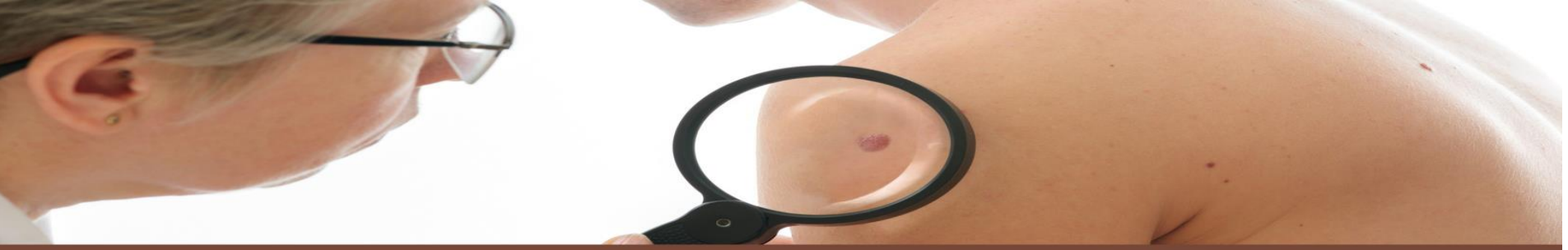

433 Teams

DERMATOLOGY

L3-General principles of eczema /dermatitis

(Atopic dermatitis and others)

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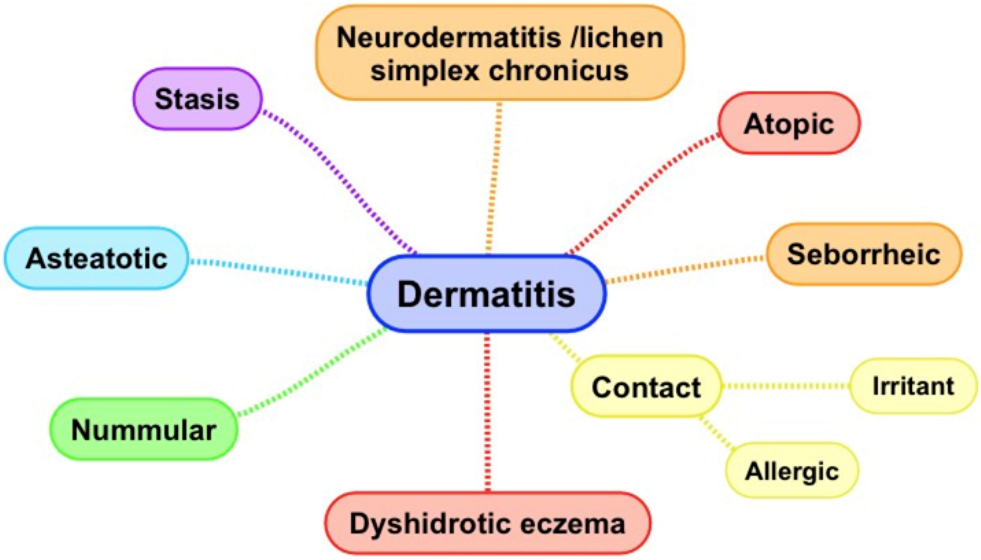
Objectives

- To know the definition & classification of Dermatitis/Eczema
- To recognize the primary presentation of different types of eczema
- To understand the possible pathogenesis of each type of eczema
- To know the scheme of managements lines

Mind Map



Hypersensitivity Reaction

Type 1: Immediate Hypersensitivity Reaction

- Mediated by IgE to specific antigens
- Mast cells stimulated and release histamine
- Reaction within 15-30 minutes of exposure
- Examples: Anaphylaxis (e.g.penicillin) ,Urticaria , Angioedema .

Type 2: Cytotoxic **Antibody** mediated Reaction

- Mediated by IgG and IgM to specific antigens
- Examples: Transfusion Reaction ,Rhesus Incompatibility (Rh Incompatibility), **Hashimoto**' thyroiditis.

Type 3: Immune Complex Reaction

- Antigen-Antibody complexes deposit in tissue
- Reaction within 1-3 weeks after exposure
- SLE, serum sickness , vasculitis:Examples

Type 4: Delayed-Type Hypersensitivity (cell mediated)

- Mediated by T-Lymphocytes to specific antigens
- Reaction within 2-7 days after exposure
- **Examples: Allergic contact dermatitis (e.g. Nickel allergy)**

1-Eczema (Dermatitis)

Definition:

is an **inflammatory** **skin** disease.

Skin inflammation characterized by→tchy, scaly, patches of erythema

Pathogenesis:

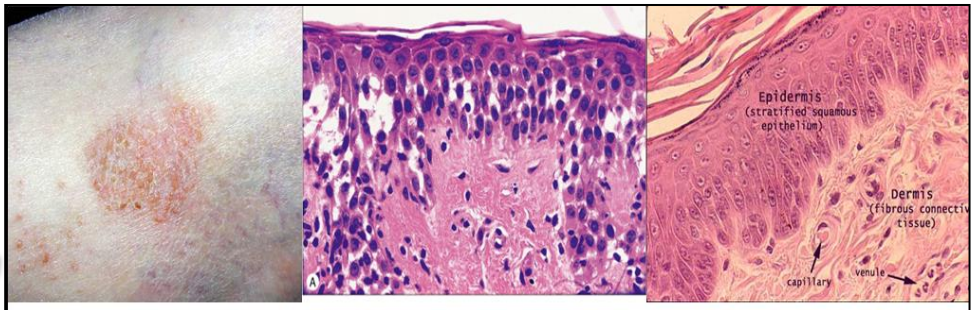
It is an epidermal reaction to specific Antigens; these antigens may be internal or external, acting singularly or in combination

Clinical picture:

Most eczemas share certain general features, and each different type of eczema will have some distinguishing markers of their own. Eczema can be broadly classified as acute, subacute, and chronic

Acute:	Subacute:	Chronic:
erythema	scales	lichenificaion
papules & vesicles	Excoriation	hyperkeratosis
oozing		

When you go from acute to chronic there well be more dryness and scaly



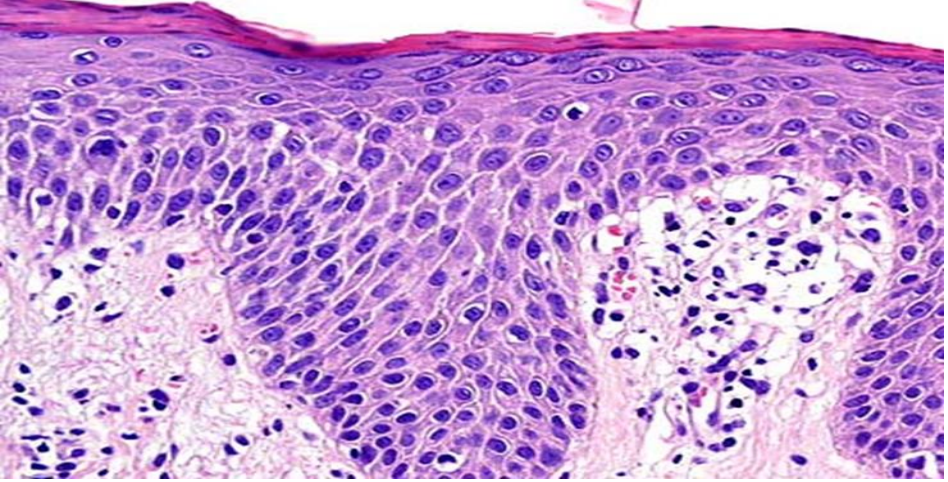
Dermatitis="Eczema"=Spongiosis : fluid (intra cellular edema) between the cells , in acute dermatitis is increased and present as vesicles

