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ECZEMATOUS DERMATITIS

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The WSCC Care Pathways provide a standardized context for clinical decision making as well as a variety of possible interventions. These pathways are not intended to replace the clinical judgment of the individual practitioner. A practitioner may vary from these guidelines, if in his or her judgment, variance is warranted to meet the healthcare needs of the patient and the variance remains within generally accepted standards of practice.

Limitations

WSCC pathways are intended for use within our clinic system. They may be useful as a seed for regional guidelines or guidelines with wider application, but caution must be exercised. The following limitations would have to be addressed. 1) The literature searches employed would need to be more exhaustive; 2) inclusion criteria for published studies would need to be more stringent; 3) a wider pool of subject-matter experts must be tapped; 4) the participants of the consensus panel would need to be drawn from a broader cross-section of the profession and perhaps other healthcare providers as well. Although individual procedures and decision-making points within the Care Pathways have established validity or reliability, the pathways as a whole are untested.

ICD-9 Codes

692.9 Eczema (acute) (allergic) (chronic) (erythematous) (occupational) (squamous)
691.8 Atopic
692.9 Contact
692.9 Dermatitis
due to specific cause (see ICD code book)
684 Impetiginous
690.12 Infantile (acute) (chronic) (due to any substance) (seborrheic)
692.9 Nummular
690.18 Seborrheic
454.1 Stasis (lower extremity)
454.2 Ulcerated

THE SCOPE OF THIS CARE PATHWAY INCLUDES THE EVALUTION AND CONSERVATIVE MANAGEMENT OF A VARITEY OF FORMS OF EZCEMA/DERMATITIS AND RELATED SKIN CONDITIONS.

On using this document…

Eczematous dermatitis has been subdivided into four categories of lesions: irritant contact dermatitis, allergic contact dermatitis, atopic dermatitis, and conditions related to atopic dermatitis, which include xerosis, nummular eczema, ichthyosis vulgaris, keratosis pilaris, seborrheic dermatitis, perioral dermatitis, dyshidrosis, stasis dermatitis, and lechen simplex chronicus. Superimposed infections are also addressed. The management section is similarly divided (as indicated by the footers on each page). Atopic dermatitis has further been divided into four major treatment components.

Search Strategy

Recent review articles and newer trials of specific conservative interventions were identified though Medline searches. These articles provided reference lists whereby additional studies could be accessed as needed. Additional conservative therapies and their references were obtained from the Textbook of Natural Medicine.*

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* Pizzorno JE Jr., Murray MT. Textbook of Natural Medicine. 2nd ed. Vol. 1. London: Churchill Livingstone, 1999.

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NOTES

BACKGROUND

Pathophysiology

The terms “eczema” and “dermatitis” are often used synonymously. Eczema is the most common form of skin disease. It is estimated that 10% of all patients who visit a general practitioner go because of a skin condition. Of these, one third has some kind of eczema.

Eczema is defined as *an itchy dermatitis characterized by an eruption with serous exudate and inflammation*.

The condition may be acute, subacute or chronic. Clinically, there is often a scale or crust composed of dried serous exudate, inflammatory cells and keratin. The skin usually has a spongy consistency (spongiosis).

In chronic eczema, histological changes include hyperkeratosis of the epidermis and thickening of the collagen bundles in the dermis. In chronic cases, persistent rubbing and scratching of the skin leads to development of roughened skin. This process is known as lichenification or lichen simplex [1, 2].

Eczematous eruptions can be triggered by a variety of stimuli such as house dust, food, chemicals, infection, climate and stress [3]. Atopic eczema has a hereditary component and runs in families [4]. Emotional factors, fatigue and psychological stress exacerbate all forms of eczema. Stimulants such as coffee, tea, tobacco and alcohol may also play a role, especially in chronic cases.

Eczematous disease may also be localized and have some unique characteristics. Examples include seborrheic dermatitis, perioral dermatitis, vesicle formation (dyshidrosis), and stasis dermatitis *(see Conditions Related to Atopic Dermatitis, p. 17).*

Eczematous dermatitis falls into three main categories, and are summarized here:

1. Contact Dermatitis

Irritant contact dermatitis. This is the most common form of occupational dermatitis. It is not associated with allergy and is triggered by irritating chemicals. All individuals are susceptible.

Allergic contact dermatitis. This form of eczema is also triggered by chemicals but is mediated by Type IV delayed hypersensitivity. A variety of chemicals and plants are associated with this form of dermatitis.

2. Atopic Dermatitis

This is the most complex form of eczema. It is hereditary and takes different forms throughout life. Patients have genetically compromised skin characterized by abnormally high levels of Immuglobin E (IgE). An enormous number of foods and environmental allergens can trigger atopic dermatitis. Skin and serological testing are useful in establishing the range of hyper-sensitivity.

3. Other Localized Eczematous Dermatitis

A number of other forms of dermatitis are related to atopic dermatitis (i.e., atopic eczema). These include chronic dry skin, lichenification, ichthyosis, seborrhea, dyshydrosis, stasis dermatitis, nummular and perioral dermatitis, lichen simplex chronicus and keratosis pilaris.

Categories of Eczematous Dermatitis

1. CONTACT DERMATITIS

Irritant Contact Dermatitis

This is the most common form of eczematous dermatitis. It is characterized by a rapid skin reaction following contact with an irritating chemical. However, it is not commonly seen in clinical practice. This is because the patient usually realizes what is happening and removes or avoids the irritant. Virtually all chemicals have the potential to cause a reaction. It is not associated with allergy, no sensitization is required, and all persons are susceptible.

Contact dermatitis is often triggered by exposure to strong, irritating chemicals such as detergents, acids, alkalis and solvents. These substances remove lipids from the epidermis, thereby exposing the lower layers of the skin to irritants. Persons at risk are those employed in occupations requiring repeated wetting and drying of the skin or contact with irritating chemicals[5]. A layman's term used to describe this condition is "dishpan" hands. Individual sensitivity varies. In some persons the skin hardens and becomes less sensitive over time[6].

Skin damage is usually apparent within several hours of contact with a strong irritant. Dryness and chapping may be followed by reddened sensitive skin[7]. In later stages, painful cracks and fissures accompanied by itching are common[8]. This is the subacute stage. Continued irritation of the skin may result in acute inflammation. At this point the lesions begin to itch and crust. Itching becomes intense and infection is common[9]. *(Management of this condition is described on p. 22.)*

Allergic Contact Eczema

This form of eczema is not as common as irritant contact dermatitis (approximately 20% of contact dermatitis is allergic)[10,11]. Allergic contact dermatitis is the result of a delayed hypersensitivity reaction. It occurs when T-lymphocytes in the skin are sensitized by primary exposure to specific sensitizing chemicals known as allergens[12,13].

Contact allergens are usually simple chemicals that link with proteins in the skin to become antigenic. Innumerable chemicals may cause allergic contact dermatitis. Sometimes chemicals related to the original allergen also trigger inflammation[14,15].

Allergens are processed by macrophages (Langerhans cells) in the epidermis. During sensitization the Langerhans cells present the allergens to large numbers of T-lymphocytes. On re-exposure to the same allergen the sensitized T-lymphocytes recruit and activate inflammatory cells, resulting in an eczematous reaction. This is a classic Type IV (delayed) hypersensitivity

reaction. Allergic contact dermatitis usually appears 24-48 hours after contact. In some cases inflammation may not occur for up to a week after exposure. The delayed reaction helps to distinguish allergic from contact dermatitis. This form of dermatitis usually subsides within 3-4 weeks. However, if exposure to the allergic chemical continues, the epidermal barrier breaks down and chronic dermatitis occurs.

Typical lesions are angular or linear streaks with sharp margins and corners[16]. Secondary bacterial infection may further complicate the condition. In some cases allergic contact dermatitis may complicate atopic dermatitis, especially if the atopic patient develops allergic sensitivity to topical medicines being used to treat the condition[11-17]. *(Management of this condition is described on p. 23.)*

2. ATOPIC DERMATITIS

Heredity and Atopic Dermatitis

This form of dermatitis is hereditary[18]. Approximately 70% of all cases occur in persons who have a family history of asthma, hay fever, eczema and dry, itchy, scaling skin. When both parents have allergic disease, the probability that it will be passed to their offspring is approximately 80%. When only one parent is allergic, the chance of disease is approximately 60%. The course of the disease is complex and determining the causes of outbreaks is often difficult.

Infant atopic dermatitis is an inflammatory skin disorder in children under 2 years old[19-21]. This skin condition is chronically relapsing and pruritic. It is often accompanied by asthma[22-25]. Atopic dermatitis affects 5-10% of children and is slightly more common in boys. It usually continues into adulthood when it typically begins to improve[27].

The common belief that an emotional disorder is the underlying cause of atopic dermatitis is a misconception. Anxiety and tension do not cause dermatitis but may contribute to increased itching and scratching, aggravating the condition. Patients may also have self-image problems and depression, related to having a chronic skin disease. Proper management of atopic dermatitis should address these concerns[26-29].

