

Eczema

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Definition

inflammatory or non-inflammatory.
✓

-
- ? ? Dermatoses : Condition of the skin. may involve the hair and nails as well.
 - ? ? Dermatitis : Inflammation of the skin.
 - ? ? Eczema : Type of dermatitis.
 - ? ? 'Ekze' in Greek means "to boil over" ^{papulo-vesicular}
 - ? ? Definition : Eczema is type of dermatitis characterized by erythema, edema papulo-vesicles, oozing in acute stage, crusting and scaling in subacute & lichenification in the chronic stages and histologically characterized by spongiosis.
 - ? "All eczemas are dermatitis, but not all dermatitis are eczemas."

Classification

- ❑ Exogenous eczemas : External cause for the eczema is identifiable.
- ❑ Endogenous eczemas : An internal cause or an inherent property of the skin is responsible.
- ❑ Some types of eczema are precipitated by both external and internal factors. Eg: Xerotic eczema

Exogenous eczemas

? Irritant dermatitis

? Allergic contact dermatitis

? Photodermatitis

Endogenous eczemas

? Atopic dermatitis

? Pityriasis alba

? Seborrhoeic dermatitis

? Discoid eczema

? Hand eczema

? Asteatotic eczema

? Gravitational eczema

? Lichen simplex chronicus ? Prurigo nodularis

Acute eczema

? Acute eczema Classical clinical features

? Intense itching

? Intense erythema *Bright red.*

? Oedema

? Papulovesicles *Blisters.*

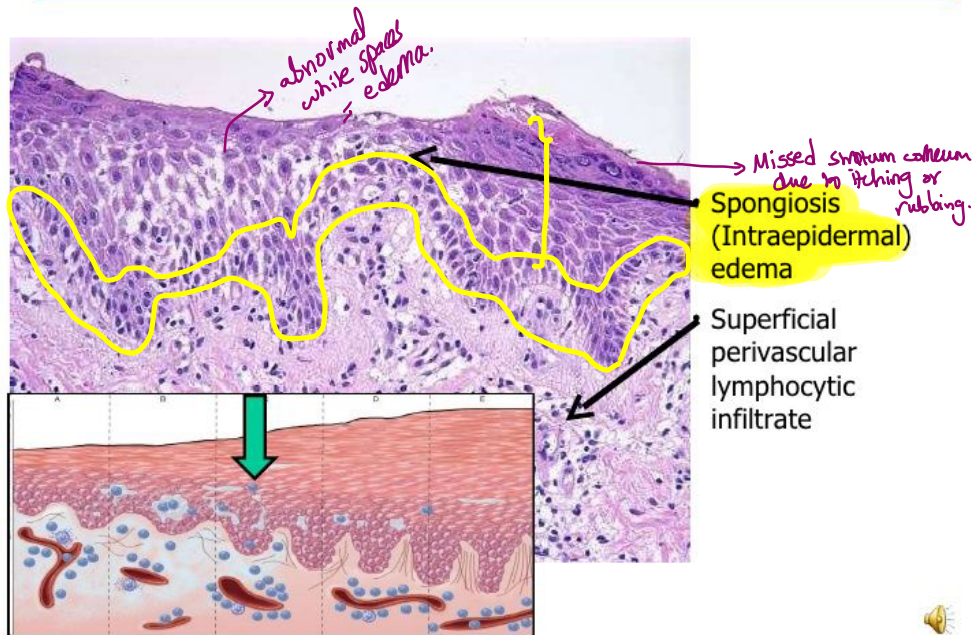
? Oozing

? exudates





ECZEMA – histology



Subacute eczema Classical clinical features

- ? Erythema (lesser than in acute stage)
- ? Crusting and scaling
- ? Fissuring
- ? Slight to moderate itching

Chronic eczema Classical clinical features

- ? Dryness of skin + scaling + hypo/hyperpigmentation
- ? Excoriation
- ? Fissuring
- ? Lichenification - combination of thickening, hyperpigmentation & increased skin markings.
 - * Acanthosis → increase in the epidermis
 - * Hyperkeratosis → increase in the stratum corneum
 - * As a defense mechanism by the body after itching
 - * not only in AD.

