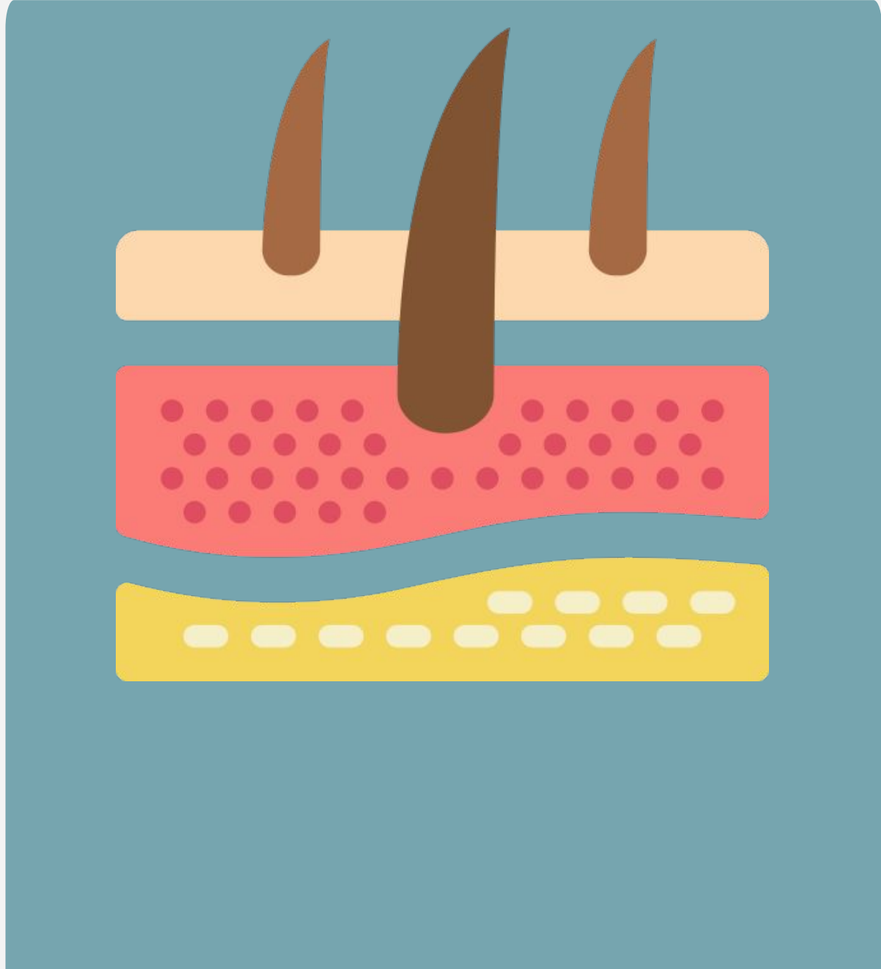


4-General Principles of Eczema/ Dermatitis

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

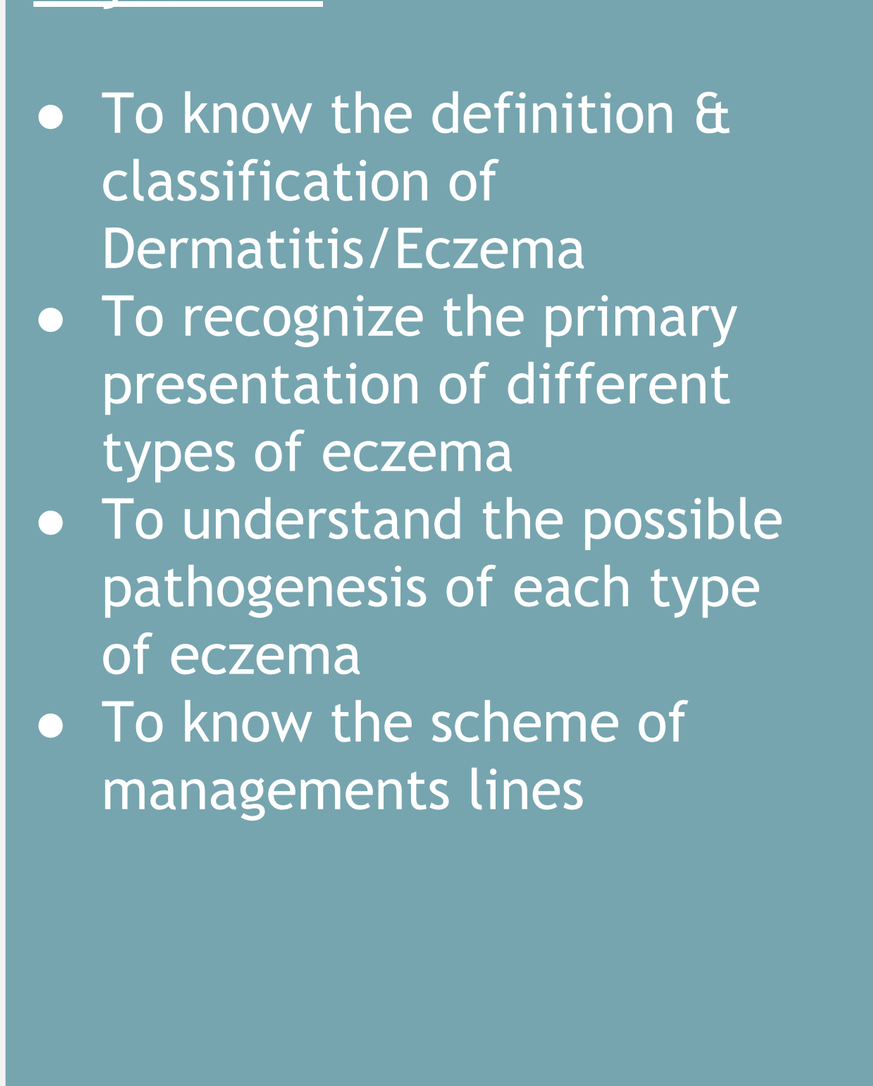
Done by:
Yazeed AlKhayyal, Homoud al zaid, Rakan Al Salhi
AlHanouf AlJaloud, Noura AlBassam, Reem AlQarni.
Revised by: Sultan Al Nasser, Rotana Khateeb

References: Doctor slides, Team 436

Color Index:

- Important
- Doctor's Notes
- Extra

Editing File

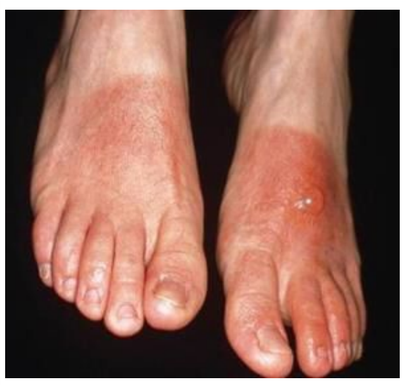
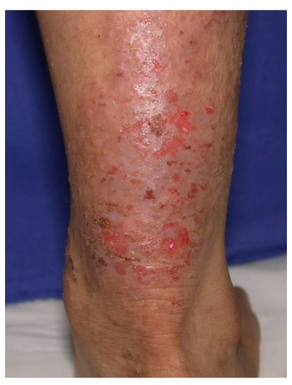
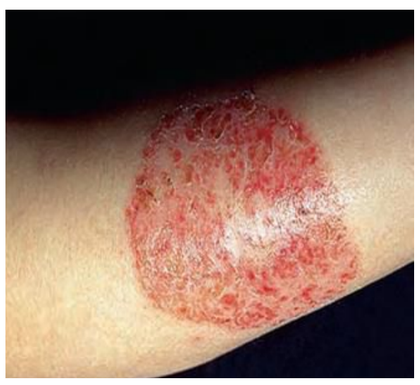
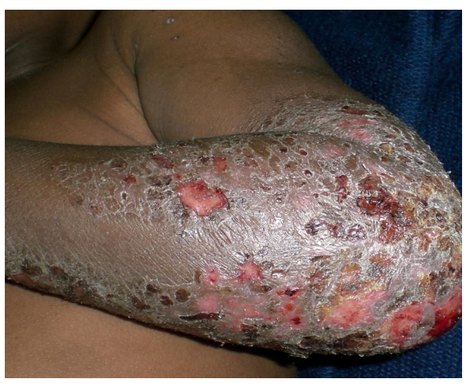
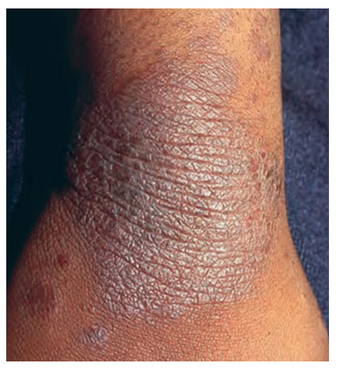
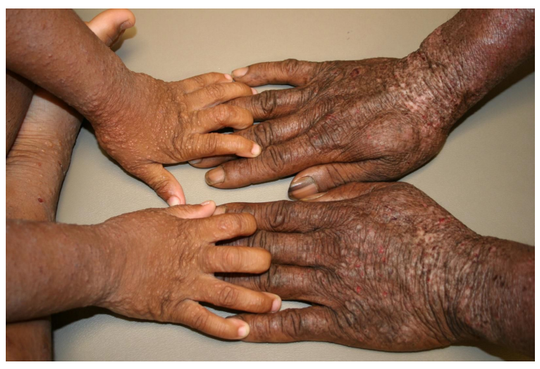


Eczema & Atopic Dermatitis

Eczema:

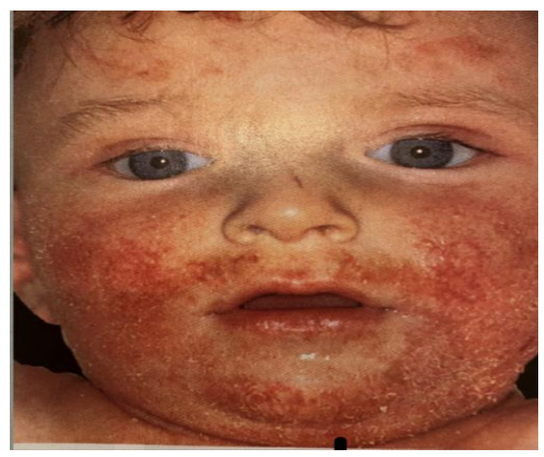
- Inflammation of the skin.
  Eczema phases:
  - **Acute eczema:** Erosion "losing part of skin if deep we call it ulcer", oozing, & vesicles "Fluids". We usually don't see vesicles because of scratching.
  - **Subacute eczema:** Redness, swelling, crust ± scales, and infection. Crust dried blood, Scale thickening of outer most layer of skin.
  - **Chronic eczema:** Lichenification, dark pigmentation, and thick papules and plaques. Lichenification: Thickening of skin due to chronic rubbing, increase skin marking.

