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ORIGINAL ARTICLE

Comparison of Two Topical Steroid Regimens in Managing Pediatric Atopic Dermatitis

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ABSTRACT:
Background: Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disease of childhood that causes intense pruritus, sleep disturbance and impaired quality of life. Topical corticosteroids are the mainstay of therapy, but the optimal regimen for sustained disease control with acceptable safety in children remains a matter of debate. **Aim:**To compare the efficacy and safety of a conventional reactive topical steroid regimen with a proactive intermittent topical steroid regimen in children with mild-to-moderate atopic dermatitis attending a tertiary care hospital. **Material and Methods:**This prospective, randomized, comparative study included 76 children aged 2–16 years with mild-to-moderate AD fulfilling Hanifin and Rajka criteria. Patients were randomized 1:1 into Regimen A (reactive) and Regimen B (proactive). Both groups received a mid-potency topical corticosteroid once daily during active disease until clinical control, along with emollients. In Regimen A, steroid was then stopped and only emollients continued; in Regimen B, the steroid was continued as proactive maintenance on previously affected sites on two consecutive days per week plus daily emollients. Outcomes assessed at baseline and follow-up included SCORAD, EASI, body surface area (BSA) involvement, pruritus and sleep disturbance visual analogue scales (VAS), Children's Dermatology Life Quality Index (CDLQI), Investigator's Global Assessment (IGA) and adverse events. **Results:**Both groups were comparable at baseline in age, sex, disease duration and severity scores. Regimen B showed significantly greater mean reductions in SCORAD (25.47 ± 7.12 vs. 18.84 ± 6.33; $p = 0.001$), EASI (4.29 ± 1.21 vs. 3.12 ± 1.08; $p < 0.001$) and BSA (10.68 ± 3.02% vs. 7.95 ± 2.44%; $p < 0.001$) compared with Regimen A. Symptom relief was also superior with Regimen B for pruritus VAS (4.16 ± 1.07 vs. 3.24 ± 1.01; $p = 0.002$), sleep disturbance VAS (2.98 ± 0.92 vs. 2.15 ± 0.87; $p = 0.001$) and CDLQI (7.11 ± 2.08 vs. 5.42 ± 1.89; $p = 0.001$). At final follow-up, "clear" or "almost clear" IGA status was more frequent in Regimen B (68.42%) than in Regimen A (39.48%), while moderate disease persisted more often in Regimen A (26.32% vs. 7.89%; $p = 0.04$). Overall adverse events were infrequent and identical in both groups (18.42%), with no serious safety concerns. **Conclusion:**Proactive intermittent mid-potency topical steroid therapy following initial control provides superior clinical improvement and quality-of-life benefits compared to a purely reactive regimen, without increasing adverse effects, and represents an effective, well-tolerated strategy for long-term management of pediatric atopic dermatitis.
Keywords: Atopic dermatitis; children; topical corticosteroids; proactive therapy; randomized study

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INTRODUCTION

Atopic dermatitis (AD) is one of the most common chronic inflammatory skin diseases of childhood, characterized by intense pruritus, eczematous lesions and a relapsing–remitting course. It imposes a substantial public health burden worldwide, with population-based data from the International Study of Asthma and Allergies in Childhood (ISAAC) Phase Three showing considerable global variation but consistently high prevalence of eczema symptoms in both developed and developing countries.[1] The disease typically begins in early childhood and may persist into adolescence or adulthood, contributing to the so-called "atopic march," in which AD is followed by allergic rhinitis and asthma. The high frequency of pediatric AD means that even modest improvements in disease control or prevention of flares can translate into significant reductions in health-care utilization and family distress.Beyond its cutaneous manifestations, AD profoundly affects quality of life for both children and their caregivers. Persistent itching leads to sleep disturbance, impaired school performance, daytime irritability and emotional distress. Parents often report exhaustion, anxiety and guilt related to the child's discomfort and visible lesions. Comprehensive reviews highlight that AD is a paradigmatic chronic, relapsing disorder with complex genetic and environmental determinants, and that it frequently coexists with other atopic diseases such as food allergy and asthma.[2] This multifaceted burden underscores the need for treatment strategies that are not only effective during acute flares but also acceptable and sustainable for long-term management in young children.Current understanding of AD pathophysiology emphasizes a central role for epidermal barrier dysfunction in genetically

