

In the Clinic

Atopic Dermatitis (Eczema)

Prevention	page ITC5-2
Diagnosis	page ITC5-3
Treatment	page ITC5-7
Practice Improvement	page ITC5-13
Tool Kit	page ITC5-14
Patient Information	page ITC5-15
CME Questions	page ITC5-16

Physician Writer
Susan V. Bershada, MD

Section Editors
Deborah Cotton, MD, MPH
Darren Taichman, MD, PhD
Sankey Williams, MD

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CME Objective: To review current evidence for the prevention, diagnosis, and treatment of atopic dermatitis (eczema).

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Recommendations from the American Academy of Pediatrics for Atopic Disease in High-Risk Infants*

Evidence supports:

Exclusive breastfeeding for the first 4 months of life

Avoiding food introduction for the first 4–6 months

Breastfeeding plus hydrolyzed (or elemental) formula after 4 months

Evidence does not support:

Maternal dietary manipulation during pregnancy and breastfeeding

Delaying food introduction after 4–6 months

*Those who have first-degree relatives with histories of Ig-E mediated disorders, principally atopic dermatitis, asthma, seasonal rhinitis, and/or food allergies.

Eczema and *atopic dermatitis* (AD) are often used synonymously. The distinction is that eczema can mean inflamed skin from any cause, whereas AD is the relapsing–remitting pruritic rash that occurs at typical sites, mainly the face and skin creases, and is associated with other type I allergic disorders, such as asthma, food allergies, and allergic rhinitis.

AD is the most common skin condition of early childhood. Its prevalence may be increasing in developing countries but remains stable at about 17% in the United States (1). The disorder begins in the first 18 months of life in approximately 65% of patients and persists beyond adolescence in 40%. In addition to causing discomfort, sleep loss, and psychosocial challenges, AD can impose major financial burdens on families for direct medical care, household accommodations, and missed work (2). In the United States, the total estimated direct costs of AD range from about \$400 million to \$4 billion per year (3).

AD is a multifactorial disorder involving a genetic predisposition and environmental triggers. There is no single biological marker. At-risk individuals manifest at least 19 different associated genes. Mutations in filaggrin, an epidermal-barrier protein, occur in about 15% of patients and are considered major predisposing factors (5, 6). Controversy surrounds recent proposals that there are 2 types of AD, intrinsic and extrinsic (7). The extrinsic type is described as classical AD that occurs in conjunction with elevated IgE to various airborne and food allergens and is likely to show filaggrin mutation, whereas intrinsic AD is associated with normal IgE levels and a lack of mutated filaggrin. In patients who lack allergen-specific IgE, the World Allergy Association suggests the term nonatopic eczema, rather than intrinsic AD (8), and some authors prefer the term atopiform dermatitis (9).

The most widely held theory of AD pathophysiology is that it begins with a compromised skin barrier caused by defects in filaggrin or other components of the cellular envelope. Epidermal barrier function is further compromised by environmental factors that dry the skin, such as indoor heating and harsh cleansers. Subsequent penetration by allergens, irritants, and bacteria is facilitated, thus triggering the hyperimmune response found in AD (6). Characteristically, skin dendritic cells bearing IgE receptors become polarized as a result of cytokines derived from injured epidermal keratinocytes, resulting in Th2-dominated responses in acute AD lesions and Th22 predominance in chronic lesions (10).

Prevention

Can AD be prevented?

Efforts at primary prevention of AD have focused on prenatal and early childhood exposure to, or avoidance of, various dietary and environmental factors, including baby formulas and breast feeding; allergenic foods such as peanuts, soy, cow's milk, and eggs; probiotic supplements; and household pets. By and large, these studies have not demonstrated the usefulness of such measures.

When contemplating parenthood, patients with past or current AD, asthma, or other type I allergies

should be aware of the American Academy of Pediatrics recommendations for early nutritional interventions to prevent atopic disease, summarized in a 2008 position paper based on extensive review of high-quality evidence (11) (Box: Recommendations from the American Academy of Pediatrics for Atopic Disease in High-Risk Infants). Although exclusive breastfeeding of newborns has been associated with a lower risk for AD in some studies, others have shown no benefit, and this issue remains controversial.

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In contrast to a prior 2001 systematic review of breastfeeding for AD prevention, a 2009 meta-analysis examining 21 studies found that there is no strong evidence that breastfeeding has a protective effect, except when exclusive breastfeeding was

compared with conventional (cow's milk) formula feeding. Comparisons with newer formulas, inherent lack of blinding, and observer bias are possible explanations for the disparity in findings among breastfeeding-versus-formula studies (12).

Prevention... Primary prevention of AD is difficult because heredity plays a large role in its development. Expectant parents with a history of AD, asthma, or food allergies should be made aware that risk might be decreased or delayed in their offspring by exclusive breastfeeding and avoidance of food introduction for 4 to 6 months. Extensively hydrolyzed and elemental baby formulas after 4 to 6 months seem to be safer than traditional cow's milk or soy formulas for high-risk infants.

CLINICAL BOTTOM LINE

What items in the history and physical examination suggest AD?

History and physical examination are the keys to AD diagnosis. Many versions of the classical criteria proposed by Hanifin and Rajka in 1980 (13) have been used to identify AD for research purposes. Of these, the most extensively validated are the UK Working Party Criteria (14), which have been adapted to clinical practice as a concise list of minimum criteria (Box: Diagnostic Criteria for Atopic Dermatitis).

What elements of the history should clinicians focus on in evaluating patients with AD?

Elements of the patient's history that strongly suggest AD are early childhood onset, generalized dry skin, pruritus and scratching, other family members with AD, and a personal or family history of such allergic disorders as asthma, allergic rhinitis, and food allergies. If treatment has been tried with topical corticosteroids, a full or partial response and subsequent relapse are exceedingly common (Table 1).

How should clinicians carry out a skin examination?

A thorough skin examination requires observation of the morphology of the lesions, pattern of

distribution, and concomitant findings (Table 1).

AD lesions are usually discrete but vaguely delineated, erythematous, and scaly. They can take the form of flat patches, edematous plaques, or closely grouped raised papules, on a background of dry skin. Cracking, excoriations, bleeding, crusting, hyperpigmentation and lichenification may be present as secondary findings that reflect frictional trauma, consequent bacterial superinfection, and postinflammatory pigmentary alterations.

In adults, typical AD sites include the face, particularly the eyelids (Figure 1) and perioral area; the antecubital and popliteal fossae and other flexural creases (Figures 2 and 3); extensor surfaces of the limbs; and the hands (Figure 4) and feet. In infants and children, AD has a greater likelihood to affect the cheeks (Figure 5), chin, forehead, base of the neck, and the diaper area, in addition to the flexural surfaces and periorificial areas of the face. AD is almost always bilateral and fairly symmetrical. In severe cases, it can be widespread.

Nummular dermatitis is a pattern of AD that consists of 1 or more oval, erythematous patches (Figure 6). Chronic AD is associated with prolonged scratching that may result in

Diagnosis

Diagnostic Criteria for Atopic Dermatitis*

Patient must have pruritus in the past 12 months, plus 3 or more of the following:

- History of skin-crease dermatitis or cheek dermatitis (in infants)
- Personal history of asthma or hay fever (or first-degree relative if < age 4 y)
- History of generalized dry skin in past year
- Visible skin-crease dermatitis (or dermatitis of the cheeks, forehead, or outer limbs if < age 4 y)
- Onset before age 2 years (not used in patients < age 4 y)

*Adapted from reference 15.