Sub-acute and chronic eczema

□ Edema tends to diminish, and acanthosis (an increase in thickness of the spinous layer) develops. In addition, there are parakeratosis and hyperkeratosis. *→ in stratum corneum.*

□ The increase thickness of the epidermis with the underlying edema and infiltrate produces areas of skin in which the normal skin lines become greatly exaggerated, so the skin looks rather like the bark of tree (lichenification). *↑ incomplete maturation.* *↑ pathophysiology of lichenification.*

□ This often associated with severe itching.



Pathophysiology: of atopic dermatitis

① Genetic factors in endogenous eczema, filaggrin gene defects lead to impaired skin barrier. (More water loss + ↑ infections).

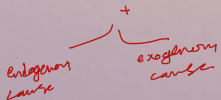
The balance between T helper 2 (vs) Th1 and Th 17. is lost.

* $TH2 > TH1 - TH17$ in eczema. → AD

psoriasis

- Atopy → Genetic susceptibility for having type I IgE reaction

(Both have genetic factors + defective skin barrier)



Complications

✓ viral bacterial
Defective skin barrier.
infection, hyper/hypopigmentation superimposed
allergic reaction and impaired quality of life.

• Superficial bacterial infection → Impetigo.

Staph
↓
Staph.

staph aureus impetigo → honey in color.

• Viral infections → HSV eczema herpeticum
HPV

Causes of eczema

1.Exogenous cause(due to external factors)

contact dermatitis:

- ☐ Irritant contact dermatitis
- ☐ Allergic contact dermatitis
- ☐ Photosensitive dermatitis

2.Endogenous cause(constitutional)

- ☐ Atopic eczema.
- ☐ Seborrhoeic eczema.
- ☐ Gravitational (varicose) eczema.
- ☐ Asteatotic eczema.
- ☐ Discoid eczema.
- ☐ lichen simplex
- ☐ Juvenile plantar dermatosis

Atopic eczema

* **ATOPY** : A GENETICALLY MEDIATED PREDISPOSITION TO AN EXCESSIVE IGE REACTION (Type 1 hypersensitivity, Immediate).

* **ATOPIC TRIAD** : ECZEMA, ASTHMA, ALLERGIC RHINITIS ask about all in history

* IT RUNS IN FAMILIES. ENVIRONMENTAL IMMUNOLOGICAL Dust, Pollens, irritants, stress

* MOSTLY AFFECTS INFANTS AND YOUNG CHILDREN

IN INFANTS : MOSTLY AFFECTS FACE AND SCALP

< 1y.o

more localization → IN CHILDREN : MOSTLY AFFECTS FLEXOR SURFACES AS POPLITEAL FOSSA, AND WRIST • while in psoriasis vulgaris → on the extensors.

* RESOLVES IN CHILDHOOD, REMAIN SUSCEPTIBLE TO THE EFFECTS OF PRIMARY IRRITANTS => RECRUDESCENCE OF ECZEMA some patients might outgrow the symptoms But atopy is still

ATOPIC ECZEMA

patches of red, dry, itchy scaly skin

Later on : lichenification may be seen *chronic eczema.*



-eczema herpeticum:

Eczema herpeticum is herpes simplex viral infection superimposed onto the skin affected by eczema (usually in atopics).

There is frequently a history of close contact with an adult with herpes labialis

Clinically, there are multiple small 'punched-out' looking ulcers, especially around the neck and eyes.

Eczema herpeticum is a serious complication of eczema that may be life threatening (systemic acyclovir)



Figure 4.8 Eczema herpeticum.

- Herpes → small, clustered vesicles (punched out) due to erosion and deroofing.

"Herpetiform"

* Yellow color indicates impetigo ~> gasni superimposed infection by staph.

life-threatening * Herpes labialis → disseminate easily, in head and neck could cause encephalitis.



* Melanocytes carry melanin by melanosomes $\xrightarrow{\text{to}}$ Keratinocytes } Normally }



- on sun exposed area
- can have other characteristics of AD.

