

Variation in Clinical Presentation of Herpes Zoster in HIV Positive and Negative Patients

Aishni Shah, Juhi Shah, Khushboo Modasia and Rita Vora*

*Department of Dermatology, Pramukhswami Medical College, Shree Krishna Hospital, Gujarat, India.

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ABSTRACT

In immunocompetent individuals herpes zoster usually presents with typically grouped vesicles on erythematous base involving single dermatome with self-limiting nature, while it may present in extensive form involving multiple dermatomes or disseminated form in immunocompromised, especially in Human Immunodeficiency Virus (HIV). HIV-positive patients with lower CD4 counts have higher risk of HZ. Disseminated and multiple dermatomal involvement, with more severity is more commonly involved among HIV-positive patients when compared to HIV-negative patients and they also have higher incidence of post herpetic neuralgia.

Keywords: Herpes zoster, Human immunodeficiency virus, Immunosuppression

INTRODUCTION

Herpes Zoster (HZ) is characterized by unilateral radicular pain and grouped vesicular eruption involving dermatome innervated by a single spinal or cranial sensory ganglion, as a result of reactivation of persistent latent form of Varicella Zoster Virus (VZV) within the sensory ganglion [1]. Although more than 90% of adults have serologic evidence of VZV infection and are therefore at risk of developing HZ, many times HIV-positive patients have been found to have higher incidence than the general population [2]. HZ usually occurs as an early manifestation in the course of HIV infection and when compared to HIV-negative individuals it shows higher incidence of post herpetic neuralgia, recurrences, disseminated zoster, ulcerated lesions and chronic verrucous lesions [3]. There have been various studies showing the difference in HZ presentation between HIV positive and negative individuals.

DISCUSSION

Herpes zoster is a viral infection caused by varicella-zoster virus, which occurs more frequently in the elderly and those with any form of immunodeficiency. Hence, the incidence of HZ is observed to be higher in the immune-deficient condition 'Acquired Immuno-Deficiency Syndrome' (AIDS) with more severity, multiple dermatomal involvement, dissemination and systemic involvement [4]. The prevalence of HIV in general population of Gujarat is around 0.38% [5]. It ranges from 5.6% to 22.5% in various similar studies [6]. HZ occurs primarily in adults older than 50 years although it can occur at any age [7]. Its incidence rate ranges from 1.2

to 3.4 per 1,000 person-years among younger healthy individuals, increasing to 3.9-11.8 per 1,000 person-years among those older than 65 years [8]. Association of VZV reactivation with immunosuppression and age suggests if the immune system is effective, it prevents viral replication [9]. Therefore immunosuppression, especially organ transplantation, hematologic malignancy and HIV infection increases the rates of HZ occurrence. One study has showed that 30% of the AIDS cases have history of HZ [10]. Unlike typical HZ which is characterized by unilateral, segmental painful vesicles over certain affected dermatomal region, HZ in the immunosuppressed can be ulcerative and necrotic and scar more severely. These patients have relatively increased severity of cutaneous lesions and course of disease is prolonged, persistent or recurrent with attacks, showing unusual morphologies like verrucous and hyperkeratotic lesions [11]. HZ has high risk of dissemination up to 40%, in immunocompromised persons and in HIV infections [12], which is defined as more than 20 vesicles outside the

Corresponding author: Dr. Rita Vora, Senior Professor, Skin OPD no. 111, Department of Dermatology, Pramukhswami Medical College, Shree Krishna Hospital, Karamsad-388325, Gujarat, India, Tel: 9879290417; E-mail: ritavv@charutarhealth.org

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primary and immediately adjacent dermatomes and is followed by visceral involvement like lungs, liver, brain in 15% of high-risk patients. In a recent study, multiple dermatomal involvement and disseminated zoster in HIV-positive patients is statistically significant when compared to that of HIV-negative patients, yet among HIV-positive patients, 82.85% HZ patients had localized dermatomal involvement [13]. Thus, HIV-positive patients can present with typical, disseminated or atypical lesions of HZ. Cervical, ophthalmic dermatomes are more commonly involved in HIV-positive and thoracic dermatome was more commonly involved in HIV-negative patients. CD4 cell count below 500 cells/mm³ and higher HIV RNA level are associated with high HZ incidence [14]. A study showed that HIV-infected individuals at the highest stratum corneum CD4 cell count (750 cells/mL) are at a nearly 9-fold higher risk of developing HZ, while HIV-infected women with the lowest CD4 cell counts (200 cells/mL) are nearly 25-fold more likely to develop HZ than HIV-uninfected women [14]. HZ is more often found to precede AIDS in high risk individuals like old age or immunodeficiency, but if it occurs in asymptomatic HIV cases, it is considered a bad prognostic marker for progression to symptomatic HIV. It often precedes other symptoms such as thrush or oral leucoplakia by an average of 1.5 years, which in turn precedes to other AIDS defining opportunistic infections by 2-3 years.[15] Post Herpetic Neuralgia (PHN) can be caused by persistence of VZV following HZ or by increased neuronal excitability which causes alteration of pain perception [16]. Thus, lower cell-mediated immunity may lead to higher levels of virus during acute infection and thus an increased risk of PHN, proving that immunosuppression such as in HIV, is strongly associated with greater PHN risk. Although death due to HZ is likely to be exceedingly rare in HIV-infected patients, it is an important cause of morbidity. The development of HZ in otherwise asymptomatic individuals at high risk for HIV represents an early clinical sign that should alert the physician to consider the possibility of the impending development of an immune deficiency. In India, where diagnostic facilities are often limited, HZ may be used as a sentinel event for estimating the number of HIV-infected patients in a given population who will be requiring further screening tests for HIV.