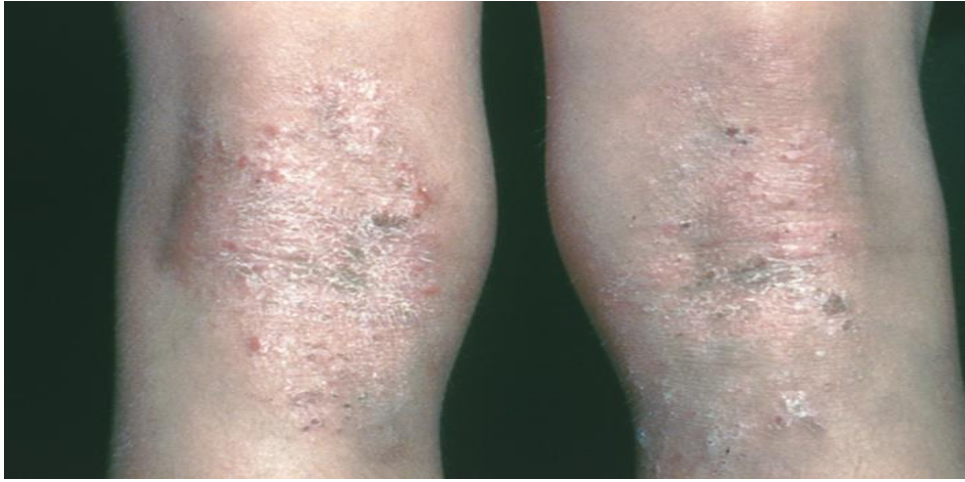
Acute Dermatitis

erythema and edema

papules, vesicles, and sometimes bullae accompanied by exudation and crusting

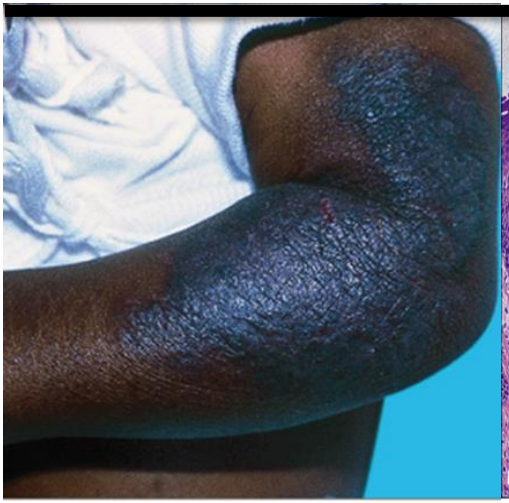
Clinically an eczematous disease may start at any stage and evolve into another

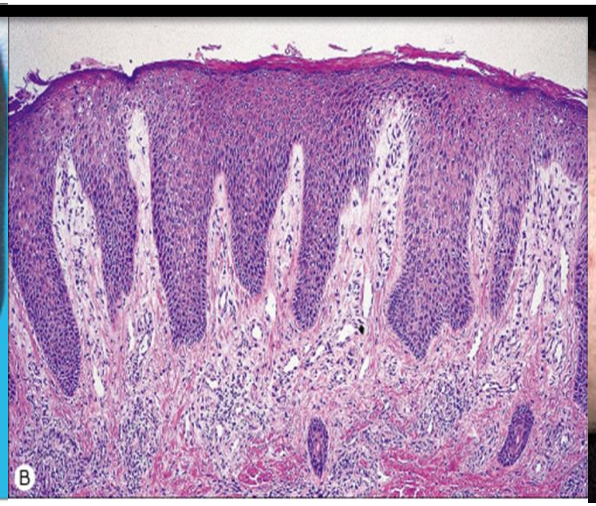


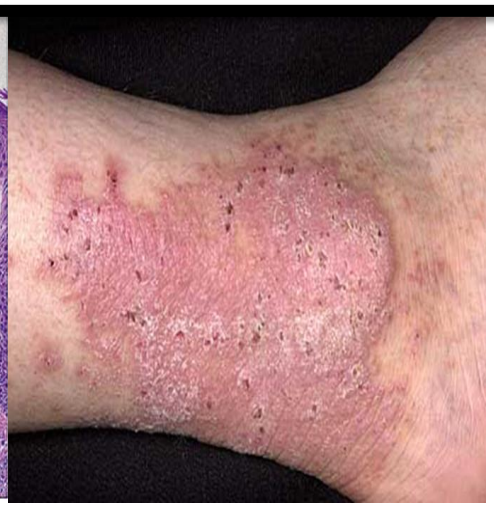


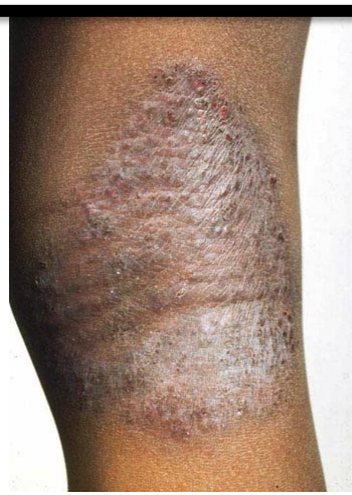
Subacute Dermatitis

Commonly misdiagnosed as tinea









Chronic Dermatitis

Commonly misdiagnosed as psoriasis

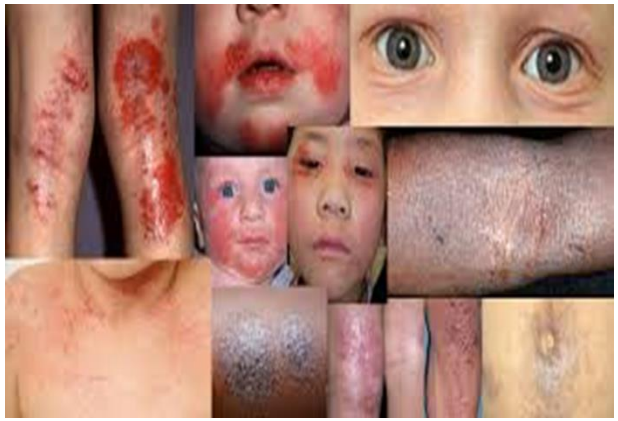
less erythema and edema. presence of lichenification, scaling,

*and fissuring more thickening and dryness(acanthosis)

*acanthosis: thickening of epidermis (stratum corneum)

Atopic Dermatitis

chronic relapsing itchy skin disease in genetically predisposed patients
associated with personal or family history of other atopic conditions as asthma, allergic rhinitis, conjunctivitis or atopic eczema



- Affects 15-30% of children, 2-10% of adult
- 60% begin during the first year
- 85% begin before 5 years
- Up to 70%: spontaneous remission before adolescence

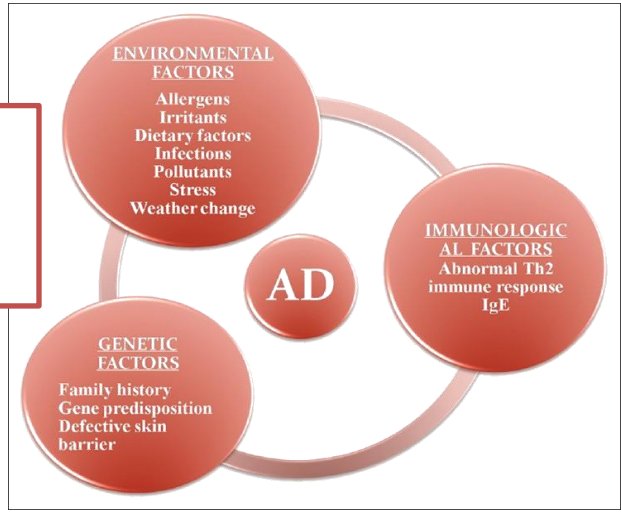
Pathogenesis:

No single etiology =No "magic bullet" cure

-Genetic predisposition
-immune mediated (increase IgE), T-helper cell2 activation
-Impaired skin barrier.(defect in stratum corneum envelop)

defective epidermal differentiation (filaggrin mutations) and resultant impaired barrier function of the skin

