Eczema and dermatitis

- Allergic contact dermatitis
- Irritant contact dermatitis
- Atopic dermatitis
- Microbial eczema
- Seborrheic dermatitis

Allergic contact dermatitis

makes 5 – 15% of all dermatoses

- **Prevalence – 1,5-3%**
- **Incidence – 5-10 / 1000** per year
- **Hypersensitive reaction of the IVth type according Coombs & Gell**

Allergic contact dermatitis

contact allergens – molecules smaller than 500 D – *penetration through the skin barrier*

inding of the molecule – hapten - to pt‘s own proteins in the skin forms an antigen – with the molecular weight at least 5000 D

- **the conjugation of haptens with proteins takes place in LC (antigen presenting cells)**

Allergic contact dermatitis

Induction phase enetration of allergen through the stratum corneum teraction with APC

hagocytosis of antigen

subsequent expression of antigen on the surface of **LC** igration to regional lymphatic nodes and presentation of the antigen to naive **T–lymphocytes**

Allergic contact dermatitis

Elicitation phase – in case of sensitization

roliferation of specific

Clone of **effector T–lymfophocytes** igration

to the site of allergen penetration

ytotoxic effect of T–lymphocytes releasing cytokines leading to inflammation

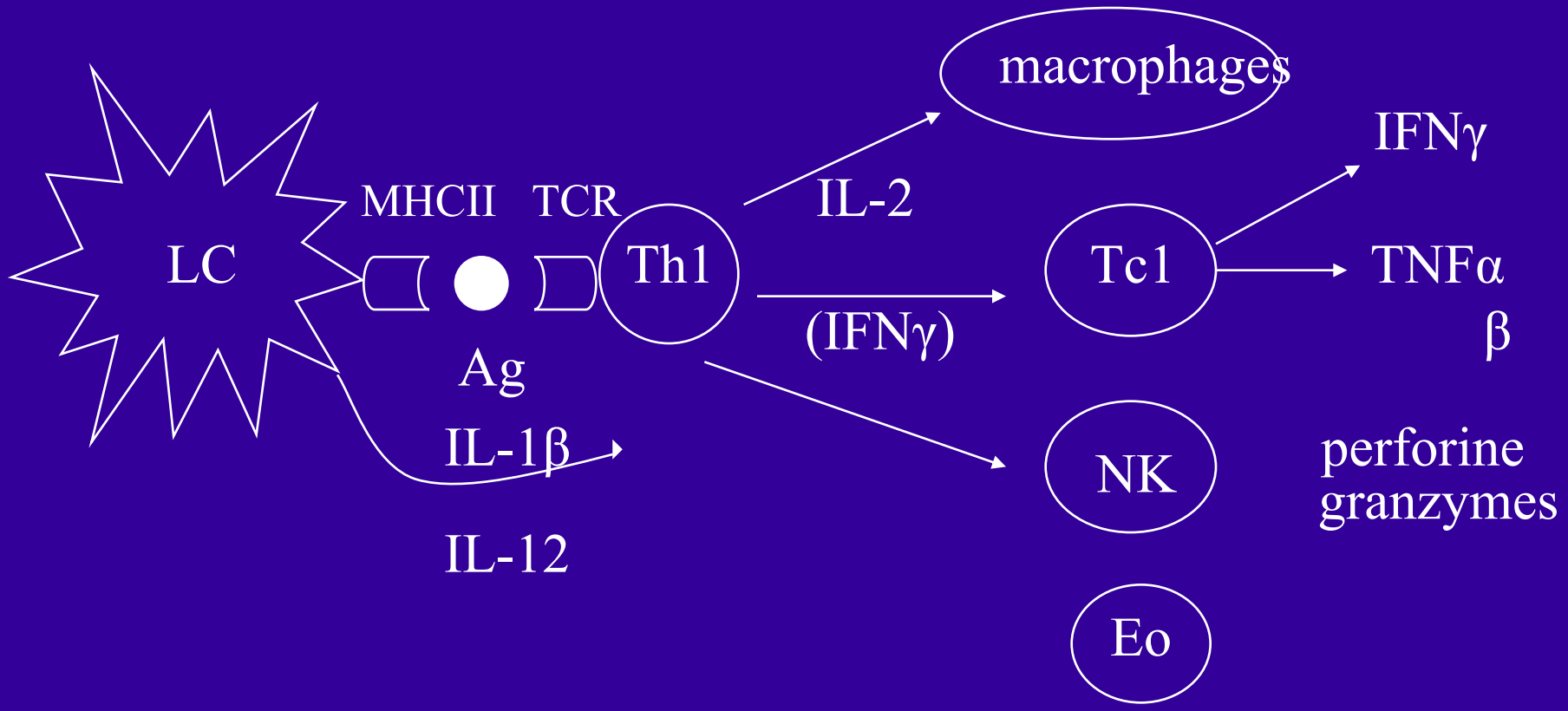
→ **allergic contact dermatitis**

Shortest time to sensitization: 5-14 days

migration of LC to regional LN takes about 5-24 hours

proliferation of T-lymphocytes – 5-10 days)

Patophysiology of the late-type hypersensitivity



Allergic contact dermatitis

Factors influencing the ease of sensitisation:

- **Chemical structure of allergens**
- **Patient** – skin barrier status (fissures, maceration)
  localisation (eyelids x soles)
  age

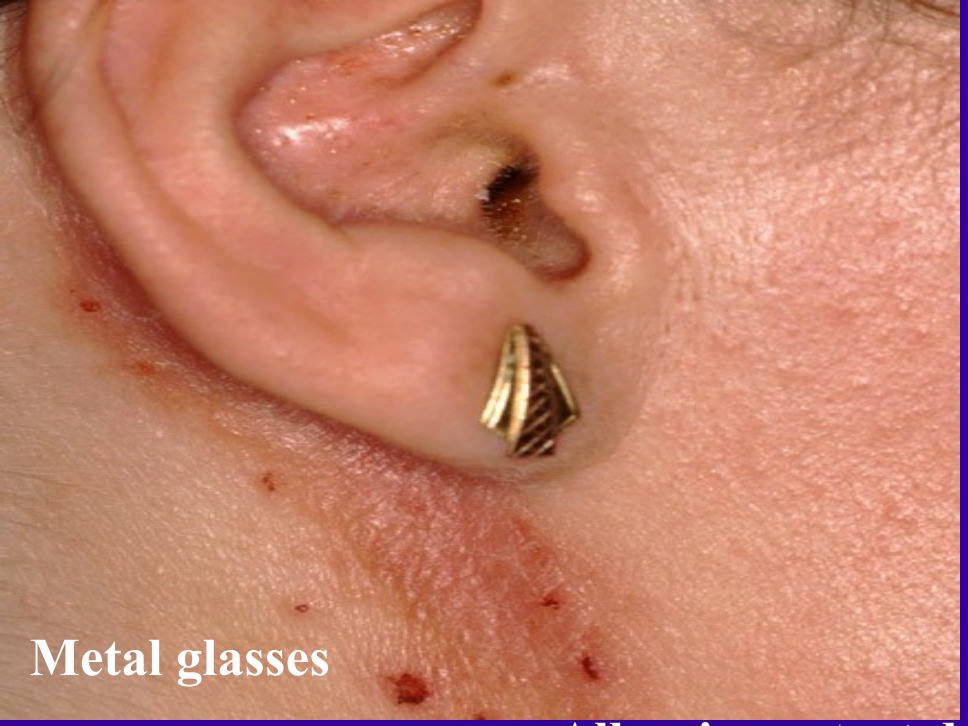
- **Duration of hypersensitivity**
  - **survival time of memory** T-lymphocytes
  - character of allergens

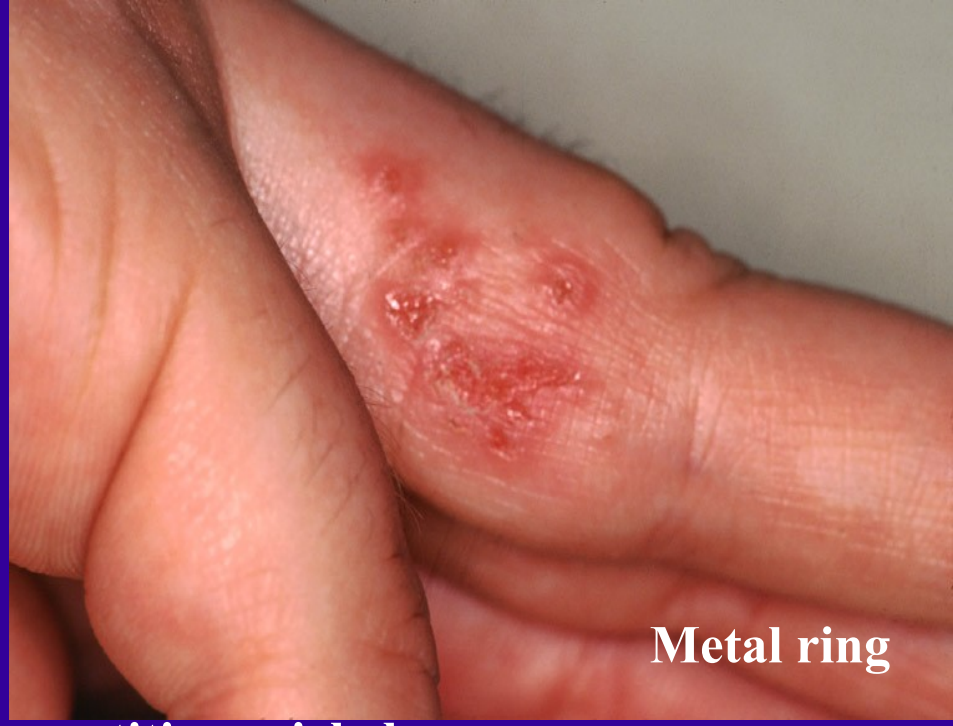
European Standard Series

- Potassium dichromate O,5 % pet.
- Neomycin sulphate 20 % pet.
- Thiuram mix 1% pet.
- Paraphenylenediamine 1% pet.
- Cobalt chloride 1% pet.
- Caine mix 10% pet
- Formaldehyde 1% aq.
- Colophony 20% pet.
- Hydroxyethyl metacrylate 2% pet.
- Balsam of Peru 25 % pet.
- N-isopropyl-N-phenyl-4-phenylenediamine 0,1% pet.
- Wool alcohols 20% pet.
- Mercapto mix 2% pet.
- Epoxy resin 1% pet.
- Paraben mix 16%

