Lecture No. 24

Eczema

Eczema defines an inflammatory skin disorder with many possible causes, the hallmark being a histological process called spongiosis: the accumulation of oedema fluid within and between keratinocytes in the epidermis, giving a "spongy" appearance. It is common, affecting between 5 and 10% of the population, and 10-20% of children.

The main symptoms are **itching or, in the infant the appearance of dry, red patches with occasional vesicles overtaken by crusting**. In infancy, the cheeks, abdomen and limb surface are involved, whilst in older children the classical distribution is on the elbow, knee and wrist flexor surfaces. Prolonged scratching leads to the development of discoloured plaque with a leathery texture (lichenification). In approximately 75% of cases the disorder is self-limiting and clears in the first few years of life. The pathogenesis of allergic eczema is less clear.

The relationship between diet and eczema is perhaps the most intriguing and controversial aspect of the disease. Well designed studies in which potential triggers in food (**cow's milk, hen's egg proteins, peanuts**) are avoided appear to bring about an improvement in eczema.

The diagnosis is made on **history, examination and skin testing** (skin prick test evokes a wheal and flare response but not eczematous lesions, it is possible to replicate eczema like plaques by patch testing with allergens such as house dust mite extracts in sensitive individual), with **the total serum IgE level** also usually raised and IgE directed against airborne and food allergens is a prominent feature.

Contact dermatitis

Contact dermatitis is an inflammatory skin disease caused **T_H1-cell mediated (type IV) hypersensitivity** to external agents (see figure below) which come into contact with the skin. These agents (known as **haptens**) are usually molecules of relatively of low molecular weight (<1 KDa) and are not immunogenic, but they can penetrate the epidermis and bind to certain proteins in the skin (carrier proteins) and become highly reactive molecules.

Classification

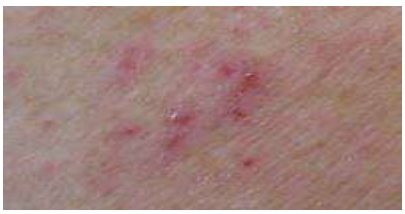
1. Acute toxic dermatitis.
2. Cumulative dermatitis.
3. Allergic Contact dermatitis.

Pathogenesis

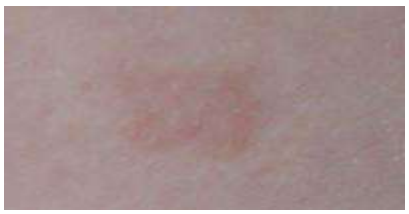
Two phase of pathogenesis are recognized: an **induction phase**, from time of initial antigen contact to sensitization of T lymphocytes, and an **elicitation phase**, from antigen re-exposure to the appearance of dermatitis. In **the induction phase**, Langerhans cells bind the hapten-carrier protein complex and present it in association with MHC class II antigens to T lymphocytes ($CD4^+$). Induction of cellular immunity to a contact skin sensitizer can occur within 7-10 days of first contact but it usually happens after many months or years of exposure to small amounts of antigens. Individual sensitivity varies according to the nature of the agent, its concentration and the genetic susceptibility of the person exposed. Re-exposure to the relevant antigen triggers **the elicitation phase** which produce dermatitis. In this phase, effector T lymphocytes carried via the circulation to the skin meet the antigen (composed of hapten complexed to carrier protein) presented by Langerhans cells and other antigen-presenting cells in the epidermis. Activation of T lymphocytes releases cytokines which cause induce skin inflammation, with keratinocytes proliferation, hyperplasia of the epidermis and consequent protective thickening.

Diagnosis

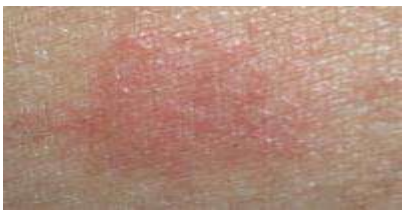
The diagnosis of contact dermatitis depends on **a careful medical history, the distribution of the lesions, and patch testing**. In the patch test, a suspected contact sensitizer is applied to normal skin (usually on the upper back) and covered for 48h. The reaction is read after 2 and 4 days. In a positive response, there is inflammation and induration at the test site.



