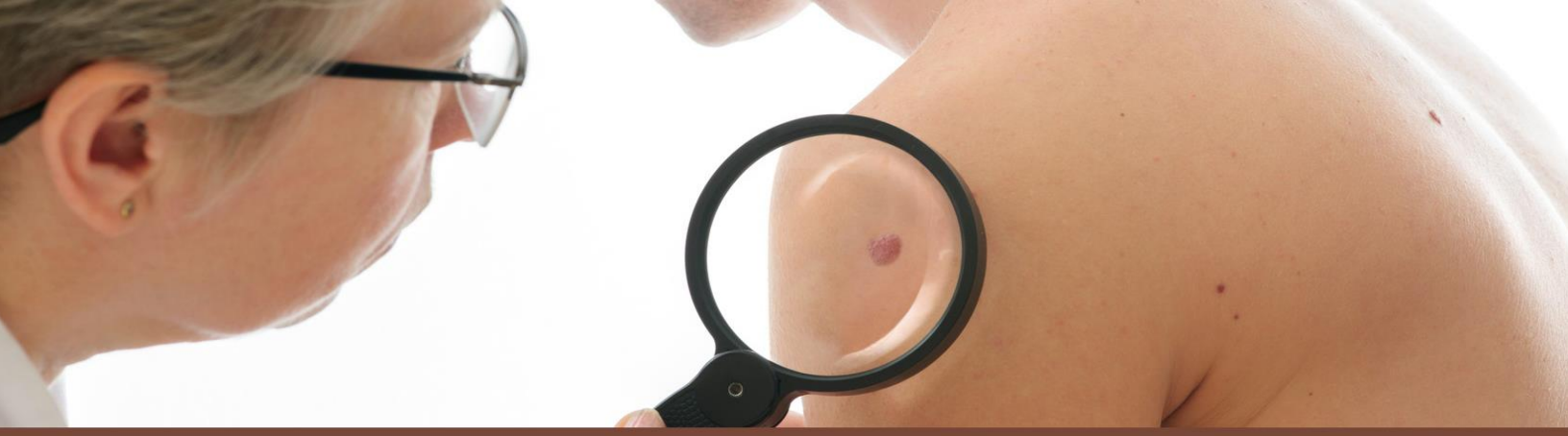

433 Teams

DERMATOLOGY

Lecture (3)

General principles of eczema /dermatitis (Atopic dermatitis and others)

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جامعة الملك سعود
King Saud University

مظلل بالأصفر كلام ذكرته الدكتوره بالمحاضره –
- الكلام اللي باللون الرمادي من الانترنت .

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
- **Color index: slides, doctor notes,**

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
Examples: SLE, serum sickness , vasculitis

Type 4: Delayed-Type Hypersensitivity:
Mediated by T-Lymphocytes to specific antigens
Reaction within 2-7 days after exposure (do you expect the patient will develop this reaction in the first exposure? No it should be after exposure.)
Examples: Allergic contact dermatitis (e.g. Nickel allergy)

Eczema (Dermatitis):

Definition:

is an inflammatory skin disease.

Skin inflammation characterized by:

-itchy, scaly, patches of erythema

Pathogenesis:

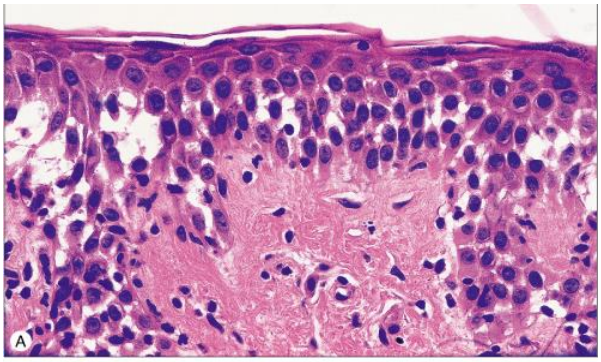
It is an epidermal reaction to specific Antigens; these antigens may be internal or external, acting singularly or in combination.

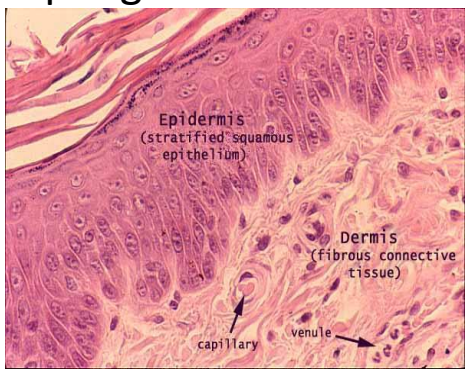
Clinical picture:

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.

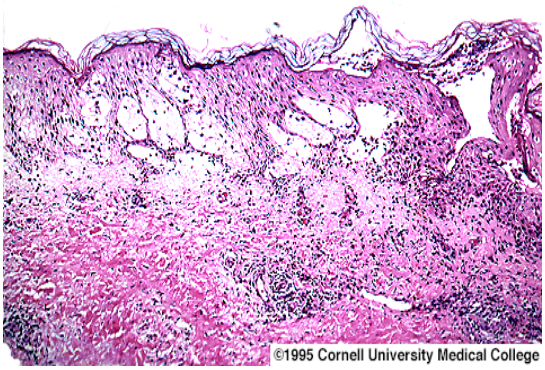
Acute	Subacute	Chronic
-eryhema - papules (elevation less than 0.5 cm)& vesicles(fluid filled elevation less than 0.5 cm) - oozing	more dry lesion - scales - Excoriation = linear extraction or linear erosions	lichenificaion = execution of skin margins (skin marking) & hyperkeratosis Clinically an eczematous disease may start at any stage and evolve into another

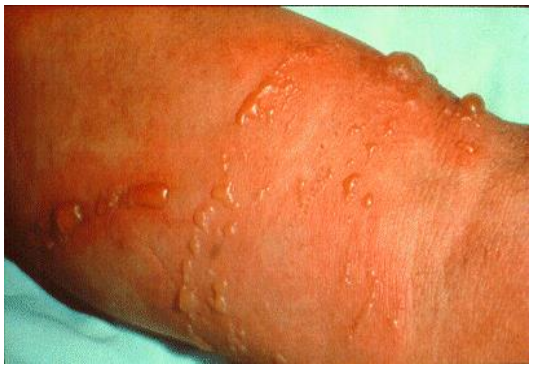
Dermatitis: present histologically with
Spongiosis =intercellular edema
Dermatitis="Eczema"=Spongiosis



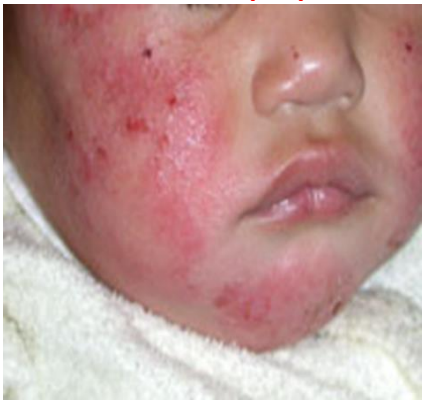


Normal epidermis ,normal keratocyte and increasee the space between the keratocyte due to edema .



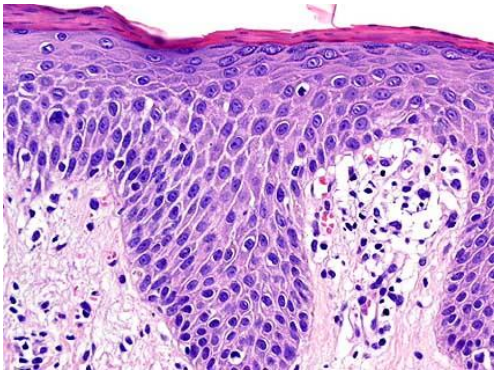
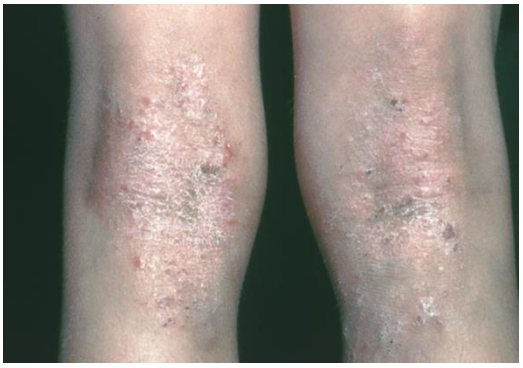


left picture = acute dermatitis with infiltration of lymphocytes.
right picture = acute dermatitis clinically with erythema ¸ vesicles and papules.