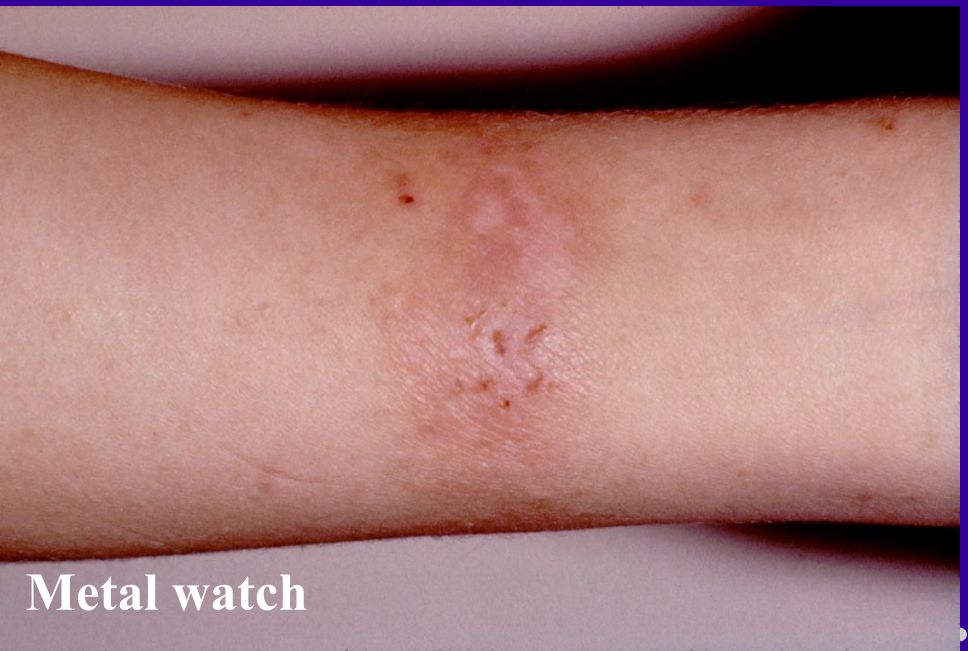
European Standard Series

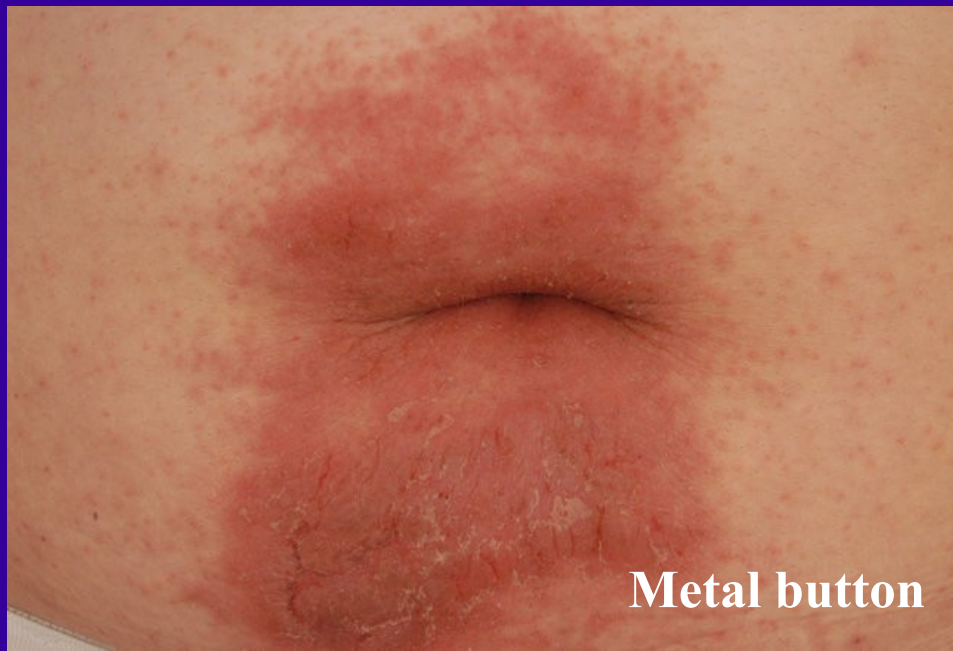
- pet.4-t- butylphenol formaldehyd resin 1% pet.
- Fragrance mix 8% pet.
- Quaternium 15 1% pet.
- Nickel sulphate 5% pet.
- Kathon CG 0,01% aq.
- Mercaptobenzothiazole %pet.
- Sesquiterpenlactone mix 0,1% pet.
- Propolis 10% pet.
- Tixocortol-21-pivalate 0,1% pet.
- Budesonide 0,01% pet.
- Methyldibromoglutaronitrile (1,2-dibromo-2,4-dicyanobutane)*
- Fragrance II 14% pet.
- Lyral 0,5 % pet.
- Methylisothiazolinone 0,02% aq
- Textile dye mix 6,6% pet.