A second misconception is that atopic dermatitis is entirely due to allergies[30-31]. There is now substantial evidence that atopic dermatitis is precipitated by environmental stress on genetically compromised skin, accompanied by immune dysregulation[32]. A characteristic and important symptom of atopic dermatitis (Type I hypersensitivity) is elevated levels of immunoglobulin E (IgE). Patients with atopic dermatitis have decreased cyclic adenosine monophosphate (cAMP) levels due to increased Camp-phosphodiesterase activity and a decreased level of prostaglandin precursors.

Mediators of inflammation, such as platelet activating factor (PAF) also play an important role in inflammation.[33] The lack of intracellular cAMP results in increased histamine release, activation of eosinophils, decreased bactericidal activity, and changes in intestinal sensitivity to antigen[34].

Patients with atopic dermatitis also have a deficiency resulting in low levels of circulating CD8+ suppressor T-cells. In persons with normal immunity, the role of

the suppressor cells is to regulate and limit production of IgE by B-lymphocytes.[35-36] In atopic persons low levels of suppressor T-cells result in high levels of IgE[37]. Attachment of IgE to mast cells causes them to become hypersensitive and release high levels of histamine when triggered by specific allergens. Numbers of eosinophils also increase. Increase in IgE is the main cause of acute inflammation in atopic dermatitis.[38] These immune defects normalize during clinical remission and become abnormal again during recurrences of dermatitis. Decrease in components of the alternate complement pathway results in a defect of serum bactericidal activity, increasing susceptibility to infection.

The role of IgE in immediate hypersensitivities is well known. However, there is also considerable evidence that IgG, and particularly the IgG4 subclass plays an important role in delayed reactions. IgG has a circulating half life of 21 days, compared to a circulating half life of 1-2 days for IgE. IgG attached to the surface of a mast cell can remain there for as long as 2-3 months. IgG4 is the only subclass that induces basophil degranulation. This action triggers the release of histamine and other potent vasoactive chemical mediators in response to exposure to specific antigens. It is a common mechanism of allergic reactions[39-40].

TRIGGERING FACTORS

Triggering factors for atopic dermatitis include house dust, dust mites, cockroach feces, dog and cat dander, mold, grass and foods. Removal of suspected allergens may be of benefit in the management of this disease. Other factors are also important in allergic disorders. For example, environmental pollutants such as sulfur dioxide, nitrogen oxides, diesel exhaust particulates and fly ash may increase mucosal permeability and enhance allergen entry and raise IgE levels. Low levels of cigarette smoke have also been shown to increase IgE levels.

Food Allergies

Approximately 70-80% of children and adults with atopic dermatitis have food allergies[41]. This includes reactions to eggs, milk, wheat, nuts, soy, fish and shellfish. In some cases a small percentage of patients may have IgE mediated allergies, including edema, urticaria (hives), itching, wheezing and even anaphylaxis[42]. These are immediate symptoms, usually within 15 minutes of ingestion of the food. There is now evidence that food additives such as tartrazien, benzoate and nitrite may aggravate atopic dermatitis through increased sulfidoleukotriene production[43].

True food allergy may be confused with food intolerance, especially in infancy. In food intolerance, an adverse reaction occurs when food molecules are absorbed and reach the atopic skin. Many young children outgrow food intolerance within 6 months to a year, 40% by 5 years. It has been suggested that the early introduction of solid foods, before the age of four months, contributes to food intolerance. In general, breastfeeding is beneficial in reducing allergies. However, allergic allergens in breast milk may trigger reactions in atopic babies[44-59].

Infection

Patients with atopic dermatitis are also more susceptible to infections of the skin by bacteria and viruses. This is from breaks in

the skin due to scratching and cracking. Infection is also associated with reduced immunity due to dysfunction of neutrophils and lymphocytes. Atopic individuals are particularly prone to virus infections. Herpetic eczema occurs when viruses spread through damaged skin. Molluscum contagiosum and warts are less common but tend to spread. *Staphylococcus aureus* infections characterized by pustules may also become widespread. Infections of this type typically last 1-2 weeks[60-62].

(Management of atopic dermatitis is described on p. 26.)

EVALUATION

Key Signs, Symptoms and Historical Clues

Observation comes first. The practitioner looks at the lesion and determines if the following are present:

- ✓ Itching
- ✓ Red inflammation
- ✓ Eruption, vesicles, blisters, serous exudate (common in acute eczema)
- ✓ Scales, fissures
- ✓ Thickening of the skin (common in chronic eczema)
- ✓ History of recent contact with fabrics, chemicals, etc. (contact dermatitis)

To make the diagnosis, the patient must demonstrate three or more major findings* AND *three or more minor findings (see Table 1).

DIFFERENTIAL DIAGNOSIS IN INFANTS

Other skin disorders may resemble atopic dermatitis in infancy. Differentiation is based on family history of atopic eczema or psoriasis as well as location and appearance of the lesions.

1. Seborrheic dermatitis. Most common locations are scalp, cheeks, nasolabial folds, axillae, trunk and diaper area. Greasy yellowish scales are on an erythematous background. In contrast to atopic dermatitis seborrheic dermatitis is usually not itchy. It may occur as early as one month and often affects the groin and armpits.

TABLE 1. CRITERIA FOR DIAGNOSIS OF ATOPIC DERMATITIS

MAJOR FINDINGS. Three or more major features must be present.

- Pruritus (itching)
- Typical morphology and distribution
- Facial, flexural and extensor involvement in infants
- Facial and flexural involvement in children
- Flexural lichenification in adults
- Personal or family history of atopy: asthma, allergic rhinitis, atopic dermatitis

MINOR FINDINGS. Three or more of the minor features must be present.

- Cataracts
- Cheilitis (inflammation of the corners of the mouth)
- Conjunctivitis, recurrent
- Eczema
- Elevated IgE
- Facial pallor or erythema
- Food intolerance
- Hand dermatitis
- Ichthyosis (“fish scale” skin)
- Immediate (type 1) skin test reactivity
- Infections (Staphylococcus, Streptococcus, Herpes simplex)
- Infraorbital fold (Dennie-Morgan lines)
- Itching when sweating
- Keratoconus (elongation and protrusion of the corneal surface)
- Keratosis pilaris (acneform lesions)
- Nipple dermatitis
- Orbital darkening
- Pityriasis alba (scaly hypopigmented patches)
- Skin creases of the palms accentuated
- White dermographism (propensity for rapid formation of wheals and hives)
- Wool intolerance
- Xerosis (dry scaly skin)

2. Psoriasis. Psoriatic lesions are characterized by fine silvery scales on erythematous plaques. Distribution is most common on scalp, elbows, genitalia and knees. Unlike atopic dermatitis, the lesions are well defined. In addition there may be yellow discoloration and lysis of the nails.

3. Allergic contact dermatitis. Although this condition is uncommon in infants, it can occur in response to topical neomycin, nickel and synthetics such as vinyl. It closely resembles atopic dermatitis and may be difficult to differentiate. There is usually a sharp line of demarcation between normal and irritated skin.

4. Irritant contact dermatitis. This form of dermatitis results from chronic exposure to an irritating substance. Diaper dermatitis results from prolonged exposure to urine, feces and talcum powder. Irritant dermatitis on the extremities may be due to rough bed sheets. This condition may resemble atopic dermatitis but is usually less itchy.

5. Ichthyosis vulgaris. Common ichthyosis usually appears after 3 months. It is characterized by scales ranging from fine to coarse. It is distributed on the extensor surface of the extremities and sometimes on the face. Ichthyosis differs from atopic dermatitis in lacking erythema, papules and itching. Many infants with atopic dermatitis also have ichthyosis. Both conditions feature palmar hyperlinearity and keratosis palmaris (roughened, thickened skin on palms).

6. Tinea corporis (ring worm). Tinea infections typically form red, well delineated annular or circular plaques. There is usually peripheral scaling and central clearing. It may sometimes form vesicles and eczema. The fungus may also secondarily infect eczema in atopic children. Observation of a 10% potassium hydroxide slide at 200X readily detects fungal cells (hyphae).

7. Scabies infestation. Infestation with the itch mite *Sarcoptes scabiei* produces itchy papules, vesicles and burrows. There are usually excoriations and crusting. In infants lesions occur mainly on the neck, palms, soles, finger webs and axillae. Mites and eggs are detected by scraping affected areas with a scalpel and collecting the scrapings in mineral oil. Microscopic examination detects mites and eggs.

8. Pityriasis rosea. This skin condition produces a scaly, itchy rash. A “herald patch” typically precedes it.

9. Primary immunodeficiency. The following immunodeficiency disorders may initially present as eczematous dermatitis. Confirmation requires appropriate laboratory testing.

- Wiskott-Aldrich syndrome
- Selective IgA deficiency
- Hyper IgE deficiency
- X-linked agammaglobulinemia
- X-linked immunodeficiency with hyper-IgM
- Ataxia-telangiectasia
- Schwachman syndrome
- Chronic granulomatous disease

10. Metabolic and endocrine disorders. The following metabolic and endocrine disorders may initially present as eczematous dermatitis.

Confirmation requires appropriate laboratory testing.

- Biotin deficiency
- Maple syrup urine disease
- Cystic fibrosis
- Netherton syndrome

11. Proliferative Disorders.

- Langerhans cell histiocytosis

EVALUATION STRATEGY

Much of the art of diagnosis and management of eczematous dermatitis depends on understanding the underlying mechanisms and causes. The practitioner should have the ability to distinguish acute, subacute and chronic eczema. Since many forms of eczema have a similar appearance, it is necessary to carefully question patients in order to determine the origin and classification of their condition.

