Thermal Trigger: An Unusual Case of Herpes Zoster Reactivation in the Trigeminal Dermatome Following Hot Water Exposure

Termal Tetikleyici: Sıcak Suya Maruz Kalmanın Ardından Trigeminal Dermatomda Olağandışı Bir Herpes Zoster Reaktivasyonu Vakası

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Atıf/Cite As: Semet C. Thermal Trigger: An Unusual Case of Herpes Zoster Reactivation in the Trigeminal Dermatome Following Hot Water Exposure. Hitit Med J 2023;5(2): 122-125. https://doi.org/10.52827/hititmedj.1299831

Abstract

Herpes zoster, or shingles, is a viral disease caused by the reactivation of the varicella-zoster virus. While reactivation is typically associated with aging, immunosuppression, or stress, this case report presents an unusual trigger: exposure to hot water. A 76-year-old woman developed herpes zoster localized to the trigeminal nerve dermatome following accidental exposure to hot water. The patient was successfully treated with acyclovir, with symptoms resolving within 14 days. This case highlights the importance of considering unusual triggers for varicella-zoster virus reactivation and underscores the need for prompt diagnosis and treatment to prevent complications.
Keywords: Heat shock proteins; Herpes zoster; Hot water exposure; Varicella-zoster virus

Özet

Herpes zoster veya zona, varisella-zoster virüsünün reaktivasyonu sonucu ortaya çıkan viral bir hastalıktır. Reaktivasyon tipik olarak yaşlanma, immünosupresyon veya stres ile ilişkilendirilirken, bu vaka raporunda alışılmadık bir tetikleyici sunulmaktadır: sıcak suya maruz kalma. 76 yaşında bir kadın hastada, kazara sıcak suya maruz kalmasının ardından trigeminal sinir dermatomunda lokalize herpes zoster gelişmiştir. Asiklovir ile başarılı bir şekilde tedavi edilmiş ve semptomları 14 gün içinde düzelmiştir. Bu vaka, varisella-zoster virüs reaktivasyonu için olağandışı tetikleyicileri dikkate almanın önemini vurgulamakta ve komplikasyonları önlemek için hızlı tanı ve tedavi ihtiyacının altını çizmektedir.
Anahtar Kelimeler: Isı şok proteinleri; Herpes zoster; Sıcak su maruziyeti; Varisella-zoster virüsü

Geliş Tarihi/Date of Submission: 20.05.2023
Kabul Tarihi/Date of Acceptance: 08.06.2023
Yayım Tarihi/Date of Publication: 30.06.2023

Peer Review: It was evaluated by independent reviewers working in three different institutions appointed by the relevant field editor.
Ethical Statement: Not required.
Plagiarism Checks: Yes - iThenticate
Conflict of Interest: No conflict of interest was declared by the authors.
Complaints: hmj@hitit.edu.tr
Informed Consent: Permission was obtained from the patient for the case report.
Financial Disclosure: No financial support.

Introduction

Herpes zoster (HZ), or shingles, is a viral disease characterized by a painful skin rash with blisters in a localized area. Typically, the rash occurs on either the left or right side of the body or face in a single stripe pattern. It is caused by the varicella-zoster virus (VZV), the same virus that causes chickenpox. After an individual recovers from chickenpox, the virus can remain dormant in the nervous system and reactivate years later, causing herpes zoster (1). The reactivation of VZV is usually associated with aging, immunosuppression, or stress (2).

It has been reported that approximately 10-20% of people infected with VZV will go on to develop shingles (2,3), and involvement of the trigeminal nerve as a result of herpes zoster virus infection is common (4). The ophthalmic portion of the trigeminal nerve and then the eye is usually affected by VZV reactivation. This condition is herpes zoster ophthalmicus (HZO) (5). Accurate and timely diagnosis of HZ is crucial to minimize morbidity, as HZO can lead to vision loss.

This report delves into an intriguing case of a 76-year-old woman who experienced an unexpected reactivation of HZ, localized to the trigeminal nerve dermatome, following exposure to hot water. This unique instance suggests that there could be a broad range of potential triggers for VZV reactivation, some of which may be entirely unexpected. The significance of this case lies in its potential to reshape our understanding of the triggers of HZ reactivation and its management.

As we delve deeper into this unusual case, we hope to highlight the importance of early diagnosis and prompt systemic antiviral treatment, particularly for HZO, to avert severe consequences, including potential vision loss. We also aim to underscore the need to consider HZ in the differential diagnosis of burn lesions, especially among the elderly.

Case

A 76-year-old woman visited our outpatient clinic, complaining of erythema, increased temperature, and a headache on her forehead and scalp, which developed two days after she accidentally poured hot water on her head. She had no systemic symptoms, including high fever, nausea, vomiting, and systemic diseases like diabetes, or hypertension. Physical exam was regular with full consciousness, orientation, and coordination. Her body temperature was 36.8°C, her pulse rate was 86 beats per minute, and her arterial blood pressure was 110/65 mmHg. There were vesicular lesions on an erythematous background extending from the right side of the forehead to the scalp, and there was edema and hyperemia around the eyes (Figure 1). Yellow scabs were present in some lesions. Visual movements were unrestricted, with no significant anomalies and facial asymmetry of extremities.

