Eczema & Dermatitis

Eczema is an inflammatory reactive pattern of skin to many and different stimuli characterized by itching, redness, scaling and clustered papulovesicles.

Eczema and dermatitis are synonymous terms. These are the most common skin conditions seen in the dermatology clinics all over the world with a prevalence of 10% in the general population.

Clinical features:

The clinical signs are similar in all types of eczema and vary according to the duration of rash.

Acute eczema reaction:

1- Redness and swelling, usually with ill- defined margins.
2- Papules, vesicles and, more rarely, large blisters.
3- Exudation and cracking.
4- Scaling.

Chronic eczema reaction:

1- May show all the above features, but it is usually less vesicular and exudative.
2- Lichenification, a dry leathery thickening and increased skin markings, is secondary to rubbing and scratching.
3- Fissures and scratch marks.
4- Pigmentation changes (hypo- and hyper-).

Histopathological features:

The hallmark of acute eczema is edema of the epidermis (spongiosis) and of the chronic eczema is hyperkeratosis and acanthosis. Upper dermal vasodilatation and perivascular inflammatory cells infiltrate occur in all stages of eczema.

Classification:

A. Exogenous:
   1- Irritant contact dermatitis.
   2- Allergic contact dermatitis.
   3- Photodermatitis.
   Others
B. Endogenous:
   1- Atopic dermatitis.
   2- Seborrhoeic dermatitis.
   3- Discoid eczema.
   4- Asteatotic eczema.
   5- Gravitational eczema.

6- Lichen simplex chronicus.
7- Pompholyx.
8- Pityriasis alba.
Others

Complications:

1- Heavy bacterial colonization and super infection.
2- Local superimposed allergic contact dermatitis.
3- Severe forms of eczema affect the quality of life.

Treatment:

1- Topical treatment:

Acute weeping eczema:

Bed rest.
Liquid applications (e.g. K+permanganate soaks).
Topical steroid lotions or creams.
Non sticky dressings and clothes.

Subacute eczema:

Steroid creams are the mainstay of treatment.

Chronic eczema:

Best is steroid in an ointment base.

2- Systemic treatment*:*

1- Antibiotic for bacterial superinfection,
2- Antihistamines for itching, and
3- Short courses of systemic steroids may be needed.

Topical steroids:

These are commonly classified into 4 classes of potency:

Class	Potency	Example
Class I	Super potent	0.05% Clobetasol propionate
Class II	Potent	0.1% Betamethasone valerate
Class III	Moderately potent	2.5% Hydrocortisone
Class IV	Weak steroids	1% Hydrocortisone

The potency of steroid used and the duration depend on the type of lesion, its severity and the type of patient, e.g:

• Nothing more potent than 1% hydrocortisone should be used on:
the face,
intertrigenous area, or
in infancy
except in specialized circumstances.
• Very potent steroid should not be used for long time. (>2 weeks).

Exogenous Dermatitis

These are *mainly* caused by exogenous (contact) factors:

1- Irritant contact dermatitis.
2- Allergic contact dermatitis.
3- Photodermatitis.
Others

Irritant Contact Dermatitis

Is an inflammatory reaction of the skin occurs from exposure to an irritant for sufficient time and in sufficient concentration. Immunological process is *not* involved and pervious sensitization is *not* required for dermatitis occur.

Irritant is any substance capable of inducing cell damage if applied for sufficient time and concentration. These may be weak or strong irritants.

There is a wide range of susceptibility for weak irritants and they usually cause chronic dermatitis after repeated exposures while strong irritants produce acute reactions after brief exposure.

The main irritants are alkalis (detergents, soaps, bleaching agents), acids, solvents, petroleum and dusts. Water can cause irritant contact dermatitis.

Treatment is based on:

Avoidance of irritant (or protection from it)
The use of topical steroids and emollients.

Examples of Irritant Contact Dermatitis

1- House-wife dermatitis

A very common disease frequently seen among housewives (or males in certain jobs) due to frequent washing. Water and detergents cause the irritation. The eruption begins with dryness and redness of fingers especially the tips, chapping is seen on the back of hands, and erythematous hardening of the palms with fissures develop. Allergic contact dermatitis may develop secondarily.

2- Napkin eczema

Mainly affects infants and babies at the site of underwear due to repeated and sustained exposure to urine and faeces. Elderly incontinent people may be affected also. Glazed and sore

erythema affects the napkin area and usually spares the skin folds. Bacterial or candida superinfection is common.

3- Lip licking dermatitis
Usually seen in young children due to frequent wetting of the lip by licking. Repeated cycles of wetting and drying will eventually cause chapping and then eczema.

Allergic Contact Dermatitis

An acquired sensitivity to various substances that produces an inflammatory reaction in those (& *only those*) who have been exposed to the allergen previously.

The mechanism is delayed type IV hypersensitivity reaction. First exposure to the allergen is required to induce hypersensitivity but no clinical reaction. The allergen is then carried by langerhans cells to local lymph nodes where memory T-cells are produced. This is called (*the induction phase*) and take about 3 weeks.

Second exposure to the same allergen will stimulate the memory cells that induce lymphocytes proliferation and cytokine release, producing the dermatitis. This is called (*the elicitation phase*). Sensitization is systemic, persist indefinitely and desensitization is seldom possible.

Common allergen are:

Nickel	(jewelry),
Dichromate	(cement, leather),
PPD	(hair dye),
Parabens	(cosmetics),
Colophony	(plaster),
Neomycin	(topical antibiotic).

Examples of Allergic Contact Dermatitis :
1- Earlobes dermatitis: due to nickel-containing ear ring. Similar reaction can occur on the wrists, and/or fingers of nickel-sensitized individuals..
2- Cement dermatitis: due to dichromate found in cement.
3- Eyelid dermatitis: may be caused by nail varnish, preservatives in local medications, or it could be air born ACD.
4- Lipstick dermatitis, hair dye dermatitis, shoes dermatitis.

Diagnosis:
Is confirm by (Patch Test), which depend on type IV cell mediated hypersensitivity reaction. The test is don by applying the suspected allergens on the back of the patient under occlusive aluminum discs or patches for 48 hrs, the removed. Positive reactions produces erythema, papules and vesicles at the site of the allergen disc or patch.

Treatment :
Same as in irritant contact dermatitis.

Main differences between two types of contact dermatitis

Feature		Irritant CD	Allergic CD
1	Cause :	Irritant.	Allergen.
2	Previous exposure:	Not required.	Essential.
3	Affected sites:	Sites of direct contact with little extension.	Sites of contact and distant sites.
4	Timing:	Rapid onset (4-12 hours) after contact. Lesions develop at first exposure	Onset generally after 24 hours or longer after exposure. No lesions after first exposure.
5	Mechanism:	Direct effect (non immunological).	Type IV hypersensitivity reaction (immunological).
6	Susceptibility:	Everyone susceptible in varying degrees to appropriate concentration.	Only some patients susceptible.
7	Patch test	No role	Helpful to confirm diagnosis.