A clinician's paradigm in the treatment of atopic dermatitis

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Successful management of atopic dermatitis requires a multipronged approach that includes skin barrier function care, use of topical or systemic agents, and identification and elimination of precipitating or exacerbating factors. Because the origin of atopic dermatitis is multifactorial and trigger factors differ among patients, treatment plans must be specific to the individual patient. This article offers an example of a permutational, or flexible, treatment paradigm. The approach utilizes 4 topical regimens—high-potency topical corticosteroids, lowest effective potency topical corticosteroids, topical calcineurin inhibitors (TCIs), or topical corticosteroid/TCI combinations—as initial therapy in a variety of induction protocols, as determined by the severity of a patient's condition and history. The paradigm permits treatment to progress from a chosen induction therapy to maintenance therapy. During the patient's induction therapy, as soon as an acceptable level of clearance is achieved, therapy should be adjusted to a maintenance regimen, such as monotherapy with either a TCI or a lowest effective potency topical corticosteroid (the latter used intermittently) or an alternation of the two agents. If there is no clearance or positive response with the initial induction protocol, the clinician should move to one of the alternative regimens. (J Am Acad Dermatol 2005;53:S70-7.)

Successful management of atopic dermatitis (AD) requires a multipronged approach that includes repair of barrier function and general skin care, use of topical or systemic agents, and identification and elimination of precipitating or exacerbating factors.[1] Because the origin of AD is multifactorial and trigger factors differ among patients, treatment plans should be individualized. Thus the ideal algorithm or paradigm for treatment of this disorder should be permutational, that is, the order of its components should be interchangeable (easily individualized) and centered around preventive measures.[2] This article discusses the various approaches to the treatment of AD, including prevention and available topical, systemic, and phototherapies (Table I), as well as combination regimens; it offers an example of a permutational paradigm.

Abbreviations used:

AD: atopic dermatitis
BMV: betamethasone valerate
FDA: Food and Drug Administration
IFN-γ: interferon gamma
IgE: immunoglobulin E
PUVA: psoralen plus UVA
TCI: topical calcineurin inhibitor
UVA: ultraviolet A
UVB: ultraviolet B

PREVENTIVE MANAGEMENT

Because AD is a chronic, often lifelong condition, identification of precipitating factors or triggers is a high priority and their avoidance should be an ongoing process. Immunologic triggers of AD vary for different patients and can include various foods, aeroallergens, irritants and contactants, hormones, stress, climate, and microorganisms such as *Staphylococcus aureus*.[3]

Proper skin care may also help to minimize disease exacerbations. Hydration of the skin is particularly important to offset xerosis resulting from reduction in skin ceramide concentrations, which, in turn, leads to water loss across the epidermal skin layer.[4] This dryness creates microfissures and cracks

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This article is part of a supplement supported by Connetics Corp, Palo Alto, California.
Disclosure: Dr Abramovits has received research support and/or is a consultant and lecturer for Fujisawa Healthcare, Novartis Pharmaceuticals, GlaxoSmithKline, Ferndale Laboratories, Abbott Laboratories, Connetics Corp, and Galderma.
0190-9622/$30.00
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doi:10.1016/j.jaad.2005.04.032

in the skin, which allow for entry of pathogens, antigens, or irritants, and causes general irritation. Such irritation then predisposes the patient to scratching of lesions, thereby exacerbating (or perpetuating) the disease process.[1] Several substances may worsen xerosis and should be avoided, including those detergents or soaps that promote significant defatting; smoke; toiletries that contain alcohol, preservatives, solubilizers, fragrances, or astringents; abrasive clothing; and various chemicals.[5] Appropriate clipping of nails and wearing of gloves also may minimize disease aggravation related to scratching, particularly during sleep.

Proper hydration may include application of an occlusive emollient after showers or baths (which should be warm, not hot), use of hydrophilic ointments and ceramide-rich barrier repair mixtures,[2,6-8] and the application of wet dressings in severely affected areas and chronic lesions.[1,5]

PHARMACOLOGIC TREATMENTS

Topical therapies

Topical corticosteroids form the cornerstone of pharmacologic treatment of AD and are the standard of care against which all other agents are compared.[9] These agents, which range in potency from ultra high to low (classes 1 to 7), have broad anti-inflammatory properties. In general, when treating AD, the lowest-potency topical corticosteroid likely to be effective may be considered initially and the patient switched to a higher potency corticosteroid as necessary. For some patients, initiating corticosteroid topical therapy with a higher potency (class 1 or 2) agent may be appropriate when the pruritus is intense, the lesions overtly inflammatory or lichenified, and to gain the patient's trust and improve compliance. However, higher potency agents are usually inappropriate for very young children and for highly permeable areas, such as the face and intertriginous areas in adults. In addition, most class 1 agents should not be used for longer than 2 weeks to minimize the likelihood of side effects. Topical corticosteroids are available in a variety of vehicles, including ointment, cream, lotion, oil, gel, foam, aerosol spray, and shampoo and thus afford the clinician a number of options when selecting a treatment regimen.

The topical calcineurin inhibitors (TCIs), tacrolimus[10-12] and pimecrolimus,[2,13,14] have also demonstrated effectiveness in AD. Tacrolimus is available as an ointment and is indicated for the treatment of moderate to severe AD[15] (and as cream and gel formulations are undergoing Food and Drug Administration [FDA]—approved clinical trials); pimecrolimus, available as a cream, is indicated for the treatment of mild to moderate disease.[16] Tacrolimus appears to carry a higher incidence of transient local side effects,[17] which appears to correlate with baseline disease severity.[18,19] Recent studies suggest that tacrolimus possesses superior efficacy compared with pimecrolimus in AD.[20]

Coal tar preparations also have been used to treat AD, but these agents are generally unacceptable to patients because of their propensity to stain clothing and other materials, and their offensive odor.[21]

Table I. Examples of available therapies for atopic dermatitis[5,59-66]

Topical agents
Corticosteroids
Topical calcineurin inhibitors
Tacrolimus
Pimecrolimus
Tar preparations
Anti-infectives
Antibiotics
Mupirocin
Antifungals
Azoles (eg, ketoconazole, clotrimazole)
Antihistamines
Doxepin
Barrier-repairing agents
Ceramide-based emollients and lipid mixtures
Phototherapy
Narrowband UVB
Wideband UVB
High- or medium-dose UVA
PUVA
Systemic agents
Antihistamines
Antibiotics
Corticosteroids
Interferon gamma*
Cyclosporine*
Mycophenolate mofetil*
Methotrexate*
Azathioprine

PUVA, Psoralen plus UVA; *UVA,* ultraviolet light A; *UVB,* ultraviolet light B.
*These drugs are not approved by the FDA for use in the treatment of AD.

Systemic therapies

Antihistamines. Oral antihistamines are sometimes prescribed to relieve the pruritus associated with AD, although their effectiveness has been questioned. The rationale for the use of antihistamines is that activation of histamine H_1 receptors is involved in the pathogenesis of pruritus, and blocking these receptors should ameliorate this symptom.

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