if you see more erythema and edema, papules, vesicles, and,sometimes bullae accompanied by exudation and crusting = **Acute**

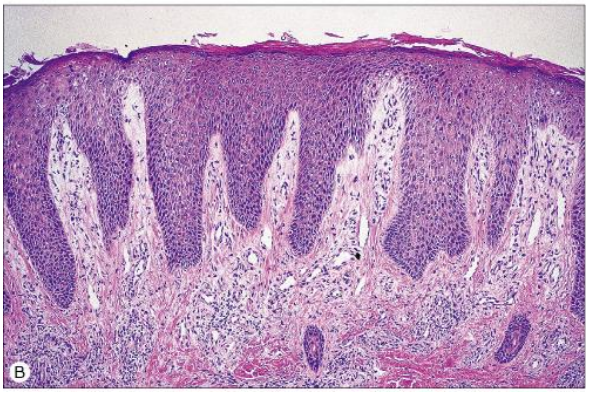
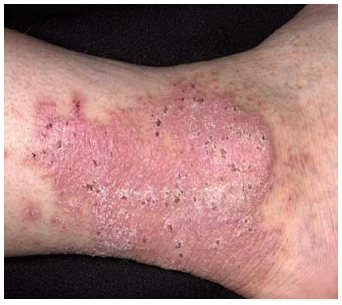
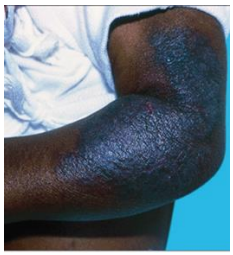
Subacute Dermatitis:

left picture : less intercellular edema more hyperkeratosis

right picture : scaling and less erythema

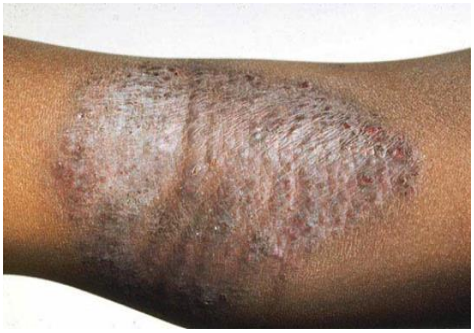
Chronic Dermatitis:

left picture : thickened epidermis and hyperkeratosis .

here is significant epidermal acanthosis, which may show a psoriasiform pattern with hyperkeratosis

right picture: lichenificaion skin marking .



In chronic , less erythema and edema presence of lichenification, scaling, and fissuring .

1- Atopic dermatitis:

chronic relapsing itchy skin disease in genetically predisposed patients associated with personal or family history of 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 or at least improve .

Pathogenesis:

1-Genetic predisposition.2-immune mediated (increase IgE), T-helper cell2 activation (type IV reaction) .

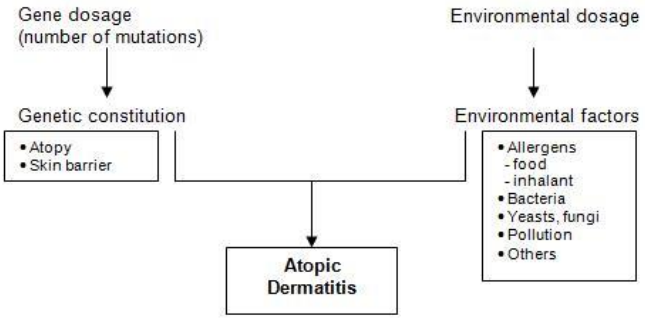
3-Impaired skin barrier ,defective epidermal differentiation (filaggrin mutations) patient with atopic dermatitis have mutation which result in impaired barrier function of the skin. Terminal differentiation of keratinocytes within the epidermis results in the formation of a densely packed and extensively crosslinked lipid-protein matrix, which forms an impenetrable barrier (known as the stratum corneum) that is the uppermost layer of the epidermis. As keratinization takes place within the keratinocyte, filaggrin-aggregated tonofibrils collapse the cytoskeleton, forming flattened squamous cells. Keratinization also destroys the cell nuclei in a mediated (timely) cell death. Further heavy cross-linking of this protein and lipid , protective epidermal barrier.These "dead" cells migrate into the upper layers of the epidermis, where they are sloughed off in the normal course of one's day, while being continually replaced with the squamous cells migrating from below. Deficient or missing filaggrin proteins compromise the epidermal barrier.

4- Allergy, increased tendency to certain allergens

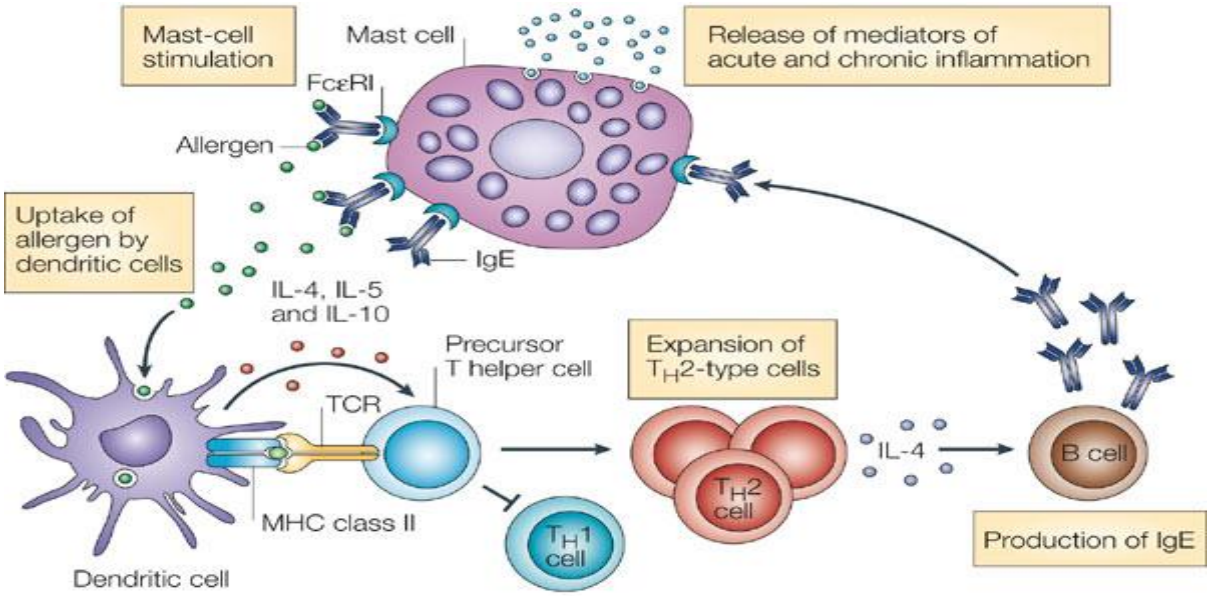
5- Infection : skin of pts with AD is colonized by S aureus. infection with S aureus often causes a flare of AD.-

6- AD and Food! minor role . The major roles is genetic and 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.



No single etiology
=
No "magic bullet" cure



Allergens are taken up by dendritic cells and presented to T cells. In the absence of childhood microbial exposure, the balance between T helper 1 (TH1) and TH2 cells is altered. TH2 cells encourage the production of immunoglobulin E (IgE) by B cells. Allergen-specific IgE then binds to the high-affinity receptor for IgE (FcepsilonRI) on mast cells. 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.

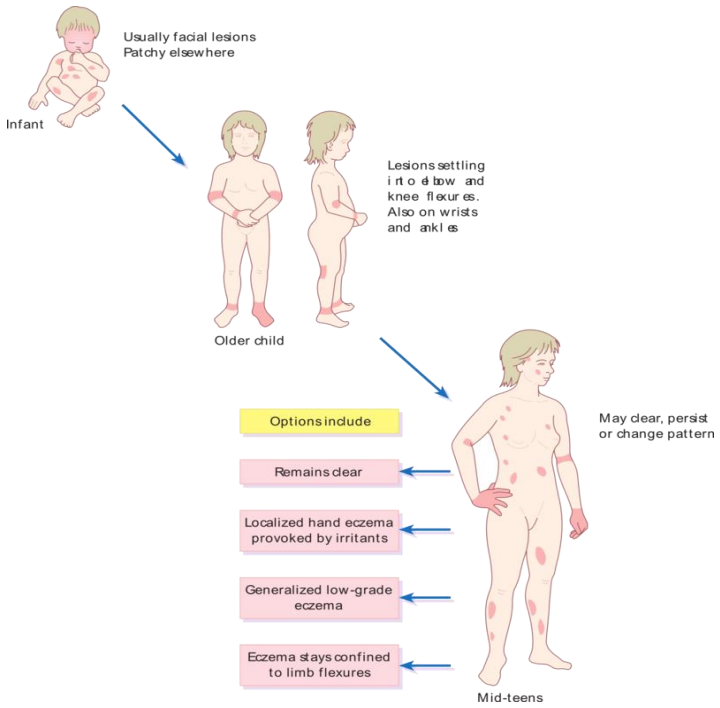
Three stages:1- Infantile . 2-Childhood .3-Adulthood.

