SUPPLEMENT

Successful Strategies In Atopic Dermatitis Management

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OFFICIAL JOURNAL OF THE DERMATOLOGY NURSES' ASSOCIATION
Successful Strategies in Atopic Dermatitis Management

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Noreen Heer Nicol and Mark Boguniewicz

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Noreen Heer Nicol, MS, RN, FNP, disclosed that she is a consultant and on the presenters’ bureau for Unilever and OrthoNeutrogena.

Mark Boguniewicz, MD, disclosed that he has received grant/research support from Novartis and Sinclair; and is on the advisory board of Graceway and Unilever.

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Successful strategies for managing atopic dermatitis require an accurate diagnosis, identification and elimination of exacerbating factors including irritants and allergens, adequate hydration of the skin, control of pruritus and infections, and appropriate use of topical anti-inflammatory and other medications. Proper patient education increases the chances of successful therapy.

As the Dermatology Nursing journal celebrates its 20th anniversary, the authors of this review celebrate working together for 20 years caring for patients with atopic dermatitis (AD). Over the past 2 decades, a great deal of progress has been made in the understanding of AD. Atopic dermatitis remains the most common chronic, relapsing skin disorder of infants and children, but can affect patients of any age. The prevalence of AD has increased globally and more than half of these patients go on to develop asthma and allergies (Kapoor et al., 2008). Atopic dermatitis places a significant economic burden on the patient, family, and society (Boguniewicz et al., 2007).

Successful strategies, particularly in those patients with moderate-to-severe disease, have been dependent on the commitment and expertise of the multidisciplinary approach led by physicians and nurses (Boguniewicz, Nicol, Kelsay, & Leung, 2008; Nicol, 1990) (see Figures 1a, 1b, 1c, & 1d). The patient and family with chronic AD have usually seen multiple health care providers who at times have given them confusing information or conflicting treatment plans. Frustrated, they come to the next evaluation hoping not only for consistency and answers, but also a cure. Learning about the chronic relapsing nature of atopic dermatitis, exacerbating factors, and appropriate treatment options is important for both patients and family members (Nicol & Boguniewicz, 1999). The purpose of this article is to review a treatment model developed and utilized by the Atopic Dermatitis Program (ADP) at National Jewish Health in Denver, Colorado for more than 20 years.

Epidemiology: Too Much Hygiene?

The prevalence of atopic dermatitis has increased more than three-fold since the 1960s (Schultz-Larsen & Hanifin, 2002). Atopic dermatitis is a global public health problem, with prevalence up to 20% in children and approximately 3% of adults in the

**OBJECTIVES**

Objectives

This continuing nursing educational (CNE) activity is designed for nurses and other health care providers who care for and educate patients and their families regarding atopic dermatitis. For those wishing to obtain CNE credit, an evaluation follows. After studying the information presented in this article, the nurse will be able to:

1. Heighten his/her awareness of the prevalence of atopic dermatitis, its impact on quality of life, and association with asthma and allergies.
2. Examine a multi-faceted approach to management of patients with atopic dermatitis including non-pharmacologic and pharmacologic interventions.
3. Summarize common interventions including hydration, moisturizers, and pharmaceutical agents.
4. Discuss appropriate safety issues related to topical and systemic therapies.

This article and the CNE answer/evaluation form are also available online at www.dermatologynursing.net

Complimentary CNE for this Supplement is available on page 19 and at www.dermatologynursing.net
United States and other industrialized countries (Williams et al., 1999). There is also a female preponderance for AD, with an overall female/male ratio of 1.3:1. There is evidence that atopy comes before the appearance of AD and that the skin disorder often precedes the development of other atopic diseases including asthma and allergic rhinitis, known as the “atopic march” (Boguniewicz, Eichenfield, & Hultsch, 2003; Nicol, 2005a). Epidemiologic studies indicate that more than 50% of children with AD will go on to develop asthma, allergic rhinitis, and other allergic diseases, often prior to age 3 (Kapoor et al., 2008). Since wide variations in prevalence have been observed within countries inhabited by similar ethnic groups, environmental factors seem to play a key role in determining disease expression. Some of the potential risk factors that have been associated with the rise in AD include small family size, increased income and education, migration from rural to urban environments, and increased use of antibiotics (a “Western lifestyle”) (Strachan, 1989; von Mutius, 2000). The “hygiene hypothesis” suggests that allergic diseases (“T helper-2-type”) might result from a lack of infections in early childhood (Leung, Boguniewicz, Howell, Nomura, & Hamid, 2004). More recently, this theory has been modified to account for the concomitant increase in autoimmune (“T helper-1-type”) diseases, and abnormalities in regulatory T cells (Chatila, 2005).

Although the outcome of AD may be difficult to predict in any given patient, the disease generally progresses to periods of remission as the patient grows older. Spontaneous
resolution of AD has been reported to occur after age 5 years in 40%-60% of patients affected during infancy, particularly if their disease is mild. In addition, more than half of adolescents treated for mild dermatitis may experience a relapse of disease as adults.

Pathophysiology and Genetics: Outside-In or Inside-Out?

Atopic dermatitis is an inflammatory skin disease that occurs in genetically prone individuals resulting in a defective skin barrier, defects in the innate immune system, and abnormal immunologic responses to irritants, allergens, and microbial organisms (Leung et al., 2004). Whether the pathophysiology of this disease favors an “inside-out” (immunologic dysregulation leading to skin barrier abnormality) or an “outside-in” explanation (barrier dysfunction causing immunologic perturbations), remains an area of active research.

Atopic dermatitis is characterized by abnormal skin barrier function associated with abnormalities in cornified envelope genes, reduced ceramide levels, increased levels of endogenous proteolytic enzymes, and enhanced transepidermal water loss (Cork et al., 2006). Use of soaps and detergents that raise skin pH can increase activity of endogenous proteases, leading to further breakdown of epidermal barrier function. The epidermal barrier may also be damaged by exposure to exogenous proteases from house dust mites and Staphylococcus aureus. This is worsened by the lack of certain endogenous protease inhibitors in atopic skin. These epidermal changes may contribute to increased allergen absorption into the skin and microbial colonization. Exposing the immune system of the skin to allergen compared to systemic measures directed at maintaining a healthy skin barrier. It is important to note that beside filaggrin gene mutations, other gene mutations affecting the skin barrier can occur. In addition, T helper-2-type cytokines can decrease expression of filaggrin and other skin barrier proteins (Howell et al., 2007; Kim, Leung, Boguniewicz, & Howell, 2008). Thus other gene products must also be involved in AD pathology. The “outside-in” or “inside-out” debate remains to be resolved.