Figure 1. Acute episode of seasonally recurring eyelid dermatitis in a 29-year-old man. This finding is typical in individuals with pollen-specific IgE. Dennie-Morgan folds (extra creases) are apparent in the infraorbital region. Photo credit: Caroline Halverstam, MD.



Figure 2. Chronic antecubital atopic dermatitis showing follicular prominence, lichenification, and hyperpigmentation in a young woman. Photo credit: Susan V. Bershad, MD.

lichenification, a grayish to purple-hued skin thickening; patterns of severe lichenification include plaques, known as lichen simplex chronicus, and papules, referred to as prurigo nodularis (Figure 7).

Associated skin findings that help confirm the diagnosis of AD, although they are not always present, are ichthyosis vulgaris, pityriasis alba, keratosis pilaris, hyperlinear palms, and infraorbital creases, also called Dennie-Morgan folds (Figure 1).

How should the severity of AD be classified?

Establishing the severity of AD is an essential element of the initial assessment and follow-up visit.

In general, mild AD causes minimal pruritus and sleep disturbance and displays barely noticeable or minor findings on skin examination. Moderate AD is likely to cause episodes of marked pruritus, sleep disruption, and apparent physical signs. Severe AD is associated with extreme or constant pruritus, sleep deprivation, and major widespread skin signs that include bleeding, crusting, weeping, and lichenification.

Validated instruments used for clinical monitoring and research assessments (SCORAD, EASI, and POEM), are advocated by professional organizations to improve the quality of clinical care of AD (Table 2) and to provide reproducible evaluation standards. Although each

Table 1. Elements of the History and Physical Examination for Atopic Dermatitis

Category	Element	Ask About, or Look for . .	Notes
History	Duration	Age at onset Acute, intermittent, or chronic	Onset of AD is usually before age 2 y
	Symptoms	Pruritus; sleep disturbance	AD is always pruritic
	Other allergic disorders	Personal history of asthma, seasonal allergies, food allergies	Ask if suspected food and airborne allergens were confirmed by RAST or skin prick tests
	Prior treatments	Regular emollient use; corticosteroids; calcineurin inhibitors; oral antihistamines; antibiotics; natural remedies; other medicines; light treatments (e.g., phototherapy with UVA, narrow-band UVB)	Dry skin is a common precipitant of AD; response and relapse to topical medication is typical
	Environment	Personal care products; dust; pollen; animal fibers in clothing; goose down and feathers in bedding	Traditional recommendations include neutral-pH soap, fragrance-free products, cotton clothing, hypoallergenic bedding
	Family history	Atopic dermatitis, asthma, seasonal allergies, and food allergies in first-degree relatives	Family history is almost always positive for one or more type I allergies
Physical examination	General skin examination	Dry skin (xerosis)	
	Distribution of lesions	Face and scalp; flexural creases; extensor surfaces; hands and feet; widespread	Adults are more prone to AD lesions on dry extensor surfaces and are less prone to cheek and forehead lesions; genital and web space lesions suggest scabies.
	Morphology of lesions	Flat scaling patches; edematous plaques; grouped papules; nummular (oval) patches	Unusual patterns, such as widespread vesicles, bullae, honey-colored crusts, or pustules, suggest viral or bacterial infection
	Intensity of lesions	Degree of erythema; oozing/crusting; excoriation; cracking/fissuring; flaking; bleeding	
	Concomitant skin findings	Ichthyosis vulgaris; Dennie-Morgan folds; pityriasis alba; keratosis pilaris; hyperlinear palms	
	Other systems	Enlarged lymph nodes; wheezing	Lymphadenopathy may indicate chronic superinfection; AD patients are at greater risk for asthma and seasonal allergies

AD = atopic dermatitis; RAST = radioallergosorbent testing; UVA = ultraviolet A; UVB = ultraviolet B

Table 2. Severity Scoring tools for AD*

Outcome Measure	EASI	SCORAD	POEM
Extent/body sites affected	X	X	
Symptoms			
Pruritus		X	X
Sleep disturbance		X	X
Intensity of lesions			
Erythema	X	X	
Edema/induration	X	X	
Oozing/crusting		X	X
Excoriation	X	X	
Lichenification	X	X	
Dryness		X	X
Cracking/fissuring			X
Flaking			X
Bleeding			X

*AD = atopic dermatitis. Adapted from reference 16. EASI = Eczema Area and Severity Index (17); SCORAD = SCORing Atopic Dermatitis (18); POEM = Patient-Oriented Eczema Measure (19).

produces a cumulative score that correlates with global severity, they do not provide a strict formula to grade overall severity. Describing AD as mild, moderate, or severe is ultimately a matter of judgment that involves the patient and caregiver.

SCORAD is the most comprehensive, whereas EASI is based solely on the extent and severity of 4 physical signs; both involve a multistep calculation of the total score. POEM is the simplest to administer—the patient can fill it out in the waiting room and it takes only a few seconds to score, but it only assesses only the frequency of 7 characteristic signs and symptoms and not the extent or severity of lesions.

What other conditions should be considered in the differential diagnosis?

When a diagnosis of AD is being considered, the site and distribution of a rash are important distinguishing features (Table 3). When the eyebrows, nasolabial creases, inner cheeks, scalp, and chest are involved, one must consider seborrheic dermatitis. In seborrheic

dermatitis, exfoliated skin scales tend to be larger, whiter, and greasier than the fine dry scales or yellowish crusts of AD. Although both disorders respond to topical corticosteroids, seborrheic dermatitis responds to more frequent skin cleansing and shampooing, which tend to exacerbate AD.

Psoriasis is another erythematous scaling eruption. It affects teenagers and adults more often than infants and children. Typically, psoriasis plaques are more raised and have a thicker scale than AD lesions, and they tend to occur on extensor surfaces, sometimes making them difficult to distinguish from the nummular (oval) form of AD. Lesions on the hands and feet may also present diagnostic challenges when other characteristic AD sites are unaffected.

When a suspected case of AD is refractory to treatment, one should consider allergic contact dermatitis. A thorough history may reveal recent exposure to a new perfume or grooming product or a common allergen, such as latex, nickel in jewelry or belt buckles, or a poison plant. When examining the patient, look for a pattern that is asymmetrical or limited to areas of contact.

It is important to remember that infections and infestations are common and can coexist with AD. When scaly patches are present on the scalp, one must consider the possibility of dermatophyte infection (tinea capitis) and perform fungal culture of hair and skin scrapings, because treatment requires systemic antifungal therapy. Nummular dermatitis can be virtually indistinguishable clinically from tinea corporis, which generally responds to topical antifungal agents. Likewise, even in a patient with known AD, scabies infestation must be considered when a pruritic eruption involves the skin folds, especially around the wrists, genital area, and finger and toe web spaces. In such cases, microscopic examination of skin scrapings is often



Figure 3. Acute exacerbation of flexural dermatitis on the wrist of a 29-year-old fair-skinned man with a history of atopic dermatitis from infancy. Photo credit: Caroline Halverstad, MD.



