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# Complications related to fracture treatment in HIV patients A case report

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ABSTRACT

We present the case report of a 40-year-old woman who was HIV-positive in Highly Active Anti-Retroviral Therapy (HAART) and affected by femural pertrochanteric fracture, which was treated by endomedullary nailing. Two years after the surgical operation, the woman developed an aseptic symptomatic osteolysis around the implant. Hardware removal was resolutive. Aseptic and septic hardware mobilization, hardware removal, and implant decision in HIV patients with pertrochanteric fractures is discussed. The authors suggest close follow-up and prompt hardware removal, as soon as X-rays demonstrate healing signs, in HIV patients with fracture fixation, if general condition allows.



## Introduction

According to 2012 UNAIDS Report on AIDS epidemic, the number of people living with HIV in North America and Western Europe was 2.3 million in 2011, which is 32% more then in 2001. The spread of HIV amounts to 0.6% in the US population, and 0.4% in the Italian population [1]. A 1996 study states that between 0.2% and 8.9% of patients who reach the Emergency Room are HIV positive, and between 0.1% and 7.8% of those accepted in hospitals are HIV positive [2].

HIV-positive subjects may encounter severe complications because of cellular immunological, humoral, and non-specific deficiencies [3]. HIV-positive patients in highly active antiretroviral therapy (HAART) have a higher incidence of osteopenia–osteoporosis [4–10], osteonecrosis [11], and pathological fractures [12–14]. Yin et al. evaluated the incidence and predictors of fractures in 4640 HIV patients and found that fracture rates were higher within the first 2 years after initiation of antiretroviral therapy (ART) compared with subsequent years, but continuation of ART was not associated with increasing fracture rates in these relatively young HIV-positive individuals [15].

Calmy et al. in 2013, found that premenopausal HIV-positive women had trabecular and cortical bone alterations compared with those who were HIV-negative, and that this could contribute to bone fragility [16].

HAART has had a considerable impact on the natural history of AIDS as it has improved life expectancy and quality of life [4–6,17]. One consequence of the combination of long active life expectancy and bone deficiencies is that HIV-infected patients are more likely to need surgical fracture treatment in their lifetime.

In the past, indications or contraindications for surgery were often based on personal preconceptions instead of an objective clinical judgement [3], with some surgeons reluctant to operate on HIV patients due to unjustified fear of complications or transmission of the virus [2].

Fortunately, HIV patients with fractures are now treated surgically in the same way as patients who are not infected with HIV, but the possible rise of long-term or late complications of HIV infection and their treatment after a hardware insertion may become an issue for the future.

Yoo et al. in 2010, reported that there were no significant complications in HIV patients after hip surgery when their preoperative immunity was optimal, and that the safety of medical personnel could be assured when the operation is performed in line with the guidelines of HIV infection control [18].

This case report introduces the concept of possible hardware-related complications and their preventive treatment in HAART-HIV patients.

## Case report

The patient was a 40-year-old hairdresser who was a former drug addict. She was HIV-positive at the age of 20 (at the time, stage B1 car. CDC), with chronic hepatitis C, affected with chronic

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gastritis and a hiatal hernia. She was in ART with stavudine 30 mg × 2, lamivudine 150 mg × 2, and nelfinavir 250 mg × 2.

On September 27, 2004, the patient was involved in a road accident. The patient was transported to the closest Trauma Centre and there underwent standard trauma screening. X-rays showed a pertrochanteric fracture, an ileo-pubic fracture and an ischio-pubic fracture, all on the left side (Fig. 1).

Three days later, the patient underwent an operation to receive a Proximal Femoral Nail (PFN, Synthes USA). The patient recovered from surgery without any complications. The patient was transferred to a long-term internal medicine ward 15 days later, where she developed an acute case of *Clostridium difficile* enteritis, and was transferred to an infection ward. She was discharged 40 days after the trauma.

Two months after the operation the patient could walk with complete-weight bearing and without pain, and went back on HAART with atazanavir, ritonavir, tenofovir, and lamivudine.

Twelve months after surgery, the patient was hospitalised in an infectious disease ward for slight fever, loss of weight and diarrhoea. The first immunological tests showed a good immunological situation with CD4 + T lymphocytes 784 cell/mmc equal to 33% of total lymphocytes, the T4/T8 lymphocyte ratio was 0.62 and HIV viral load was 2000 copies/mL; white blood cell (WBC) was 4080 cell/mmc, haemoglobin (Hb) 11.6 g/dL, platelets 278,000 plt/mmc, gamma-glutamyl transpeptidase (GGT) 52 U/L, erythrocyte sedimentation rate (ESR) 19 mm, C-reactive protein (CRP) 0.30 mg/dL, ferritin 21.9 ng/mL, and folates 3.3 ng/mL. *Salmonella typhi* was diagnosed and appropriate therapy was started. During hospitalisation, ART was suspended because the patient reported tremor in her limbs. Electromyography was within the limits, whereas the neurological check-up showed bilaterally highly excitable osteotendon reflexes. She was discharged with a prescription of gabapentin and immunological monitoring to start back on her therapy as soon as possible.