Innate Immunity and Atopic Dermatitis: The Basic Immune Response

An intact skin barrier is the first line of defense against microbial organisms. In addition, we are born with an innate immune system which, unlike our adaptive immune system, does not require prior exposure and education to respond appropriately. Keratinocytes play a key role in the skin’s innate immune response. They express toll-like receptors and secrete pro-inflammatory cytokines, as well as anti-microbial peptides (such as human beta defensins and cathelicidins) in response to tissue injury or invading microbes (McGirt & Beck, 2006). Several studies have now demonstrated that keratinocytes from patients with AD produce reduced amounts of antimicrobial peptides due to suppression by Th2-type cytokines and this may predispose such individuals to skin colonization and infection with S. aureus, viruses, and fungi (Ong et al., 2002).

Clinical Findings: Recognizing the Disease

Despite all the advances over the past 20 years in understanding the pathogenesis of atopic dermatitis, there continues to be no single distinguishing feature of AD or a diagnostic laboratory test. The diagnosis is still based on the constellation of clinical findings described in 1980 by Hanifin and Rajka (1980) and major criteria listed in Table 1. Laboratory testing is not needed in the routine evaluation and treatment of uncomplicated AD. Serum IgE is elevated in the majority of patients with AD but levels do not always correlate with disease severity and do not point to specific triggers.

Atopic dermatitis typically presents during infancy and early childhood. These children frequently have a family history of asthma or allergies. The key feature of AD is pruritus that can disrupt sleep and interfere with daily activities. Atopic dermatitis in infants usually presents on the cheeks or scalp. Involvement of the extensor aspects of extremities and trunk is common, but the diaper area is typically spared. Infantile AD lesions tend to be symmetric, scaly, and erythematous. Weeping and crusting may be present in more severe or infected cases. Generalized dryness is common. The childhood phase occurs from age 2 to puberty. Flexural surfaces of the extremities, especially the antecubital and popliteal fossae, are most typically affected. Other involved skin areas include the neck, wrists and ankles, and the creases between the thighs and buttocks. Lichenification or an accentuation of skin markings associated with thickening of the skin due to repeated scratching can be a prominent feature (Nicol, 2003). Although most children outgrow their atopic dermatitis, almost all patients have persistent dry skin and some patients continue to have AD in adulthood or, in a minority of cases, have their onset after puberty. The adult form usually involves flexural aspects of the extremities and can be focal although, in some patients, it can be more diffuse. Hand and foot dermatitis can be a difficult-to-treat com-
ponent of eczema and in adults may be the only manifestation of AD for some patients. Patients may also have eyelid dermatitis. Lichenification is more prominent in older patients with chronic eczema.

Differential Diagnosis: Getting the Diagnosis Right

A number of inflammatory skin diseases (e.g., psoriasis), immunodeficiencies (e.g., Wiskott-Aldrich syndrome), skin malignancies (e.g., cutaneous T-cell lymphoma), genetic disorders (e.g., immune dysregulation polyendocrinopathy X-linked syndrome), infectious diseases (e.g., HIV), and infestations (e.g., scabies) may have symptoms and signs similar to atopic dermatitis. In an adult who presents with an eczematous dermatitis with no history of childhood eczema, respiratory allergy, or atopic family history, cutaneous T-cell lymphoma must be ruled out. Allergic contact dermatitis may also be in the differential. Ideally, biopsies should be obtained whenever the diagnosis is in question. The biopsies should be taken from three separate sites, because the histology may show spongiosis and cellular infiltrate similar to AD.

Current Management of Atopic Dermatitis: The Nuts and Bolts and Beyond

Successful strategies for managing AD require a systematic, multi-pronged approach. This approach emphasizes patient education; identifying and eliminating flare factors such as irritants, allergens, and emotional stressors; and incorporates skin hydration, pharmacologic, and non-pharmacologic therapies (Boguniewicz, Nicol et al., 2008; Boguniewicz & Nicol, 2002). Treatment plans should be individualized to address each patient’s skin disease reaction pattern, including the acuity of the rash. In patients refractory to conventional forms of therapy, alternative anti-inflammatory and immunomodulatory agents may be necessary.

Multidisciplinary Approach

While patients with atopic dermatitis of all severities could benefit from a multidisciplinary approach, the current health care system often creates roadblocks to such an approach by requiring or denying consults to appropriate specialists. Patients who are “failing” conventional therapy, those labeled as polyallergic (especially those believed to be allergic to multiple foods), patients with recurrent skin infections or on frequent courses of antibiotics, patients with concerns about medication side effects, patients whose disease is causing a significant impact on their or their family’s QOL, and those with need for in-depth education are all candidates for multidisciplinary management. It is worth remembering that families may have different levels of tolerance for a disease, thus, this approach may benefit more than just patients with severe AD.

At National Jewish Health in Denver, CO, the team is composed of pediatric allergist-immunologists with extensive experience in basic and clinical research in AD, a nurse practitioner/dermatology clinical specialist, pediatric psychiatrist, psychologists, allergy-immunology fellows-in-training, physician assistants, nurse educators, child life specialists, creative art therapist, social workers, dietitians, and rehabilitation therapists. Dermatologists are available for consultation if the diagnosis of AD is in question or alternative therapies, such as phototherapy, are being considered.
The philosophy of care is in keeping with the center’s approach to individualized medicine and patients undergoing comprehensive evaluation and treatment tailored to their needs and goals of the patient. The ADP provides single-day consultations, multidisciplinary team interaction, and continuity of care. This unique program allows for comprehensive evaluation and treatment of patients in an outpatient setting, typically over 5 to 10 days. In this controlled environment, patients and caregivers interact with members of the multidisciplinary team between 8 a.m. and 5 p.m., as well as overnight, if necessary, especially when evaluating sleep disturbance and response to interventions (Bender, Ballard, Canono, Murphy, & Leung, 2008). Importantly, patients and caregivers interact with other patients and families in formal and informal settings. The ongoing evaluation and response to therapy in the day program are reviewed in clinical review meetings with the patient/caregiver and plan of care conference that involves input from the various services. Rarely, patients may also be admitted as inpatients. By educating and caring for patients and families, the ADP can lead to sustained improvements in outcomes and quality of life (Kelsay, Carel, Bratton, Gelfand, & Klinnert, 2006).

Educating Patients and Caregivers

National Jewish, as a center of excellence for both asthma and atopic dermatitis, has used a similar multidisciplinary approach for these chronic inflammatory diseases. This approach confirms the importance of teaching patients skills to self-monitor and manage disease in a stepwise manner to a written action plan. The National Asthma Education and Prevention Program’s Expert Panel Report 3 (EPR-3) (2007) confirms the importance of teaching patients skills to self-monitor and manage asthma and to use a written asthma action plan, which should include instructions for daily treatment and ways to recognize and handle worsening disease. The EPR-3 emphasizes the use of a stepwise approach for a chronic disease like asthma, and has provided a good model for AD.

