

DEFINITION

1. **Eczema**, rather than being a disease as such, is a pattern of acute, subacute or chronic inflammatory response of the skin to multiple exogenous and endogenous agents. The histological changes of eczema are confined to the dermis.
2. The terms '**eczema**' and '**dermatitis**' are often regarded as being synonymous. However, some authors still use the term 'dermatitis' to include all types of cutaneous inflammation, dermal and epidermal. Thus, all eczema is dermatitis but not all dermatitis is eczema.

CLINICAL MANIFESTATIONS

3. The condition presents with itching, redness, scaling, clustered papulovesicles, crusting and discharge. The clinical pattern of the condition may be altered by the site of the body affected and by the nature of the normal skin. This is particularly seen in the hands and feet, where characteristic patterns called pompholyx and cheiropompholyx are seen.
4. The classification of clinical eczema is difficult and the nomenclature is controversial. Many cases are of multiple aetiology and two or more forms of eczema may be present in the same patient.

AETIOLOGY

5. The aetiology of eczema is unknown. The eczemas are broadly classified into **exogenous eczemas** and **endogenous eczemas**. Exogenous eczemas are associated with identifiable external factors. It was formerly considered that such external agents would lead to eczema in any exposed individual. Endogenous eczemas, on the other hand, were considered to result not from environmental factors but to be constitutionally determined. However, there is now growing evidence of an interaction between external and constitutional factors. Thus, the classification into exogenous and endogenous eczema is not absolute.

EXOGENOUS ECZEMAS

6. Identifiable external factors may cause a skin reaction, either by a direct local effect on the skin at the point of contact or by an immunological mechanism. When there is a direct local effect, a skin reaction can occur at the time of the initial contact. In other cases it is a dose-related phenomenon which does not become clinically manifest until there have been multiple exposures.
7. Eczema involving an immunological mechanism does not become clinically apparent at the initial exposure. Initially, the body's immune system recognises the allergen as foreign and mounts an immune response. When the individual next encounters the offending external agent, the body is primed to react and a skin reaction results.

8. **Irritant Dermatitis.** This is due to contact with irritants, both chemical and physical. It may be acute, occurring at the first exposure, or cumulative, when the skin reaction becomes apparent after repeated exposure.
9. **Allergic Contact Dermatitis.** This is a delayed or cell-mediated immune reaction to allergens which are of low molecular weight. These are usually simple chemicals, classically nickel or chromium. In this type of eczema there is no reaction at the initial contact, but only on subsequent challenge.
10. **Infective Dermatitis.** This is caused by contact with micro-organisms or their products. This type of eczema is not universally recognised by dermatologists. There may be difficulty in making a firm diagnosis because it may be difficult to determine whether the eczema arose from primary exposure to the infective agent or whether it is an eczema of some other origin which has become infected.
11. **Photo-allergic contact dermatitis.** This form of eczema is immunologically based. It can be elicited in persons who have been sensitised by previous exposure to a photosensitiser and at the same time to light. There are many potential photosensitising drugs, including phenothiazines, topical non-steroid anti-inflammatory drugs and topical sulphonamides.
12. **Eczematous dermatophytosis.** This is due to an allergic reaction to a dermatophyte infection elsewhere in the skin. A classic example is where a person has a localised fungal infection of the feet, which is later followed by a widespread generalised eczematous eruption.

ENDOGENOUS ECZEMAS

13. **Atopic eczema.** Atopy is a genetically determined disorder in which there is an increased liability to form IgE antibodies. This leads to an increased susceptibility to asthma, hay-fever and dermatitis. In atopy, true eczematous changes are not always present. The fundamental cause remains unknown, but the production of clinical disease depends on an interplay of constitutional and environmental factors.
14. There is a family history in 70% of cases of atopic eczema. Associated with the excess IgE production is a tendency to anaphylaxis and a decrease in cell-mediated hypersensitivity.
15. Atopic eczema may become clinically apparent at any age, the onset not being confined to infancy and childhood. In adults the characteristic clinical feature is lichenification, particularly of the flexures and the hands.
16. Atopic persons are more likely to have anaphylactic reactions to drugs or insect bites, and food allergy is more common than in non-atopics. Patients with both active and quiescent atopic eczema are liable to acute systemic infection with Herpes simplex (eczema herpeticum) and vaccinia virus (which results in Kaposi's varicelliform eruption).
17. Conjunctivitis is common in atopics. Bilateral cataract occurs in up to 10% of severe adolescent or adult cases. The peak age of onset is between 15 and 25. On examination of the eyes, there is a characteristic slit lamp appearance.

18. **Seborrhoeic dermatitis.** This occurs chiefly in areas of the body which are rich in sebaceous glands, such as the scalp, face and upper trunk. The condition may be precipitated by infection with *Pityrosporum ovale*. It has a high incidence in coal miners, soldiers in wartime and in those suffering from AIDS. It may also occur as a complication of Parkinsonism, where seborrhoea is common.
19. **Asteatotic eczema** (eczema craquele, winter eczema). This occurs where the skin has a decreased lipid content. It affects the legs, arms and hands and is often seen in the elderly. It may be a presenting sign of myxoedema, or it may relate to diuretic therapy, cimetidine therapy, the use of topical corticosteroids or gross zinc deficiency.
20. **Nummular eczema** (discoid eczema). This consists of discrete coin-shaped plaques of eczema with well demarcated edges. It is of unknown aetiology. On present evidence it appears to be a reaction which may be precipitated by many factors, including infection, dry skin, emotional stress and local physical or chemical trauma.
21. **Pityriasis alba.** This is a condition of unknown aetiology in which erythematous patches are followed by depigmentation of the affected skin. It predominantly affects children.
22. **Gravitational eczema** (venous eczema). This is eczema secondary to venous hypertension. The exact mechanism by which it is produced is unknown. The condition was formerly known as varicose eczema, but this term is inappropriate as varicosity of veins is not necessary for development of the condition. Rather, it is associated with increased perfusion of the tissues of the lower leg. It usually occurs on the inner aspect of the lower leg and it may be associated with deep venous thrombosis. This type of eczema may be complicated by secondary contact dermatitis, caused by infection or rubbing.
23. **Juvenile plantar dermatosis.** This is characterised by dry fissured dermatitis of the plantar surface of the forefoot. It occurs almost exclusively in children aged 3 to 14 years. It is considered that resins and dyes used in the manufacture of socks and shoes may be responsible for its cause. Friction, sweating and atopy may also play a part.
24. **Metabolic eczema.** Eczema may be associated with systemic disease, including diabetes mellitus, gout, asthma, malabsorption, Wiskott-Aldrich syndrome, Job's syndrome, hypogammaglobulinaemia, Jung's disease, pellagra and phenylketonuria.

CONCLUSION

25. **Eczema** is not a disease entity but, rather, a reaction of the skin to varied and complex internal and external factors. Its precise aetiology is unknown. The clinical syndrome results from an interplay between endogenous factors and environmental factors. The clinical picture varies according to the type of eczema and the site of the body involved.

REFERENCES

Burton J L. Eczema, Lichenification, Prurigo and Erythroderma. In: (Eds) Champion R H, Burton J L and Ebling F J G. Textbook of Dermatology. Oxford. Blackwell Scientific Publications. 5th Ed. 1992:537-589.

Champion R H and Parish W E. Atopic Dermatitis. In: (Eds) Champion R H, Burton J L and Ebling F J G. Textbook of Dermatology. Oxford. Blackwell Scientific Publications. 5th Ed. 1992:589-611.

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