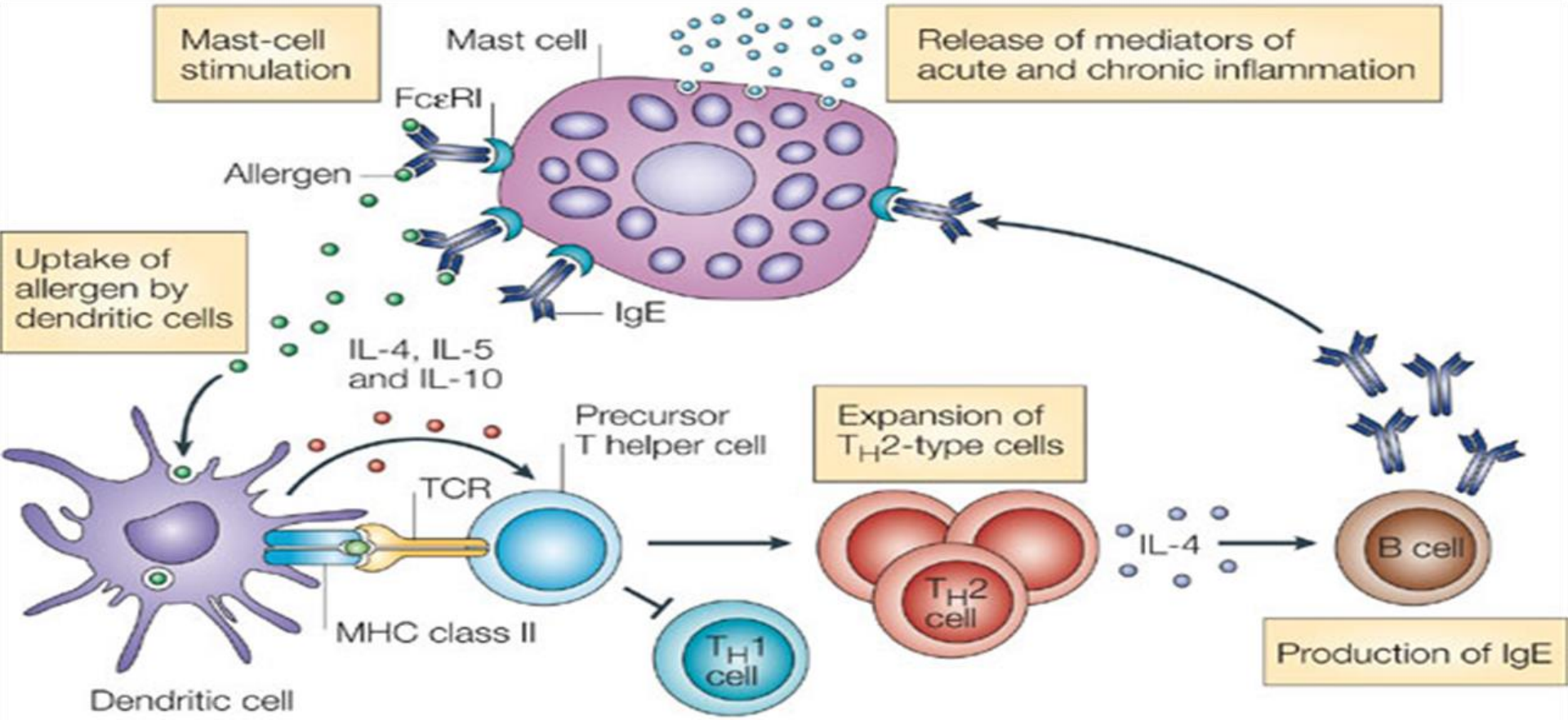
- Allergy, increased tendency to certain allergens
- Infection : skin of pts. with AD* is colonized by S aureus.
infection with S. aureus often causes a flare of AD
- AD and Food! minor role most important factor is impaired skin barrier



- **AD associated with local infiltration of Th 2 that secrete IL-4, IL-5, IL-13, IL-31**
- **More than 50% develop asthma**
- **75% develop Allergic Rhinitis**
- **Complex interrelationship of genetic, environmental, and immunologic**

*** AD: atopic dermatitis**

more explanation of Pathogenesis→ in Next slide



1. Allergens are taken up by dendritic cells and presented to T cells.
2. In the absence of childhood microbial exposure, the balance between T helper 1 (TH1) and TH2 cells is altered.
3. TH2 cells encourage the production of immunoglobulin E (IgE) by B cells.
4. Allergen-specific IgE then binds to the high-affinity receptor for IgE (FcepsilonRI) on mast cells.
5. Allergen exposure induces crosslinking of receptor-bound IgE with subsequent mast-cell degranulation and the release of pro-inflammatory molecules.

IL, interleukin; TCR, T-cell receptor.

Clinical picture of Atopic Dermatitis:

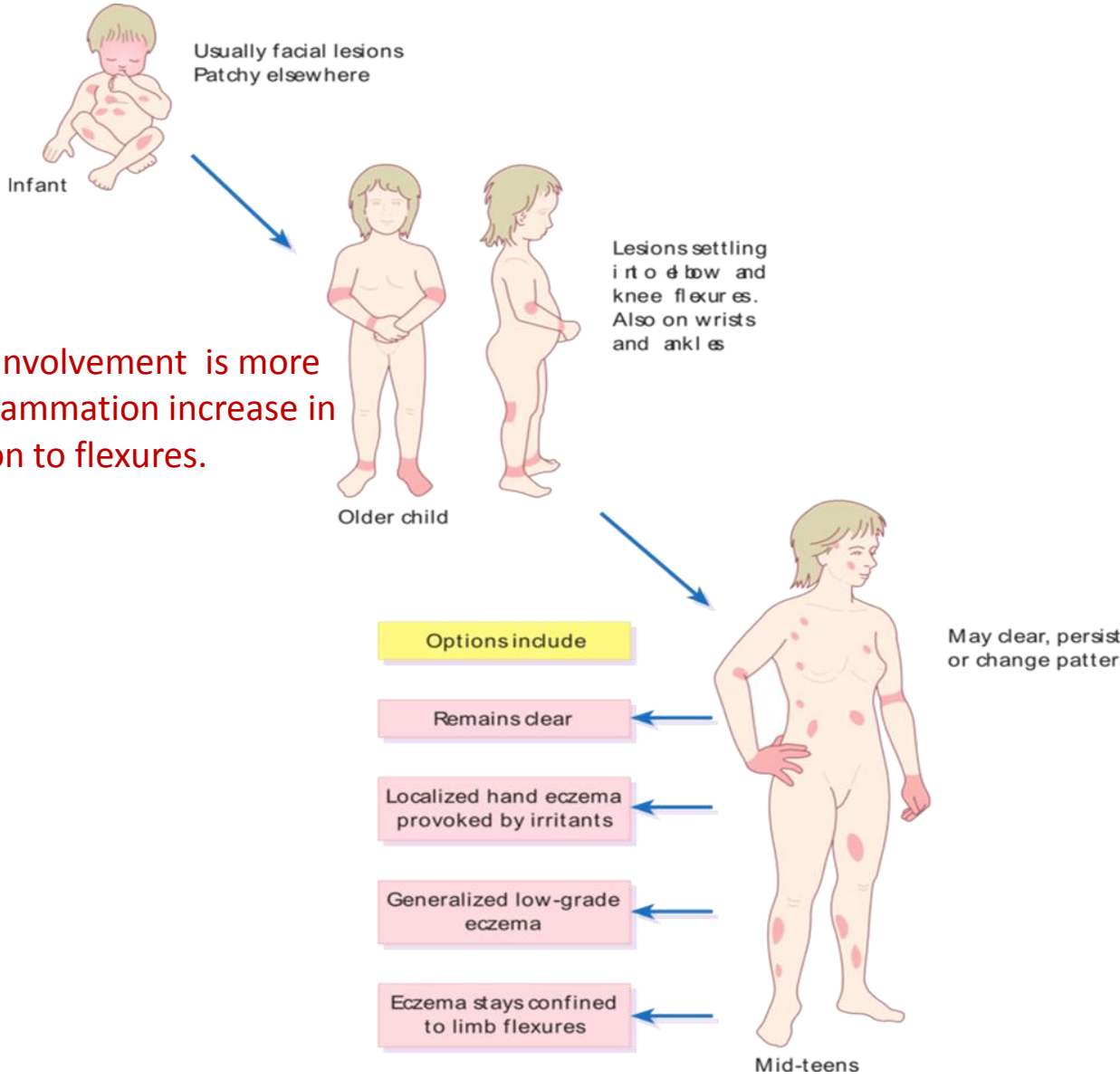
same as dermatitis

Three stages:

-Infantile
-Childhood
-Adulthood

Next slides

Acute inflammation & extensor/facial involvement is more common in infant whereas chronic inflammation increase in prevalence with age as does localization to flexures.