predisposed individuals, interacting with immune dysregulation and environmental triggers. Defects in structural proteins such as filaggrin, alterations in epidermal lipids and increased transepidermal water loss facilitate penetration of irritants, allergens and microbes, thereby amplifying cutaneous inflammation.[3] This "outside–inside" concept complements earlier immune-centered views by suggesting that restoration and maintenance of barrier integrity is a cornerstone of AD management. In clinical practice, this has translated into strong emphasis on daily emollient use and avoidance of irritants as the foundation of all treatment regimens, with anti-inflammatory therapies layered on as needed for disease control.Diagnosis of AD in children remains clinical and is guided by established criteria. The classic Hanifin and Rajka criteria, based on combinations of major and minor features such as pruritus, typical morphology and distribution, chronic course and personal or family history of atopy, continue to inform both routine care and research definitions.[4] Over time, standardized outcome measures such as the Scoring Atopic Dermatitis (SCORAD) index, the Eczema Area and Severity Index (EASI) and validated quality-of-life instruments have been developed to quantify disease severity and treatment response. These tools are essential for comparing therapeutic regimens, designing clinical trials and evaluating the real-world effectiveness of different management strategies in pediatric populations.Topical therapy remains the mainstay of AD treatment, with international and national guidelines traditionally advocating a stepwise approach in which emollients are used regularly and topical anti-inflammatory agents are introduced according to disease severity and body site. A landmark systematic review of treatments for atopic eczema highlighted that, despite the proliferation of complementary and alternative interventions, the strongest randomized controlled trial evidence supported the use of topical corticosteroids, ultraviolet light and, in more severe cases, systemic immunosuppressants.[5] Within this framework, mid-potency topical corticosteroids are widely used for short courses to control acute flares in children with mild-to-moderate AD, especially when lesions involve the trunk and limbs rather than delicate areas such as the face or intertriginous regions.Topical corticosteroids (TCS) exert potent anti-inflammatory, immunosuppressive and antiproliferative effects and have been shown to rapidly reduce erythema, edema and pruritus in AD. Their efficacy and short-term safety in pediatric patients have been demonstrated in multiple clinical trials, including randomized, double-blind, placebo-controlled studies of fluticasone propionate 0.05% lotion applied once daily in subjects as young as three months, which confirmed significant improvement in investigator global assessments and lesion scores without clinically relevant systemic adverse effects over four weeks of treatment.[6] Nonetheless, concerns about local adverse effects such as skin atrophy, telangiectasia and striae, as well as potential systemic absorption, remain central when planning repeated or long-term use in children.In addition to objective safety considerations, parental and patient perceptions about topical steroids substantially influence adherence. "Steroid phobia," encompassing fears of dependence, skin thinning and systemic harm, has been documented in both adults and parents of children with AD and is associated with under-use of prescribed TCS and suboptimal disease control.[7] These concerns are particularly salient in pediatric practice, where caregivers may hesitate to continue treatment once visible inflammation improves, leading to rapid relapse and a "flare-treat-flare" cycle that further reinforces negative attitudes toward steroids. Any regimen intended for long-term management must therefore balance efficacy with a dosing schedule that is acceptable and reassuring to families.

MATERIALS AND METHODS

This was a prospective, randomized, comparative study conducted in the Department of Dermatology of a tertiary care teaching hospital. The study was designed to compare the efficacy and safety of two topical corticosteroid regimens in children with atopic dermatitis (AD). All procedures were performed in accordance with the institutional ethical standards, and approval was obtained from the Institutional Ethics Committee prior to initiation. Written informed consent was obtained from parents or legal guardians, and assent was taken from children whenever appropriate.A total of 76 pediatric patients with clinically diagnosed atopic dermatitis were included. Children of either sex, aged between 2 to16 years, fulfilling the Hanifin and Rajka diagnostic criteria for AD and presenting with mild to moderate disease severity were eligible. Patients with severe AD requiring systemic immunosuppressants, concurrent skin infections requiring systemic antibiotics, known hypersensitivity to topical corticosteroids, presence of other chronic dermatological conditions that could interfere with assessment (e.g., psoriasis, ichthyosis), or those who had received systemic steroids or immunomodulatory drugs shortly before enrollment were excluded. Children with significant systemic comorbidities (such as uncontrolled asthma or immunodeficiency) were also excluded.

