Derma Team 436

Eczema and atopic Dermatitis

Objectives :

- Not given

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Before you start.. CHECK THE EDITING FILE

Sources:doctor's slides and notes + FITZPATRICK color atlas +435 team
[Color index: Important| gold|doctor notes|432 team|Extra]

Eczema/dermatitis

A spectrum of inflammatory related disorders with pruritus being the hallmark of the disease (inflammation of the skin).

- eczema = dermatitis
- it has different types
- when we say eczema without naming a type we usually mean (atopic dermatitis) which is the most common type

types of dermatitis:

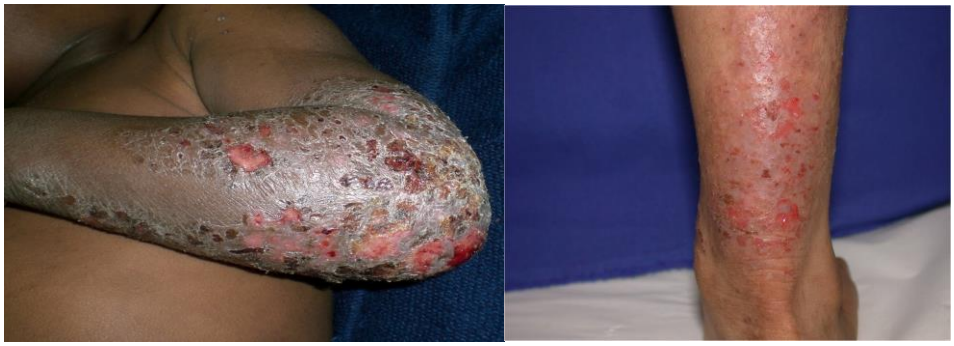
- **Atopic**, more common in children
- **Seborrheic** (oily skin)- (like naso-labial folds, scalp, ears)
- **Contact dermatitis**, substance cause eczema
  - Allergic
  - Irritant
- **Nummular**, coined shape, usually in the shin.
- **Asteatotic**, no seborrhea
- **Stasis** associated with venous insufficiency of the lower extremities.
- **Neurodermatitis** (acute) /**Lichen Simplex Chronicus** (chronic).

- It classified as:

Acute: characterized by erosion, vesicles, oozing, crusting, erythema and papules.
we usually don't see the vesicles because of scratching.
Subacute: clinically it is represented by erythema, scaling, and crusting.

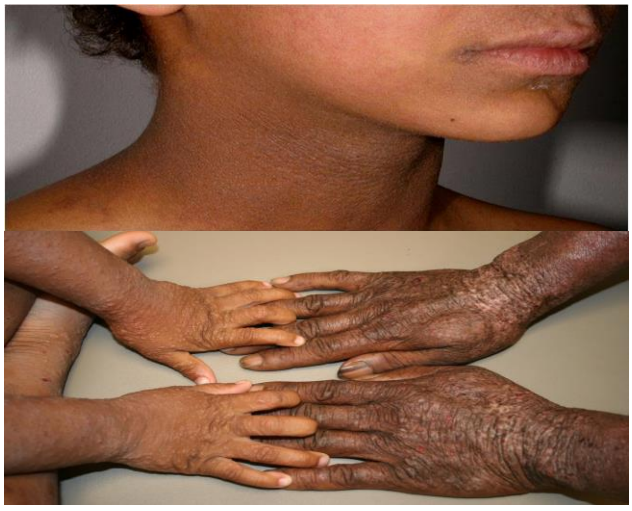
Chronic: presents with thickening of the skin, skin markings become prominent (lichenification); dark pigmentation and thick papules and plaques.

Acute eczema :



we can see erosions, and crust (crust indicates infection).

Chronic eczema:



Thickening of the skin on the neck

Thickening of skin

Atopic Dermatitis (AD):

Definition: chronic relapsing itchy skin (pruritis is the hallmark) disease in genetically predisposed patients.

