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

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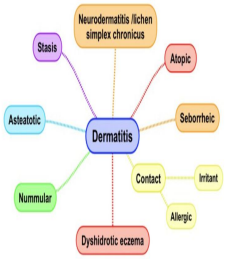
[Color index : Important | Notes | Extra]

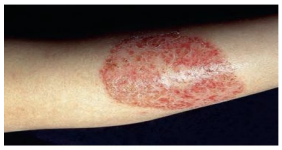
Eczema (Dermatitis)

Mind Map

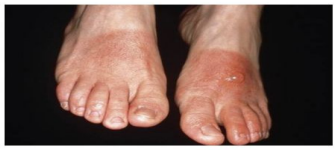


- **Definition:** A spectrum of inflammatory related disorders with pruritus being the hallmark of the disease (inflammation of the skin). **Phases** of eczema according to the onset:
- **Acute eczema:** erosion, oozing and vesicles. weepy, erythematous, edematous, pruritic, scaly plaques with superficial fissuring.
- **Subacute eczema:** Redness+ swelling, crust-+ scale +infection لو صارت فيه إنفكشن راح تتكون قشرة صفراء أو بنية = impetigo (honey-colored crust)
- **Chronic eczema:** lichenification, dark pigmentation and thick papules and plaques.gradual onset; hyperkeratotic, hyperpigmented, pruritic plaques. It has erythema and edema + presence of lichenification (secondary to scarring) + more thickening and dryness (acanthosis) >> so it is commonly misdiagnosed as psoriasis.Both of them might contain vesicles.

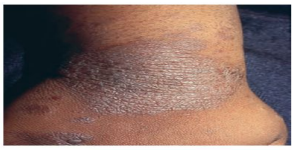
★ **In general lesions in eczema are ill defined**



Subacute



Acute Background erythema With vesicles.



lichenification with pigmentation (chronic)

It's ill defined

Types:

1- Atopic dermatitis

2- Seborrheic dermatitis

3- Contact dermatitis**:**

- Allergic
- Irritant

4- Dyshidrosiform dermatitis

5- Asteatotic dermatitis

6- Stasis dermatitis

7- Neurodermatitis (Lichen Simplex Chronicus)

1)ATOPIC DERMATITIS (AD):

- Chronic relapsing **itchy** skin disease in genetically predisposed patients. atopic لاتقولين أبداً itchy مو إشي!
- **Incidance:** up to 15-20 % in early childhood More in male
- **Associated diseases:** bronchial asthma, allergic rhinitis, allergic conjunctivitis(personal or family Hx) Associations NOT causes
- **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 (IMP) لو راحوا جدة ولا الشرقية بيتحسنون
- More than 40% will remit completely during childhood, and another 40% will have only mild symptoms.

- **Pathogenesis: (Multifactorial)**

- We can not say that atopic dermatitis is a hypersensitivity reaction, it's a mixed of type 4 (delayed hypersensitivity) and T-cells activation so we call this "Immunodysregulation". Contact dermatitis is Type 4 (pure type 4).

- **Defective barrier (skin).** Loss of moisture which makes them dehydrated. Mutation of a gene called (filaggrin) which is essential for the synthesis of skin barrier بالتالي مهما سووا يبقى الجلد ناشف نتيجة للطفرة في الجين
- **Immune dysregulation.**

- Both of these 2 factors results in increased skin colonization by S aureus leading to superantigenic infections.

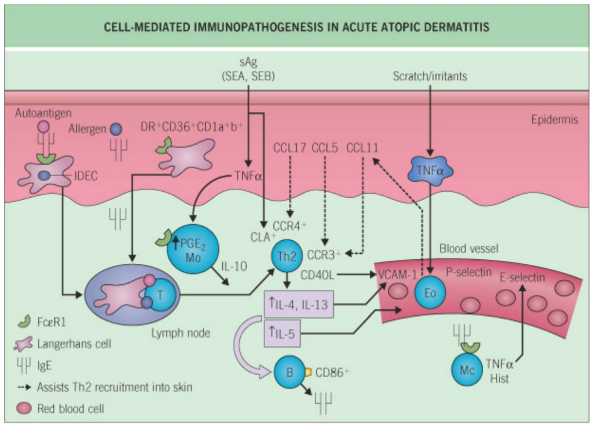
- **Atopy:** Genetic predisposition. It causes defect in skin barrier
- T cell activation
- IgE? (Epiphenomenon)