Summary of Evaluation Steps

Step 1: Physical examination to confirm the diagnosis of eczematous dermatitis. ***(See p. 12.)***

- Determine location and appearance of lesions
- Do the lesions itch? Is inflammation or exudate present? If not, reconsider the diagnosis of eczema.
- Determine if the rash is a form of contact dermatitis.
- Eliminate similar conditions such as psoriasis, etc.

Step 2: Identify the stage of eczematous dermatitis (acute, subacute, or chronic). ***(See p. 13.)***

Step 3: Take the patient's history and identify the category and cause of eczema:

- **Category 1:** Contact dermatitis. Reaction is immediate or within a few hours. Lesions may be acute or chronic. *(See p. 13.)*
- **Category 2:** Allergic contact dermatitis. Reaction occurs after 12-24 hours or even several days. *(See p. 13)*
- **Category 3:** Atopic dermatitis. A chronic condition appearing first in infancy. A family history of atopic asthma/rhinitis or other allergies is common. *(See p. 14)*
- **Category 4:** Other localized eczematous dermatitis. These include chronic dry skin, lichenification (roughening), ichthyosis (fish scale disease), keratosis pilaris (acne-like lesions), seborrhea, dyshidrosis, stasis dermatitis, nummular and perioral dermatitis, and lichen simplex chronicus. *(See p. 17.)*

Step 4: Determine if there is a superimposed infection. ***(See p. 19.)***

STEP 1: Physical Examination To Confirm the Diagnosis of Eczematous Dermatitis

Divide the skin surface into sections. For example, the face may be divided into the forehead, area around the eyes, lips, ears, etc. Carefully examine the skin in the area of complaint (best done by a window in natural light). A magnifying glass (10-20X) is often helpful.

Examine the rest of body for other lesions (arms, legs and torso). Note any differences. *(See Appendix II: Distribution of Lesions.)*

If there are signs of infection, check lymph drainage. Check the clinic laboratory manual to determine the proper method for

collecting specimens from the lesions. *Staphylococcus*, *Streptococcus* and Herpes simplex infections may be confirmed by sending specimens for appropriate laboratory testing. Fungal infections are more likely to be seen on warm, moist parts of the body such as feet, groin and arm pits. Fungal infections may be confirmed by examining skin scrapings mounted in 10% KOH with a microscope. Wood's lamp is considered to be less reliable for diagnosis.

Examine the skin carefully and see if it has the common characteristics of an eczema (see key signs above). Signs of eczema include itching, indistinct borders, vesicles, and intracellular edema and weeping, resulting in a sponge-like consistency of the skin (spongiosis). Related eczematous eruptions may be differentiated as follows. (See Steps 2 and 3.)

STEP 2: Identify the Stage of Eczematous Dermatitis

Acute. This form of eczema is characterized by bright red inflammation and formation of vesicles (pompholyx) and blisters. Itching is intense. It is usually caused by a wide variety of contact allergens (poison oak, cosmetics, rubber, etc.), severe irritation (wet cement), or stasis dermatitis.

Subacute. Mild to intense red inflammation with scales and fissures. Moderate itching, stinging and burning. Causes include irritant contact dermatitis, allergic contact dermatitis, atopic dermatitis, stasis dermatitis, fingertip eczema, and fungal infection.

Chronic. Moderate to intense itching results in habitual scratching leading to thickening of the skin (lichenification), cracks, fissures and excoriations. This pattern is characteristic of atopic dermatitis.

STEP 3: Take the Patient's History and Identify the Category and Cause of Eczema

The Four Categories of Eczema

1) Irritant contact dermatitis
2) Allergic contact dermatitis
3) Atopic dermatitis
4) Conditions related to atopic dermatitis

CATEGORY 1. Irritant Contact Dermatitis

Irritant contact dermatitis is often triggered by exposure of skin to strong irritating chemicals such as detergents, acids, alkalis and solvents. *Hands and fingertips are most commonly involved*. Approximately 80% of all dermatitis is contact dermatitis. However, the reaction to a chemical is usually rapid, the association obvious, and many cases are self-diagnosed. Therefore, the physician should consider all forms of eczematous dermatitis in evaluating each patient[9]. The patients who consult a physician about their condition are more likely to have allergic contact or atopic dermatitis[63].

CATEGORY 2. Allergic Contact Dermatitis

The major allergen in allergic contact dermatitis can usually be confirmed by taking a good history[64]. This is a classic Type IV (delayed) hypersensitivity reaction.

Allergic contact dermatitis usually appears 24-48 hours after contact. In some cases inflammation may not occur for up to a week after exposure. The delayed reaction helps to distinguish allergic from contact dermatitis[65].

The intensity of inflammation depends on individual sensitivity and the concentration of the allergen. The reaction is often confined to the area of exposure. For example, scalp and ear inflammation may be caused by shampoo, hair dye, earrings and eyeglasses. Allergy to rubber in shoes may cause foot eczema, a metal wrist watch band can cause dermatitis[66].

Patch testing

Patch testing is the accepted method of confirming allergic contact dermatitis when the cause is not obvious. A standard battery of allergens (T.R.U.E. Test) is available. This consists of a variety of common allergens. These are applied to the skin on adhesive strips. The strips are left in place for 48 hours and read. A second reading is done 48 hours later. Interpretation of the entire test series requires experience.

In many cases a more practical approach is to make a patch utilizing the suspected allergen. For example, a simple and convenient method for analyzing foot dermatitis is to remove one-inch square piece of shoe lining. All layers of the patch are separated, wet with water and taped to the skin of the outer upper arm. Patches are read after 48 hours. Similar topical patches can be used to test many kinds of fabric, rubber gloves, topical medications, cosmetics and creams[67,68].

CATEGORY 3. Atopic Dermatitis

Characteristics of Atopic Dermatitis

There is usually a family history and the patient has had the condition for a long time. The appearance of atopic eczema is variable and changes with age and environment. It tends to be less common around the nose and mouth.

The skin rash can be red, edematous and oozing. Hyperpigmentation accompanied by lichenification (lichen simplex chronicus) is common. Atopic dermatitis is characterized by roughening and thickening of the skin due to constant scratching. There may be a combination of signs but the condition always starts with itching. Signs of IgE-mediated atopy include allergic shiners, Dennie Morgan lines (infra-orbital fold), hyperlinear palms and boggy, pale swelling around the nose. The presence of wheezing indicates that other organ systems besides the skin are involved.

Atopic inflammation begins abruptly with erythema and severe pruritus. During recovery the skin becomes dry, and scaly, a condition known as xerosis. There is no single primary lesion in atopic dermatitis; rather several types of lesions depending on external stimuli, infection, scratching, etc.

Stages of Atopic Dermatitis

Infancy. In infants atopic eczema is typically red and oozing and appears on the scalp, face, torso and extremities. Both flexural and extensor surfaces of the arms and legs may be affected[69]. The usual onset is between 2-6 months of age. Itching, nocturnal restlessness, irritability and crying are common. There is now evidence linking T-cell deficiency and atopy in bottle fed

babies. Incidence of eczema in children is reduced if they are breast fed. There is additional evidence that bottle-feeding with cow's milk in infancy reduces the number of regulatory T-cells, and increases levels of IgE[72].

Childhood. In childhood atopic eczema is typically chronic. At about 18 months eczematous patches are replaced by lichenification as a result of scratching. Elbows, knees, dorsal hands, axilla, wrists and feet are commonly involved. Continuous scratching may destroy melanocytes giving rise to patches of hypopigmentation. Intensive hair loss may also occur. Inflamed fissures, especially in intergluteal areas, are known as intertrigo. The eczema typically goes through periodic exacerbations and remissions. These changes may be seasonal and are individualistic. In severe cases, growth retardation and significant emotional upset may occur[70].

Adults. The adult pattern of atopic eczema usually begins around the time of puberty. The skin becomes more stable and outbreaks are less common. Acute outbreaks are uncharacteristic[71]. Adult atopic eczema is most common on the face and hands but may also occur on the flexural surfaces. The skin around the eyes and on the arms is often involved due to the thinness and sensitivity of those areas. Recurrent eruptions of minute, itchy vesicles on fingers, palms, and soles is known as dyshidrosis (pompholyx). The vesicles may burst to form dyshidrotic eczema. Hand dermatitis is the most common symptom of adult atopic dermatitis and is most severe in females[72]. It may be difficult to distinguish this form of dermatitis from allergic contact dermatitis. The key to differential diagnosis is the patient's long history of atopic dermatitis.

Prognosis of Atopic Dermatitis

If present, the following factors indicate an *unfavorable* prognosis:

- Persistent dry or itchy skin in adults
- Widespread dermatitis and eczema in children
- Allergic rhinitis
- Family history of atopic dermatitis
- Bronchial asthma
- Early age of onset
- Female sex

Diagnosis of Food Allergies in Atopic Dermatitis

Summary of tests

- Skin prick test
- Provocation food tests
- RAST tests
- Serological tests

Diagnosis of food allergies is difficult. It is based on a careful clinical history. The prevalence of clinically relevant food allergy in patients with atopic dermatitis is not known. Estimates range as high as 80%[73-74]. Skin tests should be used to support, or discount, a diagnosis of allergy. The skin prick test is the method of choice for diagnosing immediate-type (IgE mediated) hypersensitivity.

