Atopic Dermatitis: An Evidence-Based Management Approach

Case Study and Commentary, Gary C. Pien, MD, PhD, and Jonathan M. Spergel, MD, PhD

A topic dermatitis (AD) is a chronic, relapsing inflammatory skin disorder that occurs in 10% to 20% of children and 1% to 3% of adults [1]. The eczematous dermatitis appears as erythematous patches, papules, and/or plaques with or without scaling. The classic distribution of these lesions varies with age. In infancy through early childhood, extensor and exposed skin surfaces are involved, whereas in older children, flexural surfaces are more characteristically affected. In adults, the face, neck, chest, hands, feet, and genitals can also be affected. Generalized involvement can occur with severe disease. Other hallmarks of AD include xerosis (dry skin) and pruritus, resulting in a vicious itch-scratch cycle. Excoriations may be apparent and, in the chronic state, result in lichenification of the skin. Recurrent skin infections by bacteria, viruses, and dermatophytes may also occur.

AD significantly impacts quality of life, resulting in anxiety, discomfort, sleep disturbance, and poor self-confidence and self-image [2–4]. In addition, AD is often the first manifestation of the “atopic march,” appearing in the first year of life in 60% of patients and in 90% by the fifth year [1]; a majority of these individuals will then go on to develop allergic rhinitis and/or asthma later in life [5]. Although chronic and relapsing, AD can be successfully controlled. This article provides a brief overview of the current understanding of disease pathogenesis with an emphasis on applying this knowledge towards evidence-based management, with the goal of optimizing clinical outcomes.

CASE STUDY
Initial Presentation

A 12-month-old boy presents to his pediatrician’s office for a well-child visit. During the interview, his mother reports that he has had eczematous lesions since early infancy, which she has successfully treated with over-the-counter moisturizing lotions. However, the eczema is now widespread with significant nocturnal pruritus, and the lotions no longer alleviate the child’s symptoms.

• What is the initial approach to evaluation of eczema?
Eczema is a general term referring to a form of dermatitis that can result from a number of pathologic processes. Its appearance varies with chronicity of the lesions, but it is typically characterized by erythema, scales, vesicles, and pruritus. AD is arguably the most common cause of eczema. The hallmarks of AD are pruritus and eczematous eruptions localized to classic anatomic sites. In infants and young children, lesions typically occur on the face, scalp, and extensor surfaces of the extremities. In older children and adults, lesions appear over flexural surfaces, and the skin may be lichenified as a result of chronic inflammation and scratching. However, a variety of other skin disorders, immunodeficiencies, autoimmune diseases, malignancies, infections, and metabolic derangements can mimic AD and must be considered in the differential diagnosis (Table 1). Investigation of suspected AD requires a careful history and physical examination, which can be helpful in weighing alternate etiologies of a patient’s constellation of symptoms. Once other diagnostic entities have been ruled out and AD is identified, the search can begin for triggering/exacerbating factors, barriers to therapy, and optimal management strategies.

**History**

The child was born full-term via spontaneous vaginal delivery. The pregnancy and the child’s postnatal course were uneventful. He was exclusively breastfed for 3 months and transitioned to a milk-based formula without incident. Solid foods were introduced at 6 months of age without difficulty. The eczematous lesions first appeared at 1 month of age on the cheeks and were mild. At age 6 months, the lesions also appeared over the extensor surfaces of the patient’s elbows. The eczema would remit and recur episodically and had previously responded to hydration with over-the-counter lotions. However, for the past month, the patient has had persistent eczema over his elbows, knees, abdomen, and back. In addition, he has been irritable at night and has been having difficulty sleeping due to constant scratching of the lesions. Past medical history and systems review is otherwise unremarkable. He has normal growth and development. He is attentive and wary of the examiner but in no apparent distress. Lymphatic, cardiac, pulmonary, abdominal, genital, neurologic, and musculoskeletal examinations are normal. Examination of the skin reveals diffuse xerosis with multiple 3- to 4-cm erythematous plaques over the extensor aspects of the elbows, knees, torso, and back. There are linear excoriations over the areas within arms’ reach, without erythema, edema, weeping, or tenderness. The patient’s right thumb and both wrists are lichenified.

