Eczema? Psoriasis? Or else?

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Focused Practice Dermatology

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Declaration of Conflicts of Interest

- This talk has no grants/research support
- I have no honoria or consulting fees
- I have no patents in relation to the topic or material presented
- I receive no funding or support for mentioning a brand name which is done simple for illustration purpose
- I am not a FRCPC Dermatologist!

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Summary of presentation

- Eczema—etiology and clinical presentation
- Eczema—dermatoscopic features and differentials
- Eczema—treatment and prognosis
- Psoriasis—etiology and clinical presentation
- Psoriasis—dermatoscopic features and differentials
- Psoriasis—treatment and prognosis
- Or Else?
- When to refer?
- To steroid or not to steroid?

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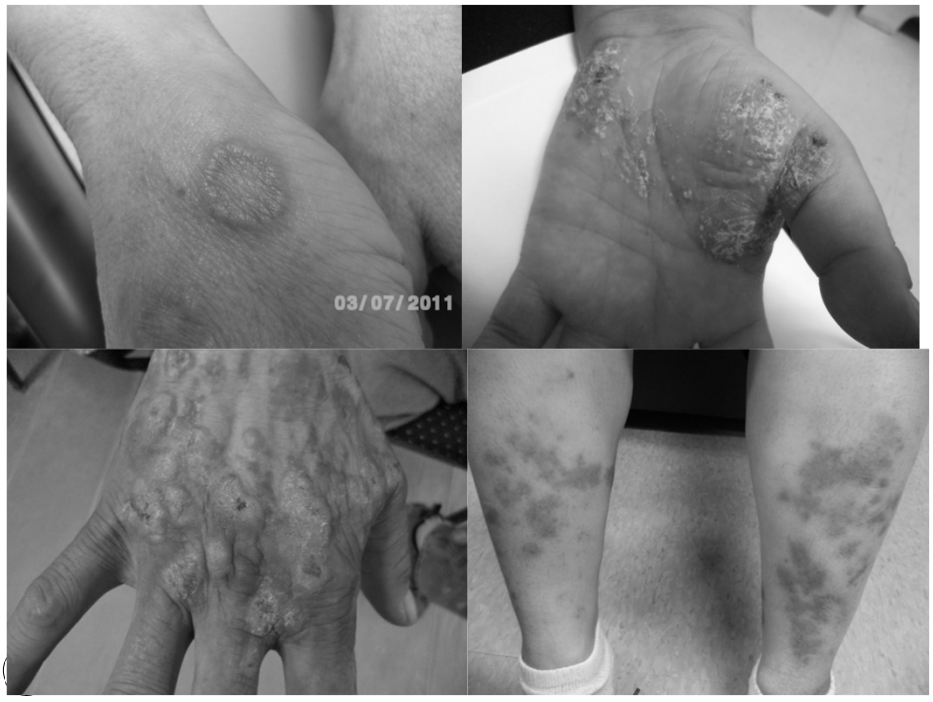

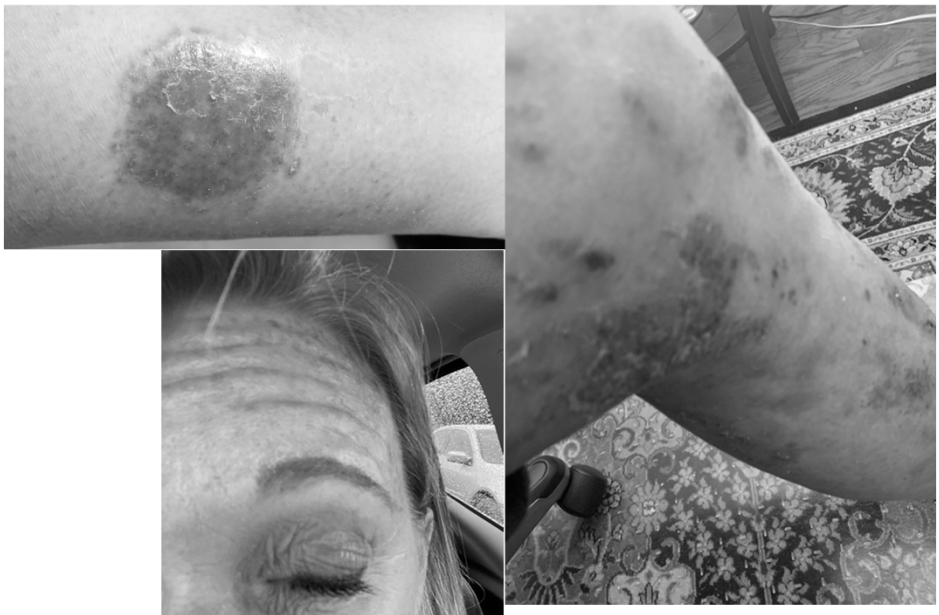

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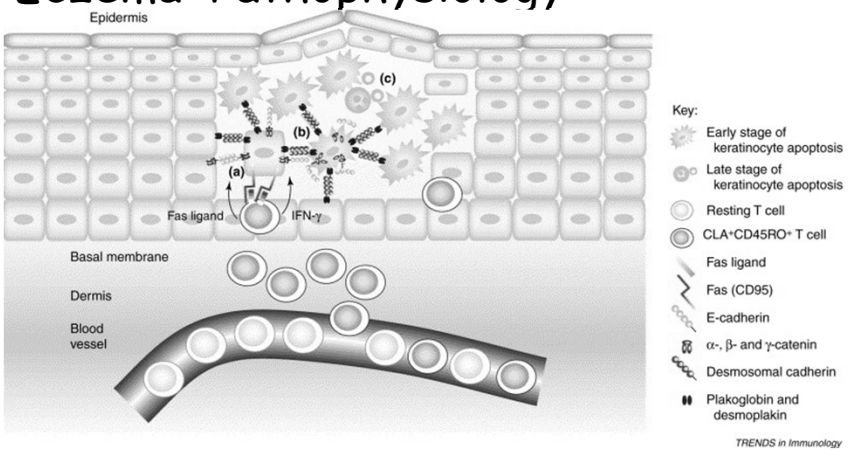
Eczema: Pathophysiology