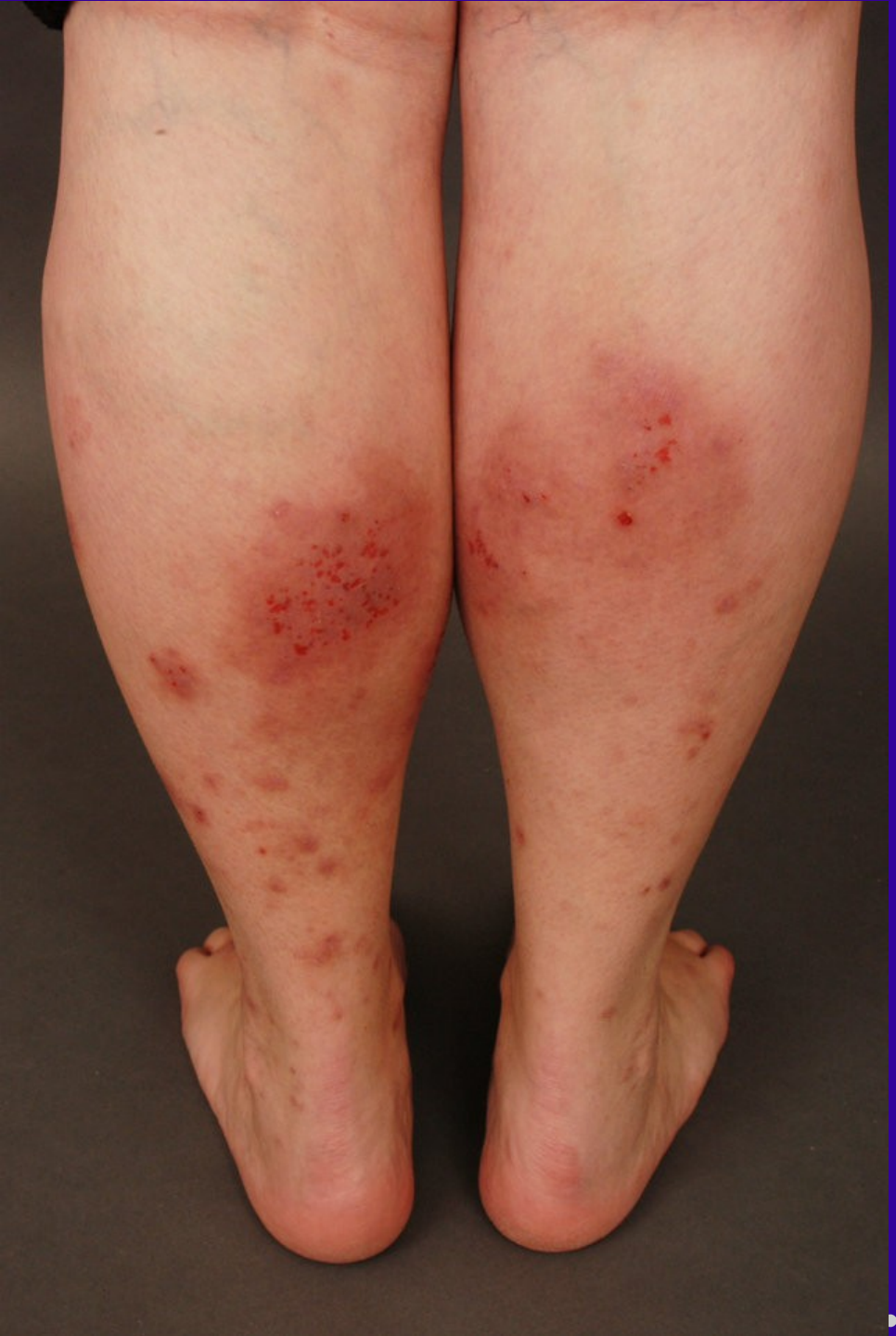




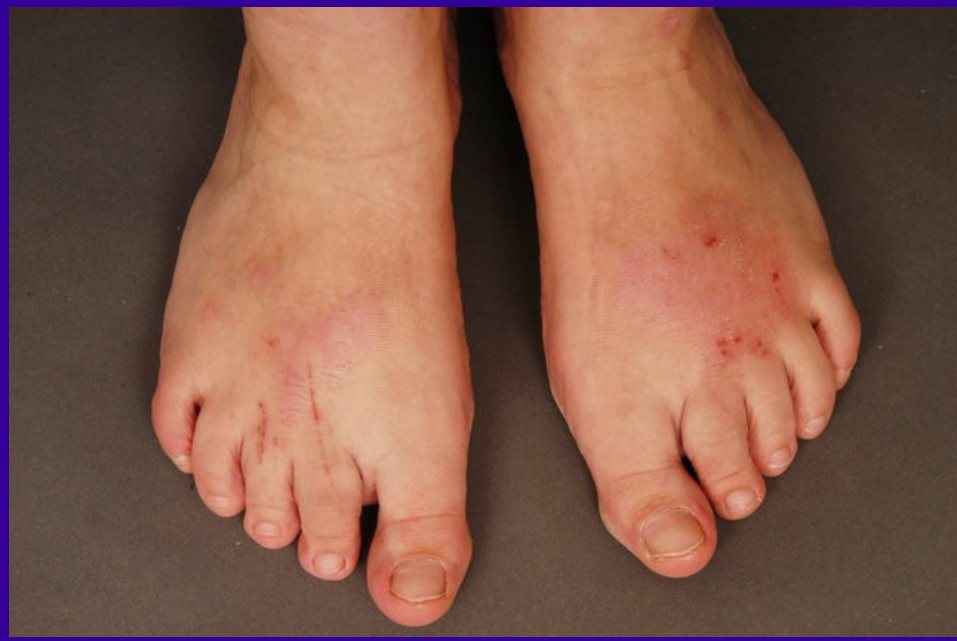
Allergic contact dermatitis – nickel

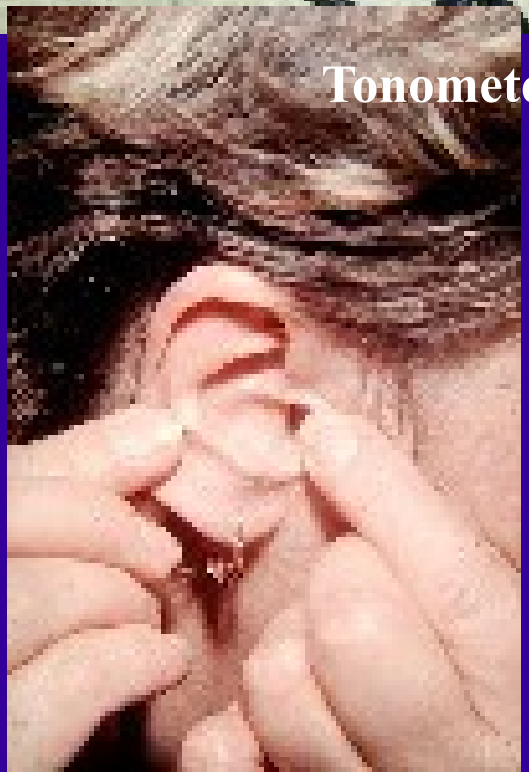






ACD to chromium from leather boots

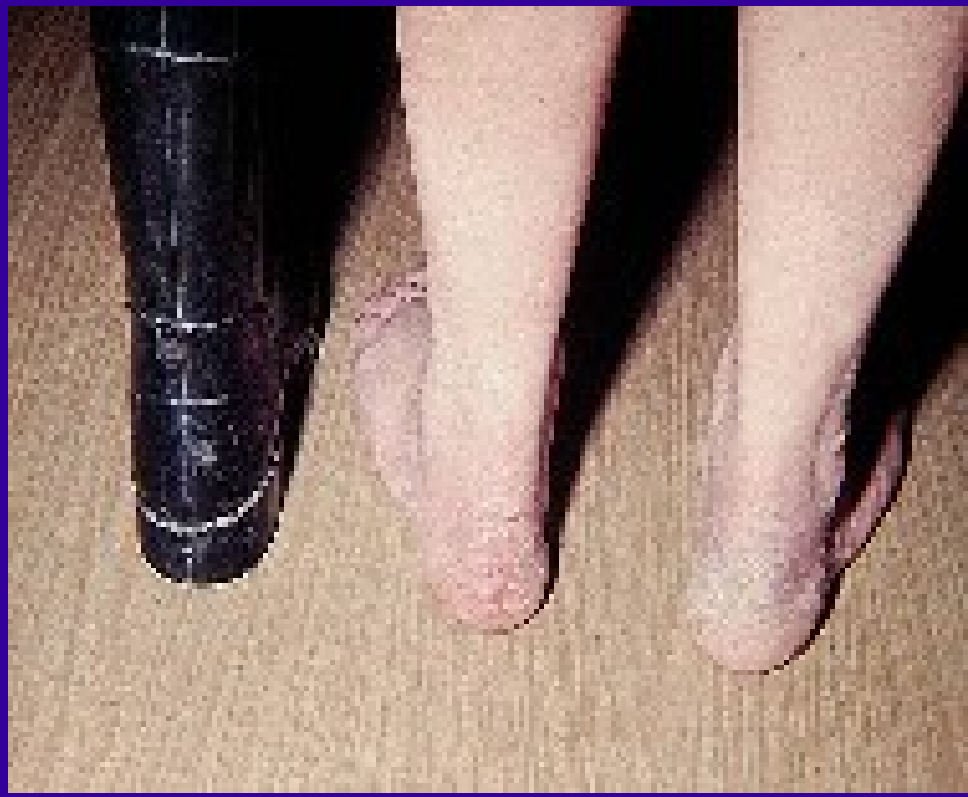




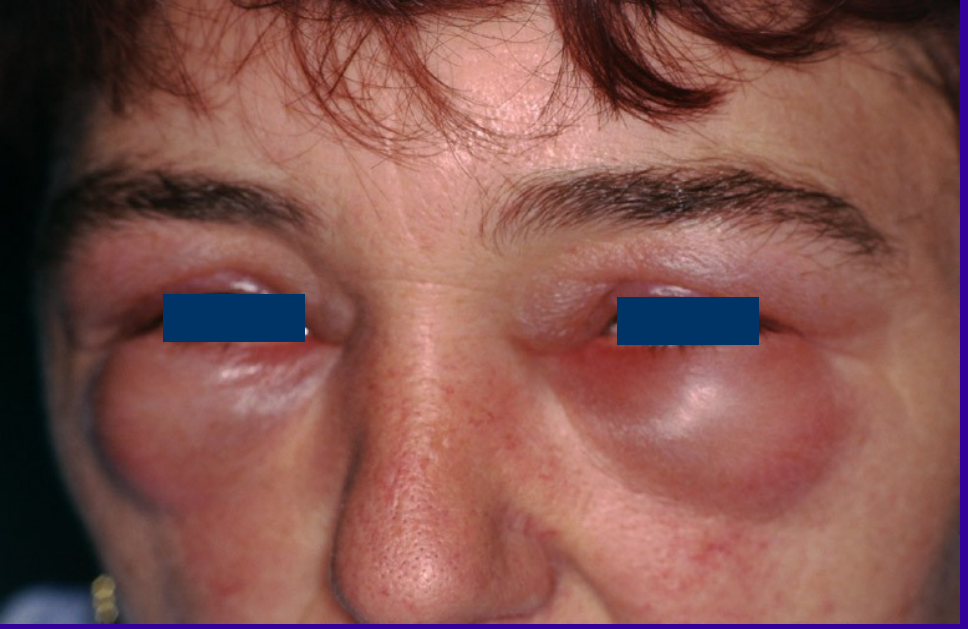
Tonometer, stethoscope

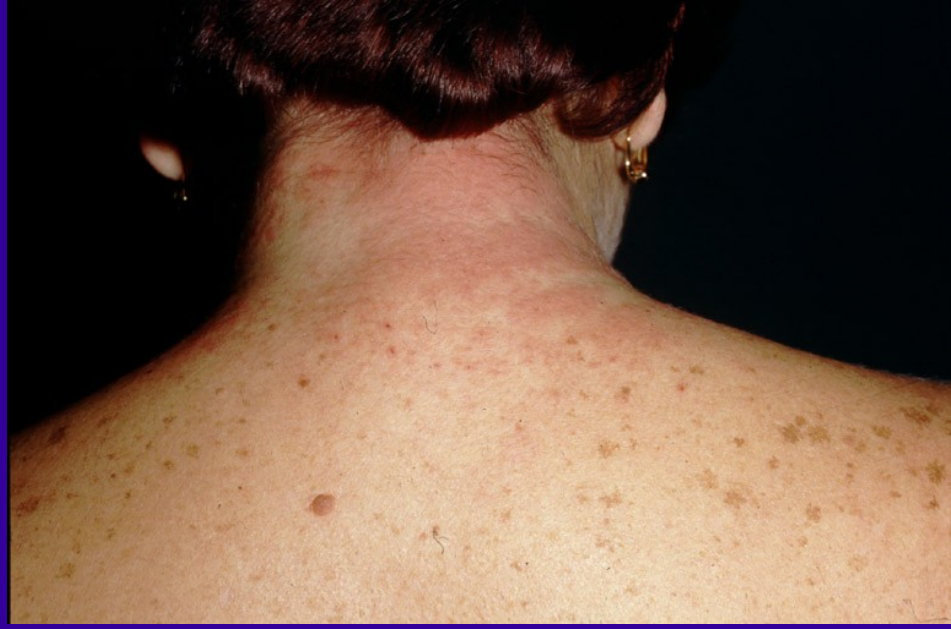
(nurse)

