Ann Dermatol. 2025 Aug;37(4):201-215
https://doi.org/10.5021/ad.24.153
pISSN 1013-9087·eISSN 2005-3894

AD ANNALS of DERMATOLOGY

Original Article

Consensus-Based Guidelines for the Treatment of Atopic Dermatitis in Korea (Part I): Basic Therapy, Topical Therapy, and Conventional Systemic Therapy

Hyun-Chang Ko [1,*], Yu Ri Woo [2,*], Joo Yeon Ko [3], Hye One Kim [4], Chan Ho Na [5], Youin Bae [6], Young-Joon Seo [7], Min Kyung Shin [8], Jiyoung Ahn [9], Bark-Lynn Lew [8], Dong Hun Lee [10], Sang Eun Lee [11], Sul Hee Lee [12], Yang Won Lee [13], Ji Hyun Lee [14], Yong Hyun Jang [15], Jiehyun Jeon [16], Sun Young Choi [17], Ju Hee Han [14], Tae Young Han [18], Sang Wook Son [19], Sang Hyun Cho [2]

[1]Department of Dermatology, Pusan National University School of Medicine, Busan, Korea
[2]Department of Dermatology, Incheon St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Seoul, Korea
[3]Department of Dermatology, Hanyang University College of Medicine, Seoul, Korea
[4]Department of Dermatology, Hallym University Kangnam Sacred Heart Hospital, Hallym University College of Medicine, Seoul, Korea
[5]Department of Dermatology, Chosun University Medical School, Gwangju, Korea
[6]Department of Dermatology, Soonchunhyang University Seoul Hospital, Seoul, Korea
[7]Department of Dermatology, Chungnam National University College of Medicine, Daejeon, Korea
[8]Department of Dermatology, Kyung Hee University College of Medicine, Seoul, Korea
[9]Department of Dermatology, National Medical Center, Seoul, Korea
[10]Department of Dermatology, Seoul National University Hospital, Seoul National University College of Medicine, Seoul, Korea
[11]Department of Dermatology, Gangnam Severance Hospital, Cutaneous Biology Research Institute, Yonsei University College of Medicine, Seoul, Korea
[12]Department of Dermatology, Soonchunhyang University Bucheon Hospital, Bucheon, Korea
[13]Department of Dermatology, Konkuk University School of Medicine, Seoul, Korea
[14]Department of Dermatology, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Seoul, Korea
[15]Department of Dermatology, Kyungpook National University Hospital, School of Medicine, Kyungpook National University, Daegu, Korea
[16]Department of Dermatology, Korea University Guro Hospital, Korea University College of Medicine, Seoul, Korea
[17]Department of Dermatology, Chung-Ang University Gwangmyeong Hospital, Chung-Ang University College of Medicine, Gwangmyeong, Korea
[18]Department of Dermatology, Nowon Eulji Medical Center, Eulji University, Seoul, Korea
[19]Department of Dermatology, Korea University Ansan Hospital, Korea University College of Medicine, Seoul, Korea

Received: Jan 8, 2025
Revised: Apr 29, 2025
Accepted: May 14, 2025
Published online: Jun 11, 2025

Corresponding Authors:
Sang Hyun Cho
Department of Dermatology, Incheon St. Mary's Hospital, College of Medicine, The Catholic University of Korea, 56 Dongsu-ro, Bupyeong-gu, Incheon 21431, Korea.
Email: drchos@yahoo.co.kr

Sang Wook Son
Department of Dermatology, Korea University Ansan Hospital, Korea University College of Medicine, 123 Jeokgeum-ro, Danwon-gu, Ansan 15355, Korea.
Email: skin4u@korea.ac.kr

*Hyun-Chang Ko and Yu Ri Woo contributed equally to the work as first authors.

ABSTRACT

Background: Atopic dermatitis (AD) is a common skin disease with a wide range of symptoms. Due to the rapidly changing treatment landscape, regular updates to clinical guidelines are needed.

Objective: This study aimed to update the guidelines for the treatment of AD to reflect recent therapeutic advances and evidence-based practices.

AD ANNALS of DERMATOLOGY

Methods: The Patient characteristics, type of Intervention, Control, and Outcome framework was used to determine 48 questions related to AD management. Evidence was graded, recommendations were determined, and, after 2 voting rounds among the Korean Atopic Dermatitis Association (KADA) council members, consensus was achieved.
Results: The guidelines provide detailed recommendations on foundational therapies, including the use of moisturizers, cleansing and bathing practices, allergen avoidance, and patient education. Guidance on topical therapies, such as topical corticosteroids and calcineurin inhibitors, is also provided to help manage inflammation and maintain skin barrier function in patients with AD. Additionally, recommendations on conventional systemic therapies, including corticosteroids, cyclosporine, and methotrexate, are provided for managing moderate to severe AD.
Conclusion: KADA's updated AD guidelines offer clinicians evidence-based strategies focused on basic therapies, topical therapies, and conventional systemic therapies, equipping them to enhance quality of care and improve patient outcomes in AD management.

Keywords: Atopic dermatitis; Consensus; Guidelines; Republic of Korea; Treatment

INTRODUCTION

Atopic dermatitis (AD) is a common, chronic, relapsing, inflammatory skin disorder driven by immunological, genetic, and environmental factors, along with skin barrier dysfunction[1]. In South Korea, AD affects 10%–20% of children and 3%–4% of adults[2,3]. Asian AD patients exhibit distinct clinical and immunological features, including sharply demarcated lesions and lichenification, reflecting a greater epidermal inflammatory response[4]. Higher immunoglobulin E and eosinophil levels, along with stronger T helper (Th)2/Th17/Th22 cytokine skewing, further differentiate the Asian AD phenotype from the European/American subtype[4,5]. Its varied clinical presentations and complex etiology pose challenges for standardized treatment[6,7].

To address these challenges, the Korean Atopic Dermatitis Association (KADA) has updated its consensus guidelines, incorporating the latest evidence on basic, topical, and systemic therapies, including newly approved biologics and Janus kinase (JAK) inhibitors. Divided into 2 parts, this guideline provides practical, evidence-based recommendations to optimize AD management, with Part I focusing on basic therapies, topical treatments, and conventional systemic therapies.

MATERIALS AND METHODS

The KADA task force team, composed of 10 dermatologists, conducted a comprehensive, up-to-date literature reviews on AD management. Following a thorough literature review, the task force team developed 48 Patient characteristics, type of Intervention, Control, and Outcome (PICO)-based questions for AD management and sought expert opinions (**Table 1**)[8].

Database and literature searches

The task force team conducted a comprehensive search of various databases, including PubMed, Scopus, the Cochrane Library, and KoreaMed. This search included articles published up to December 31, 2023. The search queries included a combination of keywords: "atopic eczema," "atopic dermatitis," "moisturizer," "cleansing," "bathing," "education," "allergen," "avoidance," "wet wrap therapy," "topical," "topical agents," "topical corticosteroids," "topical calcineurin inhibitor," "pimecrolimus," "tacrolimus," "topical phosphodiesterase 4 inhibitors," "crisaborole," "topical JAK inhibitors," "ruxolitinib," "topical anesthetics," "topical capsaicin," "topical doxepin," "corticosteroids," "cyclosporine," "azathioprine," "methotrexate," "mycophenolate mofetil," and "alitretinoin." In addition to database searches, the team also conducted manual searches by reviewing the reference lists of relevant systematic reviews and guidelines issued by other research groups.

Evaluation of the literature

The quality of the evidence was evaluated and then the strength of the recommendation for each PICO statement was determined. The evidence for each statement was assessed using the following grading system: level 1a, systematic review (with homogeneity) of randomized controlled trials (RCTs); level 1b, individual RCT (with narrow confidence interval); level 1c, all or none; level 2a, systematic review (with homogeneity) of cohort studies; level 2b, individual cohort study (including low-quality RCTs); level 2c, "outcome" research; level 3a, systematic review

https://doi.org/10.5021/ad.24.153

Table 1. Expert consensus recommendations for the treatment of AD

PICO	Recommendation strength	Grade of evidence	Mean agreement score	% of respondents
Basic therapies				
We recommend the use of moisturizers to improve AD symptoms and prevent acute exacerbations.	A	1a	9.56	98
We suggest the avoidance of house dust mite in house dust mite-sensitized AD patients with a history of apparent skin exacerbation associated with house dust mite exposure.	B	2b	8.46	94
We recommend a structured educational program with a multidisciplinary medical team approach for the effective management of AD.	A	1b	9.28	94
We suggest conducting patient education using well-structured educational materials for effective patient management in AD.	B	2b	8.60	92
Topical therapies				
We recommend the use of topical corticosteroids in AD to control AD symptoms.	A	1a	9.40	100
We recommend selecting and using an appropriate strength of topical corticosteroids for AD patients based on severity, treatment area, and age.	A	1a	9.78	100
We suggest considering wet wrap therapy with diluted topical corticosteroids to achieve rapid improvement for acute exacerbated lesion of AD.	B	1b	8.32	92
We recommend the use of topical calcineurin inhibitors in AD to control AD symptoms.	A	1a	9.42	96
We suggest using an initial application of topical corticosteroid before switching to topical calcineurin inhibitors to reduce local skin reactions like burning sensation.	B	5	7.92	72
We recommend the use of topical calcineurin inhibitors for sensitive areas such as the face, intertriginous, and genital regions.	A	1b	9.44	98
We recommend proactive therapy with a moderate potency topical corticosteroid or topical calcineurin inhibitors 2 to 3 times a week to prevent flares or relapses in the improved areas.	A	1a	8.98	94
Systemic therapies				
We recommend the use of cyclosporine for patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	A	1a	9.36	98
We recommend regular blood pressure monitoring and laboratory testing to identify adverse effects of cyclosporine in patients with AD.	A	1a	9.58	98
We suggest the selective use of methotrexate for patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	B	1b	8.04	84
We suggest the selective use of azathioprine for patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	B	1b	6.54	58
We propose the limited use of mycophenolate mofetil for patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	C	4	6.20	62
Long-term use of systemic corticosteroid is not recommended due to potential for side effects.	D	5	9.46	96
We propose the limited use of alitretinoin to improve hand eczema in patients with AD.	C	4	7.22	70
Biologics				
We recommend the use of dupilumab in adult, adolescent, and pediatric patients over 6 months of age with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	A	1a	9.54	98
We recommend the use of tralokinumab in adult and adolescent patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	A	1a	9.32	97
We recommend the use of lebrikizumab in adult and adolescent patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	A	1b	9.21	97
We suggest considering the use of nemolizumab in patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	B	1b	7.92	86
We propose the limited use of omalizumab in patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	C	3b	5.32	34
We propose considering the selective addition of systemic immunosuppressants or oral JAK inhibitors in patients with moderate to severe AD who are not adequately controlled by biologic agents.	C	4	7.80	74
We suggest considering switching to another biologic or oral JAK inhibitor in patients with moderate to severe AD if there is an insufficient response* to biologic therapy or an inability to use current biologic treatment due to side effects.	B	Jak inhibitors 1b/ Biologics 4	9.32	96
We suggest considering selective dosing intervals according to the patient's symptoms in patients with AD on biologics.	B	Tralokinumab 1b/ Lebrikizumab 1b/ Dupilumab 4	8.50	90
JAK inhibitors				
We recommend the use of baricitinib, an oral JAK 1/2 inhibitor, in adult patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	A	1a	8.80	94