Figure 4. Recurrent hand dermatitis with cracking and honey-colored crusts due to *Staphylococcus aureus* superinfection in a 40-year-old woman with a long history of atopic dermatitis and dry skin in winter. Photo credit: Jacob Levitt, MD.



Figure 5. An infant girl with atopic dermatitis and secondary *Staphylococcus aureus* impetigo on the cheeks and neck creases. Photo credit: Susan V. Bershad, MD.

Table 3. Differential Diagnosis of Atopic Dermatitis

<i>Disease</i>	<i>Typical Demographics</i>	<i>Physical examination</i>	<i>How to Diagnose</i>	<i>Notes</i>
Seborrheic dermatitis	Newborns and infants; teens and adults	Erythema and coarse, greasy scale, located on the face (especially eyebrows and nasolabial folds), scalp, presternal area; can be widespread (rare)	Physical examination; positive response to frequent cleansing and shampooing, which make AD worse; skin biopsy shows non-specific features	One of the most common skin disorders after puberty
Psoriasis	Any age, but seldom seen in infants; most common in teens and young to middle-aged adults	Erythematous, raised plaques covered by thick, silvery scale, located on the scalp, extensor surfaces (especially elbows and knees), hands, feet, and trunk	Physical examination; family history is often positive for psoriasis; skin biopsy shows characteristic features	Inverse psoriasis shows a flexural or intertriginous pattern that can be confused with AD; guttate psoriasis can appear acutely as dozens of small lesions, characteristically following an upper respiratory infection; presents on rare occasions as generalized erythroderma with widespread scaling
Allergic contact dermatitis	Any age; certain types of workers are more prone due to occupational exposure to contact allergens, for example: chemicals, nickel, or latex gloves	Erythema, scaling, blisters, or crusts, sometimes asymmetrical, following a pattern of contact with the offending substance	History of exposure to a contact allergen; physical examination; skin patch testing; skin biopsy is nonspecific	Poison ivy is the most common cause; may also be related to other plants, costume jewelry, belt buckles, tools, rubber derivatives, cosmetic ingredients, topical medications (especially antibiotics and anesthetics), preservatives, and dozens of other everyday exposures
Tinea capitis (scalp dermatophyte infection)	Infants, children, and teens; relatively rare in adults	Pruritic scaling, crusting, and erythema of the scalp; may be localized to oval patches or widespread, with possible	Scalp scrapings may show fungal hyphae on direct KOH exam; scalp biopsy usually not needed but may show fungal elements on PAS stain; fungal culture of scrapings usually positive within 2 weeks	False-negative KOH preparations are common, therefore fungal cultures are needed for confirmation; requires at least 6 weeks of oral antifungal therapy
Tinea faciei, corporis, pedis, or cruris (dermatophyte infection of the face, body, feet, or groin area)	Any age; tinea and cruris are more common after puberty	Inflammatory type appears as typical "ringworm," with active red scaling border and central clearing; noninflammatory type appears as dry scaling patches of skin or maceration between the toes	Skin scrapings may show fungal hyphae on direct KOH examination; skin biopsy shows fungal elements on PAS stain; fungal culture of scrapings usually positive within 2 weeks	False-negative KOH preparations are common, therefore fungal cultures are needed for confirmation; localized cases on nonhairy areas can be treated with topical antifungal therapy
Scabies infestation	Any age	Extremely pruritic, excoriated, small erythematous papules on typical sites, such as the axillae, inner wrists, groin, genitals, intergluteal area, and finger and toe web spaces; may be widespread	History of recent onset of an itchy rash in the index patient and other affected household members can be highly suggestive; wet preparation of skin burrow scrapings or skin biopsy may show scabies mites, eggs, or feces; skin biopsy is often characteristic of arthropod-induced eruptions	Burrows are not always seen; empirical treatment is recommended when scabies cannot be ruled out clinically; can be seen as a comorbid condition with AD
Systemic immune disorders (severe combined immune deficiency; the hyper IgE syndrome; the Netherton syndrome; the Wiscott-Aldrich syndrome; phenylketonuria)	Infants and children	Recalcitrant widespread AD, or an eruption that resembles AD, starting during infancy or early childhood, with recurrent systemic and local infections	History and physical examination for typical features; immunologic, metabolic, and genetic evaluations as needed; skin biopsy usually nonspecific.	The possibility of an immune disorder should be considered in cases of severe refractory AD when seen in infants and children who fail to thrive

AD = atopic dermatitis; KOH = Potassium hydroxide prep of skin scrapings; PAS = Periodic acid–Schiff histologic stain

negative, and empirical treatment with a scabicide is warranted.

AD patients presenting with crusting or blistering lesions must be evaluated for secondary infections, including impetigo caused

by *Staphylococcus* and *Streptococcus* organisms, and herpes simplex and varicella-zoster infections (i.e., eczema herpeticum). Smallpox vaccination, although rarely given today, can cause serious widespread secondary infection of

AD, a condition known as eczema vaccinatum.

What is the role of laboratory testing in evaluation of AD?

Laboratory testing is not needed for a diagnosis of classical AD, but a skin

biopsy may be helpful in atypical cases. Although the histologic findings of AD are nonspecific, some differential diagnoses, particularly psoriasis, scabies, and dermatophytosis, usually exhibit diagnostic features on microscopic examination. Dermatophyte infections, such as tinea capitis and tinea corporis, can also be diagnosed by direct microscopic examination of skin scrapings mounted in potassium hydroxide.

Certain laboratory tests are used principally to assess complications and to guide management. When lesions are crusted, bacterial culture and sensitivity are used to select adjunctive topical or systemic antibiotic treatment. Viral studies, including direct fluorescent antibodies and culture for herpes simplex and varicella zoster, are needed to confirm suspected cases of eczema herpeticum requiring systemic antiviral therapy.

In a patient with refractory dermatitis, allergic contact dermatitis should be considered and, if necessary, arrange for skin patch testing to detect type IV delayed hypersensitivity to common contact allergens, including nickel, rubber, and fragrance ingredients.

Radioallergosorbent testing (RAST) for allergen-specific IgE has a high predictive value for diagnosis of AD, because more than half of patients with AD have IgE sensitivity to at least one airborne or food allergen. Skin prick tests are also used to identify and confirm such sensitivities.

In an international study of 2184 infants with AD, 53% had increased total IgE and 56% showed allergen-specific IgE. Sensitivity to the most common aeroallergens (house dust mites, cat dander, grass pollen) were universal, but specific food sensitivities varied widely among countries (20).

How common are food allergies, and how are they related?

At least 170 foods have been reported to cause IgE-mediated allergic reactions. Of these, 8 major

food allergens have been identified: peanuts (0.6% prevalence in the U.S. population); tree nuts (0.4%); fish and crustacean shellfish (0.6% in children and 2.8% in adults); cow's milk (2.2% in a Danish cohort); hen's eggs (1.6% in a Norwegian cohort); wheat; and soy (21). The vast majority of patients eventually develop tolerance to cow's milk, eggs, soy, and wheat, but not to peanuts OR tree nuts. Curiously, fish and shellfish allergies are more prevalent in adults than in children.