ACD- IPPD, antioxydant of black rubber

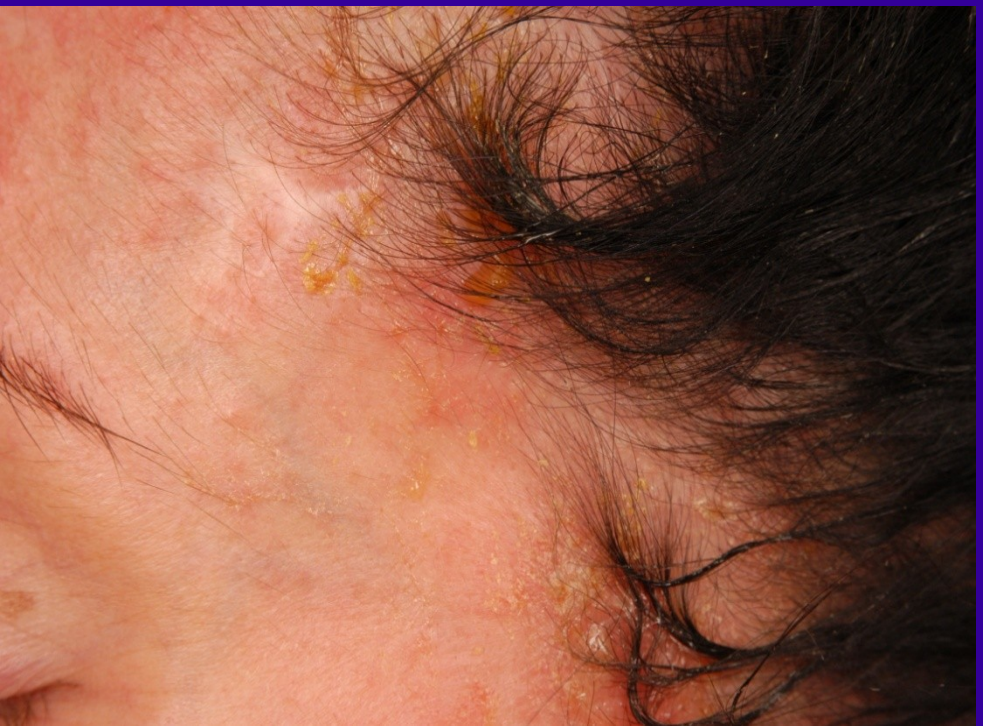


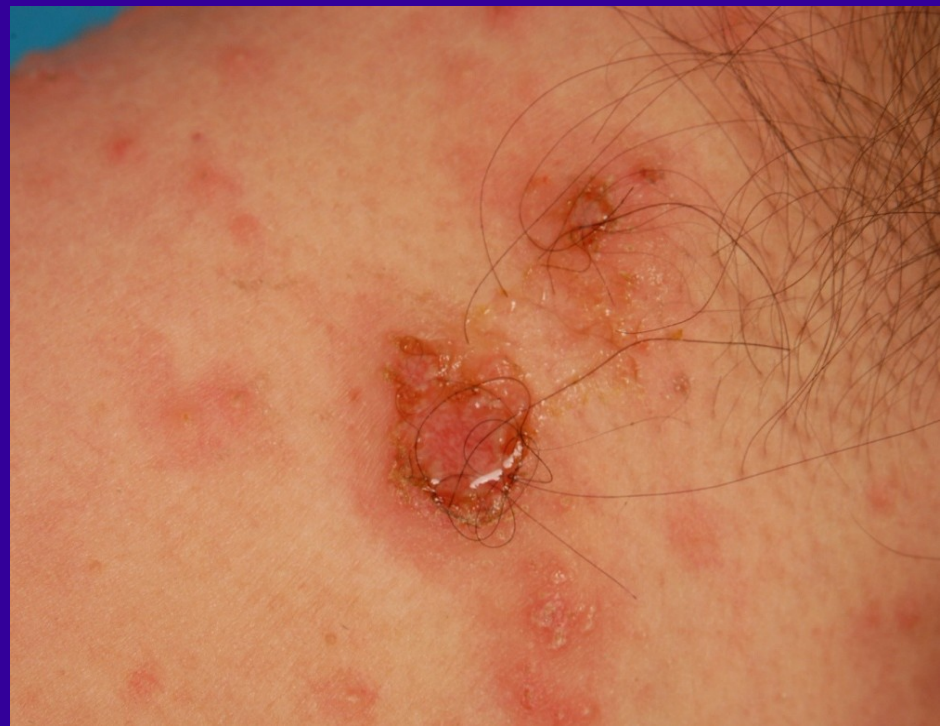
Rubber boot

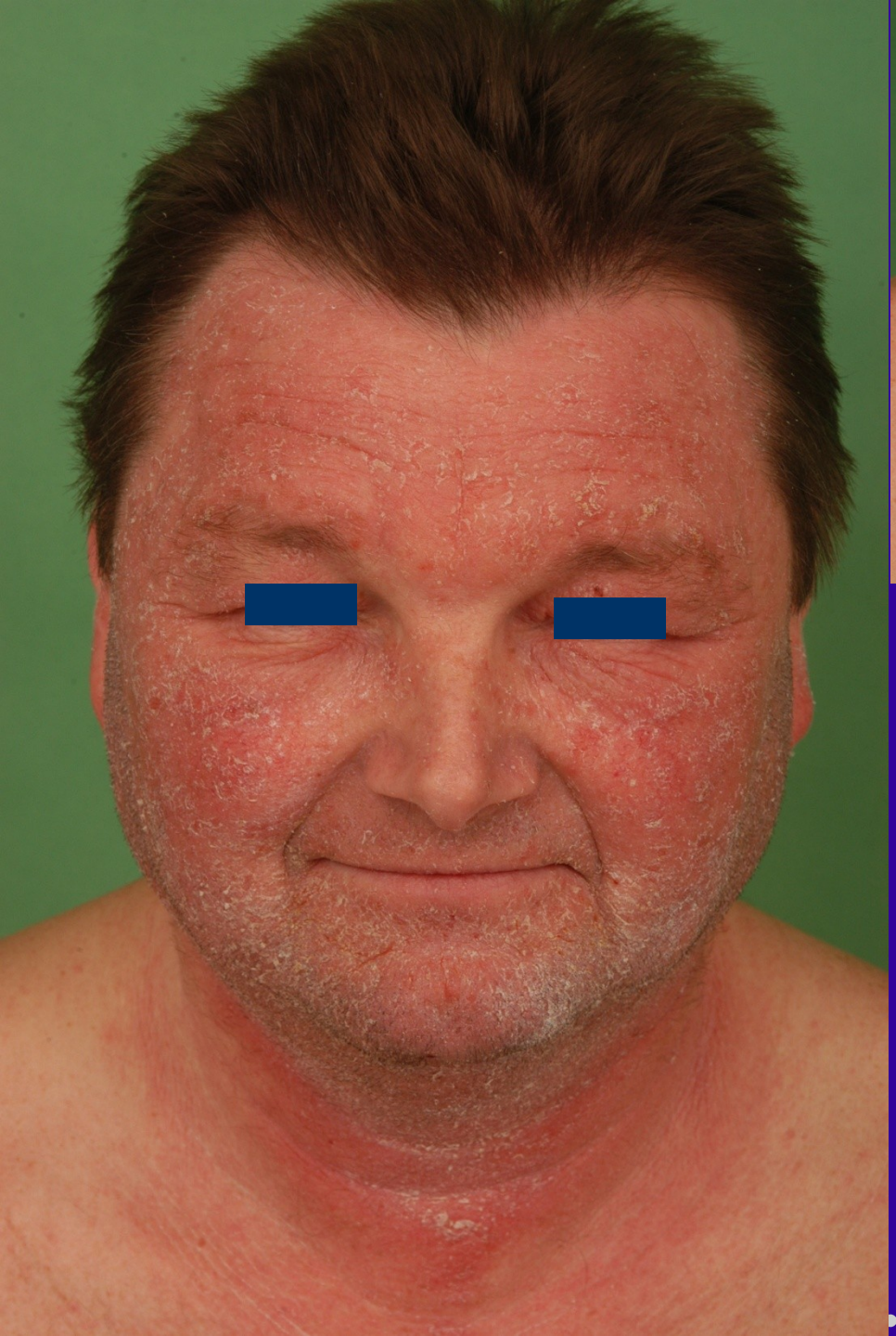


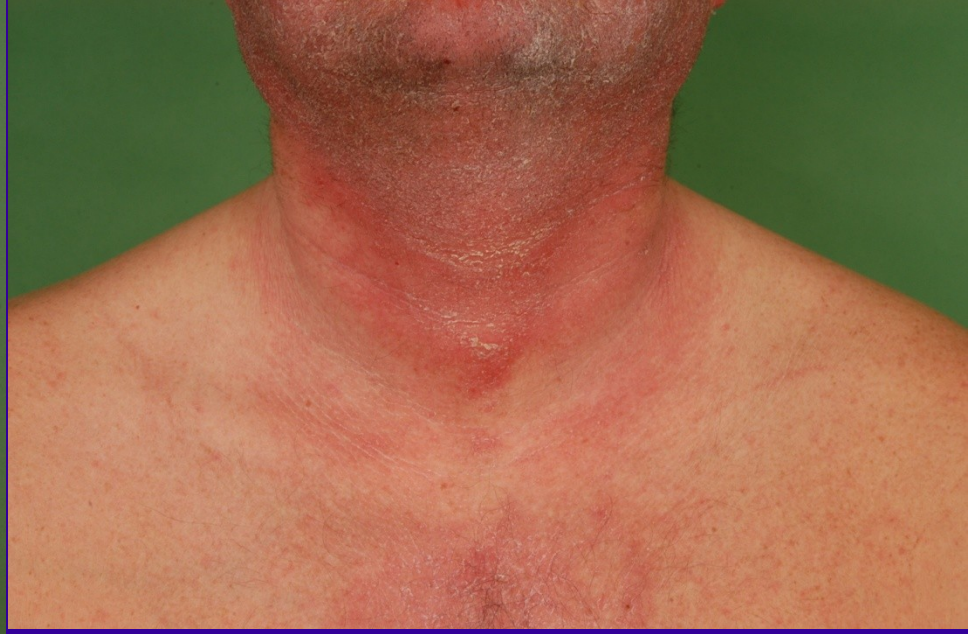


ACD to PPD from hair dyes

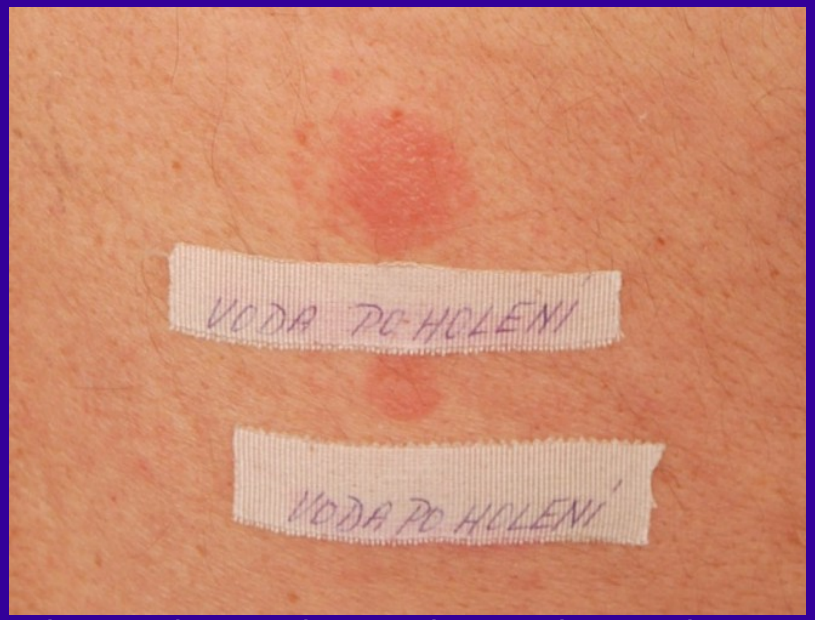








