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Complicated Herpes Zoster: Literature Review and Clinical Case Report

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Abstract

Herpes zoster (HZ) results from reactivation of latent varicella-zoster virus (VZV) in sensory ganglia. Facial involvement and neurological complications—including postherpetic neuralgia, peripheral facial paralysis, and severe ocular involvement—account for most of the associated morbidity and mortality. The adjuvanted recombinant vaccine has transformed prevention strategies in adults aged ≥ 50 years and immunocompromised individuals aged ≥ 19 years. However, gaps persist in the early diagnosis of atypical forms, risk stratification for postherpetic neuralgia (PHN), and equitable access to immunization in middle-income regions, including Latin America. We present the case of a female patient in her fourth decade of life, with a history of HIV infection, who presented with painful blistering and desquamative lesions of five days' evolution, with severe involvement. The patient showed an adequate clinical response to the instituted medical treatment with acyclovir. This review synthesizes recent evidence on this infectious disease, which remains a frequent health concern in geriatric and vulnerable populations.

Keywords

Ophthalmic Herpes Zoster, Postherpetic Neuralgia, Vaccine, Acyclovir, Ramsay Hunt

1. Introduction

Varicella-zoster virus (VZV), a neurotropic alpha-herpesvirus, establishes latency after primary infection (varicella) in dorsal root and cranial nerve ganglia [1]. Throughout life, processes such as immunosenescence and/or immunosuppression facilitate its reactivation, leading to herpes zoster (HZ). It is estimated that up to one-third of the population will develop HZ at some point, with a rising incidence after age 50 and an exponential increase in those over 70 [1] [2]. The clinical impact centers on acute neuropathic pain and chronic complications such as postherpetic neuralgia (PHN), ophthalmic involvement, peripheral facial paralysis associated with Ramsay Hunt syndrome, and vascular events—including a transient increased risk of stroke and prolonged functional impairment [3].

Facial involvement poses a risk of epithelial, stromal, and neurotrophic keratitis, anterior and posterior uveitis, scleritis, acute retinal necrosis, and secondary glaucoma, with potential for irreversible vision loss [2] [3]. In parallel, Ramsay Hunt syndrome—characterized by herpes zoster oticus with seventh cranial nerve palsy—is associated with more severe sensorineural hearing loss and poorer facial recovery compared to idiopathic Bell's palsy [4]. PHN, typically defined as neuropathic pain persisting ≥ 90 days after rash onset (although some definitions use ≥ 30 days), represents the main chronic complication, with a negative impact on quality of life due to its association with sleep disturbances, affective symptoms, and healthcare costs [1] [3].

The socioeconomic burden includes recurrent medical visits, loss of productivity, and hospitalizations, particularly in high-risk populations such as immunocompromised individuals, those with complicated ophthalmic presentations, or patients with intractable pain [1] [2]. The introduction of the adjuvanted recombinant vaccine has achieved > 90% efficacy against HZ and PHN in immunocompetent adults, with robust real-world effectiveness. However, vaccine coverage remains suboptimal in many countries, and integration into immunization schedules for immunosuppressed patients is progressing unevenly [5].

Critical gaps remain, including the underdiagnosis of prodromal presentations without rash or atypical forms such as zoster sine herpete. Additionally, delayed initiation of antiviral therapy is common, despite the optimal window being within the first 72 hours. Inadequate pain management is also an issue, with underutilization of early multimodal analgesia that could reduce the risk of PHN [1] [2] [6]. Moreover, regions such as Latin America lack robust community-based incidence data, hindering the development of locally adapted cost-effectiveness models [7].

We present a clinically relevant case of an immunosuppressed patient with HIV who developed severe ophthalmic involvement, showing optimal response to early initiation of antiviral therapy.

2. Clinical Case

A 34-year-old female patient with a five-year history of human immunodeficiency virus (HIV) infection, under antiretroviral therapy with dolutegravir (DTG) plus tenofovir alafenamide (TAF)/emtricitabine (FTC), with an undetectable viral load and a CD4+ count of 510/mm^3, presented to the emergency department with a five-day history of left hemifacial pain that progressed to painful vesicular lesions involving the left frontal and ophthalmic regions. The patient also exhibited marked palpebral edema, erythema, ocular pain, and associated serous discharge. She had self-medicated with cephalexin for two days without improvement, prompting medical consultation.