Extra: Any inflammation of the skin is called dermatitis, when we say eczema without naming a type we usually mean atopic dermatitis, which is the most common type.

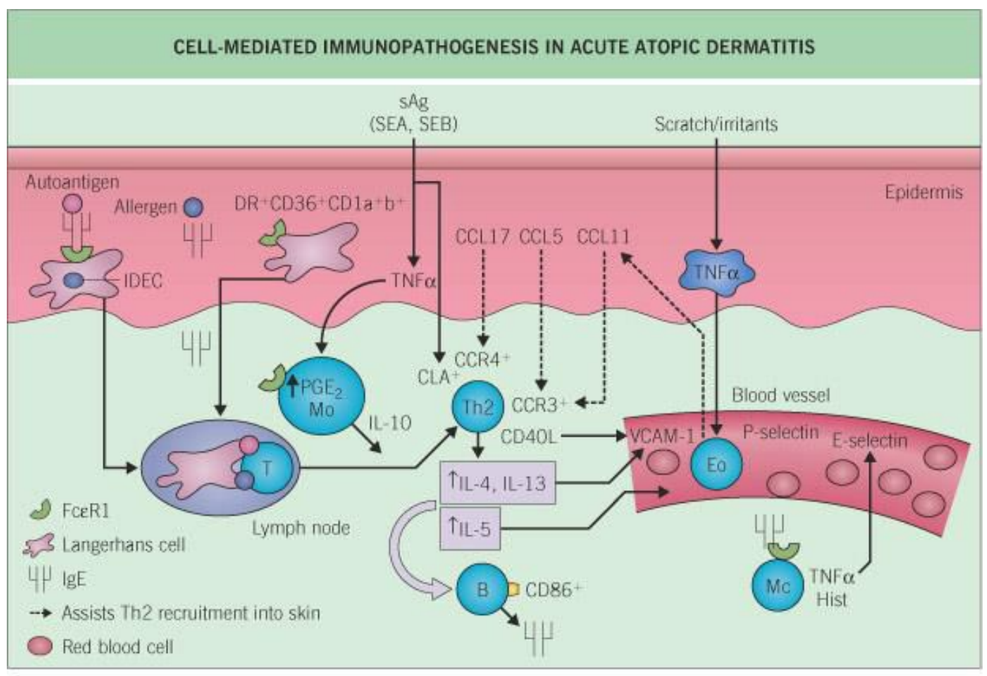
 	 	 
Presence of vesicles (**Acute**)	Scaley, crust, erythematous but not yet lichenification (**Subacute**)	Increased skin lines indicating lichenification (**chronic**) 2nd picture demonstrate the genetic inheritance between the father & the son

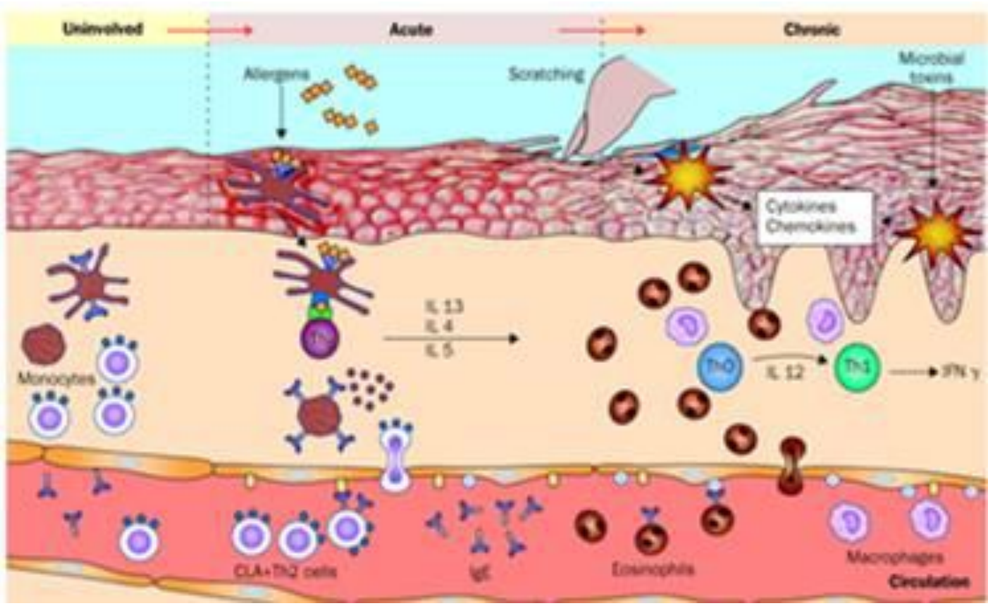
Atopic Dermatitis: حساسية تأتبية

- **Definition:** Chronic relapsing itchy skin disease in genetically predisposed patients.
- **Associated diseases:** Bronchial asthma, allergic rhinitis, allergic conjunctivitis.
- **Incidence**: up to 15-20% in early childhood.
- (personal or family hx) there's no causal relationship but they're associated
- Grow out tendency. Tends to be less severe as they grow up.
- More common in male.
- **Age of onset:**
  - 60%: First 2 months of life.
  - 30% By age of 5 years.
  - 10% Between ages of 6-20 years.
- Improves in summer and flares in winter.
  Its chronic disease but does it continue till adulthood? Only 10%, either incidence (first time) or continuation.



- **Pathogenesis:**
  - **Cause**;
  - Complex interaction of skin barrier, genetic, environmental, pharmacological and immunological factors.
    - **"Atopy"**: genetic predisposition: A protein in the skin called **Filaggrin**, genetic mutations in this gene results in dry skin
    - Mutation of FLG (encodes filaggrin) distributed skin barrier and function skin barrier defect: Dry skin (decreases production of moisturizing and lipid; sebum)
    - **Dry (atopic) skin** Xerosis (decrease human B-defensin 3 predisposing patients to frequent infections).
    - Immune dysregulation:
      - **T-Cell** activation (elevated Th2 cytokines & increased IgE production).
      - IgE (epiphenomenon)
    - Recent studies showed a potential role for the **Th17 pathway**, with increased circulating Th17 cells in atopic patients, & increased Th17 in acute eczematous lesions. A decreased Th17 in chronic eczema argues for a dynamic role for the Th17 pathway.





Through this cascade (first pic) we know that the disease is associated with IgE and Th2. The autoantigen that starts the cascade is *unknown.*

In the epidermis, the antigen presenting cell (langerhans cell in skin) meets with the allergen, and they both react in the lymph node > activation of lymph node > activation of T-cell > transformation into T helper 2 (Th2), which:

1. Increases IL-5: Increases Eosinophils
2. Increases IL-4 & IL-13: Activate the Vascular-Endothelial system > Inflammation (WBC, Cytokines..etc).

- IgE & eosinophils will be high when you do CBC
- Acute phase Th2 patterns & Chronic phase Th1 patterns, immunoregulation is complex. There is involvement of langerhans, IgE, and eosinophils which is imp in the treatment.
- Now the treatment against IgE **omalizumab**, but it's not effective on all types of atopic dermatitis because of different patterns.
- In **acute phase the skin is red and inflamed**, convert into thick and **lichenified in chronic stage,** cytokines change in chronic stare.
- Steroids consider anti-inflammatory on every cytokines any AD patterns, that's why we favour it, better if used topically.

- **<u>Triggers:</u>**
  - Allergies - increased tendency to certain allergens (autoallergen)
  - Infections: skin of patients with AD is colonized by S. Aureus. Infection often causes a flare.
  - AD and food = minor role. Parents often ask about egg, chicken, and milk worsening the condition, but it doesn't have anything to do with AD on the basis of pathology. Maybe there is association, but no causation or activation.

    Dr said "the best test is if the parents suspect certain foods make it worse, to stop it for 2wk, then reintroduce it. If it improves and worsen then they stop it". Allergens (like food) may cause eczema, asthma or anaphylaxis.".
- **<u>Prevalence:</u>**
  - Prevalence is almost 20% in the US, representing a marked increase during the past several decade.
  - Studies before 1960 estimated the prevalence to be up to 3%.
  - AD is often the 1st manifestation of the "atopic march".
  - AD → Asthma → Allergic rhinitis.
  - Asthma occurs in up to 50% of children who develop AD during the first 2 years of life.
  - Allergic rhinitis develops in 43-80% of children with AD.
  - In general, children showing more severe dermatitis have a higher risk of developing asthma, as well as sensitization to foods and environmental allergens.
  - AD occurs more frequently in urban areas than rural, in smaller families, and in higher socioeconomic classes.
  - Ultimately 80% of patients will develop increased IgE levels.

Loss of function mutations in profilaggrin (FLG) causes ichthyosis vulgaris, a common genetic disorder characterized by dry scaling skin, and hyperlinear palms that has long been known to be common in individuals with AD. Distinct mutations in FLG has been discovered in the European and Japanese populations, but all are strongly linked with AD, particularly of early onset.

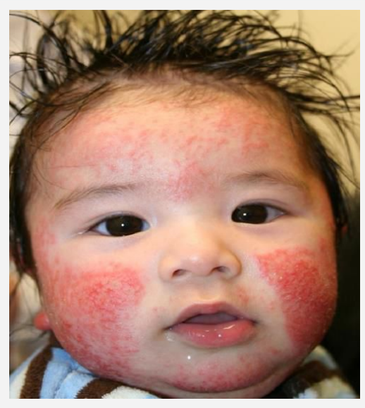
- **<u>Histology:</u>** rarely done because it's a clinical diagnosis
  - Edema within the epidermis (spongiosis) and infiltration with lymphocytes and macrophages in the superficial dermis.

