REVIEW ARTICLE

Total Joint Arthroplasty in Human Immunodeficiency Virus Positive Patients

Stephanie Swensen, BSc[1], Ran Schwarzkopf, MD, MSc[2]

[1]Department of Orthopaedic Surgery, New York University Hospital for Joint Diseases, New York, New York, USA; and [2]Department of Orthopaedic Surgery, University of California Irvine, Irvine, California, USA

Recent advances in the medical management of patients with human immunodeficiency virus (HIV) have led to improvement in their life expectancy. The growing numbers of HIV-positive patients are now living long enough to develop end-stage arthritis, as well as other long-term musculoskeletal complications of HIV infection and treatment. This has resulted in an increased demand for total joint arthroplasty among these individuals. However, the safety and outcomes of such procedures are frequently questioned in published reports. Although increased complication rates have often been reported, most studies have reported on joint arthroplasties in HIV patients with hemophilia. The most widely reported complications in both HIV-negative and positive hemophiliac patients are aseptic loosening and postoperative infection. A possible relationship between the rate of these complications and cluster of differentiation (CD4) lymphocyte count has also been proposed. In addition to hemophilia, other factors frequently comorbid with HIV infection, such as intravenous drug use, can further complicate the clinical outcomes of these individuals following total joint replacement procedures. Physicians treating HIV positive patients must remain aware of the risks and outcomes of total joint surgery in this group when counseling them on treatment options.

Key words: Arthroplasty; Human immunodeficiency virus; Treatment outcome

Introduction

The human immunodeficiency virus (HIV) causes systemic infection with diverse multi-organ system manifestations. The incidence of HIV in the USA is steadily rising, an estimated 56,300 Americans becoming infected each year[1]. There have been numerous advances in the treatment of HIV since the advent of highly active antiretroviral therapy (HAART) in 1991. HAART treatment has altered the course and nature of the disease, resulting in significant reductions in HIV-related morbidity and mortality[2]. Combination antiretroviral therapy, surveillance of cluster of differentiation (CD)4+ T-cell counts and HIV RNA viral load has brought about a dramatic increase in life expectancy among HIV-infected patients, thus contributing to the overall increase in individuals living with HIV[3].

The musculoskeletal system is particularly affected by HIV infection. Musculoskeletal symptoms, such as arthralgias and myalgias, commonly occur during the acute phase of this infection, often being among the first clinical indications of the presence of this disease[4]. Numerous well-known musculoskeletal complications of HIV have been documented, including reactive arthritis, psoriatic arthritis, HIV-associated arthritis, musculoskeletal tumors, opportunistic bone infections, osteonecrosis, and osteopenia[4–7]. Many authors have suggested that the musculoskeletal manifestations of the disease are a result of a multitude of confounding factors[4–7]. In addition to the disease process itself, antiretroviral treatments contribute to the musculoskeletal pathology of HIV-positive patients. Development of osteonecrosis appears to be particularly associated with HIV infection and treatment therapies[8,9]. Morse *et al.* reported a 100-fold greater risk of developing osteonecrosis in HIV-positive patients than in the general population[10]. Furthermore, the increased incidence of osteonecrosis correlates with increased use of antiretroviral drugs such as protease inhibitors and corticosteroids[6,7,10,11]. The most frequently involved sites of osteonecrosis in HIV-infected

Address for correspondence Ran Schwarzkopf, MD, MSc, 101 The City Drive South Pavilion III, Building 29A, Orange, California, USA 92868 Tel: 001-714-4567012; Fax: 001-714-4567012; Email: schwarzk@gmail.com

Disclosure: Each author certifies he or she has no commercial associations that might pose a conflict of interest in connection with the submitted article.

Received 26 February 2012; accepted 30 July 2012

Orthopaedic Surgery

TABLE 1 (CDC) Classification Laboratory Categories in 1993

		Clinical category		
Category	CD4+ T-lymphocyte count	A (asymptomatic)	B (symptomatic)	C (AIDS-indicator conditions)
1	≥500 cells/μL	A1	B1	C1
2	200–499 cells/μL	A2	B2	C2
3	<200 cells/μL	A3	B3	C3

individuals are the hip, knee and shoulder and bilateral joint involvement is common[12].

