ISSN 2664-8075 (Print) & ISSN 2706-5774 (Online)
South Asian Research Journal of Applied Medical Sciences
Abbreviated Key Title: South Asian Res J App Med Sci

| Volume-7 | Issue-3 | May-Jun- 2025 | **DOI: https://doi.org/10.36346/sarjams.2025.v07i03.006**

Case Report

Reactivation of Herpes Simplex Virus in a Polytraumatized Patient

Grecia Chávez-Chavira[1*], Laura Patricia Camacho-Cedeño[1]

[1]Internal Medicine Department, Hospital Regional de Alta Especialidad "Bicentenario de la Independencia", Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado, State of Mexico

***Corresponding Author:** Grecia Chávez-Chavira
Internal Medicine Department, Hospital Regional de Alta Especialidad "Bicentenario de la Independencia", Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado, State of Mexico

Article History
Received: 16.05.2025
Accepted: 21.06.2025
Published: 24.06.2025

Abstract: Herpes simplex virus type 1 (HSV-1) is a common neurotropic virus that remains latent in sensory neurons after the initial infection. Reactivation can occur throughout life, especially in the presence of triggering factors such as trauma, immunosuppression, or stress. We present a case of a 72-year-old male developed herpetic stomatitis with perioral vesicular lesions following multiple traumatic injuries, including severe head trauma and subarachnoid hemorrhage.

Keywords: Dermatology, Herpes Simplex Virus, Reactivation, Polytraumatism.

INTRODUCTION

Herpes simplex virus type 1 (HSV-1), and type 2 (HSV-2) are two of the most prevalent human viruses; worldwide prevalence is estimated at up to 67% and 13% respectively. Transmission of both occurs through close contact and results in a lifelong infection [1, 2], which is characterized by periodic reactivation at the infection site. HSV-1 virus is primarily transmitted by oral-to-oral contact, while HSV-2 is almost entirely sexually transmitted, causing genital herpes [2, 3]. Latent infection occurs primarily in sensory neurons, characterized by silencing of most viral genes via epigenetic repression, HSV reaches peripheral neurons via retrograde axonal transport. Reactivation occurs in response to neuronal stress and is mediated by the JNK-DLK signaling pathway, facilitating gene expression [2]. The immune system of an individual tends to decay upon aging, trauma, stress and opportunities arise for HSV to reactivate in the organism and spread to tissues [3]. Brain injury induces systemic immunosuppression, which increases the risk of HSV reactivation; this reactivation was linked to a specific monocyte transcriptomic signature that it is characterized by impaired interferon-γ mediated and antiviral responses, suggesting that trauma-induced immune dysregulation facilitates viral reactivation and may negatively impact neurological outcomes [4]. This case report emphasizes the reactivation of HSV-1 following a multiple traumatic injury (including traumatic brain injury).

CASE PRESENTATION

72 year-old male patient who was admitted to Internal Medicine service after a multiple contusion due to a traffic accident presenting severe head trauma, subarachnoid hemorrhage, unstable thorax and patellar fracture. During his hospitalization he developed with septic shock due a urinary tract infection. Three days after multiple trauma he began with a dermatosis located in the perioral region characterized by multiple vesicles with a diameter of 1-3 mm (Figure 1), with a tendency to confluence, without internal involvement of the oral mucosa, which progressed to a necrotic warty appearance (Figure 2), a diagnosis of herpetic stomatitis was made. Management was started with supportive care measures and oral acyclovir with subsequent resolution and improvement of the condition (Figure 3).

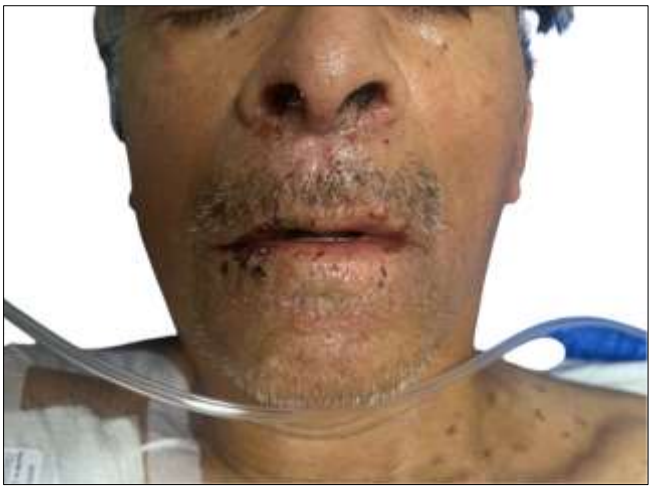

Figure 1

Figure 2

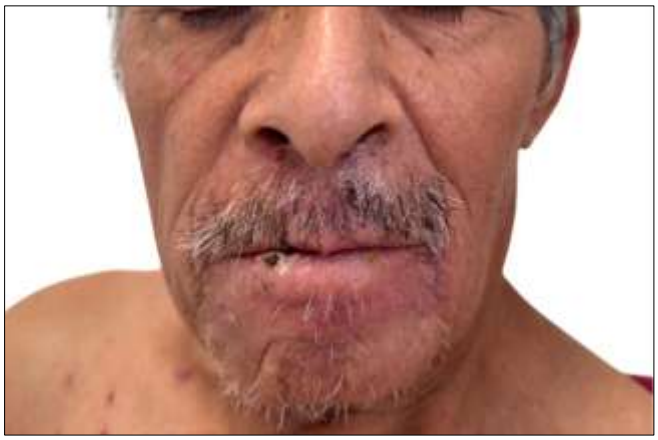

Figure 3

DISCUSSION

HSV-1 is a widely distributed neurotropic human pathogen that is transmitted mainly by intimate contact between infected and susceptible individuals, and it may cause labia or ocular infections [5], after primary infection of epithelial cells, the virus becomes latent in neurons of the peripheral nervous system and can be periodically reactivated resulting in