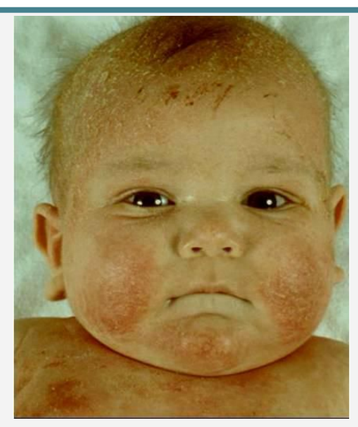
- **Clinical variants:** 1.Infantile AD, 2.Childhood AD, 3.Adult AD

Infantile AD: <2 years old, Acute - localized on face

Distribution: scalp, neck, forehead, wrist, and extensors.
Red skin, tiny vesicles on "puffy" surface. Scaling, exudate with wet crust and fissures, Dennie-morgan fold
Diaper & scalp usually spared.
60% of case AD present in the first year of life, after 2 months of age.
Begins as itchy erythema of the cheeks.

The patient
Looks well
Not ill

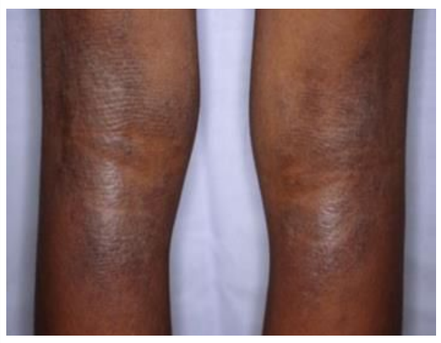




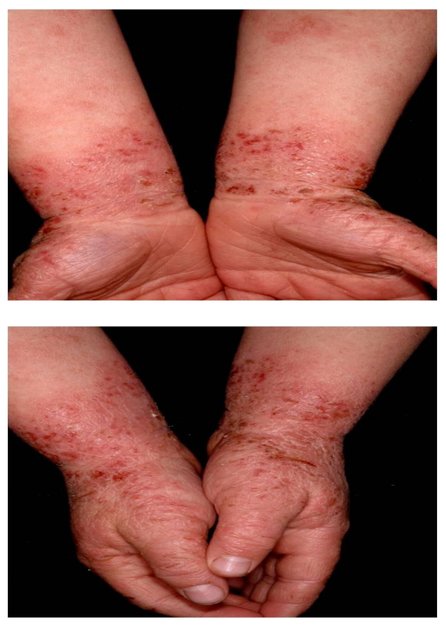
Well demarcated, Ill defined, erythematous plaques, crusty and non-scaley.
Over the cheeks and forehead sparing the nasolabial fold.

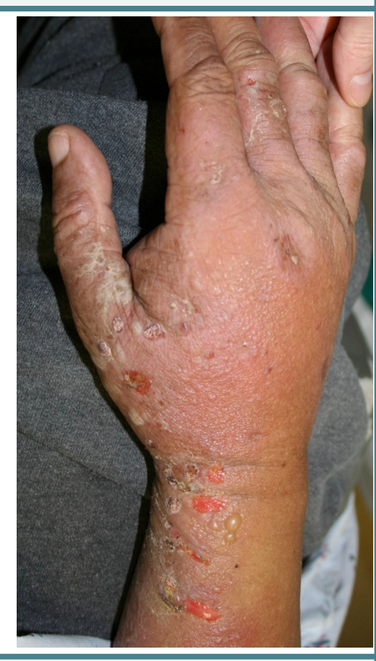
Childhood AD: 2-12 years old

Distribution: antecubital, popliteal fossae, flexor wrists, eyelids, neck, and face.
May be generalized.
Characterized by less acute lesions.
Papular, lichenified plaques, erosions, and crusts.
Severe atopic dermatitis involving more than 50% of the body surface area is associated with growth retardation.
More chronic presentation.



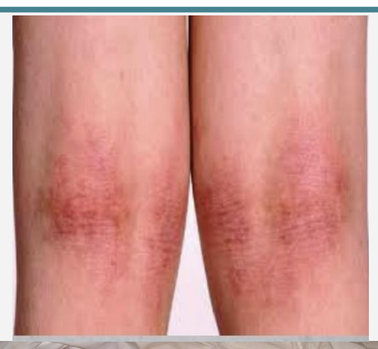
Ill demarcated hyperpigmentation with lichenification

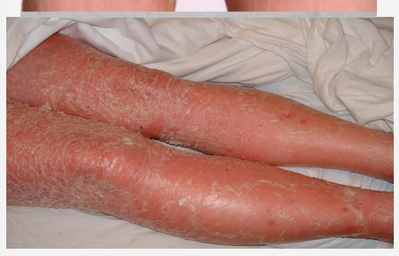


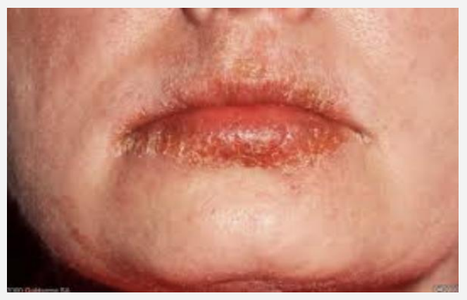


Adulthood AD: >12 years old, Chronic.

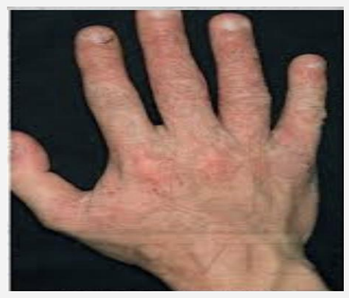
Distribution: mostly flexural, face and neck.
Usually more localized over one area only (like the neck, hands or eyelids) but may also be generalized.
May also involve classical areas (antecubital or popliteal)
Lichenification and excoriations. Its dry so you will know it from the morphology





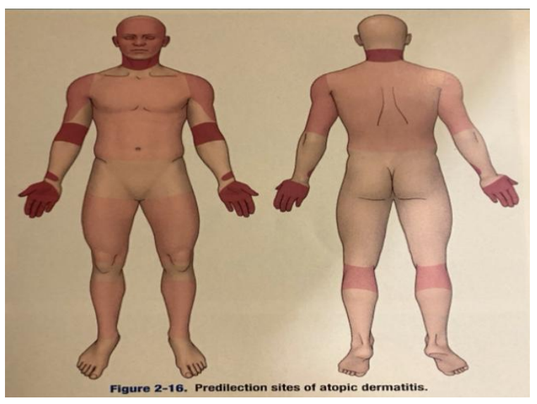


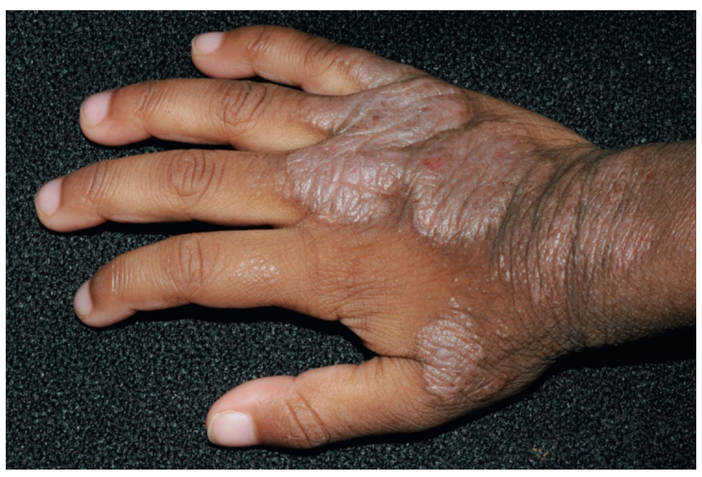
lichenification



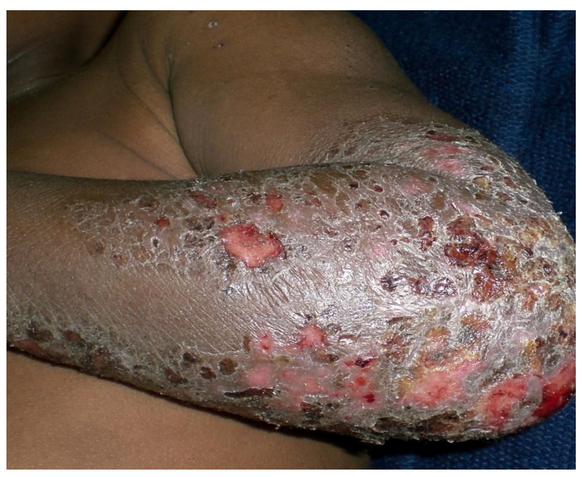
←Erythroderma:
A very rare complication of atopic dermatitis

- Acute > infantile
- Localized on hands eyelids & lips > adult
- Lichenified plaques more with adults and childhood because they are chronic

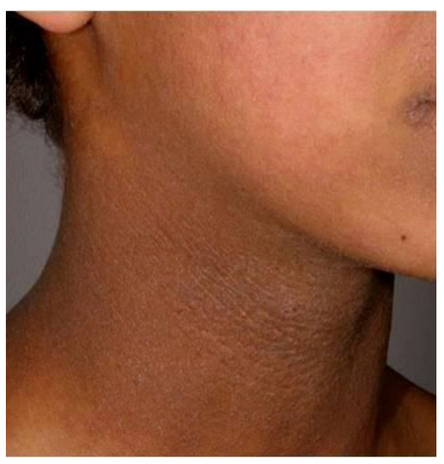




Chronic lichenified plaques, well defined, looks like psoriasis. The difference is psoriasis silvery scales and dont present here. The manifestation called "psoriasiform eczema"



There is erosions. Complication: bacterial infection, infective eczema itchy, painful fever, & discharge, if the skin barrier did not develop skin is prone to infection, most commonly staph aureus.



Eczema: lichenified plaques, its color depends on the natural skin tone thats why its darker here but you can see redness. Also it affect the lips. Its childhood pattern overlap with adult

- Atopic individuals have a distinct tendency towards an extra line or groove of the lower eyelid, so called "atopic pleat", is present at birth or shortly after and usually retained throughout life, referred to as "Dennie-Morgan fold". (They could present even without facial involvement)
- Another feature, an exaggerated linear nasal crease, caused by frequent rubbing of the nasal tip (allergic salute), although not a specific sign of AD.

- **Investigations:**
  - Atopic dermatitis is purely clinical; No investigations are needed.