Improvements in the medical management of HIV patients have lead to an increasing demand from them for total joint arthroplasty. Patients now live long enough to experience the clinical sequelae of long-term HIV infection and its treatment, as well as age-related degenerative joint disease. It is important to elucidate the potential benefits and complications of total joint arthroplasty in this group that is regarded as high-risk. Previous studies have focused on total joint arthroplasty in HIV-positive hemophiliac patients[13–22]. In these patients, pre-existing hemophiliac arthropathy is further complicated by HIV infection. The commonest complications reported in this patient group are post-operative infection and aseptic loosening[15,16,22,23]. Despite the concentration on hemophiliac HIV-positive patients, studies by Haberman *et al.* have demonstrated that non-hemophiliac HIV-positive patients appear to have the same functional outcomes after surgery as do HIV-negative patients[22]. Outcomes also appear to be affected by the CD4+ T cell count and viral load at the time of surgery[15,24]. Decisions to perform total joint arthroplasty procedures should therefore be individualized, taking into consideration the CD4+ cell count, viral load, medical management and overall state of health of the patient.

Classification

Clinical staging systems are useful for assessing the prognosis and determining treatment of HIV-infected patients. An understanding of these systems is particularly important for orthopedic surgeons who want to plan surgery for these patients. The Center for Disease Control (CDC) and the World Health Organization (WHO) created the two most widely utilized HIV classification systems. The 1993 CDC system allocates patients to one of three laboratory categories according to CD4+ T-lymphocyte counts per microliter of blood (Table 1). The CDC classification proposes three clinical categories based on the presence or absence of certain symptomatic conditions[25] (Table 2).

The WHO staging system is more commonly used by orthopedic surgeons[26]. This classification system divides patients into the following four clinical categories: stage 1 (asymptomatic), no symptoms or persistent generalized lymphadenopathy; stage 2 (mild disease), <10% weight loss, and cutaneous manifestations; stage 3 (moderate disease), >10% weight loss, severe bacterial infections, and chronic diarrhea > month; and stage 4 (severe disease, AIDS), *Pneumocystis jirovecii* pneumonia, Kaposi's sarcoma, HIV wasting syndrome, toxoplasmosis, and cryptosporidiosis.

Pathophysiology

Osteonecrosis is the leading reason for total joint replacements in HIV-positive patients. The incidence of osteonecrosis among HIV-positive patients has steadily increased since the first reported case in 1990, the annual incidence ranging from 0.08%–1.33%[27–29]. Although the exact cause of osteonecrosis in HIV-infected individuals has yet to be elucidated, many predisposing factors has been implicated[28–34]. Some of the most frequently reported risk factors for alterations in blood flow to bone are alcohol, corticosteroids, hyperlipidemia, cigarette smoking, the presence of antiphospholipid and anticardiolipid antibodies, and the duration of HIV infection and antiretroviral therapy[28–34]. Although many authors suggest that osteonecrosis is caused by the combined effects of a variety of risk factors that interrupt blood supply to bone, others offer observations supporting the possibility that HIV infection alone causes osteonecrosis[8,12,25]. Ries *et al.* reported that a significantly greater proportion of HIV positive patients with nontraumatic osteonecrosis have no known associated risk factors than do patients who are HIV negative[12].

Corticosteroid use and hyperlipidemia are common contributing factors to development of osteonecrosis in HIV-infected and other patients. Steroids modify bone marrow stromal cell differentiation and bone metabolism, causing

TABLE 2 (CDC) Classification Clinical Categories in 1993

Clinical category	Symptomatic conditions
A	Asymptomatic HIV infection, persistent generalized lymphadenopathy, acute (primary) HIV infection with accompanying illness or history of acute HIV infection
B	Bacillary angiomatosis, candidiasis, cervical dysplasia, constitutional symptoms, hairy leukoplakia, herpes zoster, idiopathic thrombocytopenic purpura, listeriosis, pelvic inflammatory disease, peripheral neuropathy
C	AIDS defining illnesses: Kaposi's sarcoma, *Pneumocystis* pneumonia, toxoplasmosis

fatty infiltration of the bone marrow and resultant obstruction of blood flow within the bone. These events result in increased intraosseous pressure. Additionally, steroids may induce fat embolization of small vessels[26–28]. Corticosteroids are often used in the management of HIV-related illnesses, such as central nervous system (CNS) toxoplasmosis and *Pneumocystis* pneumonia[11,31,32]. Miller *et al.* confirmed this association and concluded that even short courses of corticosteroid therapy may substantially increase the risk of osteonecrosis[11]. Hyperlipidemia, an established consequence of HIV infection and HAART regimens, is strongly involved in atherosclerotic pathways and is considered a causative factor in steroid-induced osteonecrosis. A number of studies have correlated high serum cholesterol concentrations and use of lipid lowering agents with the pathophysiology of osteonecrosis[12,28–32].