**Diagnosis**

The patient’s clinical history and physical examination is highly suggestive of AD. The lichenification of his thumb and wrists indicates that this condition has been chronic and likely suboptimally controlled. The classic appearance and distribution of the lesions and their pruritic nature support a diagnosis of AD. Also helpful is the family history of atopy. Although seborrheic dermatitis is a potential diagnosis, the appearance and location of this patient’s lesions are not consistent with this disease. Importantly, he has an unremarkable review of systems. These factors make other diagnostic considerations (Table 1) less likely. If the patient’s history had suggested poor growth, chronic diarrhea, and/or recurrent, persistent, severe, or unusual infections, then an immunodeficiency (eg, zinc deficiency, Wiskott-Aldrich syndrome) would be important to investigate.

**What is the general approach to management of AD?**

Once a diagnosis of AD has been established, evaluation and management should be tailored to the individual patient and the severity of the disease. Patient education is an integral part of management of AD. In addition, medical therapy for AD involves a care plan that mimics the action plans developed for asthma [6]. Patients should be provided with a daily maintenance routine with provisions for escalating therapy at the start of symptoms and for prolonged or severe symptoms. Thus, most treatment plans include a combination of skin hydration, topical anti-inflammatory agents, and possibly avoidance of exacerbating factors.

**What are the components for maintenance therapy?**

For patients with AD, daily routines are directed at maintaining good skin hygiene. Several studies have demonstrated that in AD, the skin has impaired barrier function, resulting in depletion of water and vital lipids. Transepidermal water loss measurements show that affected skin from AD patients has a decreased ability to retain water as compared with normal skin [7,8]. AD-affected skin also demonstrates
abnormal electrical impedance, which is dependent upon the lipid content within the stratum corneum [9]. Ceramides are particularly depleted in AD due to the high expression of sphingomyelin deacylase [10,11]. Recently, loss-of-function variants of filaggrin, a key protein in the formation of the cornified envelope epidermal barrier, have been identified as major risk factors for AD [12–14]. These findings help explain why xerosis is frequently observed in AD. Thus, routine hydration of the skin with emollients helps restore barrier function by providing both water and lipids.

There are many available over-the-counter emollients, such as lotions, creams, and ointments. Compared with creams and ointments, lotions generally are not as effective for maintaining skin hydration as they are of lower viscosity and do not retain moisture in the skin as effectively. Therefore, creams and ointments are recommended for maintaining

