Atopic Dermatitis

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KEYWORDS

• Atopic dermatitis • Emollients and skin care • Topical therapy • Systemic therapy • Phototherapy

KEY POINTS

- Atopic dermatitis (AD) is a common, chronic relapsing, and remitting inflammatory skin disease that is characterized by erythematous, scaly, and pruritic lesions often located over the flexural surfaces.
- Good skin hygiene and the use of emollients are recommended for chronic treatment in all patients with AD.
- Topical corticosteroids are first-line treatments during AD exacerbations.
- Systemic therapies including monoclonal antibody treatments and phototherapy are effective in patients with moderate-to-severe AD not responsive to topical therapies.

INTRODUCTION

Atopic dermatitis (AD), also known as atopic eczema, is a chronic relapsing inflammatory skin disease that is characterized by erythematous, scaly, and pruritic lesions often located over the flexural surfaces in adults (**Fig. 1**).[1] In infants, the lesions predominantly affect the face, scalp, trunk, and extensor surfaces. Acutely, the lesions are vesicular with open weeping and crusting eruption. In the subacute phase, the lesions present as scaly and dry fissures, which over time are lichenified from repeated scratching. Another feature of chronic AD is decreased erythema compared with the

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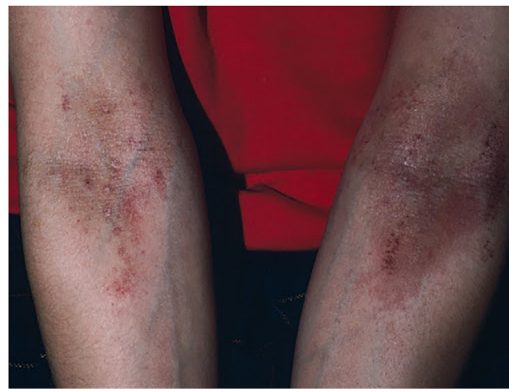

Fig. 1. Atopic dermatitis: pink scaly papules and plaques in the antecubital fossa.

acute state.[1] AD is often associated with atopic conditions, such as allergies and asthma. In addition to the association with closely related atopic conditions, AD is related to other diseases such as obesity, cardiovascular disease, and psychological diseases such as anxiety and depression.[2,3]

Multiple theories exist regarding the pathophysiology of AD and include a complex interplay of factors including genetics, the immune system, the environment, and the skin surface microbiome. Two major theories exist to explain the pathophysiology of AD: the inside-out and outside-in hypotheses. The inside-out hypothesis suggests inflammation or dysregulation of the immune system creates skin barrier defects that further allow penetration of allergens and irritants.[2,3] The outside-in hypothesis suggests that epidermal skin barrier impairments lead to immune dysregulation and allergic sensitization.[4]

Both innate and acquired immune responses have a role in the pathogenesis of AD. The Th2 response is further exacerbated by Langerhans cells in the epidermis, which are specialized immune cells that have a heightened response to allergen and irritant antigens. Implicated cytokines in this causal pathway include IL-4, IL-13, IL-31, and IL-22 many of which are targeted by new biologic medications.[5,6] IL-31 has specifically been shown to be important in the pathogenesis of pruritus. This itch–scratch cycle ultimately worsens the inflammation of AD by decreasing filaggrin (FLG), ceramides, and antimicrobial peptides while increasing bacterial colonization and infections of the skin, which become difficult to control.

The pathogenesis of AD involves skin barrier dysfunction, often times triggered by FLG mutations. Emerging evidence shows that disorder in FLG expression plays a critical role in developing AD. FLG are proteins that bind keratin in epidermal cells. Loss of FLG leads to flattening of skin surface cells, a decrease in natural moisturizing factors of the skin, and an increase in skin pH, which leads to increased activity of proteases and enzymes that break down proteins that hold skin epidermal cells together and ultimately cytokines that promote skin inflammation.[6–8]

Other factors to consider that contribute to the process of inflammation include the skin microbiome, viral, bacterial, and fungal infections, stress, climate, food, and environmental allergens.[6]

OBSERVATION/ASSESSMENT/EVALUATION

The 3 phases of AD are infantile, childhood, and adult.