Pattern of atopic eczema varies with age

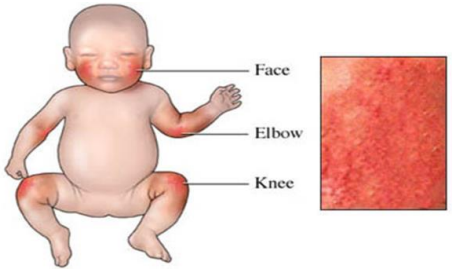
Clinical picture

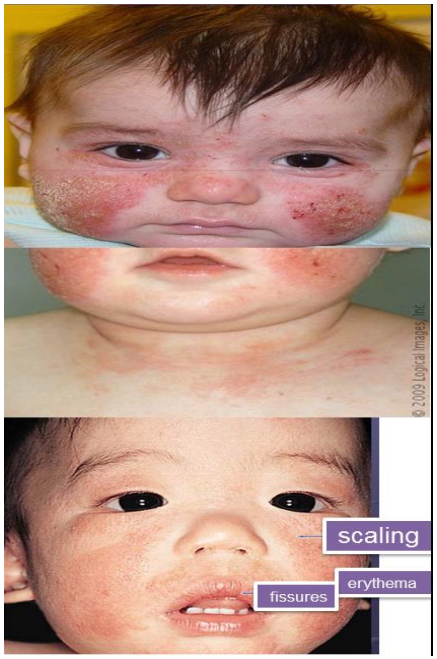
1-Infantile atopic dermatitis

Infants develop an itchy vesicular eczema(acute) on cheeks and hands often with secondary Infection

in face and extensors

- **Diaper area is usually spared**





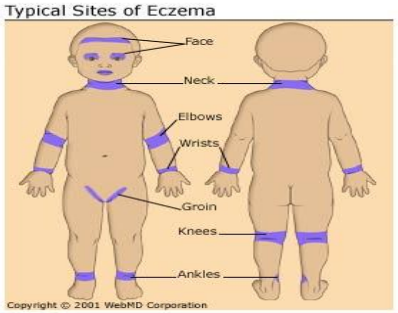
Ill-defined dusky erythematous edematous plaque, with faint scales on the cheeks, forehead, and chin.

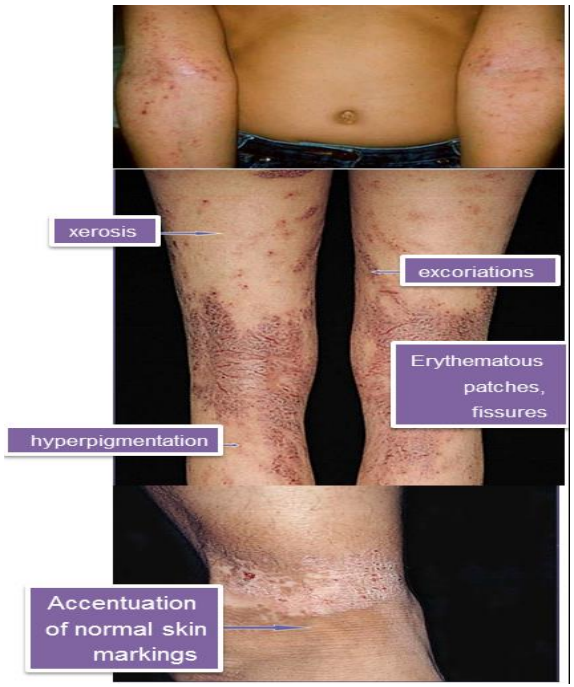
Psoriasis is well defined

2-Childhood atopic dermatitis

Children develop lesions **subacute and chronic** at antecubital and popliteal fossae, neck, wrists, and ankles. (**Flexors**)

Lichenification, excoriations, and dry skin are common as well as post-inflammatory hyperpigmentation

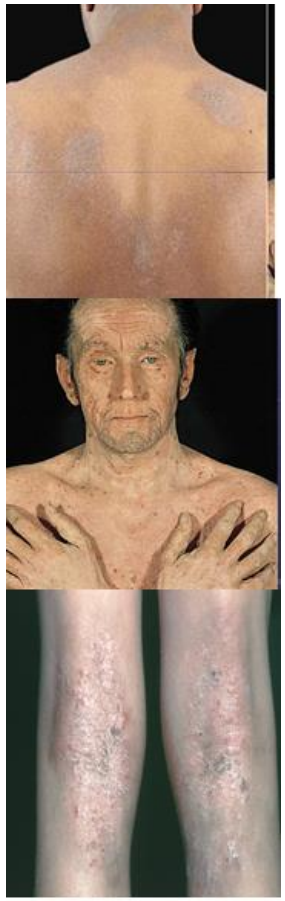




3-Adult atopic dermatitis

In adults

- most common manifestation: **flexors and hand dermatitis**.
- **sub acute or chronic** severe form of generalized and lichenified atopic eczema.



Widespread chronic atopic dermatitis

Diagnostic Features Of Atopic Dermatitis

Mainly Clinically diagnosis

criteria	Major	Minor	
	1.pruritus 2.typical morphology and distribution 3.chronicity 4.Personal or family history of atopy	Xerosis :dry skin Icthyosis/hyperlin جلد السمكear palms/keratosis pilaris: increase scaling and roughness of the hair follicles -IgE reactivity -Elevated IgE level -Early onset -Skin infection -Chelitis: eczema of lips -Nipple eczema	-Recurrent conjunctivitis -Keratoconus -Dennie morgan fold -Anterior cataract -Orbital darkening -Facial erythema -Pityriasis alba -Food hypersensitivity -White dermatographism
	3 out of 4 to diagnose	+3 minor criteria	

