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(CASE REPORT)

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HERPES SIMPLEX VIRUS REACTIVATION FOLLOWING FACIAL BURNS: A CASE REPORT

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ABSTRACT

Herpes simplex virus (HSV) reactivation is a recognized but potentially overlooked complication in burn patients, particularly following facial burns. Its clinical manifestations may mimic bacterial infection or an atypical evolution of the burn wound, potentially delaying appropriate antiviral therapy.

We report the case of a 35-year-old woman with no significant medical history who sustained mixed superficial and deep partial-thickness thermal burns involving approximately 30% of the total body surface area, including the face and neck. The facial burns were managed conservatively with daily cleansing and topical wound care. Approximately two weeks after injury, during the re-epithelialization phase, the patient developed fever associated with multiple vesicular and vesiculopustular lesions, predominantly periorificial in distribution, involving the healing facial and cervical burn wounds. The lesions subsequently evolved into erosions and crusts. Based on their characteristic morphology, distribution, and timing of onset, post-burn HSV reactivation was clinically suspected. Virological confirmation was not performed. Intravenous acyclovir therapy was initiated, resulting in progressive regression of the lesions and favorable wound healing. At three months of follow-up, the eruption had completely resolved, with no clinical recurrence.

HSV reactivation should be considered when new vesicular or erosive lesions associated with fever develop over a healing facial burn, particularly during the first weeks following injury. Early clinical recognition may allow prompt initiation of antiviral therapy and minimize disruption of the wound-healing process.

Keywords: Herpes Simplex Virus (HSV), HSV Reactivation, Facial Burn, Thermal Burn, Burn Infection, Acyclovir, Wound Healing

1 INTRODUCTION

HSV infection represents an important viral complication in the clinical course of burn patients [1,2]. Thermal injury may induce alterations in host immune defenses, creating favorable conditions for viral infection and reactivation [1,2]. Facial burns appear particularly susceptible, and HSV reactivation typically occurs during the healing phase, often within the first weeks following injury [3–5].

The clinical presentation may initially be difficult to distinguish from other infectious or noninfectious changes affecting burn wounds [1,3]. The development of fever associated with newly appearing vesicular, vesiculopustular, or erosive lesions over healing burn wounds should therefore raise suspicion of HSV reactivation [3,6]. Early recognition is clinically relevant, as active HSV infection may impair wound healing [2].

We report the case of a 35-year-old woman with extensive thermal burns involving the face, in whom a clinical diagnosis of HSV reactivation was made approximately two weeks after injury. Intravenous acyclovir therapy was initiated, with a favorable clinical response. This case highlights the importance of considering HSV reactivation in the differential diagnosis when new vesicular or erosive lesions develop over a healing facial burn.

2 CASE PRESENTATION

A 35-year-old woman with no significant medical history was admitted approximately 20 hours after sustaining an accidental thermal burn caused by a domestic gas leak in a confined space. Clinical examination revealed mixed superficial and deep partial-thickness burns involving approximately 30% of the total body surface area, affecting the face and neck as well as both upper and lower limbs. The Baux score was 65 and the Unit Burn Standard (UBS) score was 30.