Methodology

The 76 eligible children were randomized in a 1:1 ratio into two treatment groups (Regimen A and Regimen B) using a computer-generated random sequence. Allocation concealment was ensured by using sequentially numbered, opaque, sealed envelopes that were opened only after enrollment. The prescribing dermatologist was aware of the allocated regimen, while the outcome assessor was kept blinded to treatment assignment to minimize observer

bias.Regimen A received a mid-potency topical corticosteroid (for example, fluticasone propionate 0.05% cream) applied once daily to affected areas during active disease until clinical control of lesions was achieved, followed by discontinuation and use of bland emollients alone. Regimen B received the same mid-potency topical corticosteroid applied once daily to affected areas during active disease until clinical control, followed by a proactive maintenance regimen in which the corticosteroid was applied intermittently (for example, on two consecutive days per week) to previously affected, clinically stable areas along with daily emollient therapy. In both groups, parents were instructed on correct fingertip unit dosing, gentle skin care, liberal use of fragrance-free emollients, and avoidance of identified triggers. Use of non-sedating oral antihistamines for symptomatic relief of pruritus was allowed in both groups as needed and recorded as concomitant medication.

At baseline, a detailed history and clinical examination were performed, including age, sex, age at onset of AD, disease duration, family history of atopy, and previous treatments. Disease severity was assessed using the Scoring Atopic Dermatitis (SCORAD) index and the Eczema Area and Severity Index (EASI). The percentage of body surface area (BSA) involved was recorded. Investigator's Global Assessment (IGA) on a standardized 5-point scale (clear, almost clear, mild, moderate, severe) was documented. Symptom intensity was evaluated using a 10-cm visual analogue scale (VAS) for pruritus and a separate 10-cm VAS for sleep disturbance, as reported by the parent or caregiver. Health-related quality of life was assessed using the Children's Dermatology Life Quality Index (CDLQI) in age-appropriate patients. These parameters (SCORAD, EASI, BSA involvement, IGA, pruritus VAS, sleep disturbance VAS, and CDLQI) were recorded at baseline and at each scheduled follow-up visit to monitor response to treatment.

Safety evaluation included careful examination of treated areas at each visit for local adverse effects of topical corticosteroids such as skin atrophy, telangiectasia, hypopigmentation, striae, folliculitis, and acneiform eruptions. Parents were specifically questioned about stinging, burning, or irritation after application, as well as any systemic symptoms potentially related to steroid use. Need for treatment modification or discontinuation due to adverse events was documented. Use of rescue medication (such as topical calcineurin inhibitors or short courses of systemic therapies if deemed necessary by the treating dermatologist) and any intercurrent skin infections were also recorded.

RESULTS

Table 1 explains the baseline demographic characteristics of the study participants. Both treatment groups were comparable at baseline, indicating successful randomization. The mean age of children in Regimen A was 7.82 ± 3.41 years, while in Regimen B it was 8.05 ± 3.27 years, with no significant difference between the groups ($p = 0.72$). The sex distribution was similarly balanced, with males comprising 57.89% in Regimen A and 55.26% in Regimen B ($p = 0.82$). Mean disease duration was also comparable between the two groups (2.14 ± 1.01 years vs. 2.08 ± 0.97 years; $p = 0.78$). A family history of atopy was present in 39.47% of children in Regimen A and 44.74% in Regimen B, showing no significant difference ($p = 0.65$). Additionally, the age at onset of dermatitis did not differ significantly between the groups (4.25 ± 1.82 years vs. 4.11 ± 1.77 years; $p = 0.73$).

Table 2 describes the baseline severity of atopic dermatitis before treatment initiation. The mean SCORAD score was 42.36 ± 8.12 in Regimen A and 41.89 ± 7.95 in Regimen B, indicating comparable disease severity ($p = 0.78$). Likewise, EASI scores were similar between the groups (6.74 ± 2.21 vs. 6.59 ± 2.17; $p = 0.74$). The body surface area (BSA) involvement also showed no significant difference (18.42 ± 6.25% vs. 17.97 ± 6.11%; $p = 0.68$). Symptom severity parameters, including pruritus VAS and sleep disturbance VAS, were nearly identical, with pruritus scores of 7.21 ± 1.15 vs. 7.05 ± 1.12 ($p = 0.53$) and sleep disturbance scores of 5.89 ± 1.42 vs. 6.02 ± 1.38 ($p = 0.66$). Similarly, quality-of-life impairment measured by CDLQI showed no statistically significant difference at baseline (11.47 ± 3.05 vs. 11.26 ± 3.12; $p = 0.74$).