Pityriasis alba

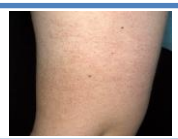
Misconception with vitamins deficiency

It is mild eczema With hypopigmentation

TX: mild topical steroid

Keratosis pilaris

Common in lateral aspect of the **thigh** and lateral side of the **arms**



Complication

Eczema Herpeticum

Caused by herpes simplex virus1 infection

Investigations: swap for viral culture + zank smear

TX: antiviral

Impetiginised dermatitis

Caused by S. aureus

TX: if immunocompetent > topical antibiotic

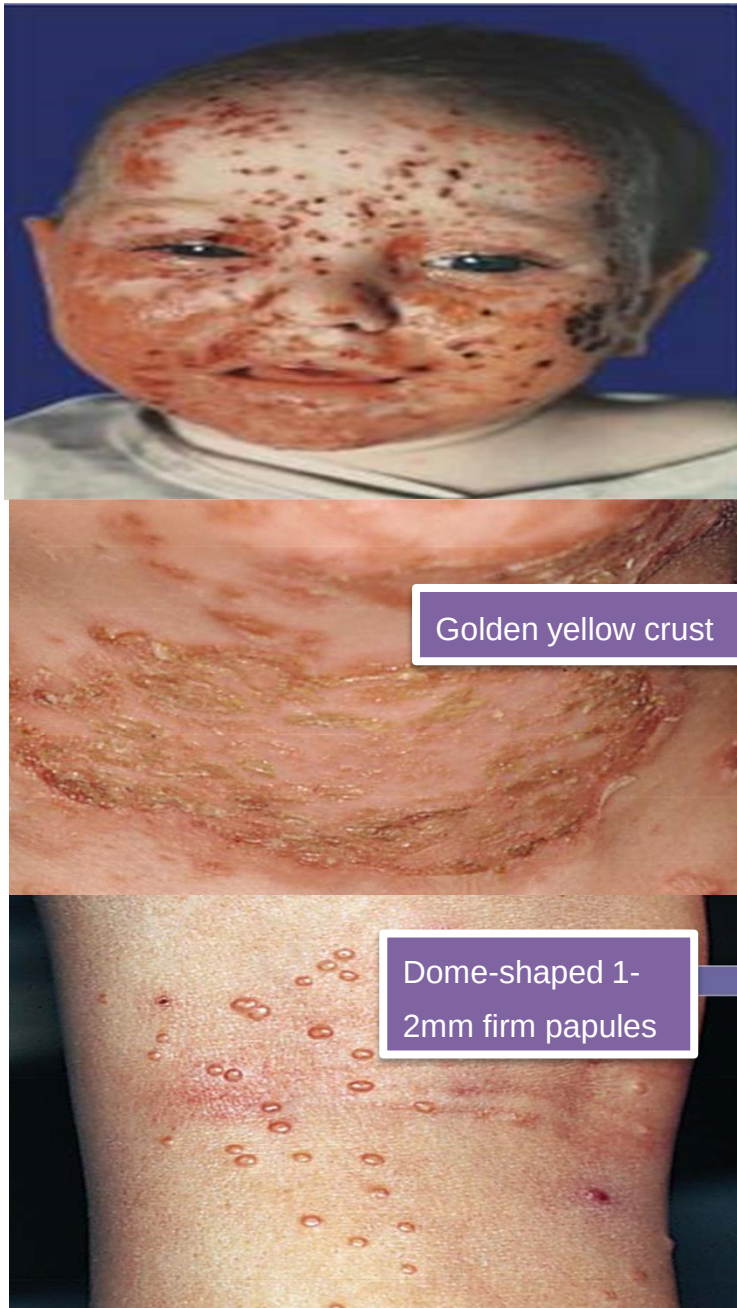
Immunocompromised > oral antibiotic

Mollusca Contagiosa

Example of papule with central implicated

Caused by Mollusca Contagiosum virus

With chronic itching



Management

Frame work for Treating Dermatitis:

Dry Skin:

Bathing, soap

Emollients/barrier repair

Inflammation:

- Topical steroids
- Topical calcineurin inhibitors

Pruritus/Sleep:

Sedating antihistamines

Infection:

Contributing factors

Dipper area doesn't affected by atopic dermatitis because it wet area

Education *important :*

1. **Emollient (motorization)**

e.g : petroleum jelly specially after the bath

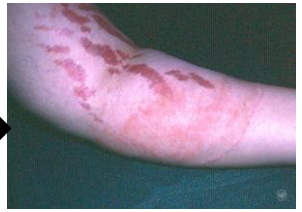
2- **Avoid irritant**

Avoid alkali soaps use non alkali soaps

Avoid woolen clothes and wear cotton instead

3- Food has a minor roll so don't stop any important element for the baby

25 y/o male treated for eczema in antecubital fossa with "some cream"



Treatment

Education.

-Emollient.

-topical steroid

Super potent : for thick skin e.g : palm – sole

Mild steroid : for thin skin and face

-**topical immunomodulators** (tacrolimus & pimecrolimus)

Don't have the side effect of topical steroid

-**oral antihistamine for sedation effect only**

-**oral Antibiotic** (for 2ry bacterial infection)

-ultraviolet light

-systemic steroid

-others: cyclosporine , methotrexate ,azathioprine, IVIG , *Biologic*

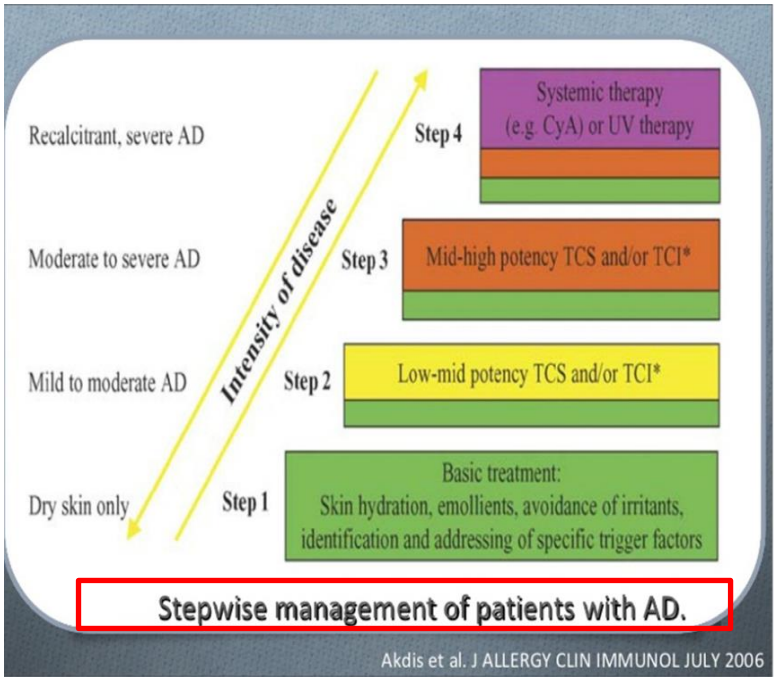
What are the side effects of topical steroid on the skin ?

Rare!

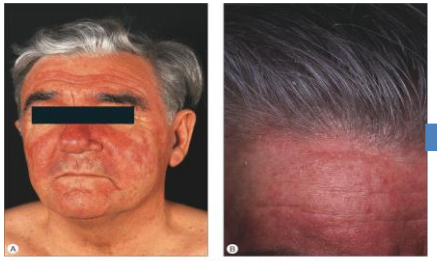
1-Reversible: **(epidermal)**

- Telangiectasia/prominent blood vessels
- Epidermal Atrophy/thin skin
- Acne/rosacea
- Prone to infection
- Delayed wound healing
- Increased hair growth
- Cushing syndrome in children with moon face

2-Non-reversible (dermal): dermal atrophy



52 y/o male with erythematous, scaly patches of face and scalp



DDx:
1- contact dermatitis
2-seborrheic dermatitis

Seborrheic dermatitis

Is a common mild chronic eczema typically confined to skin regions with high sebum production & the body folds

Pathogenesis:

-Seborrhea & abnormal sebum production.

-Commensal yeast Malassezia furfur (pityrosporum ovale)

Seborrheic dermatitis is defined by clinical parameters which include:

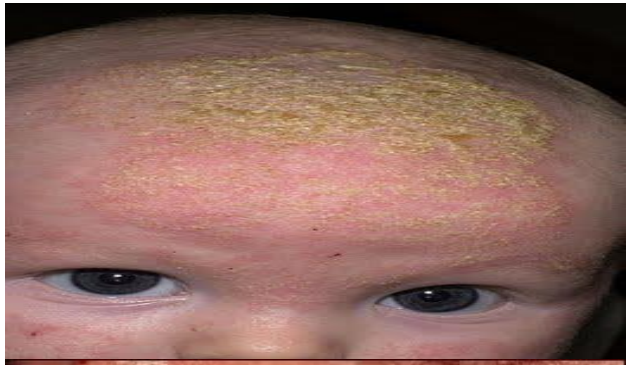
1-erythematous red-yellow , poorly circumscribed patches & thin plaques with bran-like to flaky (greasy) scales.

2-Limitation to those periods of life when sebaceous gland are active i.e. the **1st few months** of life(due to maternal androgens) & **post puberty**(due to increased androgens) (infantile & adult forms).