Skin Prick Test

The skin prick test is an excellent preliminary test to exclude food antigens. Environmental allergens may also be tested using this procedure. Elevated IgE levels due to hypersensitive basophiles are commonly associated with house dust mites, molds and pollens. A negative skin

prick test excludes immediate hypersensitivity for that allergen. The test is useful for limiting the number of foods in a challenge test. Skin test solutions must be standardized[75-78].

Provocation food tests

Double-blind placebo-controlled food challenge is considered the gold standard for establishing the role of food hypersensitivity in atopic dermatitis[79-82]. Open or single-blind testing is less accurate but more practical. It should be kept in mind that food allergies may be only one of a multitude of causes of atopic dermatitis and may affect only a subset of patients[83-87].

Suspected foods are identified by assessment of patient's history. Six major food groups account for 90% of allergic reactions. ***These are eggs, milk, peanuts, soy, fish and wheat. Minor groups include: citrus, tomatoes, strawberries, corn, chocolate, food preservatives and colorings***.

Food intolerance is often present in infancy. Therefore, the age at which atopic dermatitis was first diagnosed should be noted. Reactions to certain allergens such as peanuts are lifelong, whereas reactivity to other antigens such as milk may be "outgrown" or lost with age[97]. Dermatologists now recommend that an evaluation for food allergy be done in any pediatric patient with moderate to severe atopic dermatitis that does not respond to topical steroids, antibiotics, emollients and soaking baths. Evaluation of the patient with a possible food reaction involves a detailed history, including:

- the time between ingestion of the suspected food and the development of symptoms,
- the quantity of the suspected food ingested,
- whether similar symptoms developed on other occasions when the food was ingested, and
- how long since the last reaction to the food occurred?[43].

Suspected foods are eliminated from the diet for two weeks. This is followed by controlled reintroduction of foods, one at a time, over a period of 3 days.

Symptoms usually occur within 2 hours of ingesting the food antigen. They consist of pruritis, erythema and edema. A recurrence of pruritis may occur 6 to 8 hours later. Many patients complain of abdominal pain, nausea, vomiting or diarrhea.

Although uncommon, an important limitation of this testing is the possibility of anaphylaxis when the suspected allergen is reintroduced into the patient's diet. Patients with a previous history of anaphylaxis should only be tested under appropriate hospital supervision. Anaphylaxis is most likely to occur in recalcitrant cases of atopic dermatitis. For this reason a qualified dermatologist or allergist should always conduct a challenge of patients with severe atopic dermatitis[88-91].

NOTE: Food challenge in conservative chiropractic evaluation and practice should be limited to mild to moderate atopic dermatitis patients, with no previous history of anaphylaxis.

Radioallergosorbent (RAST) Test

The RAST test is considered less reliable than skin prick and provocation tests. High

IgE levels may interfere with RAST tests. RAST results correlate with positive double-blind food challenges only one-third of the time. However, negative tests accurately predict negative reaction to specific foods. RASTs are useful in identifying foods suspected as allergens but should not be considered definitive evidence of food hypersensitivity [92-94].

Laboratory Testing

A number of clinical laboratories now provide ELISA/EIA (Enzyme Immuno-assay) panels to test for the presence of IgG1 and IgG4 antibodies in order to determine a patient's response to numerous food antigens. High levels of circulating IgG are correlated with clinical food allergy signs and symptoms[95].

IgG1 tends to occur in high titers early in life but is transient. IgG4 occurs later in infants in response to common foods like milk and egg. In some studies increased IgA has also been observed in response to cow's milk. Increased levels of these antibodies have been reported in children and adults with atopic eczema. However, the clinical significance of these antibodies is controversial; furthermore, IgG4 levels are not well correlated with clinical improvement [96,97].

The testing laboratory usually provides therapeutic diets, based on test results, to the physician and in some cases to the patient. *Unfortunately, these tests are questionable in both theory and validity*. They tend to be costly as well as unreliable. Physicians who use these tests should send split samples to the testing lab (at the cost of the lab) in order to assess reliability[98].

CATEGORY 4. Conditions Related to Atopic Dermatitis

Chronic Dry Skin

Xerosis. (chronically dry, sensitive and damaged skin) Xerosis is characteristic of atopic dermatitis and may occur at any age. It is most common on the arms and legs. It is estimated that 50-98% of patients become worse in periods of low humidity (winter). Ichthyosis is seen in about 40% of atopic patients[99]. Keratosis pilaris is also common (see next page).

Nummular Eczema. Coin-shaped eczematous patches characterize nummular eczema. It is most common in older men and young women[100]. Lesions may be chronic or acute. They are most common on the lower legs and backs of the hands. Itching is usually present and excoriations are common. The condition is worse in winter when humidity is low. The cause of nummular eczema is unknown. It may be difficult to distinguish from other forms of eczema.

Ichthyosis vulgaris

Ichthyosis ("fish skin" disease) is an inherited disorder of keratinization. Symptoms include dryness, hyperkeratosis, scaling and exfoliation. Ichthyosis appears in infancy and tends to improve with age. This condition is often associated with atopic dermatitis[101].

Other forms of Ichthyosis. There are many variations. These include sex-linked ichthyosis, a form found in males only and characterized by large, brown polygonal scales. Lamellar ichthyosis appears in

infancy. The skin is shining, dry and thin with coarse, yellow scales, and is also known as "alligator" skin. Management is similar to that used for other forms of atopic dermatitis.

Keratosis Pilaris

This condition is characterized by the appearance of rough papules or pustules. It is more common and extensive in persons with atopic dermatitis. It peaks during adolescence so it is often confused with acne. Abrasive washing and scratching worsen the condition. Management is similar to that used for other forms of atopic dermatitis[102,103].

Seborrheic Dermatitis (Seborrhea or "dandruff")

Seborrheic dermatitis is a chronic, scaling inflammation, primarily effecting the hairy parts of the body, especially the scalp, eyebrows, eyelashes, moustache and beard areas. The recesses of the ear canal and the fold behind the ear may also be involved. Mild inflammation and loose, flaky scales are characteristic. There are dry and oily forms. Scaling of the scalp results in dandruff.

The disease closely resembles atopic dermatitis; however, it is usually not itchy and IgE levels are usually normal. *Tinea capitis* and psoriasis should also be considered in differential diagnosis. The occurrence of seborrheic dermatitis parallels increased sebaceous gland activity in infancy and puberty. Infants with seborrheic dermatitis may develop greasy scaling on the scalp known as "cradle cap". It consists of greasy scale and crusts, which typically disappear by the end of the first year. Seborrheic dermatitis is most common and severe in fair-skinned persons. It is also common, and may be severe, in AIDS patients. There is evidence that this condition is an inflammatory reaction to the yeast *Pityrosporum obiculare*. The same organism is responsible for the characteristic white spots of *Tinea versicolor*[104].

Perioral Dermatitis

This condition is most common in young women. The area around the mouth is most often involved but the eruption may also occur on the cheeks and forehead. The cause is unknown except in those cases where it is a result of chronic use of topical steroids. Fluoridated toothpaste and mouthwash have also been linked to this condition[105,106].

Dyshidrosis (Pompholyx)

Dyshidrosis is a vesicular eruption seen on the palms and soles of the feet. It is typically deep, itching and burning. After a time the vesicles may coalesce to form bullae. In this stage erythema and scaling are present. This condition has been associated with atopic eczema, stress, excess perspiration, fungal infection, and metal sensitivity[107].

Stasis dermatitis

Stasis dermatitis is a sign of venous disease and is seen only in older adults. Capillary damage in the extremities results in an eczematous eruption on the legs. Lesions occur in lichenified plaques with brown pigmentation on lower legs. There is typically a history of chronic itching and swelling of the lower legs. Varicose veins and pitting edema are common. In severe

cases thrombophlebitis and ulceration may occur. It may be subacute, acute or chronic. Remissions and reoccurrence are common. Patients with stasis dermatitis are prone to allergic contact dermatitis and must use care in applying topical lotions containing sensitizing chemicals[108].

Lichen Simplex Chronicus (Lichenification)

This condition is also known as localized neurodermatitis. Lichenification is a common reaction to repeated rubbing and scratching. It is characterized by thickening of all layers of the skin, including the dermis. It occurs in all chronic, itchy skin diseases and is especially common in atopic dermatitis.

Lesions are usually solitary, but may be multiple. Normal skin lines are accentuated. Lesions are usually without a sharply defined margin and may be excoriated. The condition is most commonly seen on the nape of the neck, ankles and lower legs, wrists, forearms, scalp and ears[1].

STEP 4: Determine If There Is a Superimposed Infection

Cutaneous Infections

Patients with atopic dermatitis have a tendency to develop viral, bacterial and fungal skin infections. These infections are associated with disruption of the barrier of the epidermis and probably reduced localized immunity [60].