ACD to fragrance (eau de toilette)



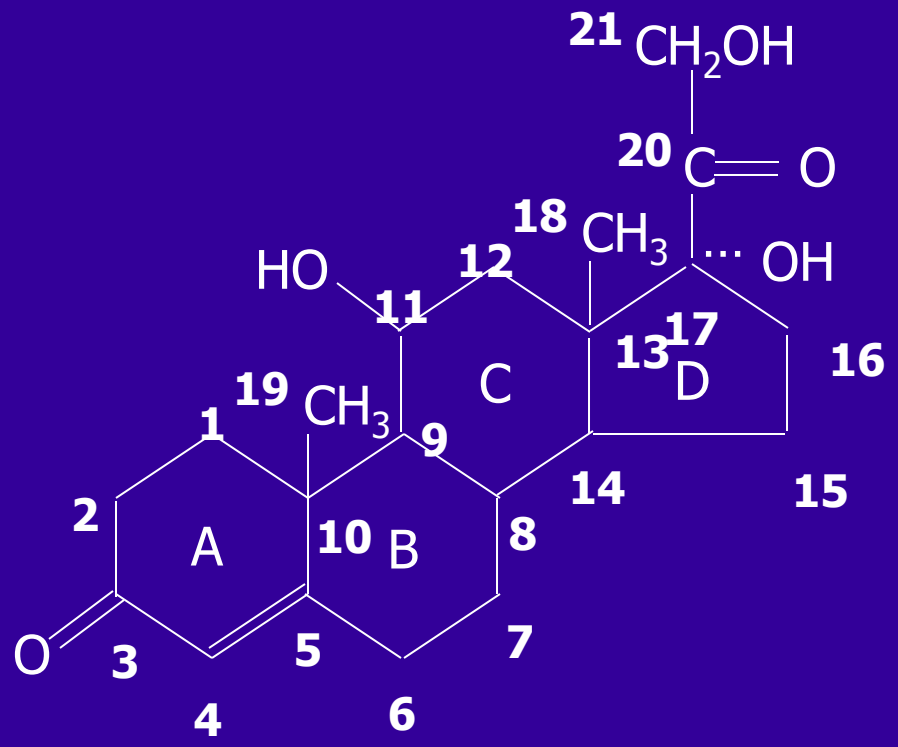
Corticosteroids

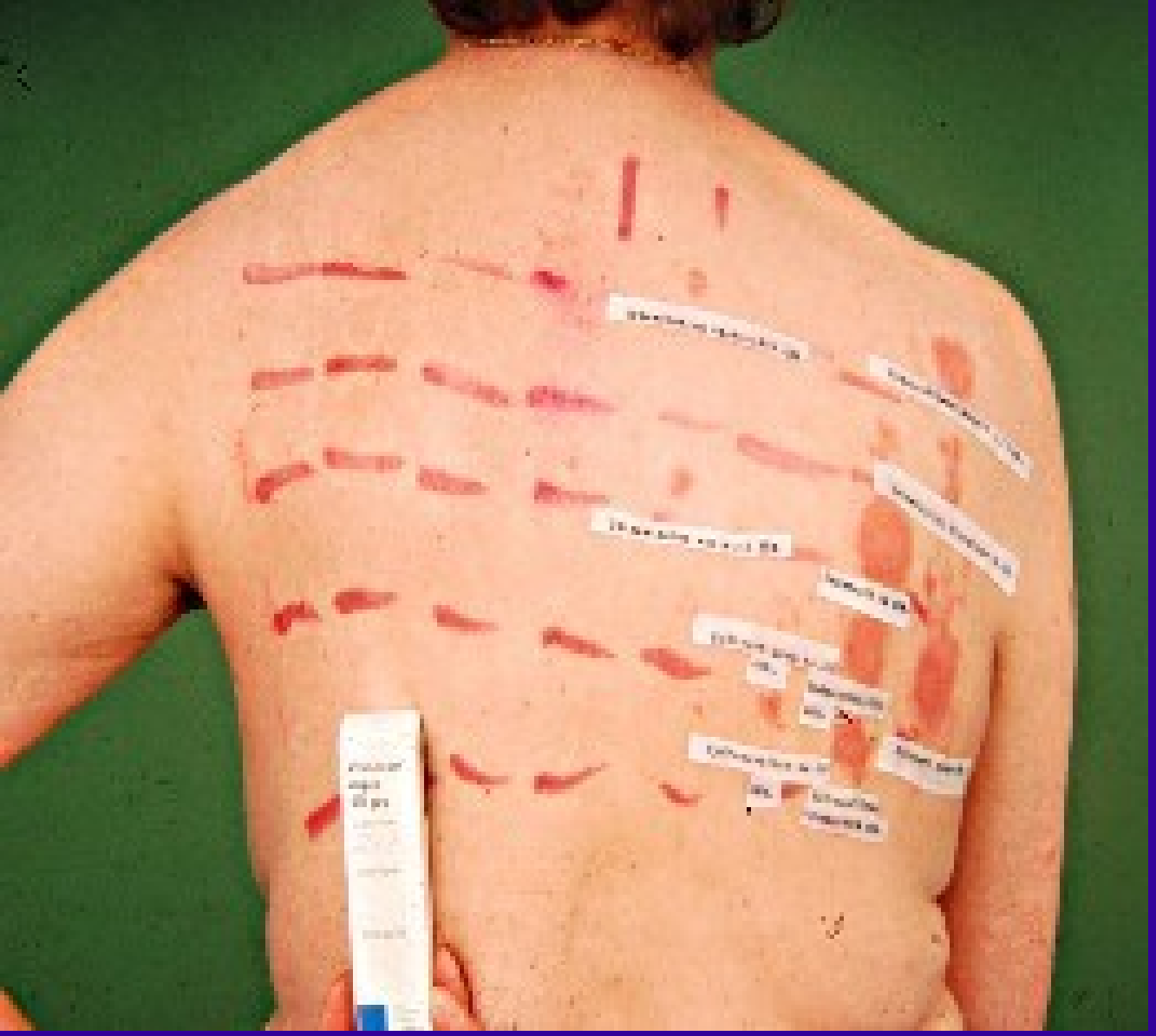
A - type **Hydrocortison**: D kruh nesubstituovaný, **C 20**, **C 21** nesubstituovaný nebo **C 17**, **C 21** krátký řetězec (acetáty nebo estery), event. **C 21**, thioester

