

Genital lesions: An indication for changing ART regimen

S. Arun Kumar, N. Kumar, N. Kumarasamy¹

Institute of Venereology, Rajiv Gandhi Govt. General Hospital, ¹YRG CARE, VHS, Taramani, Chennai, India.

Address for correspondence:

Dr. S. Arunkumar, Institute of Venereology, Madras Medical College, Chennai – 600 003, India. E-mail: arun_ssshc@yahoo.com

Abstract

Genital lesions are common in HIV positive patients and aetiology for these are mainly due to HSV, HPV or bacterial. They usually respond to HAART, antiviral or antimicrobials. We are presenting a young patient on HAART with non-healing genital ulcer lesions for sixteen months. He responded well to a change in ART regimen within a period of 15 days. This happened after a change to a more potent ART regimen.

Key words: ART Change, chronic genital ulcer, HIV, AIDS

INTRODUCTION

Genital lesions are common in HIV positive patients.^[1] Etiology of the genital lesions are mainly due to herpes simplex virus (HSV), human papilloma virus (HPV) and non-specific genital ulcers.^[2] They usually respond to the antiviral and antibacterial agents. We are presenting a young unmarried HIV positive patient who presented with non-healing genital ulcer lesions while on antiretroviral therapy and subsequently responded well to a change in ART regimen within a period of 15 days.

CASE REPORT

Thirty-five year old unmarried male, known positive for HIV infection and on highly active antiretroviral therapy (HAART) since 2001, presented with genital ulcers for a prolonged duration of 16 months. He gave past history of recurrent genital ulcers which

were responded to acyclovir. He had undergone circumcision because of recurrent herpes ulcers in 2005. He was initiated on NNRTI containing ART (AZT + 3TC+NVP) in 2001. His CD4 cell count improved in the years 2002–2006 and subsequently in late 2006 he had a decline in CD4 to 140. He underwent genotyping and found to have NRTI and NNRTI mutations. He was switched to boosted indinavir-containing second line HAART regimen in late 2006. His CD4 count improved to 290, 354, 405 and 398 cells/microliter in the follow-up visits. In spite of his improvement in CD4 he developed genital ulcers in 2007 [Figure 1], which were not responding to the usual line of management with acyclovir and antibiotics. The imprint cytology and scrapings from the lesions showed degenerated and mature squamous cells in an inflammatory background and the patient did not give consent for biopsy. Since the patient was known to have recurrent herpes genitalis, and was responding to acyclovir in the past, we diagnosed it as HSV and his VDRL was non-reactive on four repeated attempts. Dark-field examination for *Treponema pallidum* was negative on repeated attempts. He was on acyclovir and antibacterial for a prolonged period with some improvement on the genital lesions but genital lesions did not heal fully. In July 2008, to avoid indinavir toxicity he was substituted with boosted atazanavir containing PI regimen. His genital

Access this article online**Quick Response Code:****Website:**

www.ijstd.org

DOI:

10.4103/0253-7184.81253

How to cite this article:

Kumar SA, Kumar N, Kumarasamy N. Genital lesions: An indication for changing ART regimen. Indian J Sex Transm Dis 2011;32:37-9.



Figure 1: Genital ulcer before change in ART



Figure 2: Genital ulcer after change in ART

lesions healed completely [Figure 2] in 15 days with no recurrence for more than two months and his current CD4 count is 437 cells/microliter.

DISCUSSION

Herpes simplex virus type 2 (HSV-2) is the dominant primary causative agent in genital ulcerative infections^[2] in HIV positive patients. Prevalence of genital ulcer disease (GUD) is high in high-risk HIV-positive patients and increases with declining immunity.^[1] In our patient he gave past history of herpes and he was improving with acyclovir previously.

Genital herpes was the main etiology of chronic genital ulcer (CGU) in patients with HIV infection.^[1] Conversely, chancroid was the main etiology in patients without HIV infection.^[1] This finding suggests that herpetic CGU is highly suggestive of AIDS whereas chancroid CGU is not. In our patient his CD4 was improving with the indinavir-containing regimen in spite of his non-healing genital ulcers.