Table 3 presents the post-treatment improvement in disease severity scores and highlights the superior efficacy of Regimen B. Regimen B demonstrated significantly greater reductions across all clinical parameters. The reduction in SCORAD was markedly higher in Regimen B (25.47 ± 7.12) compared to Regimen A (18.84 ± 6.33), with a significant p-value of 0.001. Similarly, EASI score reduction was significantly greater in Regimen B (4.29 ± 1.21) than in Regimen A (3.12 ± 1.08), with $p < 0.001$. BSA involvement decreased by 10.68 ± 3.02% in Regimen B, significantly more than the 7.95 ± 2.44% reduction in Regimen A ($p < 0.001$). Symptom relief was also superior in Regimen B, where pruritus VAS reduction was 4.16 ± 1.07 vs. 3.24 ± 1.01 in Regimen A ($p = 0.002$). Sleep disturbance improved significantly more in Regimen B, with a reduction of 2.98 ± 0.92 compared to 2.15 ± 0.87 in Regimen A ($p = 0.001$). Quality of life also improved more in the proactive therapy group, where CDLQI reduction was 7.11 ± 2.08 vs. 5.42 ± 1.89 ($p = 0.001$).

Table 4 compares the Investigator's Global Assessment (IGA) outcomes at the final follow-up. A higher proportion of children in Regimen B achieved better clinical clearance. Specifically,

28.95% of patients in Regimen B reached the "clear" category compared to 13.16% in Regimen A, although this difference did not reach statistical significance (p = 0.08). Additionally, more children in Regimen B (39.47%) achieved an "almost clear" status compared to Regimen A (26.32%), though this difference was also not statistically significant (p = 0.22). Mild disease was observed in 34.21% of Regimen A participants and 23.68% of Regimen B participants. Importantly, the proportion of children with moderate disease was significantly higher in Regimen A (26.32%) compared to Regimen B (7.89%), with a p-value of 0.04, indicating a statistically meaningful difference. No participants in either group fell into the "severe" category post-treatment.

Table 5 outlines the adverse effects reported during treatment and shows that both regimens were comparably safe. The frequency of adverse events was low and similar across both groups. Skin atrophy occurred in only one patient (2.63%) in Regimen A, while none in Regimen B exhibited this effect (p = 0.31). Hypopigmentation was seen in 5.26% of Regimen A and 2.63% of Regimen B (p = 0.55). Folliculitis occurred in 2.63% of Regimen A and 5.26% of Regimen B, without significant difference (p = 0.55). Stinging or burning sensations were reported by 7.89% in Regimen A and 10.53% in Regimen B (p = 0.69). No cases of telangiectasia were reported in either group. Importantly, the overall incidence of any adverse event was identical (18.42%) in both regimens with a p-value of 1.00, indicating no difference in safety profiles.

Table 1. Baseline Demographic Characteristics of Study Participants (N = 76)

Parameter	Regimen A (n=38)	Regimen B (n=38)	p-value
Mean Age (years) ± SD	7.82 ± 3.41	8.05 ± 3.27	0.72
Sex: Male	22 (57.89%)	21 (55.26%)	0.82
Sex: Female	16 (42.11%)	17 (44.74%)	—
Mean Disease Duration (years) ± SD	2.14 ± 1.01	2.08 ± 0.97	0.78
Family History of Atopy	15 (39.47%)	17 (44.74%)	0.65
Mean Age at Onset (years) ± SD	4.25 ± 1.82	4.11 ± 1.77	0.73

Table 2. Baseline Disease Severity Parameters

Parameter	Regimen A (n=38) Mean ± SD	Regimen B (n=38) Mean ± SD	p-value
SCORAD Score	42.36 ± 8.12	41.89 ± 7.95	0.78
EASI Score	6.74 ± 2.21	6.59 ± 2.17	0.74
BSA Involvement (%)	18.42 ± 6.25	17.97 ± 6.11	0.68
Pruritus VAS (0–10)	7.21 ± 1.15	7.05 ± 1.12	0.53
Sleep Disturbance VAS (0–10)	5.89 ± 1.42	6.02 ± 1.38	0.66
CDLQI Score	11.47 ± 3.05	11.26 ± 3.12	0.74