Additional characteristic features of HIV infection, including increased concentrations of antiphospholipid antibodies and other thrombophilic factors, have been associated with development of osteonecrosis in HIV-positive patients. Antiphospholipid antibodies are commonly present in HIV-infected patients and are a predisposing factor for venous and arteriolar thrombosis. In a recently published report, the estimated prevalence of antiphospholipid antibodies in HIV-positive patients was 44.4%, with an incidence of clinical manifestations of 13.3%[13,14]. The role of these antibodies in development of osteonecrosis in patients with systemic lupus erythematosis has been well documented and similar mechanisms have been proposed for HIV positive patients[29,31]. In addition to antiphospholipid antibodies, other autoantibodies and thrombophilic factors have been implicated in thromboembolic complications of HIV infections. De Larrañaga *et al.* recognized the frequency of thrombophilia in HIV-positive patients, documenting abnormalities in antithrombin, protein C, and protein S[34].

Recently, researchers have focused on the potential role of antiretroviral treatments, particularly protease inhibitors, in the pathophysiology of osteonecrosis. Although the exact mechanisms by which HAART treatment may contribute to development of osteonecrosis remain unknown, associations between protease inhibitors and traditional risk factors for osteonecrosis have been identified[8,10,11,28]. Hypertriglyceridemia due to HAART treatment is a frequently suggested possible mechanism for development of osteonecrosis. Protease inhibitors may also enhance the effects of corticosteroid therapy through cytochrome P450 interactions. A recent study by Penzak *et al.* demonstrated that a significantly increased prednisolone concentrations occurred in healthy subjects who were treated with ritonavir[9]. Despite the many reported associations between HAART treatment and osteonecrosis, it is important to recognize that numerous cases of osteonecrosis were reported in HIV positive patients years before the introduction of HAART treatment, thus treatment regimens for HIV cannot be the sole cause of this pathology. A formative conclusion is that osteonecrosis is of multifactorial etiology and has a high prevalence among HIV positive patients, ultimately resulting in an increased need for total joint arthroplasties in this patient group.

Current Outcomes of Total Joint Arthroplasty in HIV Positive Patients

The increasing rate of HIV infections worldwide has lead to an increase in assessment of the outcomes of orthopedic interventions in HIV-positive patients. The outcomes and complications of total joint arthroplasties in these patients have been of particular concern because of the increasing demand for these procedures as described above. In addition to functional results, many of these studies have focused on the prevalence of post-operative infections among HIV-positive individuals. Historically, HIV has been considered an independent risk factor for infection; therefore, many have questioned the safety and benefits of performing elective surgical procedures with a high potential for postoperative infection in these patients. However, numerous recent studies of total joint arthroplasty in HIV-positive individuals have challenged this notion.

Most studies assessing outcomes of total joint arthroplasties in HIV-positive individuals have reported on HIV-positive hemophiliac patients. Hemophiliac patients have a considerably increased risk of joint degeneration because of repeated intra-articular and periarticular hemorrhage. The risk of joint arthropathy is further compounded by the increased rates of HIV infections in these patients caused by the use of contaminated blood products between the years 1979 and 1985. Contamination of transfusions of factor VIII lead to seroconversion of an estimated 80% of hemophiliac patients during that time. Prior to the widespread use of HAART treatment, early studies by Weidel *et al.*[35] and Gregg-Smith *et al.*[36] examined the incidence of infection in HIV-positive hemophiliac patients following total knee replacement surgery. In a 1989 study, Weidel *et al.* demonstrated a progressive increase in acute infections following this procedure[35]. The results of a 1993 study by Gregg-Smith *et al.*[36] supported these findings and deterred many individuals from undergoing total joint replacement surgery[19,33,34].