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

Distribution :

Childhood and adult = flexures

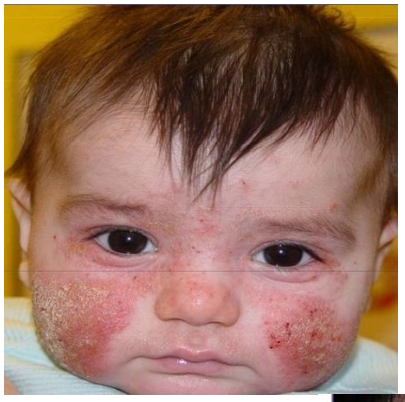
Infintile = extensors and face .

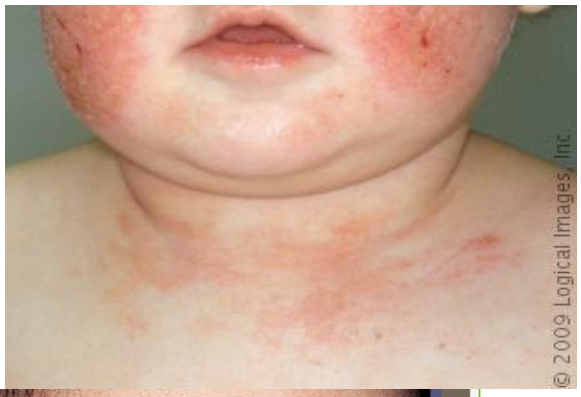


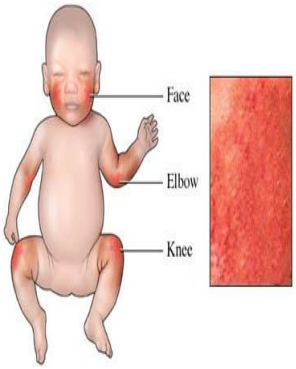
Pattern of atopic eczema varies with age.

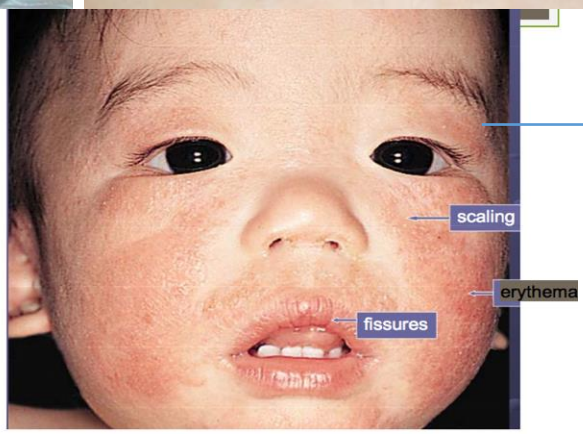
A- Infantile atopic dermatitis :

Infants develop an itchy vesicular eczema on cheeks and hands often with secondary infection.



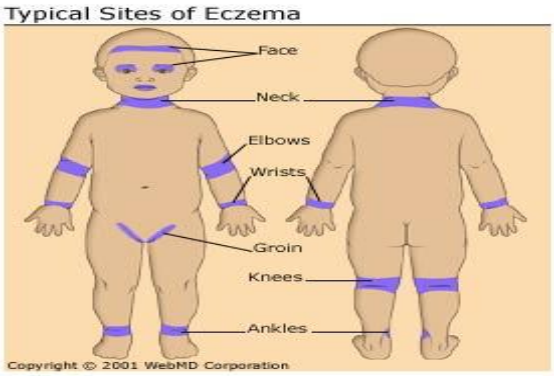


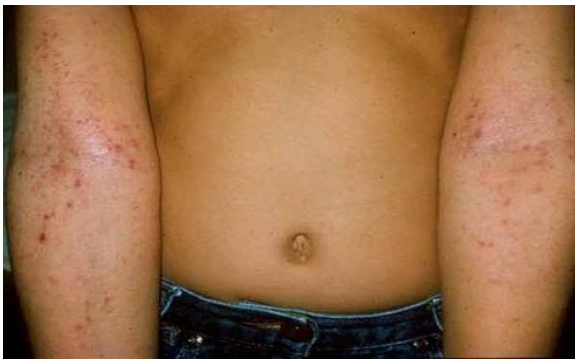




his face has chucinoid face due to chusing from disusing of steroid .

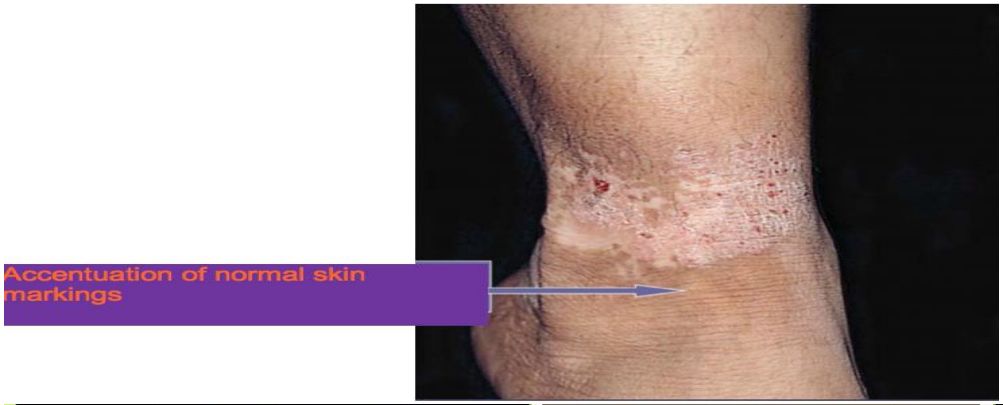
B- Childhood

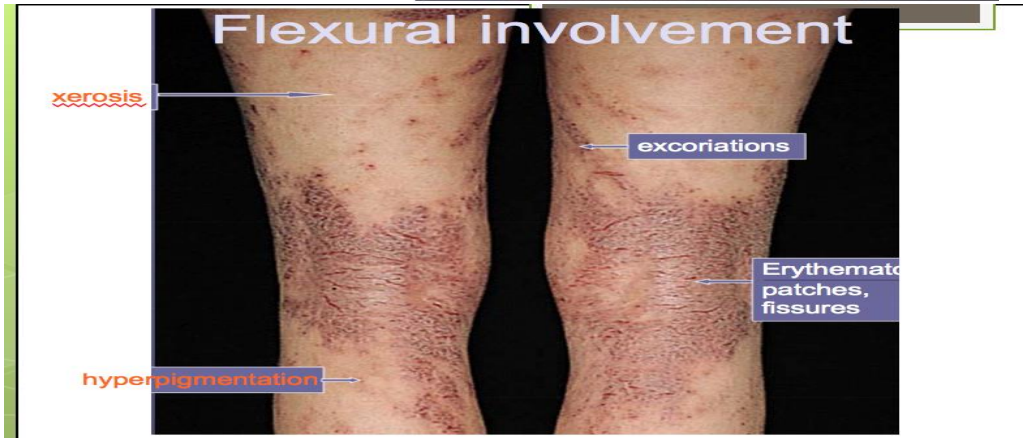




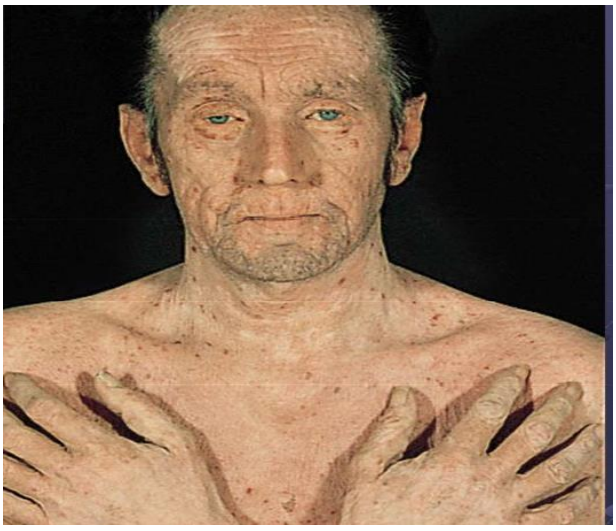
Children develop lesions at antecubital and popliteal fossae, neck, wrists, and ankles. Lichenification, excoriations, and dry skin are common as well as post-inflammatory hyperpigmentation