Table 3. Post-treatment Improvement in Disease Severity Scores

Parameter	Regimen A Mean Reduction ± SD	Regimen B Mean Reduction ± SD	p-value
Reduction in SCORAD	18.84 ± 6.33	25.47 ± 7.12	**0.001**
Reduction in EASI	3.12 ± 1.08	4.29 ± 1.21	**<0.001**
Reduction in BSA (%)	7.95 ± 2.44	10.68 ± 3.02	**<0.001**
Reduction in Pruritus VAS	3.24 ± 1.01	4.16 ± 1.07	**0.002**
Reduction in Sleep Disturbance VAS	2.15 ± 0.87	2.98 ± 0.92	**0.001**
Reduction in CDLQI Score	5.42 ± 1.89	7.11 ± 2.08	**0.001**

Table 4. Investigator's Global Assessment (IGA) at Final Follow-up

IGA Category	Regimen A (n=38)	Percentage	Regimen B (n=38)	Percentage	p-value
Clear	5	13.16%	11	28.95%	0.08
Almost Clear	10	26.32%	15	39.47%	0.22
Mild	13	34.21%	9	23.68%	0.28
Moderate	10	26.32%	3	7.89%	**0.04**
Severe	0	0.00%	0	0.00%	—

Table 5. Adverse Effects Reported During Treatment

Adverse Event	Regimen A (n=38)	Percentage	Regimen B (n=38)	Percentage	p-value
Skin Atrophy	1	2.63%	0	0.00%	0.31
Hypopigmentation	2	5.26%	1	2.63%	0.55

Folliculitis	1	2.63%	2	5.26%	0.55
Stinging/Burning	3	7.89%	4	10.53%	0.69
Telangiectasia	0	0.00%	0	0.00%	—
Any Adverse Event	7	18.42%	7	18.42%	1.00

DISCUSSION

The present study demonstrates that both topical steroid regimens were initiated in two well-matched pediatric cohorts, and that proactive maintenance with intermittent steroid (Regimen B) provided superior disease control without compromising safety. The mean age of our participants (7.82 ± 3.41 vs. 8.05 ± 3.27 years in Regimen A and B, respectively) and the slight male predominance (57.89% vs. 55.26%) are consistent with the known epidemiology of pediatric atopic dermatitis, where onset typically occurs in early childhood and prevalence in school-aged children may reach 15–20%. Atherton et al. (2003) similarly reported that AD is common in children and often begins in early life, emphasizing the need for safe long-term topical steroid strategies in this age group.[8]

Baseline disease severity in our cohort was moderate, as reflected by mean SCORAD values of 42.36 ± 8.12 and 41.89 ± 7.95 and EASI scores of 6.74 ± 2.21 and 6.59 ± 2.17 in Regimens A and B, respectively. These figures compare well with the moderate–severe childhood AD population studied by Kirkup et al., who evaluated fluticasone propionate 0.05% cream versus hydrocortisone 1% and hydrocortisone butyrate 0.1% in children aged 2–14 years and showed significantly lower Total AD Scores with fluticasone both in the acute phase (difference –2.39 vs. hydrocortisone; $p < 0.001$) and maintenance phase (difference –1.88; $p = 0.006$).[9]

With regard to treatment response, Regimen B (proactive maintenance) produced greater improvements across all objective and subjective severity indices than Regimen A (reactive use only). In our study, SCORAD reduction was 25.47 ± 7.12 in Regimen B versus 18.84 ± 6.33 in Regimen A, and EASI reduction was 4.29 ± 1.21 versus 3.12 ± 1.08, respectively. This pattern of superior disease control with intermittent maintenance corticosteroid is in line with the proactive strategy proposed by Hanifin et al. (2002), who showed in an intermittent fluticasone cream regimen that applying steroid to previously affected areas even when clinically quiescent significantly reduced the risk of relapse compared with vehicle.[10]

The magnitude of benefit seen with our proactive regimen—particularly in terms of BSA and symptom relief—parallels the large flare-prevention effect reported in the landmark study by Berth-Jones et al. (2003). In our cohort, BSA involvement decreased by 10.68 ± 3.02% in Regimen B versus 7.95 ± 2.44% in Regimen A, and pruritus VAS fell by 4.16 ± 1.07 compared with 3.24 ± 1.01. Berth-Jones et al. evaluated twice-weekly fluticasone propionate added to emollients in patients with moderate-to-severe AD and found that, during a 16-week maintenance phase, disease remained controlled in 87 patients on fluticasone versus 46 on emollient alone, with median time to relapse exceeding 16 weeks versus just 6 weeks in the control arm.[11]