Infantile Atopic Dermatitis

Clinical presentation and differential diagnosis

Infantile AD typically manifests from birth to 2 years and is part of a multitude of allergic conditions that develop during infancy known as "atopic march." This progressive atopy begins with the development of AD and subsequently allergic rhinitis and asthma in later childhood. Infants typically present with erythematous papules and vesicles seen on the cheeks, forehead, or scalp. In infants, atopic lesions often involve the face, scalp, and extensor surfaces (as opposed to adult AD), and the affected areas can have serous oozing and crusting (**Fig. 2**). The diaper area is often spared due to moisture retention of the diaper.[9]

Diagnosis and classification

The "Hanifin and Rajka" criteria and the UK Working Party's Diagnostic Criteria for Atopic Dermatitis are the most widely cited criteria used.[10–13]

Five major clinical features based on these criteria are (4 required for diagnosis) as follows:

1. pruritus
2. a chronic, relapsing course
3. typical distribution
4. family or personal history of atopy
5. onset before 2 years of age.

Minor criteria are also frequently observed and include the following:

- Early age of onset
- Xerosis
- Palmar hyperlinearity, ichthyosis, keratosis pilaris
- Immediate skin test reactivity, elevated serum IgE
- Cutaneous infection, including *Staphylococcus aureus* and Herpes simplex virus
- Nipple eczema
- Cheilitis
- Pityriasis alba
- White dermatographism, delayed blanching
- Perifollicular accentuation
- Anterior subcapsular cataracts

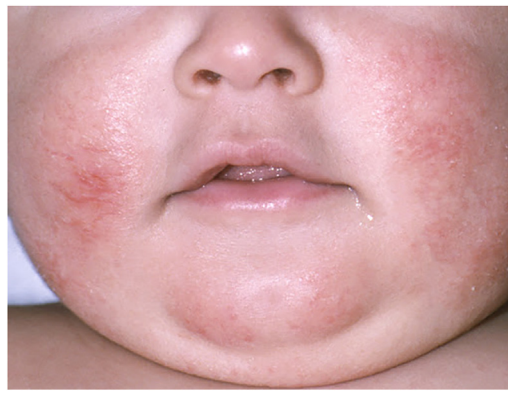

Fig. 2. Infantile atopic dermatitis: pink oozing vesicles on the cheeks.

- Itch when sweating
- Nonspecific hand or foot dermatitis
- Recurrent conjunctivitis
- Dennie-Morgan folds
- Keratoconus
- Facial erythema or pallor

Scoring tools to measure disease severity such as the Severity Scoring of Atopic Dermatitis index and the Eczema Area and Severity index can be used for research and clinical practice. It is also important to note that infantile AD affects quality of life not just for the patient but also for the parent and caretaker. Infants often have intense pruritus and sleep disturbances, and parents or caretakers are often affected by the cost of medication, poor sleep, time off of work, and emotional stress because of their care for their child with AD.[9]

Management

Treatment of infantile AD includes topical emollients and corticosteroids. Emollients improve skin hydration and have been shown to decrease the number of flares. There is little evidence to show whether certain emollients are superior to others but ideally should be free of sensitizing agents. First-line medication option in the management of AD is topical corticosteroids (TCS), which are classified into 7 groups based on potency. Low-potency TCS should be used for long-term management because children are more prone to develop side effects. Because infants have thinner skin and a larger surface-area-to-weight ratio, they are likely to absorb increased amounts of the medication compared with adults. Commonly used TCS approved for infants aged 3 months and older include low-to-medium potency TCS such as desonide, fluocinolone acetonide oil, hydrocortisone butyrate, or triamcinolone. Ointment is based in petrolatum and is more effective at healing the skin than lotions or creams that are water based. Current treatment guidelines do not recommend more than twice-daily application of TCS. Side effects include skin atrophy, striae, acne, and telangiectasia. For this reason, they should be used for the shortest duration needed to control the flare-up and on an as-needed basis after that.