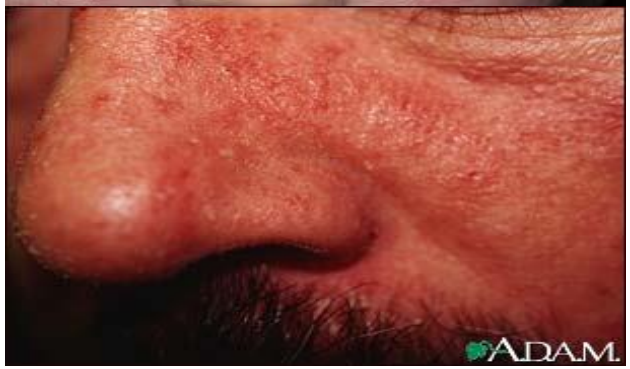
3- A predilection for areas rich in sebaceous glands

e.g: scalp , face, ears , presternal region & flexural areas (axillae, inguinal & inframammary folds , umbilicus).

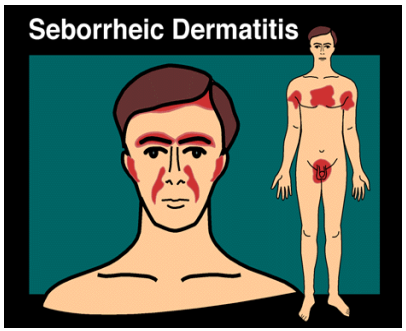
4-A mild course with moderate discomfort.

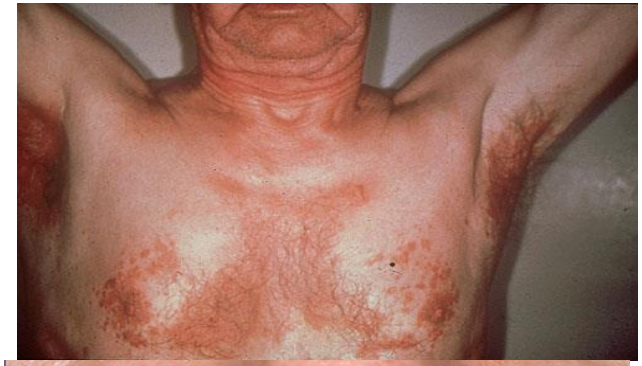


Cradle cap: is coherent scaly & crusty mass covering most of the scalp & can be seen in infanile seborreic dermatitis. We treat these patients with keratolytic

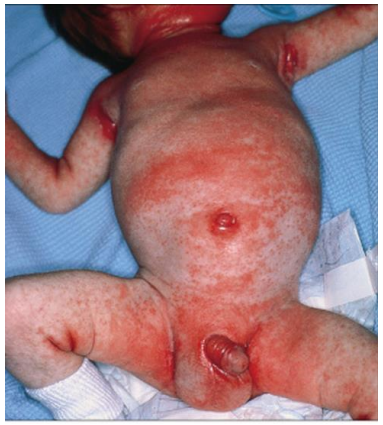


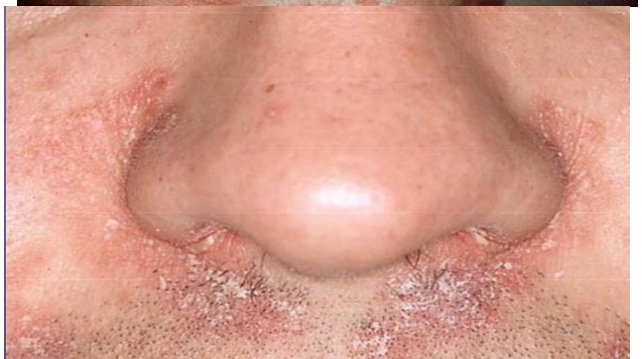
Seborrheic dermatitis with orangish, erytrhematous, thin plaques and patches over the nasal area





Seborrheic dermatitis of presternal area





Seborrheic dermatitis

Treatment:

-Medicated shampoo (e.g. containing coal tar, selenium sulfide or ketoconazole)

-Topical antifungal.

-low potency topical steroid.

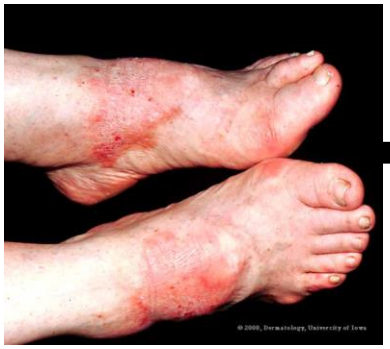
- Topical immunomodulators (tacrolimus & pimecrolimus)

- A preparation of salicylic acid (2–5%, depending upon the scaling) can be used for the scalp.

note(**seborrheic dermatitis** most of the time start at the **first 3 months** of life and get better after it while **atopic dermatitis start after 3 months**) but all can occur at any age

Infantile atopic dermatitis	***Infantile Seborrheic dermatitis***
-Markedly pruritic -Presents as erythema, papules and vesicles -Prominent on the cheeks and extensor surface of the limbs.	-Asymptomatic -Present as greasy scales over an erythematous base -Prominent on the scalp , nasolabial fold and body folds

Contact Dermatitis



24 y/o male 2 year h/o red, scaly feet
Lesion description: (bilateral, symmetrical well defined with irregular edges erythematous scaly thin plaques)
Diagnosis: contact dermatitis

Dermatitis precipitated **by an exogenous agent**:
-**Allergic contact dermatitis(immune mediated)**
-**Irritant contact dermatitis(cytotoxic)**

Definition:
Dermatitis resulting from type 4 reaction following exposure to topical substances in sensitized individuals (requires induction and elicitation phase (lag time to reaction). We have acute, subacute and chronic.

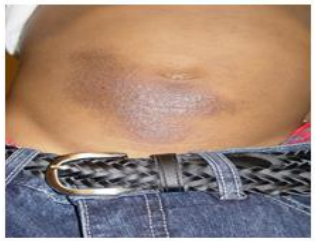
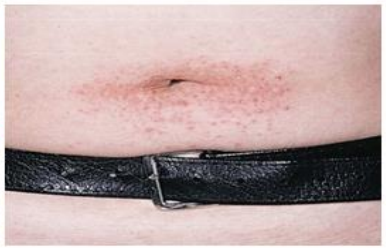
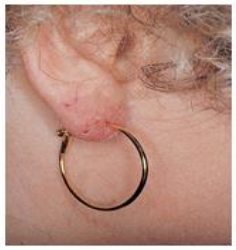
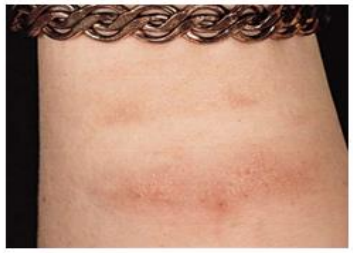
-Acute form:
present with crusted erythematous papules, vesicles & bullae that is well demarcated & localized to the site of contact with the allergen.
-ACD can be more diffuse in distribution.

