Scientific Research Publishing

Open Journal of Pediatrics, 2024, 14, 981-987
https://www.scirp.org/journal/ojped
ISSN Online: 2160-8776
ISSN Print: 2160-8741

Eczema Herpeticum in Pediatrics: A Case Report

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How to cite this paper: Kassal, G., El Moussaoui, S., Lahmini, W. and Bourrous, M. (2024) Eczema Herpeticum in Pediatrics: A Case Report. *Open Journal of Pediatrics*, **14**, 981-987.
https://doi.org/10.4236/ojped.2024.146094

Received: September 20, 2024
Accepted: November 2, 2024
Published: November 5, 2024

Abstract

Background: One of the most common viral infections occurring in atopic dermatitis (AD) patients is eczema herpeticum (EH): herpes simplex virus (HSV) skin infection. Despite being comparatively uncommon affecting just 3% of AD patients, EH can progress from a local disease to a potentially fatal systemic infection. Up till now, the pathophysiology of EH is mainly unclear. **Case Report**: We report the case of a 2-year-old boy who was well-vaccinated and had a history of untreated atopic dermatitis from infancy and presented to the emergency department with increasingly itchy diffuse lesions, red eyes, profuse tearing, chills and malaise. The diagnosis of EH with bilateral ocular involvement was highly suspected. The patient had received intravenous acyclovir with a notable clinical improvement and a complete remission at the two-week follow-up.

Keywords

Atopic Dermatitis, Eczema Herpeticum, Herpes Simplex Virus, Acyclovir

1. Introduction

Kaposi's varicelliform eruption (KVE), also known as eczema herpeticum, is a rare and potentially severe viral infection, essentially caused by reactivation of herpes simplex virus (HSV), especially type I. [1] Other viruses such as Coxsackie A 16, vaccinia [2], and varicella zoster [3] have also been implicated in its pathogenesis. Preexisting skin conditions [4], mostly atopic dermatitis (AD) [2], and immunodeficiency [5] are considered risk factors.

The pathology manifests as widespread clusters of umbilicated vesico-pustules

and eroded vesicles, interspersed with punched-out ulcers overlaid by hematic crusts, primarily distributed across the cephalic, cervical, and truncal regions. Systemic involvement may precipitate fatal outcomes. Diagnosis hinges on clinical observation, while prompt initiation of antiviral therapy is imperative to mitigate significant morbidity and mortality risks [4] [5].

2. Case Presentation

We present a case involving a 2-year and 3-month-old male child, brought to the emergency department due to an acute onset of high-grade fever accompanied by a cutaneous rash. The fever commenced 2 days prior to presentation, peaking at 39.6˚C, with partial alleviation following administration of paracetamol. Subsequently, the patient developed a multitude of painful, pruritic vesiculopustular eruptions with oozing, involving the lips, along with redness of the eyes and profuse tearing, leading to oral feeding intolerance. Concurrently, the rash progressively extended to encompass the facial region, while sparing the genitalia. Notably, there was an absence of upper respiratory tract symptoms, abdominal discomfort, or alterations in urinary or bowel patterns. Furthermore, the patient had no documented exposure to sick individuals and was compliant with his scheduled vaccination regimen. The patient had a history of atopic dermatitis, which had not been previously addressed. Upon examination, the child exhibited signs of systemic illness, characterized by severely fissured and exuding lips. Additionally, there were diffuse clusters of umbilicated papulo-vesicles with purulent fluid and crusts predominantly localized in the periorificial and cervical regions. Ophthalmological assessment using a slit lamp and fluorescein eye stain demonstrated bilateral keratoconjunctivitis. Vital signs were recorded as follows: pulse rate of 118 beats per minute, blood pressure of 124/80 mmHg, respiratory rate of 24 breaths per minute, and a temperature of 39˚C. Examination of the head and neck revealed an erythematous and congested pharynx, with no palpable cervical lymphadenopathy. Cardiorespiratory and abdominal examinations yielded unremarkable findings.

Initial laboratory assessments revealed a hemoglobin level of 12.1 g/dL, leukocyte count of 11,500/μL, with neutrophile count of 6050 and normal lymphocyte count (3800), platelet count of 455,000/μL, and C-reactive protein concentration of 200 nmol/L. Cultures obtained from lesion swabs exhibited no bacterial growth. Tzank smear test wasn't performed due to lack of reagent. Chest radiography depicted clear lung fields devoid of opacities. Due to the patient's inability to tolerate oral feeding, admission was deemed necessary for comprehensive evaluation and therapeutic intervention. Subsequently, the dermatology team evaluated the patient and confirmed clinically the condition as eczema herpeticum.

