ISSN: 2476 - 2415

Case Report

International Journal of Clinical & Experimental Dermatology

Pseudotumoral Chronic Genital Herpes Mimicking Squamous Cell Carcinoma: A Case Report

M. Hammouch*, H. Rachadi, F. Anejjar and S. Chiheb

Dermatology and Venereology Department, CHU Ibn Rochd, Morocco

***Corresponding Author**
Meryame Hammouch, Dermatology and Venereology Department, CHU Ibn Rochd, Morocco.

Submitted: 2026, Jan 27; **Accepted:** 2026, Feb 25; **Published:** 2026, Mar 12

Citation: Hammouch, M., Rachadi, H., Anejjar, F., Chiheb, S. (2026). Pseudotumoral Chronic Genital Herpes Mimicking Squamous Cell Carcinoma: A Case Report. *Int J Clin Expl Dermatol, 11*(1), 01-04.

Abstract

Background: *Genital herpes is a common sexually transmitted infection, usually caused by herpes simplex virus type 2 (HSV-2). In immunocompromised patients, especially those living with HIV, chronic and atypical presentations can mimic neoplastic lesions.*

Case Presentation: *We report a 48-year-old HIV-positive male with a 7-month history of an ulcerative-vegetative lesion on the glans. Antiretroviral therapy had been interrupted for one year, resulting in elevated viral load. Initial biopsies suggested no malignancy. The lesion worsened after corticosteroid therapy for suspected pyoderma gangrenosum. Cytodiagnosis (Tzanck smear) confirmed herpes simplex virus infection. Intravenous acyclovir combined with topical imiquimod and resumption of antiretroviral therapy led to rapid clinical improvement and normalization of viral load and CD4+ count.*

Conclusion: *Pseudotumoral chronic genital herpes is a rare but important differential diagnosis for genital lesions in immunocompromised patients. Early recognition and appropriate antiviral therapy, along with immune restoration, are essential for lesion resolution and prevention of recurrence.*

Keywords: Chronic Genital Herpes, HSV, HIV, Pseudotumoral Lesion, Case Report

1. Introduction

Genital herpes is a common sexually transmitted infection, primarily caused by herpes simplex virus type 2 (HSV-2), although HSV-1 is increasingly implicated in genital cases [1]. It classically presents as grouped vesicles on an erythematous base, progressing to painful erosions, with acute onset and spontaneous resolution in immunocompetent individuals [2].

In immunocompromised patients, particularly those living with HIV, clinical manifestations may be atypical, more severe, and prolonged [3]. Chronic ulcerative-vegetative or hypertrophic forms have been described, sometimes mimicking genital squamous cell carcinoma [4,5]. These unusual presentations pose diagnostic challenges and may delay appropriate management.

We report a rare case of pseudotumoral chronic genital herpes mimicking penile squamous cell carcinoma, highlighting the importance of considering herpes in the differential diagnosis of chronic suspicious genital lesions in immunocompromised patients.

2. Case Report

A 48-year-old man with HIV infection, off antiretroviral therapy for one year due to incarceration, presented with a seven-month history of a progressively enlarging, painful ulcerative-vegetative lesion on the penis (Figure 1). At admission, his plasma viral load was elevated at 120,000 copies/mL, and his CD4+ count was low at 150/mm³.

Figure 1 : Ulcerative-Vegetative Lesion on the GLANs at Initial Presentation

Two successive biopsies revealed no malignancy. Immunohistochemistry suggested possible *Treponema pallidum* infection, but syphilis serology was negative. Empirical penicillin therapy showed no improvement.

Rapid worsening of the lesion after biopsies, with marked extension and significant inflammation, led to suspicion of genital pyoderma gangrenosum, and oral corticosteroids were initiated. However, this therapy caused rapid lesion aggravation and the appearance of multiple peripheral vesicles (Figure 2).

Figure 2 : Lesion after Corticosteroid Therapy Showing Exacerbation with Vesicles at the Periphery

Tzanck smear revealed multinucleated giant cells consistent with herpes simplex virus infection.

The patient resumed his antiretroviral therapy, alongside targeted antiviral treatment: oral valaciclovir (initially ineffective), followed by intravenous acyclovir combined with topical imiquimod. This combined regimen led to rapid and progressive lesion regression (Figure 3).

Figure 3 : Regression of the Lesion after Intravenous Acyclovir and Topical Imiquimod

At follow-up, the viral load had normalized to <50 copies/mL, and the CD4+ count rose to 420/mm³, emphasizing the key role of immune restoration in clinical improvement and recurrence prevention.

3. Discussion

Chronic genital ulcerations in HIV-positive patients represent a diagnostic challenge due to the broad spectrum of infectious, inflammatory, and neoplastic etiologies. HSV can cause atypical chronic ulcerative-vegetative or hypertrophic lesions ("pseudotumoral") due to persistent viral replication facilitated by impaired cellular immunity, particularly low CD4+ counts [3].

Lesions can mimic squamous cell carcinoma, often prompting biopsies to exclude malignancy [4,5]. Aggravation under corticosteroids supports active HSV infection, as steroids promote viral replication [6]. PCR for HSV is the ideal diagnostic tool, but Tzanck smear can rapidly suggest the infection [2].

In HIV-positive patients, chronic HSV may require higher doses or intravenous antivirals if oral therapy fails. Topical imiquimod may serve as an adjuvant in hypertrophic or refractory cases [7].

This case underscores the importance of systematically considering HSV in chronic genital lesions in immunocompromised patients and highlights the value of a rigorous diagnostic approach coupled with appropriate antiviral therapy.

4. Conclusion

Pseudotumoral genital herpes, though rare, may mimic squamous cell carcinoma, particularly in immunocompromised patients. Awareness of this atypical presentation is critical in the differential diagnosis of chronic genital lesions, and effective antiviral therapy can lead to significant clinical improvement. Immune monitoring in HIV-positive patients remains essential to prevent recurrence and complications.

References:

1. Looker, K. J., Magaret, A. S., May, M. T., Turner, K. M., & Vickerman, P., et al. (2015). Global and regional estimates of prevalent and incident herpes simplex virus type 1 infections in 2012. *PloS one, 10*(10), e0140765.
2. Johnston, C., & Corey, L. (2016). Current concepts for genital herpes simplex virus infection: diagnostics and pathogenesis of genital tract shedding. *Clinical microbiology reviews, 29*(1), 149-161.
3. Patel, R., Kennedy, O. J., Clarke, E., Geretti, A., & Nilsen, A., et al. (2017). 2017 European guidelines for the management of genital herpes. *International journal of STD & AIDS, 28*(14), 1366-1379.
4. Fatahzadeh, M., & Schwartz, R. A. (2007). Human herpes simplex virus infections: epidemiology, pathogenesis, symptomatology, diagnosis, and management. *Journal of the American Academy of Dermatology, 57*(5), 737-763.
5. Mosunjac, M. B. (2009). *Arch Pathol Lab Med*.133(9), 1516–1520.
6. Hung, C. C. (2000). *J Microbiol Immunol Infect*. 33(4), 237–242.

7. Erlich, K. S., Mills, J., Chatis, P., Mertz, G. J., & Busch, D. F., et al. (1989). Acyclovir-resistant herpes simplex virus infections in patients with the acquired immunodeficiency syndrome. *New England Journal of Medicine, 320*(5), 293-296.