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Poncet’s disease in a patient with AIDS

Sir, We report the development of Poncet’s disease (tuberculous rheumatism) in a 27-yr-old heterosexual Zairian man with AIDS. He had been living in the UK since 1991 and was diagnosed HIV-1 antibody-positive in October 1997. He had no previous AIDS-defining illnesses and was receiving no anti-retroviral therapy. In December 1997 he was admitted with a history of fever, night sweats and persistent cough. A chest radiograph and CT scan showed hilar lymphadenopathy with no parenchymal lung lesions. Gram and Ziehl-Neelsen stains and cytological preparations of bronchoalveolar lavage fluid were negative. Subsequently, Mycobacterium tuberculosis (MTB) was cultured after 6 weeks and he was started on anti-tuberculous treatment with isoniazid, rifampicin, pyrazinamide and ethambutol. Just prior to commencing anti-tuberculous therapy, he was readmitted with a 3-week history of acute onset of pain, and swelling and stiffness of both elbows, wrists, knees and ankles. There was no history of diarrhoea, rashes, genito-urinary symptoms or eye problems. On examination, he was febrile (38°C). He had bilateral effusions of knee and ankle joints with restricted movements. There was active synovitis of the small joints of the hands and feet. There was no skin rash, mouth ulcers, conjunctivitis or urethral discharge.

Investigations showed the following: haemoglobin 9.9 g/l (13.5–17.5 g/l), white cell count 13.1 × 10^9/l (4–11 × 10^9), platelets 166 × 10^9 (150–400 × 10^9), CD4 count 0.1 × 10^9 cells/l (0.455–1.32 × 10^9). Urea, electrolytes and liver function tests were normal. Radiographs of the knees and ankles were normal. Aspiration of synovial fluid from the knees revealed a large number of white cells with 90% neutrophils. There were no crystals and no organisms were identified on Gram or Ziehl-Neelsen staining. Blood and synovial fluid cultures were negative for mycobacteria and other pathogens. Other causes of arthritis were excluded: rheumatoid factor, ANA, antibodies to extractable nuclear antigens, brucella, and hepatitis C, hepatitis B surface antigen, and syphilis serology. Urethral cultures for Chlamydia trachomatis and Neisseria gonorrhoea were all negative.

The patient’s polyarthritis and effusions improved rapidly and he was able to walk and climb stairs within 3 weeks of commencing anti-tuberculous therapy. MTB was subsequently found to be resistant to isoniazid and the therapy was changed according to the sensitivity of the organism. He continued appropriate anti-tuberculous treatment for 1 yr. His pulmonary tuberculosis had resolved and he had no further evidence of joint inflammation. A diagnosis of Poncet’s disease was made on the basis of a symmetrical, seronegative polyarthritis involving both large and small joints occurring in association with active pulmonary tuberculosis, synovial fluid which was negative for MTB both on Ziehl-Neelsen staining and culture, and prompt resolution of symptoms with anti-tuberculous therapy.

A variety of rheumatic disorders have been described in association with HIV infection. Reiter’s syndrome and other reactive arthritides are the most frequently reported joint diseases. However, organisms known to trigger this type of arthritis, such as C. trachomatis and N. gonorrhoea, are rarely identified in these patients. A study in Zimbabwe showed that arthritis associated with heterosexually acquired HIV infection was most commonly characterized by oligoarticular, asymmetrical, large joint arthritis with or without Reiter’s syndrome [1]. Poncet’s disease was first described in 1897 and is a reactive polyarthritis associated with non-articular tuberculosis. Cases of reactive arthritis complicating atypical mycobacterial infection and intravesical instillation of Calmette-Guérin bacillus (BCG) have also been described [2, 3], but these should not strictly be considered as Poncet’s disease.

It is unclear why sterile polyarthritis should sometimes complicate tuberculosis. It has been suggested that the pathogenic mechanism could be T-cell-mediated cross-reactivity between mycobacterial antigens and cartilage proteoglycans [4]. Alternatively, patients with or
without HIV disease may exhibit hypersensitive immune responses to mycobacterial antigens [5]. A genetic predisposition may also be required, as persons positive for HLA DR3 and HLA DR4 have been shown to be hyper-responsive to mycobacterial antigens [6–8].

In HIV-infected patients presenting with reactive polyarthritis, Poncet’s disease should be considered, particularly in those groups at increased risk of tuberculosis. Only two cases of Poncet’s disease in HIV-infected patients have been reported so far in the biomedical literature [5, 9]. This is the first case report of Poncet’s disease in a patient with HIV infection in the UK. As in previously reported cases, polyarthritis resolved promptly with anti-tuberculous therapy.

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