Education strategies include one-on-one communication, direct demonstration with reinforcement, group discussions, classroom teaching, written materials, and Atopic Dermatitis Home Care or Action Plan (see Table 2). This treatment model requires that all members of the multidisciplinary team teach the same key concepts and reinforce the messages being delivered to the patients and caregivers regardless of which educational strategy is incorporated. National Jewish has been providing patients who have participated in the ADP detailed, written step-care plans for the 20-year history of the program. Historically, these individualized teaching tools were extremely labor intensive and included a personalized booklet with annotated photographs, reflecting stages of disease and remission of each patient with appropriate skin care (see Figure 2). While current home care plans continue to be individualized, they follow a more standardized format and address treatment in a step-care manner (see Table 2). Since patients or caregivers may forget or confuse skin care recommendations given to them without a written plan, it should be reviewed and modified at followup visits. Clinical improvements achieved in the ADP are typically sustained for an extended period of time based on an outcomes study (Kelsay et al., 2006).

Direct demonstration of proper skin care includes topical application of agents and techniques such as wet wrap therapy. Watching the patient’s or caregiver’s current technique often reveals fundamental errors which helps providers understand why a patient may not be showing the expected therapeutic response. The

Table 2.
National Jewish Atopic Dermatitis Program Step-Care “AD Action” Plan

<table>
<thead>
<tr>
<th>MAINTENANCE OR DAILY CARE</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Take at least one bath or shower per day; use warm water, for 10-15 minutes.</td>
<td></td>
</tr>
<tr>
<td>Use a gentle cleansing bar or wash in the sensitive skin formulation as needed such as Dove® or Olay®.</td>
<td></td>
</tr>
<tr>
<td>Pat away excess water and immediately (within 3 minutes) apply moisturizer, sealer, or maintenance medication if directed.</td>
<td></td>
</tr>
<tr>
<td>Use cleansers as above.</td>
<td></td>
</tr>
<tr>
<td>Bathe as above for 10-15 minutes, once (and possibly twice) daily.</td>
<td></td>
</tr>
<tr>
<td>Use moisturizers as above to healed and unaffected skin, twice daily especially after baths and at mid-day total body.</td>
<td></td>
</tr>
<tr>
<td>Apply to affected areas of face, groin and underarms twice daily especially after baths low-potency topical corticosteroid, or topical calcineurin inhibitors, or other topical preparation as directed.</td>
<td></td>
</tr>
<tr>
<td>Apply to other affected areas of the body twice daily especially after baths low to mid- potency topical corticosteroid, or topical calcineurin inhibitors, or other topical preparation as directed.</td>
<td></td>
</tr>
</tbody>
</table>

Table continues on page 8
Table 2. (continued)
National Jewish Atopic Dermatitis Program Step-Care “AD Action” Plan

<table>
<thead>
<tr>
<th>MODERATE-TO-SEVERE ATOPIC DERMATITIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bathe as above for 10-15 minutes, two times a day, once before bedtime.</td>
</tr>
<tr>
<td>Use cleansers as above or consider an antibacterial cleanser (eg., Lever 2000®)</td>
</tr>
<tr>
<td>Use moisturizers as above to healed and unaffected skin, twice daily especially after baths and at mid-day total body.</td>
</tr>
<tr>
<td>Apply to affected areas of face, groin and underarms twice daily especially after baths _____________________ (low-potency topical corticosteroid), or _______________ (topical calcineurin inhibitors), or other topical preparation as directed _______________ (topical barrier repair cream, eg., Atopiclair® three times daily).</td>
</tr>
<tr>
<td>Apply to other affected areas of the body twice daily especially after baths _______________ (mid-to-high-potency topical corticosteroid), or _______________ (topical calcineurin inhibitors), or other topical preparation as directed.</td>
</tr>
<tr>
<td>Use wet wraps to involved areas selectively as directed.</td>
</tr>
<tr>
<td>Add other medications as directed: _____________________ (eg., oral sedating antihistamines, topical or oral antimicrobial therapy)</td>
</tr>
<tr>
<td>Pay close attention to things that seem to irritate the skin or make condition worse.</td>
</tr>
<tr>
<td>Contact your health care provider for additional evaluation or therapies. Oral steroids are not usually recommended.</td>
</tr>
<tr>
<td>Step down to moderate plan above as the skin heals.</td>
</tr>
</tbody>
</table>

REDUCE SKIN IRRITATION

- Wash all new clothes before wearing them. This removes formaldehyde and other irritating chemicals.
- Add a second rinse cycle to ensure removal of detergent. Residual laundry detergent, particularly perfume or dye, may be irritating when it remains in the clothing. Changing to a liquid and fragrance-free, dye-free detergent may be helpful.
- Wear garments that allow air to pass freely to your skin. Open weave, loose-fitting, cotton-blend clothing may be most comfortable.
- Work and sleep in comfortable surroundings with a fairly constant temperature and humidity level.
- Keep fingernails very short and smooth to help prevent damage due to scratching.
- Carry a small tube of moisturizer/sunscreen at all times. Daycare/school/work should have a separate supply of moisturizer.
- After swimming in chlorinated pool or using hot tub, shower or bathe using a gentle cleanser to remove chemicals, then apply moisturizer.

NOTES:

Seek psychosocial support.
Use reliable resources for information on atopic dermatitis:
National Jewish Health 1400 Jackson Street Denver, CO 80206 1.800.222.LUNG www.nationaljewish.org
National Eczema Association 4460 Redwood Hwy. Ste. 16-D San Rafael, CA 94903-1953 415.499.3474 / 800.818.7546 www.nationaleczema.org
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It is important to stress to patients and caregivers that they should review
advice or tips from outside sources with their clinicians. Even small changes to a treatment regimen can be detrimental or of little benefit, and can add significantly to the cost of therapy. Often, patients are switched to new prescriptive treatments on followup visit if the treatment response has not been optimal without first reviewing current care to see if it is being done properly and what else might be contributing to poor therapeutic response (ongoing exposure to an irritant, etc.). The costs of these changes are often borne by the family and contribute to non-adherence and frustration. An open and ongoing dialog between patients, caregivers, and their clinician improves the likelihood of adherence with the treatment plan and leads to improved outcomes.

**Proper Daily Skin Care**

Patients often are unclear about the role of skin care in management of atopic dermatitis. They frequently state that little time has been spent clarifying skin care instructions. Education regarding proper skin care is increasingly difficult to accomplish in the typical clinic visit due to time constraints. Studies have shown that patients fail to receive adequate explanation of the causes and triggers of AD or are not taught how to apply topicals, even though instruction and practical demonstrations may be associated with dramatic improvement in the treatment outcomes (Nicol, 2005b).

**Hydration**

Xerosis contributes to the development of epithelial microfissures, which favors the entry of microbial organisms, irritants, and allergens. This problem can become aggravated during the dry winter months and in certain work environments.