(continued to the next page)

Table 1. (Continued) Expert consensus recommendations for the treatment of AD

PICO	Recommendation strength	Grade of evidence	Mean agreement score	% of respondents
We recommend the use of upadacitinib, an oral JAK1 inhibitor, in adult and adolescent patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	A	Adults 1a/ Adolescents 1b	9.24	96
We recommend the use of abrocitinib, an oral JAK1 inhibitor, in adult and adolescent patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	A	Adults 1a/ Adolescents 1b	8.84	94
We recommend periodic monitoring and careful consideration of the benefit-risk ratio when using oral JAK inhibitors for maintenance therapy in patients with moderate to severe AD.	A	1b	9.22	96
We suggest considering switching to another biologic or oral JAK inhibitor in patients with moderate to severe AD if there is an insufficient response* to an oral JAK inhibitor or an inability to use current oral JAK inhibitor due to side effects.	B	4	8.88	92
We suggest considering selective dose adjustments according to the patient's symptoms in patients with AD on oral JAK inhibitors.	B	5	8.50	90
Other therapies				
We suggest considering the selective use of phototherapy for patients with moderate to severe AD.	B	2b	8.18	92
We suggest selective consideration of the additional use of oral H1 antihistamines in patients with AD.	B	2b	8.60	94
We recommend a short-term use of topical or systemic antibiotics in patients with AD with clinically apparent bacterial infections.	A	1a	9.34	98
We suggest considering the selective use of oral antifungal agents for symptomatic relief in AD patients with head and neck dermatitis uncontrolled by conventional therapy.	B	2b	7.20	72
We suggest considering the selective use of allergen-specific immunotherapy for symptomatic relief in patients with moderate to severe AD sensitized to inhaled antigens.	B	1b	7.14	66
We propose the limited use of probiotics/prebiotics for symptomatic relief in patients with AD.	C	2b	6.68	52
We propose the limited use of evening primrose oil for symptomatic relief in patients with AD.	C	2b	7.52	80
We propose the limited use of vitamin D for symptomatic relief in patients with AD.	C	2b	6.76	56
Special consideration for children and adolescents				
We suggest considering the selective use of short-term systemic corticosteroids for acute exacerbation in children and adolescent patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	B	2b	8.06	90
We suggest considering the selective use of systemic immunosuppressive agents for children and adolescent patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	B	2b	8.72	98
We recommend the use of biologics for children and adolescent patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies.	A	1b	9.18	96
Special consideration for elderly patients				
We suggest the treatment for AD be considered in elderly patients with persistent chronic pruritus with skin lesions if other causes of pruritus have been ruled out by a dermatologist.	B	5	7.78	80
We suggest considering the selective use of biologics and low-dose JAK inhibitors in elderly patients with AD with refractory to conventional topical therapy, taking into account the benefit-risk ratio and comorbidities.	B	5	7.90	82
We suggest considering the selective use of topical calcineurin inhibitors for elderly patients with AD when prolonged use of topical steroids is necessary.	B	5	8.62	92
Special consideration for pregnant and breastfeeding women				
We suggest considering the use of topical corticosteroids during pregnancy or lactation in patients with AD.	B	2b	8.20	90
We propose the limited use of oral cyclosporine during pregnancy in patients with AD based on benefit-risk ratio.	C	4	6.56	76

AD: atopic dermatitis, EASI: Eczema Area and Severity Index, JAK: Janus kinase, PICO: Patient characteristics, type of Intervention, Control, and Outcome.
*Insufficient response is defined as a patient with AD who fails to achieve EASI-50 or who meets one or more of the following criteria after 3 months of appropriate treatment: daytime or nighttime itch with itch numeric rating scale score ≥4, or Dermatology Life Quality Index ≥6 according to a previous Korean expert consensus[8].

(with homogeneity) of case–control studies; level 3b, individual case–control study; level 4, case series (and poor-quality cohort and case-control studies); and level 5, expert opinion.

We applied the modified Grading of Recommendations Assessment, Development and Evaluation system for determining the strength of recommendations. In addition to the level of evidence and the balance of benefits and harms, we comprehensively considered various important factors such as the feasibility of implementing the recommendations in primary care settings, acceptability, and level of utilization. The strength of the recommendation was classified as follows: A, strong recommendation for using an intervention. The benefits of this intervention significantly

outweigh potential harms (generally recommended); B, weak recommendation for using an intervention. The benefits of this intervention outweigh potential harms, but there is some uncertainty (recommended selectively); C, weak recommendation against using an intervention. The harms of this intervention outweigh benefits, but there is some uncertainty (not recommended unless there are specific considerations); D, strong recommendation against using an intervention. The harms of this intervention significantly outweigh benefits (generally not recommended).

Consensus process

A total of 56 council members of the KADA were asked to provide their level of agreement with each draft statement. A voting scale ranging from 1 to 10 was used, where 1 indicated strong disagreement, and 10 indicated strong agreement. The voting scores were categorized into one of 3 groups: 1 to 3 (indicating disagreement), 4 to 6 (indicating neutrality), and 7 to 10 (indicating agreement). In this context, consensus was defined as a situation where at least 70% of the participants provided scores falling within the 7 to 10 range, indicating agreement. The process of arriving at consensus recommendations involved 2 rounds of voting.

RESULTS

Basic therapies

1) Moisturizer

> We recommend the use of moisturizers to improve AD symptoms and prevent acute exacerbations (Recommendation strength: A, Grade of evidence: 1a, % of respondents [agreement score ≥7]: 98%).

Moisturizers are defined as externally applied compounds made up of multiple components, that aim to maintain skin integrity and appearance[9]. They restore skin barrier function, reduce skin sensitivity to irritants, and alleviate symptoms[10,11]. RCTs have demonstrated that consistent use of moisturizers reduces the frequency of flare-ups, prolongs remission periods, and decreases reliance on corticosteroids[12,17]. Despite these benefits, moisturizers are not intended for use on acute inflammatory lesions and should be combined with topical or systemic anti-inflammatory therapies as needed, depending on disease severity[10,18].

The moisturizers containing physiologic lipid mixture can alleviate the clinical severity and symptoms of AD, and restore skin hydration[19,20]. Ceramide-dominant formulations with an optimized 3:1:1 ratio of ceramides, cholesterol, and fatty acids are particularly effective in repairing the epidermal barrier[20]. Additionally, patients should select fragrance-free and hypoallergenic options to minimize the risk of irritation.

There is limited evidence for the optimal frequency and amount of moisturizer in patients with AD. A liberal and frequent use of at least 250 g of moisturizer per week is generally recommended for adults[21-23]. Additionally, the fingertip unit (FTU) is a practical measure for topicals, representing the amount dispensed from a 5-mm nozzle along the length of an adult's distal phalanx of the index finger[24]. One FTU (about 0.5 g) covers an area equivalent to 2 adult palms or 2% of the body surface area[22,24]. Several AD consensus guidelines suggest the FTU to estimate the optimal amount of moisturizer[25,26]. Regarding the optimal frequency of moisturizer applications, it is generally suggested to apply moisturizer at least twice daily to maintain skin moisture[27-31].

As for the preventive measures, the moisturizer can be applied as either a secondary or tertiary preventative strategy for AD. However, additional confirmation is needed regarding the role of moisturizers in infancy as a primary prevention for AD. Systematic reviews show conflicting results regarding the role of moisturizers in preventing AD onset, with some studies highlighting potential benefits for high-risk infants[32], while others report no significant effects[33,34].

2) Cleansing and bathing

Bathing is essential in AD management, supporting hydration, removing irritants, and improving treatment absorption[35]. General recommendations include daily bathing with warm or lukewarm water (27°C–30°C) for 5–10 minutes and avoiding vigorous scrubbing. Non-soap cleansers with neutral to acidic pH are preferred over alkaline soaps to preserve the skin barrier[36]. Further details are provided in the supplementary materials (**Supplementary Data 1**).

3) Avoidance of allergens

> We suggest the avoidance of house dust mite in house dust mite-sensitized AD patients with a history of apparent skin exacerbation associated with house dust mite exposure (Recommendation strength: B, Grade of evidence: 2b, % of respondents [agreement score ≥7]: 94%).

Patients with AD exhibit an increased prevalence of allergic sensitization[37,38], with house dust mites (HDMs) being key antigens that impair skin barrier[39-42] and activate protease-activated receptor-2, leading to non-histaminergic itch[43]. Although HDM avoidance shows mixed effectiveness in preventing flares[44-48], we suggest avoiding HDM exposure in HDM-sensitized AD patients who have a history of apparent skin exacerbation associated with HDM exposure. HDM avoidance strategies include controlling indoor humidity, washing bedding in hot water (55°C–60°C), using high efficiency particulate air (HEPA) filters or filter bags, and reducing the use of fabric furnishings, but they are not recommended for

primary prevention of AD[49].

