

Prolonged remission of pediatric pityriasis lichenoides et varioliformis acuta after systemic corticosteroid therapy: a case report and scoping review

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Abstract

Background

Pityriasis lichenoides is a rare inflammatory dermatosis characterized by a heterogeneous clinical course and poorly standardized treatment strategies. Approximately 20% of cases occur during childhood, yet evidence regarding systemic corticosteroid therapy remains limited.

Methods

We report the case of a 2-year-old boy with pityriasis lichenoides et varioliformis acuta (PLEVA) treated with systemic corticosteroids. A scoping review of the literature was also performed to summarize current therapeutic approaches in pediatric PLEVA.

Results

The child developed PLEVA after an episode of acute gastroenteritis. Initial treatment with acyclovir and azithromycin resulted in minimal clinical benefit. A subsequent severe relapse with intense pruritus and secondary impetiginisation required hospitalization. Systemic corticosteroid therapy administered with gradual tapering produced rapid and marked clinical improvement, followed by complete remission within two weeks. A later flare associated with febrile pharyngotonsillitis responded promptly to another short steroid course. No additional relapses occurred during 15 months of follow-up.

Conclusions

Although current evidence does not support systemic corticosteroids as standard therapy for PLEVA, selected clinical experiences suggest that carefully tapered regimens may contribute to rapid disease control and sustained remission. Larger prospective studies are needed to better define their efficacy and safety profile.

Introduction

Pityriasis lichenoides (PL) comprises a spectrum of uncommon inflammatory skin disorders ranging from the acute form, pityriasis lichenoides et varioliformis acuta (PLEVA), to the chronic form, pityriasis lichenoides chronica (PLC), the most frequent one [1]: the mixed form (MPL) is characterized by both PLEVA and PLC lesions. A distinct variant is febrile ulceronecrotic Mucha–Habermann disease (FUMHD) [1–8]. In PL, the definitions “acute” and “chronic” describe more the morphology of the lesions than the duration of the disease itself. PLEVA is characterized by erythematous macules that may evolve into papules with micaceous scale free at the periphery; these can progress into vesicular, pustular, necrotic, or varioliform lesions and subsequently heal with atrophic scarring, present in 36% of a large series [1],

and post-inflammatory hyper- and hypopigmentation [1–8]. The eruption is polymorphous, as lesions exist in all stages of development, and occasionally small lesions of oral mucosa can be appreciated [1]. Conversely, PLC typically presents with persistent flattened red-brown papules covered by fine mica-like scales evolving into hypochromic patches without scarring; it is generally asymptomatic and typically follows a relapsing course characterized by prolonged periods of remission. Mixed pityriasis lichenoides (MPL) includes features of both forms. In PLEVA, PLC, and MPL, lesions may spontaneously regress, although relapses or persistent disease lasting months to years are common [1–8].

Approximately one-fifth of PL cases occur during childhood, with peak incidence reported between 5 and 10 years of age [5, 6] and PLEVA with a younger median age of appearance [7]. Several studies have described a male predominance [1–10] which seems much more evident in childhood [1]. No definitive associations with ethnicity, geographical distribution, or environmental risk factors have been established [11]. The disease course is generally benign. Reports of progression to cutaneous lymphoma are exceedingly rare and almost exclusively described in adults [1, 12, 13]. Moreover, large pediatric follow-up studies have not demonstrated progression to lymphomatoid papulosis or other cutaneous lymphomas [7, 10].

Methods

A scoping review was conducted to map the available evidence regarding PL in pediatric patients, with particular attention to therapeutic strategies and the potential role of systemic corticosteroids. The literature search was conducted up to January 2026. Articles published in English were eligible for inclusion. The scoping review was conducted and reported in accordance with the PRISMA Extension for Scoping Reviews (PRISMA-ScR) guidelines. Literature searches were performed in PubMed, Scopus, Web of Science, Embase, and the Cochrane Library. The search strategy combined Medical Subject Headings (MeSH) and free-text terms, including: “pityriasis lichenoides,” “children,” “pediatric,” “management,” “systemic,” “corticosteroids,” “treatment,” “therapy,” and “prognosis.” A search of references was performed to identify additional relevant articles. Eligible studies included case reports, case series, literature reviews, systematic reviews, retrospective analyses, clinical trials, randomised controlled trials, observational studies, clinical guidelines, and previous scoping or narrative reviews. Titles and abstracts were screened independently by two authors, and potentially eligible articles underwent full-text review. In accordance with scoping review methodology, no formal risk-of-bias or quality assessment was performed. The results were synthesized narratively, with particular attention to therapeutic regimens and outcomes. A PRISMA flow diagram summarizing the study selection process is provided in Figure 1, and the PRISMA-ScR checklist is available as supplementary material.

Case presentation

We report the case of a previously healthy boy aged 2 years who developed PL following an episode of acute gastroenteritis during international travel.