On admission, the patient appeared in significant distress. Vital signs showed a blood pressure of 123/78 mmHg, heart rate of 78 bpm, and respiratory rate of 18 breaths per minute. Physical examination revealed crusted and blistering lesions over the left hemiface, including the frontal and periorbital regions, with marked eyelid edema and complete limitation of ocular opening (Figure 1). Laboratory workup showed a white blood cell count of 7000/μL with 70% neutrophils, hemoglobin of 10.8 g/dL, elevated C-reactive protein (18 mg/L), serum creatinine of 0.7 mg/dL, and normal electrolyte levels.

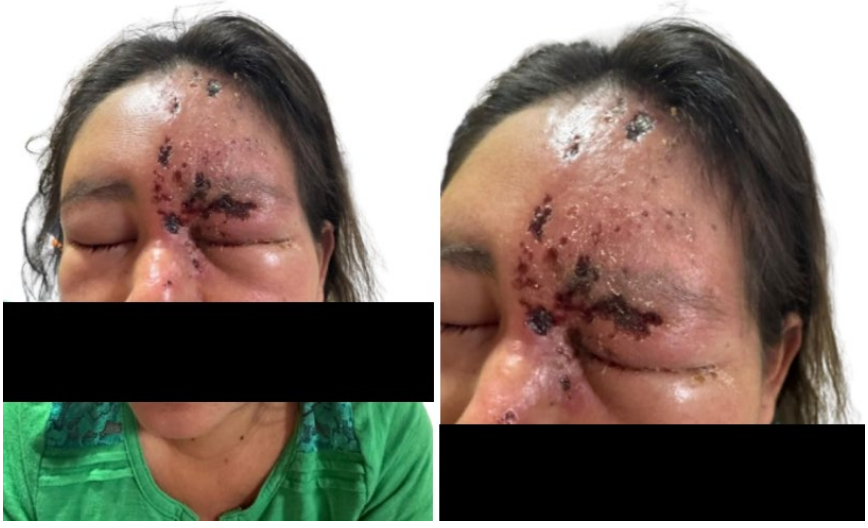

Figure 1. Admission images showing erythematous and edematous lesions with crusted vesicles and marked palpebral edema.

The case was classified as severe herpes zoster with ophthalmic involvement; the diagnosis of herpes zoster was established clinically, based on the presence of a painful, unilateral vesicular rash with a V1 dermatomal distribution. Ophthalmologic evaluation corroborated ocular involvement by documenting decreased

visual acuity and corneal edema on slit-lamp examination, findings consistent with HZ. Based on this, intravenous acyclovir was initiated at a dose of 10 mg/kg for 14 days, along with close clinical monitoring, supported by high-level clinical guidelines, such as the Sanford Guide and IDSA. Additionally, a short course of corticosteroids was added—specifically, a single dose of 8 mg of dexamethasone, with the aim of accelerating functional recovery and reducing visual sequelae. Its use is justified in the presence of significant ocular involvement, always in combination with acyclovir. The risks of hyperglycemia, elevated blood pressure, immunosuppression, and, at the ocular level, increased intraocular pressure and delayed corneal re-epithelialization were cautioned and closely monitored. The patient subsequently showed marked clinical improvement, achieving ocular opening, resolution of serous discharge from the affected eye, and significant improvement in the skin lesions (Figure 2).

Regarding pain management, a multimodal analgesic regimen was established to address both somatic and neuropathic pain. The patient was started on pregabalin 50 mg once daily, acetaminophen 1 g every 8 hours, and a rescue analgesic plan with low-potency opioids, including tramadol 50 mg, which was required only once on the first day of hospitalization.

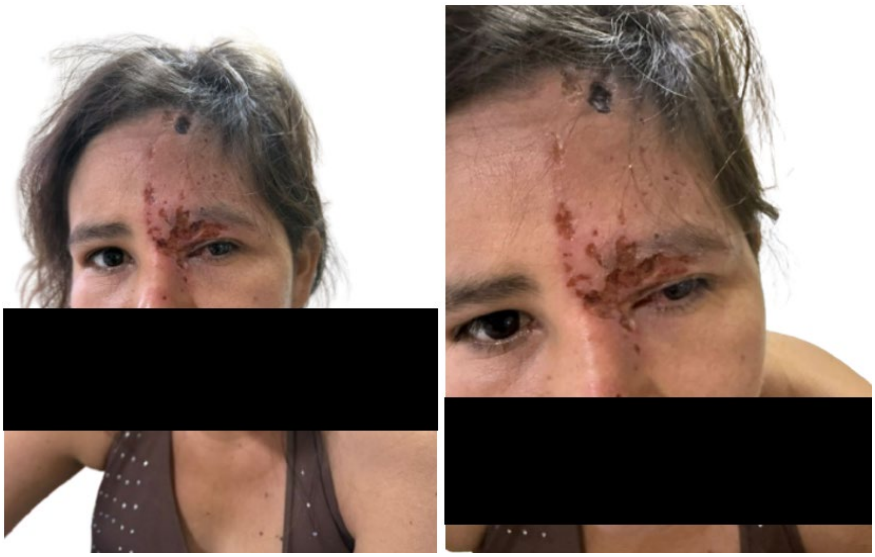

Figure 2. Follow-up image after completion of antiviral therapy showing significant improvement of the lesions.