* ^{white} Pityriasis alba is a variant of atopic eczema in which ^{scaly} pale patches of hypopigmentation develop on the face of children.

Color gets there normally after a while but emollients are beneficial.

Juvenile plantar dermatosis is another variant of atopic eczema in which there is dry cracked skin on the forefoot in children

no signs of inflammation.



ATOPIC ECZEMA

* The most common complication is **2ry bacterial infection** causing **impetigo**

- Viral warts

- Pityriasis alba

- Juvenile plantar dermatosis

* Eczema herpeticum is a life threatening complication

* Treatment :

- Identify and avoid irritants

- Emollients at bath time

- Topical steroids , mild for young children, potent for older

- If 2ry infection occurs then use systemic antibiotics

- Antihistamines , Tacrolimus (topically) , cyclosporine , UV light may be used also



✓ FLEXURAL INVOLVEMENT IN ATOPIC DERMATITIS

✓ THE PRESENCE OF LICHENIFICATION AND FISSURING TELLS US THIS IS CHRONIC ECZEMA

- People who have this have constitutional (Not curable" (can control the symptoms not the cause of the disease itself)

الأَكزيما الدهنية

Seborrhoeic eczema = seborrhoeic dermatitis

- * linked to malassezia furfur part of the normal flora.

papulosquamous disease, which characteristically involves areas rich in sebaceous glands with high sebum production and large body folds.

- * In general it appears as ^{thick} greasy ^{plaques} yellowish scaling over red inflamed skin

- * Bimodal age, men > women infants very active sebaceous glands at puberty

- * DDx : psoriasis

- * Very mild of seborrhoeic dermatitis → Dandruff.



Figure 15.2 Seborrhoeic dermatitis.



Figure 16.8 Seborrhoeic dermatitis.



Figure 19.14 Seborrhoeic dermatitis.

Clinical features (Infants)

sebacous glands' activity will be reduced later on.

- ✓ [?] Commonly affects within first 3 months of life; rare after 6 months of age; affects both sexes equally.
- ✓ [?] Usually starts in 1st week after birth.
- ✓ [?] Affects the scalp (vertex and frontal areas; the 'cradle-cap' area), diaper area, face (forehead, eyebrows, eyelids, nasolabial folds, temples), retroauricular folds, neck and the axillae.
- ✓ [?] Lesions comprise tiny papules covered with yellow, greasy scales; and redness in the diaper area and axillae.

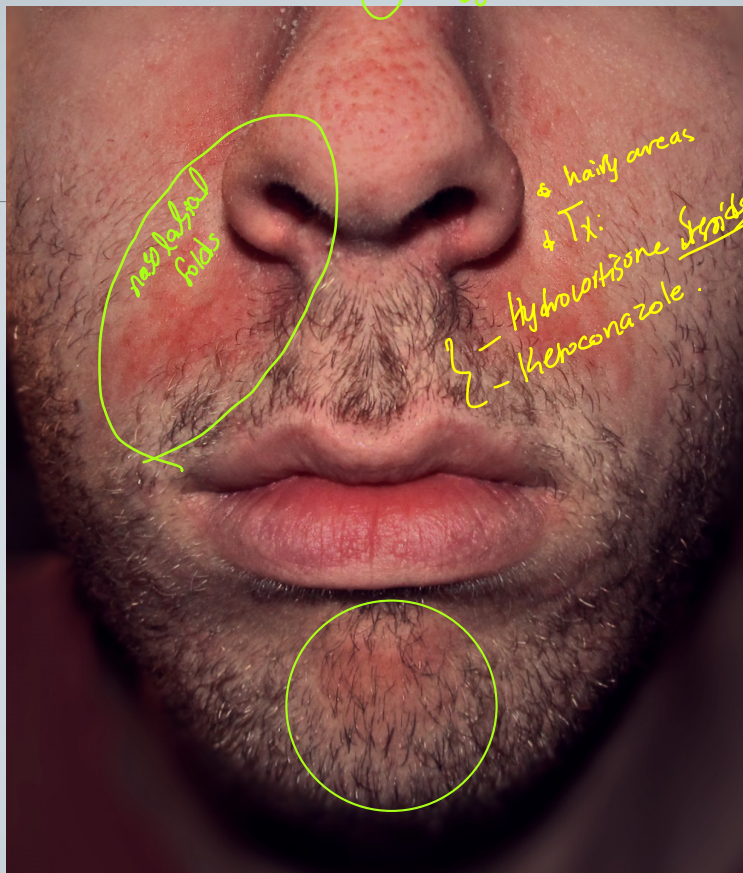
Clinical features (Adults)