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 (eczema is not related to food vs urticaria which is type I and is related to food)

مب مطالبين بالتفاصيل بس اعرفوا القصة:

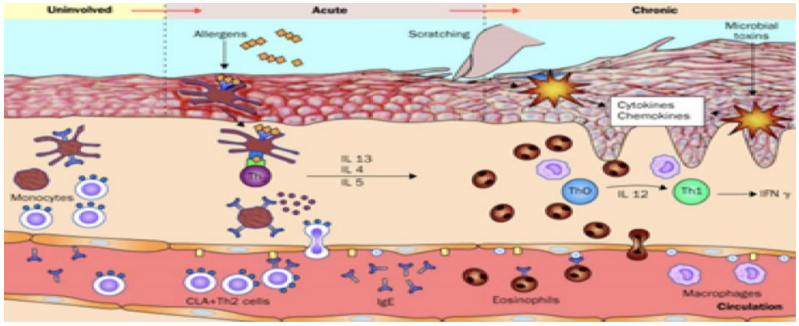
- For any immune reaction, there should be an antigen, either bacterial (staph aureus) or irritant كريم أو صوف أو عطر or autoantigen خلل من جوا سبب القصة هذي كلها.
- Dermis contains lymphatics مكان القصة
- Antigen enters > مين بيستقبله؟ > Antigen presenting cells (dendritic cells; langerhans cells)
- T cells في الوضع الطبيعي نايمة وماتدري وش يصير عليها > If activated it either gives Th1 or Th2 وتحدد بناءً على الانتجين اللي جاها > if intracellular virus (Th 1)/ if bacteria or allergen (Th2) اللي تعطينا IL-4, IL-13 and IL-5 "IL-5 = Eosinophils".
- The role of histamine is very limited. If you are going to prescribe an antihistamine, give sedative antihistamine. عشان ينام بدون حكة



1. Allergens are taken up by dendritic cells and presented to T cells.
2. In the absence of childhood microbial exposure, the balance between Th1 and Th2 cells is altered (activation).*
3. **TH2 cells encourage the production of IgE by B cells.**
4. Allergen-specific IgE then binds to the high-affinity receptor for IgE (FcepsilonRI) on mast cells.
5. Allergen exposure induces crosslinking of receptor-bound IgE with subsequent mast-cell degranulation and the release of pro-inflammatory molecules.
6. It is mixture of type I and type IV hypersensitivity reaction

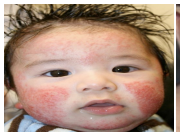
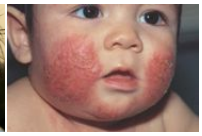
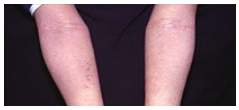
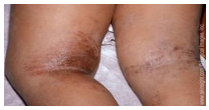
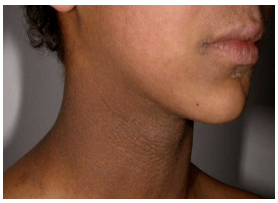
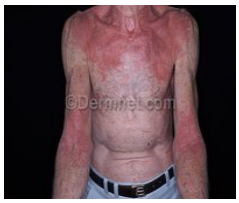
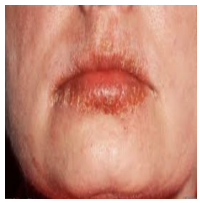
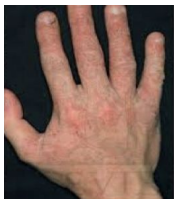
***We don't actually know the cause of the disease but there are 2 theories:**

1) People with AD have a problem in their skin development (caused by a gene variation), which makes them more susceptible to allergies
2) Children have more microbial exposure, so their immune system is familiar with foreign antigens ,but when they get older they start taking better care of their hygiene which causes their immune system to forget self and non-self antigens which makes it attack parts of the skin causing AD.