Later studies by Hicks *et al.*[15] and other researchers further demonstrated the increased risks associated with total joint arthroplasties in HIV infected hemophiliac patients and suggested a possible relationship between the rate of these complications and CD4 lymphocyte count. In a large multicenter retrospective study, Hicks *et al.* analyzed the outcomes of 102 replacement arthroplasties in 73 HIV-positive hemophiliac patients[15]. During a median follow-up of five years, the overall rate of deep surgical site infection was 18.7% for primary procedures and 36.3% for revisions. The mean preoperative CD4 count was less than 0.2×10^9/L in 62.5% of the infected group, compared with 16.7% of the individuals in the non-infected group. Ragni *et al.*[24] also demonstrated an elevated rate of post-operative infections in knee and hip arthroplasty procedures in HIV-positive hemophiliac patients with CD4 counts below 0.2×10^9/L. A 1995 retrospective survey of 115 hemophilia centers in the USA reported an infec-

tion rate of 15%, which the authors noted is three times greater than the current risk of surgical site infection among HIV-negative arthroplasty patients.

Current large-scale studies have failed to demonstrate an increased risk of complications after total joint replacements in this patient group. Powell *et al.* examined the rate of deep infections following total knee and hip arthroplasties in HIV-positive and HIV-negative patients with hemophilia from 1975 to 2002[37]. Primary joint infections occurred in three of the 30 joints of HIV-positive patients, compared with two of 21 joints of HIV-negative patients. This study did not find that the relative risk of deep infection in patients with HIV was increased ($RR = 1.49$), leading the authors to conclude that total joint replacement is a reasonable option for individuals with hemophilic arthropathy and concomitant HIV infection. Studies by Unger *et al.* also supported the relative safety of these orthopedic procedures in HIV-positive hemophiliac patients. In their study of 26 knee arthroplasties performed in 15 patients with HIV and hemophilia A, all patients had a functional improvement following surgery and no surgical site infections occurred during a 6.4-year follow-up period[19].

Later studies have focused on the outcomes after total joint arthroplasty procedures in non-hemophiliac HIV-positive patients. In a study of total knee and hip arthroplasties in 21 HIV-positive patients, Parvizi *et al.* found a markedly high rate of post-operative complications[16]. At follow-up evaluation, 12 of the 21 arthroplasties required revision due to deep infection and aseptic loosening. They reported a statistically significant association between the immune status of the patients and occurrence of deep infection (six joints). In this study, Parvizi *et al.* found a high prevalence of *Staphylococcus aureus*, *Staphylococcus epidermidis*, and *Pseudomonas aureginosa* among the deep infections[16]. From our experience, the above organisms are the commonest in periprosthetic infections in HIV-positive patients. Treatment algorithms should be the same as used with HIV-negative patients, two-stage revision surgery being the gold standard. We do recommend longer treatment with intravenous antibiotics: up to 6 months instead of 6 weeks. Patients' medical condition, including CD4 count and viral load, should be optimized. When cultures are taken, emphasis should be put on longer incubation periods, and assessment for tuberculosis and uncommon organisms.

Similar to previous studies, Habermann *et al.* noted an increased total complication rate of 12.7% in HIV-positive hemophiliac and non-hemophiliac patients undergoing total joint replacements[22]. However, these researchers reported that the non-hemophiliac HIV-positive subgroup had the same functional outcome after surgery as HIV-negative patients[22]. Mahoney *et al.* also demonstrated good functional outcomes in a recent study of total hip arthroplasty in non-hemophiliac patients with HIV[38]. Of the 40 patients studied, only one patient with a known intravenous drug abuse history developed a clinically significant antibiotic-sensitive *Staphylococcus aureus* infection 3 years after total hip arthroplasty.

In addition to hemophilia, other frequent comorbidities with HIV infection, such as intravenous drug use, further complicate the clinical outcomes following total joint replacement procedures. Lehman *et al.* determined the rate of deep periprosthetic infection in patients with HIV and intravenous drug use (IVDU) after total joint arthroplasty[23]. In this study, there was a surgical site infection rate of 40% in patients with concomitant HIV infection and IVDU. No infections occurred in the four HIV-positive patients without associated comorbidities (hemophilia, IVDU). The results of this study further suggest the need to assess the risks and benefits of total joint arthroplasty procedures on an individualized basis.