Proper daily skin care emphasizing hydration remains a cornerstone to a successful treatment plan. At the authors’ center, “soak and seal” was developed as a fundamental concept to teach proper skin care emphasizing use of hydration, moisturizers, cleansers, topicals, and medications to help maintain an intact skin barrier (Nicol, 1987; Nicol & Boguniewicz, 1999). Unfortunately, there has been confusion about how to hydrate and moisturize the skin. Thus, water avoidance is often mistakenly recommended even for patients with severe xerosis. Typically, evaporation and microfissuring occur when wet skin is not immediately covered by a protective layer of moisturizer, occlusive, or medication. In contrast, proper soak-and-seal method leads to re-hydration, sealing in of moisture, and repair of the damaged epidermal barrier.

Proper bathing or soaking the affected area should be done at least once per day for approximately 15 minutes in warm water making sure that involved areas are covered to avoid evaporation. A wet washcloth or towel can be used to cover face, head, neck, or body not covered my water to increase hydration (see Figure 3). Adding age-appropriate toys will help young children cooperate with the bath. Baths can be increased to up to three times daily during AD flares. Young children must be supervised during baths. Water temperature should feel comfortable to the patient, as the oft recommended “tepid” is usually too cool for most patients. Showers may be appropriate in patients with mild disease.

Bathing may also remove allergens from the skin surface and reduce general colonization by *S. aureus*. Additives to the bath remain at times unproven or controversial. Addition of oatmeal to the bath water may be soothing to patients but does not promote skin hydration, while bath oils may give the patient a false sense of lubrication and can make the tub slippery. The addition of bleach to bath water may be beneficial to patients with recurrent infections, particularly methicillin-resistant *S. aureus* infections; however, the amount of bleach per volume of water and frequency of baths have not been well studied. Bleach baths have the potential to cause significant skin irritation.

Another important reason why bathing is avoided is that patients may complain of discomfort or pain when
bathing and thus this fundamental skin care measure is avoided. This often results in progressive worsening of the atopic dermatitis. For pain and discomfort, patient or caregiver should be taught to avoid any irritating additives to the bath and to consider premedication for pain relief. This may include acetaminophen or ibuprofen, sedating agent (under close supervision in the tub!), or anxiolytic. However, distraction or other means of comforting the child are preferred by the therapists in our ADP.

**Cleansers**

The use of appropriate cleansers plays an important role in dry skin conditions such as atopic dermatitis. It is important that patients with AD are not using cleansers that have ingredients which are drying or irritating. Cleansers with minimal defatting activity and a neutral pH are preferred. Formulations that are dye-free and fragrance-free are less irritating and more appropriate for atopic skin. Our center recommends sensitive skin formulations of Dove®, Oil of Olay®, Vanicream®, and others. Antibacterial cleansers such as Lever 2000® may be helpful for patients with frequent folliculitis or recurrent skin infections. Patients should be instructed not to scrub with a washcloth.

**Moisturizers**

Patients frequently do not understand how the various vehicles of skin care products such as ointments, creams, lotions, and oils can affect treatment outcomes. In general, ointments seal the best and can be the most hydrating when used after bathing and they are formulated with the fewest additives. Since they are the most occlusive, in a hot, humid environment, they may trap perspiration, which may result in increased pruritus. Lotions and creams may be irritating due to added preservatives or fragrances. In addition, lotions contain more water than creams and may have a drying effect due to evaporation. While oils may go on easily, they are often less-effective moisturizers. Patients should be encouraged to carry moisturizers in small tubes with them at all times and to keep a separate supply in the daycare, school, or work environment.

Topical therapy to replace abnormal epidermal lipids, improve skin hydration, and decrease skin barrier dysfunction may be useful therapeutically. Recommending the use of moisturizers together with hydration may help re-establish and preserve the skin barrier (Lodén, 1995). Moisturizers can improve skin barrier function and reduce susceptibility to irritants (Lodén, Andersson, & Lindberg, 1999). Adding a moisturizer to a low-potency topical corticosteroid can improve clinical parameters in patients with AD (Hanifin et al., 1998). Moisturizers can also

Following hydration of the skin, patients should gently pat away excess water with a soft towel and apply the appropriate topical moisturizer or medication to prevent evaporation which completes “soak and seal.” Application of appropriate moisturizers or medications should occur within 3 minutes (“3 minute rule”). This rule has been promoted to patients by organizations such as the National Eczema Association (www.nationaleczema.org). Moisturizers should be obtained in the largest size available (typically one pound/480 g jars) since they typically need to be applied several times each day on a chronic basis. Plastic spoons or wooden tongue depressors should be used to remove topicals, especially ointments or creams from large jars to avoid contamination. Recommended moisturizers which are available in a one pound jar include Aquaphor® Ointment, Vanicream®, CeraVe® Cream, Ceta-phil® Cream, and Eucerin® Creme. Vegetable shortening (Crisco®) can be used as an inexpensive moisturizer. Patients and caregivers need to understand that petroleum jelly (Vaseline®) is a good occlusive preparation to seal in water; however, since it is a sealer, not moisturizer, it should be used after hydrating the skin. Of note, even young patients can be taught to apply moisturizers, which allows them to participate in their skin care. Moisturizers should be applied routinely rather than over or immediately prior to topical medications to avoid dilution or blocking of penetration of medication into skin.

 Patients and caregivers should understand that frequent and proper use of moisturizers together with hydration may help re-establish and preserve the skin barrier (Lodén, 1995). Moisturizers can improve skin barrier function and reduce susceptibility to irritants (Lodén, Andersson, & Lindberg, 1999). Adding a moisturizer to a low-potency topical corticosteroid can improve clinical parameters in patients with AD (Hanifin et al., 1998). Moisturizers can also

**Figure 3.** Bathing of child with atopic dermatitis with head involvement.
decrease the need for topical corticosteroids (Lucky, Leach, Laskarzewski, & Wenck, 1997).

A number of studies suggest that AD is associated with decreased levels of ceramides, contributing not only to a damaged permeability barrier, but also making the stratum corneum susceptible to colonization by *S. aureus* (Macheleidt, Kaiser, & Sandhoff, 2002). A ceramide-dominant emollient added to standard therapy in place of moisturizer in children with “stubborn-to-recalcitrant” atopic dermatitis resulted in clinical improvement (Chamlin et al., 2002). Ceramide-containing creams include TriCeram®, EpiCeram®, and CeraVe®. In addition, patients may benefit from other non-steroidal creams such as MAS063DP (Atopiclair®) (Boguniewicz, Zeichner et al., 2008), especially given concerns of some patients and caregivers regarding use of topical corticosteroids and topical calcineurin inhibitors.

**Topical Corticosteroids**

Topical corticosteroids have been the mainstay of treatment for atopic dermatitis for many years. Because of potential side effects, most physicians use topical corticosteroids primarily to control acute exacerbations. Studies suggest that once control of AD is achieved, long-term control can be maintained with twice weekly applications of topical corticosteroid to areas that have healed, but are prone to relapse (Berth-Jones et al., 2003; Van Der Meer, Glazenburg, Mulder, Eggink, & Coenaads, 1999).