<table>
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<th>Table 1. Differential Diagnosis of Atopic Dermatitis</th>
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<tr>
<td><strong>Distinguishing Features</strong></td>
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<tr>
<td><strong>Skin disorders</strong></td>
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<tr>
<td>Contact dermatitis                          Intensely pruritic, erythematous papular eruption after contact with irritant or allergic trigger</td>
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<tr>
<td>Ichthyoses                                    Group of disorders characterized by fish-like scales and dry, thickened skin</td>
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<tr>
<td>Netherton’s syndrome                         Erythroderma, ichthyosis linearis circumflexa, bamboo hair, failure to thrive, atopy</td>
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<tr>
<td>Nummular eczema                               Highly pruritic, eczematous, coin-shaped patches with papules, scaling, crusting, and weeping; typically distributed over trunk, lower extremities</td>
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<tr>
<td>Psoriasis                                    Erythematous papules and plaques with silver scaling; variable forms and presentations</td>
</tr>
<tr>
<td>Seborrheic dermatitis                        Erythema with greasy scales, classically located on scalp, face; can be on back, chest, and skin folds</td>
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<td><strong>Immunodeficiencies</strong></td>
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<tr>
<td>Hyper-IgE syndrome                           Recurrent staphylococcal infections, skin and sinopulmonary abscesses, elevated serum IgE, eczema, retained primary teeth, coarse facial features, scoliosis, pathologic bone fractures, hyperextensible joints</td>
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<td>Severe combined immunodeficiency (SCID) with maternal engraftment</td>
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<tr>
<td>SCID with Omenn’s syndrome                   Exudative erythroderma, lymphadenopathy, hepatosplenomegaly, eosinophilia, diarrhea</td>
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<tr>
<td>Wiskott-Aldrich syndrome                     Triad of recurrent infections, microcytic thrombocytopenia, and eczema with petechiae</td>
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<tr>
<td><strong>Autoimmune diseases</strong></td>
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<tr>
<td>Dermatitis herpetiformis                     Pruritic, papulovesicular lesions over extensor surfaces of the extremities, trunk; commonly associated with celiac disease</td>
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<tr>
<td>Dermatomyositis                              Heliotrope rash over eyelids, Gottron’s papules over joint extensor surfaces, sun sensitivity, periangual erythema, telangiectasis, symmetric proximal muscle weakness, calcinosis</td>
</tr>
<tr>
<td>Pemphigus foliaceus                         Scaly, crustng erosions over erythematous base on face, scalp, chest, and back</td>
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<tr>
<td><strong>Malignancies</strong></td>
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<tr>
<td>Letterer-Siwe disease                        Seborrheic-like eruption over scalp, face, trunk, and groin; associated with fever, hepatosplenomegaly, lymphadenopathy, cytopenias</td>
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<tr>
<td>Cutaneous T-cell lymphoma                    Indolent eruption of erythematous, scaly patches or plaques that may be pruritic; evolving to generalized erythroderma with atrophic or lichenified skin</td>
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<td><strong>Infections</strong></td>
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<tr>
<td>Dermatophytosis                              Can affect scalp, body, hands, feet, nails, or crural folds; erythematous, well-defined patches with scaling; spreads centrifugally with central clearing and vesicular borders</td>
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<td>Scabies                                      Small, erythematous papules and linear burrows along intertriginous areas; pruritus</td>
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<tr>
<td><strong>Metabolic derangements</strong></td>
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<tr>
<td>Multiple carboxylase deficiency              Ataxia, seizures, alopecia, eczematous eruptions, failure to thrive</td>
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<td>Niacin deficiency                            Dry, hyperkeratotic, hyperpigmented skin over sun-exposed areas; vomiting, diarrhea, weakness, dementia</td>
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<tr>
<td>Phenylketonuria                              Mental retardation, light pigmentation, eczematous eruptions, may smell “mousy”</td>
</tr>
<tr>
<td>Zinc deficiency                              Bullous, pustular dermatitis with alopecia, failure to thrive, diarrhea, and recurrent infections; accentuated in peri-orificial and acral distributions</td>
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skin hydration. Several prescription-only, nonsteroidal barrier creams have been approved by the U.S. Food and Drug Administration (FDA), including N-palmitoylethanolamine cream (MimyX, Stiefel Laboratories, Coral Gables, FL) [15], MAS063DP cream (Atopiclair, Sinclair Pharmaceuticals, Godalming Surrey, UK) [16], and ceramide-dominant cream (Epiceram, Ceragenix, Denver, CO). Another ceramide-dominant cream (TriCeram, Osmotics, Denver, CO) is available without a prescription [17]. The effectiveness of these nonsteroidal barrier creams has yet to be rigorously evaluated. Nevertheless, the routine use of emollients can help restore and maintain normal skin hydration and barrier function. Emollients, with or without occlusion, are often most effective when applied to damp skin. This can be done after daily lukewarm bath soaks for 15 to 20 minutes to help retain moisture and provide symptomatic relief. Data supporting the effectiveness of lukewarm baths or mild soaps are lacking; however, the use of no soap, a mild soap, or synthetic surfactant-based detergent bars typically results in less drying of the skin [18].