- Allergens/irritants→activate T-cell immunity→inflammatory cytokines→apoptosis and spongiosis of keratinoctyes + loss of desmosomes→ itch + weeping + blistering

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Eczema: classification

- By origin: endogenous (i.e., atopic dermatitis) Vs exogenous (i.e., contact dermatitis)
- By anatomical sites: face/hand/trunk/eyelid
- By pattern: flexural Vs extensor surface (reverse eczema)
- By duration: acute Vs chronic
- By appearance : eczema craquelé, nummular (discoid) eczema, lichenified, nodularis

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Assessment of eczema

- BSA : body surface area covered in%
- EASI score (0-72): Eczema Area and Severity Index: assess body/UL/LL/HN for thickness, redness, scratching, lichenification and % area affected; mild < 7, severe > 21
- SCORAD (0-103) : 60% to intensity, 20% extent, 20% subjective feeling. Mild < 25, severe > 50
- IgE levels : ↑↑ (>10,000) associated with persistent widespread eczema
- DLQI : Dermatology Life Quality Index (0-30): measures health-related QoL, not specific to eczema.

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Diagnosis of Eczema

- History of atopy (other 2 of the triad) or contact with exogenous agents
- *Symptoms* : itch, scratch, forming a vicious itch-scratch cycle
- *Distribution of rash* : typical (atopy), print-impression of allergens (contact dermatitis)
- *Morphology* : erythematous, maculopapular +/- vesicular, margins not well defined, excoriation, +/- exudes, +/- Staph infections
- *Beware of* : unusual distribution, failure to treatment, seeking disability, weird beliefs ←-dermatitis artefacta

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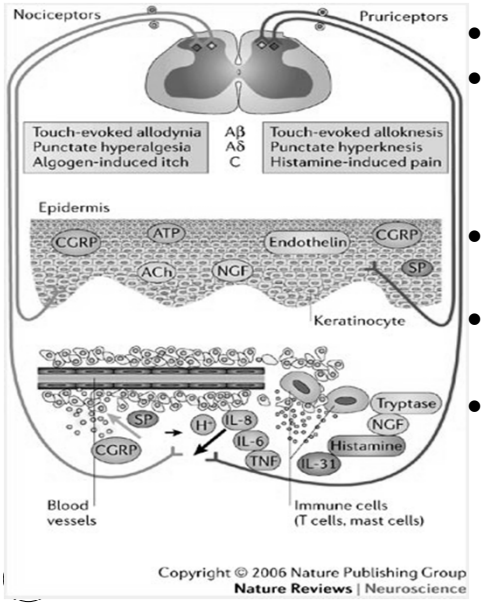
Pathophysiology of skin itch



- Itch is a mild form of pain
- At least 2 sets of afferent fibers (A-δ, A-β and C) serving the pain receptors and the itch receptors
- Both sets converge at the spinal cord
- Mediators involved: SP, Histamine, ILs, ACh, ATP
- Acute itch ≠ chronic itch (sensitisation)

Akihiko Ikoma et al., The Neurobiology of Itch. Reviews Neuroscience 7, 535-547 (July 2006)

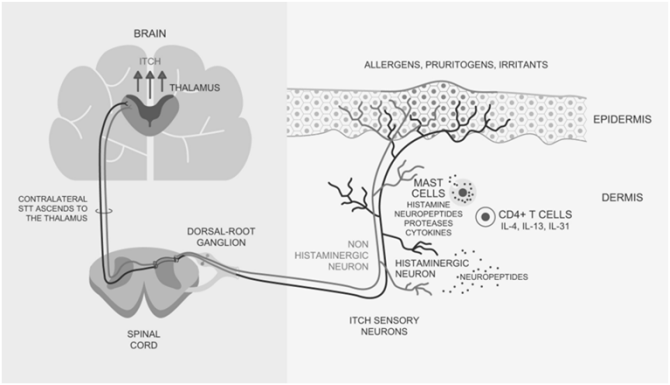


FIGURE 1. Initiation of itch. Allergens, pruritogens, and irritants are exogenous substances which interface with the skin in acute and chronic itch. Branching terminal fibers of afferent neurons which sense these substances reach the epidermis, the uppermost viable layer of skin immediately below the stratum corneum barrier. The sensory neurons are considered histaminergic or non-histaminergic. Neural activity drives the recruitment of immune cells, including mast cells and CD4+ T cells among others. These cells release mediators that activate cognate receptors on sensory neurons to release neuropeptides to contribute to the itch-scratch cycle. Messages are relayed from the peripheral afferents to their cell bodies in dorsal root or trigeminal ganglia followed by synapsing with second-order neurons in the spinal cord. The thalamus then assists in the interpretation of messages encoding itch. IL, interleukin; STT, spinothalamic tract.

