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 irreversibly 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 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.

DANIEL J CLAUW
JEFF A ALLOWAY
Division of Rheumatology, Immunology and Allergy
Department of Medicine
Georgetown University Medical Center
3800 Reservoir Road NW
Washington DC 20007-2197
USA


Postural variation in von Willebrand factor antigen

Sir: von Willebrand factor antigen (vWF Ag) is the antigenic component of von Willebrand factor and is synthesised by endothelial cells and megakaryocytes. Raised concentrations of vWF Ag 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 Ag between these groups might be due to posture and activity.

We tested the effect of different postures on circulating levels of vWF Ag 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 Ag estimated by enzyme linked immunosorbent assay (ELISA). Mean (SD) concentrations of vWF Ag 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 Ag concentrations were 950 (270) IU/l, but after 25 minutes sitting blood levels increased to 1070 (200) IU/l (p<0.05).

This small study identified changes in vWF Ag concentrations depending upon variations in posture and activity. As vWF Ag has been shown to rise after exercise,2 this finding is not surprising. It points, however, to a possible artefact in vWF Ag determination in patients. This may become important in the case of patients with, for example, rheumatoid arthritis, who are often admitted to hospital for bed rest. Reductions in vWF Ag 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.

A BLANN
M V JAYSON
Rheumatic Diseases Centre
University of Manchester
Hope Hospital
Salford M6 8HD
United Kingdom

M H POPE
A M KAGLE
D WILDER
Orthopaedic and Rehabilitation
University of Vermont
Burlington VT05404, USA

J W WEINSTEIN
University of Iowa Hospitals and Clinics
Iowa 52202, USA

Correspondence to: Professor Jayson.


Hypertrophic osteoarthropathy and AIDS

Sir: Rheumatologic manifestations of human immunodeficiency virus (HIV) are diverse. AIDS conditions appear to be more common in 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 anerobic 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 haemoptoic, fever, and pleuritic chest pain. He had lost 10 kg of weight.

On examination his past medical history was unremarkable, and 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. Heart sounds were normal, with a systolic murmur which radiated to the back. No pericardial friction was heard.

Blood studies showed haemoglobin 90 g/l, leucocytes 7.3 x 10^9/l (lymphocytes 1965 x 10^9/l, neutrophils 55 x 10^9/l, CD4 + CD8 + ratio 0.5), 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, 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 = 3.0-35). Tests for IgG and IgM, rheumatoid factor, antinuclear antibodies, the Veneral Disease Research Laboratory test, tests for thyroid and other hormones, and somatomedics 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-dermureaction with tuberculin ZU 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 potato 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 osteoarthritis (figure). Synovial fluid from one knee was mildly inflammatory, it contained 3.5 x 10^6 cells/ml, with more than 90% lymphocytes. The glucose concentration was 3.9 mmol/l, C3 was 500 mg/dl, 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 osteoarthritis. He was treated with clindamycin, indomethacin, and 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 osteoarthritis and AIDS has been published, being a young male abuser of parenteral drugs with Pneumocystis carinii pneumonitis.1 The severe osteoarthropathy, 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 osteoarthritis was due to lung infection. As the patient had severe manifestations we do not exclude a possible role of fungi or its effects on immunity as modulator or amplifier of the mechanisms that led to hypertrophic osteoarthritis. Some authors have proposed immune mechanisms in the pathogenesis of hypertrophic osteoarthritis.3 Further studies are needed before this possibility can be confirmed.

**LOURDES GIL-GARCIA J MARIEL MARTIN-SANTOS MANUEL BLANCO-CABERO ANGEL TAPIAS DEL POZO FELICIDA MARTINEZ-BARRERO Department of Internal Medicine Hospital Rio Hortega, Valladolid, Spain**

Correspondence to: Dr J M Martin-Santos, Unit of Rheumatology, Hospital 'Del Rio Hortega', 2Cardenal Torquemada s/n, 47010 Valladolid, Spain

**DIGUO ROIGIN FIGUEROA EDUARDO LOZA CORTINA SENEN GONZALEZ SUAREZ CELIA ERAUSQUIN ARRIAGABARENA JOSE L PENA SARMIENTO Rheumatology Division, University Hospital Marques de Valdecilla, Santander, Spain**

Correspondence to: Dr Rua-Figueroa, Hospital Universitario Marques de Valdecilla, Rheumatology Division, Avda de Valdecilla s/n, 39080 Santander (Cantabria), Spain.

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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 white blood cell sedimination rate was 75 mm/h, a result of a routine biochemistry test and urine analysis were normal and serological tests for syphilis and brucellosis were negative. The hands, wrists, knees, and pelvis radiographs were normal. Total haemolytic complement (CH50) was 0 U/ml.

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.2 C2 deficiency is the most common complement deficiency, often associated with immune complex disease and recurrent sepsis.4 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 elderly1; patients with disseminated gonococcal infection are usually younger than 40 years.4 The importance of suspecting this diagnosis, even in elderly patients without an appropriate history, is that the organism may be missed. Correct cultures are taken.5 N gonorrhoeae is recovered from the sites 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 wide ranges of microorganisms in elderly patients with septic arthritis.

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*Images of consolidation and multiple cavitations in the superior lobe of the right lung, compatible with necrotising pneumonitis.*
Hypertrophic osteoarthropathy and AIDS.

L Gil-García, J M Martín-Santos, M Blanco-Cabero, J A Tapias del Pozo and F Martínez-Barrero

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