Associated diseases: bronchial asthma, allergic rhinitis, allergic conjunctivitis

Incidence: up to 15% in developed countries

Grow out tendency: It tends to become less severe as they grow up

Pathogenesis:
- **Multifactorial**
- **"Atopy":** genetic predisposition
- **Dry (atopic) skin** (decrease human B-defensin 3 predisposing patients to frequent skin infections).
- **T-Cell** (elevated Th2 cytokines and increased **IgE** production.
- 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.
- **Allergy,** increased tendency to certain allergens.

Age of onset:
- 60% first 2 months of life
- 30 % by age of 5
- 10% between age 6- 20 years (Improves in summer and flare in winter)
- More than 40% will remit completely during childhood, and another 40% will have only mild symptoms

Triggers:
- Allergy, increased tendency to certain allergens (Auto allergen)
- 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

  Dr. said "the best test is: if the parents suspect certain food make it worse, they stop it for two weeks then reintroduce it. If it improves and worsen then they stop it". Allergens (like food) may cause eczema, asthma or anaphylaxis.

Prevalence and association with other atopic disorders:
- Prevalence is almost 20% in US, representing a marked increase during the past several decades.
- 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**

*refers to the natural history or typical progression of **allergic** diseases that often begin early in life. These include **atopic** dermatitis (eczema), food allergy, **allergic** rhinitis (hay fever) and asthma.

- Asthma occurs in up to 50% of children who develop AD during the first 2 years of life.
- Allergic rhinitis develop 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 in rural areas, in smaller families, and in higher socioeconomic classes.
- Ultimately 80% of patients will develop increased IgE levels.

profilaggrin (FLG) and Atopic dermatitis:

- Loss of function mutations in profilaggrin (FLG) (filament aggregating protein) is a filament-associated protein that binds to keratin fibers in epithelial cells, cause 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 have been discovered in the European and Japanese populations, but all are strongly linked with AD, particularly of early onset.

Histology:

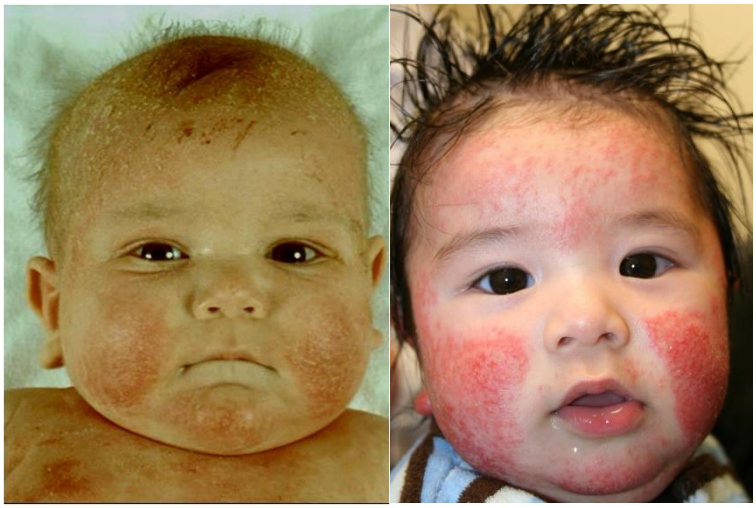
- Edema within the **epidermis (spongiosis)** and infiltration with lymphocytes and macrophages in the superficial dermis.

Clinical Variants:

- **Infantile AD**
- **Childhood AD**
- **Adult AD**

Infantile AD:

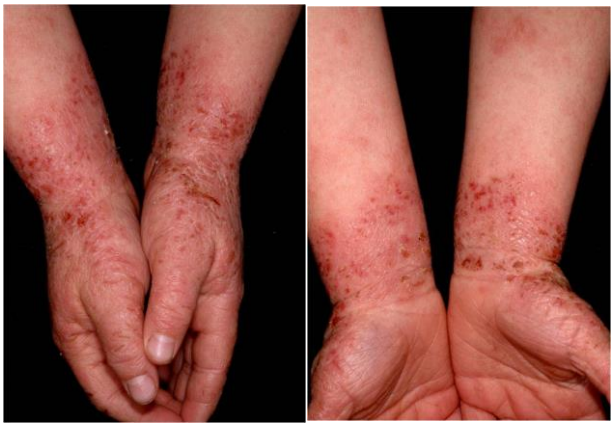
- 60% of case AD present in the first year of life, after 2 months of age
- Lesions in the face, scalp, neck, forehead, wrist, and extensors (Begin as itchy erythema of the cheeks)
- Present with itchiness, Red skin, tiny vesicles on “puffy “surface. Scaling exudate with wet crust and fissures. (subacute)
- Diaper area is usually spared in contrast to seborrheic dermatitis

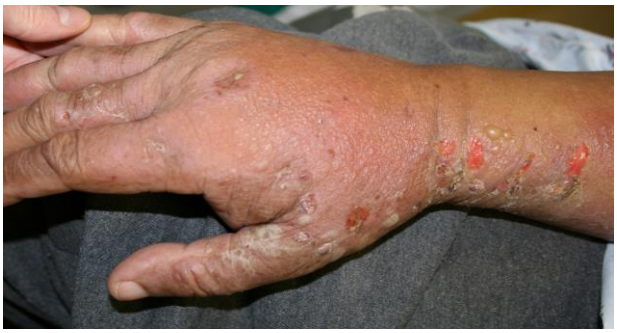


Involvement of the cheeks is characteristic of the infantile pattern of AD.

Childhood AD:

- Characterized by less acute lesions
- Distribution: antecubital and popliteal fossae, flexor wrist, eyelids, and face.
- Severe atopic dermatitis involving more than 50% of body surface area is associated with growth retardation.
- Eczematous plaque, early onset, history of atopy in the patient or the family and chronic course.

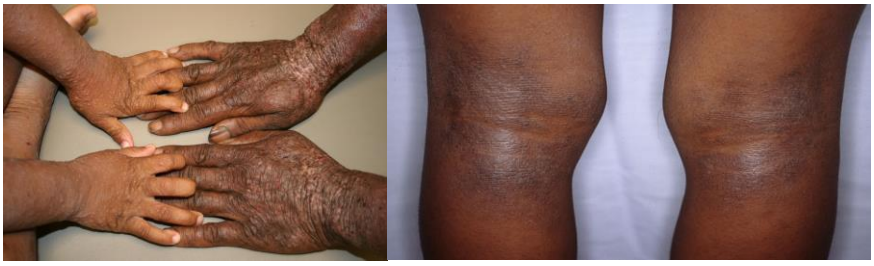




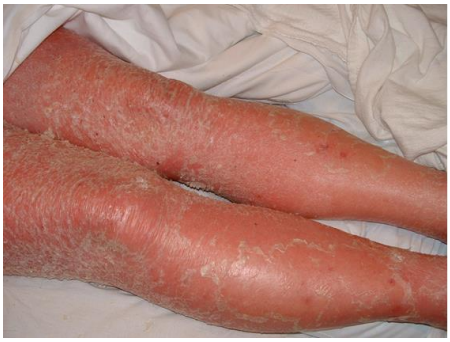
Vesicles can be seen here

Adult AD:

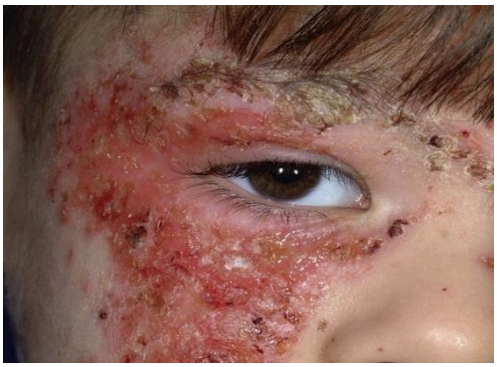
- Distribution: antecubital and popliteal fossae, the front side of the neck, the forehead, and area around the eyes.
- Atopic individuals are at greater risk of developing hand dermatitis than are the rest of the population
- 70% develop hand dermatitis sometimes in their lives.



