SPECIAL ARTICLE

Skin Allergy Research Society and Society for Eczema Studies Joint Task Force Guidelines of Care for Management of Atopic Dermatitis for Adults, Children, and Special Populations in India: An Evidence-Based Review and an Expert Consensus

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Abstract

Atopic dermatitis (AD) is a chronic inflammatory skin disease with significant morbidity. Recognising the need for region-specific guidance, the Skin Allergy Research Society and Society for Eczema Studies have collaborated to develop updated, evidence-based guidelines tailored to the Indian context. These guidelines address AD management across all age groups, special populations while considering local epidemiology, healthcare infrastructure, and treatment accessibility. A structured Delphi consensus process was conducted among 23 dermatology experts over 3 months through virtual and in-person meetings. Literature from MEDLINE, Cochrane, and Google Scholar was systematically reviewed, and the GRADE (Grading of Recommendations, Assessment, Development, and Evaluations) approach was used to assess evidence quality. Clinical recommendations were refined through multiple voting rounds, leading to consensus statements. Recommendations are based on an extensive literature review up to December 2024. This document updates the 2019 Skin Allergy Society guidelines, reinforcing global recommendations while allowing local adaptability. These guidelines provide updated recommendations for topical, systemic, phototherapy, and biologic therapies in AD. Key advancements include the introduction of topical crisaborole and JAK inhibitors for mild to moderate AD, along with a focus on emerging systemic therapies, such as biologics and systemic JAK inhibitors. In the Indian context, the guidelines define the roles

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Access this article online

Quick Response Code:

Website: https://journals.lww.com/ijd

DOI: 10.4103/ijd.ijd_421_25

How to cite this article: Dhar S, De A, Rajgopalan M, Godse K, Patil A, Chakraborty D, *et al*. Skin allergy research society and society for eczema studies joint task force guidelines of care for management of atopic dermatitis for adults, children, and special populations in India: An Evidence-based review and an expert consensus. Indian J Dermatol 0;0:0.

Received: May, 2025. **Accepted:** May, 2025.

of dupilumab and abrocitinib while also addressing the off-label use of tofacitinib and baricitinib in resource-limited settings. Specific recommendations are provided for children, elderly patients, and pregnant women, emphasising safety considerations for systemic and biologic therapies. These guidelines align with global AD management while incorporating India-specific adaptations based on epidemiology, accessibility, and affordability. They serve as a key reference for dermatologists, pediatricians, and general practitioners in India and other resource-limited settings. Though tailored for India, they are also relevant to dermatologists in developing countries, guiding treatment selection based on disease patterns, environmental factors, and medication availability.

Key Words: *Atopic dermatitis, eczema, guidelines, phototherapy for eczema, systemic therapies for eczema, topical therapies for eczema*

Introduction

Background of the guidelines

The Skin Allergy Research Society of India (SAS) released India's first atopic dermatitis (AD) guidelines in 2018–19.[1] Since then, rapid advancements in research and treatment have emerged. To integrate these updates, SAS and the Society for Eczema Studies (SES) have collaborated to develop the Combined Guidelines of Care for Atopic Dermatitis in India, covering children, adolescents, adults, and special populations.

Why India needs dedicated AD guidelines

Global AD guidelines exist but are shaped by region-specific factors like ethnicity, socioeconomic status, healthcare infrastructure, and demographic variations. India faces distinct challenges, including a lack of local data on AD's natural history, pathogenesis, epidemiology, and clinical presentation. This guideline represents the collective expertise of SAS and SES, offering an evidence-based, India-specific consensus on AD management.

Scope of the guidelines

These guidelines cover AD management across all age groups and severity levels but exclude other dermatitis types (e.g., irritant or allergic contact dermatitis). These guidelines serve as an update to the 2019 Atopic Dermatitis guidelines published by the Skin Allergy Research Society. Recommendations are based on an extensive literature review up to December 2024. Targeted at dermatologists, paediatricians, general physicians, and other healthcare providers treating AD in India, the guidelines update prior therapy recommendations and include all approved treatment options available in the country.

Methodology

These guidelines were developed through a structured, multi-phase process. A panel of 23 experts (seven from each society) collaborated over 3 months via virtual and in-person meetings. They conducted extensive discussions, a Delphi consensus, and jointly drafted the guidelines.

A comprehensive literature search was performed using MEDLINE, Google Scholar, Cochrane, and other reputable sources. The multidisciplinary team systematically reviewed the effectiveness and safety of currently approved topical agents for adult AD in India, applying the GRADE (Grading of Recommendations, Assessment, Development, and Evaluations**)** approach to assess evidence quality and form clinical recommendations.

Each expert independently rated preselected recommendations on an "agree" or "disagree" scale, considering the Indian clinical context. The final insights were consolidated, and a treatment algorithm for AD management was developed. The strength of recommendations and supporting evidence are detailed in Tables 1A and 1B.

Atopic Dermatitis: General Aspects

Definition of AD

AD, or atopic eczema, is a chronic inflammatory skin disorder that typically begins in infancy or childhood. It follows a relapsing course with periods of exacerbation and remission, characterized by intense itching and eczematous dermatitis, often linked to elevated serum IgE levels. AD frequently progresses to food allergies, allergic rhinitis, and asthma, a pattern known as the "atopic march", especially in those with a personal or family history of atopic diathesis (a genetic tendency toward allergic diseases).[1]

AD presents with pruritic, eczematous lesions that often follow a symmetrical distribution across the body. The specific areas affected vary with age: While it commonly appears on the face and extensor surfaces in infants, it shifts to flexural areas in older children and adults. Although AD may resolve during childhood, it can persist into adulthood in a chronic, relapsing form [Box 1].

Box 1: Definition of atopic dermatitis

Atopic dermatitis is a chronic, recurrent inflammatory skin condition, often beginning in childhood. It is marked by itchy, eczematous lesions that tend to appear in specific areas, particularly in the folds of the skin. This condition is frequently found in patients with a personal or family history of *atopic diathesis*, which includes:

A personal or family history of bronchial asthma, allergic rhinitis, conjunctivitis, and/or atopic dermatitis.

Epidemiology

The incidence of AD varies globally, ranging from 11% to 21% based on age and region. In a study of North Indian children, the average age of onset was 4.2 months for "infantile AD" and 4.1 years for "childhood AD," with

Table 1A: Strength of recommendations for atopic dermatitis interventions

Recommendation Strength	Suggested Wording	Implications
Strong recommendation for use	"We recommend…"	Benefits are significantly higher than risks; applies to most patients in common scenarios.
Strong recommendation against the use	"We recommend against…"	Risks substantially outweigh benefits; applies to most patients in common scenarios.
Good practice statement	"We recommend…"	High certainty in benefits over harms; based on significant indirect evidence and seen as essential for practice.
Conditional recommendation for use	"We conditionally recommend…"	Benefits and risks are closely balanced; and may vary based on patient or stakeholder values.
Conditional recommendation against the use	"We conditionally recommend against…"	Risks and benefits are closely balanced; and may vary based on patient or stakeholder values.

Table 1B: Certainty levels of evidence for atopic dermatitis guidelines

Evidence Quality	Suggested Wording (Certainty of evidence)	Interpretation
High	High	Strong confidence that the estimated effect is close to the actual effect.
Moderate	Moderate	Moderate confidence in the estimated effect; likely close but with some chance of substantial difference.
Low	Low	Limited confidence; the actual effect may differ significantly from the estimate.
Very Low	Very Low	The effect estimate is very uncertain; the actual effect could differ substantially from the estimate.

Table 1C: Classification of moisturisers[17]

Class	Mechanism of Action	Similarity to Natural Skin Components	Examples
Occlusive	Forms a barrier on the skin to reduce transepidermal water loss (TEWL) and protect against irritants.	Intercellular lipid layers, such as ceramides, cholesterol, and fatty acids	Beeswax, Lanolin, Mineral oil, Paraffin, Petrolatum, Propylene glycol, Silicones, Squalene
Humectants	Low-molecular-weight ingredients that draw moisture to the skin from deeper layers, boosting hydration.	Natural moisturising factors in the stratum corneum	Alpha hydroxy acids, Glycerin, Hyaluronic acid, Propylene glycol, Urea
Emollients	Helps fill gaps between skin cells in dry areas, enhancing skin smoothness, flexibility, and softness.	Natural skin lipids	Lauric acid, Linoleic acid, Linolenic acid, Oleic acid, Stearic acid

urban cases significantly outnumbering rural ones. Another hospital-based study found AD to be the most common skin condition in children at a paediatric dermatology clinic, with a prevalence of 29.9% and a male-to-female ratio of 2.25:1. The average onset age was 4.5 months. Infants showed more facial involvement and acute eczema, while older children had non-specific distribution and chronic forms of eczema, with most cases being mild to moderate. Seasonal worsening in winter was observed in 62% of cases. However, a lower prevalence of 0.42% was reported among dermatology outpatients in eastern India.[2]

Aetiology and pathogenesis of atopic dermatitis

AD is a multifactorial disease influenced by various genetic and environmental factors. It involves a breakdown in skin barrier function and hypersensitivity reactions, leading to a diversity of symptoms.

Genetic factors

Both genetic predisposition and environmental triggers influence AD onset and flares. Key genes include CTLA4, IL-18, and TLR9, with genome-wide studies identifying chromosomal regions linked to AD, particularly in Japanese populations.[3]

Immunological mechanism

In the acute phase, AD is driven by Th2-related cytokines like IL-4 and IL-13 and chemokines such as TARC and eotaxin. As AD becomes chronic, Th1 cells and their cytokines, including IFN-γ and IL-12, dominate. Langerhans cells and mast cells, expressing high-affinity IgE receptors (FcεRI), contribute to inflammation by releasing histamine and cytokines. Th2 cell cytokines, IL-4 and IL-13, also stimulate fibroblasts to produce periostin, which induces thymic stromal lymphopoietin (TSLP) production in keratinocytes. This, in turn, promotes TARC/CCL17 release by dendritic cells. In

eczematous lesions, antimicrobial peptides like defensins and cathelicidins are suppressed, compromising skin defense and allowing allergens to penetrate more easily.[4]

Immune responses and pruritus

Helper T cells differentiate into Th1 and Th2 cells, where Th2 cells play a central role in allergic responses. Th2-associated cytokines (IL-33, IL-25, TSLP) direct these cells to AD lesions, stimulating IgE antibody production. Langerhans and mast cells, with FcεRI receptors, release inflammatory mediators like histamine upon binding IgE, driving allergic inflammation. AD lesions release pruritogens (e.g. IL-31, IL-4, TSLP) that activate nerves, leading to intense itching and subsequent scratching. Chronic scratching worsens dermatitis, contributing to heightened skin sensitivity. Over time, nerve fibres extend closer to the skin surface, amplifying sensitivity to external stimuli and leading to hypersensitivity reactions. Imbalances in the autonomic nervous system, along with psychological factors, can further intensify itching and AD severity.[3,4]

Factors involved in onset and exacerbation

Various triggers contribute to AD onset and flares, including environmental exposures (allergens, irritants, temperature changes) and physiological factors affecting skin function. Common triggers include heat, sweating, wool fibres, stress, certain foods, alcohol, infections, and climate shifts. Scratching, microbes, and contact allergens further aggravate symptoms. Managing these factors is key to controlling AD.[5]

Course

AD follows a variable course, often chronic or recurrent. While spontaneous remission is possible, around 30% of affected children may continue to experience episodes into adulthood. Educating patients and parents about the long-term nature of AD is recommended.

