■ ASPECTS OF CURRENT MANAGEMENT

HIV/AIDS in trauma and orthopaedic surgery

W. J. Harrison

*From the Beit Trust
Cure International
Hospital, Blantyre,
Malawi*

Immune deficiency associated with pneumocystis carinii pneumonia and Kaposi's sarcoma was first recognised in the United States in 1981. The causative virus, now known as the human immunodeficiency virus (HIV), was identified in 1983 by Barre-Sinoussi, Montagnier and colleagues at the Institut Pasteur, Paris.^{1,2} The resulting disease has been known as acquired immune deficiency syndrome (AIDS).

In 1983 Bayley³ described aggressive cases of Kaposi's sarcoma in Zambia. In the same hospital in Lusaka, Jellis⁴ highlighted the musculoskeletal manifestations of HIV-AIDS.

The author's interest in the relation of HIV to the practice of orthopaedic and trauma surgery began during post-graduate training in Bulawayo, Zimbabwe in 1994, and has continued in Blantyre, Malawi since 1999.

HIV is a retrovirus which encodes its genome in RNA and transcribes genome copies in DNA using the enzyme reverse transcriptase. This occurs within host cells such as the human CD4 (T helper) lymphocyte. HIV is

marked by a fall in the CD4 cell count with an associated decrease in immunity, particularly in humoral immunity.

Antiretroviral therapies such as nucleoside analogues and protease inhibitors reduce the viral load in the host serum and restore the numbers of host CD4 cells. The infected individual is not cured but their immunity is at least partially restored.

Classification

The most widely used classification originates from the Center for Disease Control in the United States.⁵ The 1993 system combines clinical features and laboratory findings to divide infected patients into three clinical (A, B and C) and three laboratory categories (1, 2 and 3). The combined effect is a potential of nine groupings (Table I).

The orthopaedic literature has tended to favour the WHO staging system⁶ which groups infected individuals into four stages according to the clinical features (Table II). Further categorisation can be made into three laboratory-

Table I. Center for Disease Control 1993 classification⁵

	Clinical category A [*]	Clinical category B [†]	Clinical category C [‡]
1. CD4 > 500	A1	B1	C1
2. CD4 200 to 499	A2	B2	C2
3. CD4 < 200	A3	B3	C3

* asymptomatic HIV infection; persistent generalised lymphadenopathy; acute HIV infection with accompanying illness

† bacillary angiomatosis; candidiasis, oropharyngeal or vulvovaginal; cervical dysplasia; constitutional symptoms > 1 mth; hairy leukoplakia, oral; herpes zoster; idiopathic thrombocytopenic purpura; listeriosis; pelvic inflammatory disease; peripheral neuropathy

‡ AIDS defining conditions: oesophageal candidiasis; cytomegalovirus retinitis; mycobacteriosis; Kaposi's sarcoma; pneumocystis carinii pneumonia; toxoplasmosis of brain; pulmonary tuberculosis

Table II. World Health Organisation (WHO) staging system⁶ for HIV infection and disease

WHO stage	Characterised by:	Examples*
I	Acute (primary) HIV infection, or latent, asymptomatic, or persistent generalised lymphadenopathy	Acute seroconversion illness in some patients
II	Cutaneous manifestations	Herpes zoster. Seborrheic dermatitis. Recurrent URI. < 10% body weight loss
III		Pulmonary TB < 1 yr ago. Severe bacterial infections. Weight loss > 10%. Chronic diarrhoea > 1 mth.
IV	AIDS defining illnesses	Pneumocystis carinii pneumonia, toxoplasmosis, cryptosporidiosis, CMV disease or retinitis

* URI, upper respiratory tract infection; CMV, cytomegalovirus

■ W. J. Harrison, FRCS (Trauma & Orth), Consultant Orthopaedic Surgeon, Cure International & Honorary Senior Lecturer Beit Trust Cure International Hospital, P.O. Box 31236, Chichiri, Blantyre 3, Malawi, Central Africa.