Hanifin criteria

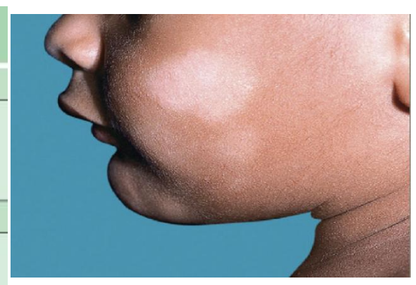
DIAGNOSTIC FEATURES OF ATOPIC DERMATITIS

Major features (3 of 4 present)

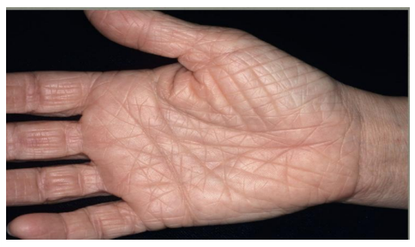
- Pruritus
- Typical morphology and distribution of skin lesions
- Chronic or chronically relapsing dermatitis
- Personal or family history of atopy

Minor features (3 of 23 present)

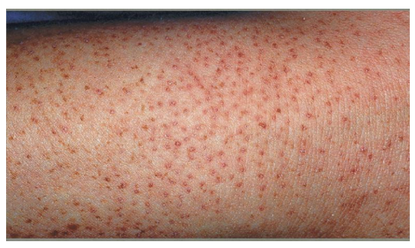
- Xerosis
- Ichthyosis/palmar hyperlinearity/keratosis pilaris
- Immediate (type I) skin test reactivity
- Elevated serum IgE
- Early age of onset
- Tendency towards cutaneous infections/impaired cell-mediated immunity
- Tendency towards non-specific hand or foot dermatitis
- Nipple eczema
- Cheilitis
- Recurrent conjunctivitis
- Dennie–Morgan infraorbital fold
- Keratoconus
- Anterior subcapsular cataract
- Orbital darkening
- Facial pallor/erythema
- Pityriasis alba
- Anterior neck folds
- Pruritus when sweating
- Intolerance to wool and lipid solvents
- Perifollicular accentuation
- Food intolerance
- Course influenced by environmental/emotional factors
- White dermographism/delayed blanch



Pityriasis alba (hypopigmentation), Not vitiligo which is depigmentation



Hyperlinearity



Keratosis pilaris

Updated criteria,

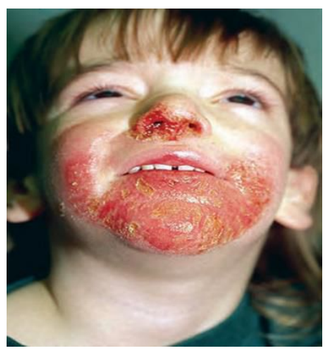
Table 5.I. Revised criteria for the diagnosis of atopic dermatitis[4]

a. Must have:
- Pruritus

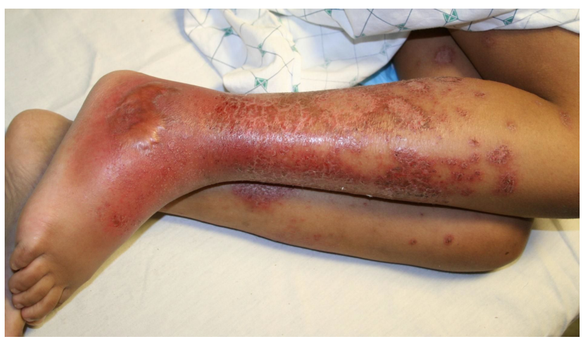
b. Plus 3 or more of the following:
- History of involvement of skin creases (front of elbows, back of knees, front of ankles, neck, around the eyes)
- History of a generally dry skin in the past year
- Personal history of asthma or hay fever
- Onset under the age of 2 years
- Visible flexural dermatitis

The diagnosis of atopic dermatitis in adults is primarily clinical; special investigations only contribute in identifying external aggravating factors.

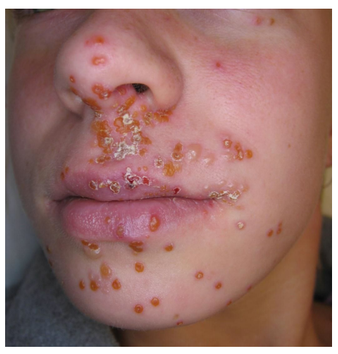
- **Complications:**
  - Secondary infections the skin acts as a barrier so once it is diseased it becomes easily infected by Strep or Staph. Aureus, and the skin will become crusty with yellow exudates. In *Impetigo* you see **honey-colored** crust skin and if not treated may lead to sepsis.
  - Eczema herpeticum caused by herpes simplex virus: grouped vesicles, the patients seems very unwell and has a fever. Usually adults that are seen in the ER. Management: it's an EMERGENCY SO admit, IV acyclovir, analgesia and call for an ophthalmologist.
  - Growth retardation due to chronic steroid use. If the baby doesn't sleep well due to itching he wont eat well either.
  - Psychological Missing school or work due to their appearance.
  - PIH Post Inflammatory Hyper/hypo pigmentation



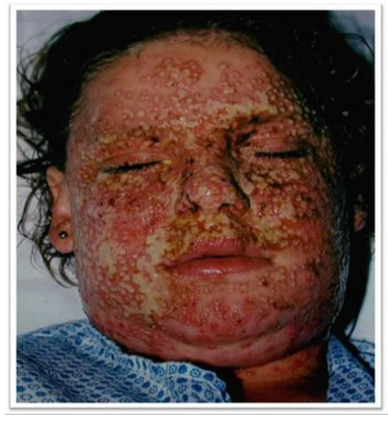
Secondary infections



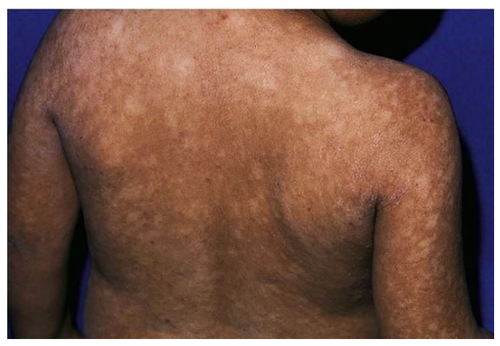
Cellulitis
S. aureus



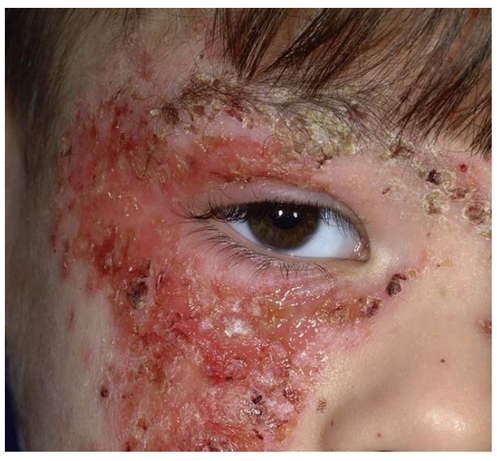
Impetigo caused by s. Aureus or streptococcus



Eczema herpeticum



PIH



Secondary bacterial infection around eczematous plaque

- **Management:**
  - Education! Education! Education! Educating the patient/parent about the disease, prognosis & management > they understand the chronic and relapsing nature of the disease > better compliance > avoidance of complications of topical steroids.
  - Psychological support.
  - Skin care: moisturizing skin. Every 2h, anything that suits the patient, using moisturizers with no perfumes may be best.
  - Topical therapy: (topical steroids, Calcinuerin inhibitors, Tacrolimus (not for acute cases) , Pimecrolimus) Mild-mid potent for babies, Calcineurin inhibitors such as Tacrolimus & Pimecrolimus are good for areas prone to side effects of steroid, and for maintenance since CS can't be good for long periods of time. Calcineurin inhibitors when used on an active disease may cause stinging. Tacrolimus & Pimecrolimus used for maintenance not acute phases, they are anti inflammatory non steroidal.
  - Phototherapy. Also used in psoriasis and vitiligo. Not much S/E mainly dryness & tanning so patient need to mostirouz. Anti inflammatory, lower the cytokines and t cells without any medication.
  - Antibiotics: (Antistaphylococcal drugs) if there's an infection.
  - Sedative antihistamine (Oral H1 antihistamine) to control itching and help sleep. If itchiness is severe and prevents the baby from sleep. Histamine has no role in the pathogenesis of AD, giving non sedative antihistamines would be useless.
  - Systemic therapy: (if not improved with topical) Steroids (should be avoided except in sever disease in
  - adult), Cyclosporin (Can cause renal toxicity and HTN) , Methotrexate, Azathioprine.
  - In severe cases we use steroid as first line then we decrease the dose and start tacrolimus and others .
  - If you use topical steroid on large area of skin it might act like systemic steroid and cause systemic side effects will happen, like cataract, HTN, DM, & adrenal suppression.

EXTRA from 435:

- **First line:**
  - Topical corticosteroids
  - Topical calcineurin inhibitor (Tacrolimus "Protopic" & Pimecrolimus) - Doesn't have the side effect of topical steroids.
  - Oral H1 antihistamine for sedation effect only.
  - Oral antibiotic treatment of bacterial infection in patient with eczema: antibiotics + corticosteroids.
- **Second line:**
  - Systemic steroids.
  - Phototherapy (PUVA, NBUVB).
  - Immunosuppressive therapy.