Complications

Infections are common in AD, including bacterial, viral, and fungal infections. Rare complications may involve eye diseases (such as glaucoma, keratoconus, retinal detachment, and, in severe cases, blindness), alopecia areata, growth delays, and associated conditions like ichthyosis vulgaris.[5]

Characteristics of eruption

Infancy (<2 years): Begins as dry skin on the cheeks, forehead, or scalp, progressing to papules. Exudative erythema may appear in skin folds (neck, armpits, elbows, knees), with lesions also affecting the trunk and limbs.

Childhood (2–12 years): Facial eruptions subside, with lesions shifting to neck, armpits, elbow/knee creases, groin, wrists, and ankles. Severe cases may involve the face and limbs, with scratching leading to erosions, scabs, lichenification, and prurigo. The skin may develop a dry, goosebump-like texture.

Adolescence/Adulthood (≥13 years): Eruptions commonly appear on the face, neck, chest, and back. Severe cases may cause widespread prurigo and progress to erythroderma.

General Features: AD presents with acute and chronic lesions. Dry skin (xeroderma) worsens with dermatitis. Acute lesions show erythema, papules, vesicles, or erosions, while chronic scratching leads to lichenification and prurigo nodules.[5]

Diagnosis

Diagnostics criteria for atopic dermatitis

The diagnosis of AD is primarily clinical, relying on the patient's history, lesion morphology, distribution, and associated clinical signs. Several formal criteria have been developed to aid in accurate diagnosis.

Hanifin and Rajka criteria

Introduced in 1980, the Hanifin and Rajka criteria are one of the earliest and most widely recognised diagnostic tools for AD. Diagnosis requires the presence of at least three out of four major criteria and three out of 23 minor criteria.[5]

United Kingdom Working party criteria

The UK Working Party criteria include one mandatory criterion and five major criteria, making them straightforward and suitable for use without laboratory testing. While the original criteria were not designed for very young children, revisions have since adapted them for infants [see Table 2]. The Hanifin and Rajka criteria are used in 44% of clinical trials, while the UK criteria are used in 12%.[6]

American Academy of Dermatology criteria

In 2003, a consensus led by the American Academy of Dermatology proposed a streamlined version of the Hanifin and Rajka criteria, designed to be more user-friendly and applicable across all age groups.[7]

Disease severity scales for atopic dermatitis

More than 28 scales have been developed to measure the severity of AD, though none are considered the gold standard. The most widely used scales are the SCORAD index, Eczema Area and Severity Index (EASI), Patient-Oriented Eczema Measure, and the Six Area, Six Sign Atopic Dermatitis Severity Score.

SCORAD

The SCORAD index is an internationally recognised scale and one of the most commonly used in English-language literature. It combines both objective assessments by physicians (extent and severity) and subjective

Table 2: Classification of Topical Corticosteroids by Potency[1]

Class	Potency Level	Examples
Class I	Very Potent	Clobetasol propionate 0.05%
Class II	Potent	Beclometasone dipropionate 0.025%, Betamethasone valerate 0.1%, Betamethasone dipropionate 0.05%, Diflucortolone valerate 0.1%, Fluocinolone acetonide 0.025%, Hydrocortisone butyrate 0.1%, Mometasone furoate 0.1%, Triamcinolone acetonide 0.1%
Class III	Moderate	Alclometasone dipropionate 0.05%, Betamethasone valerate 0.025%, Clobetasone butyrate 0.05%, Fluocinolone acetonide 0.00625%, Fluocortolone 0.25%
Class IV	Mild	Hydrocortisone 0.1%-2.5%, Fluocinolone acetonide 0.0025%

assessments by patients (itch and sleep loss). The maximum SCORAD score is 103, and scores can be calculated using an online tool (http://adserver.sante.univ-nantes.fr/Scorad.html).

Eczema Area and Severity Index

EASI is an objective tool focussed solely on physician assessments of disease extent and severity in AD. Recommended by the Harmonising Outcome Measures for Eczema (HOME) initiative – an international group standardising AD research outcomes – EASI is widely used in clinical research. The EASI score chart and training resources are accessible online at HOME for Eczema.

Assessment of pruritus

Itching is a key symptom of AD, yet it is challenging to measure objectively. The visual analogue scale (VAS) and numeric rating scale (NRS) are often used for subjective patient assessment. In the VAS, patients mark a point on a 100-mm line from "no itch" (0) to "worst imaginable itch" (100), while the NRS asks patients to verbally rate itch from 0 to 10. Both scales are correlated with itching severity, although the SCORAD index is better suited to assess pruritus and insomnia due to itch.

Patient-reported severity

The Patient-Oriented Eczema Measure (POEM) is a questionnaire-based tool for patients or caregivers to assess AD severity. POEM allows patients and physicians to align on treatment goals and has shown a good correlation with physician assessments.

Quality of Life Assessment

AD can significantly impact patients' quality of life (QOL) due to itching, appearance concerns, and treatment demands. To ensure QOL-centered care, validated QOL questionnaires are used to gauge and address these impacts effectively.[7]

Differential diagnosis and associations

The differential diagnosis of AD is broad as it can share symptoms with several other dermatologic and systemic conditions. Key conditions to consider include:

- Contact Dermatitis: Often triggered by direct exposure to irritants or allergens, this condition can mimic AD but is generally limited to areas of contact and responds well to allergen avoidance.[8]
- Seborrheic Dermatitis: Characterised by oily, scaly patches, seborrheic dermatitis commonly affects the scalp, face, and chest. While it can coexist with AD, its presentation and distribution are distinct.
- Prurigo Simplex: Intensely itchy, isolated nodules; lacks widespread eczematous lesions seen in AD.
- Scabies: Nighttime itching with characteristic burrows (fingers, wrists); confirmed via microscopy.
- Miliaria (Heat Rash): Small, clear vesicles in hot, humid conditions; resolves with cooling, unlike AD.
- Ichthyosis: Dry, scaly skin affecting large areas; lacks AD's inflammation but commonly coexists with AD.
- Xerotic Eczema (Winter Itch): Dry, cracked skin in cold months; improves with emollients and lacks AD's inflammation.
- Non-Atopic Hand Dermatitis: Occupational/environmental irritant-induced; localised to hands, unlike AD.
- Cutaneous Lymphoma: Initially eczema-like, but progresses to plaques/nodules; biopsy confirms diagnosis.
- Psoriasis: Thick, silvery scales, well-defined plaques; often involves nails and joints, unlike AD.
- Immunodeficiency Diseases: (e.g., Wiskott-Aldrich, Hyper-IgE Syndrome) Mimic AD but present with systemic symptoms; diagnosed via immunologic tests.
- Collagen Vascular Diseases: (e.g., Lupus, Dermatomyositis) Rashes with systemic involvement; serological markers differentiate from AD.
- Netherton Syndrome: Rare genetic disorder with ichthyosis, AD-like inflammation, and hair abnormalities; diagnosed via genetic testing.

Each of these conditions has specific characteristics that help differentiate them from AD. Accurate diagnosis may require a combination of clinical evaluation, patient history, and, in some cases, laboratory or biopsy findings.

Diagnostic aids

About 80% of AD patients have elevated serum IgE, while 20% do not. AD is classified into extrinsic (high IgE) and intrinsic (normal IgE) types, though this remains debated. IgE elevation is often secondary to skin barrier damage, leading to sensitization. IgE antibodies against allergens like mites, foods, and pets are common but non-specific, occurring in 55% of the general population.

Mast cell and eosinophil elevations may be present but do not reliably indicate severity. Biomarkers like

CD30, TARC, and cytokines (IL-12, IL-16, IL-18, IL-31) have been explored, but none are specific or sensitive enough for routine use. No biomarkers are currently recommended for diagnosing or assessing AD severity. Routine IgE monitoring and allergy testing are unreliable due to high false-positive rates, and advanced tests like Phadiatop or ELISpot are more predictive when negative than positive.

Histopathology shows spongiosis and lymphocytic infiltration in acute AD, while chronic AD presents with hyperkeratosis and acanthosis. However, biopsies and atopy patch tests are not recommended for standard AD diagnosis.[9]

The atopy patch test is an epicutaneous test that evaluates delayed hypersensitivity reactions to allergens known to trigger IgE-mediated responses, with results assessed after 48–72 hours. It helps identify patients whose atopic flares are linked to extrinsic allergens, enabling targeted avoidance counselling. However, it should not be used in isolation but rather alongside clinical history, examination, skin prick tests, and serum-specific IgE for a comprehensive assessment in selected patients.[10]

Useful biomarkers for diagnosis and prognosis

High serum IgE levels are common in AD, often exceeding 500 IU/mL in severe cases. While elevated IgE suggests an allergic tendency rather than active disease, a gradual decline may indicate improvement. Specific IgE tests can detect sensitisation to allergens like dust, pollen, and food, but positive results do not always correlate with symptoms, making a detailed history essential.

Peripheral eosinophil count is often higher in AD than in other allergic conditions and tends to rise with disease severity, making it useful for monitoring progression. Serum LDH levels increase with inflammation in severe AD and usually normalise as lesions heal, though persistent elevation may signal complications.

TARC levels, which guide Th2 cell migration, strongly correlate with AD severity and are more reliable than IgE, LDH, or eosinophil counts. They can help track disease progression and treatment response, but results should be interpreted with caution in children under two as naturally higher levels may affect accuracy.[9]

Controlling Factors Responsible for Exacerbation Of AD

Food allergens

Evidence on specific food allergens and their association with AD remains mixed. In a review of eight studies, Werfel *et al*.[11] reported a 33%–63% prevalence of food allergies, confirmed through double-blind, placebo-controlled food challenges. Common triggers include milk, peanuts, eggs, soy, wheat, seafood, and shellfish. The consensus advises against dietary exclusions for AD management in patients without confirmed food allergies. Food allergies should only be diagnosed when symptoms consistently appear after specific food intake; broad allergy testing without a relevant clinical history is discouraged.

Clothing

To prevent skin irritation, coarse fabrics should be avoided, along with tight, heat-retentive clothing. Some research indicates that silver-coated fabrics may reduce *Staphylococcus aureus* colonisation and alleviate AD symptoms. Derma silk, known for its sericin-free, non-irritating, antibacterial qualities, has also been suggested. In the Indian context, the consensus recommends using non-irritant cotton fabrics.

Sweating

Sweating is a known trigger for AD exacerbations. Regular bathing to remove sweat can alleviate symptoms, and minimising exposure to high heat and humidity, whether through occupational or recreational activities, can help reduce flare-ups.

