Gonococcal arthritis caused by auxotype P in a man with HIV infection

G Moyle, S E Barton, J Midgley, I F Rowe, A C Keat, A G Lawrence

Abstract

The development of gonococcal arthritis is reported in a man with HIV infection and CDC Stage IVC2 disease. The diagnosis of disseminated *Neisseria gonorrhoeae* was facilitated by microbiological examination of a joint aspirate. The auxotype identified by culture was moderately resistant to penicillin, a characteristic which is highly unusual for an organism causing disseminated gonococcal infection. This case serves as an example of the role of HIV infection in the modification of host response to common pathogens and the need for clinicians to modify their management of disseminated gonococcal infection especially in immunosuppressed persons.

Case report

A 25 year old, deaf and dumb, homosexual man presented with a two week history of generalised myalgia and multiple painful joints for one week. His symptoms had persisted despite a five day course of oral erythromycin 500 mg qds. He had no history of urethral, rectal or pharyngeal symptoms. He had been found to be human immunodeficiency virus (HIV) antibody positive in 1985 whilst asymptomatic. Subsequently he developed persistent generalised lymphadenopathy and had recently suffered an episode of oral candidiasis which resolved with topical nystatin therapy. He had not received any other recent medication.

Physical examination revealed red, swollen, hot and tender areas around his left shoulder, left great toe metatarsophalangeal (MTP) joint, right third toe MTP joint (fig) and over his left tibial tubercle. The skin overlying the right third toe was necrotic, but there were no other cutaneous or mucosal lesions. He was afebrile but appeared sweaty and generally

Figure Right third toe prior to aspiration of proximal interphalangeal joint.

unwell. Radiographs of all the affected joints were normal, as were routine biochemical tests including renal and liver functions, serum urate and alkaline phosphatase levels. An autoantibody screen was negative and the C-reactive protein level was not raised. His current CD4 T lymphocyte count was $114/\text{mm}^3$ and HIV p24 antigen present in the serum at a level of $140 \mu\text{g/ml}$.

Aspiration of the right toe proximal interphalangeal joint yielded 3 ml of pus. Gram staining and light microscopy of this aspirate revealed gram negative diplococci. The pus was inoculated onto a modified Thayer Martin medium which enabled the identification of *Neisseria gonorrhoeae*. This isolate

Kobler Centre, St Stephen's Clinic, London UK
Department of Genitourinary Medicine
G Moyle, S E Barton, A G Lawrence
Department of Microbiology
J Midgley
Department of Rheumatology
I F Rowe, A C Keat

was sent to the Gonococcus Reference Unit of Bristol Public Health Laboratory and was reported as auxotype P, serotype 1B7, moderately resistant to penicillin (MIC = 0.16 mg/l). Throat, rectal and urethral swabs were also taken but no evidence of *N gonorrhoeae* was detectable on microscopy or culture. Examination of the genitourinary tract and rectum revealed no abnormality. No organisms were grown from repeated blood cultures.

The patient was admitted for bed rest and analgesia. Treatment with intravenous benzylpenicillin 24 megaunits daily for three days, then 8 megaunits daily for four days, resulted in an excellent clinical response. This was followed by treatment with ampicillin 2 g daily for a further week. By this stage recovery was complete.

Direct questioning revealed that he had recently engaged in passive anal intercourse with four casual male sexual partners: all were untraceable. Safer sexual practice practices were once again discussed.

In the six months following this episode, he has commenced therapy with zidovudine and co-trimoxazole (as prophylaxis against *Pneumocystis carinii* pneumonia) and his symptomatic HIV disease has not progressed further from the CDC Stage IV C2 with which he presented.

Discussion

Disseminated gonococcal infection in an HIV infected man has not been previously reported. Indeed, the reported incidence of disseminated gonorrhoea has declined over recent years to only nine cases in the UK in 1988 (personal communication from CDSC, Colindale), of which four were isolated from joints. This reflects both the overall reduction in cases of gonococcal infection and the specific decline in the prevalence of strains of *N gonorrhoeae* which are associated with disseminated infection.¹ The strains most commonly identified in cases of disseminated infection are usually highly sensitive to penicillin² and found to have selective growth requirements for arginine, hypoxanthine and uracil.³ However, cases of disseminated gonococcal infection caused by multiple-resistant organisms have recently been reported.⁴

The diagnosis of a sexually acquired infection in this man and his recent history of unprotected, penetrative sexual intercourse with casual partners indicates a failure of the counselling and education which he had previously received. Special facilities for disabled patients, particularly those with communication problems, need to be made more widely available. The failure to detect *N gonorrhoeae* in the urethra, rectum or pharynx of this man may have been due to the effect of the erythromycin taken prior to his presentation.

It has been suggested that HIV infection results in a reduction in the host resistance to bacterial infec-

tion,⁵ possibly due to B cell and neutrophil dysfunction and alterations in interferon production. Among HIV infected patients an increased incidence of metastatic complications of superficial bacterial infections has been reported⁶ and opportunistic organisms have been isolated from aspirates of synovial fluid in AIDS patients.^{7,8}

The occurrence of joint disease in HIV infected patients is becoming increasingly recognised.⁹ In particular, aseptic seronegative arthropathies, Reiter's syndrome and psoriatic arthritis appear to occur more frequently in HIV infected patients.^{10,11}

An increasing prevalence of resistant strains of *N gonorrhoeae* causing dissemination prompts the need for reassessment of our management of this disease, especially on the background of immunosuppression.

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Correspondence to: Dr G Moyle, Department of Genitourinary Medicine, Kobler Centre, St Stephen's Clinic, Fulham, London SW10 9TH, UK

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