Although there are no high-quality studies of food allergies in adults, food sensitivity in children is more common in atopic versus nonatopic individuals, and it is also a risk factor for greater severity of disease.

A cross-sectional study of over 2000 patients with AD aged 13–24 months showed the following rates of elevated IgE: 7% to hen's eggs, 4% to peanuts, and 3% to cow's milk. There was a direct correlation between the frequency of positive IgE responses and the severity of AD (22).

The National Institute of Allergy and Infectious Diseases (NIAID) recently developed “best practice” clinical guidelines for the diagnosis and management of food allergy (21). The Guidelines distinguish between food sensitization, meaning laboratory evidence of elevated allergen-specific IgE, and food allergy, meaning occurrence of clinical symptoms from exposure to particular foods. Aside from systemic symptoms, such as wheezing, tachycardia, nausea, dizziness, and anaphylaxis, food reactions can include such skin findings as AD, urticaria, angioedema, flushing, and pruritus.

Food allergies are identified by a combination of allergen-specific IgE, skin prick test to the specific food, improvement with food elimination, and relapse with subsequent oral challenge.



Figure 6. Nummular pattern of atopic dermatitis on the lower extremity of an adult male, showing grouped papules coalescing into oval plaques. Photo Credit: Jacob Levitt, MD.

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When should a dermatologist or an allergist be consulted for diagnostic help?

A dermatologist can be helpful for atypical cases of AD that fail to respond to initial treatment or when a skin biopsy is being considered to rule out other disorders. Certain

dermatologists perform skin patch testing, which is necessary to diagnose suspected allergic contact dermatitis.

An allergist should be consulted to perform and interpret RAST or skin prick tests when needed to evaluate possible airborne or food allergies.

Diagnosis... AD diagnosis is based on clinical criteria that require a history and physical findings confirming early onset of generalized dry skin with pruritus, predominantly on the face and flexural areas of the limbs. There are several validated instruments that are helpful for grading severity. In the differential diagnosis, seborrheic dermatitis, psoriasis, and allergic contact dermatitis can usually be excluded on clinical grounds but occasionally require skin biopsy or patch testing. Other laboratory tests are rarely needed for diagnosis but may be helpful to detect bacterial, fungal, or viral comorbid conditions and to identify contributory airborne and food sensitivities.

CLINICAL BOTTOM LINE

Treatment

What nonpharmacologic measures should be used in management? How should patients with AD care for their skin?

Conservative dry-skin care can be very beneficial for patients with AD. Moisturizers, also known as emollients, reduce transepidermal water loss and reduce skin barrier compromise. Daily use of an emollient product is universally recommended to enhance and protect the skin barrier (23, 24), but there have been no randomized, controlled trials (RCTs) of AD treatment with emollients alone, except for one study that showed mild efficacy that was inferior to that of topical corticosteroid cream (25). Clinical trials using emollients as adjunctive therapy have shown them to be effective corticosteroid-sparing agents (26, 27). An emollient ointment, cream, or lotion is particularly recommended after bathing. Many dermatologists recommend pure petrolatum or emollients that contain a high ratio of petrolatum to water. Medium-quality evidence shows that AD patients can also be treated successfully with nongreasy moisturizers containing ceramides.

In an uncontrolled trial of 29 patients (ages 5–19 y) with mild-to-moderate AD applying

ceramide-containing lotion for 4 weeks, 20 patients reported complete resolution of symptoms, and stratum corneum hydration increased significantly (28).

Over the past decade, wet-wrap dressings have gained in popularity for AD therapy. While somewhat cumbersome, they have been shown to improve skin barrier function and AD symptoms. Such dressings must be changed frequently to prevent bacterial colonization (29).

Time-tested measures that are commonly used and believed to be helpful but lack clinical trials include the use of neutral-pH unscented soaps, nonsoap hypoallergenic cleansers, and additive-free laundry detergents; wearing comfortable clothing made of cotton instead of wool and synthetic fibers; and avoiding pillows and bedding that contain feathers, down, or animal fibers.

Is there a role for complementary and alternative medicine?

Traditional Chinese herbal medicines and other so-called natural remedies, such as aloe vera gel, evening primrose oil, and St. John's wort, are popular nonprescription

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treatments for AD, although systematic reviews show no firm evidence of their effectiveness (30, 31).

Are measures to control airborne allergens and food allergens helpful?

Numerous studies have confirmed the benefit of house dust mite reduction for controlling AD (32, 33). For patients with positive dust-mite skin prick tests and allergen-specific IgE, such measures as thorough vacuuming, eliminating old carpeting, and occlusive mattress covers may be helpful. Immunotherapy for dust mites has been successful in patients with elevated dust-mite-specific IgE (34), but it has limited usefulness in the general AD population because there are many other airborne triggers, such as plant allergens and animal dander.

Despite evidence that many AD patients have positive IgE to cat and dog dander, there is some evidence that early pet exposure may be helpful in reducing the risk for AD.

A cohort study of 4578 German children recruited at birth showed a statistically significant negative association between the development of AD and exposure to dogs as household pets during the first 2 years of life. There was a borderline protective effect of keeping small furry animals (hamsters, rabbits, and guinea pigs) as pets during the first year (35).

Food and airborne allergens are evaluated with RAST testing as noted in the Diagnosis section. In the presence of food allergy (i.e., allergen-specific IgE together with clinical symptoms), the NIAID Guidelines recommend avoidance of the food, along with nutritional counseling and growth monitoring. Attempts to modify food-induced reactions through drugs, immunotherapy, or desensitization are not recommended for the treatment of AD.

What are the roles of sunlight and phototherapy?

Moderate exposure to natural sunlight remains a simple and cost-free adjunct

to AD therapy. Moisturizing sunscreens that have a moderate, rather than maximum, sun protection factor are recommended, to avoid completely interfering with the beneficial effects of UV rays for AD while still protecting against deleterious overexposure. Sunburn, windburn, and overheating can worsen AD, and therefore prolonged exposure to the elements should be avoided.

Phototherapy with PUVA (oral psoralens plus ultraviolet A exposure), narrow-band UVB, UVA-1, and combined UVA/UVB have all been used successfully, particularly for adult AD, but these are considered second- to third-line treatments (36). Their use is generally reserved for chronic AD that responds inadequately to topical mid-strength to superpotent corticosteroids and calcineurin inhibitors. Disadvantages include relatively high expense; inconsistent third-party coverage; inconvenient treatment schedules (generally 3 times/wk for 4–8 wk, followed by weekly maintenance); short-term risk for sunburn, causing AD exacerbation; and long-term risks for freckling, hyperpigmentation, premature aging, and skin cancer.

When and how should topical corticosteroids be used?

For decades, topical corticosteroid therapy has remained the cornerstone of AD treatment (23), but it can be challenging to choose the optimum preparation from among the dozens that are available over-the-counter and by prescription. Because AD is associated with dry skin, corticosteroid ointments are generally preferred by patients and caregivers over lotions, creams, and foams that are less moisturizing.

Treatment should be initiated with the lowest potency corticosteroid that will clear AD (Table 4). The more potent the medication, the greater the risk for side effects, including local acne-like eruptions, skin atrophy, and stretch marks; systemic effects, such as



Figure 7. Prurigo nodularis resulting from chronic excoriation of atopic dermatitis on the forearm extensor surface of a teenager. Photo credit: Susan V. Bershad, MD.