The initial eruption consisted of polymorphic papular lesions involving the trunk, with a varioliform appearance. Empirical treatment with topical and oral acyclovir proved ineffective, and the skin manifestations showed recurrent phases of partial improvement and exacerbation.

One month later, following pediatric dermatologic evaluation, a diagnosis of pityriasis lichenoides et varioliformis acuta (PLEVA) was established; skin biopsy was not performed because the diagnosis was considered clinically typical by the consulting pediatric dermatologist. Laboratory investigations performed during the diagnostic work-up included sweat testing, immunoglobulin levels, lymphocyte subsets, Epstein–Barr virus serology, serum protein electrophoresis, thyroid function tests, iron studies, and ferritin levels; all results were within normal limits.

Initial treatment consisted of a short course of oral corticosteroids associated with two azithromycin cycles of 3 days. Although partial clinical improvement was initially observed, the patient subsequently experienced a severe relapse characterized by intense pruritus and secondary impetiginisation, leading to hospitalization.

At admission, the child was afebrile but appeared mildly distressed and restless because of severe pruritus. Skin lesions (Figure 2) were predominantly distributed over the trunk and proximal upper and lower limbs. The eruption consisted of markedly erythematous polymorphic papules, including vesicular lesions, papulopustules, and crusted papules, particularly involving the flanks and buttocks. Several lesions showed signs of secondary bacterial impetiginisation. The face and distal extremities were spared. Laboratory investigations revealed no significant abnormalities.

Systemic corticosteroid therapy was initiated with oral betamethasone at 0.1 mg/kg/day, followed by prednisone at approximately 1 mg/kg/day. Topical fusidic acid was initially prescribed for impetiginized lesions. Rapid clinical improvement was observed within a few days. The child was discharged after six days of hospitalization in good clinical condition and a complete remission was evident after approximately 14 days of treatment (Figure 3). The total duration of corticosteroid therapy was 28 days, including gradual tapering. He remained stable for 4 months. A further slight flare occurred 5 months later following febrile pharyngotonsillitis but responded promptly to a short course (4 days) of systemic corticosteroids administered at home. At 15-month follow-up, the patient remained in good general health with no additional relapses.

Discussion

The pathogenesis of PL remains incompletely understood. Proposed mechanisms include post-infectious inflammatory activation [12], clonal T-cell proliferation [13, 14], and immune complex-mediated vasculitis [2, 14]. Infectious triggers, particularly viral or bacterial upper respiratory and gastrointestinal infections, have frequently been reported before disease onset or relapse [12]. More recently, the potential involvement of plasmacytoid dendritic cells and type I interferon signalling pathways has been suggested [15, 16].

Clinically, PLEVA is characterized by a highly polymorphic eruption and, due to the relapsing nature of the disease, lesions at different evolutionary stages commonly coexist. The trunk, the buttocks and proximal extremities are typically symmetrically involved, whereas the face and distal limbs are usually spared [1–10]. Symptoms include pruritus and, occasionally, fever and arthralgias [1–8]. Diagnosis is primarily clinical and supported by the characteristic morphology and disease course.

Histopathological examination may be useful in atypical presentations or when differential diagnoses such as lymphomatoid papulosis, cutaneous T-cell lymphoma, or Langerhans cell histiocytosis are considered. In an older series, biopsy was performed in 61% of PL patients [7]. In PLEVA, histopathology typically shows vacuolar degeneration of the basal layer, necrotic keratinocytes, and a dermal lymphocytic infiltrate predominantly consisting of CD8-positive, CD4- and CD30-negative lymphocytes [1, 2, 3, 7, 17]. The differential diagnosis for PLEVA in children includes principally varicella, Gianotti-Crosti syndrome, Langerhans cell histiocytosis, arthropod bite reactions, erythema multiforme, and pityriasis rosea, whereas guttate psoriasis, vasculitis, secondary syphilis, and lymphomatoid papulosis are much more common in adulthood [3].

In the present case, PLEVA appeared to develop following an infectious trigger, consistent with current pathogenetic hypotheses suggesting infection-driven immune dysregulation. The extent of cutaneous involvement was remarkable, consistent with previous observations that lesional severity may be greater in children than in adults [10].

