Sero-negative rheumatoid arthritis associated with AIDS

Sir: Acquired immunodeficiency syndrome (AIDS) is a disease of protein manifestations. It is almost certainly caused by a retrovirus: human immunodeficiency virus (HIV). Its major presentations are those of recurrent infection and neoplasia.

Musculoskeletal manifestations have been reported and occur in up to 70% of patients.

Arthritis is less common but certainly does occur. Reiter’s syndrome and reactive arthritis are the most commonly associated. Infectious arthritis due to organisms such as cryptocoecus and mycobacteria has been reported. Psoriatic arthritis occurs. An AIDS associated arthritis has also been described.

Rheumatoid arthritis like disease has rarely been reported as coexisting with AIDS. The patient presented here had criteria for classical seronegative rheumatoid arthritis. His case is presented to alert clinicians to this association and to report that AIDS does not exclude rheumatoid arthritis.

The patient, a white homosexual man, aged 28, presented in January 1986 with pneumonia. He was admitted to hospital and recovered uneventfully from a diagnosis of Streptococcus pneumoniae pneumonia. At that time he was noted to have cervical adenopathy and a packed cell volume of 0.34 with a normochromic normocytic blood smear. In April 1986 he developed swelling and pain in his feet and then his knees, with generalised arthralgia. He was evaluated for this in May 1986 and was found to be HLA-B27 positive with no sacroilitis or other stigmata of Reiter’s spondylitis, which was considered to be a seronegative white blood cell with 78% polymorphonuclear cells. No crystals were noted on polarisation microscopy. A test for HIV was positive by enzyme linked immunosorbant assay (ELISA) and confirmed by Western blot. A lumbar puncture showed normal cerebrospinal fluid and a Venerole Disease Research Laboratory test was negative. His synovitis progressed and by August 1986 he had symmetrical polyarthritis affecting the metacarpophalangeal joints, wrists, elbows, shoulders, knees, ankles, and metatarsophalangeal joints.

He had pain at night and early morning stiffness, which lasted more than two hours. He noted profound fatigue and a weight loss of 5–7 kg with weakness, particularly of the quadriceps muscles. The erythrocyte sedimentation rate was 135 mm/h (Westergren).

In October 1986 a biopsy of a solitary lesion on the right thigh showed Kaposi’s sarcoma, and a diagnosis of AIDS was made. Retrospective biopsy of lesions had probably been present for 10 months before biopsy.

Laboratory data shortly after included a T4 cell count of 11% (normal 39–63), T8 cells 69% (normal 16–38), and a T4/T8 ratio of 0.16.

Despite treatment with zidovudine, doxorubicin, and other drugs his Kaposi’s sarcoma spread to the skin and bowel. He had two episodes of shigella diarrhoea, both treated with ampicillin, with no residue and no flare of his arthritis or other change in his clinical state. Over the next 20 months his major complaint was polynovasynovitis, and pain, which was poorly controlled with indomethacin and prednisolone 10–20 mg a day. Radiographs of the hands showed periarticular osteoporosis and erosions consistent with rheumatoid arthritis. The feet also showed osteoporosis, erosions, and marked deficiency of the metatarsophalangeal joints. Spinal and pelvic radiographs were normal. A synovial biopsy was performed in September 1988 but no HIV DNA was detected by polymerase chain reaction and viral cultures were negative.

Shortly thereafter he died from a massive gastrointestinal bleed secondary to his Kaposi’s sarcoma.

Winchester et al described a group of patients with AIDS with multiple criteria for Reiter’s syndrome. The arthritis in these patients and others subsequently reported was predominantly oligoarticular, and a few patients had sacroiliitis. Other clinical features of the syndrome noted were commonly present; particularly noted were heel pain and a variety of enthesopathies. Cutaneous features were common.

A subacute oligoarthritis syndrome, possibly specific for AIDS, has been reported. A more extensive report of the rheumatic manifestations of HIV also recorded Reiter’s arthritis, a painful articular syndrome, and psoriatic arthritis. One patient in that series was reported to have a polyarthritis of six months’ duration. The presentation was that of symmetrical disease affecting small joints, which was lateral and negative and clinically similar to rheumatoid arthritis. No common denominator was noted in the radiographic findings or other diagnostic features.

Autoimmune disease occurs in association with AIDS. Sjögren’s syndrome manifest mainly by sicca syndrome has been well reported. Polymyositis and vasculitis have also been described. Rheumatoid arthritis, seronegative or seropositive, has not been reported to be caused by HIV infection. Conversely, it has been stated that it does not occur. The polyarthritis seen has been thought to represent Reiter’s arthritis or reactive arthritis.

The patient presented here fulfills criteria for classical seronegative rheumatoid arthritis. He had no other features of Reiter’s syndrome or psoriasis and no enthesopathy or heel pain. The disease was prolonged with an unremitting course for about 30 months until his death. He did not respond to treatment with auranofin, penicillamine, or cytotoxic drugs and zidovudine used to treat his Kaposi’s sarcoma. His disease was moderately controlled with indomethacin 75 mg twice daily and prednisolone 10–25 mg daily. During the course of his illness treatment with prednisolone was tapered, but this resulted in a severe flare of his arthritis, though no skin rash suggestive of psoriasis.

The disease did not remit when his CD4 T lymphocyte counts were markedly reduced, suggesting that these cells were not central to the disease. The few of them that were needed, or more likely a subset of disease specific T lymphocytes was unaffected in this patient. It has been suggested that AIDS might cure or markedly attenuate arthritis, but the presence of HIV certainly did not reduce the severity of the synovitis in this patient.

A causative role for HIV was not established and no viral DNA was identified by polymerase chain reaction in the cells lines obtained from his synovial biopsy specimen.

Viral cultures of synovial tissue were likewise negative for HIV.

Possibly, the disease represented a reactive arthritis, but the symmetrical involvement and chronicity mitigate against this. It should be noted that at the onset of his disease he had no obvious infection. The natural history, immunological aspects, and pathogenesis of the arthritis seen in association with HIV are poorly understood. Although rare, a polyarthritis fulfilling current criteria for rheumatoid arthritis may occur early in the disease, and HIV should probably be tested for in all high risk patients who present with a recent onset arthritis.

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A case of chronic Lyme arthritis in England

Sir: The first British case of Lyme arthritis was reported recently, in a patient with an acute asymmetrical polyarthritis associated with erythema chronicum migrans following a definite history of tick bite. We describe a case of chronic Lyme arthritis in an individual who became infected in the British Isles. The patient had chronic symmetrical synovitis of the knees with a history of skin rash, strongly suggestive of erythema chronicum migrans, a positive serology test for Borrelia burgdorferi, but no history of tick or insect bite. Her case is described below.

A 42 year old woman noticed gradual onset of pain, swelling, and stiffness in her knee joint in February 1986. In March 1987 she was seen in our rheumatology clinic, and examination showed effusions from both knees with no evidence of active inflammation. There was no cardiac or neurological abnormality and she was otherwise well. She had no personal or family history of arthritis, psoriasis, urticaria, or bowel disorders, or urogenital infections. She was a regular practitioner in October 1985 with an annular erythematous rash on her trunk associated with malaise and fever. The rash had started with a redish macula, which gradually spread to form a plaque-like rounded lesion with some raised margin. The rash was itchy and disturbed her sleep occasionally. Her general practitioner, believing it to be a non-specific skin infection, treated her with dacyocycline as...