23. Hanifin JM, Cooper KD, Ho VC, et al. Guidelines of care for atopic dermatitis, developed in accordance with the American Academy of Dermatology (AAD)/American Academy of Dermatology Association "Administrative Regulations for Evidence-Based Clinical Practice Guidelines". *J Am Acad Dermatol*. 2004;50:391-404. [PMID: 14988682]
24. Saeki H, Furue M, Furukawa F, et al. Guidelines for management of atopic dermatitis. *J Dermatol*. 2009;36:563-77. [PMID: 19785716]
25. Sugarman JL, Parish LC. Efficacy of a lipid-based barrier repair formulation in moderate-to-severe pediatric atopic dermatitis. *J Drugs Dermatol*. 2009;8:1106-11. [PMID: 20027938]
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27. Grimalt R, Menegaud V, Cambazard F. The steroid-sparing effect of an emollient therapy in infants with atopic dermatitis: a randomized controlled study. *Dermatology*. 2007;214:61-7. [PMID: 17191050]

Table 4. Classes of Topical Corticosteroids*

Class	Description	Uses in AD†	Examples‡
1	Superpotent	Short-term treatment of severe refractory lesions in nonfacial body areas of adults	Clobetasol propionate; betamethasone dipropionate ointment; halobetasol propionate; fluocinonide 0.1%
2	Potent	Same as class 1	Betamethasone dipropionate cream; mometasone furoate ointment; diflorasone diacetate; halcinonide; fluocinonide gel/ointment; desoximetasone gel
3	Upper mid-strength	Longer-term for moderate to severe nonfacial lesions in adults; short-term for severe nonfacial lesions in children	Fluticasone propionate ointment; fluocinonide-emollient cream 0.05%; betamethasone valerate; desoximetasone
4	Mid-strength	Same as class 3, plus short-term for moderate nonfacial lesions in children	Flurandrenolide ointment; mometasone furoate cream/lotion; triamcinolone acetonide; fluocinolone acetonide ointment; hydrocortisone valerate ointment
5	Lower mid-strength	Same as class 4, plus intermediate-term for nonfacial lesions in children	Fluocinolone acetonide shampoo; flurandrenolide cream/lotion/tape; fluticasone propionate cream/lotion; prednicarbate; desonide lotion; hydrocortisone valerate cream; fluocinolone acetonide cream
6	Mild	Intermediate to longer term for all ages and body areas	Alclometasone dipropionate; fluocinolone acetonide oil/cream/solution; desonide cream/ointment/foam/gel
7	Least potent	Same as class 6	Hydrocortisone acetate lotion/cream/ointment/spray

Source: National Psoriasis Foundation.

*The lowest potency corticosteroid that clears AD should be used. AD = atopic dermatitis.

†Short-term = 1–2 weeks; Intermediate-term = 2–4 weeks; Longer-term = more than 1 month.

‡Unless otherwise noted, the generic name refers to all commercially available forms. Nonfacial areas do not include areas of thin skin (i.e., flexural creases, neck, axillae, and genital/groin area), which should be treated the same as facial skin.

28. Na JJ, Hwang JS, Park HJ, et al. A new moisturizer containing physiologic lipid granules alleviates atopic dermatitis. *J Dermatolog Treat*. 2010;21:23-7. [PMID: 19626524]

29. Lee JH, Lee SJ, Kim D, et al. The effect of wet-wrap dressing on epidermal barrier in patients with atopic dermatitis. *J Eur Acad Dermatol Venereol*. 2007;21:1360-8. [PMID: 17958842]

30. Zhang W, Leonard T, Bath-Hextall F, et al. Chinese herbal medicine for atopic eczema. *Cochrane Database Syst Rev*. 2004;CD002291. [PMID: 15495031]

31. Ernst E, Pittler MH, Stevinson C. Complementary/alternative medicine in dermatology: evidence-assessed efficacy of two diseases and two treatments. *Am J Clin Dermatol*. 2002;3:341-8. [PMID: 12069640]

32. Sanda T, Yasue T, Oohashi M, et al. Effectiveness of house dust-mite allergen avoidance through clean room therapy in patients with atopic dermatitis. *J Allergy Clin Immunol*. 1992;89:653-7. [PMID: 1545086]

suppression of the hypothalamic–pituitary–adrenal axis (37); osteoporosis; and ocular problems like cataracts and glaucoma that are concerns when treating the eyelids.

Mid- to high-potency corticosteroids in classes 1 to 5 can be highly effective for AD but should be reserved for nonfacial, nonintertriginous skin areas in adults and adolescents. Corticosteroid classes 6 and 7, the lowest-strength nonfluorinated compounds, are recommended for AD in many clinical situations, including the care of young children and treatment of the face, neck, groin area, and skin creases in all age groups. Recommendations are based primarily on studies of childhood AD, because large RCTs of adult AD treatment are lacking. Although twice-daily application has been the traditional practice, evidence suggests that once-daily treatment is equally effective and considerably less costly. The drawback of prescribing once-daily topical therapy is that missing 1 application can result in suboptimal therapy.

A systematic review of the clinical and cost-effectiveness of once-daily versus twice-daily therapy found equal benefits and considerable cost savings for less frequent application (38).

What is the role of other pharmacologic treatments?

A summary of drug therapy for AD is found in Table 5. Adjunctive therapy with oral antihistamines for pruritus is often recommended, although high-quality evidence of its effectiveness is lacking. Nighttime scratching not only worsens AD lesions but disrupts sleep, diminishing quality of life.

A sedating antihistamine like diphenhydramine or hydroxyzine can be used nightly for symptom relief and sleep induction. During the day, a non-sedating antihistamine like cetirizine or loratadine may be taken, although evidence supports the use of these agents only to alleviate airborne allergies that might contribute to AD, not to relieve the pruritus.

An evidence-based Cochrane review found no large RCTs examining the efficacy of antihistamines to relieve the pruritus associated with AD, and meta-analysis of poorer quality studies was inconclusive (39).