Topical calcineurin inhibitors such as tacrolimus and pimecrolimus are second-line treatment options. These nonsteroidal medications inhibit calcineurin in the skin, blocking T-cell activation, and the release of cytokines. They are approved in the United States by the Food and Drug Administration for children aged 2 years and older and are usually used in conjunction with TCS. Adverse effects include skin burning and irritation, so patients should be counseled on using sun protection.[9]

Wet wrap therapy is a useful technique to treat persistent and refractory AD. They are best done in the evening before bed where a topical steroid is placed on eczematous lesions, a moist dressing is placed over the lesion and then a dry one on top of that. Then the patient puts on their clothing and leaves them on for several hours over night. Results are quite remarkable even after a few days using this method.

Importantly, peanut introduction should not be delayed in children simply due to the presence of AD. The Learning Early about Peanut Allergy trial examined the introduction of peanuts in infants with severe AD or egg allergy, and concluded that early and sustained peanut consumption in this group resulted in notably lower rates of peanut allergy at 60 months of age.[14] In 2017, the National Institute of Allergy and Infectious Diseases developed an addendum of guidelines specifically to address the prevention of peanut allergy for infants at various risk levels[15] (**Table 1**).

Table 1
Summary of addendum guidelines for the prevention of peanut allergy

Infant Criteria	Recommendations	Earliest Age of Peanut Introduction
Severe eczema, egg allergy, or both	Strongly consider evaluation with peanut-specific IgE and/or skin prick test and, if necessary, an oral food challenge. Based on test results, introduce peanut-containing foods	4–6 mo
Mild-to-moderate eczema	Introduce peanut-containing foods	Around 6 mo
No eczema or any food allergy	Introduce peanut-containing foods	Age-appropriate and in accordance with family preferences and cultural practices

From Togias A, Cooper SF, Acebal ML, Assa'ad A, Baker JR Jr, Beck LA, Block J, Byrd-Bredbenner C, Chan ES, Eichenfield LF, Fleischer DM, Fuchs GJ 3rd, Furuta GT, Greenhawt MJ, Gupta RS, Habich M, Jones SM, Keaton K, Muraro A, Plaut M, Rosenwasser LJ, Rotrosen D, Sampson HA, Schneider LC, Sicherer SH, Sidbury R, Spergel J, Stukus DR, Venter C, Boyce JA. Addendum guidelines for the prevention of peanut allergy in the United States: Report of the National Institute of Allergy and Infectious Diseases-sponsored expert panel. J Allergy Clin Immunol. 2017 Jan;139(1):29-44.

Toddler and School Age Atopic Dermatitis

The distribution of dermatitis changes as children age. From age 2 to 12 years, with walking, flexural surface involvement (antecubital fossae, popliteal fossae, neck, wrists, and ankles) is often seen. With eating and drooling, dermatitis can spread to the mouth and chin. Lichenification due to chronicity of symptoms and scratching also occurs. Although during the school years, AD will improve, the function of the skin barrier will never be normal. The differential for manifestations of AD in this age group includes discoid eczema, pityriasis alba, and lip licker's dermatitis.[16]

Clinical presentation and differential diagnosis

Similar diagnosis strategies to that used for infantile AD can be used. See guidelines and therapeutics table for specific age-based options for treatment.

Adult Atopic Dermatitis

There is a variety of presentations of the adult phase. The flexural pattern can persist and is common or become more diffuse. Some adults who had complete resolution of AD as a child, may develop hand dermatitis due to occupational exposures. The upper eyelids and lips are other frequent affected areas in adults. Denni-Morgan folds, or a fold of skin under the lower eyelids, can be seen when chronic eyelid dermatitis develops. Dry skin, white dermographism, hyperlinear palms, ichthyosis vulgaris, and keratosis pilaris are common associated manifestations of AD in adults. Lichenification and fissuring can commonly occur with chronicity (**Figs. 3** and **4**) Adult-onset dermatitis also can occur but is unusual.[17]

Scoring tools used now to assess disease severity in patients with skin of color can underestimate the severity of disease due to erythema in adult AD in skin of color being violaceous. Postinflammatory hypopigmentation and hyperpigmentation occur