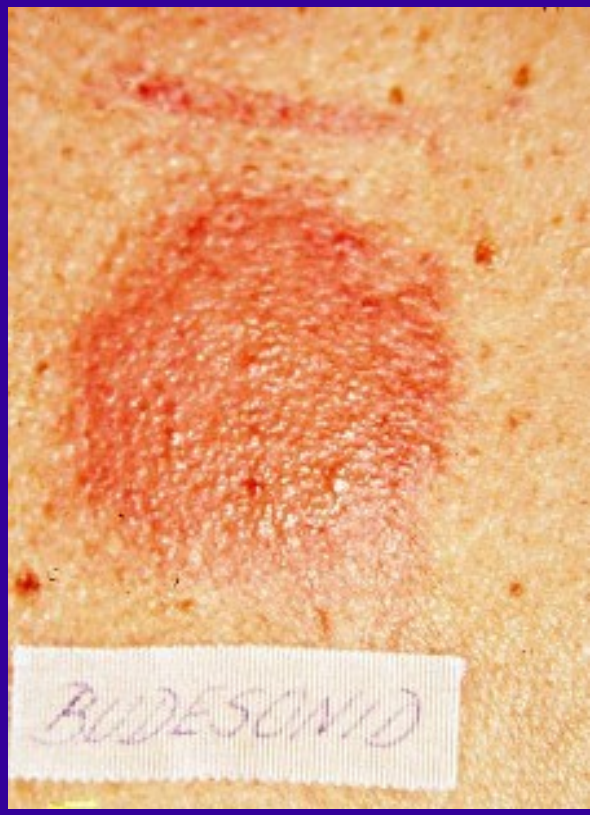
B - type **Triamcinolon acetonid**: **C 16**, **C 17** cis-ketal struktura nebo diol struktura

C - type **Betametason**: **C 16** methyl substituce

D - type **Hydrocortison butyrát**: **C 17**, a/nebo **C 21** dlouhé esterové řetězce, event. **C 16** metyl substituce







Budesonide - Apulein ung, crm, liq, Pulmicort aer inh, Pulmicort, Turbuhaler plv inh, Rhinocort spr nas

Patch test– contact allergy to Budesonide

Budesonid 1%

Betamethasoni dipropi... 0,0...

72h.

...ec.2%

Budesonid0,025%

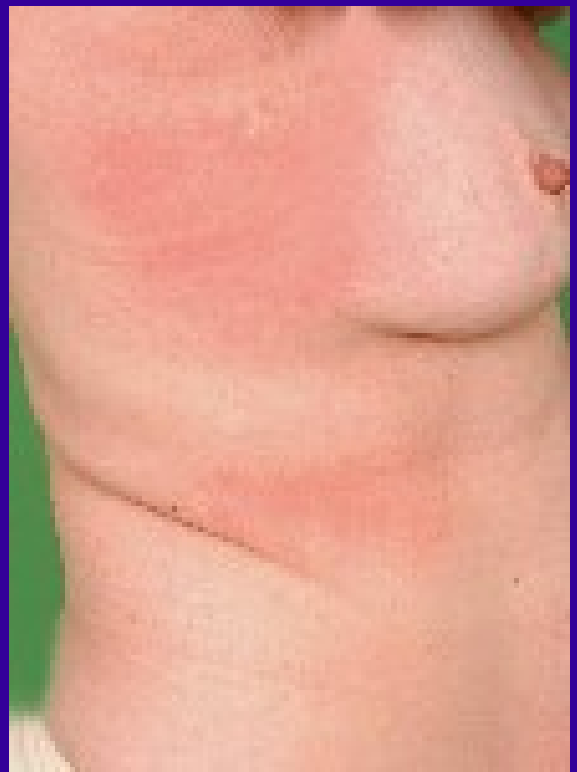
Rhinocort spray 72h.

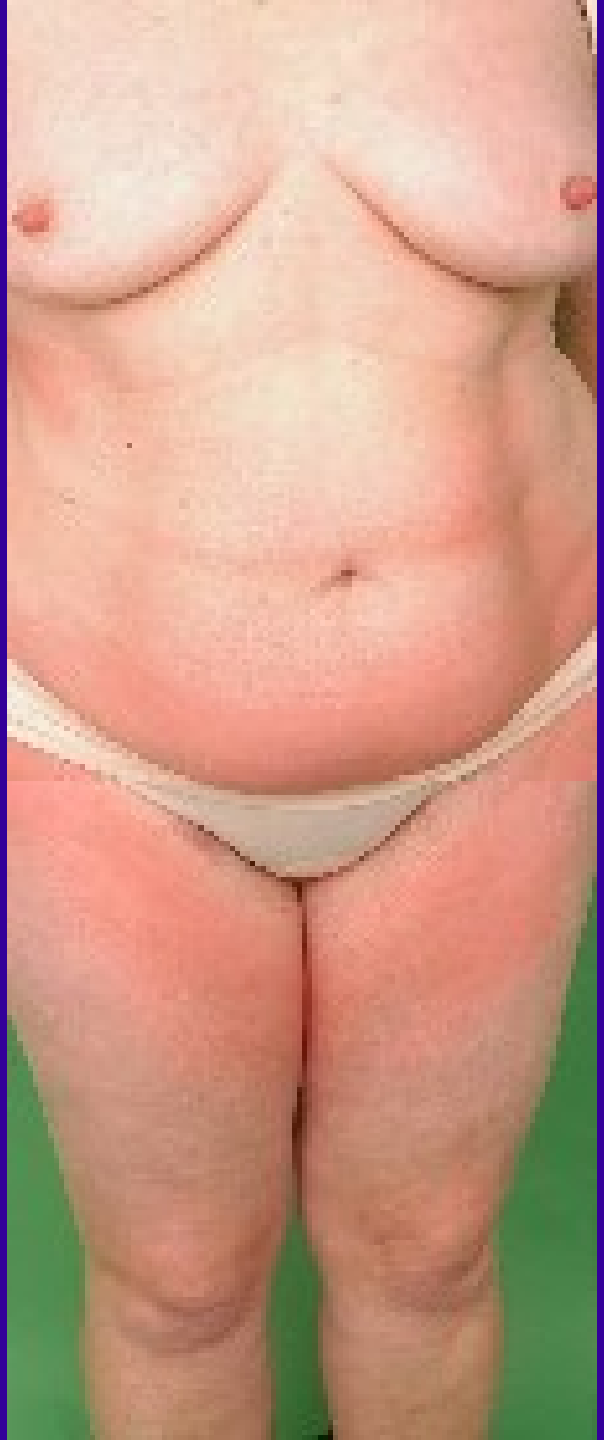
...ison ...e.1%

...n.

Hydrocortison

Drug eruption in patient sensitized to topical CS after systemic exposure to - Prednisone tbl





