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Introduction

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Eczema remains the most common skin condition seen by family physicians and dermatologists. While being a disparate group of diseases, they have some common features including the presence of itch and in the acute stages, edema (spongiosis) in the epidermis. In early disease, the stratum corneum remains intact, so the eczema appears as a red smooth edematous plaque. With worsening disease, the edema becomes more severe, tense blisters appear. If less severe or if the eczema becomes chronic, scaling and epithelial disruption occurs, giving chronic eczema the characteristic appearance. All these are phases of the reaction pattern and are known as eczema. The word eczema comes from the Greek for 'boiling'—a reference to the tiny vesicles (bubbles) that are often seen in the early acute stages of the disorder, but less often in its later chronic stages. The histological findings of eczema are depicted in Fig. 1.1. *Dermatitis* means inflammation of the skin and is, therefore, strictly speaking, a broader term than eczema—which is just one of several possible types of skin inflammation.

Though various classifications exist, we will stick to the time-honoured, division into exogenous and endogenous types (Table 1.1).

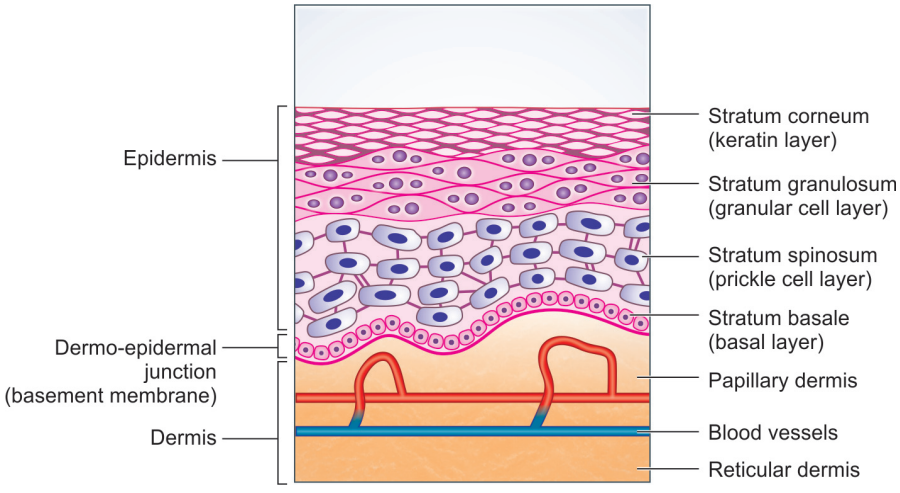

Fig. 1.1a: A depiction of the normal histology of the skin

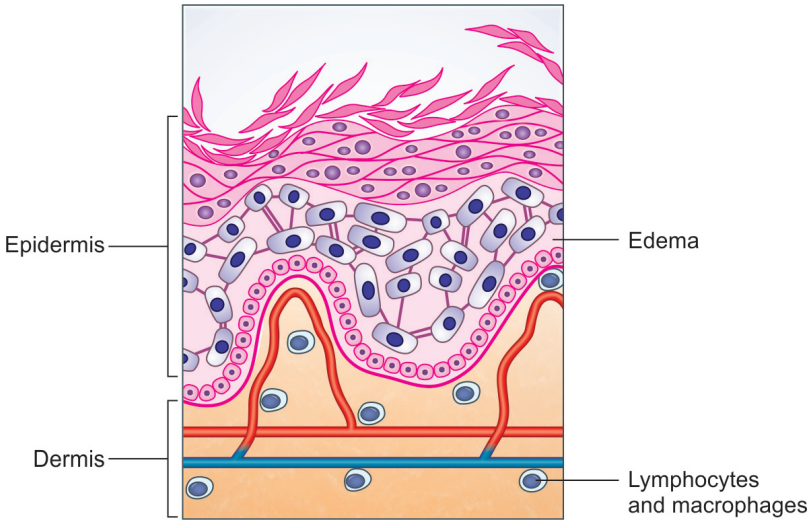

Fig. 1.1b: A depiction of the histology of eczematous skin; edema develops between the keratinocytes (spongiosis), the epidermis is thickened (acanthosis) and there are inflammatory cells in the dermis

Table 1.1: Classification of eczema

Exogenous eczemas
- ABCD
- Allergic contact eczema
- Dermatophytide
- Eczematous polymorphic light eruption
- Infective eczema
- Irritant eczema
- Photoallergic eczema
- Post-traumatic eczema

Endogenous eczema
- Asteatotic eczema
- Atopic eczema
- Chronic superficial scaly dermatitis
- Discoid eczema
- Eczematous drug eruptions
- Exudative discoid and lichenoid chronic dermatosis
- Eyelid eczema
- Hand eczema
- Juvenile plantar dermatosis
- Lichen simplex chronicus
- Pityriasis alba
- Prurigonodularis
- Seborrheic eczema
- Venous eczema

In this book, we will discuss the common types of eczema and the rare variants will be left to specialized dermatology textbooks.