- مع الوقت ممكن Th2 تتحول إلى Th1، الجهاز المناعي بعد فترة يستوعب إن عنده كثير Th2 فيقرر يحول لـ ١ بالتالي تزيد سماكة الجلد عشان كذا يلخبطون بين الاكزيما المزمنة و الصدفية (psoriasis is Th1) لأن كلهم الجلد فيهم سميك وبرضو Th1

- **Clinical variants:**

1-Infantile atopic dermatitis:	- Lesions in the face and extensors. - 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  
2-Childhood atopic dermatitis:	Lesions in the wrist , ankle and Flexors.especially on Antecubital, popliteal fossae, neck and face.May be generalized Papular, lichenified plaques, erosions, crusts.  
3-Adult atopic dermatitis:	- Mostly flexural, face and neck. May be generalized - Lichenification and excoriations - lesions are either discoid or regional (except for a minority of people who may have diffuse reaction).Most common regions are flexors and hands.     

- **Diagnosis:** IMPORTANT
- It's mainly clinical (skin biopsy and labs are not required).
- **Pruritus** MUST be present.
- Males doctor mentioned and focused on the following elements, which are slightly different than those in the following table.
  - Distribution by Hx
  - Skin dryness
  - Family Hx of atopy
  - Onset under the age of 2
  - Distribution by PE

The old criteria: الدكتورة قرأتها كاملة

DIAGNOSTIC FEATURES OF ATOPIC DERMATITIS

Major features (3 of 4 present)

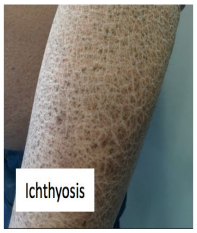
- Pruritus
- Typical morphology and distribution of skin lesions
- Chronic or chronically relapsing dermatitis
- Personal or family history of atopy

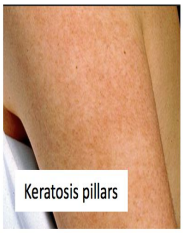
Should be present
If you have 3 out of 4 , you can directly diagnose without looking for the minor criteria

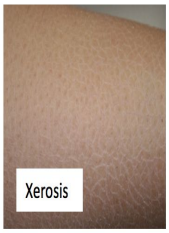
Minor features (3 of 23 present)

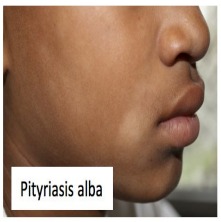
- Xerosis Dry skin
- Ichthyosis/palmar hyperlinearity/keratosis pilaris
- Immediate (type I) skin test reactivity
- Elevated serum IgE
- Early age of onset
- Tendency towards cutaneous infections/impaired cell-mediated immunity
- Tendency towards non-specific hand or foot dermatitis
- Nipple eczema
- Cheilitis inflamed lips
- Recurrent conjunctivitis
- Dennie–Morgan infraorbital fold secondary creases in the skin below the lower eyelids
- Keratoconus القرنية المخروطية
- Anterior subcapsular cataract
- Orbital darkening
- Facial pallor/erythema
- Pityriasis alba النخالة البيضاء، بقع بيضاء خفيفة
- 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

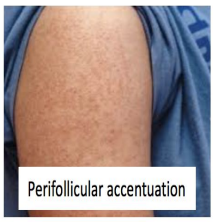
- Ichthyosis: جلد السمكة
- Keratosis pilaris or "chicken skin" is a common skin condition that causes patches of rough-feeling bumps. تقرن الجلد/خشونة/حبيبات











The new revised criteria:

