Rape and subsequent seroconversion to HIV

S Murphy, V Kitchen, J R W Harris, S M Forster

Department of Genitourinary Medicine, St Mary's Hospital, London W2 1NY
S Murphy, MRCP, *senior registrar*
V Kitchen, MRCP, *senior registrar*
J R W Harris, FRCP, *senior consultant*
S M Forster, MRCP, *senior lecturer*

Correspondence to:
Dr Murphy.

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Infection with HIV may be transmitted to a victim of sexual assault.[1,2] Seroconversion to HIV occurred in the three months after a rape in a woman who had no other identifiable risk factors for HIV infection.

Case report

In May 1987 a 24 year old woman was examined and tested for HIV antibody because she had been raped two weeks previously. A man whom she knew had forced her to have vaginal and anal intercourse. He subsequently told her that he was positive for HIV antibody. She had had no blood transfusions, used no intravenous drugs, and had no sexual contact with bisexual men or men from central Africa and no other sexual contact in the previous nine months.

The results of tests for *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, and *Trichomonas vaginalis* were negative, as were serological tests for treponemal infection and HIV antibody. Repeat testing for HIV antibody in August gave a positive result, which was confirmed by the enzyme linked immunosorbent assay (ELISA) and Envacore tests. The diagnosis was discussed with the victim, and she returned to her own country for follow up care. The assailant had attended a different department of genitourinary medicine in London. He had persistent generalised lymphadenopathy associated with HIV seropositivity when the rape occurred.

Comment

HIV infection can be transmitted during rape, but it is necessary to establish that the victim was seronegative before the incident and that the assailant was infected with HIV at the time of the assault.

Three other women who attended our unit after a rape were found to be positive for HIV antibody. None had had blood transfusions, used intravenous drugs, or been in contact with bisexual men. One had a boyfriend from central Africa. Serum taken at the time of the rape was not stored, so HIV infection could not be definitely attributed to the assault.

Routine testing for HIV infection of victims of heterosexual rape,[1] homosexual rape,[3] and child sexual abuse[2] has been discussed. After a rape many women request a test for HIV. Otherwise it may not be done or the doctor may think it is best not to do it, especially when the victim is distressed. Moreover, patients may have to wait several months for seroconversion to take place.

In Britain, unlike the United States,[4] victims of rape are not offered prophylactic antibiotic treatment for sexually transmitted diseases. We may have to consider giving prophylactic treatment with zidovudine after a sexual assault by a person known to be positive for HIV, though the after effects of zidovudine prophylaxis are not known. A trial is being conducted in the United States with medical staff who were exposed to HIV infection through needlestick injury.

An assailant may be asked to supply samples and agree to be tested for sexually transmitted diseases. A refusal may be noted in court.[5] He may prejudice the case against himself by refusing to be investigated, yet know that a positive diagnosis will alter the jury's perception of his crime.

We believe that all adult victims of rape should be offered a test for HIV, reassured that the risk of infection is low, and offered counselling. A serum sample should be taken at that time and stored in a secure freezer. A test should be considered two or three months later when seroconversion will probably have occurred if the person has become infected and when the person will be better able to make a rational decision about testing. If this sample is positive the stored sample can be analysed and if that is negative the likelihood that infection occurred as a result of the assault will be stronger.

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Osteoporosis and immunosuppression in multiple myeloma

A L Dolan, T K Wheeler, D H Jones, A J Crisp

Addenbrooke's Hospital, Cambridge CB2 2QQ
A L Dolan, MB, *senior house officer in rheumatology*
T K Wheeler, FRCR, *consultant radiotherapist*
D H Jones, FRCR, *consultant radiotherapist*
A J Crisp, MD, *consultant rheumatologist*

Correspondence to:
Dr Crisp.

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Generalised osteoporosis may be the presenting feature of multiple myeloma. We report two cases in which the only clue to the underlying diagnosis was associated immunodeficiency with suppressed serum immunoglobulin concentrations.

Case reports

CASE 1

A 56 year old man presented with a one year history of pain in the chest and thoracic spine with morning stiffness. His previous history was unremarkable. He was in severe pain. General physical examination did not show any abnormality. Peripheral joints were normal. His cervical spine was restricted with pain on flexion. There were multiple areas of tenderness throughout the thoracolumbar spine, and spinal movements were reduced and painful. Straight leg raising was full but caused back pain. There was no neurological deficit.

X Ray films showed generalised spinal osteoporosis without fractures. A bone scan suggested only minor degenerative disease of the lumbar spine. A range of blood tests, including liver function tests, yielded normal results. Immunoelectrophoresis of serum proteins gave normal results on two occasions but on a third occasion suggested immunosuppression, with IgG concentration 5·3 g/l (normal 6-13), IgA 0·7 g/l (0·8-3·7), and IgM <0·3 g/l (0·4-2·2). No serum paraprotein was identified on several occasions. Immunoelectrophoresis of urine did not detect any Bence Jones protein. A trephine biopsy of the iliac crest showed a hypercellular marrow with 25% plasma cells, including many abnormal forms.

Multiple myeloma was diagnosed. Radiotherapy followed by melphalan and prednisolone improved his pain, although he developed severe kyphoscoliosis.

CASE 2

A 72 year old woman presented with a five month history of pain of the mid-lumbar spine that had confined her to bed. Her other medical history was unremarkable. She experienced intense pain on turning in, or getting out of, bed. There was no spinal tenderness. Straight leg raising was full without neurological deficit.