The patient was initiated on antiviral therapy with intravenous acyclovir at a dose of 5 mg/kg/day, alongside empirical antibiotic coverage consisting of topical fusidic acid and oral amoxicillin-clavulanic acid, and eyes treatment with Topical Tobramycin.

Subsequent to the commencement of antimicrobial therapy, the patient exhibited

clinical amelioration, characterized by fever resolution and partial regression of the cutaneous rash within 2 days. Oral intake tolerance was restored during this period. Following a 7-day hospitalization period, the patient was discharged with a prescription for symptomatic management. At the 2-week follow-up visit, complete resolution of the rash was noted, with the patient reporting no ongoing complaints.

3. Discussion

Kaposi varicelliform eruption (KVE) typically manifests in patients with preexisting chronic dermatoses, notably atopic dermatitis. Additionally, documented associations include psoriasis, pityriasis rubra pilaris, Darier's disease, Grover's disease, Hailey-Hailey disease [6], as well as various forms of contact dermatitis [7]. It is noteworthy that a subset of patients may develop KVE in the context of treatment with immunosuppressive agents such as prednisone and cyclophosphamide [8].

The pathogenesis of Kaposi varicelliform eruption (KVE) remains incompletely understood, yet it is widely acknowledged that patients with underlying skin barrier impairments and those experiencing immune deficiencies are particularly susceptible [2]. This susceptibility is thought to arise from dysfunction in both humoral and cellular immunity [6] [9]. A study examining the replication of HSV I in cultured skin samples from patients with atopic dermatitis and psoriasis revealed accelerated replication compared to normal skin [10]. Furthermore, reduced production of certain cytokines, such as interferon or CXCL 10/IP B 10, as well as elevated levels of serum IgE, have been linked to an increased risk of KVE development [11] (**Figures 1 - 3**).

The origin of Kaposi varicelliform eruption (KVE) is believed to stem from either HSV I infection transmitted by family members or close contacts, or from endogenous recurrent infections [12]. There is speculation that HSV may gain entry into the affected skin of Kaposi varicelliform eruption (KVE) due to the overexpression of adhesion molecules [13]. KVE is more prevalent in children, primarily attributed to its association with atopic dermatitis [AD], although occurrences in healthy adults have also been documented [2] [13].

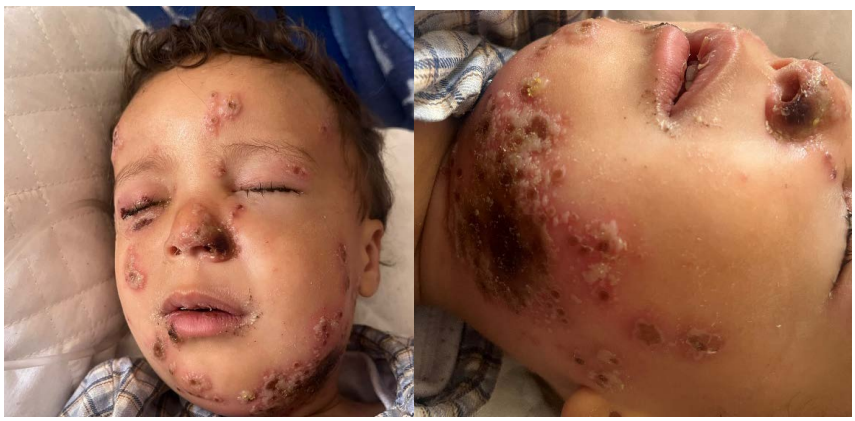

Figure 1. Kaposi's varicelliform eruption involving a child face and affecting periocular area: Erosions covered by hematic crusts day-1.

Figure 2. Kaposi's varicelliform eruption involving a child face; day-3.

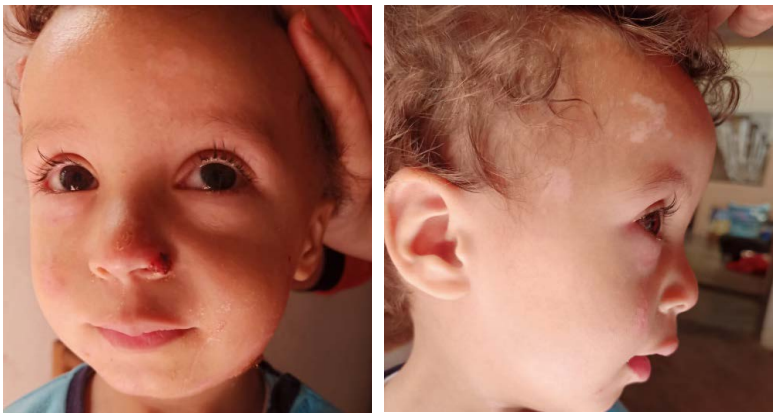

Figure 3. Complete resolution of the rash at the 2-week follow-up visit.