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Treatment of eczema

- Identification: triggers and allergens (skin tests, food journal/ exposure diary)
- Avoidance: trial of removal
- Emollients: not only for asteatotic eczema, atopic skins tend to be dry and sensitive
  - Water based with ceramide : CeraVe, Curel
  - Oil based: olive oil, pumpkin seed oil, sesame oil
  - Petroleum based: Vaseline, Aqufour, CeraVe Oint

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Treatment of eczema

- Calcineurin inhibitor: Tacrolimus 0.03% and 0.1% (Protopic©) and Pimecrolimus 1% (Elidel©)
- phosphodiesterase-4 (PDE-4) inhibitors (Eucrisa ©)
- Steroids:
  - Mild (Class 3)→0.5% (non-prescription) and 1% hydrocortisone cream/ointment (Cortate)
  - Moderate (Class 2)→betamethasone valerate 0.1% (Betnovate), Fluocinolone 0.025% (Synalar), mometasone 0.1% (Elocon), clobetasone 0.05% (Eumovate)
  - Potent (Class 1)→clobetasol 0.05% (Dermovate)

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Treatment of eczema

- Phototherapy
- Systemic Immunomodulators
  - Methotrexate—qweekly + folic acid rescue, need to do LFTs and CBC regularly
  - Cyclosporine—
  - Azathioprine
- Biologics: monoclonal antibodies blocking IL-4 (e.g., Dupilumab—Dupixent©) and IL-13 (Tralokinumab—Adtralza©) ctyokines pathway

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Complications of eczema

- 2nd bacterial infections—MSSA/MRSA (MRSA extremely prevalent in James Bay Area)
- Thickening/fissuring of skin—lichenification
- Hyper- or hypo-pigmentation (Pityriasis alba)
- Low esteem / social isolation / school problems
- Risks towards depression (OR 1.27) , anxiety (OR 2.19) and suicide (OR 1.36)

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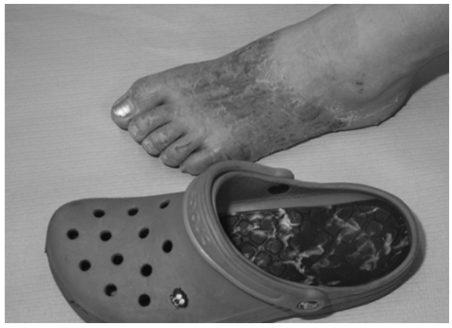
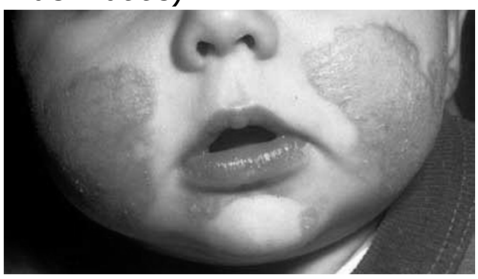
Eczema?



Exogenous (contact dermatitis)

Endogenous (Atopic dermatitis)



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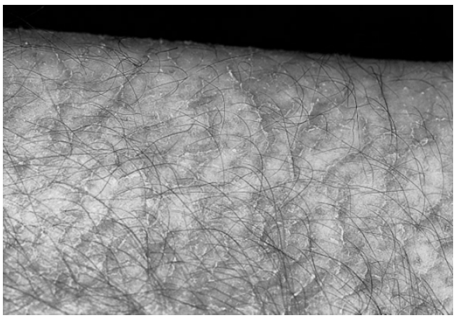
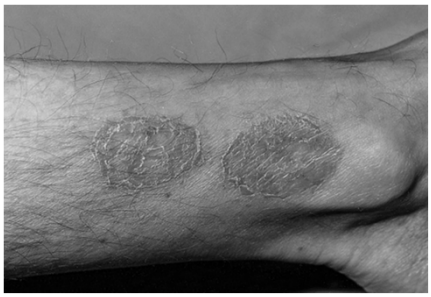
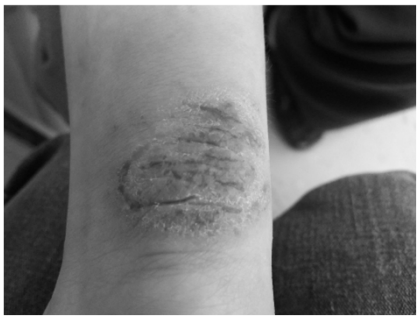
Eczema ?



Asteatotic eczema,
aka eczema craquele

Nummular/Discoid eczema





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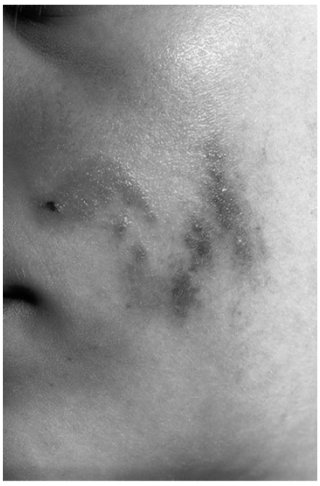
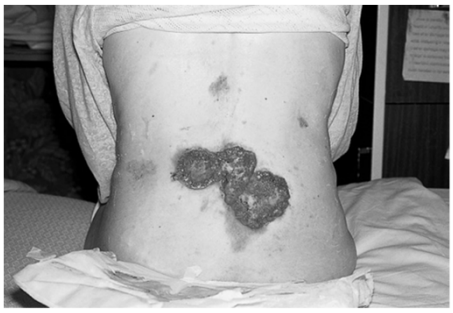
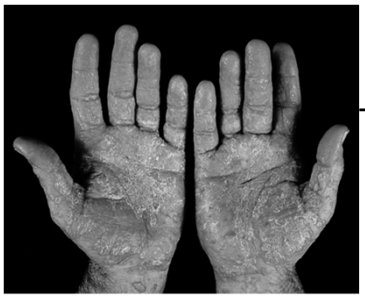
Eczema ?