Patients should be instructed carefully in the use of topical corticosteroids to avoid potential side effects (Nicol & Baumeister, 1997). On the face, the genitalia, and the intertriginous areas, only a low-potency topical corticosteroid is generally recommended. Patients should be instructed to apply topical corticosteroids to their skin lesions and to use emollients on uninvolved skin. Patients should be instructed to avoid placing moisturizer immediately over or under the topical corticosteroid. Failure of a patient to respond to topical corticosteroids is sometimes due in part to an inadequate supply. It is important to remember that it takes approximately 30 g of cream or ointment to cover the entire skin surface of an adult once. To treat the entire body twice daily for 2 weeks would require approximately two pounds or 900 grams of topical corticosteroids. An alternative that has been advocated for use in pediatrics is the finger tip unit (FTU), recently reviewed in detail by the British Dermatology Working Group (Bewley, 2008). The FTU – the amount of cream or ointment expressed from a 5-mm diameter nozzle, applied from the distal skin-crease to the tip of the patient’s index finger – can be used to calculate how much product is needed to cover affected areas, such as the face and neck, and hence the quantity which should be prescribed.

Topical corticosteroids are ranked into seven potency classes based on a vasoconstrictor assay. Because of their potential side effects, the ultra high-potency glucocorticoids should be used only for very short periods of time and in areas that are lichenified but not on the face or intertriginous areas. Mid-potency topical corticosteroids can be used for longer periods of time to treat chronic AD involving the trunk and extremities. Topical corticosteroids in gels are often in a propylene glycol base and may be irritating to the skin in addition to promoting dryness, thus limiting their use to the scalp and beard areas.

Topical corticosteroid potency and side effects are influenced by the molecular structure of the compound, the vehicle, the amount of medication applied, the duration of application, occlusion, as well as host factors including age, body surface area and weight, skin inflammation, anatomic location of treated skin, and individual differences in cutaneous or systemic metabolism. Side effects from topical corticosteroids are directly related to the potency ranking of the compound and the length of use, so it is incumbent on the clinician to balance the need for a more potent steroid with the potential for side effects. In addition, ointments have a greater potential to occlude the epidermis, resulting in enhanced systemic absorption when compared to creams. Side effects from topical corticosteroids can be divided into local side effects and systemic side effects resulting from suppression of the hypothalamic-pituitary-adrenal axis. Local side effects include the development of striae, skin atrophy, perioral dermatitis, and acne rosacea. The potential for potent topical glucocorticoid to cause adrenal suppression is greatest in infants and young children because of exposure to relatively larger body surface area. Of note, a study of children as young as 3 months of age found that flu tacson propionate 0.05% cream, a mid-potency formulation, was safe and effective even when applied on the face and over significant areas of the body for up to 1 month (Friedlander, Hebert, & Allen, 2002). It has been approved for use in children as young as 3 months for up to 4 weeks with flu tacson lotion approved for use in children 12 months and older. Topical corticosteroids continue to play a role in the management of AD, but with all the additional choices, patients and care providers need to be clear how and where they fit into the treatment plan (Bewley, 2008).

**Topical Calcineurin Inhibitors**

Topical tacrolimus and pimecrolimus have been developed as nonsteroidal topical calcineurin inhibitors (TCIs). The approval of the TCIs, tacrolimus ointment 0.03% and 0.1% and pimecrolimus cream 1%, as nonsteroidal agents for treating AD has represented a milestone in the management of this disease (Nicol, Hanifin, Tofte, & Boguniewicz, 2003). Both drugs have proven effective with a good safety profile for treatment up to 4 years with tacrolimus ointment (Berger et al., 2006) and up to 2 years with pimecrolimus cream (Paul et al., 2006). A fairly common side effect with TCIs is a transient burning sensation of the skin, although some patients may report more prolonged burning or stinging. Since treatment with TCIs is not associated with skin...
atrophy, they are particularly useful for treating eczema on the face, trigeminal regions, and atrophied skin. Use of tacrolimus ointment was associated with decreased colonization by *S. aureus*. Importantly, ongoing surveillance has not shown any trends towards increased frequency of viral infections especially eczema herpeticum or problems with responses to childhood vaccinations (Paul et al., 2006).

Currently, tacrolimus ointment 0.03% is approved for intermittent treatment of moderate-severe AD in children aged 2 years and older, tacrolimus ointment 0.1% for intermittent treatment of moderate-severe AD in adults, and pimecrolimus cream 1% is approved for intermittent treatment of patients aged 2 years and older with mild-moderate atopic dermatitis. While there is no evidence of a causal link of cancer and the use of TCIs, the United States Food and Drug Administration has issued a “black box or boxed” warning for tacrolimus ointment 0.03% and 0.1% (Protopic®, Astellas) and pimecrolimus cream 1% (Elidel®, Novartis) because of a lack of long-term safety data (see U.S. package inserts for Protopic®, Astellas and Elidel®, Novartis). The new labeling also states that these drugs are recommended as second-line treatments and that their use in children under the age of 2 years is currently not recommended. Of note, a Joint Task Force of the American College of Allergy, Asthma and Immunology and the American Academy of Allergy, Asthma and Immunology reviewed the available data and concluded that the risk/benefit ratios of tacrolimus ointment 0.03% and 0.1% and pimecrolimus cream 1% are similar to those of most conventional therapies for treating chronic relapsing eczema (Fonacier et al., 2005). In a recent case-control study of a large database that identified a cohort of 293,253 patients with AD, no increased risk of lymphoma was found with the use of TCIs (Arellano, Wentworth, Arana, Fernandez, & Paul, 2007).

Long-term safety studies with TCIs in patients with AD including infants and children are ongoing. Surveillance and recent reports have not shown a trend for increased frequency of viral superinfections especially eczema herpeticum (Hultsch, Kapp, & Spergel, 2005). The approval of topical calcineurin inhibitors for treating AD represents a significant addition to the management of this disease. There are situations in which topical calcineurin inhibitors may be advantageous over topical corticosteroids. These include treatmet of patients who are poorly responsive to topical steroids, patients with steroid phobia, and the treatment of face and neck dermatitis where ineffective, low-potency topical corticosteroids are usually used due to fears of steroid-induced skin atrophy. The potential use of topical calcineurin inhibitors as maintenance therapy is also intriguing for preventing AD flares. However, guidelines for use of topical corticosteroids versus calcineurin inhibitors in the management of atopic dermatitis are still needed. The PRACTALL Guidelines, an international initiative of the European and American Academies of Allergy, address a number of therapeutic issues in a step-wise fashion (Akdis et al., 2006).