Local cutaneous side-effects
- Atrophy
- Striae
- Periorificial granulomatous dermatitis
- Acne
- Telangiectasia
- Erythema
- Hypopigmentation

Ocular effects
- Cataracts
- Glaucoma

Systemic side-effects
- Hypothalamic-pituitary-adrenal axis suppression

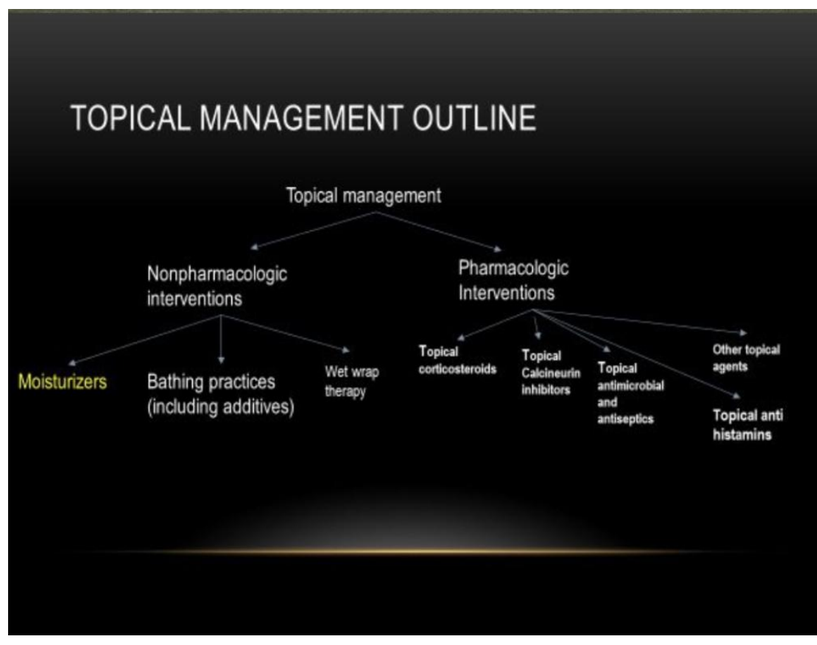
Management and treatment of Atopic dermatitis:

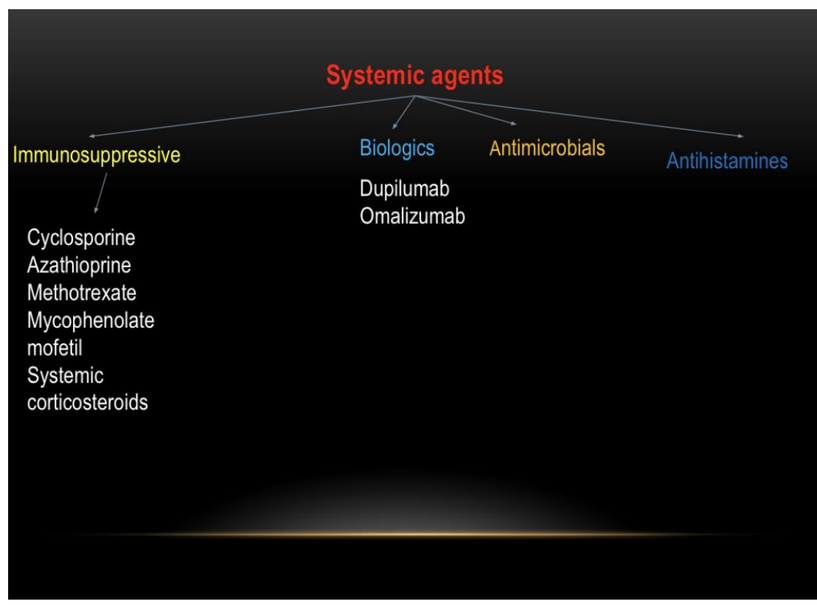
- Topical treatment
- Phototherapy
- Systemic therapy

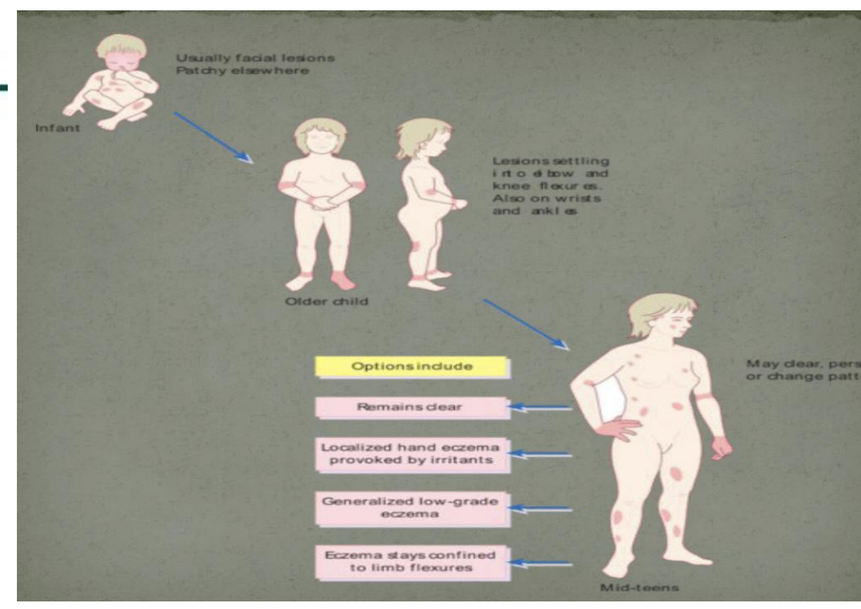
You need to know one from each class.

Topical steroid class American classification	Topical steroid class British classification	Common representative topical steroids	Indications
I	I	Clobetasol propionate 0.05% cream or ointment (Mostly to adult)	Alopecia areata
Superpotent corticosteroids	Very potent		Atopic dermatitis (resistant)
		Halobetasol propionate 0.05% cream or ointment	Discoid lupus
		Betamethasone dipropionate 0.05% ointment	Hyperkeratotic eczema
II	II	Betamethasone dipropionate 0.05% cream	Lichen planus
Potent corticosteroids	Potent	Fluocinonide 0.05% ointment	Lichen sclerosus (skin)
		Halcinonide 0.1% cream (Mild-moderate not on face)	Lichen simplex chronicus
		Mometasone furoate 0.1% ointment	Nummular eczema
III		Betamethasone dipropionate 0.05% lotion	Psoriasis
Upper mid-strength corticosteroids		Fluticasone propionate 0.005% ointment	Severe hand eczema
		Triamcinolone acetonide 0.1% ointment (Over face sometimes for only 23 days)	
		Halometasone 0.05% cream	
IV		Fluocinolone acetonide 0.025% ointment	Asteatotic eczema
Mid-strength corticosteroids		Mometasone furoate 0.1% cream or lotion	Atopic dermatitis
V	III	Betamethasone valerate 0.1% cream	Lichen sclerosus (vulva)
Lower mid-strength corticosteroids	Moderate	Fluocinolone acetonide 0.025% cream	Nummular eczema
		Fluticasone propionate 0.05% cream	Scabies (after scabicide)
		Hydrocortisone butyrate 0.1% cream	Seborrheic dermatitis
			Severe dermatitis
			Severe intertrigo (short-term)
			Stasis dermatitis
VI		Alclometasone dipropionate 0.05% cream or ointment	Dermatitis (diaper)
Mild corticosteroids			Dermatitis (eyelids)
		Desonide 0.05% cream	Dermatitis (face)
		Fluocinolone acetonide 0.01% cream	Intertrigo
		Triamcinolone acetonide 0.025% cream	Perianal inflammation
VII	IV	Hydrocortisone 1% or 2.5% cream, 1% or 2.5% lotion, or 1% (Used on face)	
Least potent corticosteroids	Mild	2.5% ointment	
		Hydrocortisone acetate (1% or 2.5% cream, 1% or 2.5% lotion, or	

Courtesy *Adapted from Ference JD, Last AR. Choosing topical corticosteroids. Am Fam Physician 2009;79:135–140



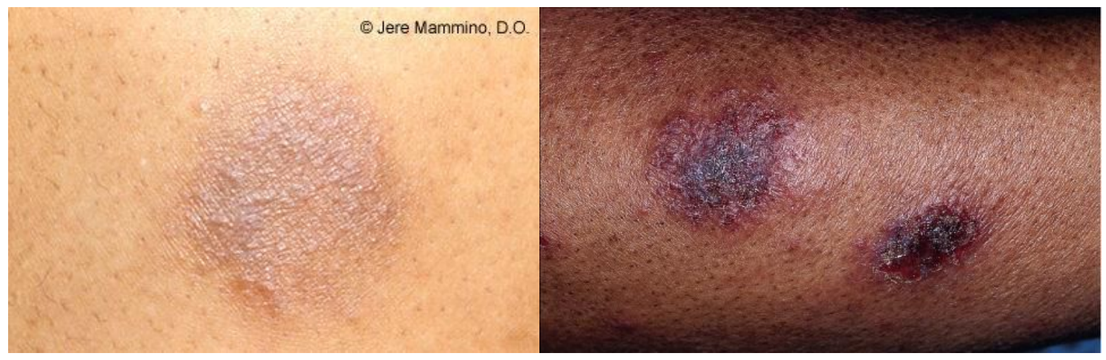




- **Prognosis:**
  - Half of the cases improve by 2 years of age.
  - Most improve by teenage years.
  - <10% of patients have lifelong problems.
  - 30-50% will develop BA or hay fever.

Nummular dermatitis:

Coin shaped patches & plaques.
Secondary to xerosis cutis
Primary symptom: itch.

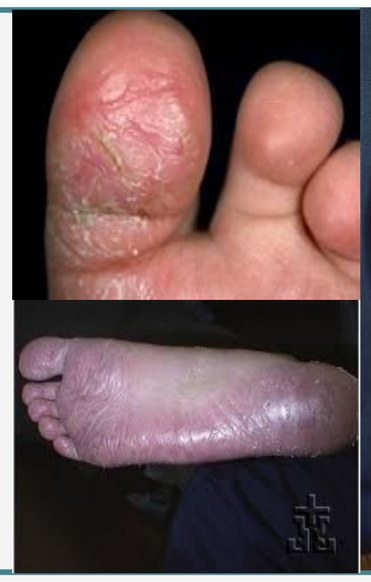


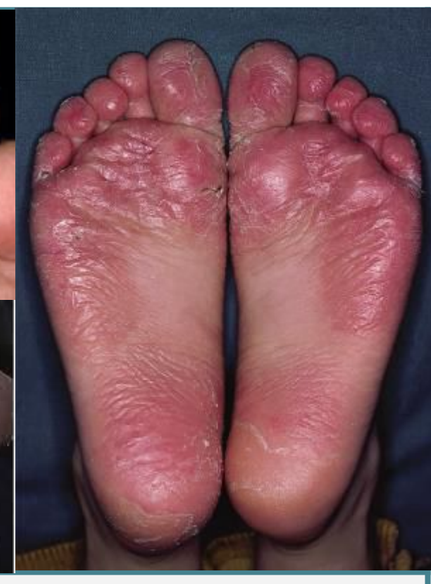
Regional eczema :

1- Juvenile plantar dermatosis:

Begins as a patchy symmetrical, smooth, red, glazed macules on the base of the great toes.
Affect ages 3 to puberty.
Symmetrical lesions on weight bearing area.
Virtually always resolve after puberty.

