

# Eczema Herpeticum in an Adult Patient with Erythrodermic Atopic Dermatitis

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## Abstract

Eczema herpeticum (EH), a life-threatening viral infection complicating atopic dermatitis, is rarely reported in adults with erythroderma. We present a 58-year-old male with untreated, severe atopic dermatitis who developed EH, manifesting as generalised erythroderma, monomorphic vesicles, and systemic symptoms with fever, hypertension. Diagnosis was confirmed by Tzanck smear, HSV-1 PCR, and bacterial culture revealing *Staphylococcus aureus*. Resource limitations necessitated oral acyclovir which achieved resolution of vesicles and fever within eight days. Concurrent management included intravenous vancomycin for bacterial superinfection, topical therapies, and cyclosporine for recalcitrant atopic dermatitis. Hypertension, a relative contraindication to cyclosporine, was controlled with nifedipine, enabling safe immunosuppression. By day 13, skin lesions normalised, pruritus resolved, and blood pressure stabilised. This case underscores oral acyclovir's efficacy in resource-limited settings and highlights multidisciplinary strategies for managing EH with comorbidities like hypertension. Early antiviral intervention, tailored antimicrobial therapy, and vigilant monitoring remain critical to optimising outcomes in severe EH-atopic dermatitis overlap.

**Keywords:** Herpes simplex virus; Hypertension; Cyclosporine; Acyclovir

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## INTRODUCTION

Eczema Herpeticum (EH) is a viral skin infection complicating pre-existing dermatoses with compromised

skin barriers, most commonly atopic dermatitis (AD).<sup>1</sup> The incidence of EH in people with AD is less than

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3%.<sup>2</sup> The condition can progress from localised mild disease to life-threatening systemic involvement if timely antiviral therapy is not initiated.<sup>2</sup> EH typically manifests with rapid-onset, morphologically uniform vesicular lesions accompanied by systemic symptoms such as fever and malaise.<sup>1</sup> While EH is frequently reported in the paediatric populations, who are more susceptible to AD, we present a case of EH in an adult patient with erythrodermic atopic dermatitis, highlighting its unique clinical features and diagnostic challenges.

### CASE REPORT

A 58-year-old male, with a history of atopic dermatitis, which had worsened over the past year but remained untreated. Ten days before admission, the patient developed scaly red skin all over the body accompanied by intense itching, leading to frequent scratching. Three days before admission, the patient developed disseminated, uniform, painful monomorphic blisters predominantly on the head and face, which rapidly spread to the trunk and upper limbs. The lower half of the body was affected by erythroderma but lacked vesicular lesions.

Alongside the blisters, the patient experienced high-grade fever and severe fatigue, prompting a visit to the dermatology department for treatment.

Upon admission, clinical examination revealed generalised erythroderma with scaling and severe pruritus across the entire body. Additionally, uniform blisters on erythematous bases were observed, distributed densely on the face and scalp, later spreading to the trunk and upper limbs. Some blisters had ruptured, leaving erosions with serous crusts, while others showed impetiginisation with pustules and yellow crusts, concentrated on the face (**Figure 1**). The patient presented with high-grade fever, fatigue, poor appetite, but no oral or genital ulcers. The patient also exhibited hypertension, with systolic blood pressure fluctuating between 180-220 mmHg.

Given the clinical features suggestive of EH, diagnostic tests were performed on blister fluid. Results showed multinucleated giant cells on Tzanck smear and HSV-1 positivity via PCR (**Figure 2**). A skin biopsy of erythematous lesions revealed spongiotic dermatitis, which is characteristic of acute atopic dermatitis (**Figure 2C**). Bacterial culture of pustules and yellow



**Figure 1:** A. Generalized erythroderma with fine scaling and dense, monomorphic vesicles on erythematous bases in the trunk. B. Ruptured vesicles with serous crusting and early impetiginisation (yellow pustules) on the face. C. Partial resolution of erythroderma and vesicular lesions after 7 days of oral acyclovir and topical therapies (betamethasone, tacrolimus). Residual erosions with crusts persist. D. Reduced facial impetiginisation following fusidic acid and salicylic acid treatment. E, F. Near-complete resolution of erythema and erosions after cyclosporine therapy (100 mg/day). Skin texture normalizes, with minor post-inflammatory hyperpigmentation.