Environmental factors

Common allergens such as mites, dust, pollen, and chemicals like formaldehyde and toluene can exacerbate AD. Early sensitisation to mites, especially in infancy, has been linked to a higher risk of asthma. Pollen seasons can also lead to periocular symptoms.

Occupational considerations

For patients with AD, it is important to assess possible occupational triggers. Where possible, reducing exposure to known triggers or implementing preventive measures is recommended. If hand eczema occurs in adolescence, avoiding wet occupations is advised.

Perinatal Prevention

A study found that eliminating high-risk food allergens (such as egg and milk) from the diet of pregnant or lactating mothers does not prevent newborn sensitisation or AD development. However, a meta-analysis supports a moderate role for probiotics in reducing AD and IgE-associated AD in infants, regardless of whether probiotics are taken during pregnancy, infancy, or both.

General Care and Non-Prescription Management of AD

Bathing recommendations

Data on bathing for adults with AD are minimal. Bathing and showering may play a key role in managing AD by removing sweat, allergens like dust or pollen, and microbes from the skin's surface. This may help to reduce the risk of infection by clearing away dirt and debris. Swimming is best avoided during active flare-ups as chlorine exposure can weaken the skin

barrier and worsen AD symptoms. Daily, short baths in lukewarm water (27–30°C) for about 5–10 minutes are recommended, ideally during the day to maintain skin hydration without overheating or overcooling the skin.[1]

Bleach bath

Bleach baths may help prevent infections and control bacterial colonisation in AD, though studies primarily focus on children. A small study in adults found bleach baths safe and well-tolerated, with no adverse effects on skin hydration, water loss, or pH after a single 10-minute session. In an 8-week study, AD patients who took bleach baths twice weekly showed significant EASI score improvements after 1 month compared to those using distilled water. Based on limited evidence, bleach baths can be conditionally recommended for the treatment and maintenance of AD.[12]

Cleansing

The skin should be gently cleansed to remove crusts and bacterial contaminants, especially in cases of bacterial superinfection. Avoid vigorous scrubbing or rubbing after bathing. Instead, pat the skin dry with a soft towel. For AD, non-soap cleansers (such as Syndet) are recommended—these should be neutral to low pH, hypoallergenic, non-irritating, and free of fragrances to minimise irritation.[1]

Allergen immunotherapy

Previous guidelines suggest allergen immunotherapy (AIT) may benefit AD. A systematic review of 23 RCTs (1957 patients, median age 19, range 4–34 years) examined AIT, primarily for house dust mites (HDMs), often alongside standard topical treatments.

Results showed AIT reduced AD severity by ≥50% in 40% of patients, compared to 26% without AIT. Both subcutaneous (SCIT) and sublingual (SLIT) immunotherapies were similarly effective. Side effects were comparable to AIT for allergic rhinitis and asthma, with local reactions in 66% (SCIT) and oropharyngeal itching in 13% (SLIT). Serious reactions were rare, requiring discontinuation in ~ 10% of SCIT and <2% of SLIT cases. The panel concluded that AIT offers moderate benefits for moderate-to-severe AD, particularly in patients with other AIT-responsive allergies. However, patient preferences may vary due to the burden of SCIT (frequent clinic visits) and SLIT (daily self-administration), along with the time required for noticeable improvement.[13]

Elimination diets

Patients with AD have a higher risk of food allergies, but evidence suggests regular oral exposure may promote tolerance, while avoidance may increase IgE-mediated allergy risk.

A systematic review (10 RCTs, 599 participants) found low-certainty evidence that elimination diets slightly improve AD severity, itch, and sleep. 50% on elimination diets saw minimal improvement versus 41% without restrictions (9% risk difference). However, Bayesian analysis suggests most patients gain little to no benefit from elimination diets.

Our panel advises against elimination diets for AD management due to the uncertain benefits and potential harms, including an increased risk of developing food allergies. This risk is particularly concerning in infants and children, but it applies across all ages, where restrictive diets may also lead to malnutrition and impose significant burdens on patients and caregivers.[14]

Avoidance of perfumes, personal hygiene, and cosmetic products

Patients with AD should avoid products containing common allergens like fragrances, formaldehyde, lanolin, nickel, neomycin, parabens, and rubber chemicals. Choosing personal care items free from perfumes, solvents (e.g. formaldehyde), and preservatives (e.g. parabens) is recommended. Scented soaps and toiletries should be especially avoided to reduce irritation.[1]

Wet Wrap Therapy for AD management

Wet wrap therapy (WWT) effectively controls AD flares, especially in resistant cases. It involves applying a topical corticosteroid (TCS) or emollient, followed by a damp cotton layer and a dry outer layer, enhancing absorption, hydration, and reducing water loss. Typically used for 1 hour to a full day, prolonged use may increase steroid absorption risks. Studies, mainly in children, show TCS-based WWT is more effective than emollient-only wraps. Given limited adult data and mixed infection risk findings, WWT should be used cautiously in moderate-to-severe cases, with proper patient education.[1]

Patient and caregiver education in AD management

Educating patients and caregivers plays a vital role in managing AD. Effective education should cover the basics of AD in simple terms, including its causes, symptoms, progression, triggers, and ways to alleviate symptoms. It should also focus on self-care strategies and coping skills.

At each visit, education is recommended and should include:

a. Proper treatment dosages and application frequency
b. Guidance on adjusting treatment as symptoms improve or worsen
c. Skincare and bathing practices
d. Infection management.

Reinforcing this information at every consultation enhances AD management and empowers patients and caregivers for better outcomes.[15]

Psychological factors and psychosomatic interventions

Stress is a common trigger for AD flare-ups, and stress reduction may help in controlling symptoms. Psychotherapeutic and behavioural therapies can be beneficial for managing emotional triggers, such as the itch-scratch cycle, coexisting anxiety or depression, and reduced QOL. Recognising the role of psychological factors and incorporating psychosomatic support in AD management is recommended.[16]

Stepladder treatment in atopic dermatitis

Treatment for AD varies with disease severity, using topical or systemic therapies as appropriate. A stepwise approach is recommended, tailored to clinical severity. After achieving remission, transitioning to proactive maintenance therapy is advised to help prevent future flare-ups.[1]

Moisturisers

Moisturisers are essential in managing AD and reducing symptoms, inflammation, and flare frequency. By minimising water loss and improving hydration, they help restore the skin barrier. While moisturisers can be effective alone in mild cases, they are generally used alongside other treatments in more severe cases.

Studies on moisturisers, including trials with over 500 AD patients, show small but notable improvements in AD severity (measured by SCORAD and EASI scores). While most research supports moisturiser use, outcomes can vary based on product formulation and ingredients. For instance, one study found that a hyaluronic acid-based moisturiser improved EASI scores, while another showed no significant difference between a glycerol-based emollient and a placebo.

Moisturisers also aid in reducing itch. In one study, a lipopolysaccharide-containing cream significantly reduced itching compared to control after 4 weeks. While some formulations show benefits, the choice of specific ingredients or moisturiser types remains limited by inconsistent evidence [Table 1C].

Moisturisers are generally safe, with mild adverse effects. Skin irritation is uncommon, though patients should consider the allergenic potential of certain ingredients. Notably, regular application (at least twice daily) is recommended, with a suggested weekly use of over 250 g for adults. Applying moisturisers within 3 minutes post-bath enhances effectiveness.

Prescription emollient devices (PEDs), designed to target AD-related skin barrier issues, offer potential benefits but are often more costly. Limited trials suggest that PEDs may improve symptoms without significantly outperforming over-the-counter options.[16,17]

Anti-inflammatory ingredients in some moisturisers may reduce the need for topical corticosteroids (TCSs). Studies with natural ingredients like aloe vera, coconut oil, and shea butter have shown mild anti-inflammatory effects, offering the potential for steroid-sparing management.

In summary, consistent use of moisturisers is recommended for AD management, helping to maintain remission, prevent flares, and support overall skin health.

Topical Prescription Therapies For AD

Topical corticosteroids

TCSs are FDA-approved, first-line treatments for mild to severe AD, reducing inflammation by suppressing proinflammatory cytokines. They are classified into seven potency levels, with lower-potency steroids preferred for sensitive areas like the face, neck, and body folds.

Some dermatologists start with high-potency TCS for rapid control, while others prefer a gradual escalation approach. Over 100 trials confirm their effectiveness in reducing itch, controlling active disease, and preventing relapses, making them highly recommended [Table 2].

Standard application is twice daily, though some potent formulations work with once-daily use. While traditionally discontinued after flare control, recent studies support intermittent use (once or twice weekly) to extend remission periods.

High-potency TCSs are effective in managing severe AD and flare-ups. A study on betamethasone dipropionate over 3 weeks found a significant response, with 94.1% of treated patients showing good or excellent results (compared to 12.5% in the control) and an 86% improvement in severity scores versus 24.9% in controls.[18] Very high-potency TCSs, such as clobetasol propionate, fluocinonide, and halobetasol propionate, are also effective for severe AD flares. In three randomised trials, 67.2% of patients achieved "clear or almost clear" skin within 2 weeks compared to 22.3% in the control group (RR: 2.76).[19] Adverse effects were minimal, with fewer therapy discontinuations in the treatment group than in the controls.

Very high-potency steroids are typically limited to short courses due to the risk of skin atrophy, while medium-potency steroids, with a better safety profile, can be used for longer durations. Daily application of fluticasone propionate 0.05% lotion over 4 weeks achieved at least 50% lesion clearance in 70.6% of AD patients, with significant symptom improvement (RR 1.86).[20] Similarly, fluticasone propionate cream showed a marked reduction in symptom severity scores at 22 days.[20] In a study of 117 adult AD patients, those on intermittent fluticasone therapy were seven times less likely to relapse than the control group.[21] Based on strong evidence, intermittent use of medium-potency TCS twice weekly is recommended to

prevent AD flares and relapses. The consensus advises against indiscriminate use of topical corticosteroids and antibiotic combination.

The incidence of adverse events with TCSs is low. Common cutaneous side effects include purpura, telangiectasia, hypopigmentation, hypertrichosis, acneiform eruptions, and striae, with skin atrophy being the primary concern. Risk factors for atrophy include high-potency TCS, application on thin skin, occlusion, older age, and prolonged use. Allergic contact dermatitis to TCS or its ingredients can be confirmed with patch testing.[22,23]

Topical Steroid Addiction (TSA) and Withdrawal (TSW) are poorly defined in literature. Recent reviews (2021) found low to very low evidence for TSA/TSW, mainly linked to prolonged potent steroid use on sensitive areas like the face. Red face and red scrotum syndrome may also occur with extended TCS use.[24]

Non-cutaneous side effects are rare. While the link between TCS and cataracts/glaucoma is unclear, limiting periocular use is advised. Long-term, high-potency TCS over large areas may suppress the HPA axis, especially when combined with systemic corticosteroids, warranting cortisol level testing. Associations with type 2 diabetes and osteoporosis need further study.

