

HIV infection and its effects on fracture healing: a literature review

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Introduction

The Human Immunodeficiency Virus (HIV) is a virus that causes Acquired Immunodeficiency Syndrome (AIDS). The disease was discovered in 1983 in the Pasteur Institute, Paris by Barre and colleagues [1]. The causative agent is a Lentivirus, a subgroup of Retroviruses that is transmitted through body fluids. The main routes of entry include sexual intercourse, shared needles, and vertical transmission from mother to child during childbirth or breastfeeding. The World Health Organization (WHO) provides a staging system for HIV infection based on the clinical manifestations of the disease [2].

HIV/AIDS is encountered by orthopaedic surgeons when managing both orthopaedic and trauma cases. Cohen et al reported a prevalence rate of 16% amongst patients undergoing orthopaedic procedures in Zimbabwe [3]. HIV is also associated with a high incidence rate of Osteonecrosis. Chokotho et al evaluated the risk factors associated with the development of non-traumatic osteonecrosis of the femoral head in HIV positive and negative patients. They concluded that, together with other risk factors such as excessive alcohol intake, HIV infection contributes to the development of non-traumatic osteonecrosis of the femoral head [4]. Osteopenia is also known to be associated with HIV infection. Whether antiretroviral treatment contributes to this condition or not remains controversial [5]. It is therefore imperative that the surgeon familiarizes him/herself with the expected implications that the disease may have on the management of the patient. This paper focuses on the effects that the infection has on the healing process of open and closed fractures. It also attempts to explore the effects that HIV infection has on the operative treatment of fractures.

HIV/AIDS in closed fractures

A closed fracture is defined as a fracture that does not disrupt the integrity of the surrounding skin [6]. There is a minimal risk of contamination in this type of fractures. HIV infection affects the rate of union in closed fractures.

Kamat et al conducted a study to evaluate the effects of HIV infection on fracture union. They studied a group of 2,376 patients with closed ankle fractures managed conservatively with below knee casts for a minimum of 6 weeks. The first group of 829 patients were HIV negative. The second group of 729 patients were HIV positive and

categorized between WHO clinical stages I and III. The third group of 755 HIV positive patients were categorized as WHO clinical stage IV. The study found that 12.45% of the patients with WHO clinical stage IV HIV had non-union compared to 1.5% and 1.25% for HIV negative and HIV stages I to III patients respectively. The study also revealed that fracture union is delayed in the third group of patients with the majority of this occurring at 8 weeks following injury. This is in contrast with the first and second groups of patients in which the majority of unions had occurred at 4 weeks after injury. The authors concluded that fracture union rates decreases with disease severity [7]. This discrepancy in union rates could be attributed to the fact that the infection alters the Cytokine environment in HIV positive patients. Cytokines are essential in fracture healing due to their role in the inflammatory phase of this process [8].

HIV/AIDS in open fractures

Contrary to closed fractures, open fractures are fractures that disrupt the integrity of the overlying skin. This type of fractures is usually associated with infection as the compromised overlying skin provides a portal of entry for infectious agents. In HIV positive patients, open fractures are associated with complications in both wound healing and fracture union. In a prospective study conducted by Harrison et al, the outcome of open tibial fractures managed by surgical debridement and external fixation was compared between HIV positive and negative patients. Results of the study showed that 5 out of 7 HIV positive patients developed deep seated wound infection compared to 4 out of 21 in the HIV negative group. The same study also revealed reduced union rates among the HIV positive patients. 3 out of the 7 HIV positive patients had non united fractures at 6 months post injury compared to only 1 of the 27 HIV negative patients [9]. O'Brien and Denton concluded that the infection rates among HIV positive patients with open fractures treated by surgical debridement was higher compared to patients without HIV infection receiving the same treatment [10].

Internal and external fracture fixation in HIV patients

The advent of fracture fixation represented a breakthrough in the care of trauma patients. Methods of fracture fixation are broadly classified as internal or external fixation. As defined by the American Association of Orthopaedic Surgeons [6]:

- Internal fixation is the surgical insertion of a device that stops motion across a fracture or joint to encourage bony healing or fusion.
- External fixation is the stabilization of a fracture or unstable joint by inserting pins into bone proximal and distal to the injury that are then attached to an external frame.

Several studies suggest that HIV is not a contraindication to internal fixation. Bahebeck et al concluded that, with prolonged preoperative prophylaxis and HAART therapy, surgical wound infection rates in HIV positive patients may approach that of uninfected patients [11]. D'Amico & Ballon-Landa compared two groups of immunocompromised patients undergoing surgery, a group of HIV positive patients and another of diabetes mellitus patients. The study concluded that perioperative infection rates are almost the same among HIV patients on HAART and patients with controlled diabetes mellitus [12]. Bates et al argued that HIV status does not affect the likelihood of the need for secondary orthopaedic procedures following internal fixation of fractures. They also concluded that such patients do not have an increased likelihood of developing chronic postoperative osteomyelitis [13]. However it is very important to preoperatively identify the risk factors in HIV positive patients. According to Abalo et al, the clinical stage of HIV positive patients undergoing surgery influences the outcome of their surgical procedures [14].

Discussion

Untreated HIV infection delays, and sometimes prevents fracture union. The effects of the disease on fracture healing increases with the increase in its severity. It is also associated with higher infection rates of both surgical incision wounds and open fracture wounds. These complications are drastically reduced with better control of the disease through the administration of HAART. Preoperative antibiotic prophylaxis also plays a vital role in preventing postoperative infections in patients with fractures undergoing trauma procedures. With HAART and preoperative antibiotics, the orthopaedic surgical outcome of HIV positive patients approaches that of the general population.

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