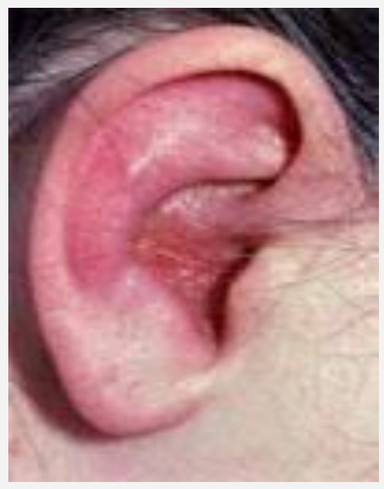
- Found in teens
- Erythema & fissures
- A defect in sweat glands (occlusion)
- Caused by wearing shoes a lot.
- Subsides on its own.
- Management: moisturize & air out





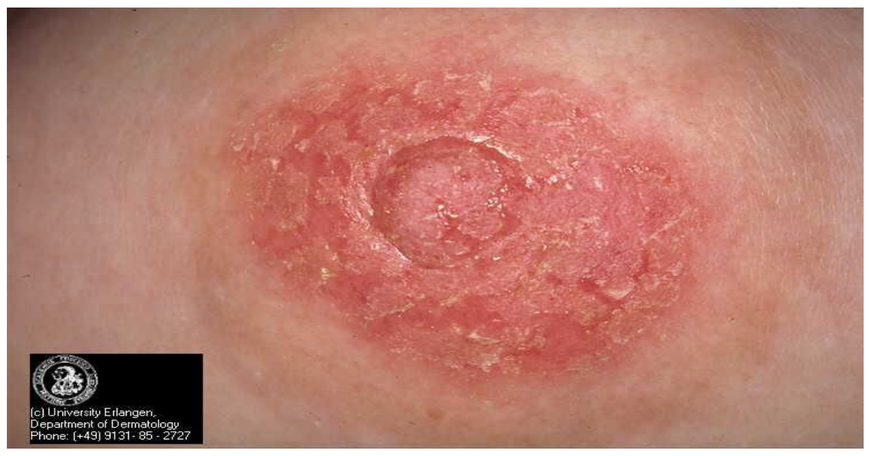
2- Ear eczema:

Most frequently caused by seborrheic or atopic dermatitis.
Staph, Strep, or Pseudomonas.
Earlobe is pathognomonic for nickel allergy.



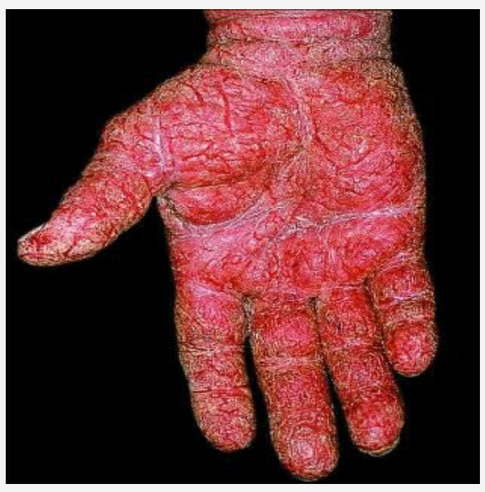
3- Nipple eczema:

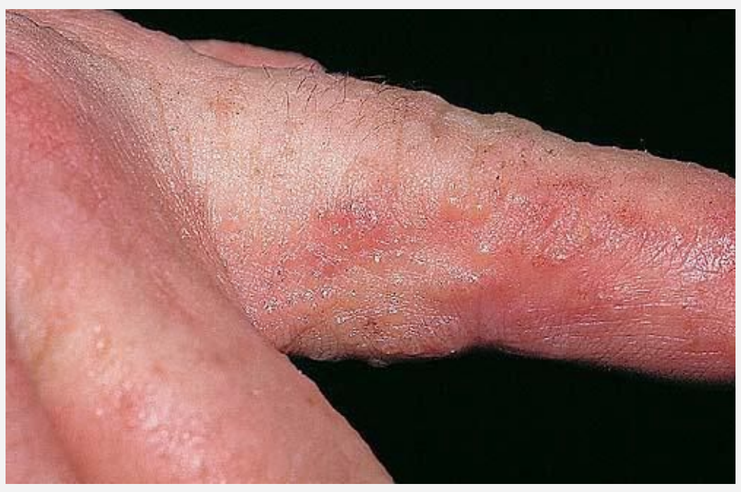
Painful fissuring, seen especially in nursing mothers.
Maybe an isolated manifestation of atopic dermatitis.
If it persists more than 3 months, and/or is unilateral, biopsy is mandatory to rule out Paget's disease.



4- Hand eczema:

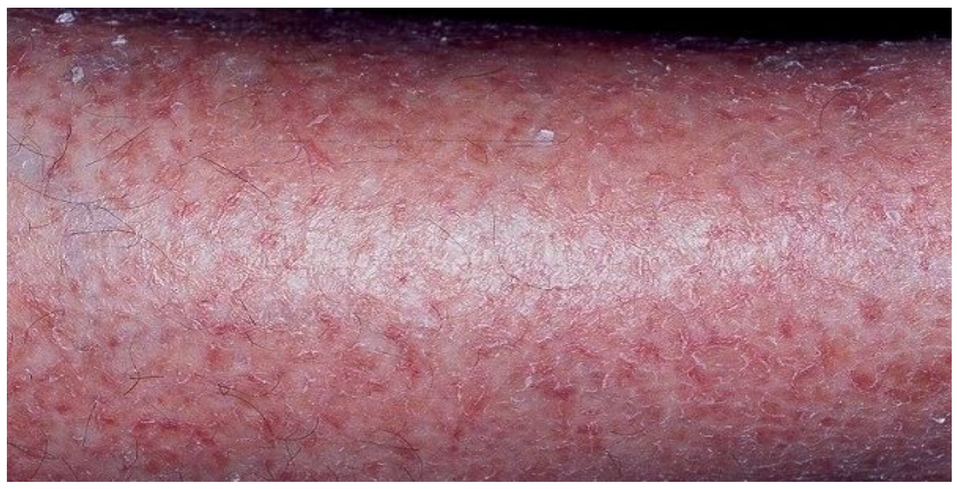
Spongiosis histologically.
Irritant hand dermatitis - seen in homemakers, nurses.
Result of excessive exposure to soaps.
Pompholyx- tapioca vesicles, on sides of fingers, palms, and soles.
Irritant, not allergic.



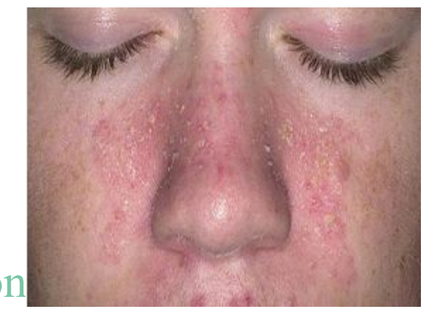


5- Xerotic eczema:

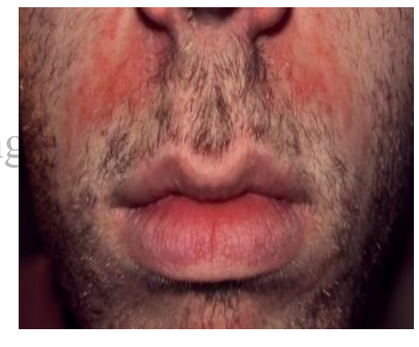
AKA winter itch, nummular eczema, eczema craquele, and asteatotic eczema.
Affects anterior shins, extensor arms, and flank.
Elderly person is predisposed.
Use of bath oils in bath water is recommended to prevent water loss.
Moisturizers - urea or lactic acid.



Seborrheic Dermatitis: الاكزيما الدهنية



Ddx: seborrhic dermatitis
Rosacea , SLE

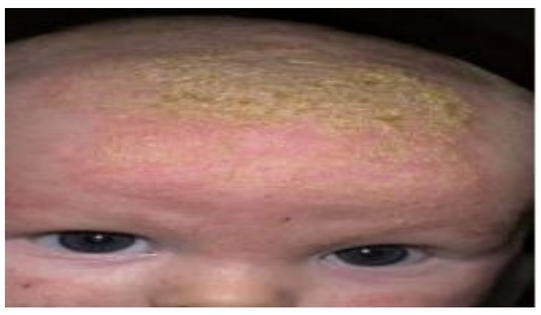


- **Definition:** redness and scaling in regions where the sebaceous glands are most active as the face, scalp, presternal area and body folds. (oily, greasy, scaly, and erythematous). The distribution is different from AD! Affects sebaceous glands(hence the distribution such as the face , scalp etc..) but the sebaceous glands are not inflamed as opposed to acne where there is inflammation of the pilosebaceous unit .
- Very common chronic dermatosis.
- **Age:** infancy, puberty, old age Commonly seen in children .
- More in males
- **Pathogenesis:** Increased Sebum (seborrheic state) - Tendency - Pityrosporum ovale overgrowth (Malassezia furfur) "dimorphic normal flora in the skin fungal type; here it only causing eczema, but in other diseases it can cause infection", concentrate on this fungus as its found in other diseases also (will be mentioned in another lecture). - More in Parkinson, HIV/AIDS patients "decrease in immunity".
- **Clinical features:**

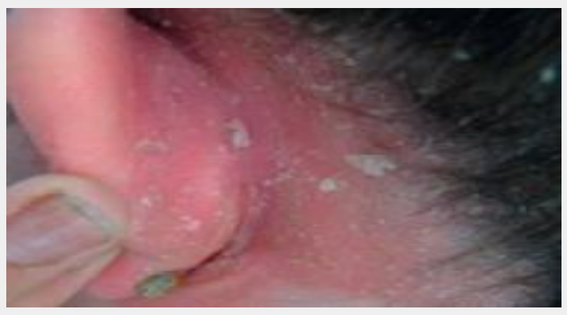
Distribution:

o Hairy area of head, cradle cap
o Face: forehead, nasolabial folds, glabella (على الأنف) and eyebrows.
o Trunk: DDx: PR vs pityriasis versicolor
o Body folds: axillae, groins, anogenital area, sub mammary areas, umbilicus and diaper area (infants)--- sharply marginated erythematous eruption, erosions and fissures
. o Genitalia : with yellow crust and psoriasiform lesions.