The cerebellar examination was regular, and there was no stiffness in the nuchal region. Laboratory tests revealed leukocytes 6310 /mm^3, hemoglobin 12.4 g/dL, platelets 272.000 /mm^3, creatinine 0.87 mg/dL, AST 27 IU/L, and ALT 24 IU/L, sedimentation 34 mm/h, C-reactive protein (CRP) 0.6 mg/dL. We ordered radiological imaging to exclude any intracranial and orbital involvement. The cranial magnetic resonance imaging (MRI) showed no intracranial space-occupying lesion with significant pathological contrast enhancement. The report indicated that the MRI of the orbit was expected, as usual. Ophthalmological examination showed normal visual acuity. Fundoscopic examination showed no pathological findings. As a result of the investigations, the patient, who had no ocular involvement, was admitted to the hospital with the diagnosis of HZ with localization to the trigeminal nerve dermatome. Due to the yellow scab lesions, we initiated treatment with Acyclovir 3 x 500 mg/day IV and ampicillin/sulbactam 4 x 1.5 g IV. On the 3rd day of treatment, the headache had resolved, and the vesicular eruptions had begun to dry up (Figure 2). On follow-up, the redness and edema around the eyes decreased. On the 7th day of treatment, all vesicular lesions were dry with a resolution of edema and hyperemia around the eyes. After completing the 14-day course of acyclovir and ampicillin/sulbactam, we discharged the patient from the hospital.

Figure I. Vesicular lesions on an erythematous background, edema, and hyperemia around the forehead

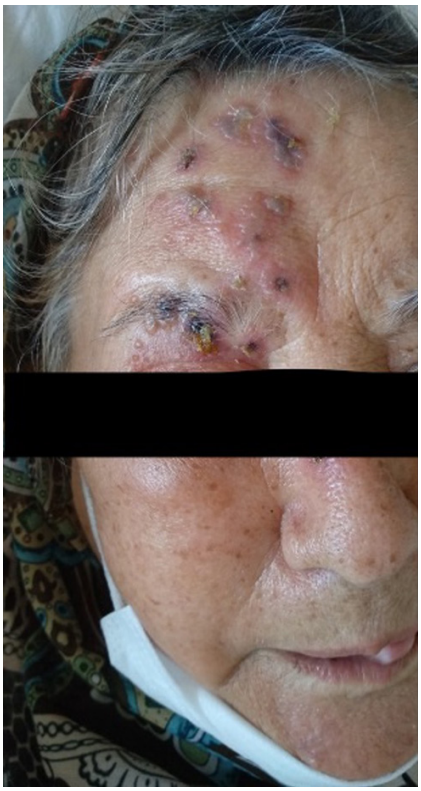
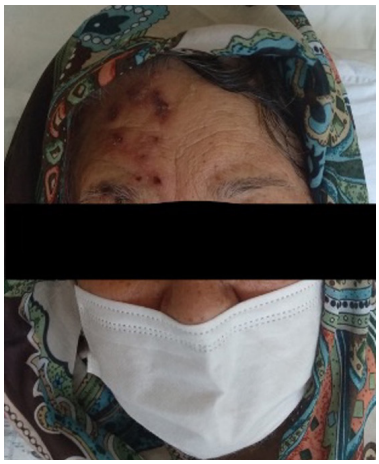


Figure II. Lesions dried and regressed after treatment.



Discussion

The case presented here is of a 76-year-old woman who developed HZ localized to the trigeminal nerve dermatome following accidental exposure to hot water. This is an unusual case as the reactivation of HZ is typically associated with factors such as aging, immunosuppression, or stress, rather than physical triggers like heat exposure.

HZ, or shingles, is a condition caused by the reactivation of the VZV, the same virus that causes chickenpox. After an individual recovers from chickenpox, the virus can remain dormant in the nervous system and reactivate years later, causing HZ. The reactivation of VZV is usually associated with aging, immunosuppression, or stress (6). However, the trigger for reactivation, in this case, appears to be exposure to hot water, which is not a commonly recognized trigger.

The pathophysiology behind this unusual reactivation could be related to the effect of heat on the nervous system. Heat stress has been shown to induce the expression of heat shock proteins (HSPs), which play a role in the reactivation of latent viruses (6). The lifetime risk of developing HZ is approximately 30% (7). A healthy immune system typically inhibits the reactivation of VZV; however, risk factors for HZ involve advanced age, psychological stress, immunosuppression due to cancer treatment, direct trauma, surgery, and sunburn (1, 4, 5, 6). HZ frequently presents with painful and unilaterally distributed vesicular lesions, affecting the thoracic and cervical regions in approximately 60% and 20% of cases, respectively. Branches of the trigeminal nerves are involved in about 20% of patients (6). It is plausible that the hot water exposure, in this case, may have induced HSPs, leading to the reactivation of the dormant VZV in the patient's trigeminal nerve.