Topical calcineurin inhibitors

Topical calcineurin inhibitors (TCIs) like pimecrolimus and tacrolimus have been approved since 2002 as anti-inflammatory options for AD, particularly useful for patients who may experience adverse effects from corticosteroids. They provide a safe and effective alternative, especially for long-term management. [Box 2]

In six randomised studies, pimecrolimus 1% cream significantly improved AD severity compared to vehicles, reducing symptoms such as itch within 1–6 weeks. A study of 198 patients found a substantial reduction in disease scores after just 7 days of pimecrolimus treatment, with improvements in itch for 81% of patients compared to 63% for those on placebo. Additionally, pimecrolimus can reduce the frequency of flares and corticosteroid use, showing a longer duration without the need for steroids in some trials.[25]

Tacrolimus ointment (0.1% and 0.03%) effectively manages adult AD, significantly reducing disease severity and itch in a 12-week trial (600 + patients). Used 2–3 times weekly, it helps control flares and maintain stability for up to a year. Tacrolimus 0.1% outperforms pimecrolimus 1%, achieving a 54.1% EASI score reduction versus 34.9% for pimecrolimus. However, tacrolimus is available only as an ointment, while pimecrolimus comes as a cream, preferred for mild AD or ointment-sensitive patients.[26]

Long-term proactive TCI use prevents relapses, offering sustained disease control without corticosteroid risks. Side effects are minimal, with localised burning resolving in a week. While TCIs carry an FDA black box warning for cancer risk, studies show no increased malignancy, even in children.

TCIs are strongly recommended for AD. Tacrolimus 0.1% is preferred for severe cases, while pimecrolimus 1% suits mild-to-moderate AD, offering a safe, effective alternative to corticosteroids.[25]

Box 2: Approval of Topical Calcineurin inhibitors for various age groups

Topical calcineurin inhibitors are approved for the following ages:

Pimecrolimus 1% cream: 2 years

Tacrolimus 0.03% ointment: 2 years

Tacrolimus 0.1% ointment: 16 years

Proactive therapy in AD

Proactive therapy helps maintain AD remission by applying topical steroids or tacrolimus (twice weekly) to flare-prone areas after acute treatment. Moisturisers support remission, while reactive therapy treats inflammation only during flare-ups. Even when inflammation appears resolved, residual inflammatory cells increase relapse risk. Proactive use of TCS or tacrolimus during this latent stage helps prevent recurrence but should begin only after full skin recovery (no itching, erythema, or raised areas) and guided by disease markers like TARC levels. Treatment must be individualised, adjusting dose, area, and duration while monitoring side effects. A physician should oversee proactive therapy, with daily moisturising essential for barrier maintenance and relapse prevention.[27]

Topical PDE-4 inhibitors

Crisaborole 2% ointment, a topical PDE-4 inhibitor approved by the FDA in 2016, is an alternative treatment for mild-to-moderate AD, particularly when corticosteroids (TCSs) or calcineurin inhibitors (TCIs) are unsuitable. In four randomised trials, crisaborole significantly improved AD symptoms compared to vehicle. In two large trials with 1016 patients on crisaborole and 506 on a vehicle for 28 days, 32.1% of crisaborole-treated patients achieved “clear” or “almost clear” skin by day 29, versus 21.7% in the vehicle group. Two Indian studies demonstrated that topical crisaborole is effective for mild-to-moderate AD, showing comparable efficacy to topical tacrolimus 0.1%.[28,29]

Crisaborole also reduced itching, as demonstrated in a study of 40 adults where treated lesions showed a greater reduction in itch scores compared to vehicle. The safety profile is favourable, with mild application-site reactions (such as burning or stinging) and a discontinuation rate similar to placebo.

Based on strong evidence, crisaborole is highly recommended for patients with mild-to-moderate AD.

Topical JAK inhibitors

Ruxolitinib 1.5% cream, FDA-approved in 2021, is indicated for short-term, non-continuous use in mild-to-moderate AD (age 12+), with a 60 g weekly limit and application on ≤20% of the body to minimise systemic absorption. Black box warnings include risks of serious infections, malignancies, cardiovascular events, and thrombosis. In two trials, 52.2% of ruxolitinib-treated adults achieved an IGA score of 0 or 1 (vs 11.1% placebo), with 52% experiencing a 4-point itch reduction over 8 weeks (vs 15.4% placebo). QOL scores improved by 73.2% (vs 19.7% placebo). Adverse events were rare, with mild site reactions.[30]

Delgocitinib, a pan-JAK inhibitor, reduces inflammation and enhances skin barrier proteins like filaggrin. In animal models, it significantly reduced dermatitis severity. A phase II study showed mEASI and IGA improvement within 4 weeks, with itch relief by day 1.[31] A long-term Japanese study (ages 16+) using delgocitinib 0.5% ointment for moderate-to-severe AD showed sustained mEASI improvement over 24 weeks. Nakagawa *et al*.[32] confirmed efficacy with marked reductions in mEASI and itch severity (NRS), and minimal adverse effects (nasopharyngitis, mild skin reactions).

Tofacitinib 2% cream in a 4-week phase 2a trial showed an 81.7% EASI reduction (vs 29.9% placebo), though 17% reported side effects (vs 9% placebo), mainly upper respiratory infections. Another study reported a 24.3% SCORAD reduction over 21 weeks in treatment-resistant AD with no major side effects.[33]

Moderate-certainty evidence supports topical JAK inhibitors for AD management, though long-term safety data are needed for future guidance updates.[34,35]

Topical antimicrobials/antiseptics and antihistamines

Antimicrobials are needed for infected AD but have limited benefit in uninfected cases. Studies on endolysin, ciclopirox olamine, sertaconazole, and hypericum showed no significant SCORAD or EASI improvement over placebo, with small samples and short durations. A double-blind trial found sertaconazole 2% cream ineffective for chronic itch.

Antiseptics like triclosan initially reduced AD severity, but benefits were not sustained. Triclocarban 1.5% soap improved lesion severity in 6 weeks but raised concerns about resistance and skin flora disruption.[36]

Topical antihistamine doxepin reduced itch by 68.6% versus 54.6% (controls) but caused drowsiness, allergic reactions, and higher withdrawal rates (12.1% vs 2.2%).[37]

Due to low-certainty evidence and risks of resistance, skin pH changes, and sensitisation, this guideline conditionally advises against topical antimicrobials, antiseptics, and antihistamines for uninfected AD.

Phototherapy for AD

Phototherapy is an established treatment for AD and psoriasis, though high-quality RCTs on AD are limited. A Cochrane review (32 trials, 1219 participants) examined NBUVB (313 nm), UVA1 (340–400 nm), and broadband UVB (290–320 nm) but lacked consistent outcome measures, making meta-analysis unfeasible. Expert consensus supports NBUVB for moderate-to-severe AD, especially when systemic treatments are contraindicated.[38]

A conditional recommendation is made for NBUVB, the most widely used and safer option, but insufficient evidence supports PUVA. Phototherapy risks include burning, heat intolerance, and long-term skin cancer risk, particularly with PUVA. While PUVA's cancer risk is well-documented, it is less concerning for other UV modalities.

Accessibility remains a major limitation, requiring 2–3 sessions per week for 10–14 weeks, often in medical settings, making it time-consuming and costly. Home UVB units could improve access, but safety and efficacy data for AD are lacking.

Phototherapy can be used alone or with topical treatments like emollients and steroids. While UVB and UVA1 are effective, side effects include skin irritation, burning, and pruritus, with rarer risks of nonmelanoma skin cancer (especially with PUVA), lentigines, and HSV reactivation. Though no standardised NBUVB protocol exists, it remains the most effective and cost-efficient phototherapy for chronic AD and is safe for children and adolescents, whereas PUVA carries higher cancer risks in children[38] [Box 3].

Box 3: Guidelines for narrowband UVB according to skin type

Skin type	Initial UVB dose	Dose increment after each treatment	Maximum dose
I	130 mJ/cm^2	15 mJ/cm^2	2000 mJ/cm^2
II	220 mJ/cm^2	25 mJ/cm^2	2000 mJ/cm^2
III	260 mJ/cm^2	40 mJ/cm^2	3000 mJ/cm^2
IV	330 mJ/cm^2	45 mJ/cm^2	3000 mJ/cm^2
V	350 mJ/cm^2	60 mJ/cm^2	5000 mJ/cm^2
VI	400 mJ/cm^2	65 mJ/cm^2	5000 mJ/cm^2

Systemic Therapies for AD

Oral antihistamines

Antihistamines, used alongside TCS, tacrolimus, and moisturisers, reduced itch in 75% of 26 RCTs, with some trials also showing lower TCS use, reduced potency, and improved inflammatory markers (sIL-2R, TARC). They are recommended as supportive therapy but lack evidence

for standalone use in AD, making combination therapy essential.

First-generation (sedative) antihistamines have stronger anticholinergic effects, causing drowsiness and impaired concentration, while second-generation (non-sedative) options are preferred due to minimal central effects (H1 receptor occupancy <30%).

Precautions apply to certain antihistamines. Ketotifen is contraindicated in epilepsy, while clemastine, hydroxyzine, cetirizine, and levocetirizine require caution in convulsive disorders. Chlorpheniramine, cyproheptadine, and loratadine have been linked to seizure risks in children, necessitating careful monitoring.[39]

Systemic corticosteroids

Systemic corticosteroids (CSs) can improve AD symptoms but are generally discouraged due to significant side effects and rebound flares. They should only be used short-term as a bridge to sustainable therapies, with long-term use not advised. No high-quality long-term RCTs support their efficacy and safety, particularly in children, and existing studies are limited. For instance, two low-quality paediatric RCTs excluded methylprednisolone, a commonly used CS.

The International Eczema Council (IEC) discourages routine CS use in AD, reserving them for rare, severe cases or acute flares. A short course may help in acute flares, but long-term use, especially in children, is not recommended due to risks like growth suppression. Gradual tapering is essential to prevent rebound when transitioning to a steroid-sparing agent.[40]

Though widely prescribed for moderate-to-severe AD, systemic CSs carry high risks. Evidence remains low certainty, with a trial comparing prednisolone to cyclosporine halted early due to rebound flares in the prednisolone group. Given the risks, systemic CSs are not recommended for AD management, but in rare cases—when no alternatives exist—they may be used temporarily.