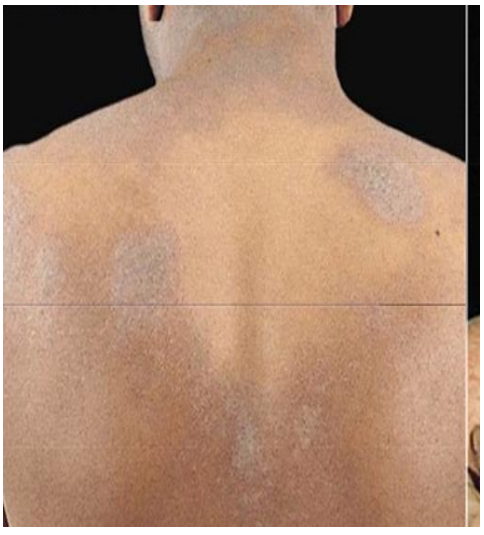
C-Adult atopic dermatit





Widespread chronic atopic dermatitis





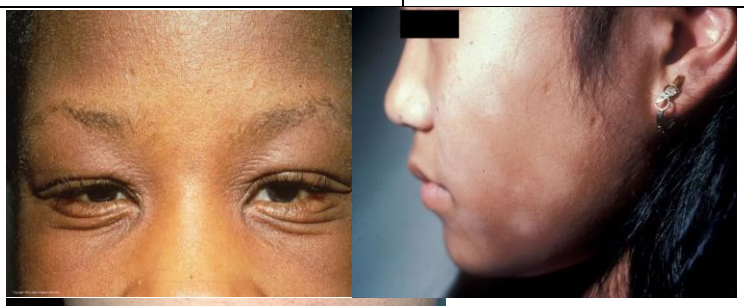
dignosis criteria : requires that patients have at least 3 of the 4 major **criteria** and 3 of the minor **criteria**.

A- Major:

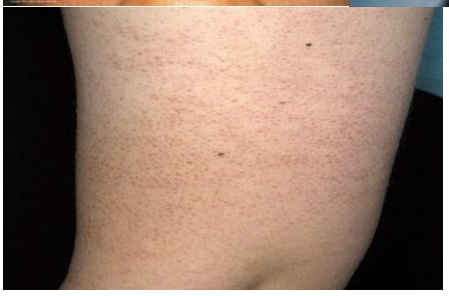
1.pruritus
2.typical morhology and distribution
3.chronicity
4.Personal or family history of atopy

B-Minor :

1. -Xerosis زي جلد السمكه
2. -Icthyosis/hyperlinear palms/keratosis pilaris.
3. -IgE reactivity
4. -Elevated IgE level
5. -Early onset
6. -Skin infection
7. -Chelitis
8. -Nipple eczema
9. -Recurrent conjuctivitis
10. -Keratoconus
11. -Dennie morgan fold
12. -Anterior cataract
13. --Orbital darkening
14. --Facial erythema
15. --Pityriasis alba
16. --Food hypersensitivity
17. --White dermatographism



right =Dennie morgan fold
left= Pityriasis alba



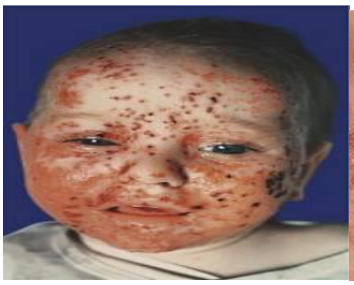
Keratosis pilaris

Complications:

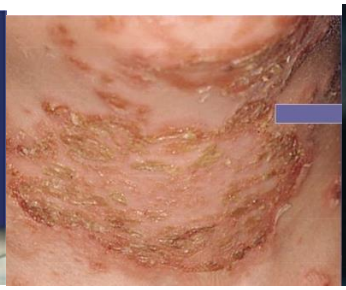
- Infection:
  - Bacterial: Impetigo(STAPH AURIOUS)
  - Viral infection: eczema herpeticum(Herpes Simplex Virus)

widespread molluscum

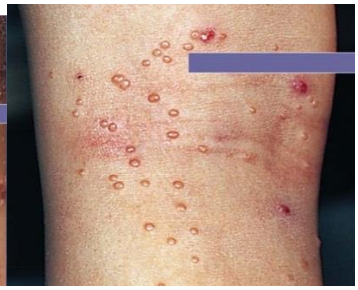
- Cataracts
- Growth retardation.



eczema herpeticum



Impetiginised dermatitis
Honey crust



Mollusca Contagiosa

MANAGEMENT

Dry Skin:   • Bathing, soap • Emollients/barrier repair	Inflammation: • Topical steroids • Topical calcineurin inhibitors
Pruritus/Sleep: • Sedating antihistamines	Infection: • Contributing factors
Education : • Avoid alkali soaps • Avoid woolen clothes and wear cotton instead.	

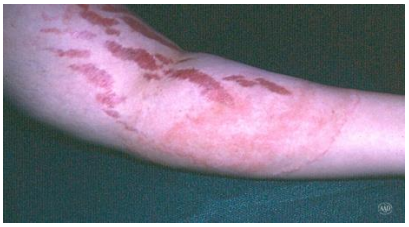
Treatment:

-topical steroid
-topical immunomodulators (tacrolimus & pimecrolimus)
-oral antihistamine
-oral Antibiotic (for 2ry bacterial infection)
-ultraviolet light
-systemic steroid
-others: cyclosporin , methotrexate ,azathioprine, IVIG , Biologic

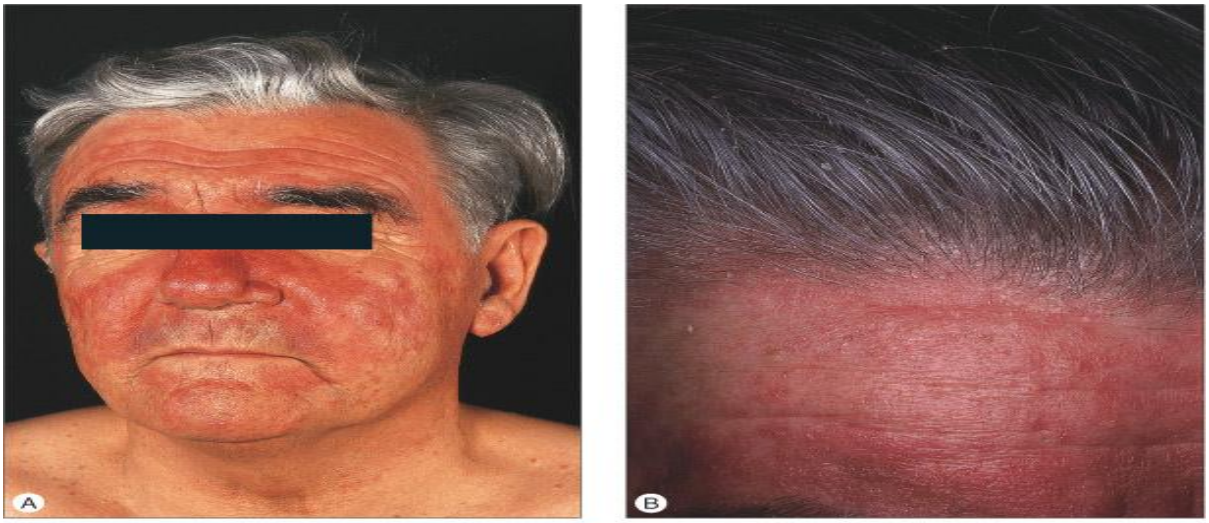
What are the side effects of topical steroid? It is rare

- Reversible:
  - Telangiectasia/prominent blood vessels
  - Epidermal Atrophy/thin skin
  - Acne/rosacea
  - Increased hair growth
- Non-reversible: dermal atrophy

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



stria



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

student saids in the lecture SLE as DDX but the libonasal fold is involves while in SLE buterfly libonasal spared.