Exposure to furred animals in sensitized patients can exacerbate AD symptoms. As a result, it is generally recommended to avoid furred animals when an allergy is confirmed[25,50]. However, exposure to furred animals during pregnancy or early childhood may lower the risk of AD development. A recent birth cohort suggested that pet ownership during infancy is inversely related to a higher risk of AD at 5 years old[51], and maternal pet ownership during pregnancy was associated with a decrease in the severity of a child's AD[52]. A meta-analysis found a 25% reduced AD risk in children exposed to dogs or cats during pregnancy, infancy, or childhood[53]. Thus, avoiding furred animals is not recommended solely for AD prevention.

Food allergies are common in children with AD[54] and linked to more severe AD[38]. A Cochrane review indicated that for those sensitized to hen's eggs, eliminating them from the diet can be beneficial in controlling AD[55]. Strict elimination diets are recommended only for specialist-diagnosed allergies, while general avoidance diets (e.g., gluten, hen's egg, cow's milk) are not recommended for patients with AD unless they have been specifically diagnosed with those food allergies[55].

4) Educational program

We recommend a structured educational program with a multidisciplinary medical team approach for the effective management of AD (Recommendation strength: A, Grade of evidence: 1b, % of respondents [agreement score ≥7]: 94%).

AD is a chronic, relapsing disease that affects all ages and often impairs quality of life[56]. Effective long-term management necessitates adherence to a multifaceted treatment approach[57]. However, misconceptions such as steroid phobia, unwarranted dietary restrictions, and reliance on unproven treatments hinder adherence, worsening disease control and increasing treatment costs[58-60]. Comprehensive patient education is key to improving treatment adherence, enhancing quality of life, and reducing the economic burden of AD.

Structured educational programs reduce disease severity and improve clinical and psychological outcomes[61-63]. Multidisciplinary approaches involving dermatologists, psychologists, and nurses have shown particular efficacy in improving disease management[63-66]. Effective educational methods include multidisciplinary medical team approach, group-based programs, family engagement, specialized nurse-led group sessions, online videos, hospital stays, leaflets, and online web[61,64,67-73]. Among these, the efficacy of a multidisciplinary team approach involving dermatologists, psychologists, nurses, and dieticians have been validated in numerous studies[63-66].

We suggest conducting patient education using well-structured educational materials for effective patient management in AD (Recommendation strength: B, Grade of evidence: 2b, % of respondents [agreement score ≥7]: 92%).

Effective patient education can be delivered through tools like written eczema action plans, leaflets, e-health programs, and online videos, depending on the national healthcare system. Written eczema action plans, in particular, have proven effective in improving treatment adherence and patient understanding[74-78]. These plans provide clear instructions for managing chronic conditions between visits, leading to reduced healthcare visits[74,75], decreased suffering, lower economic burdens, and improved clinical outcomes[77].

In addition, shared decision-making is a key aspect of patient-centered care, enabling clinicians and patients to collaborate on treatment decisions based on individual preferences, values, and lifestyles[65,79]. It enhances adherence and patient satisfaction by providing personalized treatment options and encouraging active involvement in disease management.

To ensure a comprehensive evaluation of treatment effects and facilitate personalized care, incorporating quality of life assessment tools is essential[80,81]. Tools such as Dermatology Life Quality Index, Children's Dermatology Life Quality Index, Patient-Oriented Eczema Measure, and Atopic Dermatitis Control Tool, help assess daily functioning, emotional well-being, and social impact, allowing clinicians to tailor treatments to individual patient needs[80-82].

Topical therapies

1) Topical corticosteroid (TCS)

We recommend the use of topical corticosteroid in AD to control AD symptoms (Recommendation strength: A, Grade of evidence: 1a, % of respondents [agreement score ≥7]: 100%).

TCSs are commonly used as an anti-inflammatory treatment for AD, especially during acute phase. The corticosteroids have a dual action of decreasing the synthesis of pro-inflammatory cytokines and increasing the synthesis of anti-inflammatory mediators. Numerous RCTs confirm their efficacy in managing acute flares, chronic AD, pruritus, and preventing relapses[83-86].

We recommend selecting and using an appropriate strength of topical corticosteroid for AD patients based on severity, treatment area, and age (Recommendation strength: A, Grade of evidence: 1a, % of respondents [agreement score ≥7]: 100%).

The choice of TCS strength depends on severity, anatomical site, and patient age. TCSs are classified into seven potency classes (**Table 2**)[87]. TCSs are typically applied once or twice daily[88-91], with twice-daily application recommended for acute flares. Early, intensive treatment during flare-ups reduces TCS use and minimizes adverse effects[23]. Tapering TCSs use as symptoms resolve, by reducing frequency or switching to a lower potency, helps avoid rebound phenomenon. For recurrent flares, proactive treatment is recommended. The FTU method can guide the appropriate amount of TCS application.

Side effects of TCSs include skin atrophy, telangiectasia, spontaneous scars, ecchymosis, striae cutis, hypertrichosis, rosacea-like perioral dermatitis, depigmentation, delayed wound healing, contact dermatitis, steroid acne, and skin infection, particularly with prolonged use of potent TCSs on absorbent areas like the face, neck, and intertriginous zones or use in the elderly[92,93]. They can be resolved with discontinuation or adequate treatment[94,95]. To reduce the risk of local side effects, the selection of TCS should consider potency, formulation, patient age, application site, and treatment duration.

Prolonged use of potent TCSs on the eyelid or periorbital region may increase the risk of cataracts or glaucoma, though AD itself is a cataract risk due to eye rubbing[96-98]. Maintenance treatment with topical calcineurin inhibitors (TCIs) is recommended for these areas to avoid long-term TCS use.

Systemic absorption of potent TCSs is rare but can occur with reduced skin barrier conditions, such as erythroderma, or excessive use in children, potentially causing hypothalamic-pituitary-adrenal axis suppression[93,98]. Steroid phobia is common and negatively impacts treatment adherence. Addressing these fears through education and regular monitoring during long-term TCS use is essential to improve outcomes[99401].

We suggest considering wet wrap therapy with diluted topical corticosteroid to achieve rapid improvement for acute exacerbated lesion of AD (Recommendation strength: B, Grade of evidence: 1b, % of respondents [agreement score ≥7]: 92%).

Table 2. Topical corticosteroids according to potency

Class	Drug	Dosage form	Strength (%)
I. Very high potency	Augmented betamethasone dipropionate	Ointment	0.05
	Clobetasol propionate	Cream, foam, ointment	0.05
	Diflorasone diacetate	Ointment	0.05
	Diflucortolone valerate	Ointment, lotion	0.3
	Halobetasol propionate	Cream, ointment	0.05
II. High potency	Amcinonide	Cream, lotion, ointment	0.1
	Augmented betamethasone dipropionate	Cream	0.05
	Betamethasone dipropionate	Cream, foam, ointment, solution	0.05
	Desoximetasone	Cream, ointment	0.25
	Desoximetasone	Gel	0.05
	Diflorasone diacetate	Cream	0.05
	Fluocinonide	Cream, gel, ointment, solution	0.05
	Halcinonide	Cream, ointment	0.1
	Mometasone furoate	Ointment	0.1
	Triamcinolone acetonide	Cream, ointment	0.5
III–IV. Medium potency	Betamethasone valerate	Cream, foam, lotion, ointment	0.1
	Clocortolone pivalate	Cream	0.1
	Desoximetasone	Cream	0.05
	Fluocinolone acetonide	Cream, ointment	0.025
	Flurandrenolide	Cream, ointment	0.05
	Fluticasone propionate	Cream	0.05
	Fluticasone propionate	Ointment	0.005
	Methylprednisolone aceponate	Ointment, cream, lotion	0.1
	Mometasone furoate	Cream	0.1
	Triamcinolone acetonide	Cream, ointment	0.1
V. Lower-medium potency	Hydrocortisone butyrate	Cream, ointment, solution	0.1
	Hydrocortisone probutate	Cream	0.1
	Hydrocortisone valerate	Cream, ointment	0.2
	Prednicarbate	Cream	0.1
VI. Low potency	Alclometasone dipropionate	Cream, ointment	0.05
	Desonide	Cream, gel, foam, ointment	0.05
	Fluocinolone acetonide	Cream, solution	0.01
VII. Lowest potency	Dexamethasone	Cream	0.1
	Hydrocortisone	Cream, lotion, ointment, solution	0.25, 0.5, 1
	Hydrocortisone acetate	Cream, ointment	0.5–1

https://doi.org/10.5021/ad.24.153

Wet wrap therapy (WWT) is effective for acute AD flares and recalcitrant disease. Typically, a low or mild potency TCS (or emollient) is applied to the skin lesion and wrapped around it. A wet wrap bandage consists of an inner layer of moist cotton, gauze or bandage, covered by a dry outer bandage. WWT can be used from a few hours to a day at a time. WWT rehydrates, cools the skin, reduces inflammation, relieves itching, and helps break the itch-scratch cycle. For acute exacerbations, WWT with diluted TCSs can provide rapid symptomatic relief.

2) TCIs

We recommend the use of topical calcineurin inhibitors in AD to control AD symptoms (Recommendation strength: A, Grade of evidence: 1a, % of respondents [agreement score ≥7]: 96%).

TCIs are safe and effective anti-inflammatory options for AD that is difficult to treat with TCSs due to adverse reactions. TCIs reduce inflammation by inhibiting the activity of intracellular calcineurin, which leads to the release of proinflammatory cytokines and mediators of AD[102,103]. Two TCIs, including tacrolimus ointment (0.03% and 0.1% for moderate to severe AD) and pimecrolimus cream (1% for mild to moderate AD), are approved for AD treatment[104-106]. Tacrolimus 0.03% ointment and pimecrolimus 1% cream are approved for patients aged 2 years and older, and tacrolimus 0.1% ointment is approved for patients aged 16 years and older. Although off-label use in children under 2 years is reported, more studies are needed to confirm safety[107,108].

An RCT shows tacrolimus 0.1% is more effective than pimecrolimus 1%, with a mean Eczema Area and Severity Index (EASI) score reduction of 54.1% versus 34.9%, respectively[109]. In another RCT, tacrolimus 0.1% demonstrated superior efficacy compared to pimecrolimus 1% based on the investigator's global assessment score of 0 or 1[110]. The anti-inflammatory effect of tacrolimus 0.1% ointment is comparable to that of potent TCSs.

