain


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, diarhoea, vaginal discharge, recent sexual contacts, ocular or genital 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 $8 \times 10^5$ polymorphs/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 biochemistry test and urine analysis were normal and serological tests for syphilis and brucella were negative. The blood, knee, and pelvis radiographs were normal. Total haemolytic complement (CH50) was 0 U/ml. No complement C2 was detected in two consecutive determinations.

She was treated with intravenous benzylpenicillin 1.2 g four times a day for seven days, followed by oral amoxicillin 1.5 g daily for 10 days. She required closed drainage for three consecutive days. She was discharged.

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 septis.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 correct cultures are taken.8 N gonorrhoeae is recovered from patients with 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 wide range of microorganisms in elderly patients with septic arthritis.

DANIO ROG FICUEROA EDUARDO LUZA CORTINA SENEN GONZALEZ SUAREZ CELIA ERAUSKIN ARRIABARRENA JOSÉ L PENA SASTRE Rheumatology Division, Hospital Universitario Marques de Valdecilla, Santander, Spain

Correspondence to: Dr Rúa-Figueroa, Hospital Universitario Marqués de Valdecilla, Rheumatology Division, Avda de Valdecilla s/n, 3908 Santander (Cantabria), Spain.