Bacterial Infections

Staphylococcus aureus is one of the most frequent causes of secondary infection in atopic eczema. Although *S. aureus* occurs on the skin or anterior nares in up to 30% of normal individuals, it is almost always found on patients with atopic dermatitis. It is found on normal skin as well as eczematous lesions. The early lesions are pustules, which burst to form weeping eczema and crusts. Acute infections are known as impetigo. Streptococcal infections are also common and may be concurrent with staphylococci. Cultures should be done in order to determine appropriate therapy[61].

Recent studies have shown that the organisms have a pathogenic role in the disease. Circulating anti-staphylococcal IgE antibodies have been found in up to 30% of patients with atopic dermatitis. These could cause mast cell degranulation, thus increasing inflammation. Super-antigens from *S. aureus* have also been shown to cause inflammation by stimulating T-lymphocytes and macrophages[61,62]. There is a direct link between the degree of *S. aureus* colonization and the activity of the eczema. Eczema usually improves when bacterial colonization is reduced with topical antibiotics.

Viral Infections

The most common viral infection associated with atopic dermatitis is Herpes simplex. When herpes and eczema occur simultaneously, it is known as eczema herpeticum. Vesicles containing clear fluid characterize the initial lesions of Herpes simplex. The vesicles become cloudy as they age and eventually burst to form punched out ulcers, forming crusts as they heal. These lesions are typically painful and may be associated with low-grade fever

and lymphadenopathy. Although herpes most frequently occurs on the head, the lesions can be much more wide-spread in atopic patients and may appear in atypical sites. A few have widespread eruption. Lesions should be swabbed and submitted for appropriate viral diagnosis[62].

Other viruses. Molluscum contagiosum and warts are less common in atopic dermatitis, but tend to spread and are more difficult to treat than in normal individuals.

Fungal Infections

Superficial fungal infections are also more frequent in atopic patients, characterized by erythema, scales, vesicles and fissures. Lesions may occur on any part of the body, and are more common in children over the age of 10 [62].

Candidiasis is an infection of the skin with the pathogenic yeast *Candida albicans*. In Candida infections patients usually complain of an itching and burning sensation. The rash is characterized by a scalded appearance with satellite lesions.

MANAGEMENT

In all forms of eczema, but particularly atopic dermatitis, practitioners should counsel their patients about the following issues:

- The role of emotional factors, fatigue and psychological stress in exacerbating their symptoms.
- In atopic eczema, the patient and family often want a “quick cure.” It is necessary for the physician to explain the chronicity of the disease in detail, stressing that it is not emotional in origin.
- When possible, treatment plans should help patients to form strategies to cope with stress factors.
- In addition, patients should be encouraged to reduce or eliminate stimulants such as coffee, tea, tobacco and alcohol, especially in chronic atopic eczema.

More specific therapy will depend partially on the type of eczema, as follows.

Summary of Management Sections

1. Irritant contact eczema *(See p. 22.)*

2. Allergic contact dermatitis *(See p. 23.)*

3. Atopic dermatitis *(See p. 26.)*

4. Conditions related to atopic dermatitis *(See p. 34.)*: Seborrhea, chronic dry skin, lichenification, ichthyosis, keratosis pilaris, dyshidrosis, nummular and perioral dermatitis, stasis dermatitis

1. MANAGEMENT OF IRRITANT CONTACT DERMATITIS

Management involves determining and avoiding the cause of irritation. A trial of avoidance can be used to identify or confirm the offending agent. Avoid anything that causes burning or itching of skin during the convalescence period.

Hand care
The hands should be washed as infrequently as possible with mild soap. After washing, hands should be carefully dried. Nails should be trimmed to reduce scratching[112].

Use simple lubricants such as mineral oil or petrolatum. Lotions such as Aquanil® and Cetaphil® and calendula oil also prevent excess drying and often relieve contact eczema[113].

Irritants
Avoid known irritants. These may include chemicals, vegetables, fruit and wool. Cotton, plastic or rubber gloves should be worn when working with household cleansers or detergents. To avoid irritation due to excess sweating, white cotton gloves should be worn beneath latex gloves.

Cool soaks
If inflammation and itching are significant, cool soaks may be used for a few days.

Treatment failure
If the eczema persists longer than a week, despite treatment, patch testing will be necessary to rule out other allergens, photoallergy, and contact dermatitis[109,110].

2. MANAGEMENT OF ALLERGIC CONTACT DERMATITIS

Management involves avoidance of the allergen and symptomatic therapy to promote healing.

Once the cause of the patient's allergic contact dermatitis has been confirmed, the patient must be persuaded to avoid future contact with the allergen. It is often possible to simply switch to products that do not cause the reactions[111]. However, in some cases avoidance may involve lifestyle or occupation changes. Topical corticosteroids are the mainstay of therapy. The most potent steroids are needed initially. This treatment must be prescribed by a dermatologist.

Conservative therapy includes the following: symptomatic treatment and elimination of allergens.

Symptomatic Treatment

Lubrication. The patient should not use cosmetic lubricants as they often contain allergens such as lanolin. The best lubricant is hydrated petrolatum.

Vinyl gloves are worn over the lubricant for one or two hours. This softens the skin and enhances penetration. Following this treatment the hands are rinsed and a small amount of lubricant is reapplied to prevent drying.

Coal tar cream or gel mixed with corticosteroid is used in refractory cases. Vinyl gloves should not be used in the application of tar based lubricants. Coal tar may also be used in conjunction with artificial ultraviolet B light (sunburn wavelength). This treatment is usually done 2- or 3-times weekly in the dermatologist's office. It may be done at home if the patient constructs or purchases a box. After 4 to 6 weeks of intensive treatment the treatments can be reduced to a few times a month.

Elimination of Allergens

Common Causes of Allergic Contact Dermatitis

- chromate
- formaldehyde
- ethylenediamine
- rubber
- dyes
- plants
- household or occupational chemicals
- nickel

Chromate is a common cause of dermatitis of the hands and arms. It is a common ingredient in products such as cement, detergents, bleaches, match heads, and is used in tanning leather. One of the most common problems is burning dermatitis in construction workers who come in contact with wet cement[112-115].

Formaldehyde is found in cosmetics, cigarettes, newsprint, fabric softener and coating for wrinkle-free fabrics. Formaldehyde is also found in some preservatives.

Ethylenediamine is one of the most common skin sensitizers. It is added as a preservative in some topical medications such as Mycolog cream. It is also used as a rubber accelerator and acts as a catalyst in rapid drying paints. It is a common ingredient in synthetic waxes and resins

and may induce cross sensitivity to histamines.

Rubber. Mercaptobenothiazole (MBT) and thiurams are used as accelerators in the manufacture of natural and synthetic rubber. Latex, a component of rubber boots, gloves and catheters is a common cause of hand and foot dermatitis[116,117]. This form of dermatitis may be mistaken for fungal infection. Rubber accelerators, such as thiurams, are sometimes associated with latex glove allergies. Thiurams are also used as fungicides in paint and soap. Sweat leeches out the chemicals. Absorbent powder and pads help to eliminate allergic shoe dermatitis by keeping the feet dry.

Dyes. P-Phenylenediamine (PPD) is the main component of most hair dyes and many fabric dyes. Sensitization to this chemical is a common cause of allergic contact dermatitis in men and women who dye their hair and in stylists who dye client's hair. Dyed stockings may cause leg dermatitis. Acrylic and epoxy compounds used as ink dryers have caused dermatitis in workers in the printing industry.

Plants. Plant toxins are the most common cause of allergic contact dermatitis. Approximately 50% of the population is sensitive. Plant sensitivity is an occupational hazard of florists and workers in the forest products industry. Poison oak, poison sumac and poison ivy contain powerful oleoresins, which may cause severe acute eczema.

Blocking agents may reduce severity of symptoms. Less frequently encountered toxic plants include cashews, mango, lacquer and gingko trees. Pineapple juice, carrot, parsnips, parsley, celery, spices, and lemon peel are also common allergens.

Sensitization to one plant in a family usually results in sensitivity to all. The antigen can be washed off with soap but becomes firmly fixed to skin proteins in about 15 minutes. A delayed hyper-sensitivity reaction, hours to days after exposure, is typical. New lesions may follow the primary lesions. A primary attack usually lasts 3 weeks. Subsequent exposures resolve more quickly[118].

Household or Occupational Chemicals. Acids, alkalis, detergents, medicines, cosmetics (especially fragrances), nail polish, insecticides and petroleum products are common agents of allergic contact dermatitis. Reaction may be immediate or take days or weeks, depending on the dose. Prolonged water or solvent exposure prior to contact increases the intensity of the reaction[119,120].

Nickel. In some cases a person with a severe allergy to nickel will develop generalized eczema if they eat food rich in nickel. These persons should avoid canned food and should prepare food in stainless steel utensils. Foods with high levels of nickel include herring, oysters, asparagus, beans, mushrooms, onions, corn, spinach, tomatoes, peas, whole-wheat flour, pears, rhubarb, tea, cocoa and baking powder[121-127].

Treatment of acute rhus dermatitis. Reactions to poison oak, poison ivy, and mango may be treated with cool water soaks, baths, or compresses 3 times a day for 10 to 20 minutes. Tap water or astringent dressing containing Domeboro® may be used. Aveeno® colloidal oatmeal baths reduce weeping and itching. Calamine lotion also reduces itching and is

considered to be safe and non-allergenic. These remedies are all over-the-counter (OTC). Most OTC poison ivy lotions contain antihistamines and local anesthetic and may cause an allergic reaction.