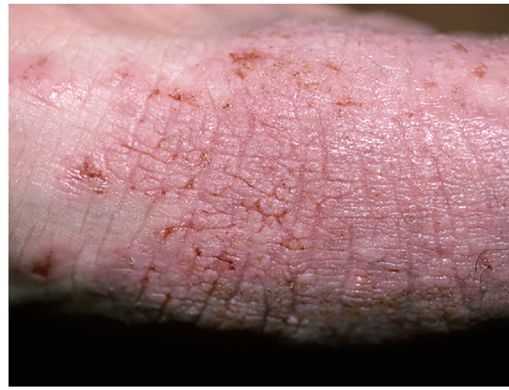

Fig. 3. Chronic atopic dermatitis: lichenified plaques on the ankle.

more commonly in skin of color and is often a source of distress (**Fig. 5**). With scratching and rubbing, patients with skin of color can also more commonly develop lichen simplex or nodular prurigo.[18]

Similar diagnosis strategies to that used for infantile AD can be used. See the guidelines and therapeutics table below for specific age-based options for treatment.

PREVALENCE/INCIDENCE

AD affects 10% to 20% of the population in developed countries.[19] It is ranked as the third most prevalent dermatologic condition but was the greatest contributor to disability-adjusted life years, which measure years of life lost due to premature mortality plus years lost due to disability or its consequences. All races are affected and people with "atopic tendency" often display a higher prevalence of hay fever, asthma, and food allergies.[20] Among populations, genetic studies show that AD affects diverse populations.[18]

Although AD most commonly affects children, all ages can be affected. In approximately 60% of cases, AD presents in the first year of life.[19,21,22] About 57% of children develop AD before age 6.[22]

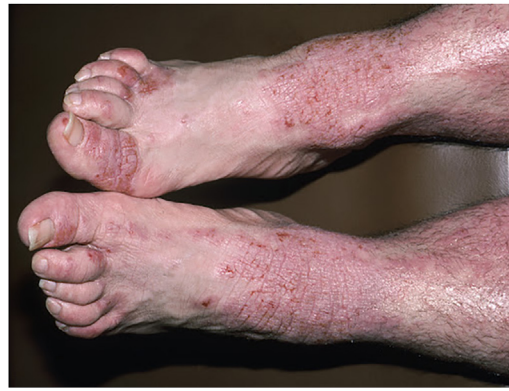

Fig. 4. Atopic dermatitis: thickened lichenified plaques on the ankles.

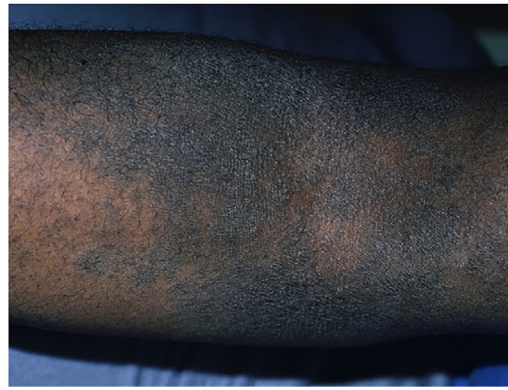

Fig. 5. Atopic dermatitis: lichenified plaques and hyperpigmentation of eczematous patch.

WORLDWIDE/REGIONAL INCIDENCE AND MORTALITY RATES

There seems to be an association between higher socioeconomic status and AD prevalence and morbidity. Lifetime prevalence worldwide of AD is greater than 15%, especially in wealthy developed countries.[18,23]

The prevalence of AD globally varies around the world. According to the latest available data, AD continues to increase in prevalence in young children, aged 6 to 7 years and 13 to 14 years, in low-income countries. Some of the countries in which this trend is noted are Latin America or in Southeast Asia.[24]

Studies show that AD is a prevalent problem all around the world, among infants, children, young adults, and adults. The global prevalence of AD is highest in younger children compared with older children, adolescents, and adults. The significant predictors of AD were age, weather, food, race, ethnicity, place of birth, sex, current working status, and family history of atopy and maternal age.[25]

DISCUSSION

Goals of Treatment

The primary goals of treatment of AD are to reduce skin inflammation and pruritus, restore skin barrier function, and improve quality of life.[26] As previously discussed, treatments can be classified into the categories of moisturizing and basic skin care, topical therapies, phototherapy, and systemic therapies.[26–28] Reduction of itching and burning, as well as complete clearing of all skin changes, are the most important treatment goals for patients.[29]

Approach

The optimal management of AD requires a multipronged approach that involves the elimination of exacerbating factors, restoration of the skin barrier function and hydration of the skin, patient education, and pharmacologic treatment of skin inflammation.