STAGES OF ECZEMA

There are three stages of eczema: Acute, subacute, and chronic. An eczematous disease may start at any stage and evolve into another. We will give detail of the various types and their treatment, though there would be variations for specific types of eczematous disorders.

1. Acute Eczema

Etiology

Inflammation is caused by contact with specific allergens such as Rhus (poison ivy, oak, or sumac) and chemicals. In the id reaction, a reaction occurs at a distant site during or after a fungal infection, stasis dermatitis, or other acute inflammatory processes (Fig. 1.2).

Clinical Findings

The classic features are:

1. Weeping and crusting
2. Blistering—usually with vesicles

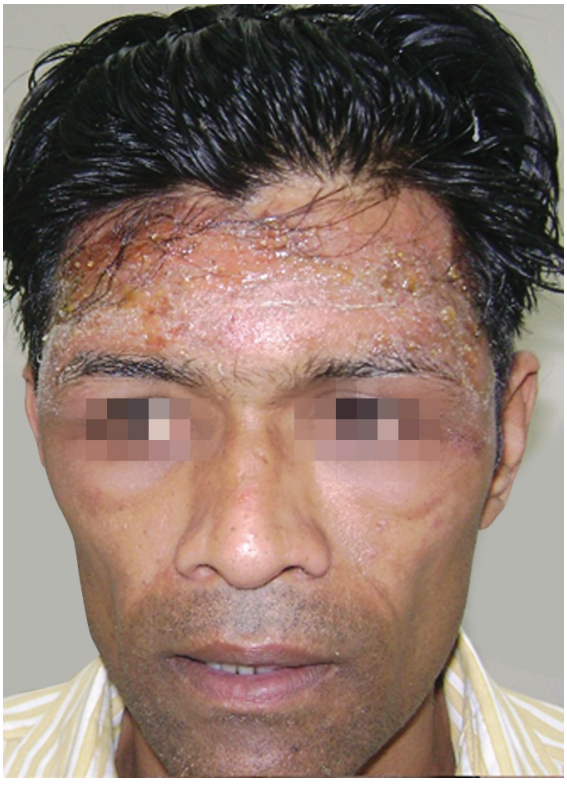

Fig. 1.2: A case of acute eczema in a patient consequent to allergic contact dermatitis to hair dye

3. Redness, papules and swelling—usually with an ill-defined border; and
4. Scaling.

There is frequently intense itching and heat and hot water can accentuate the symptoms. The condition can persist for a week or more and can evolve into a subacute stage before resolving.

Treatment

1. ***Cool, wet dressings.*** The evaporative cooling produced by wet compresses causes vasoconstriction and rapidly suppresses inflammation and itching. Either Burow's solution or normal saline can be used. A clean cotton cloth is soaked in cool water, folded several times, and placed directly over the affected areas. Evaporative cooling produces vasoconstriction and decreases serum production. Wet compresses should **not** be held in place and covered with towels or plastic wrap because this prevents evaporation. The wet cloth macerates vesicles and, when removed, mechanically debrides the area and prevents serum and crust from accumulating. Wet compresses should be *removed* after 30 minutes and replaced with a freshly soaked cloth.
2. ***Oral corticosteroids.*** Oral corticosteroids, such as prednisone, are useful for controlling intense or widespread inflammation and may be used in addition to wet dressings. A course of 20 mg dose twice or once a day for 7 to 14 days is enough in most cases though in some cases up to 21 days of therapy may be needed. Topical corticosteroids are of a little use in the acute stage because the cream does not penetrate through the vesicles.
3. ***Antihistamines.*** Antihistamines relieve itching and provide enough sedation so patients can sleep.
4. ***Antibiotics.*** The use of oral antibiotics may greatly hasten resolution of the disease, if signs of superficial secondary infection are present. Cephalexin and dicloxacillin are effective.

2. Subacute Eczema

There is erythema and scaling with an indistinct border (Fig. 1.3). The symptoms vary from no itching to intense itching.