We suggest using an initial application of topical corticosteroid before switching to topical calcineurin inhibitors to reduce local skin reactions like burning sensation (Recommendation strength: B, Grade of evidence: 5, % of respondents [agreement score ≥7]: 72%).

Long-term safety studies (10 years for tacrolimus and 5 years for pimecrolimus) confirm the safety of TCIs in AD management[111,112]. Common local side effects include burning, pruritus, and erythema, which are more frequent with tacrolimus, especially on inflamed skin. Pretreatment with TCSs or repeated application reduces these symptoms[113,114]. Bacterial or viral infections (e.g., herpes simplex, molluscum contagiosum) may occur during TCI treatment[115], though causal relationships are unconfirmed[111,116].

We recommend the use of topical calcineurin inhibitors for sensitive areas such as the face, intertriginous, and genital regions (Recommendation strength: A, Grade of evidence: 1b, % of respondents [agreement score ≥7]: 98%).

TCIs do not cause cutaneous atrophy, making them preferable for sensitive areas such as the eyelids, perioral region, genital area, axilla, and inguinal folds[117]. The U.S. Food and Drug Administration (FDA) includes a black box warning for a potential increased cancer risk with TCI use, though long-term studies show no significant rise in malignancies, including lymphoma[118]. Continued monitoring with larger studies is needed to evaluate any carcinogenic risk.

We recommend proactive therapy with a moderate potency topical corticosteroid or topical calcineurin inhibitors 2 to 3 times a week to prevent flares or relapses in the improved areas (Recommendation strength: A, Grade of evidence: 1a, % of respondents [agreement score ≥7]: 94%).

For maintenance, TCIs or moderate-potency TCSs are recommended 2–3 times weekly to prevent flares. Long-term TCI use after flare resolution effectively prevents recurrence and maintains disease control[119,120].

3) Other topical anti-inflammatory and anti-pruritic agents

Additional information on topical phosphodiesterase 4 (PDE-4) inhibitors, JAK inhibitors, and anti-pruritic agents such as topical anesthetics, topical capsaicin, and topical doxepin, including their mechanisms, indications, and associated risks, is provided in the supplementary materials (**Supplementary Data 2**).

Conventional systemic therapies

1) Cyclosporine

We recommend the use of cyclosporine for patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies (Recommendation strength: A, Grade of evidence: 1a, % of respondents [agreement score ≥7]: 98%).

Cyclosporine, a lipophilic cyclic polypeptide, inhibits interleukin 2 transcription, reducing T-cell activation pivotal to AD pathogenesis[121]. The recommended dose is 2.5-5mg/kg/day, with higher initial doses (4–5 mg/kg) providing faster relief for acute flares. A meta-analysis showed a 22% severity reduction at ≤3 mg/kg and 40% at ≥4 mg/kg after 2 weeks[122].

Cyclosporine demonstrated superior efficacy to placebo in improving clinical signs, patient-reported outcomes, and quality

of life in adults[123], and reducing clinical severity by 55% after 6–8 weeks[122]. It outperformed prednisolone, intravenous immunoglobulin, ultraviolet (UV) A and UVB, and matched the efficacy of enteric-coated mycophenolate sodium in head-to-head trial[124]. At 2.5 mg/kg/day, cyclosporine was more effective than methotrexate (MTX, 15 mg/week) for moderate to severe AD at week 8[125].

Adverse effects include nephrotoxicity, hypertension, tremors, headaches, paresthesia, hypertrichosis, gingival hyperplasia, gastrointestinal symptoms, hypertriglyceridemia, and susceptibility to infection[126,127]. Long-term use (up to 1 year) has shown tolerability but requires careful monitoring due to the open-label design and high dropout rate[124].

> We recommend regular blood pressure monitoring and laboratory testing to identify adverse effects of cyclosporine in patients with AD (Recommendation strength: A, Grade of evidence: 1a, % of respondents [agreement score ≥7]: 98%).

Blood pressure should be monitored at every visit. Laboratory tests, including complete blood count (CBC), lipid profile, and renal/liver function, should be conducted at initiation, every 2–4 weeks during dose escalation, and at least once every three months during maintenance. Pre-treatment screening for hepatitis B/C, human immunodeficiency virus (HIV), and tuberculosis is recommended[126].

2) MTX

> We suggest the selective use of methotrexate for patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies (Recommendation strength: B, Grade of evidence: 1b, % of respondents [agreement score ≥7]: 84%).

MTX reduces inflammation by modulating adenosine pathways, inhibiting T-cell activation, and suppressing the JAK/signal transducer and activator of transcription pathway[128].

For adult patients with AD, initial dose is 5–15 mg/week, up to 25 mg/week[129]. MTX is as effective as azathioprine, achieving 42% SCORing Atopic Dermatitis (SCORAD) improvement at 12 weeks[130] and similar long-term reductions[131]. MTX 15 mg/week was less effective than cyclosporine 2.5 mg/kg/day at week 8, but MTX at 25 mg/week achieved EASI-50 in 92% by week 20[125]. In a Korean study, initial responses occurred at 5.8 weeks with a median maintenance dose of 11.7 mg/week; 60.8% showed significant improvement[128].

Rare side effects of MTX include bone marrow suppression, liver toxicity, and pulmonary fibrosis. Evidence supports its safety for long-term use[128,131,132].

CBC, renal, and liver profiles should be monitored prior to treatment, every 2 weeks during the first 2 months or after dose adjustments, and subsequently every 8–12 weeks. A type III procollagen peptide assay, fibroscan or liver biopsy can be performed according to cumulative dosage[126,129]. Pre-treatment screening for hepatitis B/C, HIV, and tuberculosis is recommended.

3) Azathioprine

> We suggest the selective use of azathioprine for patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies (Recommendation strength: B, Grade of evidence: 1b, % of respondents [agreement score ≥7]: 58%).

Azathioprine is a prodrug metabolized into 6-mercaptopurine, disrupting DNA, RNA, and protein synthesis. It inhibits mitosis, neutrophil and monocyte formation, and prostaglandin synthesis[133].

Its metabolism depends on thiopurine methyltransferase (TPMT) activity. Adults with normal TPMT levels require 1–3 mg/kg/day[134]. If TPMT testing is unavailable, initiating treatment at half the standard dose for 4–6 weeks with close monitoring of CBC and liver function may be considered[129]. Dose adjustments are needed for renal or hepatic impairment[133].

Three studies evaluated the efficacy of azathioprine for AD over 12 weeks to five years. Compared to placebo, azathioprine reduced AD severity by 26%–36% after 12 weeks[135,136] and showed similar efficacy to MTX in clinical outcomes[131]. Adverse effects include gastrointestinal discomfort, hypersensitivity reaction, bone marrow suppression, and hepatotoxicity[133].

Baseline TPMT levels, CBC, renal, and liver function tests are mandatory before initiation, and azathioprine is contraindicated in patients with low TPMT activity[134]. Regular monitoring of CBC, renal, and liver profiles is required every 2 weeks for the first 2 months, then every 8–12 weeks. Pre-treatment screening for hepatitis B/C, HIV, and tuberculosis is also recommended[126].

4) Mycophenolate mofetil (MMF)

> We propose the limited use of MMF for patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies (Recommendation strength: C, Grade of evidence: 4, % of respondents [agreement score ≥7]: 62%).

MMF inhibits inosine monophosphate dehydrogenase, thereby blocking guanine nucleotide synthesis[137]. It is initiated at 0.5 g/day and adjusted to 1–2 g/day based on clinical response[138]. A systematic review reported 77.6% achieving partial or complete

remission, with an 8.2% relapse rate[137]. Initial effects were observed in 6.8 weeks, with SCORAD scores improving from 47.7 to 29.7[137]. MMF is as effective as cyclosporine for maintenance but has a slower onset of action[139].

MMF is generally well-tolerated, with gastrointestinal symptoms, headache, flu-like symptoms, and fatigue being common, while serious side effects, such as leukocytopenia, anemia, thrombocytopenia, or alteration of liver function, are rare[139].

CBC, renal, and liver profiles should be checked before treatment, every 2 weeks for 2 months, then every 8–12 weeks[126]. Screening for hepatitis B/C, HIV, and tuberculosis is recommended[126].

5) Systemic corticosteroid

> Long-term use of systemic corticosteroid is not recommended due to the potential for side effects (Recommendation strength: D, Grade of evidence: 5, % of respondents [agreement score ≥7]: 96%).

Corticosteroids inhibit the transcriptional activity of nuclear factor kappa-light-chain-enhancer of activated B cells and activator protein-1, thereby reducing the synthesis of pro-inflammatory cytokines and exerting anti-inflammatory effects[140]. Despite their dramatic efficacy in improving AD symptoms, systemic corticosteroids should generally be avoided due to adverse effects and rebound risk[126]. The recommended dose is 0.5 mg/kg/day for 1–2 weeks during acute flares.

Several RCTs showed corticosteroids were superior to placebo in improving clinical and patient-reported scores[123,141]. A trial comparing prednisolone to cyclosporine found no significant difference in SCORAD 50 rates, but cyclosporine was superior in achieving stable remission without relapse over 12 weeks[142].

Chronic corticosteroid use can cause hypertension, diabetes, osteoporosis, skin atrophy, Cushing's syndrome, and emotional lability[143]. For acute rescue therapy, laboratory monitoring should be tailored to individual patient needs[129].

6) Alitretinoin

> We propose the limited use of alitretinoin to improve hand eczema in patients with AD. (Recommendation strength: C, Grade of evidence: 4, % of respondents [agreement score ≥7]: 70%).

The biological effects of alitretinoin are mediated by retinoic acid receptor (RAR) and/or retinoid X receptor (RXR)[30]. RAR and RXR regulate skin differentiation, proliferation, and inflammation[114,144].

The dosage of alitretinoin is 10–30 mg/day. In a study of six adults with chronic hand eczema and moderate AD, 30 mg/day for 12 weeks resulted in over 50% improvement in palmar lesions and extra-palmar AD symptoms[145].

The most serious side effect is teratogenicity, with common adverse events including headaches, mucocutaneous issues, hyperlipidemia, and decreased free thyroxine (T4) and thyroid stimulation hormone (TSH)[114]. Liver enzymes, lipid profiles, TSH, and free T4 should be monitored before and during treatment.