Therapeutic management of PL remains challenging and poorly standardized, largely because of the uncertain etiology of the disease and its tendency toward spontaneous resolution. These features limit the ability of uncontrolled studies to provide reliable therapeutic recommendations: furthermore, many studies have not distinguished between PLC and PLEVA when reporting about the treatment efficacy. Our scoping review confirmed substantial variability in therapeutic strategies. Topical corticosteroids, topical calcineurin inhibitors and antihistamines have been used for symptomatic relief but appear to have limited impact on the natural course of the disease [7, 8, 10, 18–22]. Several systemic therapies have been reported with variable efficacy, including antibiotics—particularly tetracycline and macrolides such as erythromycin—, phototherapy (NB UVB, BB UVB, UVA1 and PUVA), methotrexate, pentoxifylline, pyrimethamine, trisulfapyrimidine, calciferol, quinoline and acridine derivatives, cyclosporine, bromelain, intravenous immunoglobulins, retinoids, aciclovir, valaciclovir, dupilumab, tacrolimus, and other systemic immunomodulatory agents [1, 3, 7, 8, 10–12, 22–30]. It is notable that, although robust evidence of efficacy is lacking, oral tetracycline and erythromycin, along with phototherapy, remain among the most frequently prescribed therapies, even in children [10] who typically present worse response rates to treatments compared with adults [22, 26].

In the present case the initial therapeutic approach, consisting of antibiotics and a short (3 days) corticosteroid course, resulted in only transient improvement, whereas sustained disease control was observed following prolonged systemic corticosteroid therapy. The role of systemic corticosteroids remains controversial, published evidence being mainly restricted to isolated case reports or severe

refractory presentations: notably among the 393 PL patients analysed in the review of Geller [12] only 2 received systemic corticosteroid therapy. Prednisone was administered at low doses for up to 3 weeks in 33% of patients in the series reported by Popadić: it was reserved to more severe patients but no specific details about its efficacy were presented. To our knowledge there is only one case [29] treated with systemic steroids for a long period (5 months), even though associated to erythromycin for 3 months: this adult patient remained disease free during 2 years of follow-up. It is possible that, especially in pediatric patients, the use of systemic steroids may have been limited by concerns regarding potential adverse effects; however, it is biologically plausible that systemic corticosteroids may provide clinical benefit in selected inflammatory presentations. In our patient, who was only 2 years old, systemic corticosteroids produced rapid clinical improvement with a complete remission in less than 2 weeks: it is worth noting that in a pediatric PL series, after a median disease duration of 30 months, only 20% of patients had achieved complete disease control [10] and in another paediatric population study, the median duration of PLEVA was 18 months [7].

This case highlights several clinically relevant aspects. First, diagnosis may be challenging because of the polymorphic nature of the eruption and overlap with infectious, inflammatory, and lymphoproliferative disorders: in our patient the time between the first dermatologic visit and the diagnosis was only 2 weeks, but in a previous PLEVA series the mean time from symptom onset to diagnosis was 9 months, the most frequent misdiagnoses being scabies, varicella, and arthropod bite reaction [30]. Second, empirical antimicrobial therapy alone may be insufficient in severe presentations [29]. Third, gradually tapered systemic corticosteroid therapy may contribute to rapid control of acute inflammation and possibly to longer periods of disease control in selected patients, such as the present one: furthermore, in our opinion, it should be emphasized that a 3–4 week course of home-based systemic corticosteroid therapy may have a substantially lower impact on family quality of life and daily functioning compared with phototherapy, which inevitably requires treatment over many weeks and mainly three times weekly [11]; furthermore, although NB-UVB, the most commonly used phototherapy modality in PL treatment [26], is generally considered safe, the long-term cumulative carcinogenic risk remains, especially in pediatric practice, incompletely defined. Nevertheless, caution is warranted because systemic corticosteroids may be associated with significant adverse effects, especially in children. Their use should therefore remain individualized and carefully balanced against disease severity, symptom burden, and alternative therapeutic options.

This report has several limitations. First, it describes a single patient, and therefore the observed clinical outcome cannot be generalized to the broader pediatric population with PLEVA. Second, histopathological confirmation was not obtained because the diagnosis was considered clinically typical and unequivocal by the consulting pediatric dermatologist. Third, because of the unpredictable and relapsing nature of the disease, a spontaneous remission cannot be completely excluded, although the temporal association between corticosteroid administration and the rapid clinical improvement observed in our child is highly suggestive of a therapeutic effect. Finally, the accompanying scoping review was designed to map the available evidence rather than to provide a quantitative assessment of treatment efficacy; therefore, no formal risk-of-bias evaluation or meta-analysis was performed. These

limitations highlight the need for prospective multicentre studies evaluating systemic corticosteroids and other therapeutic approaches in pediatric PLEVA.

Conclusions

PL remains a therapeutic challenge in pediatric patients because of its heterogeneous presentation and unpredictable clinical course. This case suggests that systemic corticosteroids administered with gradual tapering may represent a useful therapeutic option in selected severe pediatric presentations. However, the evidence currently available remains limited, and controlled prospective studies are required before systemic corticosteroids can be considered part of standardized treatment algorithms.

Declarations

Ethics approval and consent to participate

The clinical management of the patient was conducted in accordance with the ethical standards of the institutional and national research committee and with the principles of the Declaration of Helsinki. Written informed consent for the use of clinical data for scientific purposes was obtained from the patient's legal guardians.