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

a. Must have:
- Pruritus

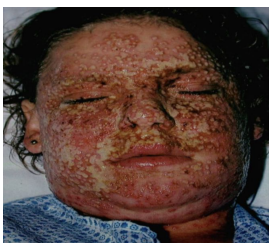
b. Plus 3 or more of the following:
- History of involvement of skin creases (front of elbows, back of knees, front of ankles, neck, around the eyes)
- History of a generally dry skin in the past year
- Personal history of asthma or hay fever
- Onset under the age of 2 years
- Visible flexural dermatitis

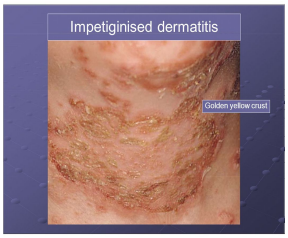
The diagnosis of atopic dermatitis in adults is primarily clinical; special investigations only contribute in identifying external aggravating factors.

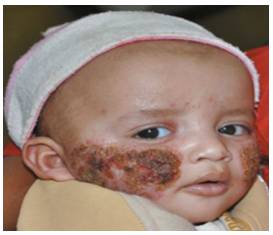
- **Complications:**

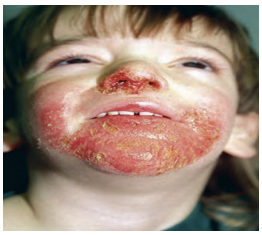
Eczema increases the risk of superinfection, especially to the skin > infection on top of eczema (unilateral).

- **Secondary infections (Impetiginized dermatitis)**: Caused by S. aureus.dermatitis with honey colored crusts. The child will have fever and you have to treat the infection first and then treat the dermatitis.
- **Eczema herpeticum**: Caused by herpes simplex virus 1 infection (Very serious, needs to be admitted and treated with IV antiviral; usually associated with severe burning sensation)
- **Mollusca Contagiosa**: Caused by Mollusca Contagiosum virus; causes chronic itching.
- Growth retardation. (Deteriorated school performance)
- Psychological
- Urticaria.
- Contact dermatitis.
- Cataract.
- Keratoconus.
- Keratoconjunctivitis.



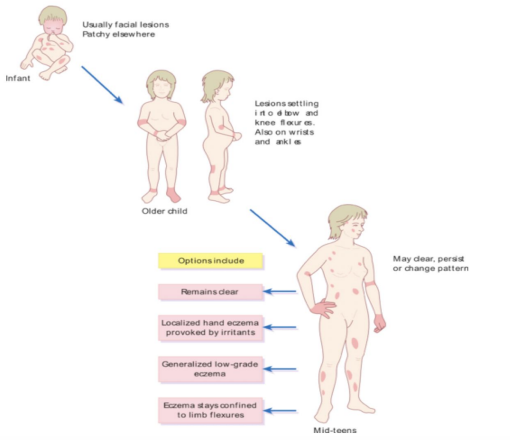






Pic (Lt) : eczema herpticum, it is an emergency you have to treat it immediately
3 next pics: secondary infection caused by staph aureus (you can see the crusts)

- **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



- **Management:**
  - Education نقول للأهل إن المرض مناعي، غير معدي، الجلد بيكون ناشف ويحتاج ترطيب دائم
  - Emollient (moisturization)
  - Psychological support
  - Avoid irritant:
    - Avoid alkali soaps and use non-alkaline soaps.
    - Avoid woolen clothes and wear cotton instead.
  - **Active Treatment:** symptomatic treatment (vs urticaria in which we treat the cause)

First line :

- Topical corticosteroid Potent steroid : for thick skin e.g palms and soles. Mild steroid : for thin skin and face
- **Topical calcineurin inhibitor (tacrolimus** "Protopic" **& pimecrolimus)** Doesn't have the side effect of topical steroid
- Oral H1 antihistamine For sedation effect only
- Oral antibiotic For secondary bacterial infection

Second line

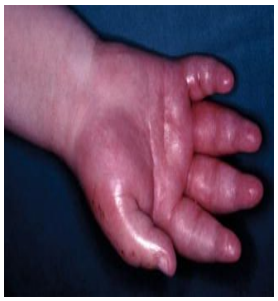
- Systemic Steroids If topical treatment didn't work or the disease is widely diffuse
- Phototherapy (NBUVB , PUVA) علاج ضوئي وليس أشعة
- Immunosuppressive therapy (cyclosporine , methotrexate، azathioprine)