DISCUSSION

The guideline underscores the importance of basic therapies as the foundation of AD management. Core elements include regular use of moisturizers, proper cleansing and bathing routines, allergen avoidance, and patient education to improve adherence and understanding of care. Topical agents, such as TCS and TCI, are recommended for effective inflammation control. For moderate to severe cases unresponsive to topical treatments, conventional systemic therapies are advised to further manage the disease. Conventional systemic therapies, such as cyclosporine, and MTX, have long been used for AD and remain effective treatment options. However, they lack extensive long-term efficacy and safety data compared to newer JAK inhibitors and biologics. Nonetheless, their lower cost makes them a practical alternative for patients with moderate to severe AD.

In summary, Part I of KADA's guidelines provides evidence-based strategies for basic, topical, and conventional systemic therapies to optimize AD management.

ORCID iDs

Hyun-Chang Ko
https://orcid.org/0000-0002-3459-5474
Yu Ri Woo
https://orcid.org/0000-0003-2903-9534
Joo Yeon Ko
https://orcid.org/0000-0003-4240-9675
Hye One Kim
https://orcid.org/0000-0001-5846-0008
Chan Ho Na
https://orcid.org/0000-0001-5259-5382
Youin Bae
https://orcid.org/0000-0003-3184-7960
Young-Joon Seo
https://orcid.org/0000-0002-4955-590X
Min Kyung Shin
https://orcid.org/0000-0001-9834-7931
Jiyoung Ahn
https://orcid.org/0000-0002-6766-9978
Bark-Lynn Lew
https://orcid.org/0000-0003-4443-4161

Dong Hun Lee
https://orcid.org/0000-0002-2925-3074
Sang Eun Lee
https://orcid.org/0000-0003-4720-9955
Sul Hee Lee
https://orcid.org/0000-0002-2990-9774
Yang Won Lee
https://orcid.org/0000-0003-1268-1248
Ji Hyun Lee
https://orcid.org/0000-0002-3671-502X
Yong Hyun Jang
https://orcid.org/0000-0003-1706-007X
Jiehyun Jeon
https://orcid.org/0000-0003-2456-7573
Sun Young Choi
https://orcid.org/0000-0003-0248-7708
Ju Hee Han
https://orcid.org/0000-0003-3194-5202
Tae Young Han
https://orcid.org/0000-0003-0317-9635
Sang Wook Son
https://orcid.org/0000-0002-3332-7056
Sang Hyun Cho
https://orcid.org/0000-0001-8289-1190

FUNDING SOURCE

None.

CONFLICTS OF INTEREST

Hyun-Chang Ko received honoraria from Sanofi, Lilly, AbbVie, Leo Pharma, and Pfizer. Jiehyun Jeon received honoraria for lectures from AbbVie, Pfizer, and Sanofi. Dong Hun Lee received grants from Sanofi, Amgen, AbbVie, Leo Pharma, Incyte, Novartis, Lilly, Galderma, Kangstem Bio, and EHL Bio. He also received honoraria for lectures from Sanofi, AbbVie, Leo Pharma, Novartis, Lilly, and Pfizer, and participated on the advisory board for AbbVie. Yu Ri Woo, Joo Yeon Ko, Hye One Kim, Chan Ho Na, Youin Bae, Young-Joon Seo, Min Kyung Shin, Jiyoung Ahn, Bark-Lynn Lew, Sang Eun Lee, Sul Hee Lee, Yang Won Lee, Ji Hyun Lee, Yong Hyun Jang, Sun Young Choi, Ju Hee Han, Tae Young Han, Sang Wook Son, and Sang Hyun Cho have no conflicts of interests to declare.

DATA SHARING STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

SUPPLEMENTARY MATERIALS

Supplementary Data 1

Cleansing and bathing

Supplementary Data 2

Other topical anti-inflammatory and anti-pruritic agents

REFERENCES

1. Leung DY. Pathogenesis of atopic dermatitis. J Allergy Clin Immunol 1999;104:S99-S108. PUBMED | CROSSREF
2. Kim KH. Current status and characteristics of atopic dermatitis in Korea. J Korean Med Assoc 2014;57:208-211. CROSSREF
3. Lee JH, Han KD, Kim KM, Park YG, Lee JY, Park YM. Prevalence of atopic dermatitis in Korean children based on data from the 2008-2011 Korean National Health and Nutrition Examination Survey. Allergy Asthma Immunol Res 2016;8:79-83. PUBMED | CROSSREF
4. Noda S, Suárez-Fariñas M, Ungar B, Kim SJ, de Guzman Strong C, Xu H, et al. The Asian atopic dermatitis phenotype combines features of atopic dermatitis and psoriasis with increased TH17 polarization. J Allergy Clin Immunol 2015;136:1254-1264. PUBMED | CROSSREF
5. Hawkins K, David E, Glickman JW, Del Duca E, Guttman-Yassky E, Krueger JG. Atopic dermatitis stratification: current and future perspective on skin and blood transcriptomic and proteomic profiling. Expert Rev Clin Immunol 2024;20:1083-1088. PUBMED | CROSSREF
6. Lee DG, Lee KJ, Kang H, Kim JE. Characteristics of atopic dermatitis according to common phenotypes. Korean J Dermatol 2022;60:16-24.
7. Chiricozzi A, Maurelli M, Calabrese L, Peris K, Girolomoni G. Overview of atopic dermatitis in different ethnic groups. J Clin Med 2023;12:2701. PUBMED | CROSSREF
8. Kim JE, Shin MK, Park GH, Lee UH, Lee JH, Han TY, et al. 2019 Consensus Korean diagnostic guidelines to define severity classification and treatment refractoriness for atopic dermatitis: objective and subjective assessment of severity. Ann Dermatol 2019;31:654-661. PUBMED | CROSSREF
9. Draelos ZD. Therapeutic moisturizers. Dermatol Clin 2000;18:597-607. PUBMED | CROSSREF
10. Lodén M, Andersson AC, Lindberg M. Improvement in skin barrier function in patients with atopic dermatitis after treatment with a moisturizing cream (Canoderm). Br J Dermatol 1999;140:264-267. PUBMED | CROSSREF
11. Lindh JD, Bradley M. Clinical effectiveness of moisturizers in atopic dermatitis and related disorders: a systematic review. Am J Clin Dermatol 2015;16:341-359. PUBMED | CROSSREF
12. Rossi AB, Bacquey A, Nocera T, Thouvenin MD. Efficacy and tolerability of a medical device repairing emollient cream associated with a topical corticosteroid in adults with atopic dermatitis: an open-label, intra-individual randomized controlled study. Dermatol Ther (Heidelb) 2018;8:217-228. PUBMED | CROSSREF
13. Wirén K, Nohlgård C, Nyberg F, Holm L, Svensson M, Johannesson A, et al. Treatment with a barrier-strengthening moisturizing cream delays relapse of atopic dermatitis: a prospective and randomized controlled clinical trial. J Eur Acad Dermatol Venereol 2009;23:1267-1272. PUBMED | CROSSREF
14. Lucky AW, Leach AD, Laskarzewski P, Wenck H. Use of an emollient as a steroid-sparing agent in the treatment of mild to moderate atopic dermatitis in children. Pediatr Dermatol 1997;14:321-324. PUBMED | CROSSREF
15. Boralevi F, Saint Aroman M, Delarue A, Raudsepp H, Kaszuba A, Bylaite M, et al. Long-term emollient therapy improves xerosis in children with atopic dermatitis. J Eur Acad Dermatol Venereol 2014;28:1456-1462. PUBMED | CROSSREF
16. Grimalt R, Mengeaud V, Cambazard F; Study Investigators' Group. The steroid-sparing effect of an emollient therapy in infants with atopic dermatitis: a randomized controlled study. Dermatology 2007;214:61-67. PUBMED | CROSSREF