Lichenification



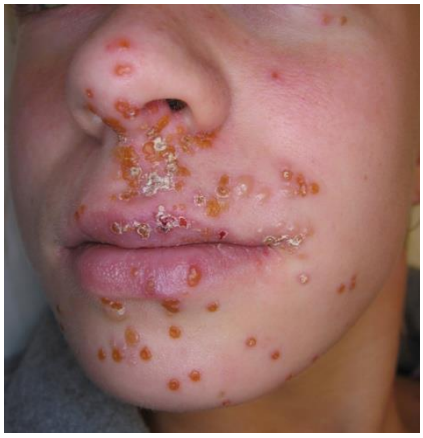
Erythroderma: is a very rare complication of atopic dermatitis



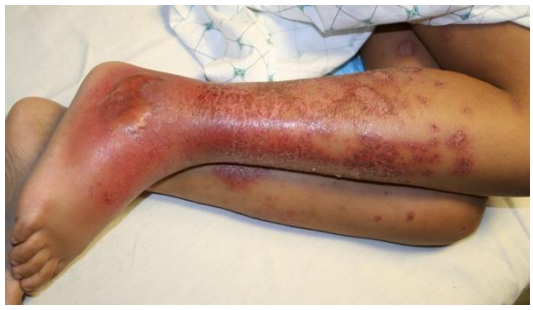
- Atopic individuals have a distinct tendency toward an extra line or groove of the lower eyelid, so called" **atopic pleat**", is present at birth or shortly after and usually retained throughout life, referred to as "Dennie-Morgan fold". (could present even without facial involvement)
- Another feature,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

Complications:

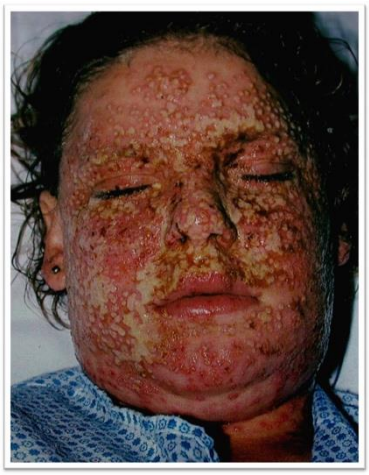
- Secondary infections: staph. aureus colonization (common)
- Eczema herpeticum
- Growth retardation (if the baby doesn't sleep well because of the itching he won't eat well)
- Psychological
- PIH (post inflammatory hyper or hypo pigmentation)

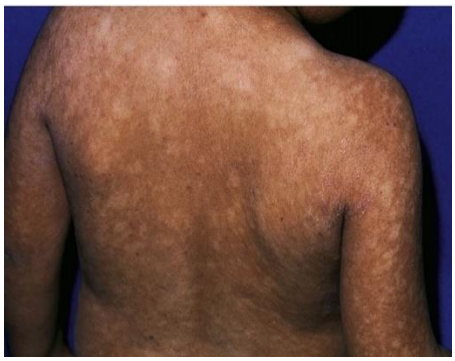


Impetigo: Bacterial infection



cellulitis





Post inflammatory Hypopigmentation

Eczema Herpiticum:

is a serious complication that needs admission and systemic antiviral

Investigations:

Diagnosis is clinical IMPORTANT

Criteria: EXTRA

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.