Genital and perianal herpetic ulcers are common in HIV-infected patients, and chronic mucocutaneous ulcers persisting for more than one month are the hallmark of active AIDS status. Atypical clinical manifestations of herpes simplex virus (HSV) may occur in immunocompromised patients presenting as tumor-like nodules or condylomatous or hypertrophic lesions, rather than a classic ulcer.^[3] Patients with AIDS may experience excessive number and size of both primary and reactivated herpetic lesions. Aggressive treatment of HSV in combination with HAART therapy provides a significant benefit.^[3]

Our patient was having the CGU for more than six

months and was treated aggressively with HAART and for HSV with acyclovir and antibiotics. Hence chronic non-healing genital ulcers is also a different type of atypical HSV infection in HIV/AIDS patients.

Male circumcision significantly reduced the incidence of human immunodeficiency virus (HIV) infection among men. In addition to decreasing the incidence of HIV infection, male circumcision significantly reduced the incidence of HSV-2 infection and the prevalence of HPV infection.^[4] Our patient had undergone circumcision in 2005 for the recurrent herpes lesions.

Our patient was diagnosed to have AIDS with HSV in 2001 and was on HAART since 2001. History of recurrent genital ulcers was present. He was treated aggressively with HAART and acyclovir on and off for the genital lesions. Initially when he was on AZT+3TC+NVP, CD4 improved till the end of 2006. CD4 declined to 140 cells/mm³ in Dec., 2006. So, he underwent genotyping and found to have NRTI and NNRTI mutations. He was shifted to boosted indinavir-containing protease inhibitor regimen in late 2006. His CD4 count improved to 290, 354, 405 and 398 cells/microliter in the follow-up visits. In spite of his improvement in CD4 he developed genital ulcers in 2007 [Figure 1], which were not responding to the usual line of management with acyclovir and antibiotics. He was on acyclovir and antibacterial for a prolonged period with some improvement on the genital lesions but genital lesions did not heal fully.

Hence the CGU in our patient is an atypical presentation of HSV not responding to the usual line of treatment due to resistance or due to constant chafing and abrasions by clothing due to lack of protective skin.^[5] In spite of his CD4 improvement

after initial ART change, he developed CGU in 2007. Due to CD4 improvement and resource limitations we did not do viral load before and after a change in ART. The patient developed the genital lesions, which were not healing in spite of increase in CD4, hence initially we thought the lesions might be due to acyclovir resistance and not in favor of incomplete suppression of HIV. We do not have facility to assess the resistance for acyclovir and viral load assessment.

Serendipitously, in July 2008, to avoid indinavir toxicity he was substituted with boosted atazanavir-containing PI regimen. His genital lesions healed completely in 15 days [Figure 2] with no recurrence for more than two months and his current CD4 count is 437 cells/microliter. Change to a more potent HAART regimen might have enabled more complete HIV suppression and immune reconstitution thus promoting healing or it raises the question if atazanavir has any role in healing of the genital ulcers. This requires further research in the future.

CONCLUSION

Atypical genital HSV lesions occur in AIDS

patients. Change in HAART with boosted atazanavir-containing PI regimen was associated with healing of chronic HSV genital lesions in an HIV/AIDS patient with improvement of CD4 count and immune reconstitution. Whether atazanavir has any direct role in healing of genital ulcers needs further investigation.

REFERENCES

1. Ghys PD, Diallo MO, Traore-Ettiégne V, Laga M, Diallo K, De Cock KM. GUD prevalence is high in HIV-positive prostitutes and increases with declining immunity. *Int Conf AIDS* 1993;9:660 (abstract no.PO-04-2659).
2. Gbery IP, Djeha D, Kacou DE, Aka BR, Yoboue P, Vagamon B, *et al.* Chronic genital ulcerations and HIV infection: 29 cases. *Med Trop (Mars)* 1999;59:279-82.
3. Mosunjac M, Park J, Wang W, Tadros T, Siddiqui M, Bagirov M, *et al.* Genital and perianal herpes simplex simulating neoplasia in patients with AIDS. *AIDS Patient Care STDS* 2009;23:153-8.
4. Tobian AA, Serwadda D, Quinn TC, Kigozi G, Gravitt PE, Laeyendecker O, *et al.* Male circumcision for the prevention of HSV-2 and HPV infections and syphilis. *N Engl J Med* 2009;360:1298-309.
5. Sorrells ML, Snyder JL, Reiss MD, Eden C, Milos ME, Wilcox N, Van Howe RS. Fine touch pressure thresholds in the adult penis. *BJU Int* 2007;99:864-9.

Source of Support: Nil. **Conflict of Interest:** None declared.