17. Åkerström U, Reitamo S, Langeland T, Berg M, Rustad L, Korhonen L, et al. Comparison of moisturizing creams for the prevention of atopic dermatitis relapse: a randomized double-blind controlled multicentre clinical trial. Acta Derm Venereol 2015;95:587-592. PUBMED | CROSSREF
18. Darsow U, Lübbe J, Taïeb A, Seidenari S, Wollenberg A, Calza AM, et al. Position paper on diagnosis and treatment of atopic dermatitis. J Eur Acad Dermatol Venereol 2005;19:286-295. PUBMED | CROSSREF
19. Na JI, Hwang JS, Park HJ, Kim DH, Park WS, Youn SW, et al. A new moisturizer containing physiologic lipid granules alleviates atopic dermatitis. J Dermatolog Treat 2010;21:23-27. PUBMED | CROSSREF
20. Chamlin SL, Kao J, Frieden IJ, Sheu MY, Fowler AJ, Fluhr JW, et al. Ceramide-dominant barrier repair lipids alleviate childhood atopic dermatitis: changes in barrier function provide a sensitive indicator of disease activity. J Am Acad Dermatol 2002;47:198-208. PUBMED | CROSSREF
21. Wollenberg A, Barbarot S, Bieber T, Christen-Zaech S, Deleuran M, Fink-Wagner A, et al. Consensus-based European guidelines for treatment of atopic eczema (atopic dermatitis) in adults and children: part I. J Eur Acad Dermatol Venereol 2018;32:657-682. PUBMED | CROSSREF
22. Chow S, Seow CS, Dizon MV, Godse K, Foong H, Chan V, et al. A clinician's reference guide for the management of atopic dermatitis in Asians. Asia Pac Allergy 2018;8:e41. PUBMED | CROSSREF
23. Wollenberg A, Christen-Zäch S, Taieb A, Paul C, Thyssen JP, de Bruin-Weller M, et al. ETFAD/EADV Eczema Task Force 2020 position paper on diagnosis and treatment of atopic dermatitis in adults and children. J Eur Acad Dermatol Venereol 2020;34:2717-2744. PUBMED | CROSSREF
24. Dekio I, Morita E. The weight of a finger-tip unit of ointment in 5-gram tubes. J Dermatolog Treat 2011;22:302-303. PUBMED | CROSSREF
25. Wollenberg A, Kinberger M, Arents B, Aszodi N, Avila Valle G, Barbarot S, et al. European guideline (EuroGuiDerm) on atopic eczema - part II: non-systemic treatments and treatment recommendations for special AE patient populations. J Eur Acad Dermatol Venereol 2022;36:1904-1926. PUBMED | CROSSREF
26. Miyachi Y. The application quantity quantification tool: how to use the FTU and precautions. J Visual Dermatol 2017;16:462-464.
27. Nakamura M, Uemura K, Nemoto O, Miyachi Y. Evaluation of an optimal method for topical application of moisturizer. Skin Res 2006;5:311-316. CROSSREF
28. Otani M, Mi O, Nozawa A, Matsumoto M, Yamamura Y, Komoda M. A study of the influence of the volume and frequency of application on the efficacy of moisturizers. Jpn J Dermatol 2012;122:39-43.
29. Kim JE, Kim HJ, Lew BL, Lee KH, Hong SP, Jang YH, et al. Consensus guidelines for the treatment of atopic dermatitis in Korea (Part I): general management and topical treatment. Ann Dermatol 2015;27:563-577. PUBMED | CROSSREF
30. Chan TC, Wu NL, Wong LS, Cho YT, Yang CY, Yu Y, et al. Taiwanese Dermatological Association consensus for the management of atopic dermatitis: a 2020 update. J Formos Med Assoc 2021;120:429-442. PUBMED | CROSSREF
31. Eichenfield LF, Tom WL, Berger TG, Krol A, Paller AS, Schwarzenberger K, et al. Guidelines of care for the management of atopic dermatitis: section 2. Management and treatment of atopic dermatitis with topical therapies. J Am Acad Dermatol 2014;71:116-132. PUBMED | CROSSREF
32. Zhong Y, Samuel M, van Bever H, Tham EH. Emollients in infancy to prevent atopic dermatitis: a systematic review and meta-analysis. Allergy 2022;77:1685-1699. PUBMED | CROSSREF
33. Xu D, Stengel R, Sun P. Effectiveness of emollients in the prevention of atopic dermatitis in infants: a meta-analysis. Dermatology 2022;238:711-716. PUBMED | CROSSREF
34. Kelleher MM, Cro S, Van Vogt E, Cornelius V, Lodrup Carlsen KC, Ove Skjerven H, et al. Skincare interventions in infants for preventing eczema and food allergy: a Cochrane systematic review and individual participant data meta-analysis. Clin Exp Allergy 2021;51:402-418. PUBMED | CROSSREF
35. Gittler JK, Wang JF, Orlow SJ. Bathing and associated treatments in atopic dermatitis. Am J Clin Dermatol 2017;18:45-57. PUBMED | CROSSREF
36. Mukhopadhyay P. Cleansers and their role in various dermatological disorders. Indian J Dermatol 2011;56:2-6. PUBMED | CROSSREF
37. Sybilski AJ, Zalewska M, Furmańczyk K, Lipiec A, Krzych-Fałta E, Samoliński B. The prevalence of sensitization to inhalant allergens in children with atopic dermatitis. Allergy Asthma Proc 2015;36:e81-e85. PUBMED | CROSSREF
38. Tsakok T, Marrs T, Mohsin M, Baron S, du Toit G, Till S, et al. Does atopic dermatitis cause food allergy? A systematic review. J Allergy Clin Immunol 2016;137:1071-1078. PUBMED | CROSSREF
39. Han Y, Cho M, Woo YR, Kim HS, Lee JD, Cho SH. Evolution in allergen sensitization in Korean patients with atopic dermatitis: a 10-year retrospective study based on the multiple allergen simultaneous test. Korean J Dermatol 2022;60:350-363.
40. Chu H, Shin JU, Park CO, Lee H, Lee J, Lee KH. Clinical diversity of atopic dermatitis: a review of 5,000 patients at a single institute. Allergy Asthma Immunol Res 2017;9:158-168. PUBMED | CROSSREF
41. Kim J, Lee S, Woo SY, Han Y, Lee JH, Lee IY, et al. The indoor level of house dust mite allergen is associated with severity of atopic dermatitis in children. J Korean Med Sci 2013;28:74-79. PUBMED | CROSSREF
42. Cid BJ, Perez-Mateluna G, Iturriaga C, Zambrano MJ, Vives MI, Valenzuela PM, et al. Is there an association between indoor allergens and the severity of atopic dermatitis? Int J Dermatol 2019;58:433-439. PUBMED | CROSSREF
43. Miller JD. The role of dust mites in allergy. Clin Rev Allergy Immunol 2019;57:312-329. PUBMED | CROSSREF
44. Oosting AJ, de Bruin-Weller MS, Terreehorst I, Tempels-Pavlica Z, Aalberse RC, de Monchy JG, et al. Effect of mattress encasings on atopic dermatitis outcome measures in a double-blind, placebo-controlled study: the Dutch mite avoidance study. J Allergy Clin Immunol 2002;110:500-506. PUBMED | CROSSREF
45. Gutgesell C, Heise S, Seubert S, Seubert A, Domhof S, Brunner E, et al. Double-blind placebo-controlled house dust mite control measures in adult patients with atopic dermatitis. Br J Dermatol 2001;145:70-74. PUBMED | CROSSREF
46. Tan BB, Weald D, Strickland I, Friedmann PS. Double-blind controlled trial of effect of housedust-mite allergen avoidance on atopic dermatitis. Lancet 1996;347:15-18. PUBMED | CROSSREF
47. Ricci G, Patrizi A, Specchia F, Menna L, Bottau P, D'Angelo V, et al. Effect of house dust mite avoidance measures in children with atopic dermatitis. Br J Dermatol 2000;143:379-384. PUBMED | CROSSREF
48. Nankervis H, Pynn EV, Boyle RJ, Rushton L, Williams HC, Hewson DM, et al. House dust mite reduction and avoidance measures for treating eczema. Cochrane Database Syst Rev 2015;1:CD008426. PUBMED | CROSSREF
49. Bremmer SF, Simpson EL. Dust mite avoidance for the primary prevention of atopic dermatitis: a systematic review and meta-analysis. Pediatr Allergy Immunol 2015;26:646-654. PUBMED | CROSSREF