Conclusion

Currently available published reports about HIV-positive patients and total joint arthroplasty provide conflicting data. While some authors have reported high complication and revision rates (up to 57%[16]) for total joint arthroplasties performed in HIV-positive patients, most of these series have included a high percentage of hemophiliac HIV-positive patients. In the absence of HIV, hemophiliac arthropathy is associated with complications after total joint arthroplasties; and is therefore a major confounding factor in the outcomes reported in previous studies. Future research should focus on outcomes in HIV-positive patients who are not hemophiliac in order to help elucidate the risks specific to HIV-positive patients undergoing total joint arthroplasty.

Our current practice is to evaluate each patient individually and strive to optimize the general medical condition of HIV-positive patients prior to surgery. Patient specific considerations, such as a history of intravenous drug abuse, CD4 count, HIV viral load, clinical classification, and the patient's overall state of health influence our decisions to recommend total joint replacement procedures. Patients should be well informed of the increased risks and incidence of perioperative complications. We believe that total joint arthroplasty may improve the quality of life of these patients.

References

1. Centers for Disease Control and Prevention (CDC). HIV prevalence estimates–United States, 2006. MMWR Morb Mortal Wkly Rep, 2008, 57: 1073–1076.

2. Chen LF, Hoy J, Lewin SR. Ten years of highly active antiretroviral therapy for HIV infection. Med J Aust, 2007, 186: 146–151.

3. Esté JA, Cihlar T. Current status and challenges of antiretroviral research and therapy. Antiviral Res, 2010, 85: 25–33.

4. Biviji AA, Paiement GD, Steinbach LS. Musculoskeletal manifestations of human immunodeficiency virus infection. J Am Acad Orthop Surg, 2002, 10: 312–320.

5. Takhar SS, Hendey GW. Orthopedic illnesses in patients with HIV. Emerg Med Clin North Am, 2010, 28: 335–342.

6. Govender S, Harrison WJ, Lukhele M. Impact of HIV on bone and joint surgery. Best Pract Res Clin Rheumatol, 2008, 22: 605–619.