Furthermore, the fifth cranial nerve, the trigeminal nerve, provides sensory innervation to the face and motor functions to the muscles of mastication. It is divided into three branches: the ophthalmic, maxillary, and mandibular nerves. In this case, the patient's symptoms and the rash's distribution suggest that the trigeminal nerve's ophthalmic branch was affected. This is consistent with the known preference of VZV for cranial nerves and the associated sensory ganglia (7). HZO is a rare form of HZ infection and involves the ophthalmic branch of the trigeminal nerve along the V1-V2 dermatomes. Vesicular eruptions of HZO usually develop in the periorbital region and forehead. In our case, the ophthalmic branch of the trigeminal nerve was partially involved. In trigeminal HZ, the involvement of the ophthalmic part is five times higher than the maxillary part. The presence of lesions on the nose of our patient was especially suspicious for HZO. This condition is known as Hutchinson's sign and indicates involvement of the nasociliary branch of the ophthalmic nerve (8). For this reason, we requested an ophthalmological consultation, and the evaluation showed no ocular involvement.

Trigeminal HZ infection may lead to encephalitis, postherpetic neuralgia, and permanent visual loss if not recognized and treated (8). Postherpetic neuralgia (PHN) is a complication of HZ characterized by persistent pain that negatively affects the quality of life and daily activities. Advanced age and immunosuppression are risk factors for PNH (9). PHN did not develop in our patient.

For HZ infections with cranial localization, we administer parenteral acyclovir (10 mg/kg every 8 hours) as treatment. (10). Starting treatment in the first 72 hours of the clinical picture decreases the duration of postherpetic neuralgia and improves clinical results (10). The course of antiviral therapy is 7-10 days (10). We should prolong the duration of treatment in elderly and immunocompromised patients because the varicella virus can remain in the cornea for up to one month (8).

In the existing literature, there are virtually no reported cases of HZ developing following exposure to hot water. Therefore, our case significantly contributes to the literature. This case not only expands our understanding of potential triggers for HZ reactivation but also underscores the importance of considering such unusual triggers in clinical practice. Further research is needed to elucidate the mechanisms behind such triggers and to determine whether they represent isolated incidents or a broader pattern.

This case also highlights the importance of a thorough clinical examination and the use of appropriate diagnostic tools. The patient's symptoms, the rash's distribution, and the absence of any significant findings on cranial magnetic resonance imaging (MRI) and ophthalmological examination led to the diagnosis of HZ. This underscores the value of clinical understanding in diagnosing conditions like HZ, where the presentation can vary, and the triggers can be unusual.

In conclusion, we presented a rare case of HZ reactivation in the trigeminal nerve dermatome following hot water exposure. The patient was successfully treated with acyclovir and ampicillin/sulbactam and showed significant improvement within seven days of treatment. Further research is needed to understand better the relationship between thermal stress and HZ reactivation and to explore potential preventative measures. Clinicians should consider HZ a possible diagnosis in patients with vesicular eruptions and a history of hot water exposure.

References

1. Werner, R., Nikkels, A., Marinović, B., et al. (2016, November 2). European consensus-based (S2k) Guideline on the Management of Herpes Zoster - guided by the European Dermatology Forum (EDF) in cooperation with the European Academy of Dermatology and Venereology (EADV), Part 2: Treatment. Journal of the European Academy of Dermatology and Venereology, 2106;31: 20-29.

2. Topçu WA, Söyletir G, Doğanay M. Enfeksiyon Hastalıkları ve Mikrobiyolojisi. Fourth Edition. Ankara: Nobel Tıp Kitabevleri, 2017:1474-1478.

3. Zaal MJW, Völker-Dieben HJ, D'Amaro J. Prognostic value of Hutchinson's sign in acute herpes zoster ophthalmicus. Graefe's Archive for Clinical and Experimental Ophthalmology 2003;241:187–191.

4. Lıesegang T. Herpes Zoster Ophthalmicus Natural History, Risk Factors, Clinical Presentation, and Morbidity. Ophthalmology 2008;115:S3–12.

5. Albietz JM, Lenton LM. Late Reactivation of Herpes Zoster Keratitis Results in Band Keratopathy. Optometry and Vision Science 2014;91:e149–155.

6. Anderson E, Fantus RJ, Haddadin RI. Diagnosis and

management of herpes zoster ophthalmicus. Disease-a-Month 2017;63:38–44.

7. Gilden D, Cohrs RJ, Mahalingam R, et al. Varicella zoster virus vasculopathies: diverse clinical manifestations, laboratory features, pathogenesis, and treatment. The Lancet Neurology 2009;8:731-740.

8. Yawn BP, Gilden D. The global epidemiology of herpes zoster. Neurology 2013;81:928-930.

9. Hong M-J, Kim Y-D, Cheong Y-K, et al. Epidemiology of Postherpetic Neuralgia in Korea. Medicine 2016;95:e3304.

10. Opstelten W, Zaal MJW. Managing ophthalmic herpes zoster in primary care. BMJ 2005;331:147-151.