Systemic antibiotics

We advise against the routine use of topical or oral antibiotics and discourage the use of combined steroid-antibiotic creams. Secondary infection should be considered in patients with moderate to severe eczema who show symptoms such as weeping dermatitis, folliculitis, clear signs of infection, or lack of response to initial topical treatments. For localised infections, topical antibiotics may be appropriate, while systemic antibiotics should be used based on the patient's overall clinical condition. Preventive application of topical antibiotics is not recommended. Antibiotics, whether topical or systemic, should only be used in short courses when there is clear evidence of infection. Long-term use is discouraged to minimise the risk of bacterial resistance and sensitisation.[1]

Cyclosporine

Cyclosporine (CsA) is an oral calcineurin inhibitor that suppresses T-cell activation, reducing IL-2 and other cytokines to control AD inflammation. Approved in Europe, Australia, and Japan, it is a second-line treatment for severe AD unresponsive to conventional therapies. Both short-term and long-term studies in adults and children demonstrate that CsA is effective. Both short- and long-term studies confirm CsA's efficacy, particularly for acute flares. RCTs using 3–5 mg/kg/day show significant symptom improvement within 6 weeks. It is recommended for adults and children (2+ years) and can be used continuously for up to 2 years. CsA is typically prescribed for patients 16+, with 3–5 mg/kg/day in two doses. Both low (3 mg/kg/day) and high (5 mg/kg/day) doses yield similar benefits within 2 weeks. Shorter courses are often preferred to limit adverse effects.[41]

CsA use carries a risk of significant adverse effects, and close monitoring is essential, particularly for renal health. Known side effects include irreversible nephrotoxicity, infection, hypertension, electrolyte imbalances, dyslipidemia, tremors, hypertrichosis, headache, gingival hyperplasia, and increased risk for non-melanoma skin cancers. Kidney function must be monitored closely as serum creatinine elevations >30% for 2 months can lead to irreversible damage. Blood pressure and lipid levels should also be tracked throughout treatment.

CsA has shown efficacy in treating AD in children, similar to its effects in adults. Studies support both continuous long-term dosing (up to 1 year) and shorter, intermittent courses of 3 to 6 months for paediatric patients. However, CsA is only recommended for children over the age of two, and careful consideration of the long-term risks, especially regarding growth and kidney function, is crucial. Currently, no definitive data exist on the safety of prolonged CsA use in children, so practitioners must proceed with caution, especially in cases requiring extended therapy.

CsA is particularly beneficial for severe AD cases in adults who do not respond to conventional treatments, especially when the condition involves 30% or more of the body surface area with marked inflammation. An initial dose of 3 mg/kg/day is typically recommended, and treatment courses usually range from 8 to 12 weeks. During CsA therapy, it is essential to monitor for nephrotoxicity, hypertension, and infections, given the potential for serious adverse events. Due to uncertainties around long-term safety, transitioning to topical therapies once symptom control is achieved is advised. For cases requiring prolonged management, intermittent dosing with extended breaks is recommended to reduce cumulative toxicity.

CsA has shown greater effectiveness than methotrexate in the first 16 weeks, with similar long-term outcomes. Azathioprine and methotrexate demonstrated comparable efficacy over 12 weeks. A network meta-analysis suggests CsA (3–5 mg/kg/day) may be more effective than both, though small sample sizes create some uncertainty. Despite this, CsA remains a preferred option for severe AD, offering rapid onset and strong immunosuppression.[42]

Monoclonal Antibodies (Biologics) Therapies: Dupilumab and Tralokinumab

Two FDA-approved biologics, dupilumab and tralokinumab, have demonstrated efficacy and safety in managing moderate-to-severe AD in adults and are recommended for patients who have not achieved adequate control with conventional treatments. Only dupilumab is currently approved in India and available in selected patient scenarios.

Dupilumab

Dupilumab, the first FDA-approved biologic for AD, is a monoclonal antibody that specifically targets the interleukin-4 receptor alpha (IL-4Rα), inhibiting the signaling pathways of both IL-4 and IL-13. These cytokines are key mediators of type 2 (Th2) immune responses, which play a central role in the pathophysiology of AD, leading to inflammation, skin barrier dysfunction, and chronic itching. By blocking IL-4Rα, dupilumab reduces inflammation and itching, addressing the core mechanisms of AD.

Dupilumab has shown significant efficacy in improving AD symptoms, reducing pruritus, and enhancing QOL across various clinical trials. In large-scale randomised controlled trials (RCTs), dupilumab demonstrated marked improvements in the EASI and Investigator's Global Assessment (IGA) scores. In a pivotal 52-week study, patients receiving dupilumab experienced sustained improvements in AD signs and symptoms, including reductions in skin redness, lichenification, and sleep disturbances.

Dupilumab has also been compared in short-term RCTs with emerging therapies, such as JAK inhibitors like abrocitinib and upadacitinib. Although these JAK inhibitors may have slightly higher efficacy in certain dosing regimens, dupilumab remains a preferred first-line biologic due to its robust safety profile. Given its extended clinical use and data from over 5 years of practice, dupilumab has established itself as a cornerstone in systemic AD therapy.[43]

Dupilumab is administered subcutaneously, starting with a loading dose of 600 mg, followed by a maintenance dose of 300 mg every 2 weeks. The FDA has approved its use for individuals 6 months and older, making it a versatile option for a wide range of patients. The self-administration aspect allows for ease of use and empowers patients to manage their treatment independently.

Dupilumab's safety profile is one of its strongest attributes, with low incidence rates of serious adverse effects. Common side effects include mild injection-site reactions and conjunctivitis. Conjunctivitis is typically self-limiting, though more persistent cases may benefit from artificial tears or ophthalmologic referral.[44,45] The lack of significant systemic side effects, along with no need for baseline or ongoing laboratory monitoring, further underscores dupilumab's safety.

Tralokinumab

Tralokinumab, a monoclonal antibody targeting interleukin-13 (IL-13), is the second FDA-approved biologic for adult AD. By selectively inhibiting IL-13, tralokinumab modulates type 2 immune responses linked to skin inflammation, itching, and barrier issues, providing a targeted approach in AD treatment.

Clinical trials confirm tralokinumab's effectiveness in reducing AD severity and enhancing QOL metrics, such as pruritus and sleep. Phase 3 studies showed significant improvement in EASI, IGA, and DLQI scores compared to placebo, with efficacy comparable to but slightly lower than dupilumab after 16 weeks.

Administered as a 600 mg initial dose followed by 300 mg biweekly, tralokinumab is approved for those aged 12 and older. Its at-home administration allows patients to integrate treatment into their routines, enhancing accessibility.[46]

Tralokinumab's safety profile is strong, with common adverse events like injection-site reactions and conjunctivitis, though studies indicate a lower incidence of conjunctivitis compared to dupilumab. Overall, tralokinumab is well tolerated, making it a valuable first-line biologic option for AD. Tralokinumab is currently unavailable in India.[47]

Recommendations and clinical scenarios

Dupilumab and tralokinumab can cause conjunctivitis, usually mild and manageable with artificial tears. Severe or persistent cases should be evaluated by an ophthalmologist, especially with vision changes or severe redness.

Dupilumab is the first-line biologic for moderate-to-severe AD unresponsive to topicals, supported by high-certainty evidence of efficacy, safety, and minimal systemic side effects. While cost and access may be barriers, its broad prescribing flexibility allows timely intervention, particularly in patients with comorbid atopic or systemic conditions.

Both dupilumab and tralokinumab are safe for older adults and those with renal/liver disease, HIV, or cancer

history. Experts favour dupilumab as a first-line systemic option for these groups due to its proven safety and added benefit in conditions like asthma, eosinophilic esophagitis, and chronic sinusitis.

Dupilumab is approved for ages 6+ months, making it essential for young children with severe AD, while tralokinumab is for ages 12+. These biologics revolutionise AD treatment by targeting immune pathways, with dupilumab as the preferred first-line choice and tralokinumab as a valuable alternative for patients needing a different approach.

Systemic JAK Inhibitors

JAK inhibitors work by blocking the JAK-STAT pathway, which mediates the inflammatory response in AD and other immune-driven conditions. Approved for moderate-to-severe AD in patients unresponsive to other treatments, selective JAK inhibitors, particularly abrocitinib, present new options for AD patients in India.

Abrocitinib is the only selective JAK inhibitor available and approved in India currently, providing a targeted approach by selectively inhibiting JAK1, which plays a role in the type-2 immune pathway relevant to AD. In clinical trials, abrocitinib (at doses of 100 mg and 200 mg) significantly improved symptoms in moderate-to-severe AD. In a study with 267 participants, abrocitinib led to rapid onset of action with improvements in skin clearance, itch reduction, and QOL, achieving strong patient response with minimal adverse effects. The JADE MONO trials reported side effects such as nausea, nasopharyngitis, and headache, but these were generally mild. One cardiac-related death occurred in the 100 mg group, though this was deemed unrelated to the treatment. Abrocitinib's main adverse effects include potential changes in platelet counts and lipid levels, which are typically managed through periodic monitoring, especially during the first 4 weeks.

Upadacitinib, originally for rheumatoid arthritis, selectively targets JAK1 and has shown high efficacy in AD. In trials, 30 mg daily led to 90% EASI improvement in 50% of patients by week 16, with strong responses across dosages in symptom reduction, itch relief, and QOL. Common side effects include acne, AD flare-ups, and upper respiratory infections.

Baricitinib, a JAK1/2 inhibitor, is approved in Europe for AD. Phase III trials (BREEZE-AD1/AD2) showed significant EASI and QOL improvements with 4 mg and 2 mg doses, with mild side effects like nasopharyngitis and elevated creatine kinase. Baricitinib is available in India.[48]

Some recent literature from India suggests oral Tofacitinib, which is a non-selective JAK inhibitor, can be used in select cases of AD where other treatment options have failed or are unavailable. However, it should be kept in mind that tofacitinib is not approved for AD, and being a non-selective JAK inhibitor has a higher potential for adverse drug events.[49]

While JAK inhibitors show promise in AD management, regulatory bodies such as the FDA caution against potential risks, including cardiovascular events, cancer, and serious infections. As a result, they recommend starting with the lowest effective dose, particularly for older adults. Additional precautions, such as pre-treatment vaccinations for herpes zoster and monitoring of liver enzymes, blood counts, and lipids, are recommended to mitigate these risks.

