

Wounds in Patients with HIV

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ABSTRACT

Highly active antiretroviral therapy has dramatically reduced morbidity and mortality among patients who are HIV-positive. A retrospective review of the authors' data separated subjects into cohorts based on HIV status and matched them for age and gender. The authors' data reveal a higher fraction of venous ulcers compared with a lower fraction of pressure ulcers in the seropositive population.

KEYWORDS: chronic wounds, HIV-positive, antiretroviral therapy, venous ulcers, pressure ulcers

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INTRODUCTION

An estimated 35.3 (32.2–38.8) million people worldwide were living with HIV/AIDS in 2012.¹ The clinical presentation of the disease can be attributed to the impairment of the immune system, primarily due to the infection of CD4⁺ helper T cells. The risk of developing opportunistic infections, especially bacterial, increases dramatically as the CD4⁺ T cell count begins to drop below 500 cells/ μ L.² With the discovery and widespread use of the highly active antiretroviral therapy (HAART) since the 1990s,³ there is now an aging HIV-positive population that experiences many of the same comorbidities as the noninfected population. Among these conditions that are of growing concern in older adult patients is the formation of chronic wounds and ulcers. Patients with HIV infection are at increased risk of developing cardiovascular disease and diabetes mellitus,⁴ which also increases the risk of developing various types of wounds and ulcers.⁵

HIV AND WOUNDS

Numerous types of ulcers exist, including, but not limited to, pressure, diabetic neuropathic, venous, and ischemic ulcers. These pose significant problems leading to pain, decreased quality of life, significant

morbidity, and prolonged hospital stay.⁶ Based on the stage or grade of a wound, certain cases will require surgical intervention to debride the necrotic tissues and avoid bacterial growth.⁷ Some perceive that surgery for HIV-positive patients poses higher risks compared with seronegative patients because their postoperative course may be complicated by poor wound healing and infection. There is no current literature to support a theory that HIV infection itself is an independent risk factor for complicated wound management. In fact, there is conflicting literature that states whether HIV and its effect on immune function play a role in surgical wound debridement. Tran et al⁸ determined that a postoperative percent CD4⁺ T cell count of less than 18 ± 3 and a preoperative to postoperative percentage CD4⁺ T cell change of 3 or greater to be independent risk factors of perioperative morbidity. In contrast, Jones et al⁹ and Avidan et al¹⁰ both determined that the most important risk factor for postoperative complications in HIV-positive patients is the general health status applied to all preoperative patients as determined by their ranking on the American Society of Anesthesiologists physical status classification system.¹¹

STUDY PURPOSE

The purpose of this study was to determine the incidence of wound formation and the prevalence of various types of wounds in HIV-positive patients compared with seronegative patients. An assessment of the complex relationship between HIV and chronic wounds

Table 1.

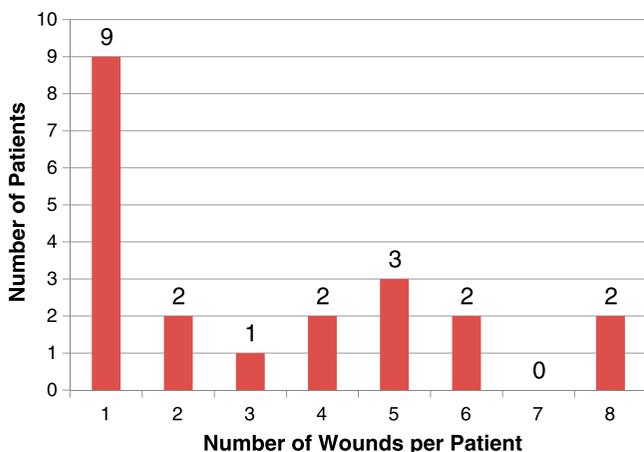
NUMBER OF PATIENTS AND WOUNDS

	Patients	No. of Wounds	Wounds per Patient
HIV positive	21	67	3.19
HIV negative	724	1967	2.72
Total	745	2034	2.73

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Figure 1.
NUMBER OF WOUNDS PER HIV-POSITIVE PATIENT



will allow clinicians to provide optimal care and improved quality of life.