7. Marquez J, Restrepo CS, Candia L, *et al*. Human immunodeficiency virus-associated rheumatic disorders in the HAART era. J Rheumatol, 2004, 31: 741–746.
8. Mary-Krause M, Billaud E, Poizot-Martin I, *et al*. Risk factors for osteonecrosis in HIV-infected patients: impact of treatment with combination antiretroviral therapy. AIDS, 2006, 20: 1627–1635.
9. Penzak SR, Formentini E, Alfaro RM, *et al*. Prednisolone pharmacokinetics in the presence and absence of ritonavir after oral prednisone administration to healthy volunteers. J Acquir Immune Defic Syndr, 2005, 40: 573–580.
10. Morse CG, Mican JM, Jones EC, *et al*. The incidence and natural history of osteonecrosis in HIV-infected adults. Clin Infect Dis, 2007, 44: 739–748.
11. Miller KD, Masur H, Jones EC, *et al*. High prevalence of osteonecrosis of the femoral head in HIV-infected adults. Ann Intern Med, 2002, 137: 17–25.
12. Ries MD, Barcohana B, Davidson A, *et al*. Association between human immunodeficiency virus and osteonecrosis of the femoral head. J Arthroplasty, 2002, 17: 135–139.
13. Galrão L, Brites C, Atta ML, *et al*. Antiphospholipid antibodies in HIV-positive patients. Clin Rheumatol, 2007, 26: 1825–1830.
14. Santos JL, Cruz I, Martin Herrero F, *et al*. Recurrent coronary thrombosis, factor V Leiden, primary antiphospholipid syndrome and HIV. Rev Esp Cardiol, 2004, 57: 997–999.
15. Hicks JL, Ribbans WJ, Buzzard B, *et al*. Infected joint replacements in HIV-positive patients with haemophilia. J Bone Joint Surg Br, 2001, 83: 1050–1054.
16. Parvizi J, Sullivan TA, Pagnano MW, *et al*. Total joint arthroplasty in human immunodeficiency virus-positive patients: an alarming rate of early failure. J Arthroplasty, 2003, 18: 259–264.
17. Kelley SS, Lachiewicz PF, Gilbert MS, *et al*. Hip arthroplasty in hemophilic arthropathy. J Bone Joint Surg Am, 1995, 77: 828–834.
18. Thomason HC 3rd, Wilson FC, Lachiewicz PF, *et al*. Knee arthroplasty in hemophilic arthropathy. Clin Orthop Relat Res, 1999, 360: 169–173.
19. Unger AS, Kessler CM, Lewis RJ. Total knee arthroplasty in human immunodeficiency virus-infected hemophiliacs. J Arthroplasty, 1995, 10: 448–452.
20. Vastel L, Courpied JP, Sultan Y, *et al*. Knee replacement arthroplasty in hemophilia: results, complications and predictive elements of their occurrence. Rev Chir Orthop Reparatrice Appar Mot, 1999, 85: 458–465.
21. Rodriguez-Merchan EC, Wiedel JD. Total knee arthroplasty in HIV-positive haemophilic patients. Haemophilia, 2002, 8: 387–392.
22. Habermann B, Eberhardt C, Kurth AA. Total joint replacement in HIV positive patients. J Infect, 2008, 57: 41–46.
23. Lehman CR, Ries MD, Paiement GD, *et al*. Infection after total joint arthroplasty in patients with human immunodeficiency virus or intravenous drug use. J Arthroplasty, 2001, 16: 330–335.
24. Ragni MV, Crossett LS, Herndon JH. Postoperative infection following orthopaedic surgery in human immunodeficiency virus-infected hemophiliacs with CD4 counts < or = 200/mm^3. J Arthroplasty, 1995, 10: 716–721.
25. From the Centers for Disease Control and Prevention. 1993 revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. JAMA, 1993, 269: 729–730.
26. Harrison WJ. HIV/AIDS in trauma and orthopaedic surgery. J Bone Joint Surg Br, 2005, 87: 1178–1181.
27. Goorney BP, Lacey H, Thurairajasingam S, *et al*. Avascular necrosis of the hip in a man with HIV infection. Genitourin Med, 1990, 66: 451–452.
28. Yombi JC, Vandercam B, Wilmes D, *et al*. Osteonecrosis of the femoral head in patients with type 1 human immunodeficiency virus infection: clinical analysis and review. Clin Rheumatol, 2009, 28: 815–823.
29. Allison GT, Bostrom MP, Glesby MJ. Osteonecrosis in HIV disease: epidemiology, etiologies, and clinical management. AIDS, 2003, 17: 1–9.
30. Mazzotta E, Agostinone A, Rosso R, *et al*. Osteonecrosis in human immunodeficiency virus (HIV)-infected patients: a multicentric case-control study. J Bone Miner Metab, 2011, 29: 383–388.
31. Brown P, Crane L. Avascular necrosis of bone in patients with human immunodeficiency virus infection: report of 6 cases and review of the literature. Clin Infect Dis, 2001, 32: 1221–1226.
32. Gutiérrez F, Padilla S, Masiá M, *et al*. Osteonecrosis in patients infected with HIV: clinical epidemiology and natural history in a large case series from Spain. J Acquir Immune Defic Syndr, 2006, 42: 286–292.
33. Tektonidou MG, Moutsopoulos HM. Immunologic factors in the pathogenesis of osteonecrosis. Orthop Clin North Am, 2004, 35: 259–263.
34. de Larrañaga G, Bottaro E, Martinuzzo M, *et al*. Thrombophilia in human immunodeficiency virus-infected patients with osteonecrosis: is there a real connection? The first case-control study. Clin Appl Thromb Hemost, 2009, 15: 340–347.
35. Wiedel JD, Luck JV, Gilbert MS. Total knee arthroplasty in the patient with haemophilia: evaluation and long-term results. In: Gilbert MS, Greene WB, eds. Musculoskeletal Problems in Haemophilia. New York: National Haemophilia Foundation, 1989; 152–157.
36. Gregg-Smith SJ, Pattison RM, Dodd CA, *et al*. Septic arthritis in haemophilia. J Bone Joint Surg Br, 1993, 75: 368–370.
37. Powell DL, Whitener CJ, Dye CE, *et al*. Knee and hip arthroplasty infection rates in persons with haemophilia: a 27 year single center experience during the HIV epidemic. Haemophilia, 2005, 11: 233–239.
38. Mahoney CR, Glesby MJ, DiCarlo EF, *et al*. Total hip arthroplasty in patients with human immunodeficiency virus infection: pathologic findings and surgical outcomes. Acta Orthop, 2005, 76: 198–203.