**Tar Preparations**

Coal tar preparations may have antipruritic and anti-inflammatory effects on the skin although usually not as pronounced as those of topical glucocorticoids (Langeveld-Wildschut et al., 2000). Tar preparations may be useful in reducing the potency of topical glucocorticoids required in chronic maintenance therapy of AD. Newer coal tar products have been developed that are more acceptable with respect to odor and staining of clothes than some older products. Tar shampoos can be beneficial for scalp dermatitis, particularly with redness and inflammation, and are often helpful in reducing the concentration and frequency of topical glucocorticoid applications. Tar preparations should be used carefully on acutely inflamed or denuded skin, because this often results in skin irritation. Side effects associated with tars include folliculitis and photosensitivity.

**Wet-Wrap Therapy**

Wet-wrap dressings have been used successfully in managing recalcitrant atopic dermatitis at National Jewish for over 2 decades (Nicol, 1987, 1990). They reduce pruritus and inflammation by cooling the skin and improving penetration of topical corticosteroids. They also act as a protective barrier from the trauma associated with scratching. These actions can help the significant sleep disruption accompanying AD. Importantly, recent work has pointed to a beneficial effect of wet wrap therapy on the skin barrier with benefits continuing even after discontinuation of this treatment modality (Lee, Lee, Kim, & Bang, 2007). Of note, wet-wrap therapy should be reserved for flares of AD and not used as routine maintenance therapy.

While different variations of this treatment have been described, at National Jewish, we use wet clothing, such as long underwear, turtle necks, pajamas, and cotton socks placed over an undiluted layer of topical corticosteroids applied after bathing followed by a dry layer of clothing such as sweat suits or footed pajamas on top (see Table 3). When doing total body wraps on small children, place wet tube socks over hands first (see Figure 4), followed by wet layer of thinner cotton pajamas. Then, place the dry pair of socks over the hands followed by the heavier-footed pajamas (see Figure 5). At present, wet-wrap therapy is not indicated over topical calcineurin inhibitors. Treatment of the head requires skilled nursing care with use of gauze bandages (Kerlix®) and surgical netting (Spandage®) (see Figure 6). It is important to emphasize that wet-wrap therapy is not the wet-to-dry dressings used for debridement of wounds. Specifics of the procedure with detailed pictures showing step-by-step have been previously published and are now also available in a new video from National Jewish (Boguniewicz & Nicol, 2002, 2008). Children tolerate both selective areas of the body wrapped as well as total body wraps, which are rarely needed. Wet-wrap therapy is not only accepted but can be enjoyed when it is done with the proper education and
Wet wrap therapy will be used to relieve inflammation, itching, and burning of atopic dermatitis. Wet wraps facilitate the removal of scale and increase penetration of topical medications in the stratum corneum. Skin protection provided by the wraps allows healing to take place. Wet wrap therapy should only be used during flares of atopic dermatitis under the supervision of a health care provider. They should not be used as routine maintenance therapy.

PURPOSE

Topical medications and moisturizers
Tap water at comfortably warm temperature
Basin for dampening of dressings
Clean dressings of approximate size to cover involved area:
  a. **Face**: 2 to 3 layers of wet Kerlix® gauze held in place with SurgiNet®.
  b. **Arms, Legs, Hands & Feet**: 2 to 3 layers of wet Kerlix® gauze held in place with Ace® bandages or tube socks, or cotton gloves, or wet tube socks followed by dry tube socks. Tube socks may be used for wraps for hands and feet, and larger ones work as leg/arm covers.
  c. **Total Body**: Combination of above, or wet pajamas or long underwear and turtleneck shirts covered by dry pajamas or sweat suit. Pajamas with feet work well for the outer layer.

Blankets to prevent chilling.
Non-sterile gloves if desired.

PROCEDURE

Be certain that the patient’s room is warm and insure privacy. Gather supplies appropriate to the individual.

If wraps are to be applied to a large portion of the body, work with two people if possible. It is necessary to work rapidly to prevent chilling.

Explain the procedure to the patient and parent.

Fill the basin with warm tap water.

Usually, the patient will have had a soaking bath prior to this procedure or will soak the area in basin to be wrapped. Pat skin dry with a towel.

Apply the appropriate topical medications to affected areas and moisturizer to non-affected areas immediately after pat drying the skin. Use clean plastic spoons or tongue depressor to avoid contamination of products in jars. This allows large areas to be covered quickly and prevent caregivers from unnecessary exposure to topical medications.

Soak the dressings in warm water. Squeeze out excess water. Dressings should be wet, not dripping.

Cover an area with wet dressing chosen for the area and the patient. Immediately after wrapping, cover with appropriate dry material such as an Ace bandage, socks, or pajamas. Start at the feet and move upward. Use wet, long underwear or wet pajamas covered by dry pajamas or sweat suit with total body involvement in place of wet gauze.

Take steps to avoid chilling. Blanket can be put in a dryer to warm up and cover patient, but do not overheat the patient. Wraps can be removed after 1-2 hours or can be re-wet. A warm blanket and snuggling help pass the time.

If patient is known or suspected to have an infection of the involved areas, place dressings accordingly. In cases of large wounds, wet dressings can also be used selectively to areas of infection. Usually, the patient will have had a soaking bath prior to this procedure or will soak the area in basin to be wrapped. Pat skin dry with a towel.

If patient is known or suspected to have an infection of the involved areas, place dressings appropriately and dispose according to infection control procedure.

REFERENCES


This may be modified and used for patient care citing National Jewish Health Atopic Dermatitis Program as source.

Table 3.
Wet-Wrap Therapy

<table>
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<tr>
<th>PURPOSE</th>
<th>SUPPLIES</th>
<th>PROCEDURE</th>
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If wraps are to be applied to a large portion of the body, work with two people if possible. It is necessary to work rapidly to prevent chilling.  
Explain the procedure to the patient and parent.  
Fill the basin with warm tap water.  
Usually, the patient will have had a soaking bath prior to this procedure or will soak the area in basin to be wrapped. Pat skin dry with a towel.  
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If patient is known or suspected to have an infection of the involved areas, place dressings appropriately and dispose according to infection control procedure.  
After all dressings are removed, moisturizers may be applied to the entire body. |
Systemic Therapy

The use of systemic corticosteroids, such as oral prednisone, is rarely indicated in the treatment of a chronic, relapsing disease such as atopic dermatitis. Some patients and physicians prefer the use of systemic corticosteroids to avoid the time-consuming skin care involving hydration and topical therapy. However, the dramatic clinical improvement that may occur with systemic corticosteroids is frequently associated with a severe rebound flare of AD following the discontinuation of systemic corticosteroids. Short courses of oral corticosteroids may be appropriate for an acute exacerbation of AD while other treatment measures are being instituted. If a short course of oral corticosteroids is given, it is important to taper the dosage and to begin intensified skin care, particularly with frequent bathing followed by application of topicals, to prevent rebound flaring of AD.