Dermatitis artefacta



Basal cell carcinoma



Tinea manuum

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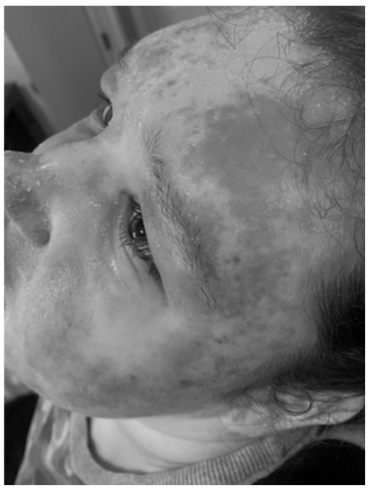
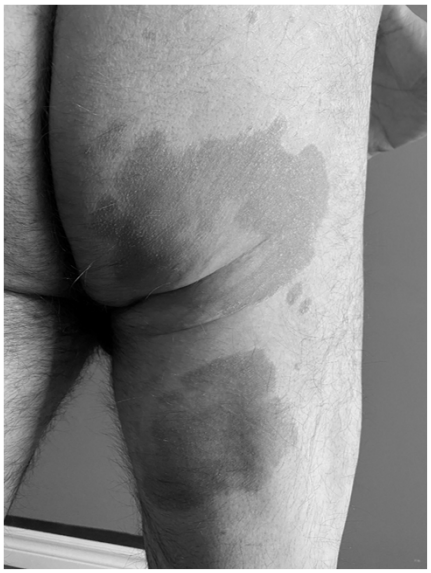
Eczema ?



Psoriasis



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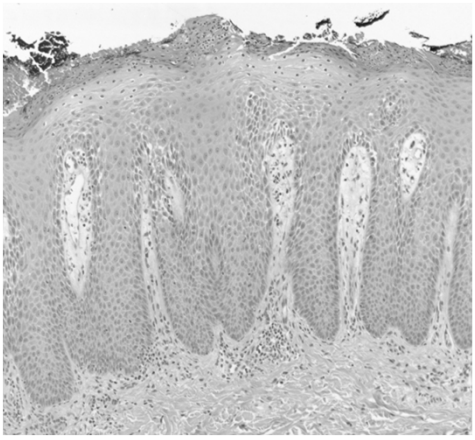
Pathophysiology of Psoriasis

- Hyper-proliferation of keratinocytes
- reduced epidermal cell transit time - from 30 days to 2-3 days (7 x faster)
- infiltration of neutrophils and activated lymphocytes
- Munro's micro-abscesses→pustular form



Source: DermNetNZ

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Etiology of psoriasis (I)

Seems to be multifactorial:

- genetic:
  - 30% of patients having one first degree relative with psoriasis
  - strong HLA associations (HLA CW6)
- infections:
  - Streptococcal pharyngitis – classic trigger for guttate psoriasis in children and young adults: onset 10-14 days
  - AIDS/HIV

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Etiology of psoriasis (II)

- stress, e.g. moving house; examinations
- trauma:
  - Koebner phenomenon – not exclusive to psoriasis
  - near scars or sites of trauma
  - drugs:
    - alcohol
    - beta blockers
    - non-steroidal anti-inflammatory drugs
    - Anti-malarials, e.g. chloroquine and mepacrine
- COVID!

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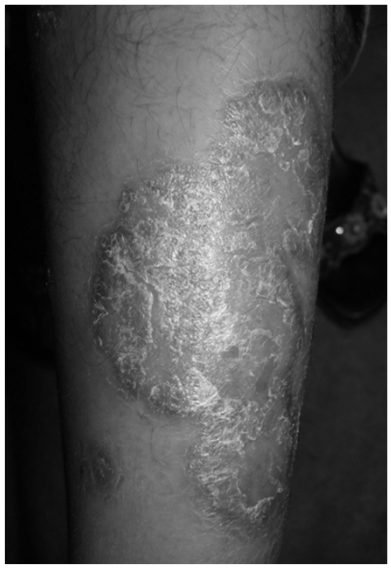
Diagnosis of Psoriasis

- Lesions with well defined edges→guttate, discoid, plaque, erythrodermic
- Prolific desquamation→silvery scales
- Auspitz sign
- Koebner phenomenon
- Itch? (often mild)



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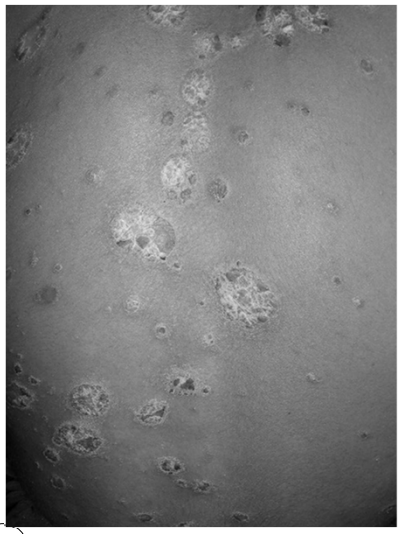
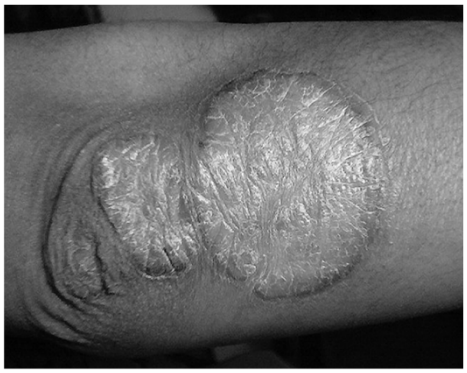
Psoriasis (plaque form)