Correspondence should be sent to Mr W. J. Harrison; e-mail: harrisons@africa-online.net

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Table III. Combined WHO clinical and laboratory staging

Clinical stage	CD4 count (cells/mm ³)		
	< 200	200 to 500	> 500
1	1A	1B	1C
2	2A	2B	2C
3	3A	3B	3C
4	4A	4B	4C

based groups according to the CD4 count. Thus a total of 12 groupings may be derived (Table III).

It is important that clinicians treating individuals infected with HIV are familiar with one or other classification as the musculoskeletal manifestations of HIV occur in different stages,⁷ and the outcome after surgery is also influenced by the stage of the disease.⁸

Trauma

The issues of particular concern are the influence of HIV on the outcome and treatment of polytrauma, open fractures and closed fractures. In all the areas discussed, the published research is based on individuals not receiving anti-retroviral therapy and the literature will doubtless be updated in the coming years.

Polytrauma. There is no literature comparing the outcome after polytrauma in symptomatic HIV-positive patients to that in previously healthy controls. However, HIV is reported as a significant prognostic indicator for a worse outcome in acute injury to the lung and adult respiratory distress syndrome patients in intensive care.⁹

It is reasonable to suppose that symptomatic HIV-positive patients are more susceptible to secondary infection after polytrauma. Furthermore, the impaired nutritional status of some individuals infected with HIV will influence a negative outcome in catabolic phases after polytrauma.

Closed fractures. There is an increasing amount of information concerning the management of closed fractures and some guidelines can be given. The main problems are the risk of wound infection after internal fixation, late sepsis around implants, union of the fracture and the functional outcome.

The early literature suggested that HIV-positive patients, in particular those with symptoms of HIV, had an increased risk of wound sepsis after internal fixation of fractures. Hoekman et al¹⁰ found a rate of infection of 24%, and Jellis⁴ of 40% in symptomatic patients. These studies highlighted a potential problem, but needed refinement. The particular areas of concern were the failure to use prophylactic antibiotics,¹⁰ a high rate of infection in controls⁴ and the retrospective nature of the studies,¹¹ which were not blind and in which the definition of infection varied.

Harrison, Lewis and Lavy¹² described a prospective, controlled, single-blind trial with a standard method of scoring the wound.¹³ The initial study was of 41 HIV-positive patients and a further trial is in progress. Our study indicated a rate of infection of 3.5% in HIV-positive

patients whether or not they had symptoms, using a strict definition of infection. Internal fixation of open fractures had a less good outcome. We concluded that the outcome was more dependent on wound contamination than on the immune status of the patient.

By employing an effective dose of prophylactic antibiotics such as a first generation cephalosporin, a clean operating environment, strict operating theatre discipline and careful soft-tissue handling, a good outcome can be expected even in those with CD4 counts below 200 cells/mm³. The organism causing infection has most commonly been *Staphylococcus aureus*. Unusual bacteria, and fungal infections have been reported, but are uncommon.

Open fractures. In open fractures, where contamination has already occurred, the frequency of wound infection is high in all published series.^{4,11,14} In our study,¹² it was 42% in HIV-positive patients compared with 11% in controls. Nevertheless, in most cases the infection settled after antibiotics and/or revision surgery with a satisfactory outcome. Only one patient had a deep chronic infection.

Open fractures of the tibia present a particular challenge. Harrison et al¹⁴ described 27 such patients, seven of whom were HIV-positive; five developed deep wound sepsis. O'Brien and Denton¹⁵ found a similar rate of wound sepsis in open tibial fractures in HIV-positive patients. All fractures of the tibia in our patients were stabilised with a monolateral external fixator. This method has proved safe. Problems at the pin site were more common but manageable in our study. We were able to salvage septic cases in severely immune-compromised patients, achieving union and a good functional outcome, but this required extensive surgery.

Union of the fracture. This may be adversely affected by HIV. The altered inflammatory response of the immune-compromised patient may mediate such a difference. We noted a preponderance for HIV-positive patients in those with delayed or non-union following treatment by internal fixation. Nevertheless, we have been able to achieve union after stable internal fixation and grafting with autologous bone.