In a study conducted by researchers in Argentina, four recent cases spanning two families were investigated: Two cases involved siblings suffering from severe atopic dermatitis (AD), both being treated with cyclosporine. The other two cases involved previously healthy cousins who developed AD shortly before experiencing Kaposi varicelliform eruption (KVE). Interestingly, there has been a noticeable increase in reported KVE cases since 1980, which is linked to the significant rise in global HSV infections [8].

The diagnosis of Kaposi varicelliform eruption (KVE) is primarily clinical. In cases of uncertainty, a Tzanck smear provides a rapid and cost-effective diagnostic approach [2] [14], although it lacks specificity. A positive Tzanck smear can help rule out smallpox and its vaccine. Additional diagnostic methods include viral DNA detection via PCR, immunofluorescence, vesicular fluid culture, viral serology, and histopathological biopsy [2] [4] [14]-[16].

In our case, no additional investigations were carried out; the diagnosis of eczema herpeticum was established based exclusively on clinical criteria, specifically the rapid clinical improvement observed after initiating acyclovir therapy.

Differential diagnoses for KVE include chickenpox, impetigo and contact dermatitis [2] [10].

The most frequent complications involve bacterial infections, which can

progress to sepsis and viremia [2] [14] affecting various organs such as the lungs, liver, brain, gastrointestinal tract, and adrenal glands. Ocular involvement can lead to vision loss [2]. With Staphylococcus aureus commonly implicated in secondary infections of erosions [6], although beta-hemolytic streptococci and Pseudomonas may also be isolated.

Acyclovir is the preferred therapeutic agent for treating eczema herpeticum. Given its limited oral bioavailability, oral administration is typically reserved for mild cases. Prompt initiation of acyclovir treatment is crucial, as delayed administration has been correlated with prolonged hospital stays [17]. Empirical antibiotic therapy has not demonstrated efficacy in reducing hospital stays or mortality rates [18].

As Acyclovir poor oral bioavailability, valacyclovir, has been developed [19]. In treatment with Valacyclovir, 500 mg orally twice a day for 5 days is recommended for EH [20]. Penciclovir (PCV) and its oral prodrug famciclovir act similarly to Acyclovir and may be used in countries where it is marketed in dosages of 5 mg/kg/dose, twice a day for 7 days [21]-[23].

Sometimes, treatments may fail due to drug resistance [21]. In a cohort of 8 patients with ADEH+, 3 were found to have acyclovir-resistant HSV [24]. This resistance is often caused by viral mutations, primarily in the UL23 gene, or in the UL30 gene. To address this resistance, alternative treatments such as foscarnet (FOS) and cidofovir [CDV] can be used, forty mg/kg 3 times a day for 2 to 3 weeks are recommended for foscarnet [20] [25].

In addition to traditional treatments, new therapeutic options should be explored, particularly for patients infected with resistant strains. One promising alternative is IFN-γ therapy, which may be especially beneficial for atopic dermatitis (AD) patients with low levels of IFN-γ [22] [24].

This treatment can help control HSV replication and spread, offering significant advantages in cases of EH. However, as of now, the clinical improvement seen in patients has not justified the very high cost of this therapy.

In cases where bacterial culture results are pending, empiric antibiotic therapy is recommended, particularly due to the high incidence of secondary Staphylococcus aureus superinfection in eczema herpeticum cases, which can occur in up to 30% of patients [17].

The case underscores the importance of identifying this condition despite its low incidence, particularly when clinical settings are indicative. Early administration of acyclovir is crucial to shorten the disease course and prevent its spread.

4. Conclusion

Eczema herpeticum poses significant risks of morbidity and mortality, especially if diagnosis and treatment are delayed. Historically, prior to the advent of acyclovir, mortality rates from eczema herpeticum were as high as 50% [1]. Complications can include severe conditions such as meningoencephalitis, keratoconjunctivitis, and disseminated intravascular coagulation. Timely recognition and

management are critical to improving patient outcomes in this condition [21] [26].

Conflicts of Interest

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

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