AD is a chronic disease that can first present in childhood and persist across a lifetime with exacerbations. The general approach toward the treatment of AD is to reduce symptoms of inflammation and pruritis, prevent exacerbations, and limit the side effects of therapies to improve the overall quality of life.[26,29] The primary approach includes the avoidance of potential triggers and skin care with emollients to limit water loss, restore the epidermal barrier, and spare the use of steroids in exacerbations. Topical therapies include the use of TCS and calcineurin inhibitors.[28]

Table 2
Frequently used topical corticosteroids for atopic dermatitis treatment

Classification	Topical Corticosteroid
Class 1 Very High Potency	• Augmented betamethasone dipropionate 0.05% (ointment, gel) • Augmented diflorasone diacetate 0.05% (ointment) • Clobetasol propionate 0.05% (ointment, gel, cream, lotion, foam, solution, spray, shampoo) • Desoximetasone 0.25% (spray) • Fluocinonide 0.1% (cream) • Flurandrenolide 4 μg/cm^2 (tape) • Halobetasol propionate 0.05% (ointment, cream)
Class 2	• Amcinonide 0.1% (ointment) • Augmented diflorasone diacetate 0.05% (cream) • Augmented betamethasone dipropionate 0.5% (cream, lotion) • Betamethasone dipropionate 0.05% (ointment) • Desoximetasone 0.05% (gel) • Desoximetasone 0.25% (ointment, cream) • Diflorasone diacetate 0.05% (ointment) • Fluocinonide 0.05% (ointment, gel, cream, lotion, solution) • Halcinonide 0.1% (ointment cream) • Mometasone fluroate 0.1% (ointment) • Triamcinolone acetonide 0.5% (ointment)
Class 3	• Amcinonide 0.1% (cream, lotion) • Betamethasone dipropionate 0.05% (cream, lotion) • Betamethasone valerate 0.1% (ointment) • Betamethasone valerate 0.12% (foam) • Diflorasone diacetate 0.05% (cream) • Fluticasone propionate 0.005% (ointment) • Triamcinolone acetonide 0.1% (ointment) • Triamcinolone acetonide 0.5% (cream)
Class 4	• Betamethasone valerate 0.12% (foam) • Desoximetasone 0.05% (cream) • Fluocinolone acetonide 0.025% (ointment) • Flurandrenolide 0.05% (ointment) • Hydrocortisone valerate 0.2% (ointment) • Mometasone furoate 0.1% (cream, lotion) • Triamcinolone acetonide 0.1% (ointment, cream) • Triamcinolone acetonide 0.2% (spray)
Class 5	• Betamethasone dipropionate 0.05% (lotion) • Betamethasone valerate 0.1% (cream, lotion) • Clocortolone pivalate 0.1% (cream) • Fluocinolone acetonide 0.025% (cream) • Fluocinolone acetonide 0.01% (oil, shampoo) • Fluticasone propionate 0.05% (cream, lotion) • Flurandrenolide 0.05% (cream) • Hydrocortisone butyrate 0.1% (ointment, cream, lotion, solution) • Hydrocortisone probutate 0.1% (cream) • Hydrocortisone valerate 0.2% (cream) • Prednicarbate 0.1% (ointment, cream) • Triamcinolone acetonide 0.025% (ointment) • Triamcinolone acetonide 0.01% (lotion)

(*continued on next page*)

Table 2
(*continued*)