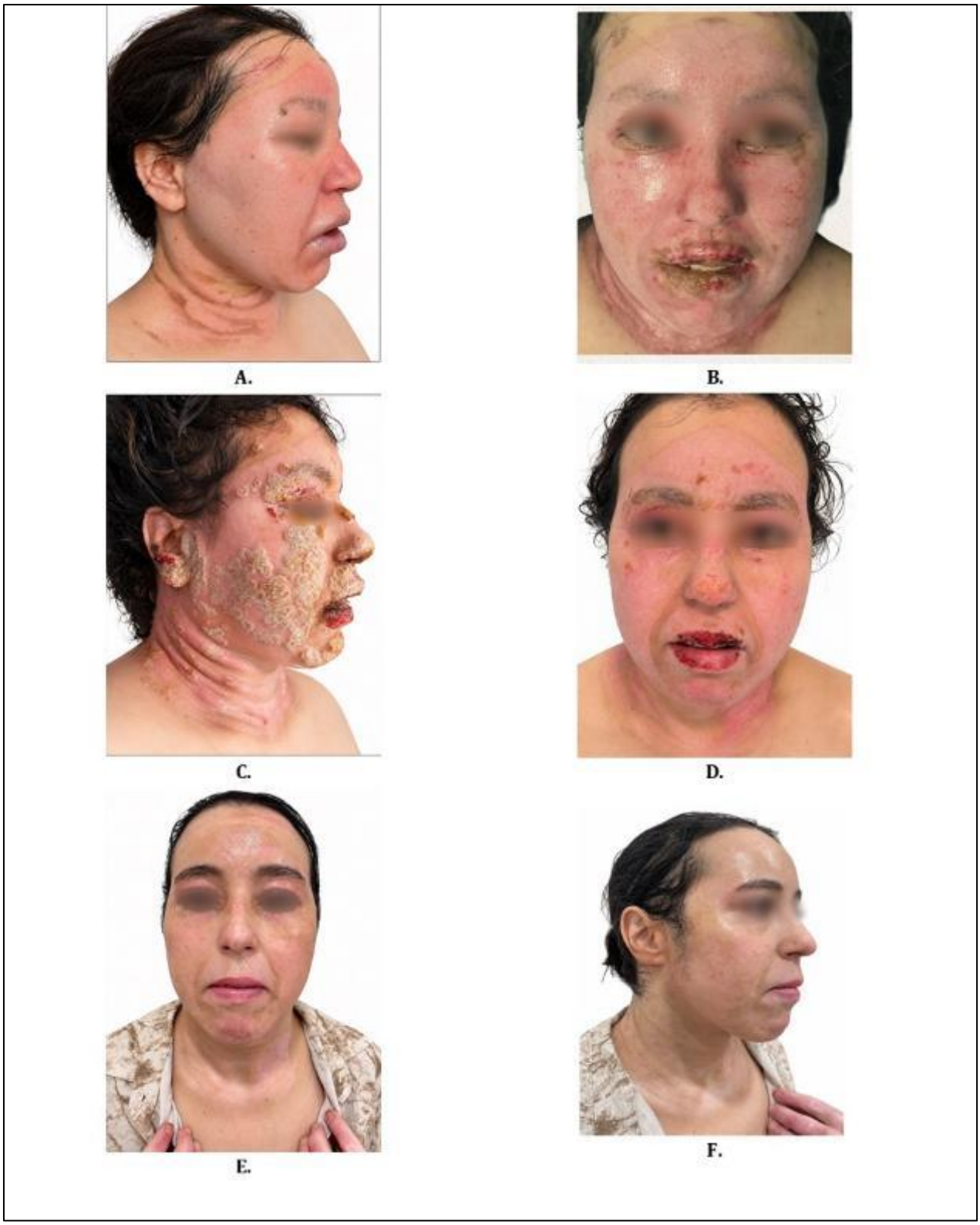
The facial burns were managed conservatively with daily local wound care. No surgical excision or skin grafting was performed on the face. The wounds initially showed a favorable course toward re-epithelialization.

Approximately two weeks after the burn injury, during the healing phase, the patient developed fever associated with the appearance of a new cutaneous eruption involving the healing facial and cervical burn wounds. Clinical examination revealed multiple vesicular and vesiculopustular lesions, predominantly periorificial in distribution, with several lesions subsequently evolving into erosions and crusts.

Given the characteristic morphology and distribution of the lesions and their onset during the healing phase, a clinical diagnosis of post-burn HSV reactivation was made. Virological confirmation by polymerase chain reaction or viral culture was not performed.

Intravenous acyclovir was initiated at a dose of 5 mg/kg every 8 hours for 10 days. A marked clinical improvement was observed within three days, with progressive regression and crusting of the lesions. Further improvement was observed over the following days, with favorable progression of wound healing.

At approximately three months of follow-up, the eruption had completely resolved without clinical recurrence. The facial burns had healed, with residual erythematous and pigmentary changes.



A. Initial facial burn at admission. B. Facial burn healing at day 4 after injury, before the onset of herpetic lesions. C. Herpetic eruption approximately two weeks after burn injury, with vesicular and vesiculopustular lesions predominantly periorificial in distribution. D. Post-herpetic evolution seven days after initiation of intravenous acyclovir, showing marked regression and crusting of the lesions. E–F. Three-month follow-up, frontal and profile views, showing complete resolution of the herpetic eruption and healing of the facial burn.

Figure 1: Clinical course of post-burn HSV reactivation.

3 DISCUSSION

Burn injury creates a biological environment that may favor HSV reactivation through the combined effects of immune dysregulation, disruption of the cutaneous barrier, and local tissue damage [1,2]. Clinically relevant HSV infection in

burn patients is thought to result predominantly from reactivation of latent infection rather than primary acquisition [1,3]. Facial burns are particularly relevant, as local tissue injury may provide an additional trigger for HSV reactivation [3]. Although the true incidence remains difficult to establish because of differences in patient populations and diagnostic methods, HSV reactivation rates of up to 25% have been reported following burn injury [3].