X Ray films showed generalised osteoporosis and a crush fracture of L3. A bone scan showed multiple sites of increased uptake throughout the spine. All blood tests, including liver function tests, gave normal results. Urinary calcium excretion was normal. Immunoelectrophoresis showed suppressed immunoglobulin concentrations: IgG 4·1 g/l, IgA <0·5 g/l, IgM <0·3 g/l. No serum paraprotein was ever identified. Immunoelectrophoresis of urine gave a positive result for Bence Jones protein on one occasion, but this was not confirmed in two further urine specimens. A trephine biopsy of the iliac crest showed that the marrow contained 30% plasma cells, including many abnormal forms. Immunohistochemical examination identified κ light chains produced by plasma cells.

Multiple myeloma was diagnosed, and she was treated with radiotherapy, melphalan, and prednisolone with satisfactory relief of pain.

Comment

Over a third of women aged over 65 suffer osteoporotic vertebral fractures.[1] Underlying diseases such as multiple myeloma or an endocrine disorder predisposing to osteoporosis should be sought as specific treatment may prevent further loss of bone. Diffuse osteoporosis is the predominant radiological feature in only 6% of cases of myeloma.[2] This loss of bone is attributed to production of lymphokine.[3] Myeloma characteristically presents with osteolytic bone lesions and secretion of a monoclonal immunoglobulin with or without Bence Jones proteinuria. An associated hypogammaglobulinaemia is found in 9% of patients.[2] Our patients had "non-secretory" myeloma, a recognised subtype, in which no detectable paraprotein is found on electrophoresis of serum and urine. It occurs in 4% of all cases of myeloma.[2] Reduced immunoglobulin concentrations were a constant feature in one subgroup of patients in a study, who were also characterised by bone loss and normal serum calcium concentrations and renal function. Two out of seven patients in this group had generalised osteoporosis.[4] Our two patients may be further examples of this pattern.

Underlying disease should always be considered in patients with apparently "simple" osteoporosis. Immunoelectrophoresis should be performed in all patients but may miss myeloma. If serum immunoglobulin concentrations are reduced in the absence of other evidence of myeloma bone marrow aspiration should be performed.

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Cost analysis of prophylaxis with antibiotics to prevent infected knee arthroplasty

Sten Bengtson, Lars Borgquist, Lars Lidgren

Department of Orthopaedics, University of Lund, S-221 85 Lund, Sweden
Sten Bengtson, MD, *senior registrar*
Lars Lidgren, MD, *professor of orthopaedics*

Department of Health and Society, Linköping University, S-581 85 Linköping, Sweden
Lars Borgquist, MD, *associate professor*

Correspondence to:
Dr Bengtson.

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Infection after a knee arthroplasty can be disastrous to the patient and puts a heavy economic burden on society. We studied the direct medical costs of infected knee arthroplasties and analysed the cost effectiveness of prophylaxis with antibiotics in patients with knee arthroplasties and concomitant skin lesions.

Patients, methods, and results

Thirty two of 1047 knee arthroplasties performed at our hospital between 1970 and 1985 became infected.[1] There were 17 haematogenous infections, and the most common distant focuses were skin ulcers and vasculitic lesions (six cases).

We calculated the direct medical costs for normal and infected knee arthroplasties from the costs of the operation, the number of days in hospital, outpatient visits, radiological examinations, and antibiotic treatment. We used the price lists of the regional hospital and of the National Corporation of Swedish Pharmacies for 1988 (£1=Swedish kr 11·20). The total direct costs averaged kr 353 951 (£31 603) for a patient with infection (range kr 77 464-692 952 (£6916-61 871)) and kr 47 868 (£4274) for a patient without infection (table).

Ainscow and Denham reported three cases of haematogenous infection among 40 cases of recurrent skin ulceration and infection.[2] By extrapolation to our study, our six cases of haematogenous infection would have occurred among 80 cases of recurrent skin ulceration and infection. We assumed that these haematogenous infections could be avoided by prophylactic treatment with antibiotics, and we calculated the cost saved per infection prevented by the formula $(C_{inf} \times SI - (C_{ab} + C_{se}))/SI$, where C_{inf}=additional costs in cases of infection, SI=number of infections prevented, C_{ab}=costs of prophylaxis, and C_{se}=costs of side effects of prophylaxis.

Thus C_{inf}=cost per case of infection minus cost per case without infection=kr 353 951−47 868 (£31 603−4274)=kr 306 083 (£27 329). From previous studies[1] we assumed the effectiveness of antibiotics to be 85%; thus SI=0·85×6=5·1. We assumed that each patient would take prophylaxis of 3 g daily for one year; thus C_{ab} for 80 patients=kr 864 000 (£77 143) for flucloxacillin (3×kr 10×360×80) and kr 950 400 (£84 857) for cephalexin (3×kr 11×360×80). The

Direct medical costs (Swedish kronas) of treatment for patients with and without infection after knee arthroplasty, Sweden, 1988

	Operations		Days in hospital		Visits as outpatient		Radiographic examinations		Days of antibiotic treatment		Total cost (kr)
	Mean No	Cost (kr)	Mean No	Cost (kr)	Mean No	Cost (kr)	Mean No	Cost (kr)	Mean No	Cost (kr)	
Patients without infection	1	26 526	14	18 312	3	1 305	3	1 260	14	465	47 868
Patients with infection	3·3	74 962	145	243 384	16	7 227	12	5 392	1 328	27 201	353 951