Classification	Topical Corticosteroid
Class 6	• Alclometasone dipropionate 0.05% (ointment, cream) • Betamethasone valerate 0.05% (lotion) • Desonide 0.05% (ointment, gel, cream, lotion, foam) • Fluocinolone acetonide 0.01% (cream, solution) • Triamcinolone acetonide 0.025% (cream, lotion)
Class 7 Lowest Potency	• Dexamethasone sodium phosphate 0.1% (cream) • Hydrocortisone 0.5%–2.5% (ointment, gel, cream, lotion, foam) • Methylprednisolone acetate 0.25% (cream)

Adapted from Eichenfield LF, Tom WL, Berger TG, Krol A, Paller AS, Schwarzenberger K, Bergman JN, Chamlin SL, Cohen DE, Cooper KD, Cordoro KM, Davis DM, Feldman SR, Hanifin JM, Margolis DJ, Silverman RA, Simpson EL, Williams HC, Elmets CA, Block J, Harrod CG, Smith Begolka W, Sidbury R. Guidelines of care for the management of atopic dermatitis: section 2. Management and treatment of atopic dermatitis with topical therapies. J Am Acad Dermatol. 2014 Jul;71(1):116 to 32.

Topic calcineurin inhibitors are second-line and are useful in patients who have not responded to TCS, in sensitive areas (eg, face, anogenital, skin folds), sites with steroid-associated atrophy, and long-term uninterrupted TCS use.[28] Phototherapy is recommended in children and adults for both acute and chronic AD not responding to topical therapies.[30] Systemic immunomodulatory agents are indicated in adult and pediatric patients in whom optimized topical regimens using emollients, topical anti-inflammatory therapies, adjunctive methods, and/or phototherapy do not provide adequate disease control.[30]

Evaluation

Dermoscopy of AD often shows focal white scales, yellow serous crust, and dotted vessels distributed in clusters or randomly with associated dilated capillaries in irregularly elongated dermal papillae. In cases where intense itching occurs, hemorrhages are also seen.[31] The diagnosis is primarily made through history and physical examination findings; however, skin biopsy may be useful in cases where there is uncertainty.

Current Clinical Guidelines

Guidelines from the American Academy of Dermatology note that topical agents form the foundation of therapy for AD.[27,28,30] These include frequent use of topical moisturizers and effective bathing practices (duration and frequency are nonstandardized but it is recommended to apply moisturizers soon after bathing and limit the use of non-soap cleansers). TCS are the mainstay of topical anti-inflammatory treatment. It is generally recommended to apply twice daily, applying every day during flares, and once or twice weekly afterward to prevent recurrence. Topical calcineurin inhibitors include tacrolimus and pimecrolimus, second-line agents that have been approved for moderate-to-severe and mild-to-moderate AD, respectively. These do not carry the risk of skin atrophy that topical steroids do, making them potentially advantageous options in sensitive areas but commonly cause localized stinging/burning. Topical antimicrobials/antiseptics have generally not been shown to be effective, with the exception that diluted bleach baths seem to improve clinical outcomes in those with moderate-to-severe AD who have frequent bacterial skin infections.[28]

Phototherapy is a second-line treatment that has also been shown to be effective for AD. Many types of light and protocols for therapy can be used, with limited

comparison trials available. It can be used on either an intermittent or continuous basis, as monotherapy or in combination with emollients and topical steroids (it is recommended to avoid combining it with topical calcineurin inhibitors). Side effects are low but it can cause erythema, burning, itching, and actinic damage.[30]

Further Treatment Options

Exciting new medications are now approved for moderate-to-severe AD. Dupilumab (Dupixent) is now FDA approved for patients with AD aged 6 months and older. It is an injection given 2 weeks apart and has a good safety profile with conjunctivitis being the most common side effect. In two phase 3 trials, the human monoclonal antibody against interleukin-4 receptor alpha dupilumab was shown to improve pruritus and increase the likelihood of achieving decreased scores on the Investigator's Global Assessment. In patients 18 years of age and older, dupilumab is administered 300 mg every other week via subcutaneous injection (after a 600 mg loading dose).[32] Dosing for the pediatric population is weight-based. Although crisaborole and dupilumab are FDA approved, their cost (ranging from US$700–3000 per month) make them impractical for many patients.[1]

JAK inhibitors such as upadacitinib and abrocitinib have been used with great success in moderate-to-severe AD.[33,34] Abrocitinib is an oral medication taken at doses of 100 or 200 mg once per day.[33] Side effect includes immunosuppression and potentially increased risk of upper respiratory tract infection, blood clots, and elevated liver enzymes. There are many other targeted treatments such as IL-13 and IL-31 inhibitors that are undergoing clinical trials.