Fragrances

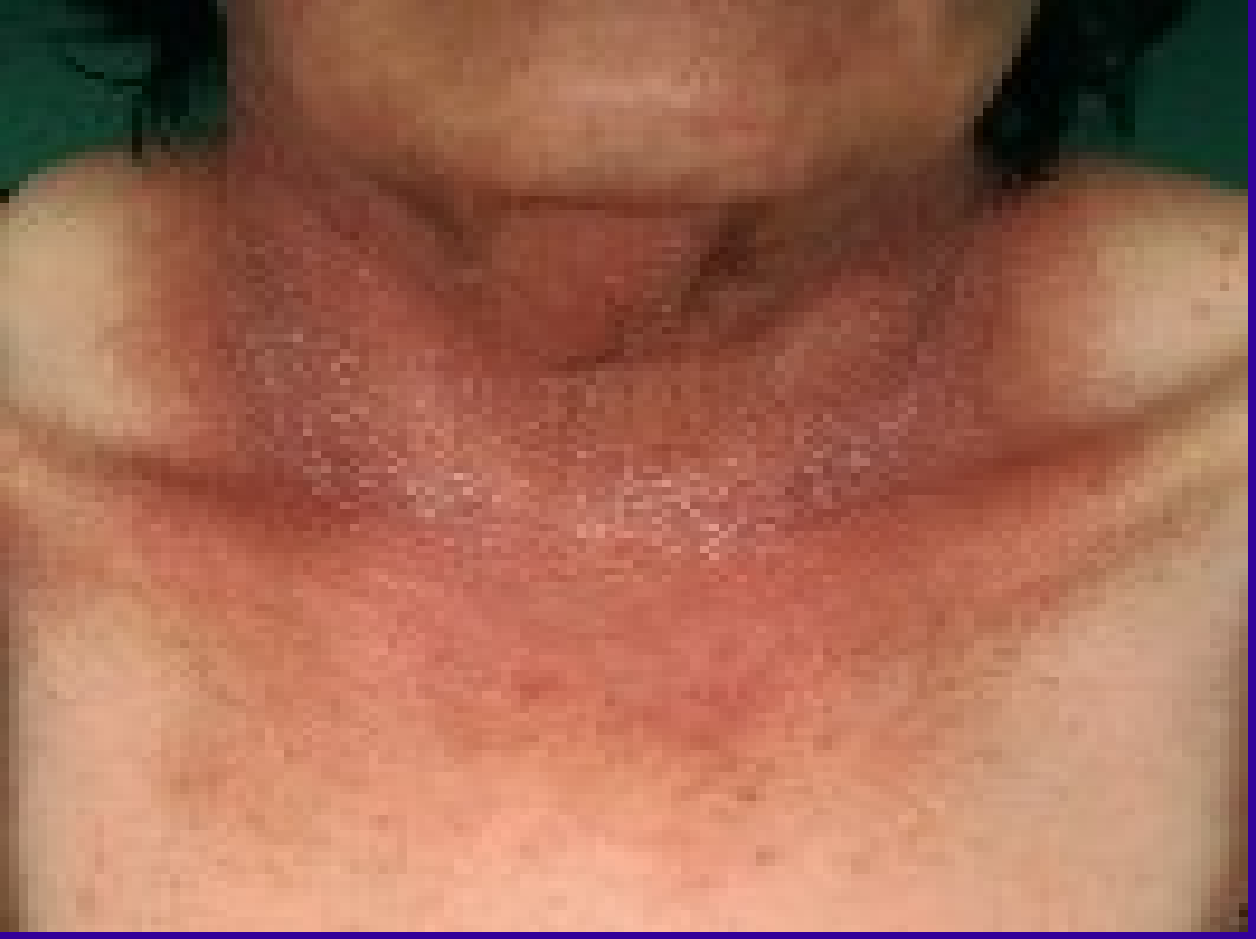
Fragrance mix I

- Cinnamic aldehyde
- Cinnamic alcohol
- [redacted]amyl-cinnamic aldehyde
- Eugenol
- Isoeugenol
- Geraniol
- Hydroxycitronellal
- Oak moss absolute (Akranorin)

Sorbitan sesquioleate (emulgator)

Frequency of sensitization:

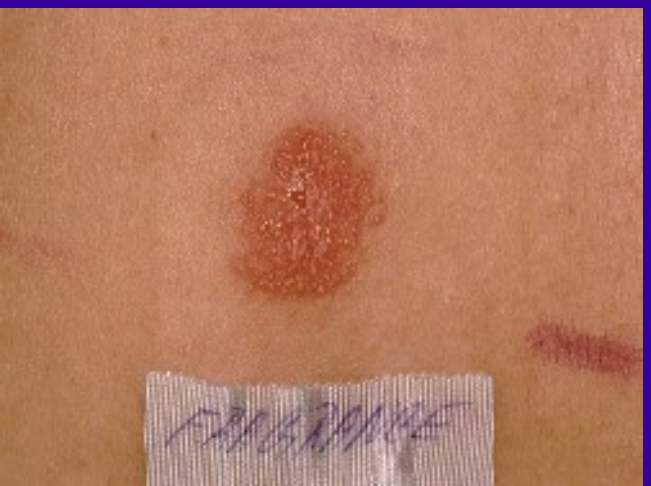
worldwide 4,7-13,3%

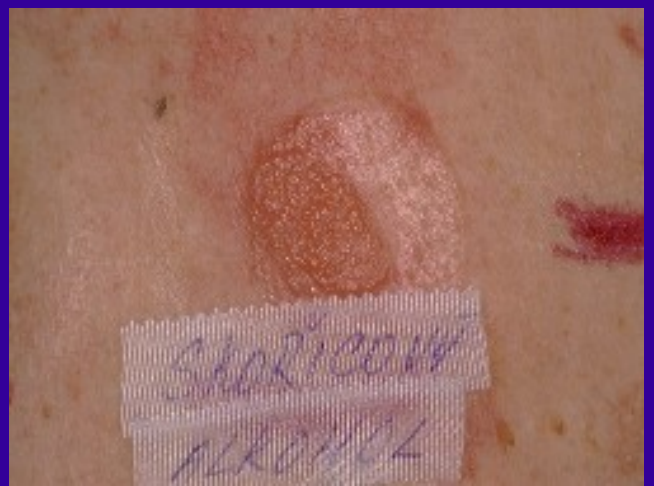


Allergic contact dermatitis–

fragrance –

cosmetic cream





Patch tests – contact allergy to fragrance and cinnamic alcohol

Fragrances

Fragrance mix II

Lyral,Citral,Farnesol,Citronellol,
hexyl cinnamic aldehyde,Coumarine

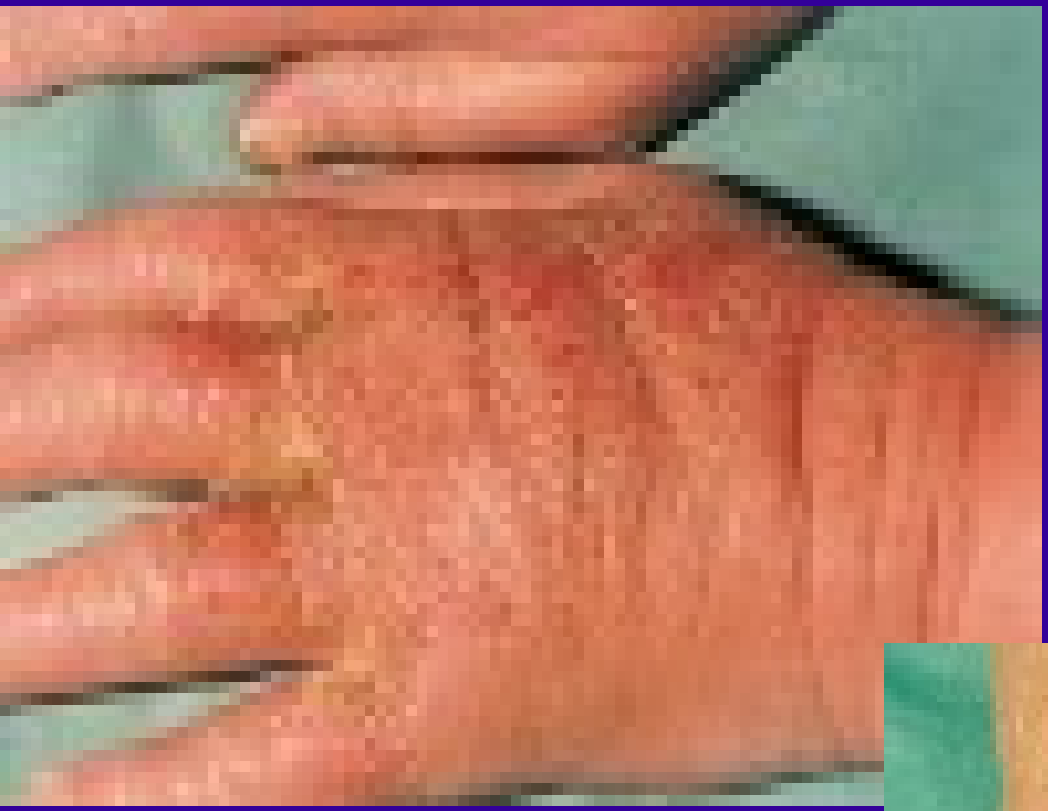
Fragrance mix III

yasmine absolute 2,0 % vaz.Amylcinnamaldehyde 2,0 % vaz.Musk ketone 1,0 % vaz.Sandalwood oil 2,0 % vaz.Musk moskene 1,0 % vaz.Ylang-ylang 2,0 % vaz.Cananga oil 2,0 % vaz.Vanilin 10,0 % vaz.Jasmine synthetic 2,0 % vaz.Geranium oil Bourbon 2,0 % vaz.Musk xylene 1,0 % vaz.Lavaner absolute 2,0 % vaz.Rose oil 2,0 % vaz.Narcissus absolute 2,0 % vaz.Methyl anthranilate 5,0 % vaz.Benzyl salicylate 2,0 % vaz.Benzyl alcohol 1,0 % vaz.

Balsam of Peru
Propolis

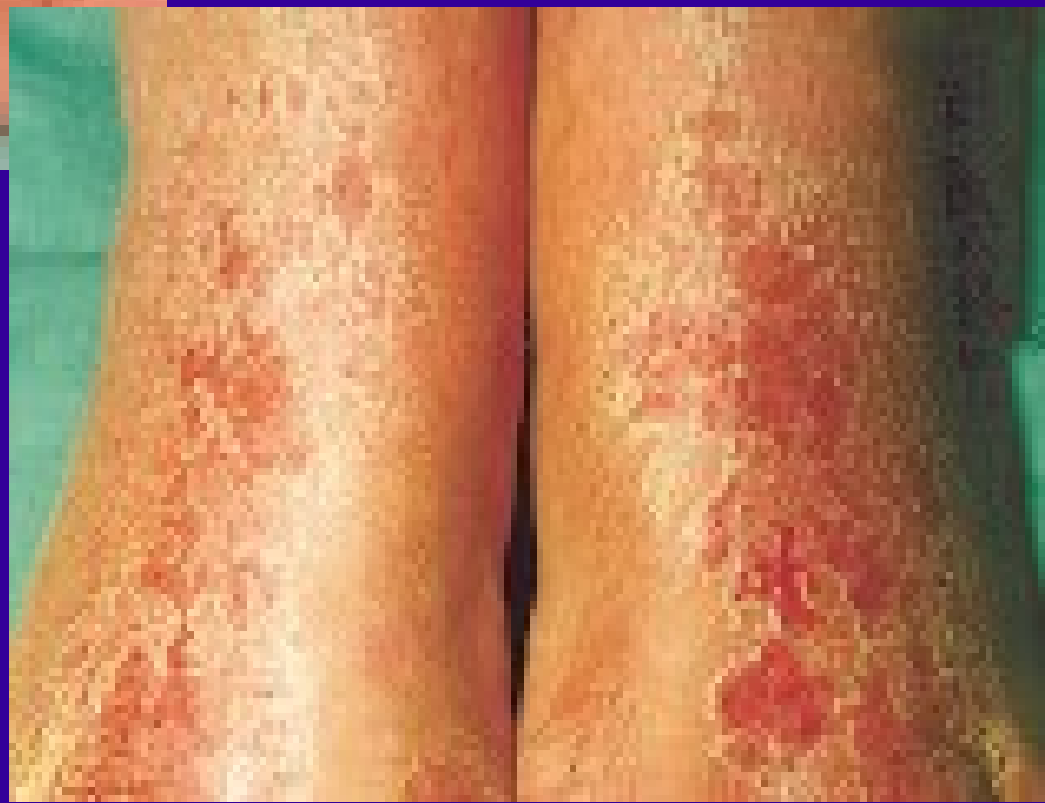
Propolis

- natural product – is a resinous mixture that honey bees collect from tree buds, sap flows, or other botanical sources. The chemical composition of propolis varies depending on season, bee species and geographic location. Propolis has approximately 50 constituents, primarily resins and vegetable balsams (50%), waxes (30%), essential oils (10%), and pollen (5%). Propolis has antibacterial, fungicidal, antipruritic and antiinflammatory effects and promotes epithelisation