METHODS

The study received approval from New York University School of Medicine's institutional review board. This is a retrospective chart review of the Wound Electronic Medical Record (WEMR) at New York University Langone Medical Center (NYULMC) from July 2008 to January 2011. The NYULMC is an 806-bed medical center located in a diverse urban setting caring for a population with a broad spectrum of medical conditions. The authors evaluated patients 18 years or older who were receiving chronic wound care at their institution. The WEMR allows digital photographs of the wound during each visit to allow objective and accurate documentation of wound management.¹² For each subject, their age, gender, type of wounds, number of wounds, and comorbidities including HIV status were recorded. Subjects were then divided into 2 cohorts, HIV-positive and HIV-negative, and were appropriately matched for their age, gender, and comorbidities. The wounds were classified into the following categories: pressure, venous, postsurgical, diabetic neuropathic, traumatic, ischemic,

inflammatory, cellulitis, and unspecified. For both cohorts, the total number of each type of wound was recorded, and the number of wounds per patient was calculated.

RESULTS

A total of 745 subjects with 2034 wounds were reviewed through the WEMR during the authors' study period. As seen in Table 1, there were a total of 724 HIV-negative patients with 1967 wounds, resulting in an average of 2.72 wounds per patient. In contrast, there were 21 HIV-positive patients with 67 wounds, resulting in an average of 3.19 wounds per patient. Figure 1 illustrates this finding by displaying the number of wounds for each HIV-positive patient. Data reveal that the number of wounds in HIV-positive patients (3.19) is not statistically significant from the matched HIV-negative patients (2.72) ($P > .05$). Table 2 shows the comparison of wound distribution in both HIV-positive and HIV-negative patients, and Figure 2 focuses on wound distribution for only HIV-positive patients. It can be observed that HIV-positive patients had 10 pressure ulcers (PrUs) (14.9%) and 26 venous ulcers (38.8%), whereas HIV-negative patients had 29 PrUs (42.6%) and 18 venous ulcers (26.5%) ($P = .0021$).

DISCUSSION

Patients with HIV/AIDS are experiencing prolonged life expectancy because of advancements in antiretroviral therapy. Consequently, they are also developing morbidities associated with aging and immobility, such as chronic wound formation. The vast majority of wounds are typically venous, diabetic neuropathic, or PrUs.¹³ These 3 types make up roughly two-thirds of all wounds in the authors' HIV-positive subjects.

The authors' data demonstrated that HIV-positive subjects tend to have a similar number ($P > .05$) of wounds per patient (3.19) compared with matched seronegative subjects (2.72). The literature is inconsistent as to whether HIV-positive patients develop wounds more commonly or are unable to heal wounds with the same ability as the seronegative population.¹⁴⁻¹⁶ HIV is often considered one of many underlying variables that can contribute to increased infection and chronic wounds in surgical patients.¹⁷ A major

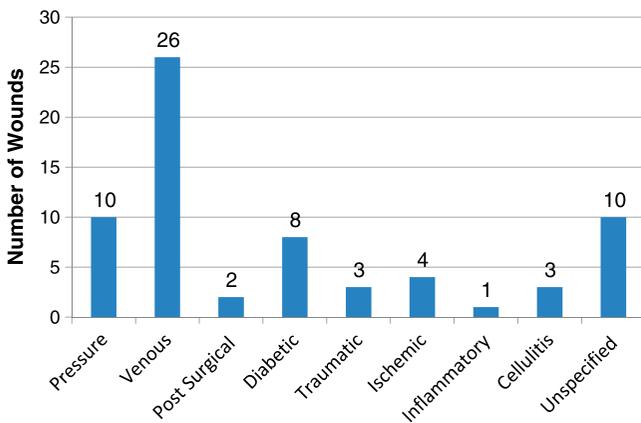
Table 2.