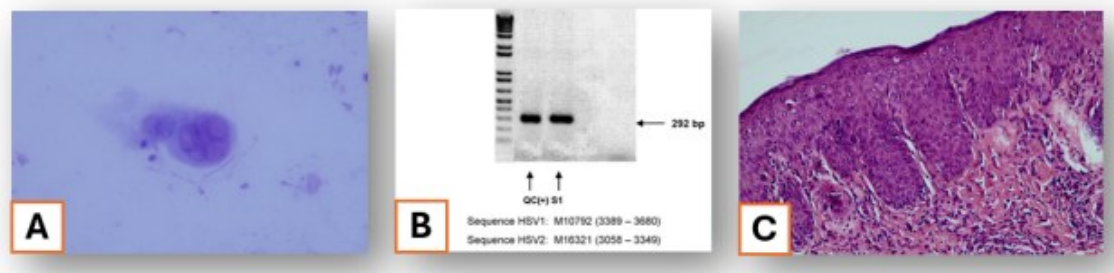


Figure 2: A. Microscopic examination of vesicular fluid reveals multinucleated giant cells, supporting the diagnosis of eczema herpeticum. B. Positive HSV-1 detection via polymerase chain reaction (PCR). A 292-bp amplicon was amplified using primers derived from the HSV-1 reference strain (GenBank accession: M10792, nucleotides 3389–3680). No HSV-2 DNA was detected (reference strain: M16321, nucleotides 3058–3349). C. Histopathological examination of the erythematous skin lesion demonstrates spongiotic dermatitis with intercellular edema (spongiosis), superficial perivascular lymphocytic infiltration, and scattered eosinophils (Hematoxylin and eosin, 20x).

crusts identified *Staphylococcus aureus* sensitive to vancomycin.

The patient was immediately initiated on oral antiviral therapy with acyclovir 800 mg x 4 tablets/day for 14 days, intravenous vancomycin 2 g/day, Ringer’s lactate infusion 500 mL /day, and paracetamol 2 g/day for fever. Hypertension was managed with nifedipine 20 mg x 2 tablets/day.

By day 3, the patient’s fever persisted at 39°C; by day 4, it decreased to 38°C, and resolved completely by day 6. Erythroderma and scaling were treated with topical betamethasone 0.064% and tacrolimus 0.03% applied to the face. Yellow pustules were managed with fusidic acid 2%, and thick yellow crusts with salicylic acid 5% ointment. Urea-based emollients with Urelid 3% for the face and 10% for other areas were applied alternately.

After 8 days of treatment, scaling resolved, and erythroderma significantly improved, with only minor erosions and crusts remaining (Figures 1C & 1D). However, generalised pruritus persisted. Cyclosporine was initiated at a low dose of 100 mg per day for patients with pre-existing hypertension alongside topical corticosteroids and urea emollients. Four days later, pruritus subsided, skin lesions normalised (Figures 1E & 1F), and the patient was discharged for outpatient management. During cyclosporine therapy, systolic blood pressure stabilised within normal limits with 100-120 mmHg.