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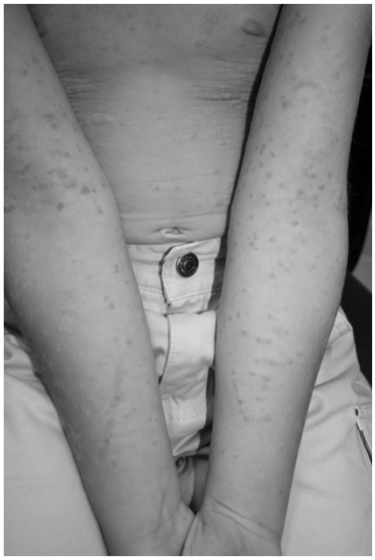
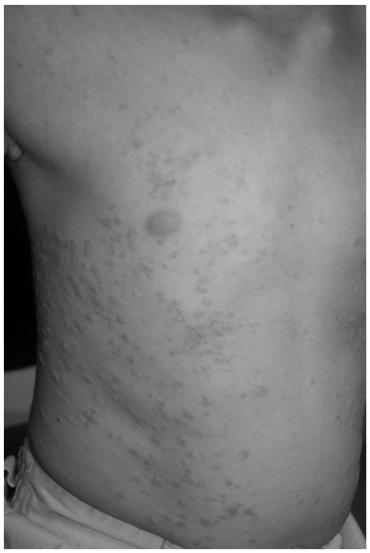
Psoriasis (guttate form)





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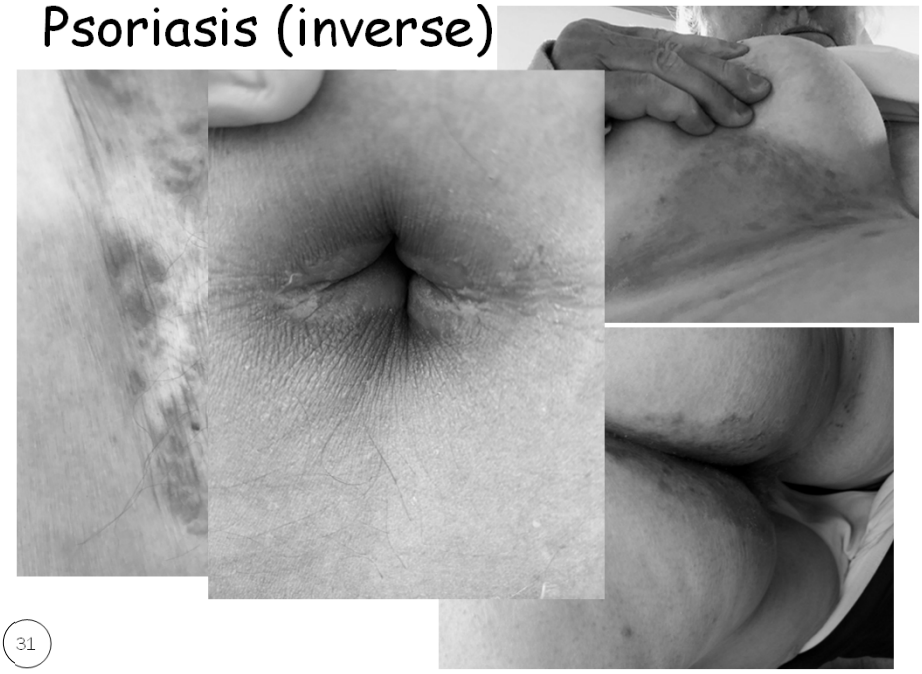
Psoriasis (inverse)



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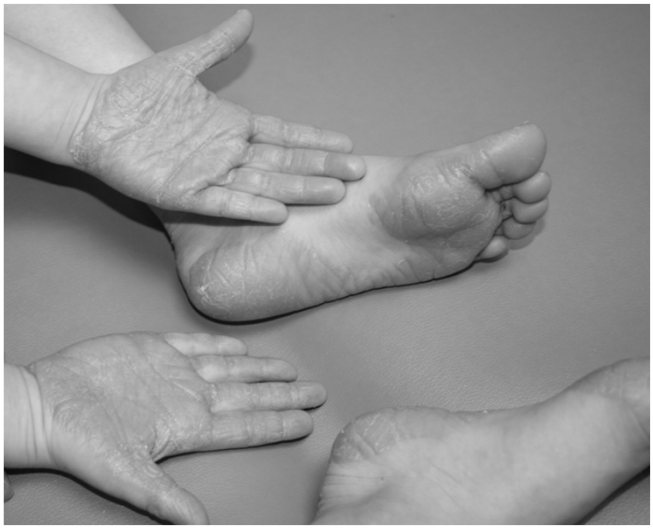
Palmo-plantar psoriasis