Table 5. Drug Treatment for Atopic Dermatitis

<i>Class (Examples)</i>	<i>Mechanism of Action</i>	<i>Dosage</i>	<i>Benefits</i>	<i>Side Effects</i>	<i>Notes</i>
Topical corticosteroids (see Table 4)	Anti-inflammatory	Apply topically once to twice daily to affected areas	Decreased pruritus and redness	Thinning of skin, striae, steroid acne; possible systemic absorption with HPA axis suppression	Class 6 and 7 are the most versatile and can be used on the face and skin creases in all ages; classes 1–5 should be reserved for short- and intermediate-term in older patients; ointments are more emolient than creams or lotions.
Topical calcineurin inhibitors (tacrolimus 0.03%, 0.1% ointment; pimecrolimus 1% cream)	Anti-inflammatory	Apply topically once to twice daily to affected areas	Decreased pruritus and redness; steroid-sparing; especially useful around the eyes	Local stinging; potential risk for rare lymphomas and nonmelanoma skin cancers	0.1% is approved for ages 15 y and older; considered second-line by most third-party insurers; useful as 1st-line therapy for “steroid phobic” patients.
Topical antipruritic agents (doxepin cream)	Tricyclic drug with antihistamine effects	Apply at bedtime	Decreased pruritus	High absorption causes drowsiness	Not studied in children.
Oral antihistamines, sedating (diphenhydramine, hydroxine)	Antihistamine	Single dose at bedtime; diphenhydramine: 12.5–25 mg (children 6 y and older), 25–50 mg (adults); hydroxine: 5 mg (under 20 lb) 10 mg (children over 20 lb) 10–25 mg (adults)	Decreased allergic symptoms and pruritus; induces sleep in most	Paradoxical hyperactivity in some children; dry mouth; urine retention	Many patients find these agents helpful for night-time pruritus, but systematic reviews have not confirmed efficacy in AD.
Oral antihistamines, nonsedating (loratidine, cetirazine, fexofenadine, acrivastine)	Antihistamine	Check recommended dose for age and weight	Decreased allergic symptoms; possible relief of pruritus	Dry mouth; urine retention; drowsiness or hyperactivity; loratidine and fexofenadine are the least sedating	No high-quality evidence of efficacy in AD.
Topical antibacterial agents (mupirocin 2% cream and ointment; retapamulin 1% ointment)	Antibacterial against <i>Staphylococcus</i> and <i>Streptococcus</i>	Apply to crusted areas, pustules, surrounding skin, nostrils, and fingernails twice daily	FDA-approved to treat impetigo, which commonly occurs secondary to AD lesions; promotes healing; reduces the spread of infection	Potential resistance	Bacterial culture and sensitivity testing of lesions is recommended before using antibacterial medications; colonization tends to recur rapidly on discontinuation of treatment; diluted bleach baths help to delay this risk
Oral antibiotic (oxacillin, cephalixin, axithromycin, doxycycline)	Antibacterial	Choose the appropriate drug based on culture and sensitivity testing; dosage and number of days as recommended for soft tissue infection	Reduced bacterial infection, usually <i>S. aureus</i> that exacerbates pruritus and inflammation; promotes healing; reduced spread of infection	Potential resistance; allergic reactions, such as pruritus, GI distress, diarrhea, and vomiting; sulfa drugs are associated with the Stevens-Johnson syndrome; tetracyclines are not for pediatric use due to dental and bone absorption	Limit oral antibiotic use to the treatment of culture-positive infection with crusting, exudates, and/or pustules
Oral corticosteroids (prednisone tablets; prednisolone oral solution)	Anti-inflammatory	Single morning dose with food, rapidly tapering to 0 over 7–10 d; prednisone 0.5–1 mg/kg on day 1; prednisolone 0.6–1.2 mg/kg on day 1	Rapid induction of temporary remission in recalcitrant AD	GI distress; steroid acne; temporary elevations of blood sugar and blood pressure; mood disorders; HPA axis suppression	Occasional judicious use can effect dramatic improvement; rebound is likely without strict adherence to a topical regimen

Table 5 (continued). Drug Treatment for Atopic Dermatitis

Oral immuno-modulators	Immune suppression	Check appropriate dosage for age and weight; begin at low end of the range	May be useful in severe refractory AD	Serious and life-threatening effects are possible, including immune suppression, anemia, hepatotoxicity, nephrotoxicity, and opportunistic infections; check individual drug side effects	Use only in consultation with a specialist in severe inflammatory skin disease
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AD = atopic dermatitis; FDA = U.S. Food and Drug Administration; GI = gastrointestinal; HPA = hypothalamic-pituitary-adrenal.

33. Tan BB, Weald D, Strickland I, et al. Double-blind controlled trial of effect of housedust-mite allergen avoidance on atopic dermatitis. *Lancet*. 1996;347:15-8. [PMID: 8531541]
34. Werfel T, Breuer K, Rueff F, et al. Usefulness of specific immunotherapy in patients with atopic dermatitis and allergic sensitization to house dust mites: a multi-centre, randomized, dose-response study. *Allergy*. 2006;61:202-5. [PMID: 16409197]
35. Zirngibl A, Franke K, Gehring U, et al. Exposure to pets and atopic dermatitis during the first two years of life. A cohort study. *Pediatr Allergy Immunol*. 2002;13:394-401. [PMID: 12485314]
36. Meduri NB, Vandergriff T, Rasmussen H, et al. Phototherapy in the management of atopic dermatitis: a systematic review. *Photodermatol Photoimmunol Photomed*. 2007;23:106-12. [PMID: 17598862]
37. www.fda.gov/ohrms/dockets/ac/05/slides/2005-4099s1_03_FDA-Cook.ppt
38. Green C, Colquitt JL, Kirby J, et al. Clinical and cost-effectiveness of once-daily versus more frequent use of same potency topical corticosteroids for atopic eczema: a systematic review and economic evaluation. *Health Technol Assess*. 2004;8(III):iv, 1-120. [PMID: 15527699]
39. Klein PA, Clark RA. An evidence-based review of the efficacy of antihistamines in relieving pruritus in atopic dermatitis. *Arch Dermatol*. 1999;135:1522-5. [PMID: 10606058]

The topical antipruritic agent doxepin, a tricyclic drug, provides effective relief but has systemic effects, such as sedation, due to high absorption (40).

What are the best approaches to treat and prevent bacterial superinfection?

Staphylococcus aureus skin colonization occurs in most patients with AD, a finding first noted by Leyden and coworkers in 1974 (41) and subsequently confirmed by others (42). Guidance from the AAD indicates that bacterial superinfection should be treated with topical and/or oral antibiotics (23). Mupirocin cream or ointment, applied twice daily, can be alternated or layered under corticosteroid preparations on fissured and crusted areas to treat culture-proven localized infection. Studies have shown that AD patients carry these organisms in their nostrils and under the fingernails; these areas should be treated with twice-daily topical applications of mupirocin for 7 to 10 days to reduce risk for reinfection. Widespread crusted lesions require appropriate oral antibiotic therapy based on bacterial culture and sensitivity results.

Recently, diluted bleach baths have become popular for prophylaxis of bacterial colonization of AD. This practice by itself has not been studied in RCTs, but it was used successfully in combination with intermittent intranasal application of mupirocin ointment for 3 months in an RCT designed to reduce the severity of AD in patients with clinical signs of secondary bacterial infections (43). Inexpensive and easy to use, this method is

accomplished by adding one-half cup of household chlorine bleach to a 40-gallon bathtub of warm water. The long-term safety of exposure to diluted bleach has not been established.

What are calcineurin inhibitors, and when should they be used?

Calcineurin is a calcium-dependent phosphatase that activates the T-cells and dendritic cells responsible for AD inflammation. There are 2 U.S. Food and Drug Administration (FDA)-approved topical drugs in the calcineurin-inhibitor class, tacrolimus and pimecrolimus. Their mechanism of action is similar to cyclosporine, an oral calcineurin inhibitor. As immune-system modulators, they are useful steroid-sparing agents and are suitable for corticosteroid-resistant AD in patients older than 2 years of age. Evidence suggests that they are more effective than class 7 topical corticosteroids (44). In several studies, tacrolimus was found to be superior to pimecrolimus (45). Tacrolimus is available in a high-potency ointment for adults and a milder formulation for ages 2 to 15 years; pimecrolimus cream comes in 1 formulation for ages 2 and up.