An ointment of crisaborole (a phosphodiesterase 4 inhibitor) was found to be effective at reducing itching faster than vehicle alone; however, it is expensive and burning is a common complaint.[35]

Therapeutic Options

(**Tables 2** and **3**)

Table 3
Therapeutic approaches

Mild AD	Moderate AD	Severe AD
Intermittent, short-term use of class 6 or 7 topical steroids (see Table) ± topical calcineurin inhibitors	Intermittent, short-term use of class 3–4 topical steroids (see Table) ± topical calcineurin inhibitors	Class 2 topical steroids for flares (see Table); class 3–5 topical steroids ± tacrolimus ointment for maintenance
Bathing and barrier repair with emollients	Oral antihistamines	Oral antihistamines
Avoidance of irritant and allergic triggers	Bathing and barrier repair	Bathing and barrier repair
Treat superinfection	Avoidance of irritant and allergic triggers	Avoidance of irritant and allergic triggers
	Treat superinfection	Treat superinfection
		Consider systemic anti-inflammatory agents, ultraviolet light therapy, biologics

Adapted from Paller A, Mancini AJ. Chapter 3 – Eczematous Eruptions in Childhood. Hurwitz Clinical Pediatric Dermatology: A Textbook of Skin Disorders of Childhood and Adolescence. 5th edition. Elsevier; 2015: 38-72e7.

SUMMARY

AD is an extremely common skin condition that significantly decreases quality of life for both the patient and their caregivers. It is important for the primary care provider to be knowledgeable on the condition and its diagnosis and management/treatment options that are available.

Emollients such as creams and ointments are extremely important for the maintenance of patients with AD. For AD flares, TCS are considered first-line treatment. In moderate and severe AD, topical calcineurin inhibitors can be used in combination with topical steroids as a steroid-sparing therapy or for areas with thin skin such as the face and intertriginous areas. Second-line treatment of moderate and severe AD that can be used is Ultraviolet light B (UVB) phototherapy. For severe AD, dupilumab is an established and effective treatment option, and Janus kinase (JAK) inhibitors are also approved and effective for severe AD. Immunosuppressants such as methotrexate, azathioprine, mycophenolate mofetil, and cyclosporine can be used for AD but they all have their own side effect profile.

Routine evaluation of AD should not include blood tests or skin prick tests. Although secondary infection is the most common complication of AD, without evidence for clinical infection in patients with AD, oral antibiotics should not be used. Lichenification and postinflammatory scarring can be seen in chronic AD. Long-term treatment of AD should not include systemic corticosteroids.

Noncompliance to treatment regimens effect treatment outcomes and patients. Every effort should be made to assess the multifactorial issues that contribute to noncompliance for a patient including cost, patient bias, complex regimens, infrequent follow-up, and lack of knowledge. Frequent follow-ups, patient education, and AD action plans can help improve patient outcomes through adherence.[1] Because this is a common and chronic condition, primary care physicians should be aware of its diagnosis and treatment and be able to set realistic expectations for these patients.

CLINICS CARE POINTS

- The diagnosis of AD is primarily based on history and physical examination findings. SORT C
- Skincare, emollients, and avoidance of triggers are recommended in all patients for chronic treatment. SORT A
- TCS are first-line treatments during AD exacerbations. SORT A
- Topic calcineurin inhibitors are useful in patients who have not responded to TCS, and for maintenance therapy in sensitive areas (eg, face, anogenital, skin folds), sites with steroid-associated atrophy, and long-term uninterrupted TCS use. SORT A
- Monoclonal antibody treatments are effective in patients with moderate-to-severe AD not responsive to topical therapies. SORT A

DISCLOSURE

The authors have nothing to disclose.

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