CONCLUSION

Although multi-dermatomal and disseminated involvement is more commonly seen in HIV-infected patients, they can also present with typical presentation of herpes zoster. Hence, the physician should screen every case of herpes zoster for HIV infection to detect the disease earlier.

REFERENCES

1. Straus SE, Schmader KE, Oxman MN (2003) Varicella and herpes zoster. In: Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, Katz SI, editors. Fitzpatrick's Dermatology in General Medicine. 6th Edn. New York: McGraw Hill 2: 2070-2085.
2. Gebo KA, Kalyani R, Moore RD, Polydefkis MJ (2005) The incidence of, risk factors for and sequel of herpes zoster among HIV patients in the highly active antiretroviral therapy era. *J Acquir Immune Defic Syndr* 40: 169-174.
3. Jacobson MA, Berger TG, Fikrig S, Becherer F, Moehr JW, et al. (1990) Acyclovir resistant varicella zoster virus infection after chronic oral acyclovir therapy in patients with the acquired immunodeficiency syndrome (AIDS). *Ann Intern Med* 112: 187-191.
4. Tappero JW, Perkins BA, Wenger JD, Berger TG (1995) Cutaneous manifestations of opportunistic infections in patients infected with human immunodeficiency virus. *Clin Microbiol Rev* 8: 440-450.
5. National AIDS Control Organization, Ministry of Health and Family Welfare, Government of India (2008) HIV estimates during surveillance in 2006.
6. Sharvadze L, Tsertsvadze T, Gochitashvili N, Stvilia K, Dolmazashvili E (2006) HIV prevalence among high risk behavior group persons with herpes zoster infection. *Georgian Med News* 132: 60-64.
7. McCrary ML, Severson J, Tyring SK (1999) Varicella zoster virus. *J Am Acad Dermatol* 41: 1-14.
8. Dworkin RH, Johnson RW, Breuer J, Gnann JW, Levin MJ, et al. (2007) Recommendations for the management of herpes zoster. *Clin Infect Dis* 44: S1-26.
9. James WD, Berger TG, Elston DM, Odam RB (2006) *Andrews' diseases of the skin: Clinical dermatology*. 10th Edn. Philadelphia: WB Saunders, pp: 379-384.
10. Hira SK, Wadhawan D, Kamanga J, Kavindele D, Macuacua R, et al. (1988) Cutaneous manifestations of human immunodeficiency virus in Lusaka, Zambia. *J Am Acad Dermatol* 19: 451-457.
11. Grossman MC, Grossman ME (1993) Chronic hyperkeratotic herpes zoster and human immunodeficiency virus infection (review). *J Am Acad Dermatol* 28: 306-308.
12. Webre DM, Pelechia JA (1965) Varicella pneumonia: Study of prevalence in adult men. *JAMA* 192: 572-573.
13. Vora RV, Anjaneyan G, Kota RKS, Pilani AP, Diwan NG, et al. (2017) Study of clinical profile of herpes zoster in human immunodeficiency virus positive and negative patients at a rural-based tertiary care center, Gujarat. *Indian J Sex Transm Dis AIDS* 38: 65-68.
14. Glesby MJ, Hoover DR, Tan T, Shi Q, Gao W, et al. (2004) Herpes zoster in women with and at risk for

HIV: Data from the women's interagency HIV study. *J Acquir Immune Defic Syndr* 37: 1604-1609.

15. Buchbinder SP, Katz MH, Hessel NA, Liu J, O'Malley PM, et al. (1992) Herpes zoster in human immunodeficiency virus infection. *J Infect Dis* 166: 1153-1156.
16. Harpaz R, Nagel MA, Schmader K, Tyring SK, Yawn BP, et al. (2012) Roundtable on post-herpetic neuralgia: What, why, how long and what's next? *Popul Health Manag* 15: 385-390.