## DISCUSSION

EH remains a persistent risk for patients with AD, particularly those with high-risk factors such as severe or poorly controlled AD, untreated AD, cutaneous

*Staphylococcus aureus* superinfection, and genetic predispositions.<sup>2</sup> This risk stems from structural and functional alterations in the skin barrier inherent to AD pathogenesis, which facilitate HSV invasion, particularly HSV-1, a highly prevalent virus in the community. These alterations include impaired skin barrier integrity, reduced antimicrobial peptides, and dysfunctional immune responses. Additionally, host genetic factors such as filaggrin mutations, defects in innate immune signaling, and dysregulation of immune components, such as suppressed antiviral interferon responses and Th2-skewed immunity which further weaken defenses against HSV. Regulatory T-cell dysfunction may also contribute to impaired viral control in AD.<sup>4</sup>

The clinical diagnosis of EH relies on key features such as disseminated, painful, monomorphic vesicles on an erythematous base, pustulation, impetiginisation, erosions with serous crusting, and occasional ulceration due to secondary bacterial infection. Lesions typically occur in areas predisposed to AD, such as the face, trunk, and extremities. Systemic symptoms, including high-grade fever, chills, rigors, and reduced oral intake with or without oral ulcers, further support the diagnosis. Although EH is clinically recognisable, atypical presentations may necessitate differentiation from impetigo, eczema coxsackium, contact dermatitis, or severe AD flares with erythroderma. In such cases, ancillary tests such as Tzanck smear, histopathology, and HSV PCR, the most sensitive diagnostic tool, are critical.<sup>5</sup>

Prior to antiviral therapy, EH mortality rates reached 50%.<sup>6</sup> Immediate antiviral treatment is mandatory upon diagnosis. For severe or extensive EH, intravenous acyclovir is preferred due to higher bioavailability.<sup>7</sup> However, oral acyclovir remains a viable alternative in resource-limited settings where IV formulations are

unavailable. Supportive measures include gentle skin cleansing, moisturisation, and targeted antibiotic therapy, such as penicillinase-resistant penicillins or cephalosporins for *Staphylococcus aureus* superinfection. Concurrent AD management with topical corticosteroids or calcineurin inhibitors is safe when combined with antivirals. Systemic AD therapies such as oral corticosteroids, cyclosporine, methotrexate, should be deferred until EH resolution.

Our patient was diagnosed with EH based on clinical features, Tzanck smear findings and HSV-1 PCR positivity. Immediate oral acyclovir was initiated due to limited access to intravenous formulations. Remarkably, the patient improved despite severe EH, underscoring oral therapy's utility in resource-constrained settings. Severe impetiginisation and systemic symptoms with fever, chills, and anorexia necessitated hospitalisation, as outpatient management would have been unsafe. Adjunct therapies include topical fusidic acid for bacterial superinfection, salicylic acid ointment for crust removal, and urea-based emollients for xerosis.

Following EH resolution, erythrodermic AD was managed with cyclosporine, selected for its rapid efficacy and favorable safety profile. Hypertension, a relative contraindication to cyclosporine, was controlled with nifedipine, enabling uninterrupted cyclosporine use. Blood pressure remained stable throughout treatment, demonstrating that hypertension is not an absolute barrier to cyclosporine when rigorously managed.

## CONCLUSION

EH complicating erythrodermic AD is a life-threatening infection requiring urgent antiviral therapy. Multidisciplinary care, integrating infection control, comorbidity management, and AD therapy, is essential. This case highlights oral acyclovir's efficacy in resource-limited settings and reinforces the feasibility of cyclosporine in hypertensive patients with careful monitoring. Early diagnosis, tailored antimicrobial therapy, and concurrent dermatological management remain pivotal to improving outcomes.

## Take Home Message

- Oral acyclovir can be an effective first-line treatment for severe eczema herpeticum in resource-limited settings where intravenous therapy is unavailable.
- Pre-existing hypertension is not an absolute contraindication for cyclosporine use in severe atopic dermatitis and it can be safely managed with concurrent antihypertensive therapy and vigilant monitoring.
- A multidisciplinary approach, integrating antiviral, antimicrobial, anti-inflammatory, and comorbidity management, is critical for successful outcomes in complex cases with life-threatening infections and significant comorbidities.

## Abbreviations

EH	Eczema herpeticum
AD	Atopic dermatitis

## Declarations

### Patient Consent

Patient consent has been obtained.

### Disclosure and Conflict of Interest

The authors declare that they have no conflicts of interest and no financial disclosures relevant to this case report.

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