- ✓ ? Affects hairy areas; mostly men (30 to 60 years).
- ✓ ? Scalp : Earliest sign is dandruff; later followed by greasy scales and retroauricular fissuring. Inflammation and itching are associated with dandruff in seborrheic dermatitis.
- ✓ ? Face : Scaling & erythema of forehead, medial portion of eyebrows, eyelids, nasolabial folds, lateral part of nose and retroauricular region.
- ✓ ? Trunk : Papules, greasy scales.
- ✓ ? Flexural areas : erythema, greasy scaling and secondary infection.

Seborrheic eczema



Adults
→.



Seborrhoeic dermatitis usually requires treatment over many years, as there is no cure for this condition. It is important to make this clear to patients, who otherwise tend to try many treatments in their quest for a permanent solution to the problem.

To reduce inflammation ↗

Topical hydrocortisone is effective, but the problem recurs when treatment is stopped.

Steroid lotions or gels and tar shampoos will help

the scalp

maintainance therapy ↗

Ketoconazole shampoo and cream

imidazole/hydrocortisone combinations, are also effective.

Eczema craquelé

Asteatotic eczema

Dry eczema.

- * Low humidity : desert, high altitude, travel
- * Excessive bathing especially using soaps and detergents
- * dry, itchy, scaly, cracked skin with network of shallow erythematous fissures in the epidermis that produce an appearance that resembles **'crazy paving'**.
- * Usually starts on the **shins**, common in elderly
- * Treated simply by emollients and mild topical steroid ointment

crazy paving



Dyshidrotic

Pompholyx – vesicular eczema

- * cheiropompholyx : endogenous eczema of **pālms**
- * Pedopompholyx : endogenous eczema of **soles**
- * Usually occurs in people aged 20– 40 years
- * itchy rash presents on plams and soles centers and with **tiny vesicles** that may evolve into bullae
- * In severe cases : nail dystrophy + paronyhcia
- * 2ry bacterial infections is common
- * Tt : potassium permanganate soaks + potent corticosteroid cream Systemic antibiotics may be needed

Dermatophytosis - vesicular eczema



Discoid eczema

- * Well defined , scattered coin
– disc shaped plaques that are
extremely itchy with vesicles
or crusting
- * Usually in the middle age *makes*
On the limbs
- * Tt : emollients and potent
corticosteroid ointment



Stasis eczema

- * Common in patients with chronic venous HTN, DVT, varicose veins, obesity, pregnancy

- * Diffuse erythema, scaling, crusting and itching

- * lower third of both legs without pain

- * Hyperpigmentation, venous ulcers are common

- * Tt : treatment of underlying condition + moderately potent corticosteroid cream + emollients

compression Before → check ABPI first to exclude arterial insufficiency thus amputation may be.

Stasis eczema



Juvenile plantar dermatosis

* Mainly due to the socks and shoes that are impermeable (low humidity , dryness)

* Weight bearing areas

* Treated by emollients and changing the type of shoes or socks



Exogenous causes “ Contact dermatitis “

- / Irritant \leadsto Toxic (alcohol, acids, alkalis ... etc.) / Depends on frequency and concentration
 - \ Allergic \leadsto All or none.
Specific
Differ from person to another.
-

very sharp demarcation.

[?] Primary Irritant Contact dermatitis

[?] A primary irritant is a substance which, if applied in high enough concentration to normal skin is capable of producing an eczematous response following a single exposure.