On areas of very thin skin, such as the face, neck, and groin, calcineurin inhibitors can be beneficial to avoid skin atrophy. These inhibitors are especially useful on the eyelids, where long-term corticosteroids may thin the skin and create risks for cataracts and glaucoma.

Topical tacrolimus and pimecrolimus, generally considered to be second-line

therapy, may be employed as first-line options when patients express so-called steroid phobia, an irrationally heightened but not uncommon fear of corticosteroid side effects.

The only local side effect of calcineurin inhibitors is occasional mild stinging. The prescriber should be aware that these drugs carry an FDA “black box” warning stating that topical use has been associated with rare cases of lymphoreticular and skin cancer. However, professional task forces of the AAD and the AAAI have disputed a causal relationship (46, 47).

When should systemic therapy be considered?

AD usually responds quickly to a topical corticosteroid with or without a calcineurin inhibitor, but treatment-resistant AD may require systemic corticosteroid therapy. Although experts agree that a short 7- to 10-day rapidly tapering course of oral prednisone or prednisolone can bring about dramatic remission, RCTS are lacking (23). Rapid rebound is a frequent consequence, particularly when patients do not adhere to an emollient regimen for dry skin and topical medications for active lesions. Long-term systemic corticosteroids are contraindicated for AD.

In a select group of adult and teenaged patients with very severe refractory AD, evidence-based practice guidelines recommend carefully weighing the risks and benefits of using systemic immunomodulating agents (23, 48). Effective drugs in this group include cyclosporine, tumor necrosis factor inhibitors, interferon gamma, mycophenolate mofetil, and azathioprine. This type of therapy should be considered only in consultation with a physician who specializes in severe inflammatory skin conditions. In experienced hands, the vast majority of cases can be managed less aggressively.

Systemic antibiotic therapy is warranted for culture-positive AD lesions that are widespread or

recalcitrant to topical antibacterial agents. For vesicular and crusted lesions that test positive for herpes simplex or varicella zoster, rapid initiation of treatment with systemic acyclovir, famciclovir, or valacyclovir is critical.

What are the complications?

The most common complication of AD is bacterial superinfection, also referred to as impetiginization, from the word impetigo, meaning a superficial skin infection with strains of *Staphylococcus* or *Streptococcus*. The usual clue to this diagnosis is a honey-colored crust overlying areas of AD, particularly those that have been subjected to excoriation and friction. Bullous impetigo, a blistering form of the infection, is relatively rare, but may occur. When vesicles and bullae are present, an urgent consideration is eczema herpeticum, a serious form of disseminated herpes simplex or varicella zoster.

AD is also frequently associated with development of asthma and allergic rhinitis.

When should a dermatologist be consulted for management of a patient with AD?

A dermatologist should be consulted for management of recalcitrant AD when topical treatment options, including corticosteroids and calcineurin inhibitors, have failed and the patient requires systemic therapy with corticosteroids, PUVA or other light treatments, or immunomodulators.

How long should therapy be continued?

The duration of treatment for AD varies greatly among patients. Although moisturizers should be used continuously for areas of dry skin in AD patients, drug therapy is required only for sites with active dermatitis. Once AD is under control, maintenance regimens, other than emollients and allergen avoidance, are unnecessary and potentially harmful.

40. Drake LA, Fallon JD, Sober A. Relief of pruritus in patients with atopic dermatitis after treatment with topical doxepin cream. The Doxepin Study Group. *J Am Acad Dermatol.* 1994;31:613-6. [PMID: 8089287]
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43. Huang JT, Abrams M, Tlougan B, et al. Treatment of *Staphylococcus aureus* colonization in atopic dermatitis decreases disease severity. *Pediatrics.* 2009;123:e808-14. [PMID: 19403473]
44. Reitamo S, Harper J, Bos JD, et al. 0.03 Tacrolimus ointment applied once or twice daily is more efficacious than 1% hydrocortisone acetate in children with moderate to severe atopic dermatitis: results of a randomized double-blind controlled trial. *Br J Dermatol.* 2004;150:554-62. [PMID: 15030341]
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49. www.aad.org/education/CPATinfo.htm.

Practice Improvement

Treatment... First-line treatment of AD consists of daily nonprescription emollients for dry skin and low-potency topical corticosteroids (classes 6 and 7) applied to facial and intertriginous active lesions. In adults and older children, lesions on other sites may be treated with limited courses of mid-strength to superpotent (classes 1 to 5) corticosteroids. Fissured and crusted areas frequently require topical or oral antibiotic therapy, based on culture and sensitivity results. Sedating antihistamines are indicated at bedtime for patients whose sleep is disrupted by pruritus. Second-line therapy includes topical calcineurin inhibitors, PUVA and other light treatments, and oral corticosteroids. The latter two modalities, as well as third-line therapy for severe recalcitrant AD with such immunomodulators as cyclosporine, may be considered in collaboration with a dermatologist specializing in inflammatory skin disorders.

CLINICAL BOTTOM LINE

What do professional organizations recommend regarding the management of patients with AD?

Official guidelines for treating AD have been established by the AAD (23) and the Joint Task Force on Practice Parameters that represents 3 societies: the American College of Allergy, Asthma and Immunology; the American Academy of Allergy, Asthma, and Immunology; and the Joint Council of Allergy, Asthma and Immunology (48).

How is quality of care for AD evaluated in the United States?

The AAD, as part of its program for maintaining professional certification, has recently developed the Clinical Performance Assessment Tool (CPAT) for dermatology, a set of Web-based disease-specific modules to evaluate quality of care in medical practices. Evidence-based treatment guidelines and evaluation standards for AD diagnosis and management, which include the severity scoring instruments listed in Table 5, are incorporated into the CPAT module titled "Atopic Dermatitis" (49).

In the Clinic Tool Kit

Atopic Dermatitis (Eczema)

PIER Module

<http://pier.acponline.org/physicians/diseases/d922/d922.html>

PIER module on atopic dermatitis from the American College of Physicians (ACP).

PIER modules provide evidence-based, updated information on current diagnosis and treatment in an electronic format designed for rapid access at the point of care.

Patient Information

www.annals.org/intheclinic/toolkit-atopic-dermatitis.xhtml

Patient information that appears on the following page for duplication and distribution to patients.

www.nlm.nih.gov/medlineplus/eczema.html

www.nlm.nih.gov/medlineplus/ency/article/000832.htm

www.nlm.nih.gov/medlineplus/ency/article/000870.htm

Information on eczema, dyshidrotic eczema, and nummular eczema from National Institutes of Health's MedlinePLUS.

www.niams.nih.gov/Health_Info/Atopic_Dermatitis/atopic_dermatitis_ff.asp

www.niams.nih.gov/Portal_En_Espanol/Informacion_de_Salud/Dermatitis_atopica/default.asp

Easy-to-read information on eczema, in English and Spanish, from the National Institute of Arthritis and Musculoskeletal and Skin Diseases.

www.skincarephysicians.com/eczemanet/proper_treatment.html

Guidelines of care for atopic dermatitis due out soon from the American Academy of Dermatology.