Cyclosporine is a potent immunosuppressive drug. Multiple studies demonstrated that children and adults with severe refractory AD can benefit from oral cyclosporine treatment (Bunikowski et al., 2001). Treatment with cyclosporine is associated with reduced skin disease and an improved quality of life. Discontinuation of treatment may result in relapse of skin disease, although some patients may have sustained remission. Elevated serum creatinine or more significant renal impairment and hypertension are specific side effects of concern with cyclosporine use.

Role of Trigger Factors

Skin hyperreactivity is and has been an important feature of atopic dermatitis. Many triggers including irritants, allergens, and psychosocial events can elicit the itch sensation in patients with AD and set off the itch-scratch cycle that results in a flare of eczema.

Irritants

Patients with atopic dermatitis are more susceptible to irritants than other individuals. Irritants can be almost anything and may include soaps or detergents, chemicals, smoke, abrasive clothing, and exposure to extremes of temperature and humidity. Alcohol and astringents found in toiletries can be drying. When soaps or cleansers are used, they should have minimal defatting activity and a neutral pH. New clothing may be laundered prior to wearing to decrease levels of formaldehyde and other added chemicals. Residual laundry detergent in clothing may be irritating. Using a liquid rather than powder detergent and adding a second rinse cycle will facilitate removal of the detergent.

Patients with AD often develop a nonspecific, irritant hand dermatitis. It is frequently aggravated by repeated wetting without use of moisturizer and by washing of the hands with harsh soaps, detergents, and disinfectants. Atopic individuals with occupations involving wet work are prone to develop an intractable hand dermatitis in the occupational setting. This is a common cause of occupational disability (Shmunes, 1986).
Recommendations regarding environmental living conditions should include temperature and humidity control to avoid problems related to heat, humidity, and perspiration. Changes in the environment, which might not affect normal individuals, can elicit the itch sensation in patients with AD. Every attempt should be made to allow patients to be as normally active as possible. Certain sports such as swimming may be better tolerated than other sports involving intense perspiration, physical contact, or heavy clothing and equipment, but chlorine should be rinsed off immediately after swimming and the skin moisturized. While ultraviolet light may be beneficial to some patients with AD, sunscreens should be used to avoid sunburn. However, because sunscreens can be irritants, care should be used to identify a nonirritating product.

Allergens

Foods and aeroallergens such as dust mites, animal danders, molds, and pollens have been demonstrated to exacerbate atopic dermatitis. Although patients with AD often have high serum IgE levels which measures total IgE, it is not useful for identifying allergens. Potential allergens can be identified by taking a careful history and carrying out selective skin prick tests or measuring specific serum IgE levels. Negative skin tests or serum tests for allergen-specific IgE have a high predictive value for ruling out suspected allergens.

The role that food allergies play in flares of AD remains an area of active research and discussion. Food allergens may play a role in a subset of patients with AD, particularly those under the age of 3 years (Sicherer & Sampson, 2006). Controlled food challenges were first reported at the authors’ center in the 1970s, recognizing that a positive skin test to a food allergen did not necessarily define clinical relevance (May, 1976). Removal of proven food allergens on the other hand from the patient’s diet can lead to significant clinical improvement. Patients typically will have clinically relevant food allergy to only a small number of foods irrespective of the number of positive skin or in vitro tests. In children who have undergone double-blind, placebo-controlled food challenges, milk, egg, peanut, soy, wheat, and fish account for approximately 90% of the food allergens found to exacerbate AD (Sicherer & Sampson, 2006). The dietician plays a key role in evaluating patients’ diets and educating patients and caregivers regarding appropriate diets (Boguniewicz, Moore, & Paranto, 2008). Avoidance of foods implicated in controlled challenges results in clinical improvement. Extensive elimination diets are rarely required. Organizations such as Food Allergy and Anaphylaxis Network (www.foodallergy.org) can provide valuable information on hidden sources of common food allergens, recognizing specific food proteins by various names on food labels and methods of preparing foods with safe substitution of allergenic ingredients.

In dust mite-allergic patients with AD, prolonged avoidance of dust mites results in improvement of their skin disease (Arlian & Platts-Mills, 2001). Avoidance measures include use of dust mite proof encasings on pillows, mattresses, and box springs; washing bedding in hot water weekly; removal of bedroom carpeting; and decreasing indoor humidity levels with air conditioning. Because there are many triggers contributing to the flares of AD, attention should be focused on identifying and controlling the flare factors that are important to the individual patient. Infants and young children are more likely to have food allergies, whereas older children and adults are more likely to be sensitive to environmental aeroallergens.

Unlike allergic rhinitis and extrinsic asthma, immunotherapy with aeroallergens has not proven efficacious in treating AD. There are anecdotal reports of both disease exacerbation and improvement. A recent study of specific immunotherapy over 12 months in adults with AD sensitized to dust mite allergen showed improvement in AD as well as reduction in topical steroid use (Werfel et al., 2006). However, well-controlled studies are still required to determine the role for immunotherapy with this disease.

Psychosocial Factors

Patients with atopic dermatitis often respond to stressful events with increased pruritus and scratching. Scratching can become habitual and occasionally is associated with secondary gain. Psychological evaluation or counseling should be considered for patients and families. It may be especially useful in adolescents and young adults who consider their skin disease disfiguring. Relaxation, behavioral modification, or biofeedback may be helpful in patients with chronic pruritus.

Role of Itch in Atopic Dermatitis

Atopic dermatitis is frequently referred to as “the itch that rashes.” It continues to be debated whether the itch or rash comes first. Itching or pruritus is the key symptom of AD. Control of itch is important because injury from scratching can induce skin cells to release pro-inflammatory cytokines leading to a vicious itch-scratch cycle that perpetuates the eczematous rash. The mechanisms of itch or pruritus in AD are not fully understood. Allergen-triggered histamine release from mast cells is only one cause of pruritus in AD, and because of this, antihistamines are only partially effective in controlling the itch of AD (Klein & Clark, 1999). T-cell derived cytokines such as IL-31 have recently been shown to play an important role in AD-associated pruritus (Sonkoly et al., 2006).

The treatment of pruritus in AD should be directed primarily at the underlying causes (Nicol, Huether, & Weber, 2006). Inhaled and ingested allergens should be eliminated if documented to cause itching and skin rash in controlled challenges. Reduction of dryness and skin inflammation with moisturization and use of topical anti-inflammatory drugs, such as topical corticosteroids and topical calcineurin inhibitors, will often
reduce pruritus. Because pruritus is usually worse at night, the sedating antihistamines or other sedating medications may offer an advantage through their sedating side effects when used at bedtime. The sedating antihistamines, hydroxyzine and diphenhydramine, remain useful treatment adjuncts. Doxepin hydrochloride has both tricyclic antidepressant and H₁ and H₂-histamine receptor-blocking effects. If nocturnal pruritus remains severe, short-term use of a sedative to allow adequate rest may be appropriate. Studies of non-sedating antihistamines show variable results in the effectiveness of controlling pruritus in atopic dermatitis, although they may be useful in the subset of patients with AD and concomitant urticaria or concurrent allergies. Topical antihistamines, as well as topical anesthetics, are not recommended as they are frequent cutaneous sensitizers in patients with AD.