Management:

- **Education! Education! Education!** (Protect from scratching or stop soaps)
- Support!
- Skin care: **moisturizing the skin** (emollients)
- Topical therapy: (topical steroids, Tacrolimus, Pimecrolimus)

(Tacrolimus & Pimecrolimus are calcineurin inhibitor they don't have steroid side effect (steroid sparing agent)

- Phototherapy
- Systemic therapy: steroids, Cyclosporin, Methotrexate, Azathioprine (in severe cases systemic steroids and cyclosporin are used)

extra from 435:

First line:

- Topical corticosteroid
- Topical calcineurin inhibitor (tacrolimus "Protopic" & pimecrolimus) Doesn't have the side effect of topical steroid
- 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

AD and Food: has minor role

Steroids side effects:

- 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

● **Prognosis**:

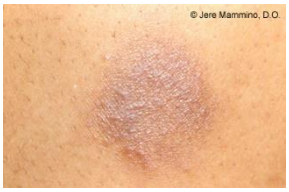
- Half of the cases improve by 2 years of age
- Most improve by teenage years
- <10% have lifelong problems
- 30-50% will develop BA or hay fever

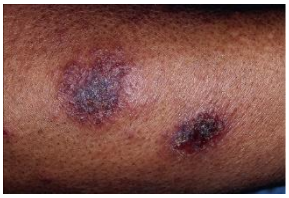
مب موجودة بالسلايدز لكن الدكتور قال لازم تعرفون انواع الستيزويدز حسب قوتها
الصورة هذي تشرحها بشكل كويس

Topical steroid class American classification	Topical steroid class British classification	Common representative topical steroids	Indications
I Superpotent corticosteroids	I Very potent	Clobetasol propionate 0.05% cream or ointment Halobetasol propionate 0.05% cream or ointment Betamethasone dipropionate 0.05% ointment	Alopecia areata Atopic dermatitis (resistant) Discoid lupus Hyperkeratotic eczema
II Potent corticosteroids	II Potent	Betamethasone dipropionate 0.05% cream Fluocinonide 0.05% ointment Halcinonide 0.1% cream Mometasone furoate 0.1% ointment	Lichen planus Lichen sclerosus (skin) Lichen simplex chronicus Nummular eczema
III Upper mid-strength corticosteroids		Betamethasone dipropionate 0.05% lotion Fluticasone propionate 0.005% ointment Triamcinolone acetonide 0.1% ointment Halometasone 0.05% cream	Psoriasis Severe hand eczema
IV Mid-strength corticosteroids		Fluocinolone acetonide 0.025% ointment Mometasone furoate 0.1% cream or lotion	Asteatotic eczema Atopic dermatitis
V Lower mid-strength corticosteroids	III Moderate	Betamethasone valerate 0.1% cream Fluocinolone acetonide 0.025% cream Fluticasone propionate 0.05% cream Hydrocortisone butyrate 0.1% cream	Lichen sclerosus (vulva) Nummular eczema Scabies (after scabicide) Seborrheic dermatitis Severe dermatitis Severe intertrigo (short-term) Stasis dermatitis
VI Mild corticosteroids		Alclometasone dipropionate 0.05% cream or ointment Desonide 0.05% cream Fluocinolone acetonide 0.01% cream Triamcinolone acetonide 0.025% cream	Dermatitis (diaper) Dermatitis (eyelids) Dermatitis (face) Intertrigo Perianal inflammation
VII Least potent corticosteroids	IV Mild	Hydrocortisone 1% or 2.5% cream, 1% or 2.5% lotion, or 1% 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

Nummular dermatitis:





Notice the surrounding xerosis

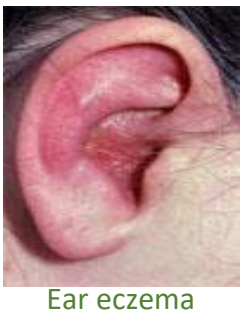
Definition: Coin shaped patches and plaques, Secondary to xerosis cutis, **Primary symptom itch**

Xerosis cutis: dry skin.