Investigator's Global Assessment (IGA) outcomes in our study likewise favor the proactive approach. In Regimen B, 28.95% of children were rated "clear" and 39.47% "almost clear," compared with 13.16% and 26.32% in Regimen A; conversely, moderate disease persisted in 26.32% of Regimen A versus only 7.89% of Regimen B. These clearance rates compare favorably with the clinical healing observed by Wolkerstorfer et al., who reported that in a small cohort of 22 children (3–8 years) treated with fluticasone propionate 0.05% once daily or clobetasone butyrate 0.05% twice daily, 16 of 21 completers (≈76%) achieved a SCORAD < 9 (clinically healed) by week 4.[12]

Symptom and quality-of-life (QoL) outcomes in our study highlight the clinical relevance of proactive control. Regimen B produced greater reductions in pruritus (4.16 ± 1.07 vs. 3.24 ± 1.01), sleep disturbance (2.98 ± 0.92 vs. 2.15 ± 0.87), and CDLQI scores (7.11 ± 2.08 vs. 5.42 ± 1.89) than Regimen A. Ben-Gashir et al. (2004) investigated the relationship between SCORAD and CDLQI in 78 children with AD and found that QoL was impaired in 92% at baseline and 77% at follow-up; SCORAD and CDLQI were significantly correlated ($r = 0.52$ and 0.59; $p < 0.001$ at both visits), with each unit change in SCORAD associated with a 0.12-point change in CDLQI.[13]

The safety profile observed in our study was favorable and comparable between the two regimens, despite more prolonged steroid exposure in the proactive arm. Any adverse event occurred in 18.42% of children in both Regimen A and B, with very low frequencies of skin atrophy (2.63% vs. 0.00%), hypopigmentation (5.26% vs. 2.63%), and folliculitis (2.63% vs. 5.26%), and no telangiectasia in either group. These findings are in keeping with the phase IV safety study by Friedlander et al. (2002), who treated 43 young children (3 months to <6 years) with extensive AD (>35% BSA; mean treated BSA 64%) using fluticasone propionate 0.05% cream twice daily for 3–4 weeks and reported no clinically relevant adrenal suppression (post-stimulation cortisol remained around 28–30 µg/dL) and no significant cutaneous atrophy.[14]

Our results also align with the broader evidence base supporting "weekend" or proactive therapy as an effective flare-prevention strategy. In our study, proactive Regimen B not only yielded larger numerical improvements in SCORAD, EASI, BSA,

and symptom VAS scores but also shifted more children into “clear” or “almost clear” IGA categories, while markedly reducing the proportion with residual moderate disease. Schmitt et al. (2011) synthesized eight vehicle-controlled randomized trials of proactive therapy and showed that topical fluticasone propionate reduced the risk of at least one flare with a pooled relative risk of 0.46 (95% CI 0.38–0.55) compared with vehicle, indicating more than a 50% reduction in flare risk.[15]

CONCLUSION

In this randomized comparative study, both topical steroid regimens were effective in managing mild-to-moderate pediatric atopic dermatitis, but the proactive maintenance regimen (Regimen B) achieved greater reductions in SCORAD, EASI, BSA involvement, pruritus, sleep disturbance, and CDLQI scores than the reactive regimen (Regimen A). Regimen B also resulted in a higher proportion of children attaining clear or almost clear IGA status and a significantly lower proportion with residual moderate disease. Importantly, the overall frequency of adverse effects was low and comparable between the two groups, indicating that proactive intermittent use of a mid-potency topical corticosteroid is both effective and well tolerated. These findings support incorporating proactive steroid-based maintenance therapy, alongside regular emollient use, into long-term management strategies for pediatric atopic dermatitis in tertiary care settings.