Irritant reaction



+/- reaction



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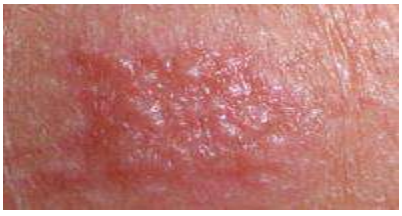

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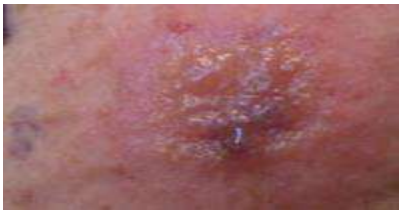

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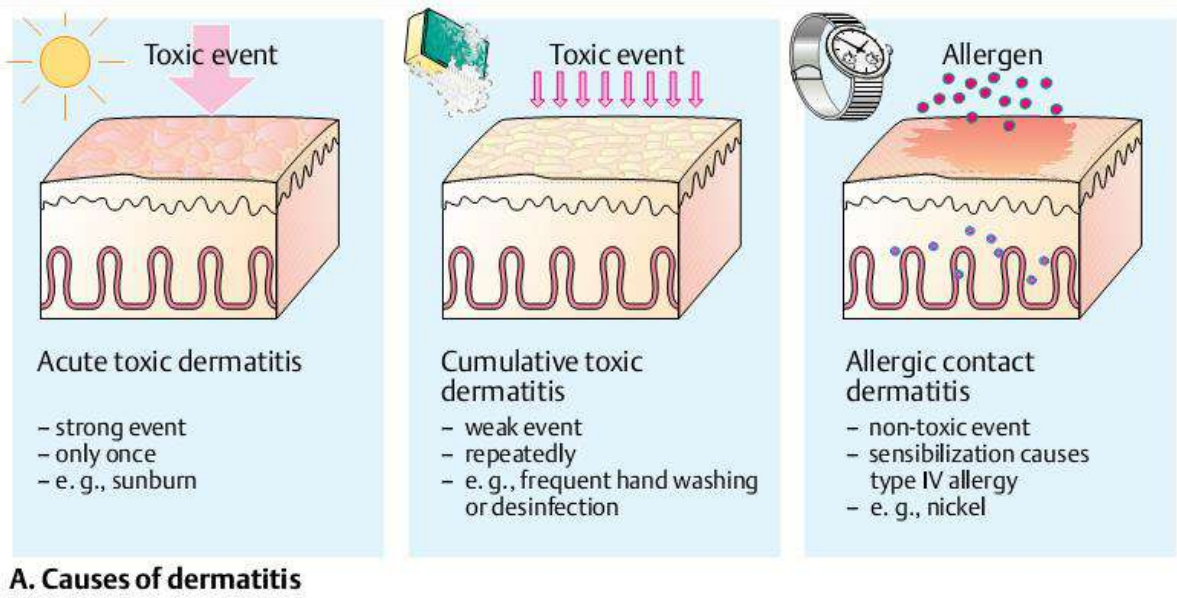
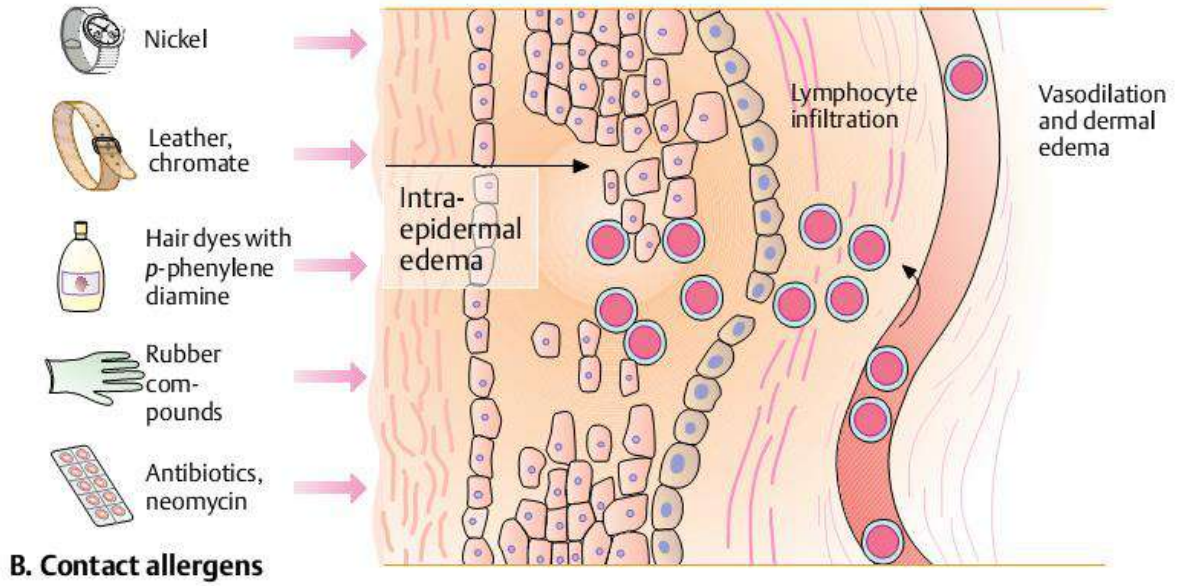
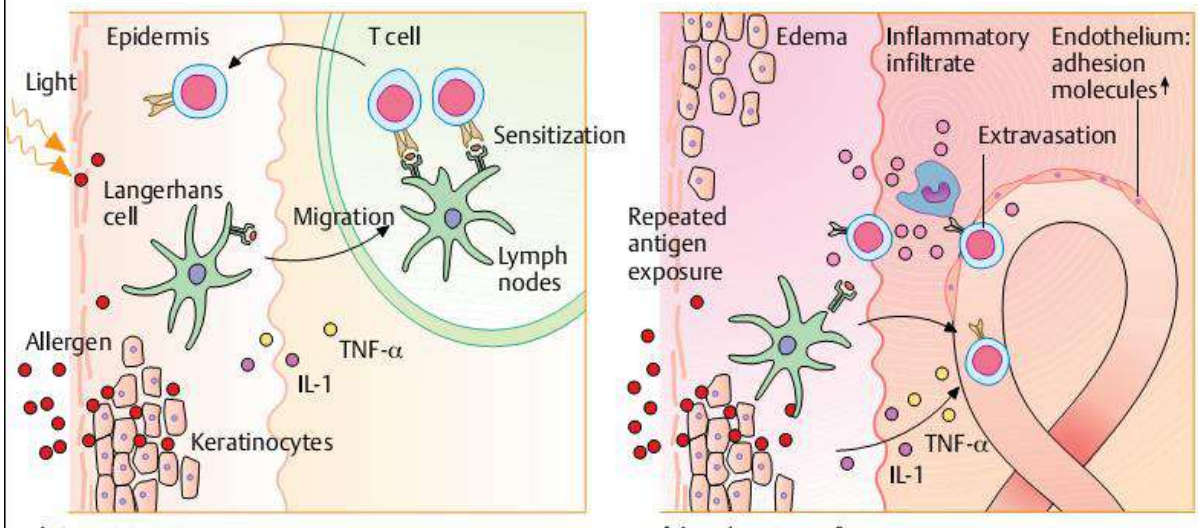


A. Causes of dermatitis



B. Contact allergens



a) Sensitization **b)** Induction of eczema

C. Pathogenesis