The conventional dermatological approach is to apply potent corticosteroids to the skin after bathing. Poison ivy oleoresin in capsules and injectable syringes for hyposensitization are considered ineffective and have been removed from the market by the FDA.

Systemic OTC antihistamines such as Benadryl® also help with itching. A 50-75 mg capsule may be taken at bedtime to aid sleep or a dose of 25 mg three times daily. The main disadvantage of antihistamines is that they cause drowsiness and they should never be taken before driving a motor vehicle. Baths and wet wraps are considered to be just as effective for managing nocturnal itching.

3. MANAGEMENT OF ATOPIC DERMATITIS including chronic dry skin

Management includes lifestyle changes designed to protect the skin from drying, infection and allergic flare-ups. Allergy elimination diets, nutrient supplementation, and botanical therapies have all proven useful in managing this condition. Controlling or eliminating allergens in the environment may be helpful in some cases.[128]

SUMMARY OF TREATMENT OPTIONS

1. Protect the Skin
- Moisturize skin *(See p. 27.)*
- Appropriate bathing strategies *(See p. 27.)*
- Control effects of temperature and humidity *(See p. 27.)*
- Control the environment *(See p. 28.)*

2. Limit the Triggers
- Reduce airborne allergens *(See p. 28.)*
- Reduce risks for contact dermatitis *(See p. 28.)*
- Minimize diet triggers *(See p. 28.)*

3. Limit Inflammatory Response
- Essential fatty acids *(See p. 29.)*
- Zinc and vitamins *(See p. 30.)*
- Lactobacilles *(See p. 30.)*
- Botanical therapy *(See p.30.)*
- Inhibition of histamine release *(See p. 31.)*
- Other herbal preparations *(See p. 31.)*
- Drug therapy *(See p. 32.)*
- Tar or ultra violet light application *(See p. 32.)*
- Tar therapy *(See p. 32)*

4. Treat Secondary Infections *(See p. 33.)*

Special consideration: pediatrics. In atopic babies, allergens in breast milk may trigger reactions. In these cases mothers should be instructed to avoid common food allergens such as milk, eggs and peanuts, and to a lesser extent, fish, soy, wheat, citrus and chocolate. In older infants peanuts are one of the most common food allergens. In most cases improvement occurs with elimination of milk products, eggs, milk, peanuts, tomatoes and artificial colors and preservatives[129-138]. In some cases avoiding allergy producing foods for one-year results in a loss of reaction to that allergen.

Elimination tests. A standard elimination diet avoiding cow's milk, eggs and tomato should help up to 75% of patients with moderate to severe atopic dermatitis. **However, if severe recalcitrant atopic dermatitis does not improve in response to the usual therapies, these cases should be referred to an appropriate specialist in allergy or dermatology.** This is especially important if a double-blind placebo-controlled food challenge is required in order to confirm suspected food allergies. The possibility of anaphylaxis upon re-introduction of food allergens must always be considered and anticipated. **A patient who has had an anaphylactic response to a specific food should never be challenged with that food[88-94].**

TREATMENT 1. Protect the Skin

Moisturize the Skin

Any factor that increases dryness of the skin exacerbates atopic dermatitis. Restoring moisture increases the rate of healing. Pure grease ointments such as petrolatum (Vaseline®) are useful for lubricating skin and reducing moisture loss. Ointments should be applied thinly. This is just as effective as a thick application and reduces the feeling of greasiness. Neutrogena® Dermatological Cream and Crisco® cooking oil are good for babies with very dry skin in a dry climate. These preparations have few additives and are unlikely to be irritating. Moisturizers should be applied as often as possible during the day, even every hour or two, especially to the infant's hands and face.

In humid weather use lighter creams such as Eucerin® and Aquaphor® (Beiersdorf), Lubriderm® (Warner Lambert Products), and Albolene® (Smith Kline Beecham). Creams are ointments with water added to make them easier to apply. They are not effective for extremely dry skin. Some ointments may have ingredients such as propylene glycol added to make them easier to apply. However, these additives are sometimes irritating to the skin. Lotions containing urea and lactic acid help to hydrate the skin. Lanolin products are often irritating to atopic skin and are not recommended.[128-132]

Appropriate Bathing Strategies

The atopic patient should avoid excess moisture. Washing and drying removes lipids from the epidermis leading to drying and cracking, and this may be controlled by reducing the frequency of bathing in hot water. Hot water dries the skin; therefore, baths should be tepid. The patient should use mild soap such as Cetaphil® (Galderma), unscented Dove® (Lever), Tone® (Dial Corporation) and Caress ® (Lever), or soap free cleansers such as Moisturel® (Squibb), or Aquanil® (Person and Covey). These products are gentle enough to use with babies. Soap substitutes such as emulsifying ointments are also available.

For adults, two or three daily hydrating tepid soaks for 20-30 minutes should be instituted with bath oil added to the tub water. For babies, one bath of 30 minutes a day in tepid water is recommended. The body and limbs should be washed using clean hands, not a wash cloth. The body is then gently patted dry (not rubbed dry) and ointment or aqueous cream applied.

Swimming is permissible, but too much chlorine is drying. The patient should use sunscreen and shower immediately after getting out of the water.

Control Effects of Temperature and Humidity

Atopic patients are intolerant to sudden temperature changes. Sweating invariably increases itching. Going from a warm to cold environment also intensifies itching. Cold air is less humid and dries the skin. These problems can be addressed at home by maintaining a constant cool temperature. Excess blankets and heavy coats that may cause sweating should be avoided. In winter months a commercial humidifier may help to reduce skin dryness.

Control the Environment

Environmental allergens should be taken into consideration. IgE-mediated allergies to house dust mites, molds and pollens play a role in pathogenesis of atopic dermatitis. Children's bedrooms should be kept as free of clutter as possible and all stuffed animals should be removed. Mattresses and pillows should be encased with plastic covers, and curtains should be washable. Pillows should be filled with synthetic material, not feathers. If possible, carpets should be replaced with washable scatter rugs. A foam cushion covered with a clean sheet should be used for playing on the floor.

Factors that contribute to the growth of mold in the home include a humidity level above 40% and the absence of a basement. It is recommended that bathrooms and kitchens be well ventilated to prevent excess humidity and the secondary growth of mold. The number of plants in the home should be limited. Children who are sensitive to mold should avoid playing near compost piles, fallen leaves or cut grass. Outdoor play should be restricted during pollen and grass booming season if there appears to be a relationship to skin sensitivity.

Tobacco smoke increases the risk of asthma and hay fever considerably[133]. Dander from cats and dogs and feathers from birds (and pillows) also increases the risk. Pets should be kept outside.

TREATMENT 2. Limit the Triggers

Reduce Airborne Allergens

Some persons have benefited from measures such as removing carpets and curtains from bedrooms, vacuuming blankets, and covering pillows and mattress with plastic. Also, air purifiers designed to remove allergens from the air are available commercially[134-5].

Rationale: Inhalation of dust mites, pollen, mold, cat and dog dander, and cockroach feces may exacerbate atopic dermatitis in some patients. The mechanism for this reaction is not clear but is thought to be related to high levels of IgG in atopic individuals.

Reduce Risks for Contact Dermatitis

Wool clothing should be avoided and perfumes and fragrances should be tested before use. It may also be helpful to reduce the amount of detergent and fabric softener when washing clothes and to double rinse clothing before drying.

Rationale: Persons with atopic dermatitis are usually less susceptible to allergic contact dermatitis due to reduced T-cell mediated delayed hypersensitivity. However, it is still wise to avoid substances that may irritate the skin.

Minimize Diet Triggers

Some foods can cause exacerbation of atopic dermatitis. The person with atopic dermatitis should maintain a bland diet. Fruits, tomatoes, nuts, eggs, shellfish, chocolate, milk, alcohol, bread, yeast, diet drinks and junk food have all been incriminated in triggering dermatitis in some individuals. Spices and caffeine should be avoided as well as foods rich in histamine

(fish, cheese, pickles, cured meat). Food allergies can usually be confirmed by the RAST test and/or food elimination diet. Foods causing skin reactions should be avoided. Elimination of allergenic foods appears to stop the development of new allergies in some individuals. Some patients who avoid offending foods for a period of up to one year may "lose" or "out grow" their allergy. A loss of food allergy was observed in 26% of patients with atopic dermatitis who avoided the five major allergens (eggs, milk, wheat, soy and peanuts) for one year[80].

Rationale: Foods can act as allergens exacerbating atopic dermatitis in some individuals.

TREATMENT 3. Limit Inflammatory Response

Consider Supplementing Essential Fatty Acids

Gamma-linolenic acid from a particular variety of evening primrose oil (Epogam) has been reported of value in the treatment of atopic eczema[139,143]. Controlled trials have shown that treatment with evening primrose oil and/or black currant seed oil (500 mg/day) helps to normalize this deficiency and reduce inflammation in some patients. In general, borage and flaxseed oils are less expensive than evening primrose oil; however, a recent placebo-controlled, double-blind trial showed no improvement after 10 to 14 weeks of treatment with borage seed oil. The recommended dosage for all of these oils is three 60 mg capsules daily until improvement is observed. Improvement should be seen between two weeks and one month. It is a subjective judgment based on significant clearing of lesions and reduction of other symptoms. Some patients have symptoms of indigestion from this quantity of oil. All patients receive 360 mg of gamma-linolenic acid daily, independent of sex and age[144-155].