Following the patient's clinical improvement, hospital discharge was indicated with continuation of oral analgesic management, including pregabalin 50 mg once daily and acetaminophen. Outpatient follow-up with the infectious diseases service was scheduled for ongoing monitoring of her chronic condition. The treatment was thus considered successful.

3. Discussion

This review integrates epidemiological, pathophysiological, and clinical evidence

that reaffirms herpes zoster (HZ) as an escalating public health concern. This trend is driven by the convergence of population-level immunosenescence, increased survival of immunocompromised patients—as exemplified by the present case—and limited vaccine coverage in middle-income regions [1] [7]. Incidence rates of 5 - 10 cases per 1000 person-years in high-income countries—with peaks ≥ 12/1000 among octogenarians—contrast with the remarkably high VZV seroprevalence (>95% in adults aged ≥ 50 years), establishing a near-universal reservoir for viral reactivation [2]. The Latin American landscape, although underdocumented, suggests incidence densities of 6.4 - 36.5 cases per 1000 person-years in high-risk cohorts, underscoring the urgency for active surveillance systems and disease burden studies to inform cost-effective vaccination strategies [2] [7].

The burden of complications, particularly postherpetic neuralgia (PHN) and herpes zoster ophthalmicus (HZO), shifts the impact of HZ from acute cutaneous morbidity to chronic disability and functional decline, with a negative impact on quality of life [3] [4]. PHN affects up to 18% of individuals over 60 and is more prevalent among immunocompromised patients, supporting the hypothesis that VZV-specific cell-mediated immunity is the main determinant of chronic pain progression [2]. This finding aligns with studies linking elevated serum IL-6 levels to increased PHN risk, opening avenues for prognostic stratification and early neuroimmune-targeted interventions [8].

From a pathophysiological standpoint, advances in understanding viral anterograde trafficking and ganglionic inflammation reinforce the recommendation to initiate antiviral therapy ideally within the first 72 hours. Early intervention not only shortens rash duration but also mitigates the inflammatory response responsible for peripheral and central sensitization [1] [9]. However, in immunocompromised patients and complicated cases, the therapeutic window is extended due to higher viral load and replication rates, justifying the use of intravenous acyclovir and early corticosteroid co-administration [10].

From a public health perspective, the adjuvanted recombinant zoster vaccine (RZV) has reshaped preventive paradigms by demonstrating > 90% efficacy against HZ and PHN, including in immunocompromised subgroups [11]. Nonetheless, its limited adoption in Latin America—due to cost, cold chain logistics, professional awareness gaps, and lack of access—continues to restrict its population-level impact [7]. Recent modeling suggests that achieving 40% coverage in adults aged ≥ 60 years could prevent up to 60% of PHN cases over ten years, generating net savings when accounting for chronic pain management and productivity loss. Incorporating RZV into national immunization programs will require microeconomic studies and sensitivity analyses that consider the often-invisible burden of chronic pain and preventable blindness from HZO [12] [13].

On the therapeutic horizon, helicase-primase inhibitors such as amenamevir and pritelivir offer a promising alternative for overcoming thymidine kinase-mediated resistance and simplifying dosing regimens, although clinical evidence in VZV remains preliminary [14]. Concurrently, mRNA vaccine platforms may en-

hance immunogenicity and facilitate large-scale manufacturing, a key factor for global equity. Additionally, the validation of biomarkers such as IL-6 levels, CD4/CD8 ratio, and predictive nomograms may enable individualized prophylaxis and tailored analgesic strategies [8] [11] [14].

4. Conclusion

Available evidence supports a dual approach, centered on universal RZV vaccination for adults aged ≥ 50 and immunocompromised individuals, alongside early antiviral and analgesic therapy to reduce complications and prevent progression to PHN. Strengthening epidemiological registries, ensuring equitable access to vaccination, and accelerating translational research in biomarkers and innovative antivirals are critical steps to reduce the growing burden of HZ and its neurological sequelae in the coming decade.

Declaration

Informed consent from the patient and the institution has been obtained for the publication of this case.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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