Examples: caustic liquids such as acids and alkalis (strong), detergents and mineral oils (mild).

A primary irritant will cause eczema in everyone if it is applied in sufficient concentration for a sufficient time.

❓ Wear and tear eczema in housework and in many industries is examples .

Diaper rash ❓ **Napkin dermatitis (nappy rash)** is another type of this eczema. In this type of eczema the skin folds are spared, whereas the reverse is true for candidosis. This due to prolonged contact with urine or faeces, and results of ammonia production by bacteria.

90% are irritant contact dermatitis but not necessarily



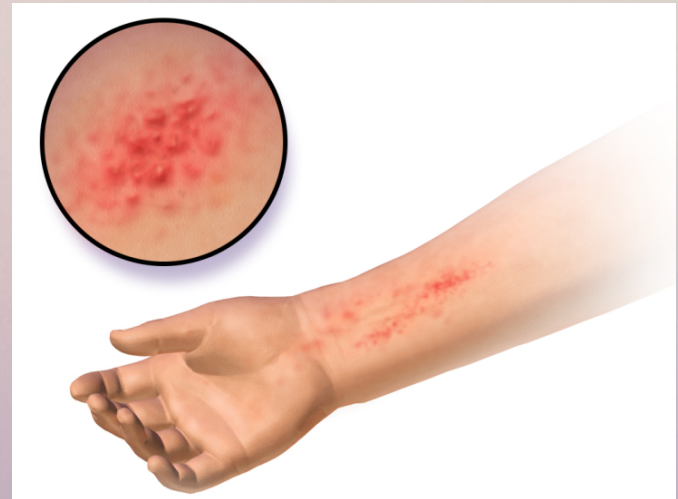
[?] Allergic Contact dermatitis (Hypersensitivity Dermatitis)

* prenatogenous spread → distant spread sometimes.
* Areas not exposed are affected

This is due to the development of delayed hypersensitivity (type 4 allergy) to a specific chemical (sensitizer or allergen). Such allergens will not cause eczema even in high concentration in a normal person, but severe eczema may be provoked by brief exposure to a very low concentration in a sensitized person.



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Differences Between Irritant and Allergic Contact Dermatitis*

		Irritant CD	Allergic CD
Symptoms	Acute	Stinging, smarting → itching	Itching → pain
	Chronic	Itching/pain	Itching/pain
Lesions	Acute	Erythema → vesicles → erosions → crusts → scaling	Erythema → papules → vesicles → erosions → crust → scaling
	Chronic	Papules, plaques, fissures, scaling, crusts	Papules, plaques, scaling, crusts
Margination and site	Acute	Sharp, strictly confined to site of exposure	Sharp, confined to site of exposure but spreading in the periphery; usually tiny papules; may become generalized
	Chronic	Ill-defined	Ill-defined, spreads
Evolution	Acute	Rapid (few hours after exposure)	Not so rapid (12–72 h after exposure)
	Chronic	Months to years of repeated exposure	Months or longer; exacerbation after every reexposure
Causative agents		Dependent on concentration of agent and state of skin barrier; occurs only above threshold level	Relatively independent of amount applied, usually very low concentrations sufficient but depends on degree of sensitization
Incidence		May occur in practically everyone	Occurs only in the sensitized

*Differences are printed in bold.

