A protein profile disclosed no abnormalities except for mild hypoalbuminaemia (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 somatomropin 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 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 malallengicus.

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

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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 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 height, knees, 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.

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 40.4 7 The importance of suspecting this diagnosis, even in elderly patients without an appropriate history, is that the organism may be missed or misidentified.5 N gonorrhoeae is recovered from 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 broad range of microorganisms in elderly patients with septic conditions.

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