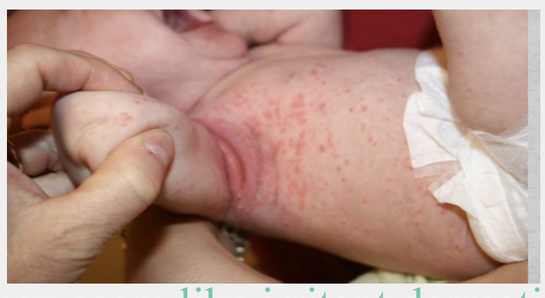
Presentation: Pruritus is variable - Gradual onset, worse in winter dry environment - Orange/red greasy scaling macules, papules of varying size - Trunk: nummular, annular - Scalp: marked scaling, diffuse involvement

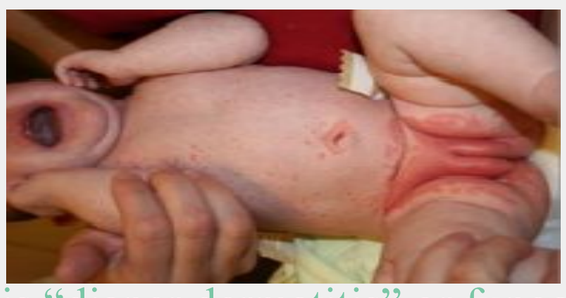


Scaly, yellowish, greasy, oily, adherent plaque, with erythema beneath it. (cradle cap).

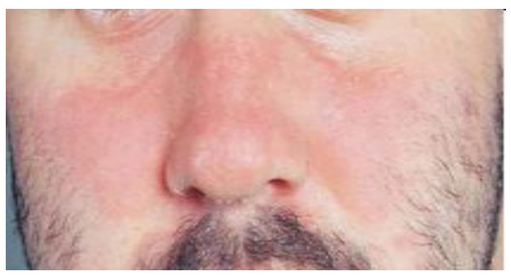


Here you can see erythema which will help differentiate it from dandruff

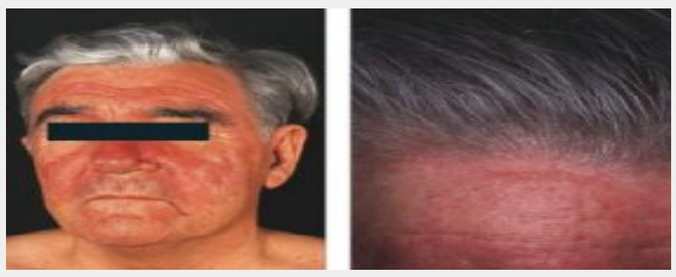




it may seem like irritant dermatitis "diaper dermatitis" or fungal infection (ddx), but here it's shiny and erythematous and other area like axilla and scalp are involved.



Ill-defined, oily, erythematous skin Distributed normally on areas with sebaceous glands.



it is not photo distribution; the nasolabial fold is involved. Unlike lupus, where the nasolabial fold is spared.

- **Management:** doesn't subside but comes and goes depending on fungal activity .
- **Scalp:** all the shampoos are anti-fungal

o Zinc pyrithione Shampoo
o Selenium sulfide 2.5% shampoo
o 2% ketoconazole shampoo o
Low potency glucocorticoid solution, lotion or gels.

Its recurrent and chronic but respond to treatment very well

- **Skin:**

Topical: antifungals, glucocorticoid, pimecrolimus
o Combined therapy antifungal and hydrocortisone > Daktacort
o You can use metronidazole instead of the steroids o Maintenance & recurrence

Contact dermatitis:

Definition: dermatitis results from contact with external materials.

Pathogenesis: Irritant (cytotoxic): not related to an immune reaction so anyone is susceptible to it vs. allergic (type IV): only genetically predisposed people are prone.

Common irritants: detergent, acids, dust, burning chemicals, etc

Common allergens: perfumes, hair dyes, nickels (e.g. watch), leathers (shoes), metals, rubbers (Gloves), latex (Material in gloves), cosmetics, etc

In allergic dermatitis; they may wear a watch and their allergy will cause itching in distant areas

Allergic CD

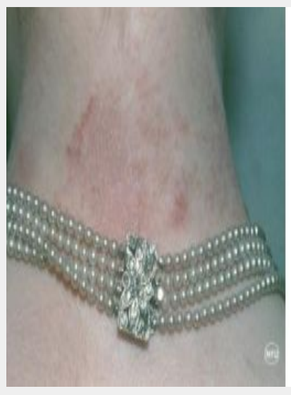
o It is caused by an allergen that triggers a type IV hypersensitivity(Delayed there's a +ve history of exposure to a specific allergen) reaction in a sensitized person. May give Distance area itching.

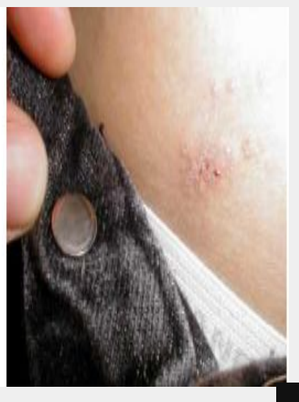
o Characteristics : - First exposure does not cause a reaction

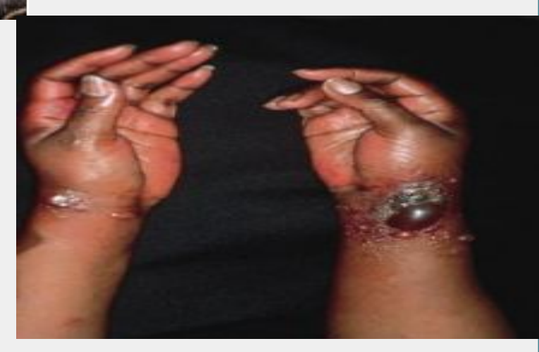
-Begins 24 h after subsequent exposure if already allergic

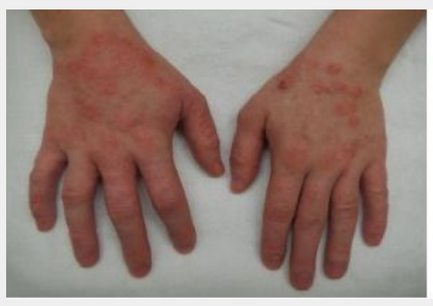
o Commonest: Nickel (the commonest) (commonly used in making watches , medals etc..) , chromates, rubber, preservatives (مواد حافظة), topical Abx, topical cs (corticosteroid).

o Diagnosis: Skin patch tests (read at 48, 96 h). it's the gold standard for diagnosis

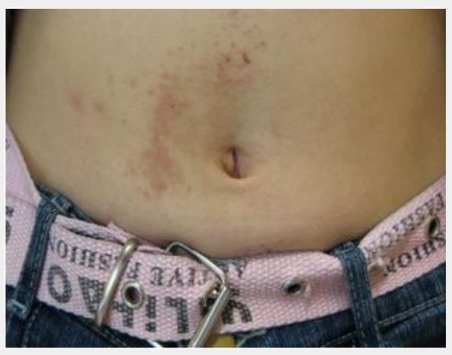






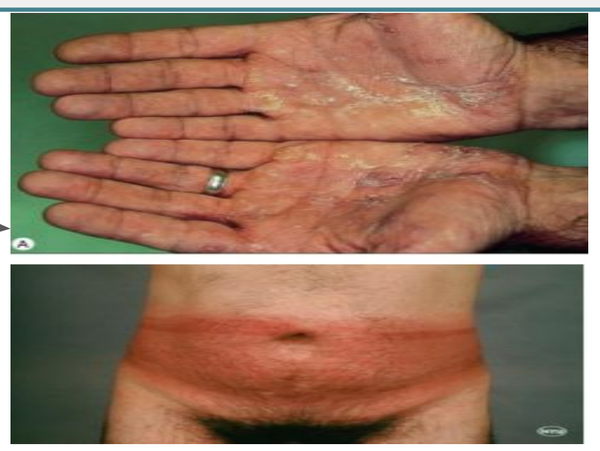


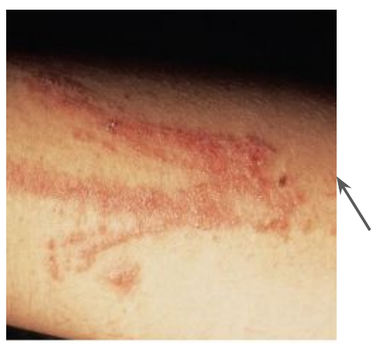
Ill-defined, erythematous plaque. Due to gloves (latex). Chronic; whenever wearing the gloves there will be itching.



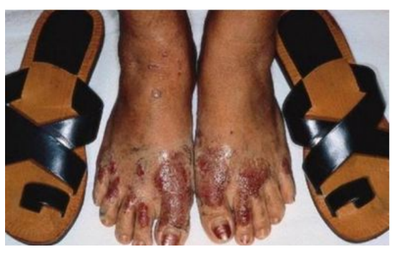
Due to the metal belt (Nickel)

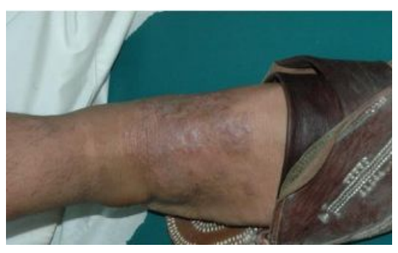
Potassium dichromate in leather

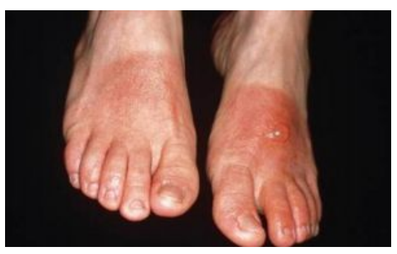




Poison Ivy/Oak/Sumac usually presents as a linear rash (as if it was stroked by a plant)







contact dermatitis at site of shoes

TOP TEN ALLERGENS AS IDENTIFIED BY THE NORTH AMERICAN CONTACT DERMATITIS GROUP

Test substance	Allergic reactions (%)	Relevant reactions (%)
Nickel sulfate	14.2	49.1
Neomycin sulfate	13.1	46.2
Balsam of Peru	11.8	82.9
Fragrance mix	11.7	86.9
Thimerosal 10.9		16.8
Sodium gold thiosulfate	9.5	40.6
Formaldehyde 9.3		63.2
Quaternium-15 9.0		88.7
Cobalt chloride	9.0	55.1
Bacitracin 8.7		50.4