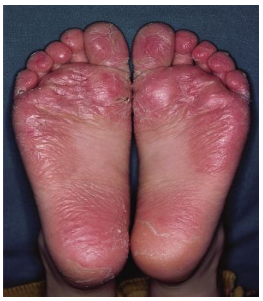
*Localized disease : topical/ diffuse disease: systemic therapy / more diffuse : phototherapy

Juvenile plantar dermatosis:

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

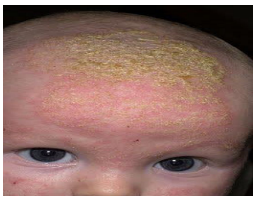
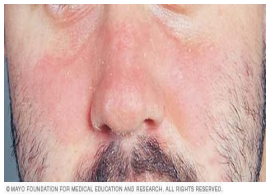
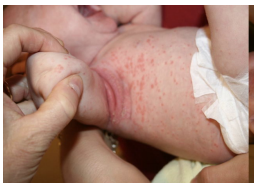
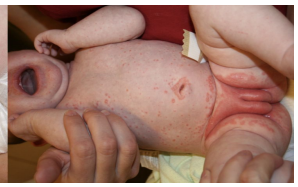
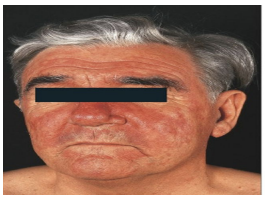
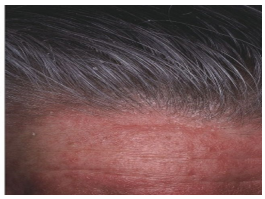
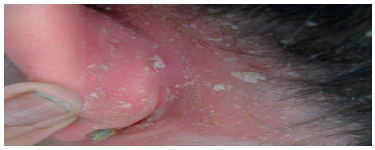
The center of the foot is spared (not weight bearing area)





2)SEBORRHEIC DERMATITIS:

It's a chronic dermatitis that often affects male between the ages of 20-50 years. Features are: it is more **Scaly**, **Yellowish**, and **Oily**. More in areas rich in sebaceous glands **e.g.:** scalp, face, ears, parasternal region & flexural areas (axillae, inguinal & inframammary folds, umbilicus). classic location is scalp, Craniofacial area and skin folds. Seborrheic = chronic = scales

Causes:

1. Hereditary Seborrheic status. بشرة دهنية
2. Pityrosporum ovale (Maalassezia furfur)over growth. It's a theory
3. Nutritional deficiencies (causes SD-like rash)
4. Tendency.

Common associated with: العلاقة مو سببية فقط ملاحظة متكررة

- Parkinson disease.
- Facial paralysis تجي لمريض مشلول نص وجه في النص المشلول بالضبط
- HIV and immune suppression. Severe eruptive lesions with acute onset. Think about it if you see a young patient with severe presentation especially if with risky behavior.

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

Distribution:

- Hairy are of head, cradle cap
- Face: forehead, nasolabial folds , glabella and eyebrows.
- Trunk: DDx: PR vs pityriasis versicolor
- Body folds: axillae, groins, anogenital area, submammary areas, umbilicus and diaper area (Difference from atopic eczema: in diaper area, oily, yellow scales) (infants)--- sharply marginated erythematous eruption, erosions and fissures
- Genitalia: with yellow crust and psoriasiform lesions.

Cradle cap: is coherent scaly & crusty mass covering most of the scalp & can be seen in infantile seborrheic dermatitis.