Regional eczema:

-Ear eczema:

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

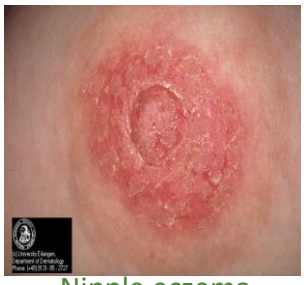


Ear eczema

-Eyelid dermatitis

-Nipple eczema:

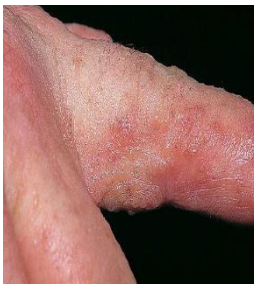
-Painful fissuring, seen **especially in nursing mothers**
-Maybe an isolated manifestation of atopic dermatitis
-If it persists more than 3 months, and/or unilateral, **biopsy is mandatory** to rule out Paget's disease

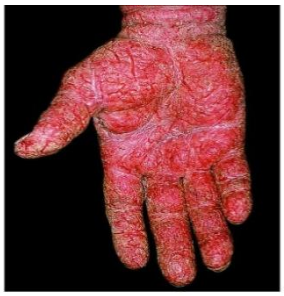


Nipple eczema

-Hand eczema:

-Spongiosis histologically
-Irritant hand dermatitis- seen in homemakers, nurses. Resulting from excessive exposure to soaps
-Pompholyx- tapioca vesicles, on sides of fingers, palms, and soles
-**Irritant** versus allergic



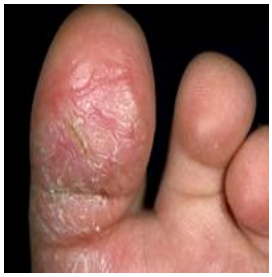


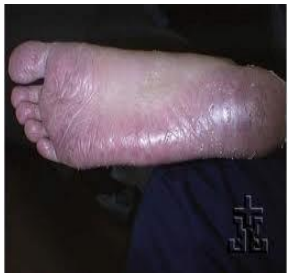
Hand eczema

-Diaper dermatitis

-Juvenile plantar dermatosis:

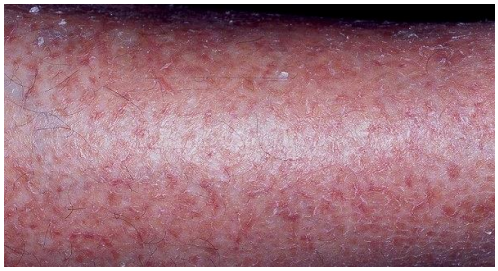
-Begins as a patchy symmetrical, smooth, red, **glazed macule** on the base of the great toes
-**Affect age 3 to puberty**.
-Symmetrical lesions on weight bearing area
-Virtually always resolve after puberty.





The center of the foot is spared (not weight bearing area)

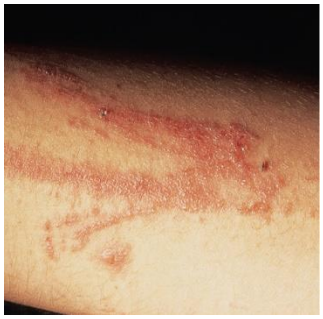
Xerotic eczema: (Asteatotic eczema)



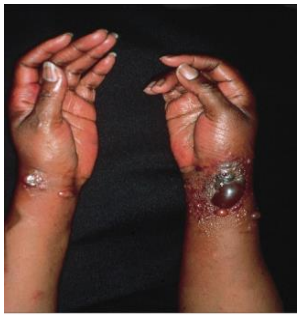
-Aka winter itch, nummular eczema, eczema craquele, and asteototic eczema.
-Anterior shins, extensor arms, and flank
-Due to decreased hydration >> usually seen in Elderly.
-Use of bath oils in bath water is recommended **to prevent water loss**
-Moisturizers – urea or lactic acid.