Consent for publication

Written informed consent for publication of the clinical details was obtained from the patient's legal guardians.

Availability of data and materials

The data supporting the findings of this study are included within the article. Additional information can be made available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

GFS, PF, and MM contributed to the clinical management of the patient and to the conceptualization of the manuscript. GFS and PF screened titles and abstracts independently. MGS, MG, FM, GFS, CM, SC and TGR collected and analysed the clinical data. SM, BT, FV and GC contributed to the literature review and drafting of the manuscript. GFS, PF, AL and GC supervised the study and critically revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

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Figures

PRISMA Flow Diagram

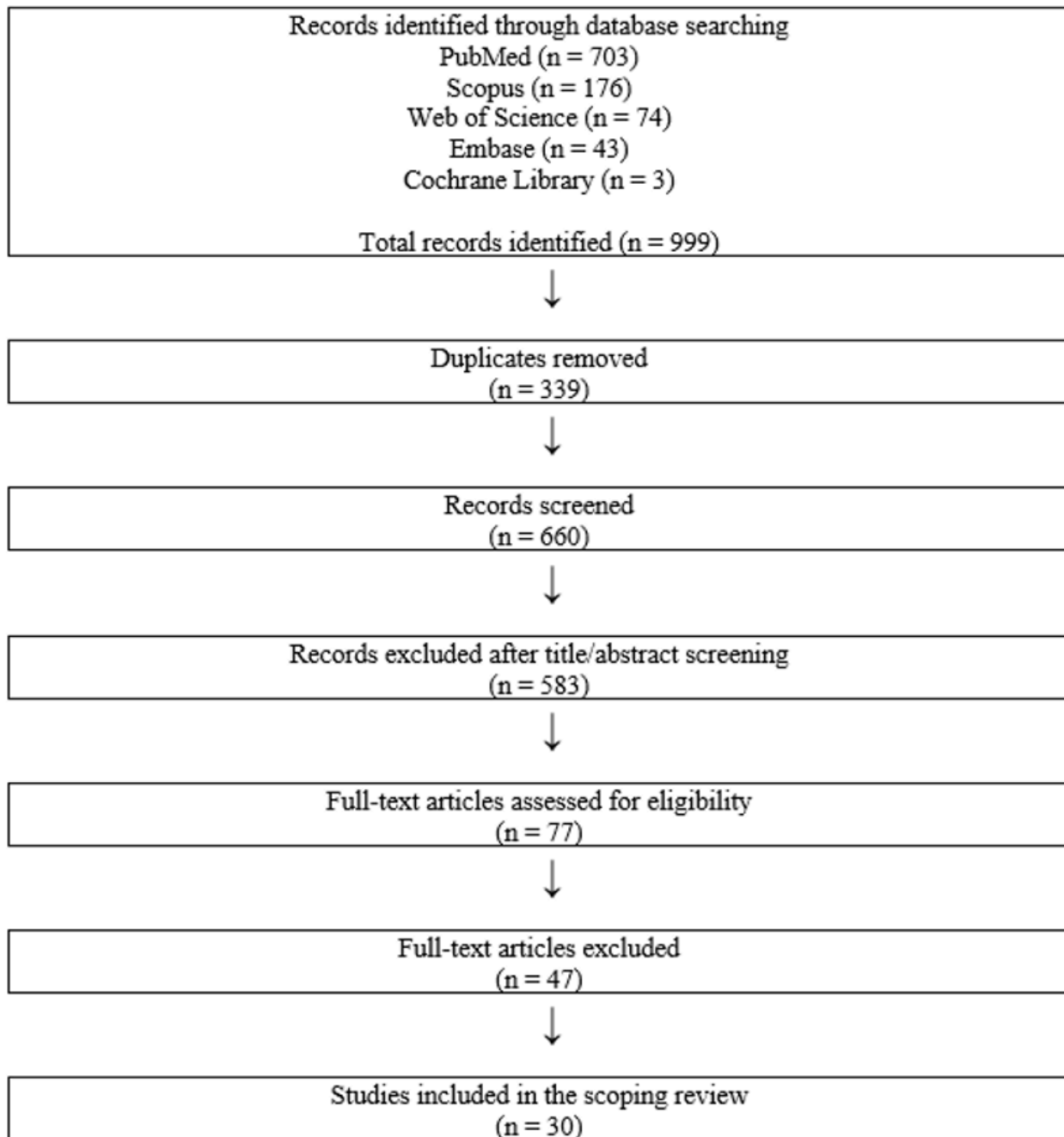


Figure 1

PRISMA flow diagram summarizing the study selection process for the scoping review



Figure 2

Erythematous lesions, polymorphic papules, including vesicular lesions, papulopustules, and crusted papules.



Figure 3

Complete remission after 2 weeks of systemic corticosteroid therapy.