The timing and clinical presentation observed in our patient are consistent with previous reports of HSV reactivation following burn injury. HSV typically becomes clinically apparent during the first to third post-burn weeks and may present in febrile patients as clusters of vesicular or vesiculopustular lesions arising within or around healing partial-thickness wounds [3]. In a retrospective series of 95 severely burned adults, Fidler et al. reported facial herpetic eruptions in 14 patients, corresponding to approximately 15% of the cohort, with the condition typically recognized during the second post-burn week [4]. More recently, Brennan et al. reported clinical signs of HSV infection in 29 of 56 high-risk burn patients; 97% of affected patients had facial burns, and the median time to clinical infection was 8 days [5]. Sheridan et al. similarly reported severe herpetic ulceration involving the face and neck in five of six burn patients [6]. These findings closely parallel our case, in which fever and predominantly periorificial vesiculopustular lesions developed over healing facial and cervical burns approximately two weeks after injury.

The diagnosis of HSV reactivation in burn patients may be challenging because its clinical manifestations can overlap with other infectious or noninfectious changes occurring in burn wounds [1]. The appearance of newly developing vesicular or vesiculopustular lesions progressing to erosions or ulcerations, particularly within or around healing burn wounds, should raise suspicion of HSV infection [3,6]. Virological confirmation should be sought whenever feasible using appropriate laboratory testing [1,3]. In our patient, the diagnosis was based on the characteristic morphology and predominantly periorificial distribution of the lesions, their occurrence approximately two weeks after facial burn injury, the associated fever, and the favorable response to acyclovir. However, the absence of virological confirmation represents the main limitation of this case.

Once clinically manifest HSV infection is suspected in burn patients, prompt antiviral therapy is generally recommended, with acyclovir being the principal antiviral agent reported in this setting [3,6,7]. Although evidence regarding the optimal antiviral regimen specifically in burn patients remains limited, previous reports have described favorable clinical outcomes following acyclovir therapy [3,4,6]. In our patient, intravenous acyclovir at 5 mg/kg every 8 hours for 10 days was associated with marked clinical improvement within three days, followed by progressive regression and crusting of the lesions. Beyond controlling the viral eruption, early recognition and treatment may also be relevant to wound healing, as active HSV infection has been associated with impaired wound healing in burn patients [2].

4 CONCLUSION

HSV reactivation should be considered in burn patients who develop fever and new vesicular or erosive lesions over healing facial burns, particularly during the first weeks after injury. Early clinical recognition and prompt antiviral therapy may promote a favorable clinical course and limit disruption of wound healing. When available, virological testing should be performed to confirm the diagnosis.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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Disclosure of conflict of interest

The authors declare no conflict of interest.

Statement of informed consent

Written informed consent was obtained from the patient for publication of this case report and its associated clinical images.

REFERENCES

[1] Baj J, Korona-Głowniak I, Buszewicz G, Forma A, Sitarz M, Teresiński G. Viral Infections in Burn Patients: A State-Of-The-Art Review. Viruses. 2020;12(11):1315. doi:10.3390/v12111315.

[2] Wurzer P, Guillory A, Parvizi D, et al. Human herpes viruses in burn patients: a systematic review. Burns. 2017;43(1):25–33. doi:10.1016/j.burns.2016.02.003.

[3] Haik J, Weissman O, Stavrou D, et al. Is prophylactic acyclovir treatment warranted for prevention of herpes simplex virus infections in facial burns? A review of the literature. J Burn Care Res. 2011;32(3):358–362. doi:10.1097/BCR.0b013e318217f6de.

[4] Fidler PE, Mackool BT, Schoenfeld DA, et al. Incidence, outcome, and long-term consequences of herpes simplex virus type 1 reactivation presenting as a facial rash in intubated adult burn patients treated with acyclovir. J Trauma. 2002;53(1):86–89. doi:10.1097/00005373-200207000-00017.

[5] Brennan PG, Wright K, Miles MVP, Lintner AC, Alexander KM, Kahn SA. Delineating the Role of Serum Immunoglobulin Titers in Burn Patients at High Risk for Herpes Simplex Virus Infection. J Burn Care Res. 2021;42(4):646–650. doi:10.1093/jbcr/irz197.

[6] Sheridan RL, Schulz JT, Weber JM, Ryan CM, Pasternack MS, Tompkins RG. Cutaneous herpetic infections complicating burns. Burns. 2000;26(7):621–624. doi:10.1016/S0305-4179(00)00025-5.

[7] Țânțu AC, Țânțu MM, Vrancianu CO, et al. Infections in Burn Patients: Pathophysiology, Prevention, and Contemporary Therapeutic Strategies in the Era of Antimicrobial Resistance. Infect Dis Ther. 2026;15:2027–2070. doi:10.1007/s40121-026-01376-7.