Shoe Dermatitis:

Causes: **a**. Rubber (most common) **b.** Chromates (in leather) **c**. Glutaraldehyde (in leather) **d.** Adhesives **e.** Dyes

Clinical Features: o Predilection sites: site of contact o Distribution & configuration : In the pictures you can tell it was due to shoes because of its morphology, try to imagine what kind of shoes they were wearing

Management: o Identification removal of causes. o Patch testing: for allergic contact dermatitis not for irritant o Avoidance allergens o Topical corticosteroids for either irritant or contact dermatitis

Irritant CD

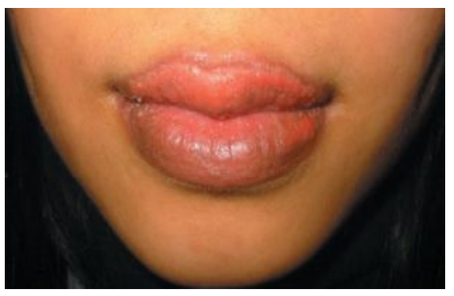
o All people will react to an irritant if applied in a high enough concentration or to sensitive skin. Only Localized itching

o At 1st exposure

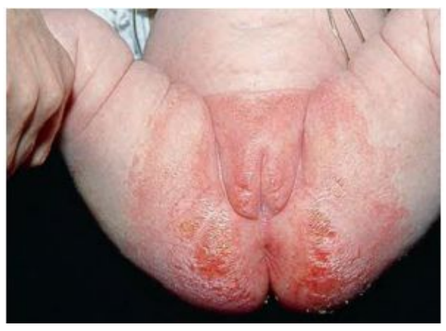
o Common causes:

- Hands repeatedly exposed to water, cleansers
- Lip-licking habit ´ wetting and drying caused by saliva
- Napkin dermatitis .

- - (irritant contact dermatitis) It accounts for 80% of occupational skin diseases
- **-Prevention is key!**
  (excessive use of hand sanitizers is a common irritant)



Irritant or allergic? its confined to the lips and there is fissure so **it's irritant**. However, with allergies it goes around the lips. Swollen due to saliva > lip-licking.



Ill-defined, erythematous scaly plaques and fissures. Due to stool or urine (cytotoxic material). - Change the diaper + use cotton + Put zinc peroxide + small amount of topical steroids for a week or so. It's treatable when managed acutely

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

Difference between allergic and irritant:

Allergic > well defined, later on after the exposure, sensitive skin.

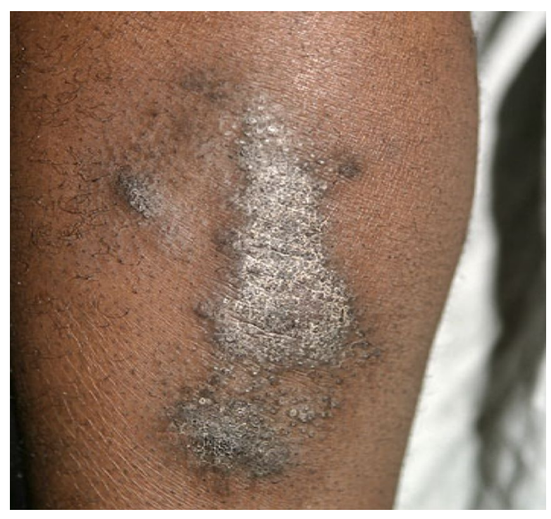
Irritant > ill defined, with the first exposure.

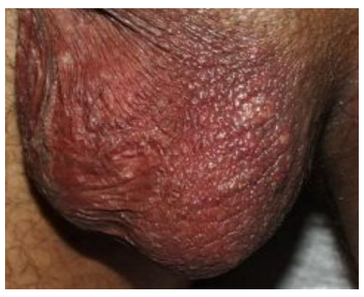
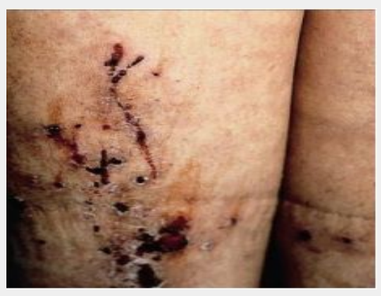
Q: Who has more sensitive skin?

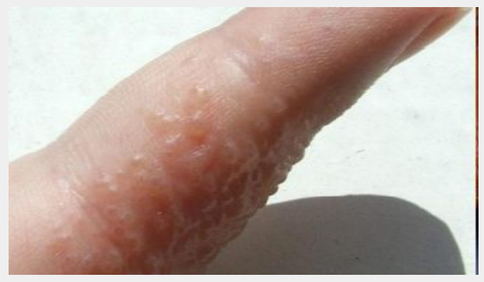
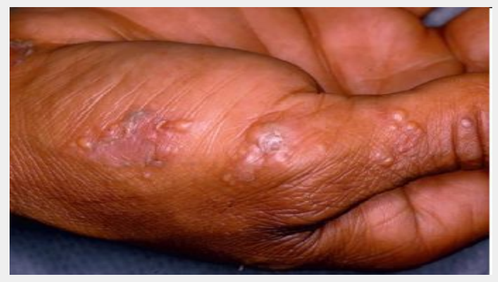
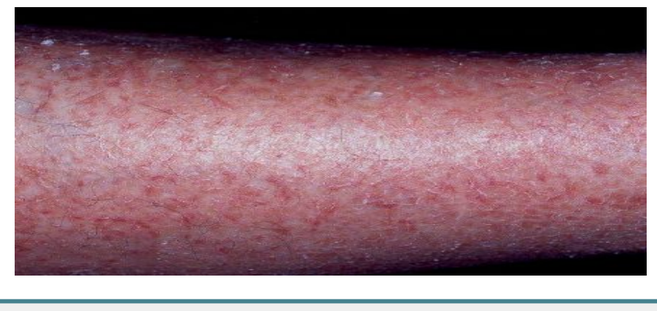
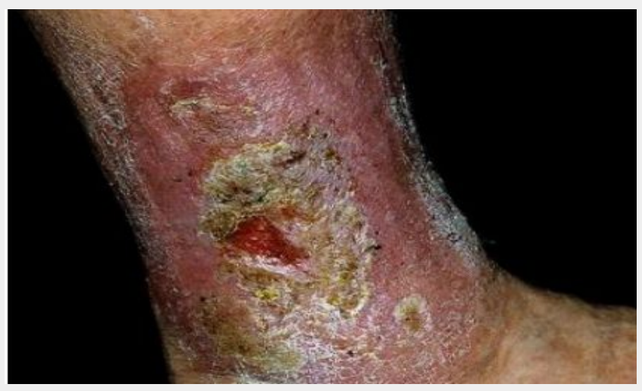
Allergic dermatitis

Neurodermatitis (Lichen Simplex Chronicus):

- This condition is usually seen in the elderly
- Paroxysmal pruritus
- Habitual **excoriating or rubbing** They don't get relieve until they see bleeding.
- Can be triggered by stress and anxiety. refer to psychiatry(areas that are hard to reach is always symptomatic free) e.g. back.
- Skin thickens to defend
- Consider underlying disease
- Lichen = thickened and lichenified plaque Simplex chronicus = chronic
- Induced by the patient (continuous itching on specific area), unilateral, not eczema nor inflammation, it's psychocatnous maybe depression or anxiety, refer to psychology, don't respond to eczema medications.



		
No fungus on the scrotum!	Prurigo simplex the patient doesn't feel pain from all the excessive itching (the pain is inhibited) this facilitates itching to the extent of causing severe damage .	Increased skin markings

Dyshidrotic Eczema : الفقاقيع -Associated with sweat glands -Deep seeded small vesicles -Poor prognosis > systemic steroids -vesicles on the sides of the fingers	 
Asteatotic Eczema : بقع اكزيما -Due to excessive dryness -In eldelry	
Stasis eczema : -Stasis of blood. -Crusts, erythema, erosions, & ulcers “venous”. -Patient with varicose veins that develop venous ulcers on the lower medial aspect of the leg + eczema surrounding that area. -Topical steroids but the vascular surgeon must treat it or else it will come back.	

Questions:

1) One-year old boy known to have atopic dermatitis presented to the emergency department with 1 day history of eruptive painful vesicles and crusted erosions over face. What is the most likely diagnosis?
A. Impetigo.
B. Pityriasis versicolor.
C. Eczema herpeticum.
D. Allergic contact dermatitis
Answer: C

2) A 6 months old infant had been very itchy, presented with Eczematous Eruption Diagnosis as Atopic Dermatitis. Which one of the following is the most common site distribution for the above patient of this disease?
A. Diaper Area.
B. Face.
C. Popliteal Area.
D. Scalp.
Answer: B

3) One-year-old boy known to have atopic dermatitis presented to the emergency department with a one-day history of painful vesicles and crusted erosions over his face associated with fever. How will you treat this patient?
A.Systemic antiviral
B.Topical steroid
C.Oral antibiotics
D.Oral Steroids
Answer: A (eczema herpeticum)

4) A-55-years-old female who works as a hairdresser presented with hand eczema. Which of the following best describes allergic and irritant contact dermatitis?
A.Patch test will be positive in irritant contact dermatitis
B.Irritant contact dermatitis is caused by delayed type hypersensitivity reaction
C.Allergic contact dermatitis occurs in previously sensitized individual
D.Allergic contact dermatitis is non-immunologically mediated
Answer: C

5) UVB narrowband treating which of the following?
A. Melanoma
B. Psoriasis
C. Atopic dermatitis
D. Urticaria
Answer: B(not sure)

6) Infant with dermatitis, diarrhea & hair loss . management?
A. Zinc supplement
B. Oral antibiotics
C. Topical steroids
D. Systemic steroids
Answer: A

7) What would you recommend to the parents of a child who was diagnosed with atopic dermatitis ?
A. using moisturizers
B. drinking fluids to prevent dehydration
C. using topical steroids every day
D. taking antibiotics to prevent infections
Answer: A

8) Which of the following is a major criterion in the diagnosis of atopic dermatitis?
A. Pruritus
B. Facial pallor
C. Dennie - Morgan folds
D. Hypopigmented patches
Answer: A