so it is **2-Seborrheic Dermaitisl:**

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)

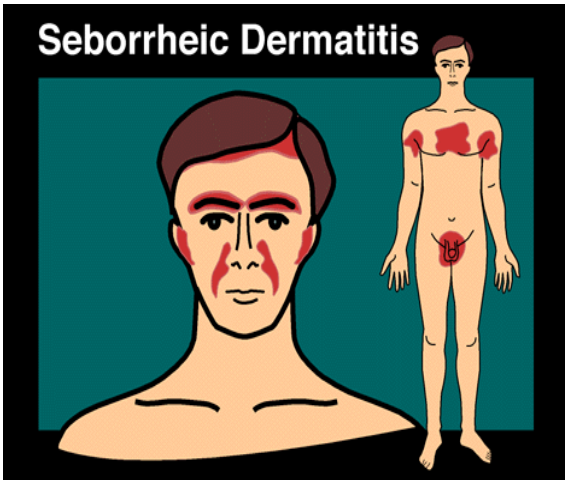
Clinical Picture:

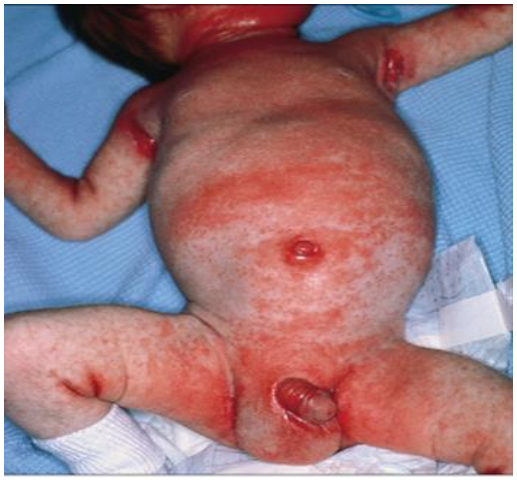
Seborrheic dermatitis is defined by clinical parameters which include:

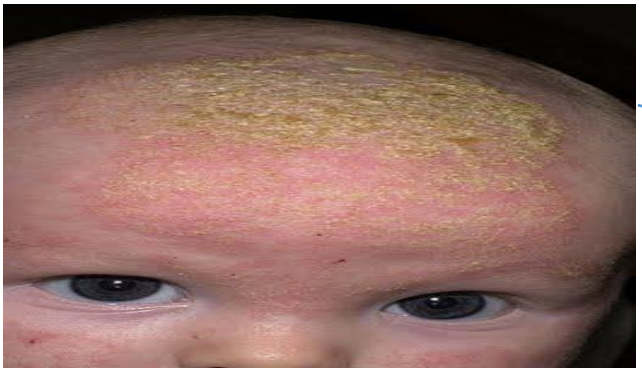
1-erythematous red-yellow , poorly circumscribed patches & thin plaques with bran-like to flaky (greasy) scales.

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

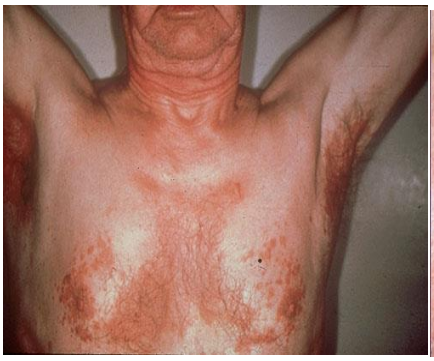
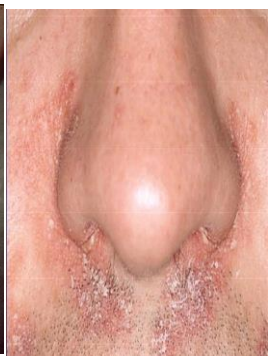
3- A predilection for areas rich in sebaceous glands
e.g: scalp , face, ears , presternal region & flexural areas (axillae, inguinal & inframammary folds , umbilicus).
4-A mild course with moderate discomfort.







Cradle cap: is coherent scaly & crusty mass covering most of the scalp & can be seen in infanile seborreic dermatitis

Treatment:

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

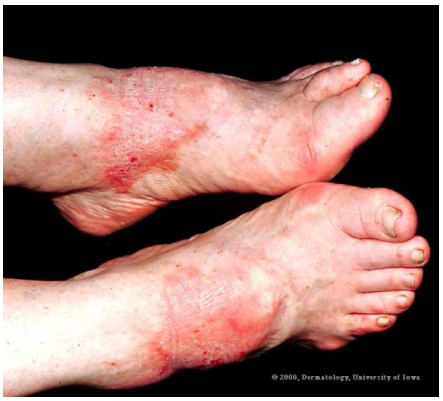
-Topical antifungal.

-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.

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



24 /o male 2 year h/o red, scaly feet

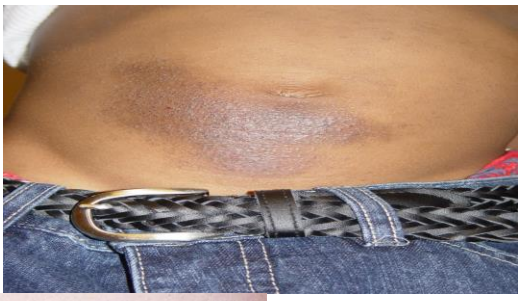
3-Contact Dermatitis

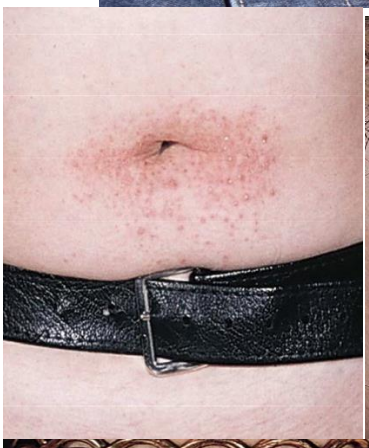
Dermatitis precipitated by an exogenous agent:

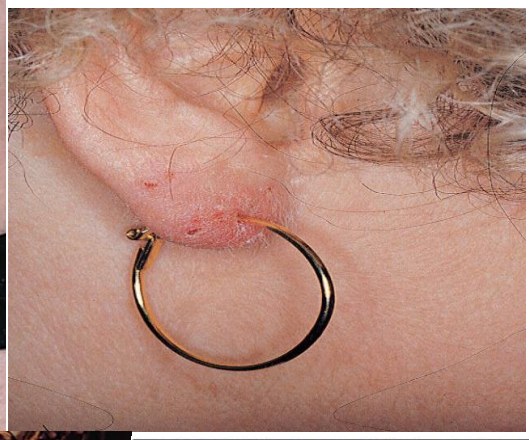
1. Allergic contact dermatitis.
2. Irritant contact dermatitis.

Definition:

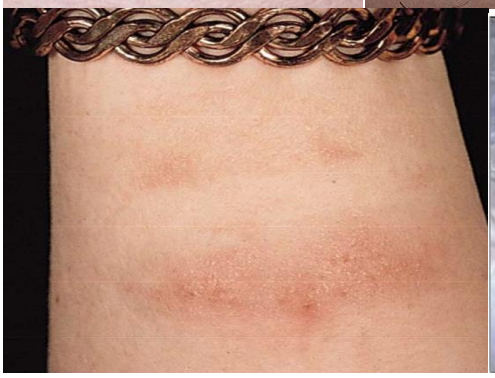
Dermatitis resulting from type 4 reaction following exposure to topical substances in sensitized individuals (requires induction and elicitation phase (lag time to reaction).Common allergens eliciting contact dermatitis: nickel (affects 10% of women and 1% of men), why ? because it happens in accssecories
perfumes, fragrances , preservatives. hair dyes, rubber latex , Acute form present with crusted erythematous papules, vesicles & bullae that is well demarcated & localized to the site of contact with the allergen.
-Acute Contact Dermatits can be more diffuse in distribution.