Irritant contact dermatitis



when exposed to irritant in low concentration for long periods
↓



**Top Ten Contact Allergens (North American Contact Dermatitis Group)
and Other Common Contact Allergens***

Allergen	Principal Sources of Contact
Nickel sulfate	Metals, metals in clothing, jewelry, catalyzing agents
Neomycin sulfate	Usually contained in creams, ointments
Balsam of Peru	Topical medications
Fragrance mix	Fragrances, cosmetics
Thimerosal	Antiseptics
Sodium gold thiosulfate	Medication
Formaldehyde	Disinfectant, curing agents, plastics
Quaternium-15	Disinfectant
Bacitracin	Ointments, powder
Cobalt chloride	Cement, galvanization, industrial oils, cooling agents, eyeshades
Methyldibromoglutaronitrile, phenoxyethanol	Preservatives, cosmetics
Carba mix	Rubber, latex
Para-phenylenediamine	Black or dark dyes of textiles, printer's ink
Thiuram	Rubber
Parahydroxybenzoic acid ester	Conserving agent in foodstuffs
Propylene glycol	Preservatives, cosmetics
Procaine, benzocaine	Local anesthetics
Sulfonamides	Medication
Turpentine	Solvents, shoe polish, printer's ink
Mercury salts	Disinfectant, impregnation
Chromates	Cement, antioxidants, industrial oils, matches, leather
Parabenes	Biocides, preservatives
Cinnamic aldehyde	Fragrance, perfume
Pentadecylcatechols	Plants, e.g., poison ivy

* Over 3700 chemicals have been reported to cause ACD.

* Most common allergen
worldwide → Nickel
in fake jewellery.

Allergic contact dermatitis



Fig. 1 Patient on postoperative day 7 following left ectropion repair.

Doxycycline

[?] Photodermatitis

The combination is the problem.

A

B

C

Interaction of light and chemicals absorbed by the skin.

Allergic vs toxic

Drugs systemic vs local.

Borolen

plants
Phytophotodermatitis due to contact with plant material and sunlight.

-Patients with chronic actinic dermatitis (chronic eczema on sun-exposed skin) are allergic to sunlight.



[?] Occupational dermatitis

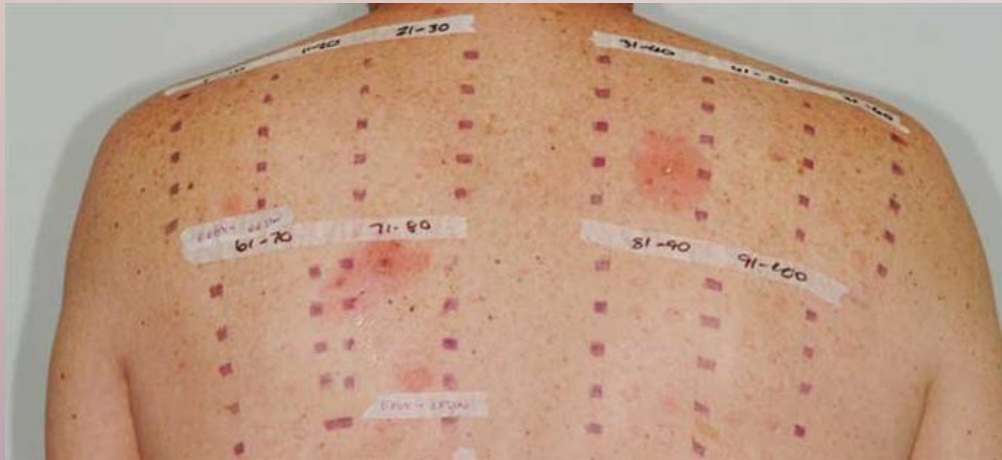
1. first occurred during employment
2. improves away from work
3. exposure to a known irritant/ allergen

Occupational irritant contact dermatitis

Occupational allergic contact dermatitis

Patch Test

- *
[?] This is a test for allergic contact dermatitis.
- [?] It may be tested by applying a substance which is suspected to cause the eczema (in a solution form) to an area of unaffected skin under a small patch of adhesive tape. The patch is removed after 48 hrs or earlier if severe irritation develops.
- [?] Positive reactions consist of erythema, some times with swelling and vesiculation.
- [?] This test should be avoided in acute phase.



-Management OF ECZEMA:

moisturizers to
regain the skin
barrier.

The general treatments are:

1- Topical steroids.

2- emollient.

-Steroids have different potencies, twice daily in general (occlusion increases the potency by 100* Folds).

-Antibiotics (macrolide, fusidic acid) for 2 weeks, (risk of resistance).

-Antihistamine (cetirizine).

-Unresponsive types of eczema could be treated with immunomodulator and inhibitors. (azathioprine, tacrolimus, ciclosporins, MTX).

Thank you for your attention