50. Saeki H, Ohya Y, Furuta J, Arakawa H, Ichiyama S, Katsunuma T, et al. English version of clinical practice guidelines for the management of atopic dermatitis 2021. J Dermatol 2022;49:e315-e375. PUBMED | CROSSREF
51. Li C, Chen Q, Zhang X, Li H, Liu Q, Fei P, et al. Early life domestic pet ownership, and the risk of pet sensitization and atopic dermatitis in preschool children: a prospective birth cohort in Shanghai. Front Pediatr 2020;8:192. PUBMED | CROSSREF
52. Milewska-Wróbel D, Lis-Święty A. Does maternal pet ownership during pregnancy influence severity of child's atopic dermatitis? Early Hum Dev 2020;151:105173. PUBMED | CROSSREF
53. Pelucchi C, Galeone C, Bach JF, La Vecchia C, Chatenoud L. Pet exposure and risk of atopic dermatitis at the pediatric age: a meta-analysis of birth cohort studies. J Allergy Clin Immunol 2013;132:616-622.e7. PUBMED | CROSSREF
54. Eigenmann PA, Sicherer SH, Borkowski TA, Cohen BA, Sampson HA. Prevalence of IgE-mediated food allergy among children with atopic dermatitis. Pediatrics 1998;101:E8. PUBMED | CROSSREF
55. Bath-Hextall F, Delamere FM, Williams HC. Dietary exclusions for established atopic eczema. Cochrane Database Syst Rev 2008;2008:CD005203. PUBMED | CROSSREF
56. Finlay AY. Quality of life in atopic dermatitis. J Am Acad Dermatol 2001;45:S64-S66. PUBMED | CROSSREF
57. Bass AM, Anderson KL, Feldman SR. Interventions to increase treatment adherence in pediatric atopic dermatitis: a systematic review. J Clin Med 2015;4:231-242. PUBMED | CROSSREF
58. Farhi D, Taïeb A, Tilles G, Wallach D. The historical basis of a misconception leading to undertreating atopic dermatitis (eczema): facts and controversies. Clin Dermatol 2010;28:45-51. PUBMED | CROSSREF
59. Jeon YH. Dietary restriction misconceptions and food allergy education in children with atopic dermatitis. Clin Exp Pediatr 2022;65:83-84. PUBMED | CROSSREF
60. Narla S, Hsu DY, Thyssen JP, Silverberg JI. Inpatient financial burden of atopic dermatitis in the United States. J Invest Dermatol 2017;137:1461-1467. PUBMED | CROSSREF
61. Stalder JF, Barbarot S. Atopic dermatitis school: therapeutic education of atopic patients. Rev Prat 2006;56:273-276. PUBMED
62. Lambert J, Bostoen J, Geusens B, Bourgois J, Boone J, De Smedt D, et al. A novel multidisciplinary educational programme for patients with chronic skin diseases: Ghent pilot project and first results. Arch Dermatol Res 2011;303:57-63. PUBMED | CROSSREF
63. Staab D, von Rueden U, Kehrt R, Erhart M, Wenninger K, Kamtsiuris P, et al. Evaluation of a parental training program for the management of childhood atopic dermatitis. Pediatr Allergy Immunol 2002;13:84-90. PUBMED | CROSSREF
64. Staab D, Diepgen TL, Fartasch M, Kupfer J, Lob-Corzilius T, Ring J, et al. Age related, structured educational programmes for the management of atopic dermatitis in children and adolescents: multicentre, randomised controlled trial. BMJ 2006;332:933-938. PUBMED | CROSSREF
65. Barbarot S, Bernier C, Deleuran M, De Raeve L, Eichenfield L, El Hachem M, et al. Therapeutic patient education in children with atopic dermatitis: position paper on objectives and recommendations. Pediatr Dermatol 2013;30:199-206. PUBMED | CROSSREF
66. Ehlers A, Stangier U, Gieler U. Treatment of atopic dermatitis: a comparison of psychological and dermatological approaches to relapse prevention. J Consult Clin Psychol 1995;63:624-635. PUBMED | CROSSREF
67. Jang YH, Lee JS, Kim SL, Song CH, Jung HD, Shin DH, et al. A family-engaged educational program for atopic dermatitis: a seven-year, multicenter experience in Daegu-Gyeongbuk, South Korea. Ann Dermatol 2015;27:383-388. PUBMED | CROSSREF
68. Barbarot S, Stalder JF. Therapeutic patient education in atopic eczema. Br J Dermatol 2014;170 Suppl 1:44-48. PUBMED | CROSSREF
69. Park GY, Park HS, Cho S, Yoon HS. The effectiveness of tailored education on the usage of moisturizers in atopic dermatitis: a pilot study. Ann Dermatol 2017;29:360-362. PUBMED | CROSSREF
70. Kupfer J, Gieler U, Diepgen TL, Fartasch M, Lob-Corzilius T, Ring J, et al. Structured education program improves the coping with atopic dermatitis in children and their parents-a multicenter, randomized controlled trial. J Psychosom Res 2010;68:353-358. PUBMED | CROSSREF
71. Barbarot S, Gagnayre R, Bernier C, Chavigny JM, Chiaverini C, Lacour JP, et al. A guide for education programs in atopic dermatitis. Ann Dermatol Venereol 2007;134:121-127. PUBMED | CROSSREF
72. Heratizadeh A, Werfel T, Wollenberg A, Abraham S, Plank-Habibi S, Schnopp C, et al. Effects of structured patient education in adults with atopic dermatitis: multicenter randomized controlled trial. J Allergy Clin Immunol 2017;140:845-853.e3. PUBMED | CROSSREF
73. LeBovidge JS, Elverson W, Timmons KG, Hawryluk EB, Rea C, Lee M, et al. Multidisciplinary interventions in the management of atopic dermatitis. J Allergy Clin Immunol 2016;138:325-334. PUBMED | CROSSREF
74. Chisolm SS, Taylor SL, Balkrishnan R, Feldman SR. Written action plans: potential for improving outcomes in children with atopic dermatitis. J Am Acad Dermatol 2008;59:677-683. PUBMED | CROSSREF
75. Ntuen E, Taylor SL, Kinney M, O'Neill JL, Krowchuk DP, Feldman SR. Physicians' perceptions of an eczema action plan for atopic dermatitis. J Dermatolog Treat 2010;21:28-33. PUBMED | CROSSREF
76. Rork JF, Sheehan WJ, Gaffin JM, Timmons KG, Sidbury R, Schneider LC, et al. Parental response to written eczema action plans in children with eczema. Arch Dermatol 2012;148:391-392. PUBMED | CROSSREF
77. Shi VY, Nanda S, Lee K, Armstrong AW, Lio PA. Improving patient education with an eczema action plan: a randomized controlled trial. JAMA Dermatol 2013;149:481-483. PUBMED | CROSSREF
78. Sauder MB, McEvoy A, Sampson M, Kanigsberg N, Vaillancourt R, Ramien ML, et al. The effectiveness of written action plans in atopic dermatitis. Pediatr Dermatol 2016;33:e151-e153. PUBMED | CROSSREF
79. Simpson EL, Bruin-Weller M, Flohr C, Ardern-Jones MR, Barbarot S, Deleuran M, et al. When does atopic dermatitis warrant systemic therapy? Recommendations from an expert panel of the International Eczema Council. J Am Acad Dermatol 2017;77:623-633. PUBMED | CROSSREF
80. Holm JG, Agner T, Clausen ML, Thomsen SF. Quality of life and disease severity in patients with atopic dermatitis. J Eur Acad Dermatol Venereol 2016;30:1760-1767. PUBMED | CROSSREF
81. Ali F, Vyas J, Finlay AY. Counting the burden: atopic dermatitis and health-related quality of life. Acta Derm Venereol 2020;100:adv00161. PUBMED | CROSSREF
82. Silverberg JI, Lei D, Yousaf M, Janmohamed SR, Vakharia PP, Chopra R, et al. Comparison of patient-oriented eczema measure and patient-oriented scoring atopic dermatitis vs eczema area and severity index and other measures of atopic dermatitis: a validation study. Ann Allergy Asthma Immunol 2020;125:78-83. PUBMED | CROSSREF
83. Sears HW, Bailer JW, Yeadon A. Efficacy and safety of hydrocortisone buteprate 0.1% cream in patients with atopic dermatitis. Clin Ther 1997;19:710-719. PUBMED | CROSSREF

84. Lebwohl M. Efficacy and safety of fluticasone propionate ointment, 0.005%, in the treatment of eczema. Cutis 1996;57:62-68. PUBMED
85. Matheson R, Kempers S, Breneman D, Draelos Z, Johnson CE, Loss R, et al. Hydrocortisone butyrate 0.1% lotion in the treatment of atopic dermatitis in pediatric subjects. J Drugs Dermatol 2008;7:266-271. PUBMED
86. Nankervis H, Thomas KS, Delamere FM, Barbarot S, Smith S, Rogers NK, et al. What is the evidence base for atopic eczema treatments? A summary of published randomized controlled trials. Br J Dermatol 2017;176:910-927. PUBMED | CROSSREF
87. Katoh N, Ohya Y, Ikeda M, Ebihara T, Katayama I, Saeki H, et al. Clinical practice guidelines for the management of atopic dermatitis 2018. J Dermatol 2019;46:1053-1101. PUBMED | CROSSREF
88. Bleehen SS, Chu AC, Hamann I, Holden C, Hunter JA, Marks R. Fluticasone propionate 0.05% cream in the treatment of atopic eczema: a multicentre study comparing once-daily treatment and once-daily vehicle cream application versus twice-daily treatment. Br J Dermatol 1995;133:592-597. PUBMED | CROSSREF
89. Williams HC. Established corticosteroid creams should be applied only once daily in patients with atopic eczema. BMJ 2007;334:1272. PUBMED | CROSSREF
90. Bieber T, Vick K, Fölster-Holst R, Belloni-Fortina A, Städtler G, Worm M, et al. Efficacy and safety of methylprednisolone aceponate ointment 0.1% compared to tacrolimus 0.03% in children and adolescents with an acute flare of severe atopic dermatitis. Allergy 2007;62:184-189. PUBMED | CROSSREF
91. Lax SJ, Harvey J, Axon E, Howells L, Santer M, Ridd MJ, et al. Strategies for using topical corticosteroids in children and adults with eczema. Cochrane Database Syst Rev 2022;3:CD013356. PUBMED | CROSSREF
92. Hengge UR. Topical corticosteroids. In: Gaspari AA, Tyring SK, editors. Clinical and basic immunodermatology. 1st ed. London: Springer, 2008: 561-577.
93. Coondoo A, Phiske M, Verma S, Lahiri K. Side-effects of topical steroids: a long overdue revisit. Indian Dermatol Online J 2014;5:416-425. PUBMED | CROSSREF
94. Furue M. Topical corticosteroids: how to use in clinical practice. Arerugi 2009;58:491-498. PUBMED
95. Furue M, Terao H, Rikihisa W, Urabe K, Kinukawa N, Nose Y, et al. Clinical dose and adverse effects of topical steroids in daily management of atopic dermatitis. Br J Dermatol 2003;148:128-133. PUBMED | CROSSREF
96. Beck KM, Seitzman GD, Yang EJ, Sanchez IM, Liao W. Ocular co-morbidities of atopic dermatitis. Part II: ocular disease secondary to treatments. Am J Clin Dermatol 2019;20:807-815. PUBMED | CROSSREF
97. Hsu JI, Pflugfelder SC, Kim SJ. Ocular complications of atopic dermatitis. Cutis 2019;104:189-193. PUBMED
98. van Velsen SG, De Roos MP, Haeck IM, Sparidans RW, Bruijnzeel-Koomen CA. The potency of clobetasol propionate: serum levels of clobetasol propionate and adrenal function during therapy with 0.05% clobetasol propionate in patients with severe atopic dermatitis. J Dermatolog Treat 2012;23:16-20. PUBMED | CROSSREF
99. Aubert-Wastiaux H, Moret L, Le Rhun A, Fontenoy AM, Nguyen JM, Leux C, et al. Topical corticosteroid phobia in atopic dermatitis: a study of its nature, origins and frequency. Br J Dermatol 2011;165:808-814. PUBMED | CROSSREF
100. Lee JY, Her Y, Kim CW, Kim SS. Topical corticosteroid phobia among parents of children with atopic eczema in Korea. Ann Dermatol 2015;27:499-506. PUBMED | CROSSREF
101. Kojima R, Fujiwara T, Matsuda A, Narita M, Matsubara O, Nonoyama S, et al. Factors associated with steroid phobia in caregivers of children with atopic dermatitis. Pediatr Dermatol 2013;30:29-35. PUBMED | CROSSREF
102. Sidbury R, Alikhan A, Bercovitch L, Cohen DE, Darr JM, Drucker AM, et al. Guidelines of care for the management of atopic dermatitis in adults with topical therapies. J Am Acad Dermatol 2023;89:e1-e20. PUBMED | CROSSREF
103. Carr WW. Topical calcineurin inhibitors for atopic dermatitis: review and treatment recommendations. Paediatr Drugs 2013;15:303-310. PUBMED | CROSSREF
104. Rubel D, Thirumoorthy T, Soebaryo RW, Weng SC, Gabriel TM, Villafuerte LL, et al. Consensus guidelines for the management of atopic dermatitis: an Asia-Pacific perspective. J Dermatol 2013;40:160-171. PUBMED | CROSSREF
105. Ashcroft DM, Dimmock P, Garside R, Stein K, Williams HC. Efficacy and tolerability of topical pimecrolimus and tacrolimus in the treatment of atopic dermatitis: meta-analysis of randomised controlled trials. BMJ 2005;330:516. PUBMED | CROSSREF
106. Paller A, Eichenfield LF, Leung DY, Stewart D, Appell M. A 12-week study of tacrolimus ointment for the treatment of atopic dermatitis in pediatric patients. J Am Acad Dermatol 2001;44:S47-S57. PUBMED | CROSSREF
107. El-Batawy MM, Bosseila MA, Mashaly HM, Hafez VS. Topical calcineurin inhibitors in atopic dermatitis: a systematic review and meta-analysis. J Dermatol Sci 2009;54:76-87. PUBMED | CROSSREF
108. Patel RR, Vander Straten MR, Korman NJ. The safety and efficacy of tacrolimus therapy in patients younger than 2 years with atopic dermatitis. Arch Dermatol 2003;139:1184-1186. PUBMED | CROSSREF
109. Paller AS, Lebwohl M, Fleischer AB Jr, Antaya R, Langley RG, Kirsner RS, et al. Tacrolimus ointment is more effective than pimecrolimus cream with a similar safety profile in the treatment of atopic dermatitis: results from 3 randomized, comparative studies. J Am Acad Dermatol 2005;52:810-822. PUBMED | CROSSREF
110. Draelos Z, Nayak A, Pariser D, Shupack JL, Chon K, Abrams B, et al. Pharmacokinetics of topical calcineurin inhibitors in adult atopic dermatitis: a randomized, investigator-blind comparison. J Am Acad Dermatol 2005;53:602-609. PUBMED | CROSSREF
111. Reitamo S, Ortonne JP, Sand C, Cambazard F, Bieber T, Fölster-Holst R, et al. A multicentre, randomized, double-blind, controlled study of long-term treatment with 0.1% tacrolimus ointment in adults with moderate to severe atopic dermatitis. Br J Dermatol 2005;152:1282-1289. PUBMED | CROSSREF
112. Sigurgeirsson B, Boznanski A, Todd G, Vertruyen A, Schuttelaar ML, Zhu X, et al. Safety and efficacy of pimecrolimus in atopic dermatitis: a 5-year randomized trial. Pediatrics 2015;135:597-606. PUBMED | CROSSREF
113. Ruzicka T, Larsen FG, Galewicz D, Horváth A, Coenraads PJ, Thestrup-Pedersen K, et al. Oral alitretinoin (9-cis-retinoic acid) therapy for chronic hand dermatitis in patients refractory to standard therapy: results of a randomized, double-blind, placebo-controlled, multicenter trial. Arch Dermatol 2004;140:1453-1459. PUBMED | CROSSREF
114. Wollenberg A, Ehmann LM. Long term treatment concepts and proactive therapy for atopic eczema. Ann Dermatol 2012;24:253-260. PUBMED | CROSSREF
115. Lübbe J. Secondary infections in patients with atopic dermatitis. Am J Clin Dermatol 2003;4:641-654. PUBMED | CROSSREF
116. Wahn U, Bos JD, Goodfield M, Caputo R, Papp K, Manjra A, et al. Efficacy and safety of pimecrolimus cream in the long-term management of atopic dermatitis in children. Pediatrics 2002;110:e2. PUBMED | CROSSREF