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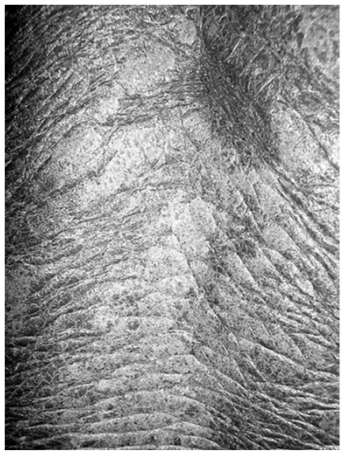
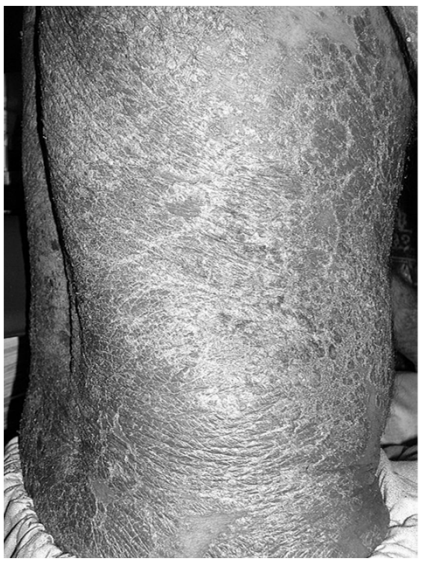
Erythrodermic Psoriasis





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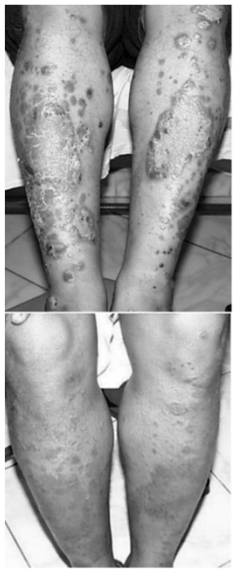
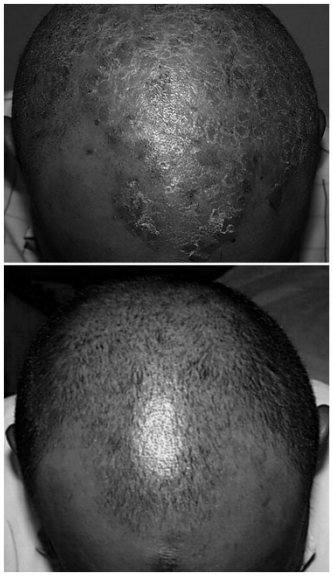
Psoriasis (with treatment)





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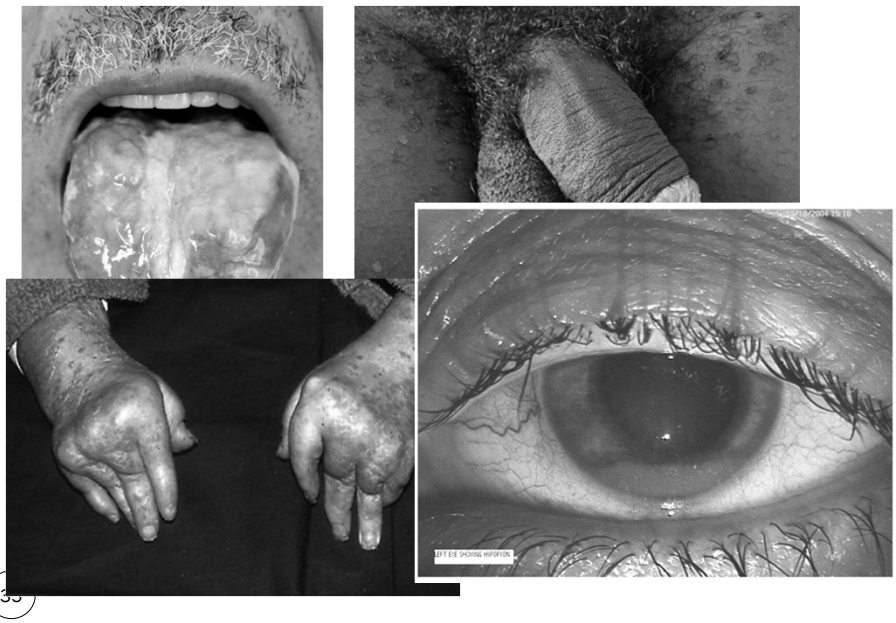
Other sites for psoriasis



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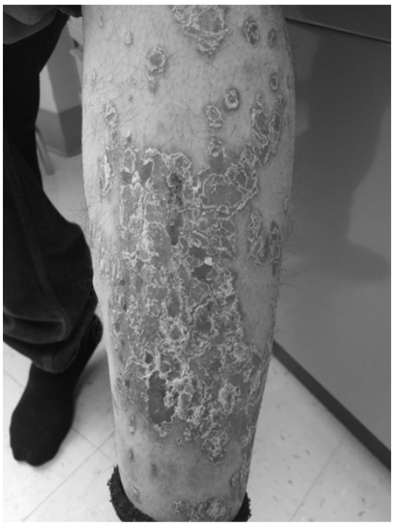
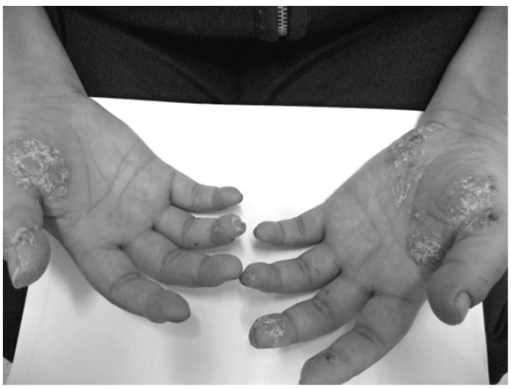
Psoriasis?