Rationale: There is evidence that some patients with atopic dermatitis have an essential fatty acid deficiency. It is thought that patients with atopic dermatitis may have a defect in delta-6 desaturase, the zinc-dependent enzyme that converts linoleic acid to gamma-linolenic acid.

Fish Oils. Therapeutic doses of fish oil are 10 grams/ day for one month. The dosage is then reduced to one gram per day. Some patients have symptoms of indigestion from this quantity of oil and may improve simply by adding more fish to their diet. Pediatric doses are usually half the adult dose.

Rationale: Fish oils have also been shown to have significant anti-inflammatory and antiallergy effects by inhibiting arachidonic acid metabolism. The main source of arachidonic acid is animal fat. Therefore, reducing animal fat in the diet and increasing vegetable and fish oils is helpful in preventing inflammation. In general, treatment with omega-3 oils appears to produce good results, as they effectively deal with prostaglandin abnormalities. One of the best sources of omega-3 oils is fatty cold water fish (mackerel, salmon and herring). Patients who supplement their diet with fish oil or eat more fish show significantly more subjective improvement in atopic dermatitis than do control patients.

Consider Zinc and Vitamins

Zinc may be taken 50mg/day until the condition clears. Discontinue at the end of

one month if no significant improvement has occurred. Zinc oxide paste applied to lesions helps to reduce itching.

Rationale: Zinc is important in essential fatty acid metabolism. It has been shown to be necessary in the conversion of fatty acids to anti-inflammatory prostaglandin. Zinc levels are often low in atopic dermatitis.

Vitamins E and A are recommended in doses of 400 IU/day and 5,000 IU/day, respectively, while vitamin C is suggested at 50-75 mg/kg.

Rationale: In a random sample of 2,633 adults from the United Kingdom, it was found that higher concentrations of vitamin E in the diet were associated with lower serum IgE concentrations and a lower frequency of allergen sensitization. Vitamin E intake was closely related to vitamin C intake, but there was no correlation between vitamin C intake and IgE concentration[156].

Consider Use of Lactobacillus

In a randomized double-blind placebo-controlled trial, 159 mothers who had at least one first-degree relative or partner with atopic eczema, allergic rhinitis or asthma were supplemented with Lacto-bacillus. The mothers took these treatments for the last 2 to 4 weeks before delivery. Breastfeeding mothers continued this same supplemental regimen for 6 months. Non-breastfed infants took the same dosage orally. At 2 years of age, atopic eczema was diagnosed in 35% of the children. The frequency of atopic eczema in the probiotic supplemented group was half that of the placebo group. There was virtually no difference between the supplemented mothers who breastfed their infants and infants who took Lactobacillus orally. It is thought that supplemental probiotics may reduce intestinal permeability and enhance gut-specific IgA responses[157].

Consider Botanical Therapy

There is evidence that some botanical products reduce histamine production, particularly those containing flavonoids[158-162]. Dosage of all of the following is three times daily.

Witch Hazel extract cream. This is an approved treatment for eczema in Europe. It has been shown to be as effective as 1% hydrocortisone[98].

Glycyrrhiza glabra (licorice root), OTC. Over 50 g/day can raise blood pressure, causing sodium and water retention, and lower potassium. Overuse may result in pseudo-aldosteronism. Other reported adverse effects include amenorrhea, cardiac arrest, congestive heart failure, headache, hyperprolactinemia, hypertension, hypokalemia, muscle weakness, myoglobinemia, myopathy and paralysis.

Contraindications include cholestatic liver diseases, liver cirrhosis, hypertension, hypokalemia, severe renal insufficiency, and pregnancy. Prolonged use and higher doses may result in mineralocorticoid adverse effects and interactions. The root should not be used for more than 4-6 weeks without consulting a physician.[163-164]

Rationale: Potent flavonoids are found in blueberry leaf, Turkish attar of rose, rue, blackthorn and hawthorn berry, and licorice root. Inulin, a component of burdock and dandelion root has been shown to reduce inflammation and it is also antimicrobial. Its

antimicrobial action is due to activation of the alternative complement pathway. This pathway promotes chemotaxis of neutrophils and lysis of viruses and bacteria[98].

Botanical medicines may be taken internally (< 50 g/day) or applied topically. Licorice appears to be useful in either application. The most prominent topical treatments are German chamomile and licorice. Licorice is most effective when lotion containing pure glycyrrhetinic acid is applied directly on the lesions.

Rationale: This substance has an action similar to hydrocortisone but without adverse effects on the skin (see drug therapy on the next page).

Consider Natural Inhibition of Histamine Release

Forskolin. Standardized extract containing 18% forskolin is given two to three times daily. Licorice is another potent cAMP stimulant.

Rationale: Some botanical agents stimulate cAMP production. This helps to reduce histamine mediated inflammation. The active ingredient of Coleus forskolii is forskolin (mint family) OTC, no side effects. Extract standardized to contain 18% forskolin, 50 mg (9 mg of forskolin), a strong cAMP stimulant. The result is inhibition of basophil and mast cell degranulation and histamine release.

Flavonoids and Ginkgo biloba. Flavonoids are also natural anti-histamines. Flavonoids are extracted from a variety of plants including green tea and Ginkgo biloba. Ginkgo biloba extract is given in two doses of 160 mg twice a day for one month. The German Commission E Monograph concludes that Ginkgo biloba is safe and efficacious[163].

Rationale: In addition to flavonoids, ginkgo extract contains terpene molecules (ginkolides) that antagonize platelet-activating factor (PAF). PAF stimulates many features of inflam-mation, including increasing vascular permeability, bronchoconstriction, and reduction in coronary blood flow.

Oolong tea consumed 3 times daily for 6 months resulted in 63% marked or moderate improvement in eczema in 118 subjects. It is suggested that the tea polyphenols such as epiogallocatechin gallate suppress the hypersensitivity reaction.

***Arctium lappa* or *Taraxacum officinale* (dandelion), OTC.** These substances have no side effects. Dried root: 2-8 gby infusion or decoction. Fluid extract (1:1): 4-8 ml (1-2 tsp). Tincture: alcohol-based tinctures of dandelion are not recommended because of the extremely high dosage required. Juice of fresh root: 4-8 ml (1-2 tsp). Powdered solid extract (4:1): 250 to 500 mg[160].

Other Herbal Preparations

The following are some traditional herbal preparations used topically to relieve the redness and itching of eczema[142].

Chamomile, calendula and chickweed creams. Chamomile and calendula have anti-inflammatory properties, while chickweed is historically used to reduce itching. Research studies have not documented the efficacy of creams of any of these three herbs for people with eczema.

Burdock root is listed in traditional herbal books for the treatment of eczema.

However, there is little evidence to support its use for this condition.

Sasparilla may be beneficial as an anti-inflammatory based on historical accounts. Capsules or tablets should provide at least 9 grams of the dried root per day, usually taken in divided doses. Tincture is taken in doses of 3 ml, three times per day.

Red clover is considered beneficial for all manner of chronic conditions afflicting the skin in traditional herbal medicine. However, the mechanism is unknown.

Wild oats have been used to treat a variety of skin conditions, including eczema, but have not been investigated scientifically.

Tannins are therapeutic components of oak bark. They bind liquids, absorb toxins, and soothe inflamed tissues. For weeping eczema, oak is applied topically. It is prepared by boiling 1 to 2 tablespoons (15 to 30 grams) of bark for 15 minutes in 500 ml (2 cups) of water. After cooling, a compress with the bark extract is applied several times a day. This remedy has not been evaluated scientifically[165-166].

Consider Drug Therapy

Corticosteroids are one of the most commonly prescribed topical medications for treating inflammation and itching in eczema. They come in various strengths. Some of the mildest, such as 1% hydrocortisone, are now available OTC. More potent prescription corticosteroids should always be used conservatively as long term use may cause thinning of the epidermis and the inflammation may rebound when the drug is discontinued [167-170].

Glycyrrhetinic acid (from licorice root) may be used in combination with topical cortisone to reduce potential side effects.

Systemic antihistamines such as Benadryl®, chlorpheniramine, hydroxyzine and promethazine are available OTC and are often recommended to reduce itching. These have marginal effectiveness and cause drowsiness. Other side effects are blurred vision, tinnitus and abdominal pain. Excessive doses can cause hyperactivity in children.

Consider Tar and Psoralen and Ultraviolet Light Application (PUVA)

PUVA treatment is used when symptoms of eczema are chronic and other treatments have not worked. The procedure is done in the physician's office. Sunbathing is also effective during warm months. PUVA has resulted in prolonged remission in some children, adolescents and adults. One drawback of this procedure is increased risk of skin cancer[171-173].

Tar treatments are used to reduce itching and inflammation. Tar may be purchased OTC. Although effective, tar has a strong odor, induces folliculitis and discolors the skin. Patient compliance is poor.