Alternative Systemic Therapies for Atopic Dermatitis

Methotrexate

For moderate-to-severe AD unresponsive to topicals, systemic treatments, and biologics, the panel conditionally advises against methotrexate due to low-certainty evidence. While it provides modest symptom relief, its slow onset and significant risks—including hepatic dysfunction, bone marrow suppression, infections, and GI issues—often outweigh benefits. Regular blood monitoring is required, and it is contraindicated in pregnancy. Though cost-effective, its uncertain efficacy and safety concerns make safer, more effective alternatives preferable.[42]

Mycophenolate mofetil

For patients with moderate-to-severe AD who have not found relief with high-potency topical or systemic treatments, the panel conditionally advises against using mycophenolate mofetil due to low-certainty evidence. Mycophenolate offers only modest potential benefits in reducing AD severity, with uncertain outcomes and associated risks, including increased chances of cancer and serious infections. Its use requires baseline and routine blood monitoring and is contraindicated in pregnancy. Patients who prioritise avoiding possible harms and prefer more established treatments may favour other options. However, individuals with coexisting rheumatologic or autoimmune conditions may consider mycophenolate beneficial for addressing multiple conditions.[42]

Apremilast

Apremilast, an oral phosphodiesterase-4 inhibitor approved by the FDA in September 2014 for moderate-to-severe plaque psoriasis, has gained interest as a potential treatment for AD. Its anti-inflammatory properties, convenient oral administration, and generally mild side effect profile make it a viable option. However, studies on its effectiveness for moderate-to-severe AD

have produced mixed results, with some open-label trials reporting moderate efficacy, while others indicate limited benefits.[42,50]

Azathioprine

Azathioprine (AZA) may be used off-label for adult AD patients unresponsive to or intolerant of cyclosporine (CsA). Placebo-controlled studies show AZA improves AD symptoms and QOL. Thiopurine methyltransferase (TPMT) levels should be tested before starting as dosing (1–3 mg/kg/day) depends on TPMT activity. Common side effects include GI issues (nausea, vomiting), headache, hypersensitivity, liver enzyme elevation, and leukopenia. While AZA is supported in paediatric AD, optimal dosing and duration remain unclear.[42]

Adjunctive Treatment for Atopic Dermatitis

Alitretinoin

Alitretinoin, also referred to as 9-cis-retinoic acid, is a newly developed retinoid derivative. It is primarily used in the treatment of chronic hyperkeratotic atopic hand eczema, showing efficacy in managing this condition.[51]

Probiotics/Prebiotics

Probiotics, such as Lactobacillus alone or in combination with Bifidobacterium, have demonstrated a protective role in preventing AD when administered during the pre- and postnatal periods. However, there is insufficient evidence to suggest any benefit of probiotics in preventing AD in infants.[52]

Essential fatty acids

Dietary supplementation with evening primrose oil or omega-3 fatty acids (e.g. docosahexaenoic acid) is considered safe and may offer some benefit in managing AD. Nevertheless, there is a lack of sufficient RCT data to confirm the clinical efficacy of these supplements, preventing them from being universally recommended for AD treatment.[53]

Vitamin D

RCTs on the therapeutic use of vitamin D supplementation in AD have yielded inconsistent results. A meta-analysis indicated that patients with AD tend to have lower serum vitamin D levels, and supplementation may represent a potential therapeutic option. Vitamin D could play an important role in improving the symptoms of AD, with some studies suggesting that it may help reduce the severity of the condition. As a result, vitamin D supplementation is considered a safe and well-tolerated treatment option.[53]

Strategies For Maintenance Therapy in Atopic Dermatitis

Proactive maintenance management of AD aims to prevent disease flare-ups and improve long-term outcomes by addressing persistent subclinical inflammation in clinically unaffected skin. This approach contrasts with reactive treatment, which typically involves corticosteroids or calcineurin inhibitors during active flare-ups.

Several studies support the efficacy of proactive therapy in preventing relapses. A meta-analysis by Schmitt *et al*. (2011)[54] showed that topical corticosteroids (fluticasone propionate) applied twice weekly were significantly more effective in maintaining remission compared to tacrolimus ointment. Similarly, a 2022 Cochrane review revealed that proactive treatment with topical corticosteroids reduced relapse rates from 58% to 25% in both adults and children, with no significant side effects such as skin atrophy or abnormal cortisol levels.[55]

Proactive therapy involves regular use of topical corticosteroids or tacrolimus (1–3 times weekly) to control subclinical inflammation, maintain skin integrity, and prevent flares, even when skin appears clear. Corticosteroids may be more effective, while tacrolimus better preserves barrier function and hydration [Table 3A-E] [Figure 1].

Special Recommendation for Children

AD differs across age groups in presentation and progression. Infants develop pruritic, exudative lesions on the cheeks, scalp, and limb extensors, while older children show flexural involvement (elbows, popliteal areas, neck). Adolescents and adults often present with hand and foot eczema, with lichenification becoming more prominent due to chronicity. AD in children features exudative lesions, perifollicular accentuation, pityriasis alba, and seborrheic-like patterns, whereas adults experience xerosis, thicker lesions, and stronger emotional triggers. Food allergies, particularly to cow's milk and eggs, are common AD triggers in children but often resolve with age. Adults, however, are more prone to respiratory allergies (rhinitis, asthma), progressing through the "atopic march." Routine allergy testing is not recommended in children unless strongly indicated as elevated IgE or allergen-specific antibodies are often clinically irrelevant. Managing food allergies in children, especially resolving cow's milk allergy, may help reduce AD severity over time.

Managing AD in children requires caution due to higher topical absorption from thinner skin and a higher surface area-to-body weight ratio. Potent corticosteroids are used sparingly, especially in children under two, to prevent adrenal suppression and skin thinning. Topical calcineurin inhibitors (TCIs) like tacrolimus and pimecrolimus are safer but have age-specific approvals. Systemic therapies, including cyclosporine, are generally avoided in children until vaccinations are

Table 3A: Consensus statements for general management of AD

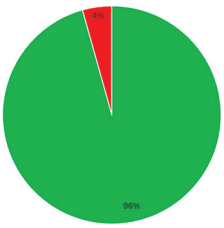
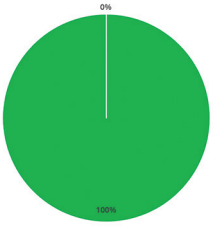
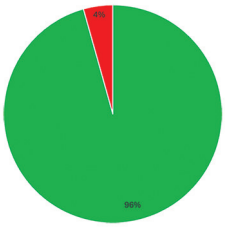
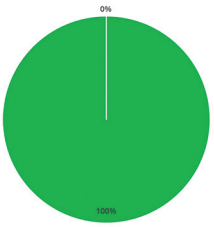
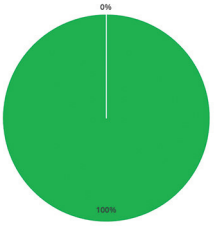
Intervention	Regulatory Status in India	Recommendation	Strength of Recommendations	Certainty of evidence
Recommendation for a specific diet in patients with AD	Not Applicable	While food allergens may contribute to eczema, empirical food restriction is not recommended in patients with AD. A specific food-free diet should only be considered when an allergy to a particular food trigger is confirmed through proper diagnostic procedures, such as a food diary and allergy tests conducted by a specialist. In infants and toddlers, dietary restrictions should be based on clinical experience and a clear food allergy diagnosis, rather than on general assumptions about food allergens.	Strong  [95.7%]	Moderate
Recommendation for Clothing in patients with AD	Not Applicable	Patients with AD should wear smooth, loose-fitting garments and avoid irritating fabrics and fibers. Woolen, acrylic, and nylon fabrics should be avoided as they can exacerbate symptoms. Cotton is the recommended fabric for its softness and breathability, providing the most comfort for individuals with AD.	Strong  [100%]	Low
Recommendation for Bathing in patients with AD	Not Applicable	Patients with AD should bathe once daily using lukewarm water (27°C to 30°C), ensuring the water is neither too hot nor too cold. The bath should be of short duration, ideally 5 to 10 minutes, and is best performed during the daytime to help maintain skin hydration and minimise irritation.	Strong  [95.7%]	Low
Recommendation for use of Syndet cleansers in patients with AD	Not Applicable	The use of non-soap cleansers (e.g., Syndet) that are hypoallergenic, non-irritating, and fragrance-free and maintain a neutral to low pH is recommended for managing AD	Strong  [100%]	Moderate
Recommendation for the avoidance of perfumes, personal hygiene, and cosmetic products in patients with AD	Not Applicable	It is recommended that individuals with AD avoid perfumes, personal hygiene products, and cosmetics containing solvents like formaldehyde and preservatives such as parabens. Additionally, perfumed soaps and other toiletries should be avoided to prevent skin irritation.	Strong  [100%]	Moderate

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Table 3A: Contd...

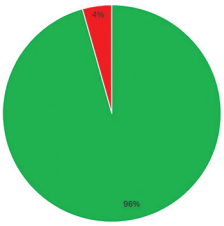
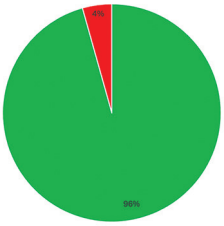
Intervention	Regulatory Status in India	Recommendation	Strength of Recommendations	Certainty of evidence
Recommendation for the Patient or Caregiver Education for Managing AD	Not Applicable	Patient or caregiver education is essential at every consultation for effective AD management. Key topics should include appropriate treatment doses and frequency, guidance on adjusting treatment, skincare and bathing practices, and infection management. This education should be reinforced at each visit to improve treatment outcomes.	Strong  [95.7%]	Moderate
Recommendation for Psychosomatic Interventions in AD Management	Not Applicable	Psychological and psychosomatic interventions are beneficial and recommended (when available) as part of the management plan for AD.	Strong  [95.7%]	Strong

Table 3B: Consensus statements for general principals of managements of AD

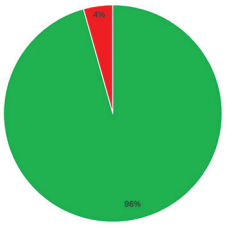
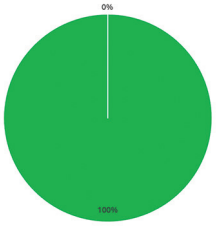
Intervention	Regulatory Status in India	Recommendation	Strength of Recommendations	Certainty of evidence
Recommendation for Disease assessments	Not Applicable	AD disease severity should be assessed through clinical signs, symptom severity, and QOL impact. Outcome measures like SCORAD, EASI, DLQI, NRS, and POEM complement the dermatological exam to monitor disease activity and guide therapy.	Strong  [95.7%]	Moderate
Recommendation for the goal of the treatment	Not Applicable	The goal of AD treatment is to achieve disease control, relieve symptoms, and enhance the patient's quality of life.	Strong  [100%]	Moderate

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Table 3B: Contd...

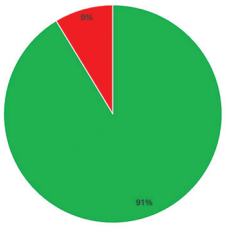
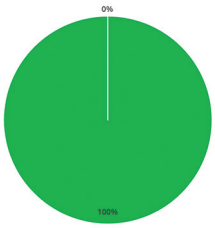
Intervention	Regulatory Status in India	Recommendation	Strength of Recommendations	Certainty of evidence
Recommendation for the target of the treatment for moderate to severe AD	Not Applicable	Key initial treatment targets for moderate-to-severe AD include a 50% reduction in SCORAD (SCORAD-50), EASI (EASI-50), at least 4 points in DLQI, 3 points in NRS, or 4 points in POEM within 3 months of treatment initiation.	Strong  [91.3%]	Moderate
Recommendation for referral before initiation of systemic therapies for moderate-to-severe AD	Not Applicable	It is recommended that physicians and paediatricians refer patients to a dermatologist to determine when to initiate systemic therapies for moderate-to-severe AD, due to the risk of misdiagnosis and potential adverse reactions.	Strong  [100%]	Not Applicable

Table 3C: Consensus statements for topical managements of AD

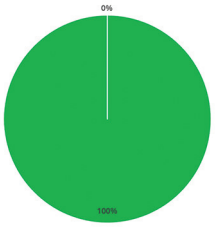
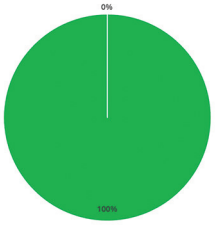
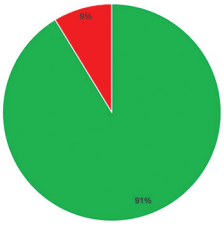
Intervention	Regulatory Status in India	Recommendation	Strength of Recommendations	Certainty of evidence
Recommendation on the use of moisturisers in AD	On Label	For patients with AD, we recommend regular use of moisturisers as a key component of overall disease management.	Strong  [100%]	High
	On Label	Consistent use of moisturizers has both short- and long-term steroid-sparing effects and helps reduce the frequency of acute flare-ups.	Strong  [100%]	High
	On Label	We suggest moisturisers be applied at least twice daily and more frequently during acute flare-ups.	Strong  [91.3%]	Low

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Table 3C: Contd...