COMPARISON OF WOUND DISTRIBUTION BETWEEN HIV-POSITIVE AND HIV-NEGATIVE PATIENTS MATCHED BY AGE AND GENDER

Type of Wound	Pressure	Venous	Postsurgical	Diabetic	Traumatic	Ischemic	Inflammatory	Cellulitis	Unspecified
HIV	10	26	2	8	3	4	1	3	10
Non-HIV	29	18	0	10	2	0	0	2	7

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Figure 2.
WOUND DISTRIBUTION IN HIV-POSITIVE PATIENTS



concern in the HIV-positive population is the CD4⁺ T cell count, which has been known to affect wound healing. Consten et al¹⁸ established a relationship between the type and extent of the procedure, CD4⁺ count, and wound healing time. They discovered that the lower the preoperative CD4⁺ count, the more at risk the patient was for compromised wound healing.¹⁹ However, these data generally predate the availability of effective HAART introduced in 1996, when most patients experienced shortened life expectancy and severely impaired immune function.^{19–21} In the modern era of potent antiretroviral therapy, studies have shown that HIV-positive patients who have achieved immune reconstitution with higher CD4⁺ T cells and lower viral loads appear to have excellent overall outcomes.^{22,23}

The authors' data supported the findings of a difference in the type of wounds in HIV-positive patients compared with a matched set of HIV-negative patients ($P = .0021$). In addition, the HIV-positive subjects in the study were more likely to develop venous ulcers than PrUs compared with the authors' matched seronegative subjects. One hypothesis is that venous insufficiency is secondary to intravenous drug use and abuse. In the United States, more than 36% of the HIV/AIDS cases can be attributed directly or indirectly to injected drug use, including 9% of newly diagnosed cases in 2009.²⁴ Because injection sites are commonly in the lower extremities, repeated use can lead to chronic vein trauma, infections, deep vein thrombosis, and collapse of veins as well as nerve and muscle damage.²⁵ These changes enhance the risk of developing chronic venous insufficiency and impact the range of motion of the ankle. Loss of range of motion causes ankle impairment of the calf muscle pump function, which results in increased venous pressure and damage to veins, leading to venous reflux.²⁶ Although venous insufficiency is necessary, it is not sufficient to cause ulceration

alone. But coupled with a deficiency in the calf muscle pump, it can significantly increase the severity of ulceration.²⁷ Therefore, HIV cases can be linked to intravenous drug use, which leads to a greater prevalence of venous ulceration in such populations.

Additional research is required to enhance the authors' understanding of the relationship between HIV and wound pathophysiology. Future studies should focus on CD4⁺ T cell count and viral load of HIV patients to evaluate their roles in risk of infection and delayed wound healing. Severe sepsis accounts for approximately 15% of diagnoses in HIV-positive patients admitted to the intensive care unit with an increased mortality as compared with those without HIV/AIDS.²⁸ These patients were found to be less frequently on HAART and consequently have worse immune status. Recent studies have found that incidence of postoperative bacterial complications and sepsis is higher in patients with lower CD4⁺ T cell count.^{23,29} Furthermore, Horberg et al²³ found that a viral load of 30,000 copies/mL or greater was associated with 3-fold increase in risk of postoperative complications.

CONCLUSIONS

In summary, the authors report their experience with chronic wounds in HIV-positive patients at the NYULMC. As antiretroviral therapy continues to advance and life expectancy of the HIV/AIDS population increases, the incidence of wounds in these patients may be expected to increase as well. Only a paucity of data exists on HIV-related wound healing in the HAART era. Many questions concerning wound healing rates and its correlation to CD4⁺ cell counts and viral load remain unanswered. By incorporating the authors' findings on HIV-positive patients with chronic wounds, additional research can further examine the role of HIV status in the formation of chronic wounds and better predict and manage risk factors for complications, such as sepsis. The authors hope their data will lead to future investigations and advances in the field of wound management in HIV-positive patients. Such investigations may include markers of inflammation and wound healing contrasting HIV versus non-HIV patients. ●

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