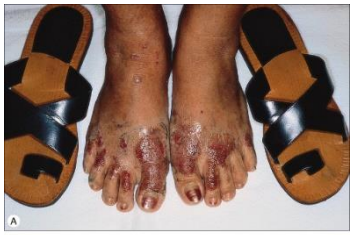
Allergic contact dermatitis:

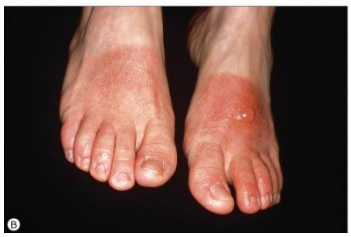
-Type 4 Hypersensitivity Response
-Classically well demarcated/patterned
-Exposure can be infrequent (once a month)
-**Patch testing** is gold standard for diagnosis



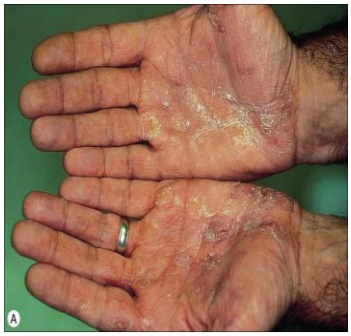
Poison Ivy/Oak/Sumac

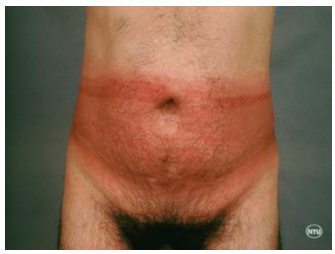


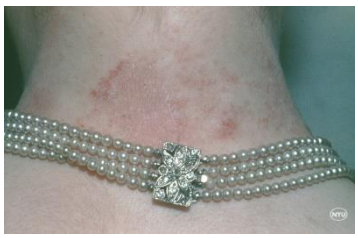


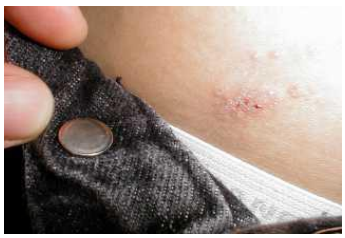


Potassium Dichromate in Leather









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

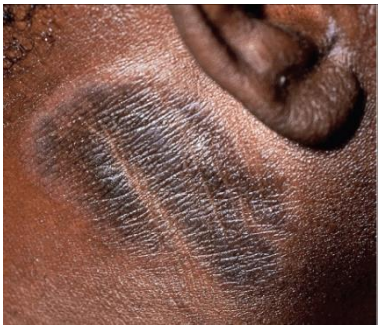
Irritant Contact Dermatitis:

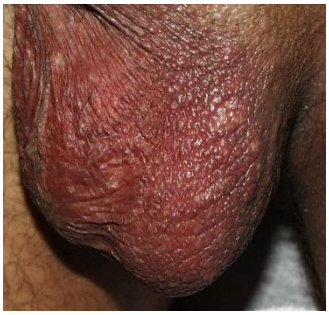
-Most contact dermatitis is irritant in nature
-It account for 80% of **occupational** skin diseases
-Irritant vs allergic
-**Prevention is key**!

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

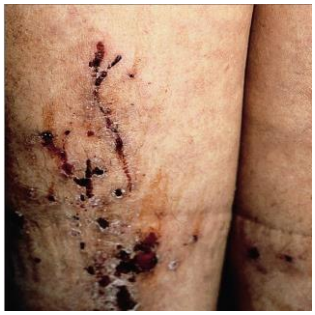
Neurodermatitis (Lichen Simplex Chronicus):

-Paroxysmal pruritus
-Habitual **excoriating or rubbing** They don't get relieve until they see bleeding.
-Can be triggered by stress and anxiety. Psychiatric disease (areas that are hard to reach is always symptomatic free) e.g. back.
-Skin thickens to defend
-Consider underlying disease





No fungus on the scrotum!



Prurigo simplex

Increased skin markings

Seborrheic Dermatitis:

-Distribution: Face, scalp, axillae, upper chest
-Pityrosprum ovale " malassezia furfur"
-Oily greasy skin
-Nasolabial folds