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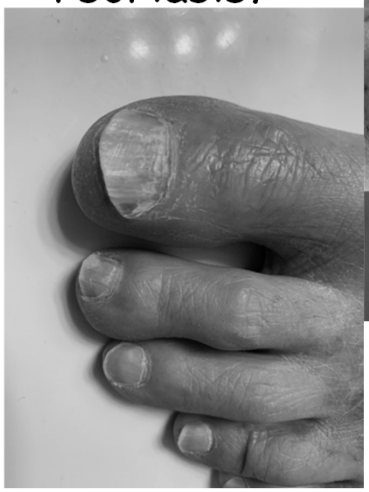
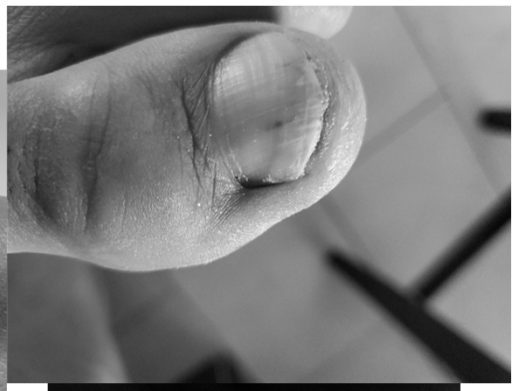
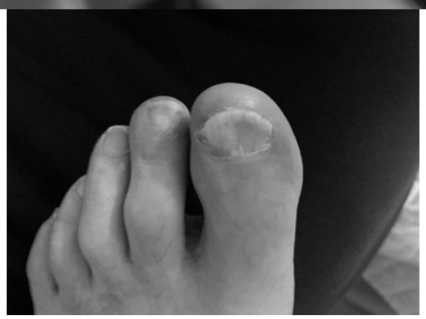
Psoriasis?







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Treatment of Psoriasis

- localised lesions: topical steroids/calcipotriol(Dovonex) or combination
- more generalised :
  - Oral agents : prednisone, retinoids (Acitretin©), cyclosporines, methotrexate, PDE4 inhibitor (Otzla ©)
  - PhotoRx--PUVA/UVB Rx
  - TNF-α inhibitors (Etanercept, Enbrel©; Infliximab, Remicade© , Humira©)
  - IL-X monoclonal antibodies [Ustekinumab(IL-12/23)--Stelara© (IL-23—Tremfya ©; Secukinumab(IL-17A), Cosentyx©]

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Before you refer….Take a good history !

- Sacred 7
- Drug use / misuse
- Travel / contacts / COVID
- Occupation /
- Co-morbidities :e.g., CRF, jaundice, diabetes, thyroid
- Stress level!

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Location	Site of the eruption?
Quality	Morphology of eruption
Severity	Extent and progress of eruption
Duration	How long has it been?
Timing	Acute/chronic/relapsing
Modifying factors	Relieving/worsening/response to treatment
Associated symptoms	Fever/systemic upset
+ PHx, FHx, SHx	+ occupational/environmental exposure

The Sacred 7+

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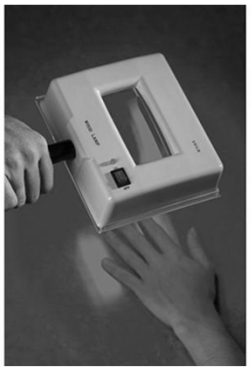
Use tools



And look critically

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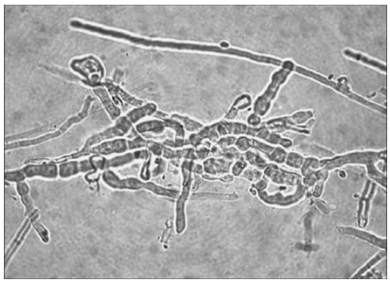
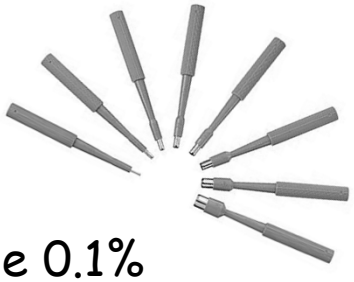
DO something….





Before you prescribe 0.1% Betaderm/Fucidin/Fucibet/Triderm Or refer to Dermatologist!

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Summary of presentation

- Eczema—etiology and clinical presentation
- Eczema—dermatoscopic features and differentials
- Eczema—treatment and prognosis
- Psoriasis—etiology and clinical presentation
- Psoriasis—dermatoscopic features and differentials
- Psoriasis—treatment and prognosis
- Or Else?
- When to refer?
- To steroid or not to steroid?

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Steroids ≠ placebo→ you'd rather give emollients