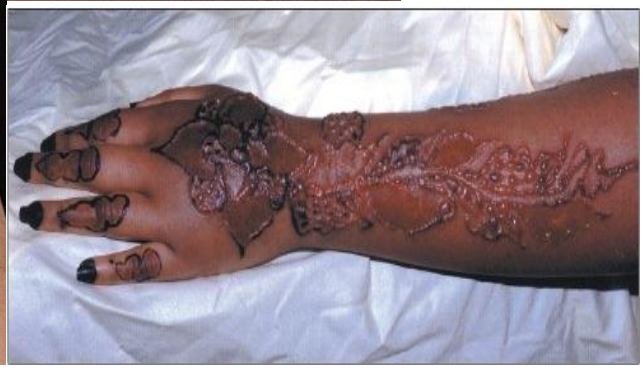






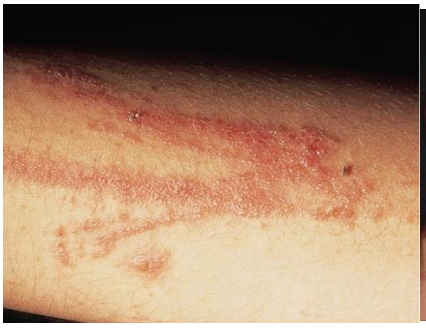
right =Nickel Allergy - belt buckle
left=ACD

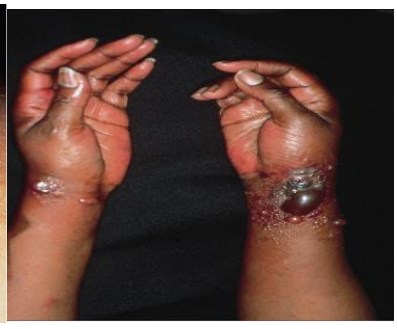




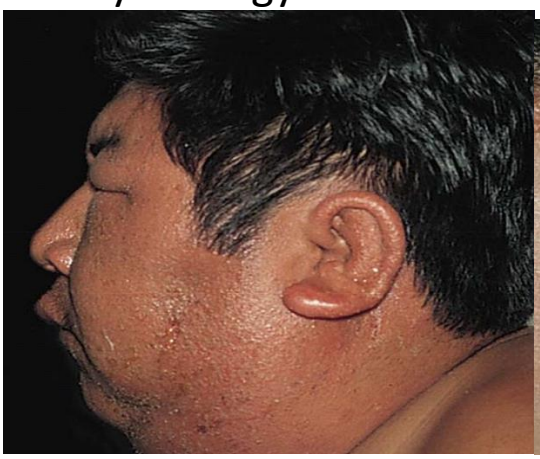
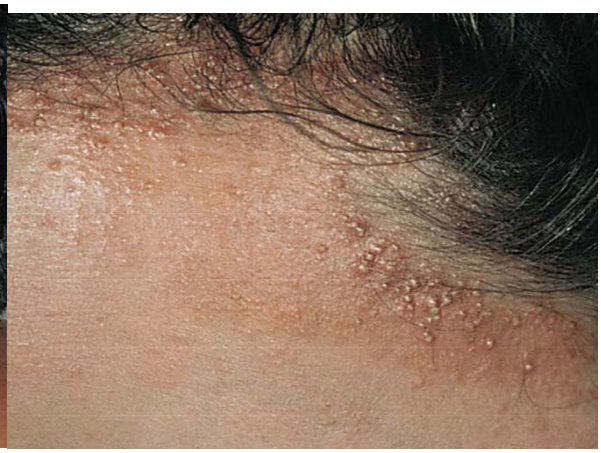
right=Nickel Allergy - bracelet
left=Suspect if dermatitis shows geometric patterns

A-Allergic Contact Dermatitis; linearity . Poison Ivy



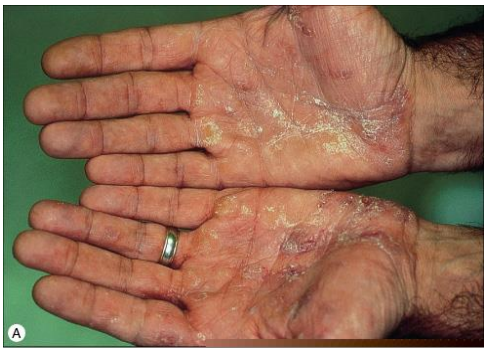


Hair dye allergy

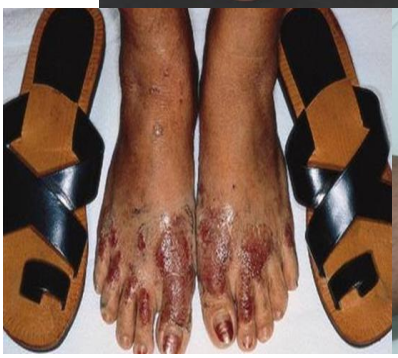
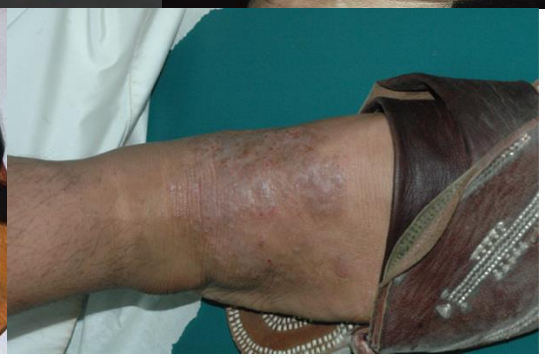
Allergic Contact Dermatitis:

- Potassium Dichromate in Leather



Allergic Contact Dermatitis:-Latex -Cleaning products – Cosmetics- Occupational

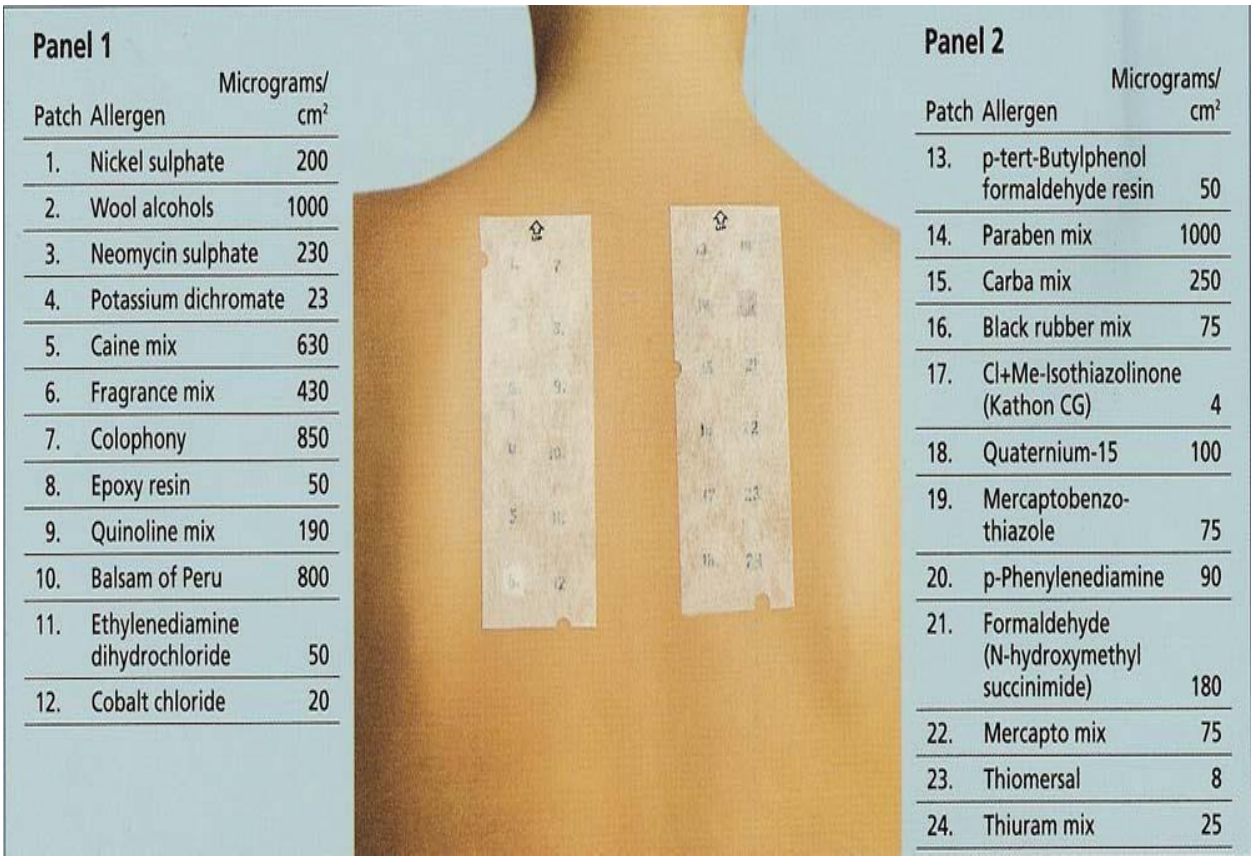


Diagnosis:

1-Hx . 2-Examination.3-PATCH testing remain the gold standard for accurate diagnosis.