1-Allergic contact dermatitis(ACD)

Common allergens eliciting contact dermatitis:
- nickel (affects 10% of women and 1% of men),
- perfumes
- Fragrances
- preservatives
- hair dyes,
- rubber latex

Examples of contact dermatitis:

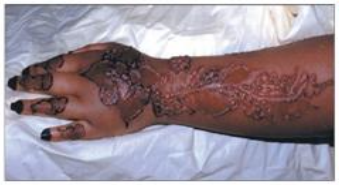
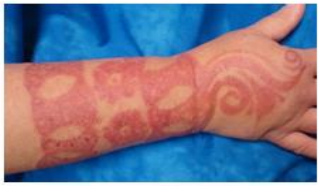
1-Nickel dermatitis

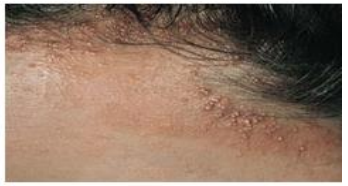





Sub acute

Acute

2-P-phenylenediamine

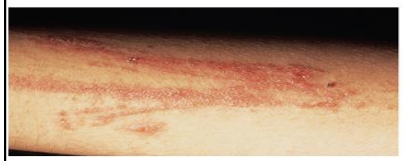





Due to black henna

Hair dye allergy

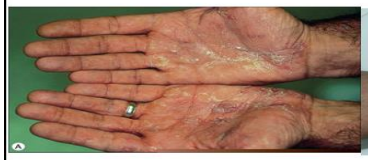
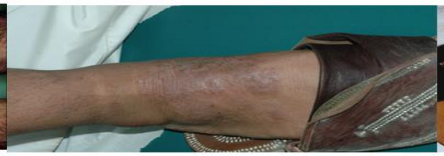
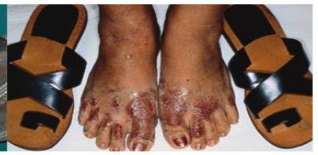
3- Poison Ivy



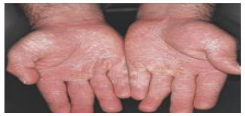
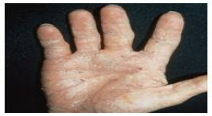

Acute due to erythema and papule

Acute due to vesiculation

4-Potassium Dichromate in Leather

5-Latex, Cleaning products, Cosmetics, Occupational exposures

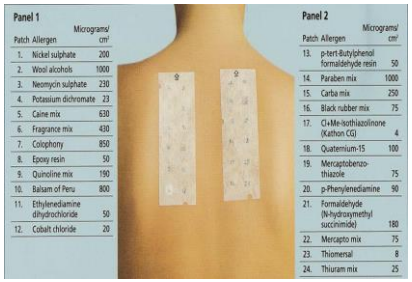
Check the feet and nails

Diagnosis:

-Hx.

-Examination.

-**PATCH testing** remain the **gold standard** for accurate diagnosis.



Patch test

Treatment of ACD:

-Avoidance.

-topical steroid

-systemic steroid

-Oral antihistamine

We have to stop any medication interfere with patch result before one week of the test e.g. (antihistamine, steroid)

2-Irritant contact dermatitis (ICD)

-Is localized non immunologically mediated inflammatory reaction.

- ICD results from direct cytotoxic effect d.t single or repeated application of a chemical substance to the skin.

Most common irritants are:

-Water

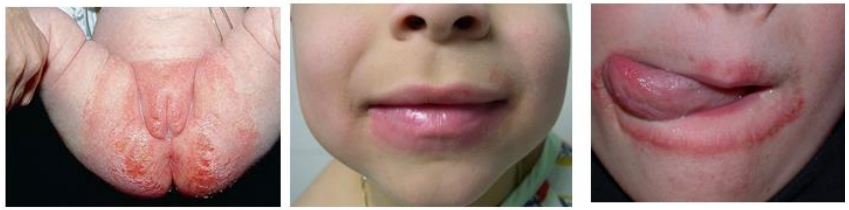
-Abrasives

-Chemicals, e.g. acids and alkalis

-Solvents and detergents

- stool(it has enzymes that my act as cytotoxic agents)
- saliva(lead to lip dermatitis)

Examples of irritant contact dermatitis:



Due to stool

Lip licker dermatitis
(Blunting of vermillion,Accentuation of angles)

Clinical picture:

-Similar to ACD but ICD **never** extend beyond the area of contact.

-tend to be painful rather than pruritic .

-can occur from the 1st exposure to the irritant unlike ACD which only occur in previously sensitized individual.

IRRITANTS AND MECHANISMS OF TOXICITY	
Irritant	**Mechanisms of toxicity**
Detergents	Solubilization and/or disruption of barrier lipids and natural moisturizing factors in the stratum corneum Protein denaturation Membrane toxicity
Acids	Protein denaturation Cytotoxicity
Alkalis	Barrier lipid denaturation Cytotoxicity through cellular swelling
Oils	Disorganization of barrier lipids
Organic solvents	Solubilization of membrane lipids Membrane toxicity
Oxidants	Cytotoxicity
Reducing agents	Keratolysis
Water	If barrier is disrupted, cytotoxicity through swelling of viable epidermal cells

Treatment:

Same as ACD

Nummular (discoid) dermatitis:

-Sharply circumscribed eczema , nummular means (coin -shaped)

-Pathogenesis:

Probably **microbial in origin** i.e. 2ry to bacterial colonization or dissemination of bacterial toxins.

Clinical picture:

-Coin shaped eczematous plaques .

-Typically affects limbs of middle-aged or elderly

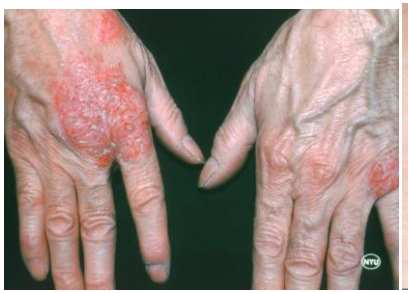
-Usually very pruritic.

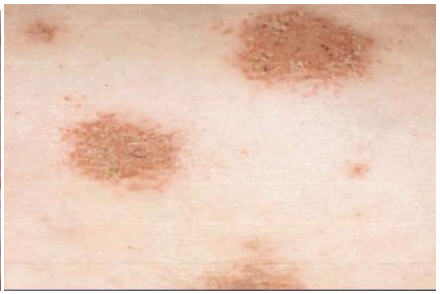
Treatment:

-Topical steroid

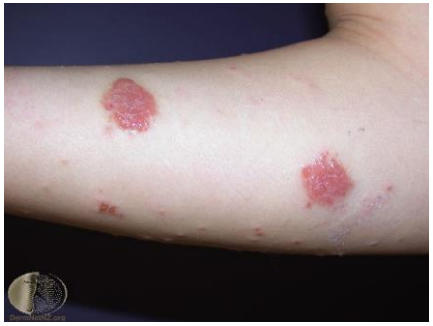
-Topical antibiotic

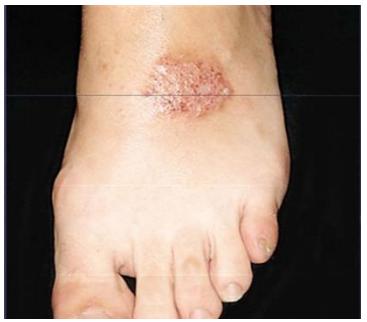
Oral antibiotic-





45 y/o female with intermittent “fungus all over”





Dyshidrotic dermatitis (pompholyx)

Acute dermatitis which is often vesicular with tiny deep seated vesicles along the sides of the fingers associated with pruritus

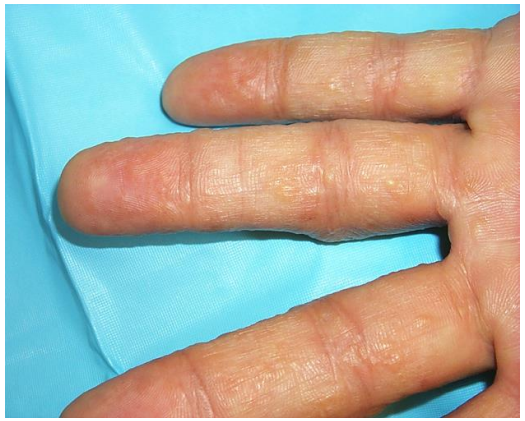
-Not considered as a separate disease

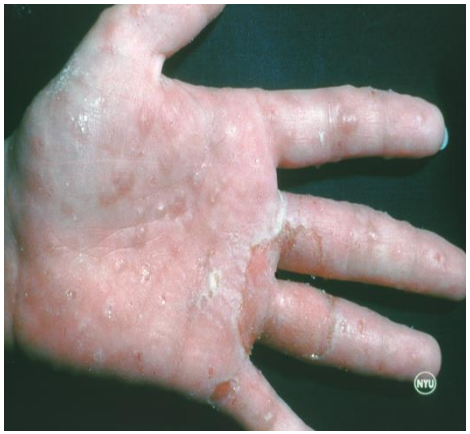
-Can be associated with atopy , of patients with dyshidrosis, 50% have atopic dermatitis.