In addition to emollients, H$_1$-antihistamines can be helpful in daily maintenance care plans for ameliorating pruritus associated with AD. Itching is one of the primary symptoms of AD, and once it has begun, the pruritus exhibits allopainasia and spreads to the surrounding skin. Controlling pruritus and scratching may provide secondary benefits, including less skin irritation, reduced lichenification, and decreased risk of superinfection from skin breakage. Oral antihistamines are preferred over topical agents due to the potential for allergic contact hypersensitivity [19,20]. Large randomized, double-blind, placebo-controlled studies on the efficacy of antihistamines in patients with AD are lacking. Indeed, data to conclusively support the routine use of sedating or non-sedating antihistamines are sparse [21–23]. This is not surprising given that inflammatory mediators other than histamine, such as eosinophil-derived products and Th2 T-cell cytokines, contribute to the disease process in AD [24]. However, a subset of AD patients may benefit from sedating antihistamines, including patients with disrupted sleep or concomitant atopic conditions such as allergic rhinitis or urticaria. The value of a restful night of sleep, aided by the soporific effects of hydroxyzine and diphenhydramine, should not be underestimated. Nonsedating agents may provide more symptomatic relief at higher doses. In a study conducted in 178 adults with AD, 20 mg to 40 mg of cetirizine resulted in less pruritus as compared with placebo or standard-dose (10 mg) cetirizine, but higher-dose cetirizine was more sedating [25]. Although evidence to support the routine use of antihistamines for relief of pruritus in AD is lacking, the decision to use such agents must be tailored to each individual and may be helpful in daily maintenance therapy.

**What are the treatment strategies for acute flares?**

Patients with AD commonly experience relapses, resulting in acute eczematous eruptions that vary in location and severity. Physicians should take these factors into consideration and recommend an AD care plan that ideally provides measures for escalating therapy commensurate with disease severity and duration. Active skin lesions contain both Th1 and Th2 inflammatory cells, which synthesize a number of proinflammatory cytokines and chemokines that may be dysregulated [26,27]. An area of current debate is whether an intrinsic skin barrier defect (eg, a mutation in filaggrin) results in dysregulated inflammation upon exposure to allergens and irritants, or whether the primary disorder is of the immune system, which in turn disrupts the skin barrier as a result of abnormal inflammatory responses. Respectively, these “outside-inside” [28–30] and “inside-outside” [24,31] hypotheses have been recently reviewed, and both provide a rationale for treating acute inflammatory skin lesions with immunosuppressive agents.

A variety of topical anti-inflammatory agents are available, including topical corticosteroids and calcineurin inhibitors. Topical corticosteroids are classified by their potency, ranging from class I (superpotent) to class VII (low potency) (Table 2). Corticosteroids exert anti-inflammatory effects through a number of mechanisms, including the inhibition of NF-κB, a family of transcription factors that up-regulate the expression of several proinflammatory cytokines [32]. Topical corticosteroids are effective in AD but are not without potential adverse effects, including skin atrophy, striae formation, acneiform eruptions, and telangiectasias [33]. Less commonly, systemic absorption with adrenal suppression has been reported, particularly in infants and small children who have a greater body surface area to mass ratio [34,35]. These risks are greatest when higher-potency topical corticosteroids are used, when they are applied to areas of thinner skin (eg, face, axilla, genitalia, and intertriginous areas), when applied under occlusion, or when used for prolonged periods of time. Of note, fluticasone propionate 0.05% cream (class V, lower-mid potency) has been used in young children with moderate to severe AD for up to 4 weeks without demonstrable evidence of adrenal suppression [36,37] or adverse skin changes [38]. Clinical judgment that includes weighing the risks and benefits of different potency topical corticosteroids is therefore instrumental in the management of AD. In general, topical corticosteroids should only be applied to eczematous lesions, with emollients used on unaffected skin. Corticosteroid potency should be titrated to disease severity, using the lowest potency agent that is still effective. A tiered approach may be helpful, with patients...
having lower potency agents available for mild to moderate symptoms, and mid- to higher-potency agents for moderate to severe flares. In addition, the maximum duration of usage is inversely proportional to corticosteroid potency. Superpotent agents should only be used for a few days, high-potency corticosteroids (classes II–IV) for up to 3 weeks, and low-potency agents (classes V–VII) for longer as necessary. High-potency, fluorinated agents should be avoided in areas of thinner skin where a low-potency corticosteroid or topical calcineurin inhibitor would be better tolerated. Once lesions begin to resolve, it may be appropriate to switch to a lower-potency agent. Once in remission, topical corticosteroids should be discontinued and daily maintenance skin care should be resumed.