117. Hong CH, Gooderham M, Bissonnette R. Evidence review of topical Calcineurin inhibitors for the treatment of adult atopic dermatitis. J Cutan Med Surg 2019;23:5S-10S. PUBMED | CROSSREF

118. Maruho Co., Ltd. Results of a post-marketing survey regarding Protopic® ointment 0.03% for children. Osaka: Maruho Co., Ltd., 2019.

119. Wollenberg A, Reitamo S, Atzori F, Lahfa M, Ruzicka T, Healy E, et al. Proactive treatment of atopic dermatitis in adults with 0.1% tacrolimus ointment. Allergy 2008;63:742-750. CROSSREF

120. Breneman D, Fleischer AB Jr, Abramovits W, Zeichner J, Gold MH, Kirsner RS, et al. Intermittent therapy for flare prevention and long-term disease control in stabilized atopic dermatitis: a randomized comparison of 3-times-weekly applications of tacrolimus ointment versus vehicle. J Am Acad Dermatol 2008;58:990-999. PUBMED | CROSSREF

121. Liu J. FK506 and cyclosporin, molecular probes for studying intracellular signal transduction. Immunol Today 1993;14:290-295. PUBMED | CROSSREF

122. Schmitt J, Schmitt N, Meurer M. Cyclosporin in the treatment of patients with atopic eczema - a systematic review and meta-analysis. J Eur Acad Dermatol Venereol 2007;21:606-619. PUBMED | CROSSREF

123. Siegels D, Heratizadeh A, Abraham S, Binnmyr J, Brockow K, Irvine AD, et al. Systemic treatments in the management of atopic dermatitis: a systematic review and meta-analysis. Allergy 2021;76:1053-1076. PUBMED | CROSSREF

124. Roekevisch E, Spuls PI, Kuester D, Limpens J, Schmitt J. Efficacy and safety of systemic treatments for moderate-to-severe atopic dermatitis: a systematic review. J Allergy Clin Immunol 2014;133:429-438. PUBMED | CROSSREF

125. Goujon C, Viguier M, Staumont-Sallé D, Bernier C, Guillet G, Lahfa M, et al. Methotrexate versus cyclosporine in adults with moderate-to-severe atopic dermatitis: a phase III randomized noninferiority trial. J Allergy Clin Immunol Pract 2018;6:562-569.e3. PUBMED | CROSSREF

126. Kim JE, Kim HJ, Lew BL, Lee KH, Hong SP, Jang YH, et al. Consensus guidelines for the treatment of atopic dermatitis in Korea (Part II): systemic treatment. Ann Dermatol 2015;27:578-592. PUBMED | CROSSREF

127. Haw S, Shin MK, Haw CR. The efficacy and safety of long-term oral cyclosporine treatment for patients with atopic dermatitis. Ann Dermatol 2010;22:9-15. PUBMED | CROSSREF

128. Lee JH, Yun SJ, Lee JB, Lee SC. Therapeutic efficacy and safety of methotrexate in moderate-to-severe atopic dermatitis: a retrospective study of Korean patients at tertiary referral hospital. Ann Dermatol 2020;32:402-408. PUBMED | CROSSREF

129. Wollenberg A, Kinberger M, Arents B, Aszodi N, Avila Valle G, Barbarot S, et al. European guideline (EuroGuiDerm) on atopic eczema: part I - systemic therapy. J Eur Acad Dermatol Venereol 2022;36:1409-1431. PUBMED | CROSSREF

130. Schram ME, Roekevisch E, Leeflang MM, Bos JD, Schmitt J, Spuls PI. A randomized trial of methotrexate versus azathioprine for severe atopic eczema. J Allergy Clin Immunol 2011;128:353-359. PUBMED | CROSSREF

131. Gerbens LAA, Hamann SAS, Brouwer MWD, Roekevisch E, Leeflang MMG, Spuls PI. Methotrexate and azathioprine for severe atopic dermatitis: a 5-year follow-up study of a randomized controlled trial. Br J Dermatol 2018;178:1288-1296. PUBMED | CROSSREF

132. Roekevisch E, Schram ME, Leeflang MMG, Brouwer MWD, Gerbens LAA, Bos JD, et al. Methotrexate versus azathioprine in patients with atopic dermatitis: 2-year follow-up data. J Allergy Clin Immunol 2018;141:825-827.e10. PUBMED | CROSSREF

133. Bracho-Borro M, Franco-Ruiz PA, Magaña M. The use of azathioprine in atopic dermatitis: a review. Dermatol Ther 2022;35:e15665. PUBMED | CROSSREF

134. Meggitt SJ, Anstey AV, Mohd Mustapa MF, Reynolds NJ, Wakelin S, Tidman MJ, et al. British Association of Dermatologists' guidelines for the safe and effective prescribing of azathioprine 2011. Br J Dermatol 2011;165:711-734. PUBMED | CROSSREF

135. Meggitt SJ, Gray JC, Reynolds NJ. Azathioprine dosed by thiopurine methyltransferase activity for moderate-to-severe atopic eczema: a double-blind, randomised controlled trial. Lancet 2006;367:839-846. PUBMED | CROSSREF

136. Berth-Jones J, Takwale A, Tan E, Barclay G, Agarwal S, Ahmed I, et al. Azathioprine in severe adult atopic dermatitis: a double-blind, placebo-controlled, crossover trial. Br J Dermatol 2002;147:324-330. PUBMED | CROSSREF

137. Phan K, Smith SD. Mycophenolate mofetil and atopic dermatitis: systematic review and meta-analysis. J Dermatolog Treat 2020;31:810-814. PUBMED | CROSSREF

138. Ballester I, Silvestre JF, Pérez-Crespo M, Lucas A. Severe adult atopic dermatitis: treatment with mycophenolate mofetil in 8 patients. Actas Dermosifiliogr 2009;100:883-887. PUBMED | CROSSREF

139. Haeck IM, Knol MJ, Ten Berge O, van Velsen SG, de Bruin-Weller MS, Bruijnzeel-Koomen CA. Enteric-coated mycophenolate sodium versus cyclosporin A as long-term treatment in adult patients with severe atopic dermatitis: a randomized controlled trial. J Am Acad Dermatol 2011;64:1074-1084. PUBMED | CROSSREF

140. Sardana K, Sachdeva S. Update on pharmacology, actions, dosimetry and regimens of oral glucocorticoids in dermatology. J Cosmet Dermatol 2022;21:5370-5385. PUBMED | CROSSREF

141. Heddle RJ, Soothill JF, Bulpitt CJ, Atherton DJ. Combined oral and nasal beclomethasone diproprionate in children with atopic eczema: a randomised controlled trial. Br Med J (Clin Res Ed) 1984;289:651-654. PUBMED | CROSSREF

142. Schmitt J, Schäkel K, Fölster-Holst R, Bauer A, Oertel R, Augustin M, et al. Prednisolone vs. ciclosporin for severe adult eczema. An investigator-initiated double-blind placebo-controlled multicentre trial. Br J Dermatol 2010;162:661-668. PUBMED | CROSSREF

143. Sidbury R, Davis DM, Cohen DE, Cordoro KM, Berger TG, Bergman JN, et al. Guidelines of care for the management of atopic dermatitis: section 3. Management and treatment with phototherapy and systemic agents. J Am Acad Dermatol 2014;71:327-349. PUBMED | CROSSREF

144. Beckenbach L, Baron JM, Merk HF, Löffler H, Amann PM. Retinoid treatment of skin diseases. Eur J Dermatol 2015;25:384-391. PUBMED | CROSSREF

145. Grahovac M, Molin S, Prinz JC, Ruzicka T, Wollenberg A. Treatment of atopic eczema with oral alitretinoin. Br J Dermatol 2010;162:217-218. PUBMED | CROSSREF