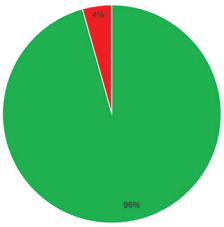
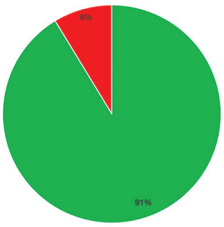
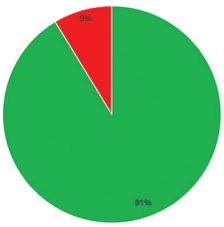
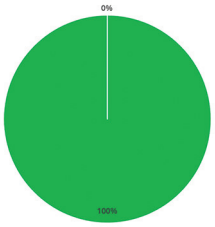
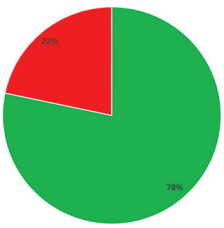
Intervention	Regulatory Status in India	Recommendation	Strength of Recommendations	Certainty of evidence
Recommendation on the use of topical corticosteroids in AD	On Label	We recommend the use of topical corticosteroids for patients with AD.	Strong  [95.7%]	High
	On Label	For maintenance therapy, we recommend the intermittent use of medium-potency topical corticosteroids (twice a week) proactively to help to reduce disease flares and prevent relapse.	Strong  [91.3%]	Moderate
Recommendation on the use of topical calcineurin inhibitors in AD	On Label	We recommend the use of tacrolimus 0.03% or 0.1% ointment and pimecrolimus 1% cream for patients with AD. Topical calcineurin inhibitors (TCIs) can be considered as first-line therapy, in conjunction with the appropriate use of moisturising agents.	Strong  [91.3%]	High
	On Label	During maintenance treatment, TCIs can be applied twice weekly to "hotspots" as a proactive management strategy.	Strong  [100%]	Moderate
Recommendation on the use of Topical PDE-4 inhibitors in AD	On Label	We recommend the use of crisaborole ointment for patients with mild-to-moderate AD.	Conditional  [78.3]	High

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Table 3C: Contd...

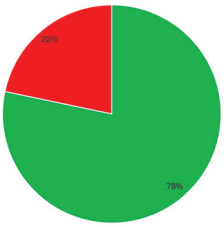
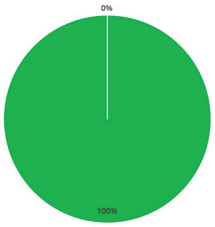
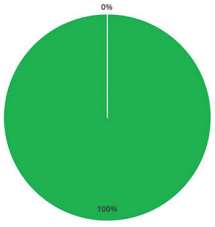
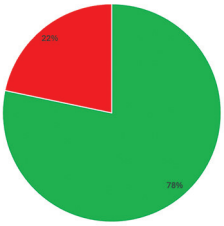
Intervention	Regulatory Status in India	Recommendation	Strength of Recommendations	Certainty of evidence
	On Label	Topical phosphodiesterase-4 inhibitors, such as crisaborole, should be considered, especially for sensitive areas (e.g., neck, eyelids, and genital areas), where the use of topical corticosteroids may lead to adverse effects.	Conditional  [78.3]	High
Recommendation on the use of Topical tofacitinib cream in AD	Off label	We suggest use of topical tofacitinib for patients with mild to moderate AD as a steroid sparing agent, particularly for sensitive areas (e.g. neck, genital area, face)	Strong  [100%]	Low
Recommendation on the use of Wet-wrap therapy in AD	Off label	Wet-wrap therapy with diluted corticosteroids or emollients can be considered for patients with moderate to severe AD, provided there is no risk of infection, to achieve a rapid reduction in AD severity.	Strong  [100%]	Moderate
Recommendation on the use of bleach baths in AD		We conditionally recommended bleach baths for the treatment and maintenance of AD.	Conditional  [78.3]	Low

Table 3D: Consensus Statements for Managements of AD with phototherapy and conventional systemic therapies

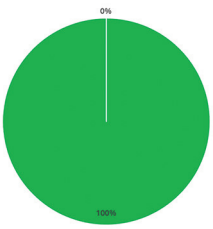
Intervention	Regulatory Status in India	Recommendation	Strength of Recommendations	Certainty of evidence
Recommendation for use of systemic corticosteroids in AD	Off Label	We strongly advise against prolonged use of systemic corticosteroids due to the unfavourable risk/benefit ratio in AD treatment.	Strong  [100%]	High

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Table 3D: Contd...

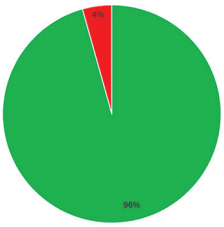
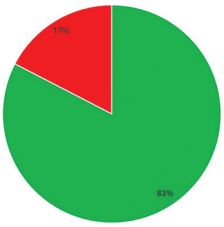
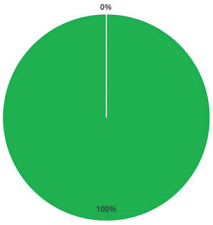
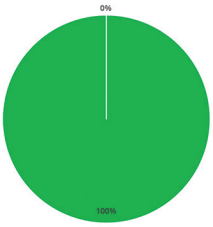
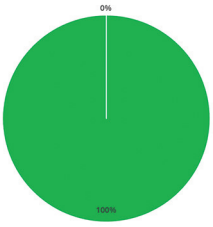
Intervention	Regulatory Status in India	Recommendation	Strength of Recommendations	Certainty of evidence
	Off Label	We conditionally recommend systemic corticosteroids be considered only as rescue therapy for acute flares of AD, and not for long-term use in chronic AD. As a rescue therapy, an initial dose of 0.5 mg prednisolone equivalent per kg/day is recommended, followed by a slow taper over 2–3 weeks to minimise the risk of rebound.	Strong  [95.7%]	Low
Recommendation of use of antihistamines in AD	Off Label	The use of antihistamines is conditionally recommended to control pruritus in AD as an adjunct to anti-inflammatory topical therapy. Antihistamines alone are not recommended due to a lack of reliable evidence for their efficacy in treating AD. Non-sedative second-generation antihistamines should be preferred as there is no difference in efficacy between sedative and non-sedative options.	Conditional  82.6%	Low
Recommendation of use of topical and oral antibiotics in AD	Off Label	We strongly recommend against the preventive use of topical antibiotics. Topical and systemic antibiotics should only be used for short courses with clinical evidence of infection.	Strong  [100%]	Moderate
Recommendation for the use of Phototherapy in AD	On Label	For chronic moderate-to-severe AD, we conditionally recommend phototherapy which can be considered as an an alternative to systemic anti-inflammatory agents. NB-UVB is preferred due to its better availability.	Strong  [100%]	Moderate
Recommendation for the use of cyclosporine in AD	Off Label	We suggest cyclosporine as the first choice among conventional systemic immunomodulators for moderate-to-severe AD patients unresponsive to topical treatments, where on-label and licensed biologics or oral selective JAK inhibitors are unavailable, contraindicated, or financially limited, as it has the strongest evidence for effectiveness.	Strong  [100%]	Moderate

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Table 3D: Contd...

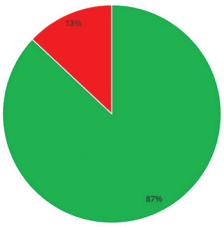
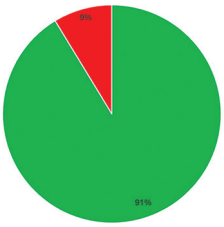
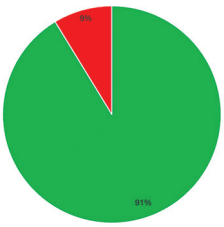
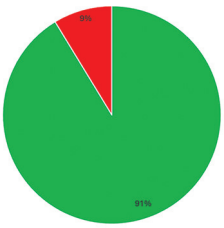
Intervention	Regulatory Status in India	Recommendation	Strength of Recommendations	Certainty of evidence
Recommendation for the use of cyclosporine in AD	Off Label	Due to the lack of sufficient evidence, we cannot make a recommendation for or against the use of azathioprine in moderate-to-severe AD patients.	Conditional  87%	Low
Recommendation for the use of methotrexate in AD	Off Label	For patients with moderate-to-severe AD who are unresponsive to topical treatments, we suggest the use of methotrexate with proper monitoring, when on-label biologics or oral selective JAK inhibitors are unavailable, contraindicated, or financially limited.	Strong  [91.3%]	Low
Recommendation for the use of Mycophenolate mofetil in AD	Off Label	Due to the lack of sufficient evidence, we cannot make a recommendation for or against the use of Mycophenolate mofetil in moderate-to-severe AD patients.	Strong  [91.3%]	Low
Recommendation for the use of Apremilast in AD	Off Label	Due to the lack of sufficient evidence, we cannot make a recommendation for or against the use of Apremilast in moderate-to-severe AD patients.	Strong  [91.3%]	Low

Table 3E: Consensus statements for managements of AD with biologics or JAK inhibitor systemic therapies

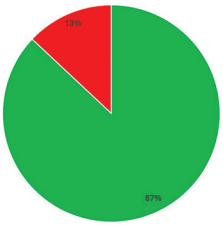
Intervention	Regulatory Status in India	Recommendation	Strength of Recommendations	Certainty of evidence
Recommendation for the use of Dupilumab in AD	On Label	We recommend dupilumab as a first line treatment for adults (>18 years of age) with moderate to severe AD.	Conditional  87%	High

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Table 3E: Contd...