Topical calcineurin inhibitors (eg, tacrolimus, pimecrolimus) are also effective for AD and are corticosteroid-sparing alternatives. Tacrolimus and pimecrolimus both inhibit calcineurin, a phosphatase necessary for activation of the transcription factor NF-AT, interleukin-2 gene transcription, and subsequent activation of T cells. Several studies comparing the efficacy of tacrolimus 0.1% ointment with topical corticosteroids in patients with AD demonstrate that tacrolimus is approximately equivalent to a mid-potency corticosteroid [39–41]. One study demonstrated pimecrolimus to be inferior to betamethasone-17-valerate 0.1% cream (class V) in AD, suggesting that pimecrolimus is equivalent to a low-potency corticosteroid [42]. Thus, pimecrolimus is usually effective for mild to moderate AD, whereas tacrolimus can be used for moderate to severe AD. Topical calcineurin inhibitors are usually well-tolerated and may be preferable in sensitive areas, such as the face, axilla, genitalia, and intertriginous skin folds. A local burning sensation is the most frequently reported side effect. Furthermore, these agents have not been associated with the adverse skin changes seen with topical corticosteroid overdose [43,44]. However, in 2006 the FDA issued black box warnings for both tacrolimus and pimecrolimus due to the potential risks for lymphoma and other malignancies from immunosuppression. Although beyond the scope of this discussion, currently available data indicate that these agents are effective, safe alternatives to topical corticosteroids for the treatment of AD [45,46].

### Initial Treatment

This patient’s history and physical examination are consistent with mild to moderate AD. The pediatrician prescribes hydrocortisone hydrochloride 2.5% cream to be applied twice daily to active lesions at sensitive sites and for mild flares, triamcinolone 0.1% ointment twice daily to remaining lesions and moderate flares, and hydroxyzine 5 mg at bedtime as needed for itching. In addition, the physician provides education on AD and recommends good skin hygiene with twice-daily application of over-the-counter cream.

### Table 2. Potency of Topical Corticosteroids

<table>
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<tr>
<th>Class</th>
<th>Potency</th>
<th>Agents</th>
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<tbody>
<tr>
<td>Class I: Superpotent</td>
<td>Betamethasone dipropionate 0.05% cream, ointment, and gel</td>
<td>Halobetasol propionate 0.05% cream and ointment</td>
</tr>
<tr>
<td>Class II: Potent</td>
<td>Aclometasone dipropionate 0.1% ointment</td>
<td>Betamethasone valerate 0.1% cream and ointment</td>
</tr>
<tr>
<td>Class III: Upper to mid-potency</td>
<td>Amcinonide 0.1% cream and lotion</td>
<td>Betamethasone dipropionate 0.05% cream</td>
</tr>
<tr>
<td>Class IV: Mid-potency</td>
<td>Clocortolone pivalate 0.1% cream</td>
<td>Prednicarbate 0.1% cream</td>
</tr>
<tr>
<td>Class V: Lower to mid-potency</td>
<td>Betamethasone dipropionate 0.05% lotion</td>
<td>Triamcinolone acetonide 0.1% cream</td>
</tr>
<tr>
<td>Class VI: Mild potency</td>
<td>Amcinonide 0.1% cream and lotion</td>
<td>Betamethasone valerate 0.1% ointment</td>
</tr>
<tr>
<td>Class VII: Low potency</td>
<td>Halobetasol propionate 0.05% cream and ointment</td>
<td>Fluocinonide 0.05% cream, gel, ointment, and solution</td>
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</table>

Table 2 outlines the potency of topical corticosteroids as determined by their concentration and clinical effect. The classification system is based on the US Food and Drug Administration’s guidelines, which categorize the potency of topical corticosteroids into eight classes (I–VII). This classification system is used to guide the selection of appropriate treatments for AD, taking into account factors such as disease severity, location of lesions, and patient history. The table includes commonly used agents and their respective potency classes. For instance, betamethasone dipropionate 0.05% cream is classified as class I (superpotent), whereas fluocinonide 0.05% cream is classified as class II (potent). The treatment plan for AD should be individualized based on the patient’s specific needs and the potential risks associated with each class of corticosteroid.
to damp skin. After 2 weeks, the mother calls and reports that the topical corticosteroid creams are helping to induce remission, but the patient's lesions quickly return after discontinuing these medications. She is concerned that food and/or environmental allergies are contributing to his disease.