Patch test : DOCTOR ASKED ABOUT WHAT IS THE POSITIVE RESULT



Panel 1

Patch	Allergen	Micrograms/ cm^2
1.	Nickel sulphate	200
2.	Wool alcohols	1000
3.	Neomycin sulphate	230
4.	Potassium dichromate	23
5.	Caine mix	630
6.	Fragrance mix	430
7.	Colophony	850
8.	Epoxy resin	50
9.	Quinoline mix	190
10.	Balsam of Peru	800
11.	Ethylenediamine dihydrochloride	50
12.	Cobalt chloride	20

Panel 2

Patch	Allergen	Micrograms/ cm^2
13.	p-tert-Butylphenol formaldehyde resin	50
14.	Paraben mix	1000
15.	Carba mix	250
16.	Black rubber mix	75
17.	Cl+Me-Isothiazolinone (Kathon CG)	4
18.	Quaternium-15	100
19.	Mercaptobenzo-thiazole	75
20.	p-Phenylenediamine	90
21.	Formaldehyde (N-hydroxymethyl succinimide)	180
22.	Mercapto mix	75
23.	Thiomersal	8
24.	Thiuram mix	25

Treatment of ACD:-Avoidance.-topical steroid-systemic steroid

-Oral antihistamine

B-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

Clinical picture:

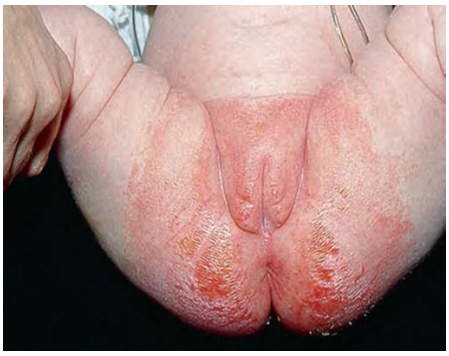
-Similar to ACD but ICD never extend beyond the area of contact.

-tend to be painful rather than pruritic .

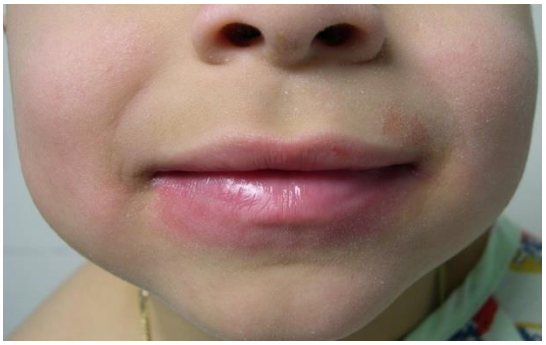
-can occur from the 1st exposure to the irritant unlike ACD which only occur in previously sensitized individual

Irritant Contact Dermatitis:

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



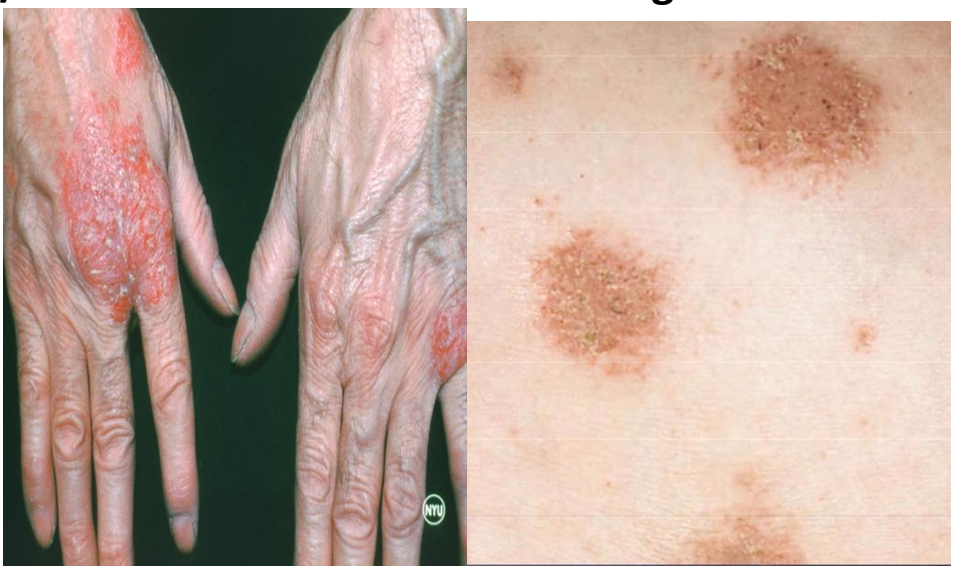
NAPKIN DERMATITIS (course from stool enzyms it will trigger the ittation)
so manage it with frequent diper change and barrier like zinc oxide .



Lip licker dermatitis caused by lip licking ,Saliva contain enzymes as irritant . manage by avoide licking and topical steroid .

Blunting of vermillion
Accentuation of angles
Treatment: Same as ACD.

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



4- Nummular (discoid) dermatitis:

Sharply circumscribed eczema , nummular means (coin - shaped)

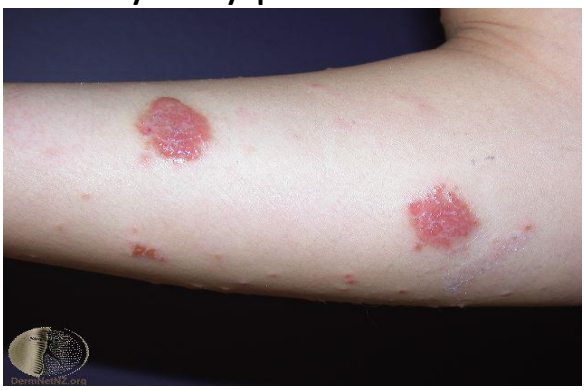
-Pathogenesis: Probably microbial in origin i.e. 2ry to bacterial colonization or disseminaion of bacterial toxins.

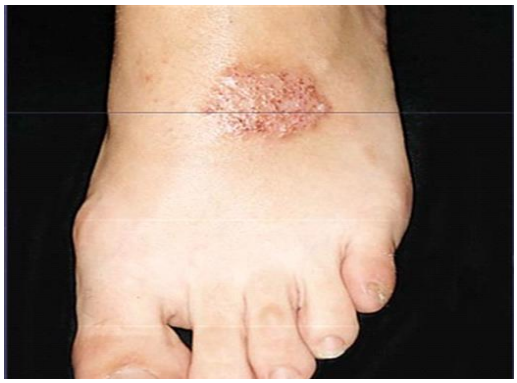
Clinical picture:

-Coin shaped eczematous plaques .

-Typically affects limbs of middle-aged or elderly

-Usually very pruritic





Treatment:

-Topical steroid

-Topical antibiotic

Oral antibiotic-

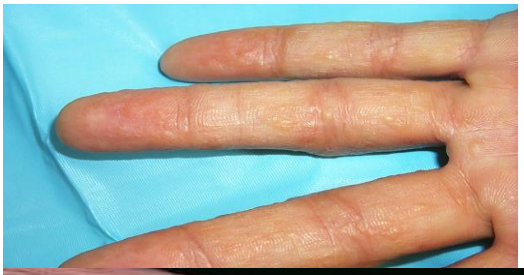
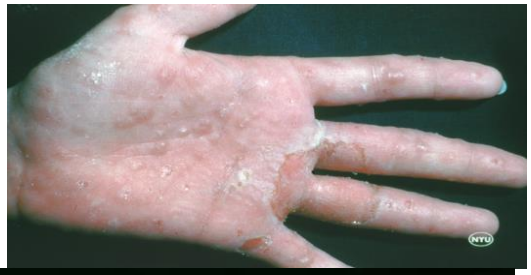
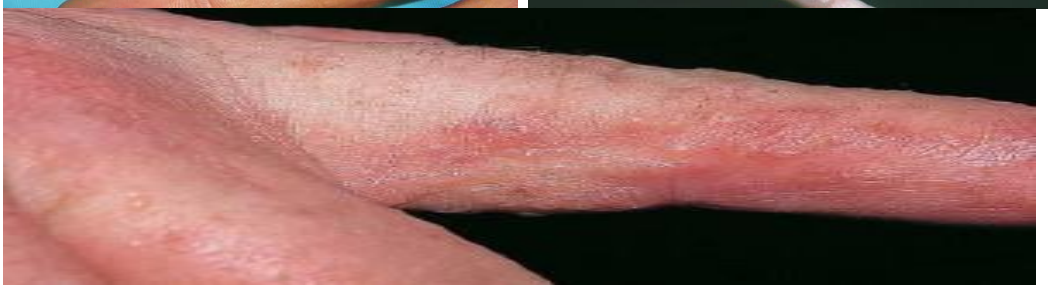
5-Dyshidrotic dermatitis (pompholyx):

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 (eg, contact dermatitis to nickel,chemicals) also play a role.-Affect hands & feet.