Treatment

Scalp:	**Skin:**
- Zinc pyrithione Shampoo - Selenium sulfide 2.5% shampoo - 2% ketoconazole shampoo - Low – potency glucocorticoid solution, lotion or gels	- Topical: antifungals, glucocorticoid, pimecrolimus - Combined therapy - Maintenance & recurrence

3) CONTACT DERMATITIS:

Dermatitis precipitated by an **exogenous** agent/external material:

1. **Allergic** contact dermatitis.(type 4)
2. **Irritant** contact dermatitis.(cytotoxic)

◆ A) Contact allergic dermatitis:

- It accounts for **7%** of occupation related diseases.
- It is caused by an allergen that triggers **type IV hypersensitivity** reaction in a presensitized person. It occurs after an exposure to a topical substance in **sensitized individuals** (requires induction "**previous exposure**" and elicitation phase 'lag time to reaction'). First exposure does not cause a reaction
- Begins 24 h after subsequent exposure if already

Common allergens eliciting contact dermatitis:

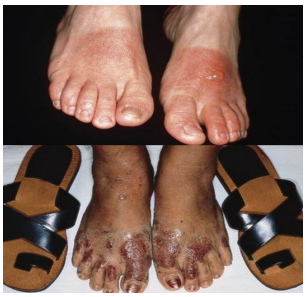
- **Nickel** (affects 10% of women and 1% of men), **why?** Because it is found in accessories perfumes, fragrances, preservatives.
- **Poison Ivy** : a plant
- **Potassium Dichromate** : in Leather
- Rubber,hair dyes, preservatives,latex, cosmetics, topical Abx, topical cs

★ **Acute form** presents with crusted erythematous papules, vesicles & bullae that is **well demarcated & localized to the site of contact** with the allergen. It can be **diffuse** as well.

★ **Shoe dermatitis:**

Causes:

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



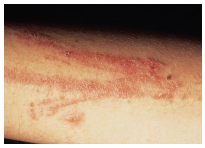
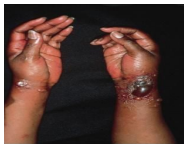
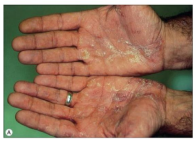
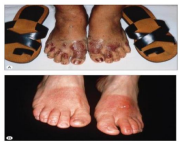
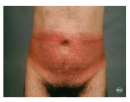
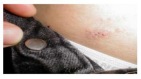
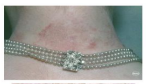
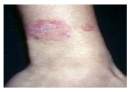
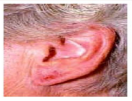
Diagnosis:

- History.
- Examination.
- PATCH testing remain the gold standard for accurate diagnosis**. (read at 48, 96 h)**

Treatment of ACD:

- Identification removal of causes.
- for allergic contact dermatitis not for irritant
  - **Avoidance allergens**
  - Topical corticosteroids

Poison Ivy | **Potassium Dichromate** | **Nickel Allergy**
most common
(belt buckle- bracelets)

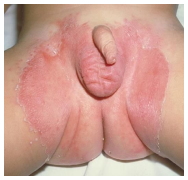
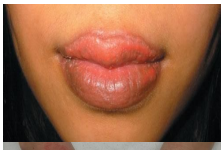
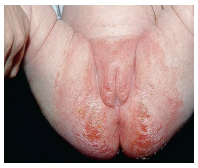
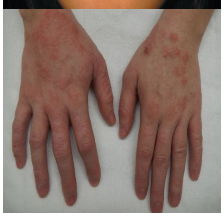










B) Contact irritant dermatitis:

It account for **80%** of **occupational** skin diseases.

Is **localized** **non-immunologically** mediated inflammatory reaction.happens at 1st exposure

- ICD results from **direct cytotoxic effect** "localized" single or repeated application of a chemical substance to the skin.
- Common causes: Hands repeatedly exposed to water, cleansers
  Lip-licking habit – wetting and drying caused by saliva,Napkin dermatitis.