• What are the potential exacerbating factors or triggers?

AD, as its name implies, can be exacerbated by exposure to allergens in sensitized individuals. Both food and environmental allergens have been implicated as offending agents in AD, but the incidence varies among pediatric and adult populations. In adults, less than 2% of patients with AD have a food allergy as an exacerbating factor [47]. In contrast, 10% to 33% of pediatric patients with severe AD cases have an associated food allergy [48,49]. The presence of a food allergy is more likely with increasing disease severity. Thus, the diagnosis of a food allergy should be considered in young children who have generalized or severe AD or those who have recalcitrant disease despite aggressive therapy. Milk, soy, egg, peanut, and wheat accounted for 90% of food allergies implicated in AD [50]. Elimination of offending foods can result in clinical improvement of AD [51,52].

In addition to foods, environmental aeroallergens can be significant triggers in AD. Aeroallergens may be more common triggers in older children and adults, as these populations are more likely to have become sensitized. More than 85% of adults with AD have evidence of IgE-mediated hypersensitivity to environmental allergens [53]. In children with AD, IgE-mediated aeroallergen sensitization first appears between the ages of 2 and 5 years and predominates over food allergies by age 5 [54]. However, in many patients, aeroallergen sensitization is only relevant for allergic rhinoconjunctivitis and not AD. Nevertheless, in a subset of AD patients, inhalational or topical exposures to aeroallergens have been shown to exacerbate AD [51,55]; therefore, measures to limit environmental allergen exposure may help improve AD [56].

Given these considerations, referral to a specialist is not routinely necessary. However, consultation with a specialist at any age is appropriate to help confirm the diagnosis of AD when the disease is recalcitrant despite aggressive skin care management. A specialist can help treat refractory disease in patients who respond poorly to conventional therapy and can help evaluate for potential exacerbating factors when clinically indicated. In limited cases, systemic immunosuppressants, including corticosteroids, cyclosporine A, azathioprine, and mycophenolate mofetil, may be useful [57]. Similarly, in the most severe cases, intensive treatment with ultraviolet light or PUVA (psoralen plus ultraviolet A) photochemotherapy may also be considered.

Referral to a Specialist

The physician explains that food allergies are more likely to exist when AD is particularly severe or refractory to aggressive topical therapy. The physician also explains that given this patient's young age, sensitization to environmental aeroallergens is unlikely. Continued patient education and aggressive skin hygiene measures are encouraged. An outpatient consultation with a specialist is arranged at the request of the parents.

A dietary inventory reveals that the toddler regularly consumes whole cow's milk, eggs, wheat, and a variety of fruits, meats, and vegetables. Skin prick testing to all foods is negative. Testing to environmental allergens, including cats, is also negative. The specialist recommends generous application of moisturizing cream to damp skin 3 times daily. For mild AD lesions, triamcinolone 0.1% cream twice daily is prescribed. After a discussion with the parents regarding using off-label medications and FDA black box warnings, tacrolimus 0.1% ointment twice daily is prescribed for moderate to severe lesions or lesions occurring at sensitive locations. Hydroxyzine 5 mg at bedtime is recommended as needed for pruritus.

One month later, the patient's AD is in remission. His skin is clear and he is sleeping through the night without any pruritus. His mother is continuing to observe good skin hygiene and is applying emollients twice daily. He has not required any topical anti-inflammatory agents for almost 3 weeks.