Dual/triple cream does not cure everything

Better do nothing and wait until biopsy or referral available

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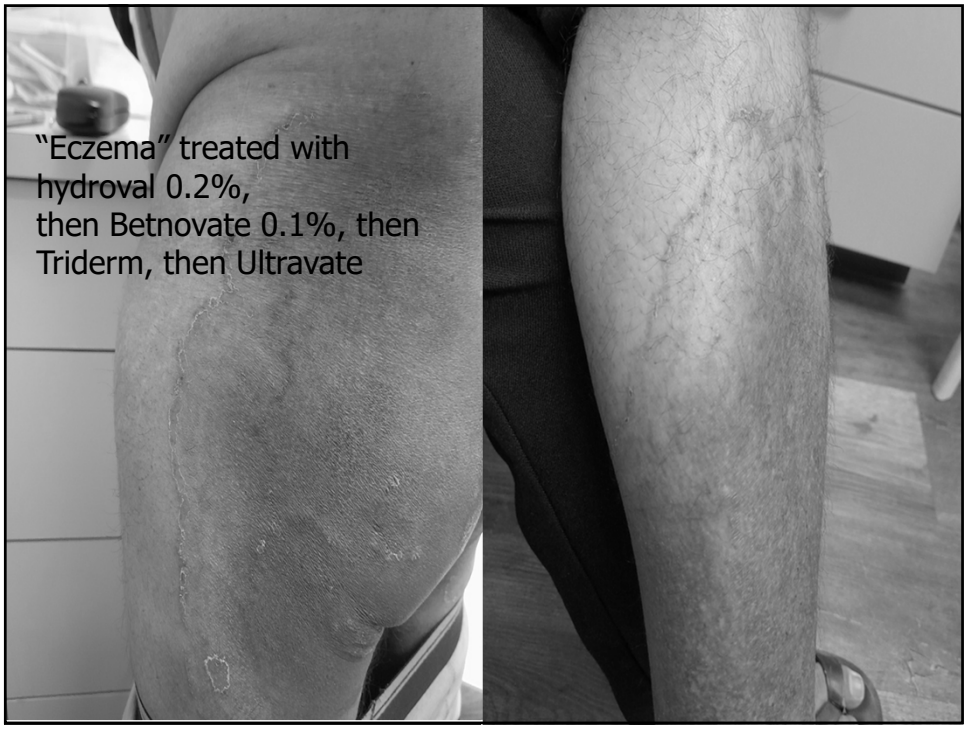
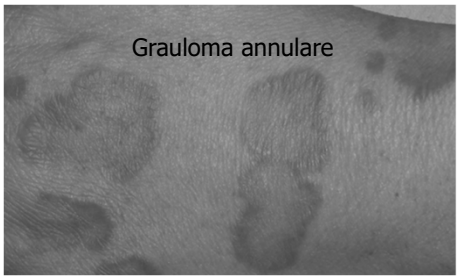
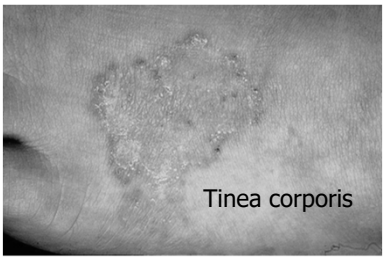
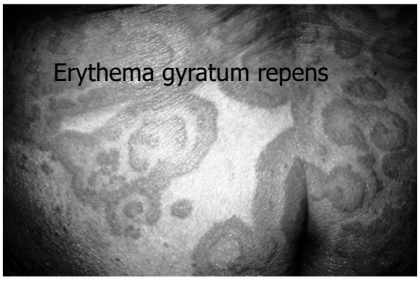
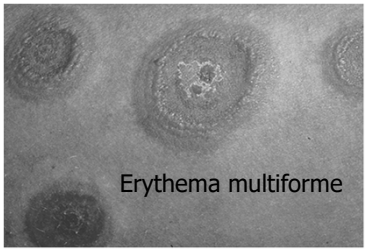




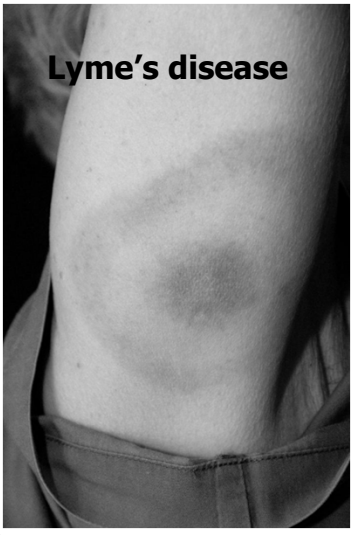


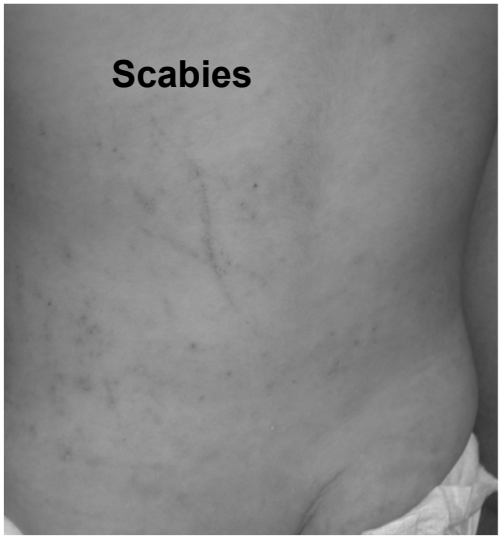




49 I have no idea, but I can tell the differences

A bit less obvious





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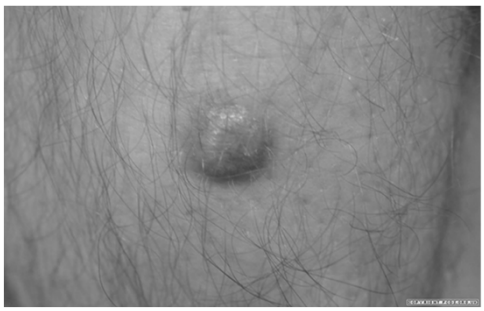
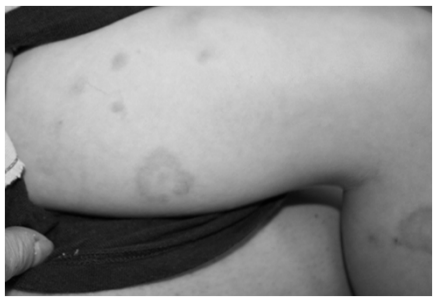
Possible presentations of insect bites



Dermatofibroma



Granuloma annulare

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