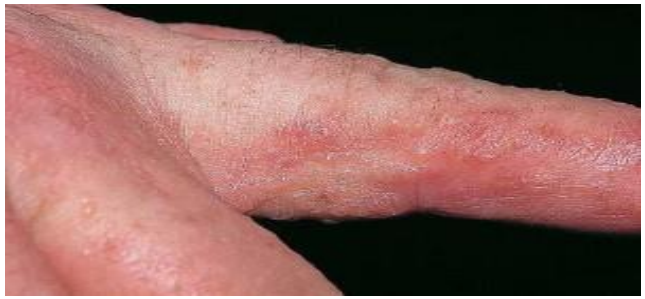
-Exogenous factors (e.g., contact dermatitis to nickel, chemicals) also play a role.
-Affect hands & feet.
-It could be a separate disease or part of atopy.
- Common in ladies who clean with water and detergents without protection.

Treatment:

-Avoidance of triggering factor.
-topical steroid.







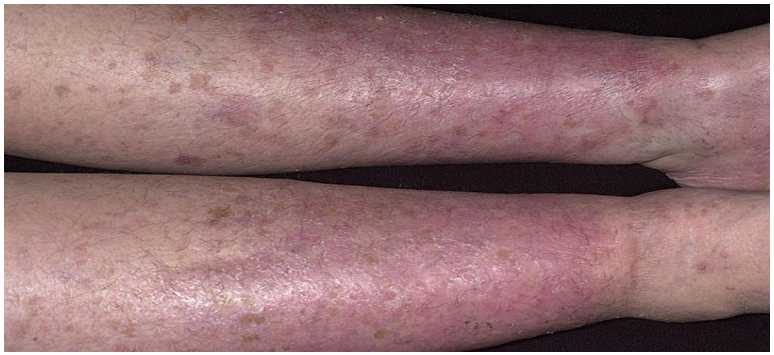
Deep seated vesicles along the sides of the finger
DDx (tinea and fungal infection which affect one hand, herpes simplex infection and dyshidrotic which affect both hands)

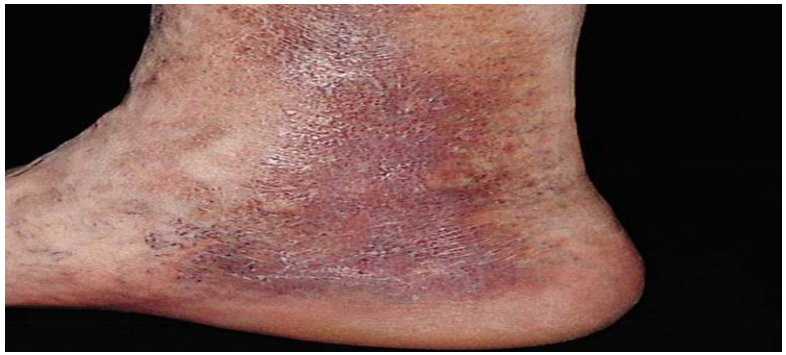
Stasis dermatitis

-seen in patient with signs of venous hypertension like chronic lower limb edema, varicose vein. We have to roll out vascular disease

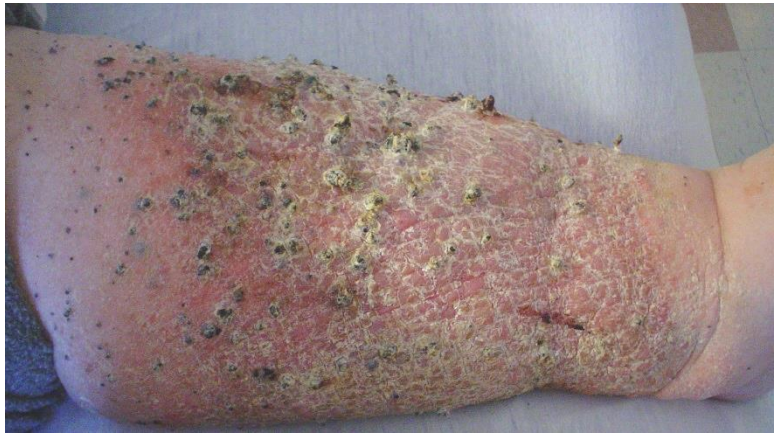
-can be complicated by superimposed allergic contact dermatitis.

- Common presentation is a bedridden patient who have venous ulcers, after treating the ulcer he may end up with allergic contact dermatitis.





Stasis eczema



Very sever type of stasis dermatitis
(Elephantiasis Verrucosa Nostras)

Treatment:
-Topical steroids
- Treat the underlying condition(venous insufficiency) (most accurate way)

Xerotic dermatitis (Asteatotic Dermatitis)

-Diffuse background skin dryness with associated dermatitis
-typically affects limbs of the elderly.

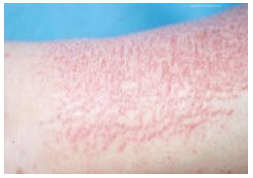
Aggravated by:

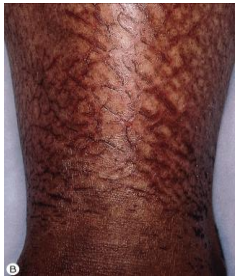
harsh cleansers, dry winter conditions, hypothyroidism, use of diuretics

-**Treat with**

emollients 1st

+/- mild steroid ointments

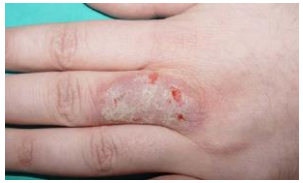




Extreme case of xerosis Riverbed type cracking

Neurodermatitis

14 y/o anxious female who can't stop itching



-Include dermatitis which results from repeated rubbing & scratching of the skin .

-Chronic itching and scratching can cause the skin to thicken and have a leather texture with exaggeration of skin marking.

-A scratch-itch cycle occurs which is difficult to break.

-Can be triggered by stress and anxiety. **Psychiatric disease (areas pts cant reach always symptomatic free) e.g. back**

-Occur commonly in atopic patient.

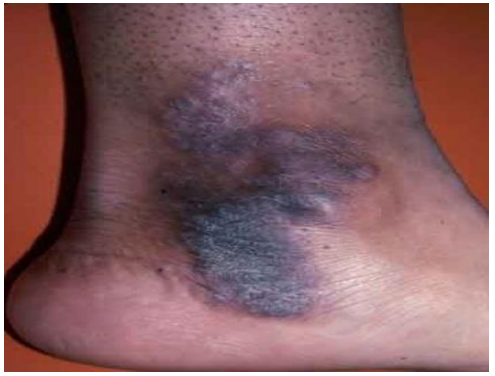
lichen simplex chronicus (example of neurodermatitis):

Present as thick hyperkeratotic plaque with accentuation of skin marking that occurs on any site that the patient can reach, including the following:

-Scalp
-Nape of neck
-Extensor forearms and elbows
-Vulva and scrotum
-Upper medial thighs, knees, lower legs, and ankles

Treatment:

-control itching (break itch scratch cycle).
-topical or intralesional steroid.
-oral antihistamine
- Oral Anxiolytic



Common pit fall:

Misdiagnosis

- Scabies (intensely pruritic, burrows/vesicles, others itch)
- Psoriasis (elbows/knees/inflammatory arthritis/nail changes
- Fungus (central sparing, well marginated, scaly border)

Summary:

Distinctive morphological features of different forms of dermatitis

Type	Features of dermatitis	Other skin findings
Atopic	Symmetry, changes with age	Xerosis
Seborrheic	Greasy scale, face and scalp affected	Oiliness
Nummular	Coin-shaped or discoid macules and patches	Xerosis
Stasis	Affects lower legs, ankles	Edema,
Xerotic	Mild, widespread; typically fall & winter	Xerosis, hyper-pigmentation
Allergic contact	sites of contact, may have geometric patterns	
Irritant contact	typically affects hands, face	Xerosis, fissuring.

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