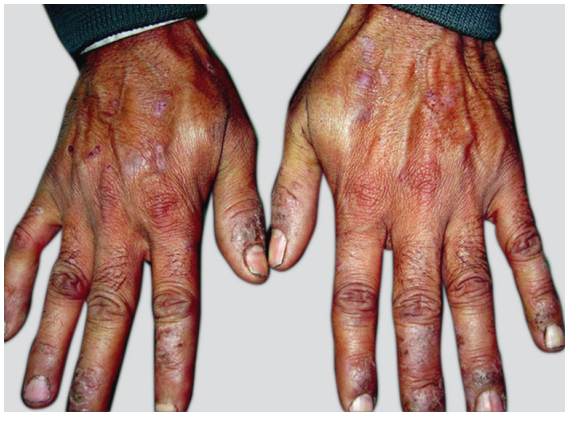

Fig. 1.3: A case of contact dermatitis to cement a prototype of subacute eczema

Subacute eczematous inflammation may be the initial stage or it may follow acute inflammation. If the inciting agent is withdrawn, the condition often resolves but excessive drying created from washing or continued use of wet dressings causes cracking and fissures (Fig. 1.3).

Treatment

It is important to discontinue wet dressings when acute inflammation evolves into subacute inflammation. Excess drying creates cracking and fissures, which predispose to infection.

1. ***Topical corticosteroids.*** These agents are the treatment of choice.
2. ***Topical macrolide immune suppressants.*** Tacrolimus ointment and pimecrolimus cream have been used for atopic dermatitis, allergic contact dermatitis, and irritant contact dermatitis and are approved for use in children 2 years or older. Response to these agents is slower than the response to topical steroids.
3. ***Lubrication.*** This is a simple but essential part of therapy. Inflamed skin becomes dry and is more susceptible to further irritation and inflammation. Resolved dry areas may easily relapse into subacute eczema, if proper lubrication is neglected. They can be applied a few hours *after* topical steroids and should be continued for days or weeks after the inflammation has cleared. Frequent application (one to four times a day) should be encouraged and using them after the skin has been patted dry following a shower seals in moisture.
4. ***Mild soaps.*** Frequent washing with a drying soap, can be avoided by using superfatted soaps.
5. ***Antibiotics.*** Eczematous plaques that remain bright red during treatment with topical steroids may be infected. Infected subacute eczema should be treated with appropriate systemic antibiotics, which are usually those active against staphylococci. Systemic antibiotics are more effective than topical antibiotics or antibiotic–steroid combination creams.

3. Chronic Eczema

Chronic eczematous inflammation may be caused by irritation of subacute inflammation, or it may appear as lichen simplex chronicus (Fig. 1.4). Chronic eczematous inflammation is a clinicopathologic entity and does not indicate simply any long-lasting stage of eczema. If scratching is not controlled, subacute eczematous inflammation can be modified and converted to chronic eczematous inflammation.

There is moderate to intense itching. Scratching sometimes becomes violent, leading to excoriation and digging, and ceases only when pain has replaced the itch. Patients with chronic inflammation scratch while asleep. They are:

1. Less vesicular and exudative.
2. More scaly, pigmented and thickened.
3. More likely to show lichenification—a dry leathery thickened state, with increased skin markings, secondary to repeated scratching or rubbing.
4. More likely to fissure.

Treatment

Chronic eczematous inflammation is resistant to treatment and requires potent steroid therapy.

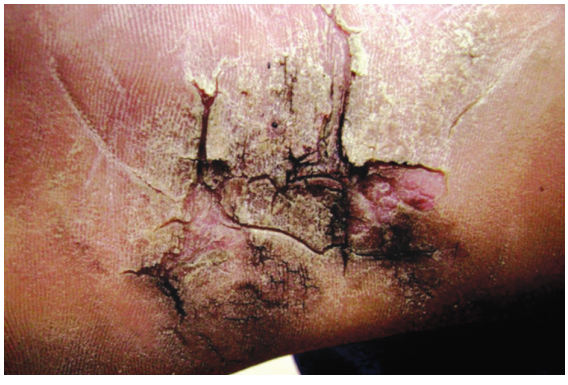

Fig. 1.4: A case of hyperkeratotic eczema, a prototype of chronic eczema

Intralesional injection. Intralesional injection is a very effective mode of therapy. Lesions that have been present for years may completely resolve after one injection or a short series of injections. The medicine is delivered with a 27- or 30-gauge needle, and the entire plaque is infiltrated until it blanches white. Resistant plaques require additional injections given at 3- to 4-week intervals.

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