<http://guidance.nice.org.uk/CG57>

Management of atopic eczema in children from birth up to the age of 12 years, from the National Institute for Health and Clinical Excellence, released in 2007.

THINGS YOU SHOULD KNOW ABOUT ATOPIC DERMATITIS (ECZEMA)

In the Clinic
Annals of Internal Medicine

What is atopic dermatitis (eczema)?

- A chronic skin disease that causes dry, irritated skin.
- The cause is unknown, but it is believed to run in families.
- Occurs most often in babies and children, but can appear at any age.
- Is not contagious.
- Allergens like dust mites, mold, pollen, or pet dander.
- Eating certain foods, such as fish, soy, wheat, milk, or peanuts.
- Skin infections.
- Long, hot baths or showers or anything else that dries the skin.

What are the symptoms?

- An itchy rash, which appears most commonly on the face, hands and feet, behind the knees, and inside the elbows.
- Scratching may cause redness, swelling, scaling, and cracked, crusting, thick skin.
- Symptoms tend to come and go, worsening sometimes and improving or clearing up other times.
- People with atopic dermatitis (eczema) are more likely to have asthma and allergies, such as hay fever.

What factors can worsen atopic dermatitis (eczema)?

- Irritants, like wool, soap, perfume, chlorine, or smoke.

How is it treated?

- Practice a skin care routine to keep skin well-moisturized.
- Avoid things that lead to flares, such as allergens or irritants.
- Follow your doctor's advice for using skin creams or ointments to control swelling and reduce allergic reactions.
- Take antibiotics as directed for any infection related to the skin disorder.
- Antihistamines may reduce nighttime scratching.
- Light therapy, sometimes combined with a drug called psoralen, may reduce symptoms.
- Consult a specialist before getting the the smallpox vaccine because it can cause serious problems.

For More Information

www.skincarephysicians.com/eczemanet/daily_care.html
www.skincarephysicians.com/eczemanet/moisturizing_cleansing.html
Articles on skin care for people with eczema, from the American Academy of Dermatology.

www.niams.nih.gov/Health_Info/Atopic_Dermatitis/default.asp
Patient handout on atopic dermatitis from the National Institute of Arthritis and Musculoskeletal and Skin Diseases.

www.skincarephysicians.com/eczemanet/index.html
EczemaNet is a comprehensive online resource for patients and caregivers from the American Academy of Dermatology.

www.nationaleczema.org/
The National Eczema Association provides sponsors support networks and education on eczema

ACP[®]

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INTERNAL MEDICINE | Doctors for Adults

1. A 35-year-old man is evaluated for an acute rash on his face. The rash is painful and somewhat pruritic, and his left eye is swelling shut. He has a history of long-standing atopic dermatitis, which has been reasonably well-controlled with regular use of emollients and topical corticosteroids. His current medications are triamcinolone ointment, tacrolimus ointment, and hydroxyzine. Lately he has been nonadherent to his medication regimen because of recent travel and therefore has been scratching more. He is allergic to sulfa.

On physical examination, vital signs are normal. The conjunctivae are inflamed, but no purulence is noted. No gross abnormalities of the cornea are visible. The left preauricular lymph nodes are slightly enlarged and tender.

Which of the following is the most likely diagnosis?

- A. Acute allergic contact dermatitis
 - B. Acute atopic dermatitis
 - C. Eczema herpeticum
 - D. Staphylococcus aureus pyoderma
2. A 51-year-old woman is evaluated for a several-year history of persistent hand dermatitis. She has worked as a hairdresser for years, and, while she has had rashes in the past, she has never before experienced such a persistent problem. Her hands are itchy and sometimes have very painful cracks. Regular emollient use has improved the rash in the past but is not helping at this time. She wears gloves at work most of the time, but she is not sure if they are latex or vinyl. She notes improvement on weekends when she does not work, although the dermatitis does not completely clear. Symptoms improved significantly when she went on vacation. She has allergic rhinitis and hypertension and has a history of mild eczema during childhood. Her mother had asthma. She currently takes hydrochlorothiazide and

nasal corticosteroids intermittently and has no known drug allergies.

On physical examination, the skin on the hands is thickened and hyperkeratotic, especially on the palms, where there are poorly defined plaques, dry scale, and fissures but no pustules. Skin on the dorsal hands is red and scaly. The surface of the nails is rough. Other areas of skin, including the soles of the feet, are not involved.

Which of the following is the most appropriate diagnostic test?

- A. Epicutaneous patch testing
 - B. Lymphocyte stimulation assay
 - C. Prick testing
 - D. Radioallergosorbent test
3. A 65-year-old man is evaluated for a generalized, intensely pruritic eruption that has been slowly progressing over the last 6 months. He has been treated with topical corticosteroids for 4 months for widespread eczema without relief of pruritus or change in clinical appearance. He has never had a skin biopsy. He does not have a personal or family history of asthma, atopic dermatitis, allergic rhinitis, or psoriasis.

On physical examination, temperature is 37.5°C (99.5°F), blood pressure is 135/85 mm Hg, pulse rate is 84/min, and respiration rate is 14/min. Skin examination reveals erythema with scale affecting greater than 90% of the body surface area. Alopecia, nail dystrophy, and ectropion (turning inside out of the eyelid) are present. There is thickening and fissuring of the skin on the palms and soles. Bilateral axillary and inguinal lymphadenopathy is present. The mucous membranes are not involved.

Which of the following is the most appropriate next step in management?

- A. Antinuclear antibody assay
- B. Cyclosporine
- C. Phototherapy
- D. Rapid plasma reagin test
- E. Skin biopsy

4. A 30-year-old woman is evaluated for a pustular rash on her central face. She has a 5-year history of dandruff and a recurrent, red, scaly, itchy rash behind and in her ears and on the medial aspects of her cheeks. For several months she has been self-treating the rash with daily application of her husband's eczema cream (triamcinolone acetone 0.1%). The rash was initially controlled using this treatment, but for the past month a new acne-like rash has appeared. She began using the triamcinolone twice daily within the last week but has noted no improvement.

On physical examination, papules and pustules on an erythematous base are observed in a perioral distribution. The remainder of the examination is normal except for atopic dermatitis involving the popliteal fossae.

In addition to discontinuing triamcinolone, which of the following is the most appropriate treatment?

- A. Clobetasol cream
- B. Benzoyl peroxide gel
- C. Neomycin ointment
- D. No further treatment

5. A 20-year-old male college student is having trouble sleeping because of an itchy rash on his hands and on the inner creases of his elbows. His pruritus has been recurring for several years, becoming more severe in the winter despite his use of a daily moisturizer. His history is remarkable for seasonal allergies, and the only medication he uses is a nasal corticosteroid in the fall.

Which of the following is the most appropriate treatment for this patient's condition?

- A. Topical corticosteroids
- B. Topical tacrolimus
- C. Oral antibiotics
- D. Topical antifungals
- E. Oral corticosteroids

Questions are largely from the ACP's Medical Knowledge Self-Assessment Program (MKSAP, accessed at http://www.acponline.org/products_services/mksap/15/?pr31). Go to www.annals.org/intheclinic/ to complete the quiz and earn up to 1.5 CME credits, or to purchase the complete MKSAP program.