Summary of the strategy. We prefer to treat patients on the merits of their injuries in the context of the resources and expertise available. In populations with a high seroprevalence of HIV it is worth screening all patients with open fractures for HIV, with the aim of avoiding internal fixation where possible. Nevertheless, the established priorities of early and adequate debridement with satisfactory stabilisation of the fracture continue to hold. Preoccupation with the HIV status must not be allowed to delay initial treatment.

Late sepsis. For those patients who do undergo internal fixation of a fracture, the clinician must decide whether to remove the implant. There may be a risk of late sepsis around implants as the immunity of the patient wanes and the disease advances. This has been seen both following trauma⁴ and arthroplasty.¹⁶ Some such infections result

from activation of latent bacteria and others may represent late haematogenous seeding.

We have followed our patients with implants to one year and in that limited follow-up period no 'late sepsis' has been seen.¹⁷ This suggests that implants can remain *in situ* long enough to allow complete union, but does not provide adequate information regarding late sepsis. We remain uncertain as to whether all implants should be removed in all HIV-positive or at-risk patients. Only further studies will guide such decisions.

Arthroplasty

Total joint arthroplasty is widely used, particularly in developed countries, as a pain-relieving operation following inflammatory or degenerative joint disease. Surgery involves the implantation of stainless steel, super-alloys, polyethylene, or ceramic components which may be supported by polymethylmethacrylate bone cement. Unlike implants for trauma which become superfluous after union of the fracture, arthroplasties must remain *in situ*.

Arthroplasty in haemophiliacs. Worldwide, arthroplasty is most commonly indicated in degenerative arthropathy in elderly patients. Arthroplasty in HIV-positive patients has been less common because they are generally young and most HIV occurs in developing countries where arthroplasty is not commonly available. The exception to this has been with haemophiliac patients in developed countries, many of whom contracted HIV from contaminated transfusion of factor VIII between 1979 and 1985. They suffer arthropathy as part of their haemophiliac disease. Most of the information on HIV and arthroplasty comes from this group of patients.

As with internal fixation of fractures, the complications of most concern are early and late sepsis around the implant. Haemophiliacs with HIV are probably a special group in that they are prone to bleeding around their joints. They may also suffer bacteraemia associated with their regular factor transfusions. Both these factors may increase the risk of sepsis, particularly late sepsis, in haemophiliacs in comparison with their non-haemophiliac HIV-positive counterparts. Haemophiliacs who are HIV negative suffer an increased rate of complications, including infection,^{18,19} following arthroplasty.

There is an increased rate of sepsis after arthroplasty in HIV-positive haemophiliacs. This increases year on year and reflects the duration of follow-up. In a large retrospective multicentre study, Hicks et al¹⁶ reported a rate of deep sepsis of 18.7% (17/91) after primary procedures, and 36.3% (4/11) after revision procedures. The mean follow-up was 5.7 years. In this study, the rate of sepsis-free survival was 95% at one year, falling to 85% at five years and 55% at 15 years.

Other studies vary in detail^{18,20,21} but all larger series show an increased infection rate. No evidence has emerged that major joint surgery accelerates the progression of HIV²² or precipitates a decline in the CD4 count.

Arthroplasty in non-haemophiliac HIV-positive patients. Whilst degenerative arthropathy is generally uncommon in HIV-positive patients, these patients do suffer from inflammatory arthropathy and from avascular necrosis,^{23,24} sometimes associated with antiretroviral therapy. These may be indications for arthroplasty, as may post-traumatic arthropathy. At present no conclusions can be drawn about joint replacement in non-haemophiliac HIV-positive patients. In our hospital we have undertaken four total hip replacements in two patients with bilateral avascular necrosis. One had a CD4 count of less than 100 cells/mm³. No sepsis has developed in these patients with a mean follow-up of two years.

For the reasons outlined above, the risk of early and late infection in these patients may be expected to be higher than that in healthy individuals, but below that in haemophiliacs with HIV.

A higher incidence of aseptic loosening has been reported for arthroplasties undertaken for avascular necrosis.²⁵ Aseptic loosening and osteonecrosis are themselves both independent risk factors for late sepsis.²⁶

Conclusions

Caution is necessary in the surgical management of patients with HIV but intervention can be very beneficial. The addition of antiretroviral therapy promises to bring further benefit, and will improve the current outcomes.

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