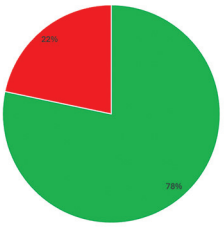
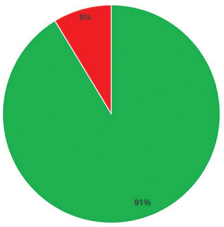
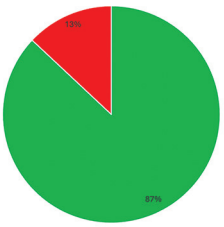
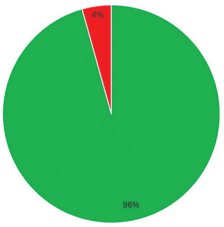
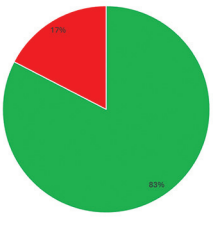
Intervention	Regulatory Status in India	Recommendation	Strength of Recommendations	Certainty of evidence
	Off Label	We conditionally recommend dupilumab as the first-line systemic treatment for children above 6 months with moderate-to-severe AD.	Conditional  [78.3]	High
	Off Label	We suggest dupilumab may be preferred for moderate-to-severe AD patients with severe comorbidities, such as end-stage organ disease, dysfunction, or cardiovascular and venous thromboembolism risk factors.	Strong  [91.3%]	Moderate
	On Label	Based on available evidence, we suggest that dupilumab is considered safe and effective in elderly patients compared to conventional systemic agents.	Conditional  87%	Moderate
	NA	We suggest that for AD patients with a history of recurrent or moderate-to-severe eye inflammation, or ocular surface disorders such as conjunctivitis or keratitis, an ophthalmologist should be consulted before starting treatment with dupilumab.	Strong  [95.7%]	Moderate
	NA	We suggest that live attenuated vaccines be avoided during dupilumab treatment. Therefore, screening for age-appropriate vaccinations should be completed at least 4 weeks prior to starting biologic treatment in AD patients.	Conditional  82.6%	Moderate

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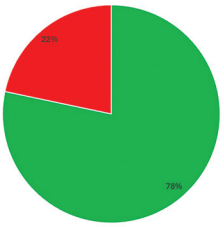
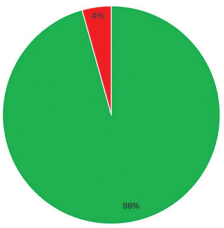
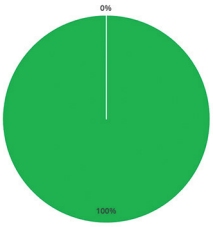
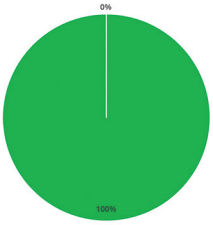
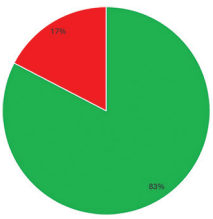
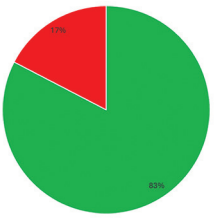
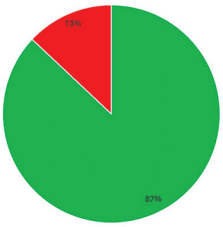
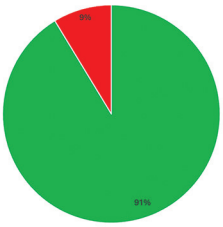
Table 3E: Contd...

Intervention	Regulatory Status in India	Recommendation	Strength of Recommendations	Certainty of evidence
	NA	We suggest that no specific laboratory tests are required to start or monitor AD patients using dupilumab.	Conditional  [78.3]	Moderate
Recommendation for the use of Abrocitinib in AD	On Label	We recommend abrocitinib, a selective JAK-1 inhibitor, for adults and adolescents with moderate to severe AD a first-line treatment.	Strong  [95.7%]	High
	NA	We suggest that abrocitinib may be preferred for adults and adolescents with moderate-to-severe AD who have severe itching.	Strong  [100%]	High
	NA	We suggest that abrocitinib may be preferred for adults and adolescents with moderate-to-severe AD who require a rapid onset of action.	Strong  [100%]	High
Recommendation for the use of tofacitinib in AD	Off Label	For patients with moderate-to-severe AD who are unresponsive to topical treatments, we suggest the use of tofacitinib with proper monitoring, when on-label biologics or oral selective JAK inhibitors are unavailable, contraindicated, or financially limited.	Conditional  82.6%	Weak

Contd...

Table 3E: Contd...

Intervention	Regulatory Status in India	Recommendation	Strength of Recommendations	Certainty of evidence
Recommendation for the use of Baricitinib in AD	Off Label (India). On label (Select countries)	We conditionally recommend baricitinib for adults with moderate-to-severe AD.	Conditional  82.6%	High
Recommendation for the use of Tralokinumab in AD	Currently off-label and unavailable in India. On label (Select countries)	We conditionally recommend Tralokinumab for adults with moderate-to-severe AD if and when licensed in India.	Conditional  87%	High
Recommendation for the use of Upadacitinib in AD	Currently off-label and unavailable in India. On label (Select countries)	We conditionally recommend Upadacitinib for adults with moderate-to-severe AD if and when licensed in India.	Strong  [91.3%]	High

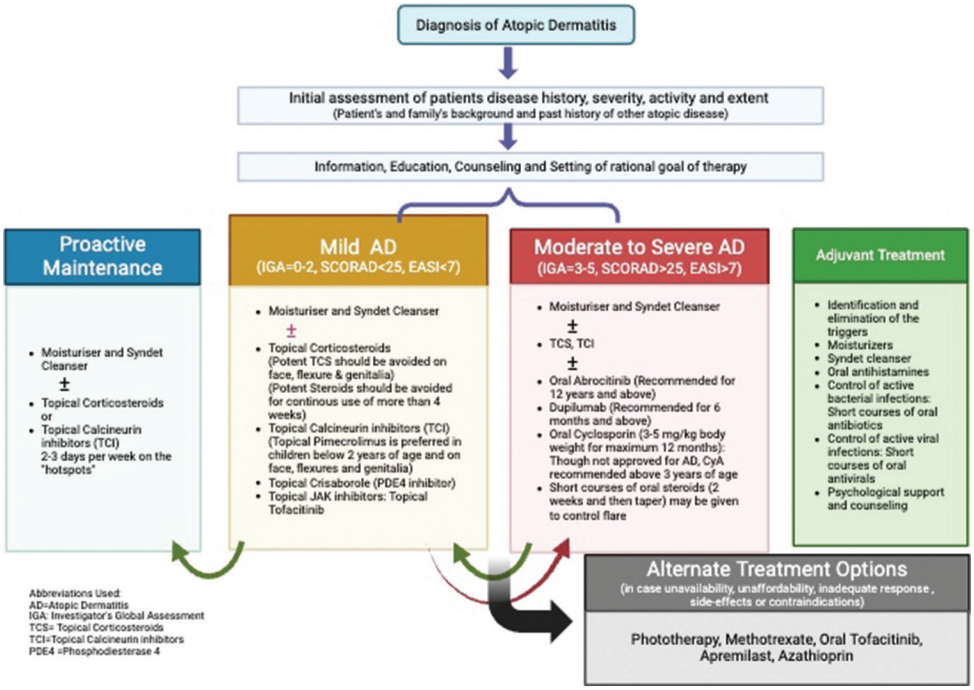


Figure 1: Algorithm for AD guidelines

complete, while adults with refractory AD may receive immunosuppressants. However, evidence for paediatric use remains limited. Biologic therapies have transformed moderate-to-severe AD treatment. Dupilumab is approved globally for patients aged 6 months and older, but in India, it is currently approved only for those aged 18 years and above. Abrocitinib is approved for use in patients aged 12 years and above globally, and in India.[56]

AD in Special Populations

Atopic dermatitis in the elderly population

AD, once considered primarily a childhood condition, is increasingly recognised in the elderly, presenting with bilateral eczematous lesions on the face, neck, and extremities. In older adults, AD may also involve the buttocks or genitals and show chronic, lichenified lesions. The disease can be exudative in acute phases and is often accompanied by xerosis and severe itching, which impacts sleep and QOL. The clinical course may be new-onset or a recurrence of earlier AD. Diagnosis follows the same principles as in younger patients but requires differentiation from conditions like contact dermatitis or psoriasis. Elevated IgE levels are not always present, making diagnosis challenging.

Managing AD in elderly patients presents unique challenges due to the prevalence of comorbidities and polypharmacy in this population. Topical treatments, such as corticosteroids and calcineurin inhibitors (e.g., tacrolimus and pimecrolimus), are effective for mild to moderate cases and should be used with caution to avoid side effects like skin thinning. Systemic treatments for more severe AD in elderly patients require careful consideration of the risks associated with comorbid conditions like hypertension, diabetes, and kidney dysfunction. Biologics like dupilumab and tralokinumab effectively manage moderate-to-severe AD in older adults, with good tolerability and no need for routine lab monitoring. JAK inhibitors, though effective, pose higher infection and malignancy risks in the elderly and should be used cautiously due to their increased infection susceptibility.[57]

Overall, AD in the elderly demands tailored management, balancing treatment efficacy with the risks associated with comorbidities and polypharmacy. New therapies, including biologics and JAK inhibitors, offer potential, but their use must be carefully monitored in this age group.

Atopic dermatitis in pregnancy

AD is the most common dermatological condition during pregnancy, with 80% of cases being new-onset and 20% representing exacerbations of pre-existing disease. Hormonal and immunologic changes during pregnancy can trigger eczematous lesions, known as atopic eruption of pregnancy (AEP), which can be difficult to diagnose, especially when occurring de novo.

Treatment must balance safety for both the mother and fetus. Emollients are essential, while topical corticosteroids and calcineurin inhibitors like tacrolimus are considered safe first-line treatments. Ultraviolet (UV) therapy can also be used if necessary. For severe cases, systemic treatments like cyclosporine A are preferred, with azathioprine an option for those already using it. Systemic glucocorticoids may be used for flare-ups, but JAK inhibitors, mycophenolate mofetil, and methotrexate are contraindicated due to fetal risks. Biologics are not generally recommended due to limited safety data, although case reports suggest cautious use may not harm maternal or fetal outcomes.[58]

AD during pregnancy is often unpredictable; so careful, individualised treatment is essential, involving a risk–benefit assessment between the healthcare provider and patient. Advances in research are slow, but probiotics and skin barrier-enhancing interventions show promise in preventing AD and food allergies.

Conclusions

These consensus statements align with international AD guidelines (European Taskforce, AAD) and support biologics and JAK inhibitors as first- or second-line options for moderate-to-severe AD in India, considering efficacy, safety, and cost. Systemic treatments should be dermatologist-guided to ensure accurate diagnosis and adverse event monitoring, with skin biopsy recommended for unclear cases, especially in the elderly.

This document updates the 2019 Skin Allergy Society guidelines, reinforcing global recommendations while allowing local adaptability. Though tailored for India, these guidelines are also relevant for dermatologists in other developing countries, helping guide treatment selection based on disease presentation, environmental factors, and medication availability.

Acknowledgement

Skin Allergy Research Society and Society for Eczema Studies.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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