TREATMENT 4:
Treat Secondary Infections

Yeast. Elevated titer of anti-candidal antibodies is common in atopic persons and may be correlated with the skin lesions. Anti-candidal therapies may result in significant improvement of atopic dermatitis in these individuals.

Rationale: An overgrowth of the yeast, *Candida albicans*, in the gastrointestinal tract has been implicated as a complication

of chronic skin conditions, including atopic dermatitis.

Echinacea purpurea has a long history of use in the treatment of inflammatory skin conditions and recurrent vulvovaginitis. Research has produced evidence that Echinacea inhibits hyaluronidase production by infectious micro-organisms, activates phagocytic activity of white blood cells, and stimulates cellular (T-cell) immunity. As an immunostimulant Echinacea is best taken three to four times daily (total 900 mg) and continued for at least 10 to 14 days. Expressed juice products are typically recommended at a dosage of 40 drops three times daily. Echinacea is essentially non-toxic when taken orally. Use is contraindicated in those with autoimmune illness and other progressive systemic diseases such as multiple sclerosis, HIV and tuberculosis.

Bacteria. Lesions of atopic dermatitis are frequently infected with bacteria such as *Staphylococcus aureus* and group A beta hemolytic streptococci. In acute exacerbation of eczema, the skin should be swabbed and cultured. Systemic antibiotics may be given immediately. Topical antibiotics of choice are the penicillinase resistant penicillins such a flucloxacillin or oral cephalosporins. Most staphylococci are sensitive to these drugs. Bacteriological findings are an essential part of management. Topical corticosteroids are contraindicated. Conservative therapy includes removal of crusts with saline or other wet soaks and application of Betadine ointment. For chronic infections, systemic antibiotics may be necessary. Antibiotics should not be given prophylactically as resistance may develop.

Herpes simplex. For herpes simplex the drug of choice is Acyclovir. This is administered orally in a dose of 200 mg orally five times daily for 5 days for adults and children over 2 years old. For children under 2 years, the dose is half the adult dose. Topical Acyclovir is considered ineffective for treatment of atopic dermatitis.

Warts and molluscum contagiosum. Treatment does not differ from non-atopic individuals. Therapy includes topical wart preparations such as Compound W, cryotherapy and cutterage. Molluscum contagiosum can be treated by mildly traumatizing the lesions and squeezing to expel the contents. Topical steroids should be avoided.

4. CONDITIONS RELATED TO ATOPIC DERMATITIS

Nummular Eczema
Natural sunlight may be beneficial if not overdone.

Seborrheic Dermatitis
Seborrheic dermatitis can usually be controlled. Scales are removed by diligently shampooing the affected areas.

Selsun Blue® shampoo is especially useful in treating Tinea versicolor because it eliminates yeast.

Peanut oil and mineral oil are also useful in lubricating the affected areas and reducing scaling.

Topical steroids should be avoided as they may cause skin atrophy and rosacea.

"Cradle cap" is a variation of seborrheic dermatitis on the scalp of infants. Dense scales may be removed by applying warm olive oil and washing several hours later with mild shampoo.

Lichenification
The first goal of therapy is to stop the scratching and rubbing.

Moist compresses and ice may help in reducing itching. Since patients scratch in their sleep, topical steroid use is almost inevitable. Tar pre-preparations also help to relieve itching. They may be applied at bedtime and removed in the morning. Keeping nails cut short is also helpful in reducing skin damage and lichenification.

Stasis Dermatitis
Moist eczema and ulcers can be treated with wet Burrow's solution compresses, or silver nitrate applied for 30 to 60 minutes several times a day.

Supportive bandages or stockings may be used.

Unna boot paste is effective in the treatment of ulcers (zinc oxide with phenol).

There is also evidence that high doses of vitamin D and zinc may promote healing.

Steroid creams interfere with the healing process. Lanolin, benzocaine and topical antibiotics should be avoided.

Patients should avoid standing for long periods. Resting in a prone position with the legs elevated may be helpful.

If patients are obese they should be encouraged to go on a weight reduction plan.

The following botanical remedies may be helpful in treating this condition:

- Horse Chestnut seed (*Aesculus hippocastanum*) is indicated for chronic venous insufficiency.

  Clinical trials have shown that oral administration, 1000 mg/day for four weeks, followed by 500 mg/day for an additional four weeks resulted in significant reduction in lower leg edema. No adverse side effects were observed.

- Bilberry (*Vaccinum myrtillus*) contains bioflavonoid complex (anthocyanoside). In doses of 480 mg/day this complex promotes formation of normal connective tissue, is venoactive, and improves blood platelet flow. At therapeutic dosages, there are no known side effects with bilberry extracts[174-176].

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APPENDIX I. TOPICAL MEDICATIONS FOR TREATMENT OF ECZEMA

Ointments

Lubricating agents

- Grease, such as Vaseline®

The thinnest possible application gives the maximum effect

- tiny amounts are dabbed onto the area to be treated and rubbed in
- may be too greasy for skin fold areas

Creams

Ointment with water added

- require preservatives and stabilizers
- not effective for extremely dry skin
- often used on the face

Lotions

Mostly water with some lipid

- used to treat moist skin folds and the scalp

Solutions

Alcohol and propylene glycol

- used for scalp and moist skin folds
- not to be used on dry skin

Astringent solutions

- Burrow's solution
- Domeboro®, applied to weeping lesions to dry them
- colloidal oatmeal (Aveeno®)
- may be followed by moisturizer

Compresses

- cool wet compresses soothe itchy skin

Gels

Gelled propylene glycol

- keratolytic (keratin softening), soften and remove scales
- may be used to apply salicylic acid or tars
- may dry the skin and cause irritation

Shake Lotions

Powder in solution

- Calamine lotion
- retards evaporation of water

Pastes

Ointment to which powder has been added

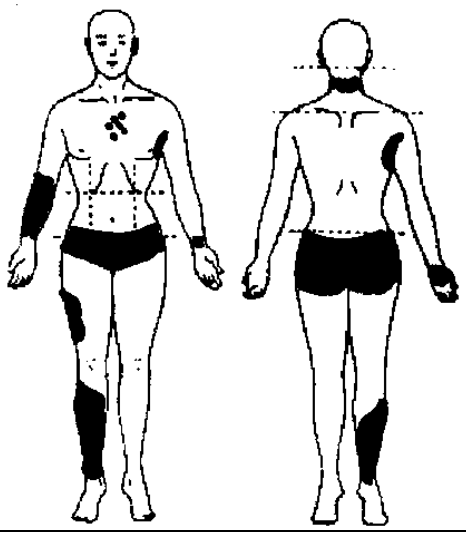
- forms protective layer
- zinc oxide paste

Powders

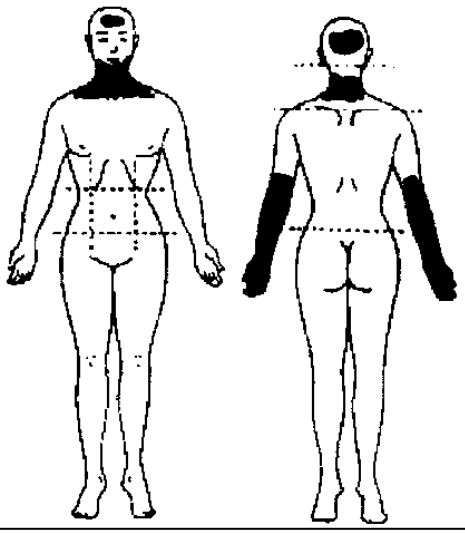
Used to dry moist skin

- may be used to distribute medication antifungal powder

APPENDIX II. DISTRIBUTION OF LESIONS



CONTACT DERMATITIS



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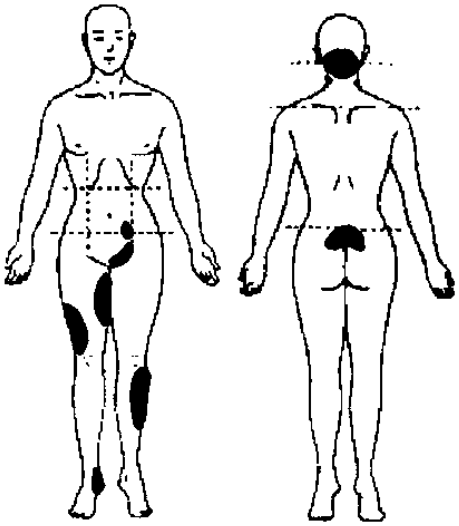
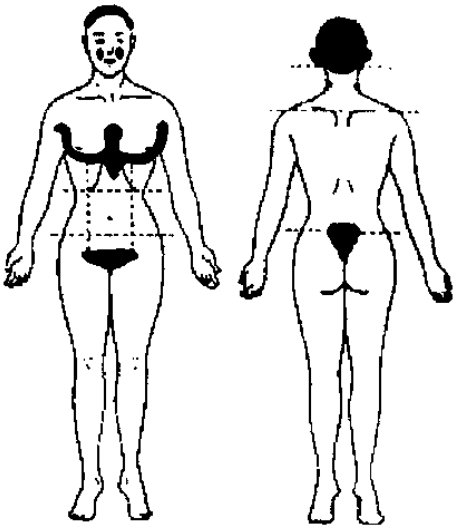


LICHEN SIMPLEX



SEBORRHEIC DERMATITIS