Role of Infections

Bacterial, viral, and fungal infections are common problems for patients with atopic dermatitis. *Staphylococcus aureus* is found on more than 90% of AD skin lesions; even normal-appearing skin of patients with AD is often heavily colonized. In contrast, fewer than 5% of normal subjects have *S. aureus* on their skin. Researchers at National Jewish found that an important strategy by which *S. aureus* enhances skin inflammation in AD is by secreting toxins. These toxins act as super-antigens which cause marked activation of T cells and antigen-presenting cells in the skin leading to significant skin inflammation (Leung, 2003). Honey-colored crusts, folliculitis, or pustules can all be indicative of secondary bacterial skin infection, usually due to *S. aureus* that requires antibiotic therapy. Methicillin-resistant *S. aureus* is becoming an increasingly important pathogen in patients with AD. It has also been a difficult surveillance and treatment issue for health care institutions to address when treating patients with AD. Culture and sensitivities may be helpful in managing these patients. Deep-seated abscesses occur rarely in AD and should raise the possibility of an immunodeficiency such as hyper-IgE syndrome.

The importance of *S. aureus* in atopic dermatitis is supported by the observation that patients with severe AD, even those without overt infection, can show clinical response to combined treatment with anti-staphylococcal antibiotics and topical corticosteroids (Nilsson, Henning, & Magnusson, 1992). Cephalexin or penicillinase-resistant penicillins (dicloxacillin, oxacillin, or cloxacillin) are usually beneficial for patients who are not colonized with resistant *S. aureus* strains. Because erythromycin-resistant strains are common, erythromycin and newer macrolide antibiotics are usually of limited utility. Topical mupirocin is useful for treating localized impetiginized lesions; however, in patients with extensive skin infection, a course of systemic antibiotics is more practical. Additionally, patients given topical mupirocin require specific instruction, reinforcing the need to use this topical three times daily to avoid developing resistance to this medication. Methicillin-resistant *S. aureus* may require culture and sensitivity testing to assist in appropriate antibiotic selection.

Infection caused by the virus herpes simplex can result in a generalized eruption termed eczema herpeticum. Herpes simplex can provoke recurrent dermatitis and may be misdiagnosed as a bacterial infection. Vesicular lesions are umbilicated, tend to crop, and become crusty. The presence of punched-out erosions, vesicles, and/or infected skin lesions, especially those that fail to respond to oral antibiotics, should initiate a search for herpes simplex virus. This can be diagnosed by a Tzanck smear of cells scraped from the vesicle base, direct immunofluorescence assay, polymerase chain reaction identification of herpes genetic material, or by viral culture. Antiviral treatment for disseminated cutaneous herpes simplex infections is of critical importance in the patient with AD because severe, even life-threatening dissemination has been reported. Acyclovir or one of the newer antiviral medications can be given orally. Intravenous treatment may be necessary for severe eczema herpeticum.

Superficial fungal or dermatophyte infections are also more common in atopic individuals and may contribute to the exacerbation of disease activity. There has been renewed interest in the yeast *Malassezia sympodialis* in AD. IgE antibodies against *M. sympodialis* are commonly found in patients with AD and most frequently in patients with head and neck dermatitis. Positive allergen patch test reactions to this yeast have also been demonstrated. The potential importance of *M. sympodialis* as well as other dermatophyte infections is further supported by the improvement of AD in some patients following treatment with topical or systemic antifungal therapy (Boguniewicz, Schmid-Grendelmeier, & Leung, 2006).

It is worth remembering that a healthy skin barrier is the best defense against all pathogens. Basic skin care measures cannot be overemphasized, particularly bathing and use of moisturizers which aid in the repair and maintenance of the skin barrier. Additionally, teaching patients to avoid sharing hygiene and skin care products from washcloths and towels to topical is important to avoid spreading infections. Dramatic clearing of *S. aureus*-infected eczema can occur in 1 week using twice daily soaking baths including a wet washcloth to the face, lower-potency topical corticosteroids followed by wet-wrap therapy, and oral antibiotics (see Figures 1a, 1b, 1c, & 1d). This illustrates what can be accomplished when proper skin care is combined with appropriate anti-infective therapy in infection-prone patients with AD.

Summary

Successful strategies for managing atopic dermatitis require an accurate diagnosis, identification and elimination of exacerbating factors including irritants and allergens, adequate hydration of the skin, control of pruritus and infections, and appropriate
use of topical anti-inflammatory and other medications. Patient education, including the fundamentals of the disease and how to do proper daily skin care, increases the chances of successful therapy for patients and their caregivers. In addition, impact of illness on patient and family quality of life needs to be considered. Treatment should be individualized according to the severity of illness and factors that trigger their atopic dermatitis. Whether at an academic center of excellence or a private practice setting, staff interested in and willing to spend the additional and necessary time educating patients and families about atopic dermatitis management is the key to successful treatment strategies.

References


Modulation of the atopy patch test reaction by topical corticosteroids and tar. Journal of Allergy and Clinical Immunology, 106(4), 737-743.


**Answer Form**

1. If you could imagine that you have fully implemented what you learned from this activity into practice, what would be different?

________________________________________________________________________________________________
________________________________________________________________________________________________
________________________________________________________________________________________________

**Evaluation**

2. By completing this offering, I am able to meet the stated objectives.
   a. Heighten his/her awareness of the prevalence of atopic dermatitis, its impact on quality of life, and association with asthma and allergies.
   b. Examine a multi-faceted approach to management of patients with atopic dermatitis including non-pharmacologic and pharmacologic interventions.
   c. Summarize common interventions including hydration, moisturizers, and pharmaceutical agents.
   d. Discuss appropriate safety issues related to topical and systemic therapies.

   | Strongly disagree | Strongly agree |
   | 1 | 2 | 3 | 4 | 5 |
   | 1 | 2 | 3 | 4 | 5 |
   | 1 | 2 | 3 | 4 | 5 |
   | 1 | 2 | 3 | 4 | 5 |

3. The content was current and relevant.

   1 2 3 4 5

4. The content was presented clearly.

   1 2 3 4 5

5. The content was covered adequately.

   1 2 3 4 5

6. I am more confident of my abilities since completing this material.

   1 2 3 4 5

7. The material was (check one) ☐ new, ☐ review for me

   Comments ____________________________________________________________

8. Time required to complete reading assignment: ___________ minutes

9. I verify that I have completed this activity __________________________________________

   Signature ________________________

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This article was reviewed and formatted for contact hour credit by Marcia J. Hill, MSN, RN, Dermatology Nursing Editor; and Sally Russell, MN, CMSRN, Dermatology Nursing Education Director.