SUMMARY

AD is a complex, multifactorial disease and is often a harbinger of the atopic march. Studies demonstrate that both skin barrier dysfunction and immunodysregulation play key roles in pathogenesis of AD. In addition to genetic factors, environmental factors can also contribute to AD in a subset of patients. Our understanding of the etiology and pathogenesis of this intricate disease continues to evolve and available treatment options allow for optimization of clinical outcomes. An exciting topic at the frontier of current research is whether repair of the defective skin barrier in AD can prevent or subdue the drumbeat of the atopic march. The epidermal barrier dysfunction in AD is thought to allow for the abnormal penetration of allergens, which then go on to induce systemic sensitization [58,59]. By aggressively managing AD early in childhood, it may be possible to alter the atopic march, thereby reducing the risk of allergic rhinitis and asthma later in life. Future research is likely to be very enlightening.

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References


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1. A 3-month-old boy presents to your office for evaluation of eczema. On review of systems, the patient’s mother reports a history of recurrent bloody diarrhea. On physical examination, eczematous lesions and petechiae are noted. In considering the differential diagnosis of atopic dermatitis (AD), which condition would be important to rule out in this patient?

A. Netherton’s syndrome  
B. Niacin deficiency  
C. Seborrheic dermatitis  
D. Wiskott-Aldrich syndrome  
E. Zinc deficiency

2. A 5-year-old girl with moderate AD presents for a follow-up appointment. Her parents report that her eczema has previously been difficult to control but finally responded to triamcinolone acetonide 0.1% ointment. The parents are concerned that the patient’s eczema may relapse and have continued to apply this ointment to her skin twice daily for the past month. On physical examination, the skin on her cheeks demonstrates mild atrophy. Which of the following would be the best intervention at this time?

A. Continue prophylaxis with routine use of triamcinolone acetonide 0.1% ointment twice daily  
B. Provide education on good skin care and consider switching the patient to a nonsteroidal agent, such as tacrolimus 0.1% ointment or pimecrolimus 1% cream, on an as-needed basis  
C. Refer the patient to an allergist for evaluation of possible food allergies  
D. Stop application of all over-the-counter and prescription topical agents  
E. Switch the patient to pimecrolimus 1% cream applied twice daily to avoid steroids

3. A 15-year-old boy presents for evaluation of a pruritic, erythematous, maculopapular eruption on his abdomen that has lasted for several weeks. On physical examination, the patient’s skin is clear except for a periumbilical, circular lesion as described. Which of the following questions would be most helpful in establishing a diagnosis?

A. Do you wear a belt with a metal buckle, pants with metal buttons, or other metal jewelry?  
B. Does the rash get worse when you are near pets?  
C. Do you have a food allergy to milk or dairy products?  
D. Do you have chronic or recurrent diarrhea?  
E. Do you have significant sun exposure, and do you use sunblock?

4. A 6-week-old formula-fed female infant presents to the pediatrician’s office for evaluation of eczema. The patient’s lesions are consistent with AD but are severe and have a generalized distribution. The parents have been applying moisturizing cream to damp skin twice daily, and triamcinolone acetonide 0.1% cream has been applied twice daily for the past 3 weeks. However, the lesions have only modestly improved. Which of the following would be the best intervention at this time?

A. Begin systemic cyclosporine A therapy for recalcitrant AD  
B. Obtain a total serum IgE level to evaluate for hyper-IgE syndrome  
C. Prescribe permethrin 5% cream to treat scabies  
D. Refer the patient to a specialist for evaluation and treatment of possible food allergies  
E. Switch the patient to pimecrolimus 1% cream applied twice daily to avoid steroids

5. A 50-year-old man presents for evaluation of an erythematos and pruritic patch on his trunk that has been slowly enlarging for the past several years. Physical examination reveals an annular, pink, dry plaque with evidence of skin atrophy. What is the most appropriate course of action at this time?

A. Advise empiric avoidance of peanuts and shellfish, as these are the most common triggers in adults  
B. Continue watchful observation as the lesion may spontaneously resolve  
C. Obtain serum zinc levels to rule out a mineral deficiency  
D. Prescribe tacrolimus 0.1% ointment twice daily  
E. Refer the patient to a specialist for a skin biopsy to evaluate for cutaneous T-cell lymphoma
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4. How helpful to your clinical practice was this article?
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5. What changes will you make in your practice as a result of reading this article?
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