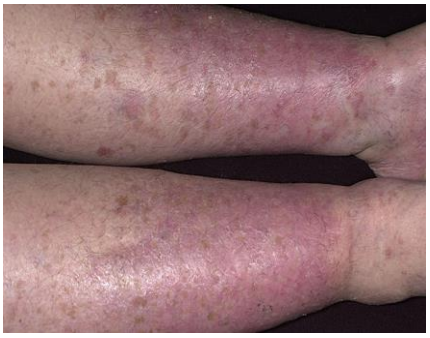
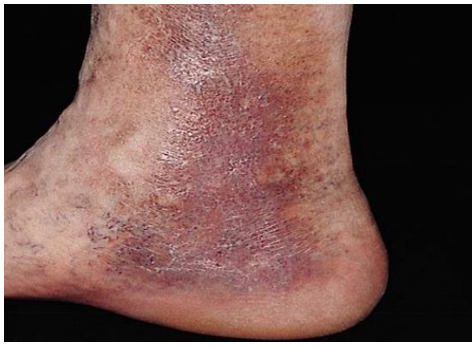




Treatment:- Avoidance of triggering factor. -topical steroid.

6- Stasis dermatitis:

seen in patient with signs of venous hypertension like chronic lower limb edema, varicose vein.

-can be complicated by superimposed allergic contact dermatitis.

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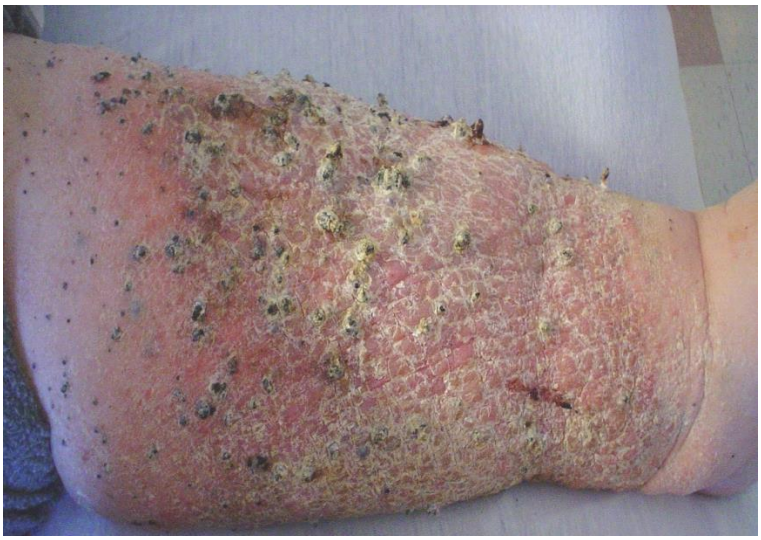
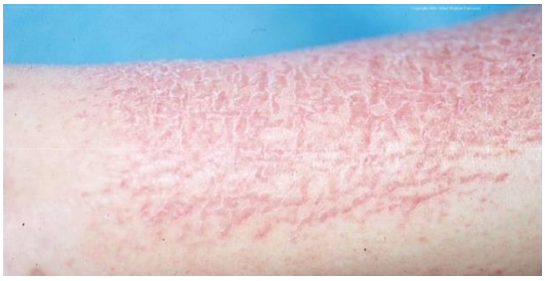


in sever cases develop to Elephantiasis Verrucosa Nostras due to lymph odema dysfunction

7-Xerotic dermatitis :

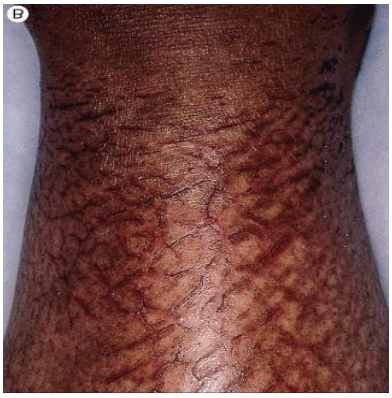


Xerotic dermatitis (Asteatotic Dermatitis)

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

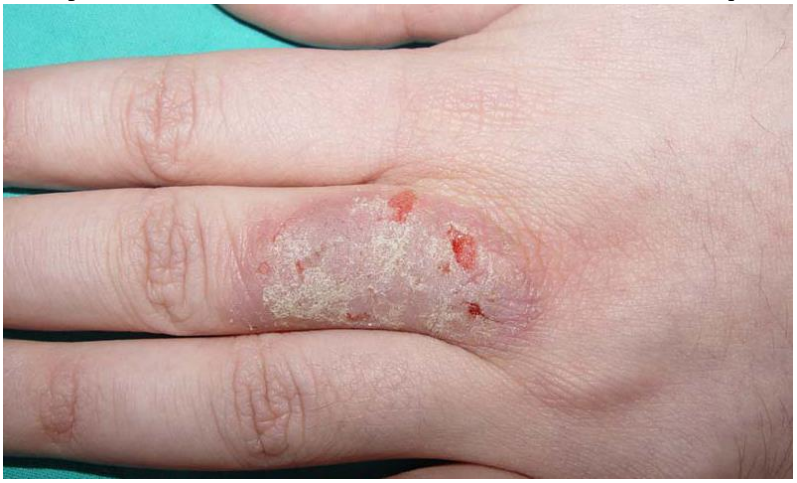
  Aggravated by:

  ✓ harsh cleansers, dry winter conditions, hypothyroidism, use of diuretics
- Treat with emollients 1st +/- mild steroid ointments .



- Extreme case of xerosis.
- Riverbed type cracking

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



in the picture we can see feutures of subaacute eczema

8- Neurodermatitis:

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. = lichenificaion

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

Can be triggered by **stress and anxiety.**

-Occur commonly in atopic patient.

Example is **lichen simplex chronicus:**

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

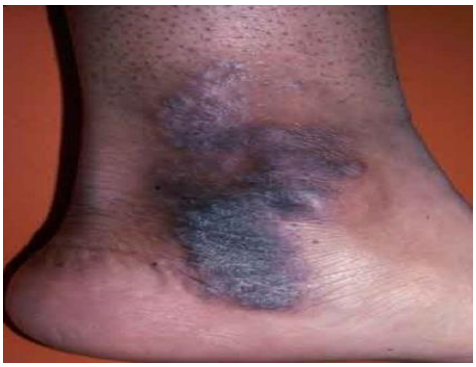
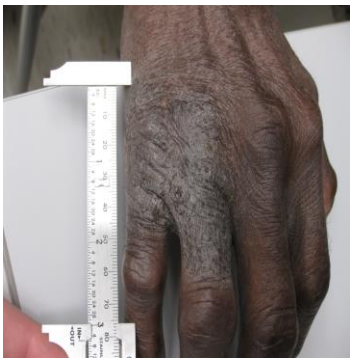
-Nape of neck

-Extensor forearms and elbows

-Vulva and scrotum

-Upper medial thighs, knees, lower legs, and ankles

lichen simplex chronicus

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

Common Pitfalls

- Misdiagnosis
  - Scabies is DDX of dermatits(intensely pruritic, burrows/vesicles, others itch), scabes is caused by mites .
  - PsoriasisDDX of dermitis becauseit hacs skaleing (elbows/knees/inflammatory arthritis/nail changes)
  - Fungus (central sparing, well marginated, scaly border)

Distinctive morphological features of different forms of dermatitis

type	Features of dermatitis	Other skin findin
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

Summary :

- Describe the cutaneous features of dermatitis.

-Differentiate acute from chronic dermatitis

- Contrast irritant versus allergic contact dermatitis
- Describe the presentation of atopic dermatitis at different ages
- Indicate cutaneous findings that are unique for each type of dermatitis

Done By: Amjad Albatli