Most common irritants are:

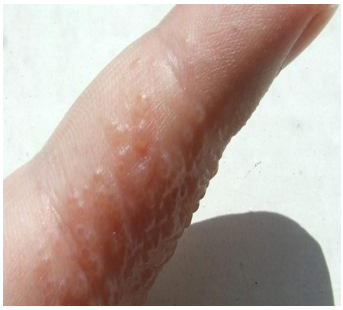
- Water
- Abrasives
- Chemicals, e.g. acids and alkalis
- Solvents and detergents

- **Diaper Dermatitis(Napkin dermatitis)** : due to stool enzymes that cause irritation >> manage it with frequent diaper changing and barriers like zinc oxide.
- **Lip licker dermatitis** : caused by lip licking, Saliva contain enzymes as irritant. Manage by avoid licking and topical steroid. Can be caused by eating citrus foods.
- **Housewife dermatitis** : from the soap that is used in washing dishes. (note that chemicals can lead to atopic, allergic contact, or irritant contact dermatitis)

Difference between allergic and irritant CD:

Contact allergic dermatitis	Contact irritant dermatitis
Type IV hypersensitivity	Direct cytotoxicity (tend to be painful more than pruritic)
In susceptible people	Can happen to anyone
Can extend beyond the area of contact (localized but can be diffuse or in distant site)	**Can never extend** beyond the area of contact (localized)
Can't occur from the first exposure	Can occur from the **1st exposure**
doesn't require significant amount	Requires significant amount
Gradual eczema. Takes 4-5 days "delayed-type hypersensitivity" for the classic presentation to appear	acute flare reaction (1-2 days) then severe hyperpigmentation

◆ Dyshidrosiform eczema:



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

Not considered as a separate disease, Can be associated with atopy, of patients with dyshidrosis, 50% have atopic dermatitis.

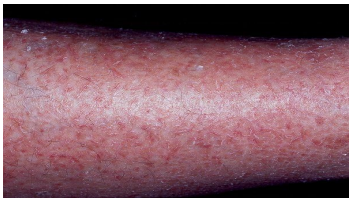
Exogenous factors (e.g., contact dermatitis to nickel, chemicals) also play a role. Affect hands & feet. -It could be a separate disease or part of atopy. Common in ladies who clean with water and detergents without protection.

Treatment:

-Avoidance of triggering factor.

-topical steroid.

◆ Asteatotic eczema (xerotic dermatitis):

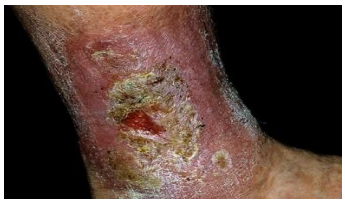


Aka winter itch, nummular eczema, eczema crackle, and asteatotic eczema. Anterior shins, extensor arms, and flank.

Due to decreased hydration >> usually seen in **Elderly**.

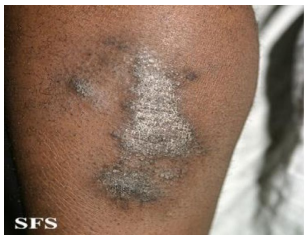
Use of bath oils in bath water is recommended to prevent water loss Moisturizers – urea or lactic acid. Bricks appearance.

◆ Stasis eczema[1]:



Seen in patient with signs of venous hypertension like chronic venous insufficiency, varicose vein. We have to roll out vascular disease can be complicated by superimposed allergic contact dermatitis. Common presentation is a bedridden patient who have venous ulcers, after treating the ulcer he may end up with allergic contact dermatitis.

◆ Lichen simplex Chronicus[2] (Neurodermatitis):



- Dermatitis which results from repeated rubbing & scratching of the skin. They don't get relieve until they see bleeding.
- Chronic itching and scratching can cause the skin to thicken and have a leather texture with exaggeration of skin marking. A scratch-itch cycle occurs which is difficult to break. Can be triggered by stress and anxiety. Psychiatric disease (areas that are hard to reach is always symptomatic free) e.g. back. Occur commonly in atopic patient. It's a secondary lesion that develop due to scratching.

Summary:

Distinctive morphological features of different forms of dermatitis

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

[1] It's common location is at lower lateral third of lower limb u can see ulcers and that will lead to infection.

[2] Because it is psychological it is in reachable area unilateral and it is induced and localized