recurrent clinical or subclinical episodes throughout life [5]. A range of stimuli, including trauma, emotional stress, hormone imbalance, UV exposure, fever, and immunosuppression, can reactivate the latent virus within infected ganglia [5, 6], on this case the reactivation was due to severe head trauma and subarachnoid hemorrhage, local injury to nerves or ganglia can precipitate viral reactivation [6]. Multiple mechanism has been described, including direct nerve injury, that can facilitate viral reactivation from latency due to direct disruption of the neuronal integrity or ganglionic microenvironments, and the associated stress response, including activation of sympathetic system and activation of the glucocorticoid receptor which can promote viral gene expression and further reactivation [2-6]. Head trauma triggers cascades of secondary injuries that promote neuroinflammation, excitotoxicity, and disruption of the blood-brain barrier. Traumatic brain injury provides a unique environment for these triggers, initiating an acute inflammatory response characterized by the release of cytokines such as IL-1β, TNF-α, and IL-6. By interfering with immune surveillance systems and neuronal homeostasis, these cytokines foster an environment favorable for HSV reactivation. Diagnosis is mainly clinical, based on the patient's history and physical examination [7, 8].

The treatment of herpetic stomatitis is primarily supportive including adequate hydration, pain control and topical anesthetic, with the addition of oral antiviral therapy in selected cases; moderate to severe cases, oral acyclovir dose ranges from 15 to 20 mg/kg/dose, up to 400 mg/dose five times daily for 7 days; other options includes valacyclovir 1 g twice daily for 7 days. In immunocompromised patients administration of acyclovir is recommended to be intravenous (5 to 10 mg/kg/dose IV every 8 hours). The administration of topical antiviral is not routinely recommended due to limited efficacy [9,10].

CONCLUSIONS

Trauma can precipitate reactivation of herpes virus, likely due to a compromised immune system or direct nerve damage. These findings underscore the importance of considering HSV reactivation in patients with head trauma.

Conflict of Interest: The authors declare that there are no conflicts of interest at the time of publication of this article.

REFERENCES

1. Zhu, Shuyong, and Abel Viejo-Borbolla. “Pathogenesis and virulence of herpes simplex virus.” Virulence vol. 12,1 (2021): 2670-2702. doi:10.1080/21505594.2021.1982373
2. James, C., Harfouche, M., Welton, N. J., Turner, K. M., Abu-Raddad, L. J., Gottlieb, S. L., & Looker, K. J. (2020). Herpes simplex virus: global infection prevalence and incidence estimates, 2016. Bulletin of the World Health Organization, 98(5), 315–329. https://doi.org/10.2471/BLT.19.237149
3. Duarte, L. F., Farías, M. A., Álvarez, D. M., Bueno, S. M., Riedel, C. A., & González, P. A. (2019). Herpes simplex virus type 1 infection of the central nervous system: Insights into proposed interrelationships with neurodegenerative disorders. Frontiers in Cellular Neuroscience, 13, 46. https://doi.org/10.3389/fncel.2019.00046
4. Chaumette, Tanguy et al. “Monocyte Signature Associated with Herpes Simplex Virus Reactivation and Neurological Recovery after Brain Injury.” American journal of respiratory and critical care medicine vol. 206,3 (2022): 295-310. doi:10.1164/rccm.202110-2324OC
5. Marcocci, M. E., Napoletani, G., Protto, V., Kolesova, O., Piacentini, R., Li Puma, D. D., Lomonte, P., Grassi, C., Palamara, A. T., & De Chiara, G. (2020). Herpes simplex virus-1 in the brain: The dark side of a sneaky infection. Trends in Microbiology, 28(10), 808–820. https://doi.org/10.1016/j.tim.2020.03.003
6. Zhang, John X et al. “Association of physical trauma with risk of herpes zoster among Medicare beneficiaries in the United States.” The Journal of infectious diseases vol. 207,6 (2013): 1007-11. doi:10.1093/infdis/jis937
7. Johansyah TKP, et al, " Dormant No More: The Neurological Impact of Herpes Simplex Virus Reactivation Following Traumatic Brain Injury. J. neuroanestesi Indones 2025;14(1): 47–52 https://doi.org/10.24244/jni.v14i1.659
8. Vannachone S, Chanthamavong A, Vongsouvath M, Sayasene P, Vongsouvath M. Case Report: Herpes simplex virus type 2 (HSV-2) meningo-encephalitis associated with traumatic brain injury – a case report from Lao PDR. Welcome Open Res. 2024;9:489. Doi: 10.12688/wellcomeopenres.22720.1
9. Coppola, Noemi et al. “Supportive care and antiviral treatments in primary herpetic gingivostomatitis: a systematic review.” Clinical oral investigations vol. 27,11 (2023): 6333-6344. doi:10.1007/s00784-023-05250-5
10. Arch Intern Med. 2008;168(11):1137–1144. doi:10.1001/archinte.168.11.1137