REFERENCES

1. Odhiambo JA, Williams HC, Clayton TO, Robertson CF, Asher MI; ISAAC Phase Three Study Group. Global variations in prevalence of eczema symptoms in children from ISAAC Phase Three. *J Allergy Clin Immunol*. 2009;124(6):1251–1258.e23. Available from: https://pubmed.ncbi.nlm.nih.gov/20004783/PubMed+1
2. Bieber T. Atopic dermatitis. *N Engl J Med*. 2008;358(14):1483–1494. Available from: https://www.nejm.org/doi/full/10.1056/NEJMra074081 PubMed
3. Cork MJ, Robinson DA, Vasilopoulos Y, Ferguson A, Moustafa M, MacGowan A, et al. New perspectives on epidermal barrier dysfunction in atopic dermatitis: gene–environment interactions. *J Allergy Clin Immunol*. 2006;118(1):3–21. Available from: https://pubmed.ncbi.nlm.nih.gov/16815133/PubMed+1
4. Hanifin JM, Rajka G. Diagnostic features of atopic dermatitis. *Acta Derm VenereolSuppl (Stockh)*. 1980;60(Suppl 92):44–47. Available from: https://medicaljournalssweden.se/actadv/article/view/10725medicaljournalssweden.se+1
5. Hoare C, Li Wan Po A, Williams H. Systematic review of treatments for atopic eczema. *Health Technol Assess*. 2000;4(37):1–191. Available from: https://pubmed.ncbi.nlm.nih.gov/11134919/PubMed+1
6. Charman CR, Morris AD, Williams HC. Topical corticosteroid phobia in patients with atopic eczema. *Br J Dermatol*. 2000;142(5):931–936. Available from: https://pubmed.ncbi.nlm.nih.gov/10809850/PubMed+1
7. Eichenfield LF, Miller BH. Two randomized, double-blind, placebo-controlled studies of fluticasone propionate lotion 0.05% for the treatment of atopic dermatitis in subjects from 3 months of age. *J Am Acad Dermatol*. 2006;54(4):715–717. Available from: https://pubmed.ncbi.nlm.nih.gov/16546598/PubMed+1
8. Atherton DJ. Topical corticosteroids in atopic dermatitis. *BMJ*. 2003;327(7421):942–943. Available from: BMJ
9. Kirkup ME, Birchall NM, Weinberg EG, Helm K, Kennedy CTC. Acute and maintenance treatment of atopic dermatitis in children – two comparative studies with fluticasone propionate (0.05%) cream. *J Dermatolog Treat*. 2003;14(3):141–148. doi:10.1080/09546630310013388. Available from: PubMed
10. Hanifin JM, Gupta AK, Rajagopalan R. Intermittent dosing of fluticasone propionate cream for reducing the risk of relapse in atopic dermatitis patients. *Br J Dermatol*. 2002;147(3):528–537. Available from: PubMed
11. Berth-Jones J, Damstra RJ, Golsch S, Livden JK, Van Hooteghem O, Allegra F, et al. Twice weekly fluticasone propionate added to emollient maintenance treatment to reduce risk of relapse in atopic dermatitis: randomised, double blind, parallel group study. *BMJ*. 2003;326(7403):1367–1370. doi:10.1136/bmj.326.7403.1367. Available from: PubMed
12. Wolkerstorfer A, Strobos MA, Glazenburg EJ, Mulder PG, Oranje AP. Fluticasone propionate 0.05% cream once daily versus clobetasone butyrate 0.05% cream twice daily in children with atopic dermatitis. *J Am Acad Dermatol*. 1998;39(2 Pt 1):226–231. doi:10.1016/S0190-9622(98)70080-5. Available from: PubMed
13. Ben-Gashir MA, Seed PT, Hay RJ. Quality of life and disease severity are correlated in children with atopic dermatitis. *Br J Dermatol*. 2004;150(2):284–290. doi:10.1111/j.1365-2133.2004.05776.x. Available from: PubMed
14. Friedlander SF, Hebert AA, Allen DB; Fluticasone Pediatrics Safety Study Group. Safety of fluticasone propionate cream 0.05% for the treatment of severe and extensive atopic dermatitis in children as young as 3 months. *J Am Acad Dermatol*. 2002;46(3):387–393. doi:10.1067/mjd.2002.118337. Available from: PubMed
15. Schmitt J, von Kobyletzki L, Svensson A, Apfelbacher C. Efficacy and tolerability of proactive treatment with topical corticosteroids and calcineurin inhibitors for atopic eczema: systematic review and meta-analysis of randomized controlled trials. *Br J Dermatol*. 2011;164(2):415–428. doi:10.1111/j.1365-2133.2010.10030.x. Available from: PubMed