Three months later, the patient developed a case of ingravescent left coxalgia with no clinical signs of surgery side acute infection. X-rays showed osteolysis around the implant (Fig. 2), while the blood tests had the following results: WBC 20,034 cells/mmc, CD4 513 cells/mmc, and T4/T8 0.60. A slight evening fever was also present.

A bone scintigraphy with anti-granulocyte antibodies showed a focal hyperaccumulation on the greater trochanter and on the subtrochanteric region on the external side. The patient was admitted to the infectious disease ward where she underwent antibiotic therapy (levofloxacine and teicoplanine). Initially her blood tests were: VES 50 mm, CRP negative, WBC 3370 cells/mmc, platelets 204,000 plt/mmc, neutrophils 39%, lymphocytes 48%, ferritin 50 ng/mL, and CD4 + T lymphocytes 270 cells/mmc equal to 31% of total lymphocytes, in a T4/T8 lymphocyte ratio of 0.42.

The patient was discharged, but then hospitalised again due to the appearance of skin rash and hyperpyrexia. The fever decreased after antibiotic interruption and the beginning of steroid therapy. The aerobic and anaerobic blood cultures were negative, as was the urine culture. At this time, blood test results were: WBC 4260 cells/mmc, with 53% neutrophils and 35% lymphocytes, and CRP 12 mg/dL. At discharge, WBC were 6180 cells/mmc, with 46% neutrophils and 35% lymphocytes, and CRP was 4.13 mg/dL, and the patient was prescribed antibiotic therapy (Linezolid).

Twenty-four days from the beginning of antibiotic therapy, 19 months after the first surgery, pain was still present and the hardware was removed. Surgery was performed without complications. No purulent material was seen. During the surgery, swabs were collected for culture tests (aerobes and anaerobes). No germs were isolated. The patient was discharged three days after surgery with a prescription to stay completely no weight-bearing.

During the post-operative period, the patient remained apyretic without any cutaneous suffering. Two months after surgery, the patient could walk without pain with full weight-bearing. After five months, the patient was put back on HAART with atazanavir, ritonavir, tenofovir, and lamivudine.

At one-year follow up, the patient had a complete and painless range of motion. There were no signs of phlogosis, and the surgical wound looked healed. The X-rays showed signs of healing, without any further sign of osteolysis (Fig. 3).

## Discussion

This is the first report on possible aseptic mobilization by intramedullary hardware in an HIV-patient. There are two possible hypotheses for this occurrence: the first is that there was an aseptic mobilization due to osteoporosis or vascular necrosis (HIV and HAART related) and the second is that the patient was infected by bacteria of such low virulence and resistance that antibiotics were able to eradicate the infection, or at least keep it under control, before the removal of the hardware.

### *Aseptic mobilization hypothesis*

Patients infected with HIV are subject to bone demineralisation because of the complex and multi-factorial metabolic modifications that are associated with this infection and the interaction of viral and pharmacological factors [14]. Osteo-apposition and osteoclasis may become difficult to balance in HIV, and may even become osteoporosis/osteopenia (as defined by the WHO [19]) and lead to pathological fractures [6,12,14], particularly of the hip and spine [12]. Approximately 22–50% of HIV-patients in ART are thought to be affected by osteopenia and 3–21% by osteoporosis [12].

Although the patient in this case report had a high-impact trauma, she also had different risk factors for osseous demineralisation. Clinical history of opioid drug addiction may have led to secondary hypogonadism [20] and then to osteopenia or osteoporosis [14]. Furthermore, although HAART increases survival of HIV-patients, it amplifies bone stock reduction because these patients

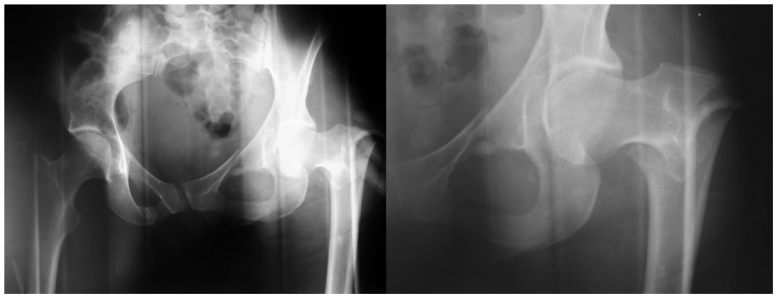

**Fig. 1.** Left hip with pertrochanteric, ileo-pubic and ischio-pubic fractures.