Allergic contact dermatitis–

propolis (folk medicine preparations)



Allergic contact dermatitis–

propolis (folk medicine preparations)

„new“ allergens

Ketoprophene – nonsteroidal antiinflammatory drug

Derivative of propionic acid

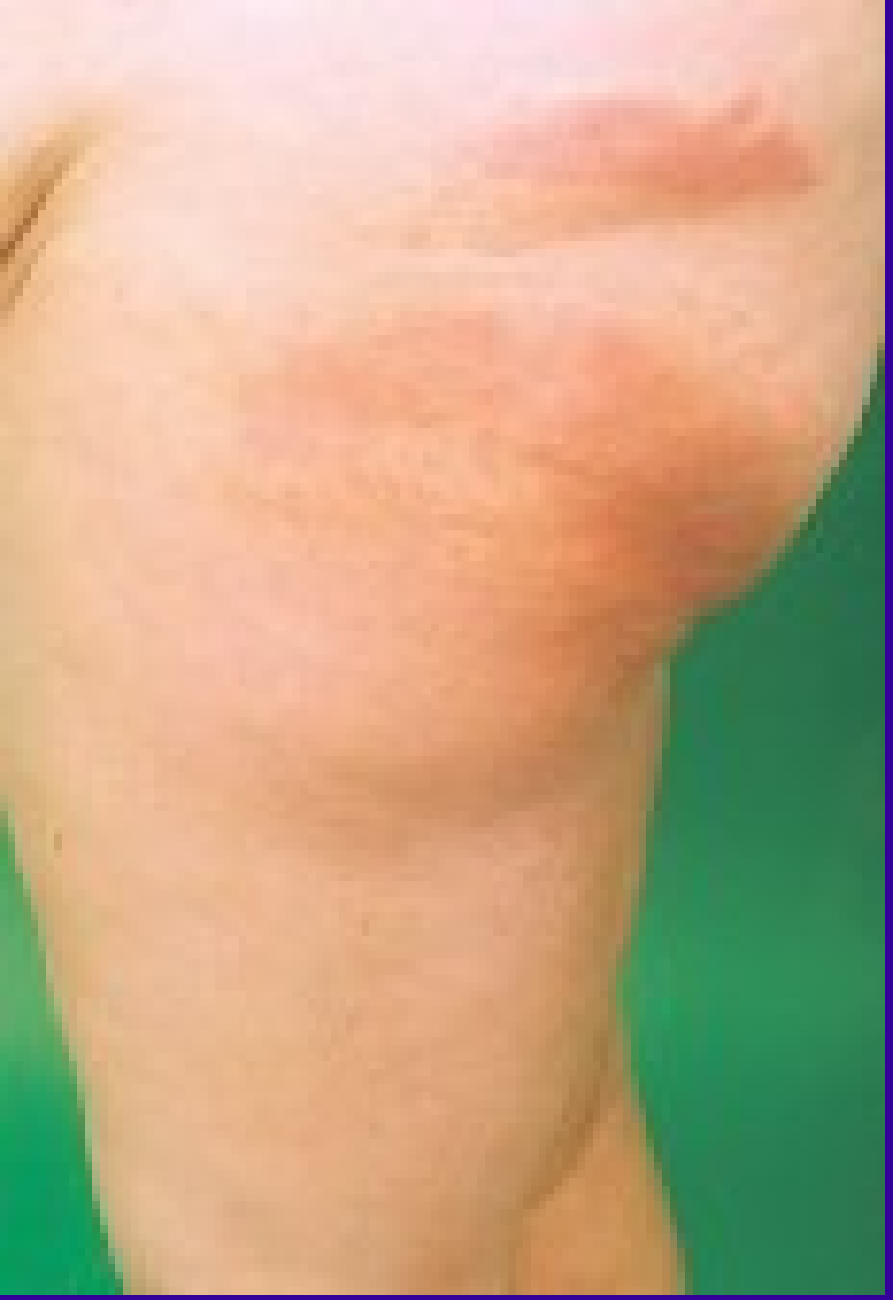
Ketoprophene – topical

Fastum
Profenid gel
Ketonal crm
and others

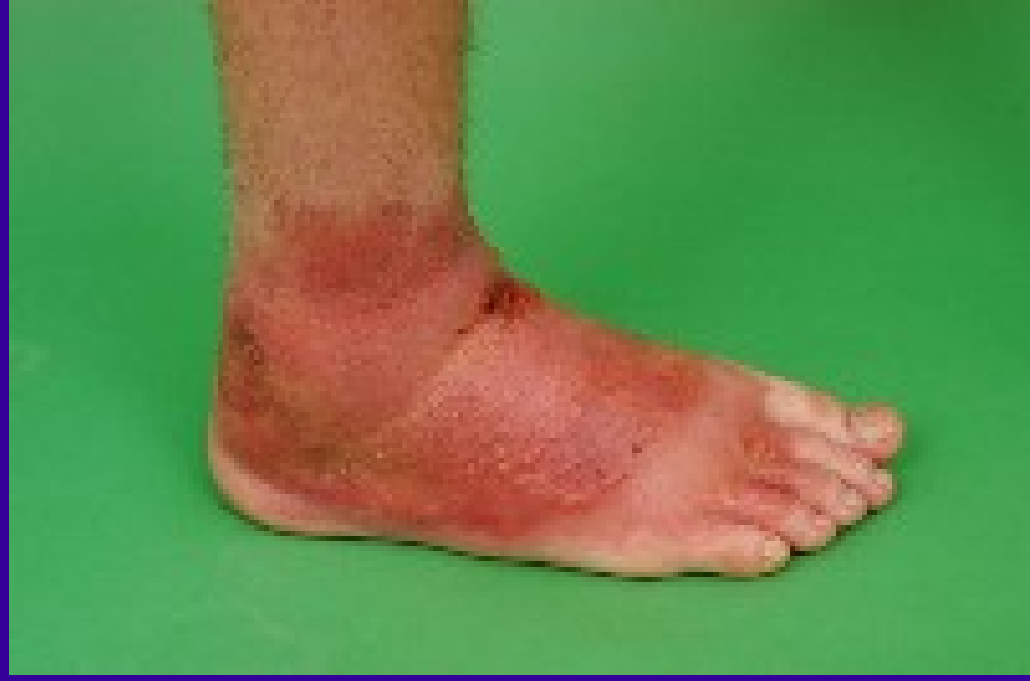
systemic

Ketoprofen tbl,sup
Ketonal cap,sup amp i.m.
Ketonal forte tbl
Ketonal ret tbl
Profenid cap,tob,sup amp
Profenid 100 mg pro inf
Toprec tbl

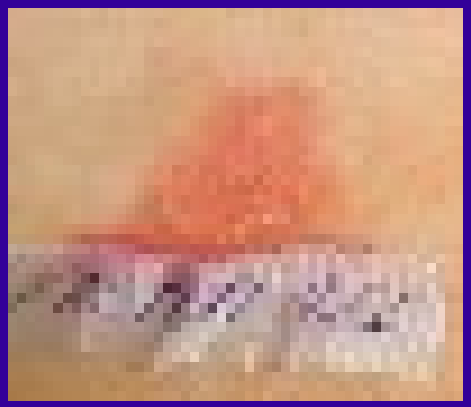
Allergy potenciated by sun exposure – photocontact allergy



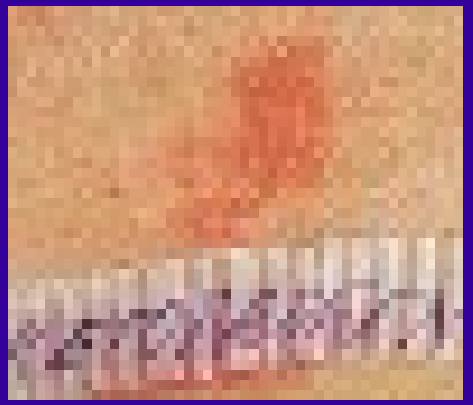
Photocontact allergy - ketoprophene - generalizace (Fastum gel)



Photocontact allergy - ketoprophene (Fastum gel)



Patch test - alergická reakce na Fastum gel



Patch test - alergická reakce na ketoprophene

Tea Tree Oil

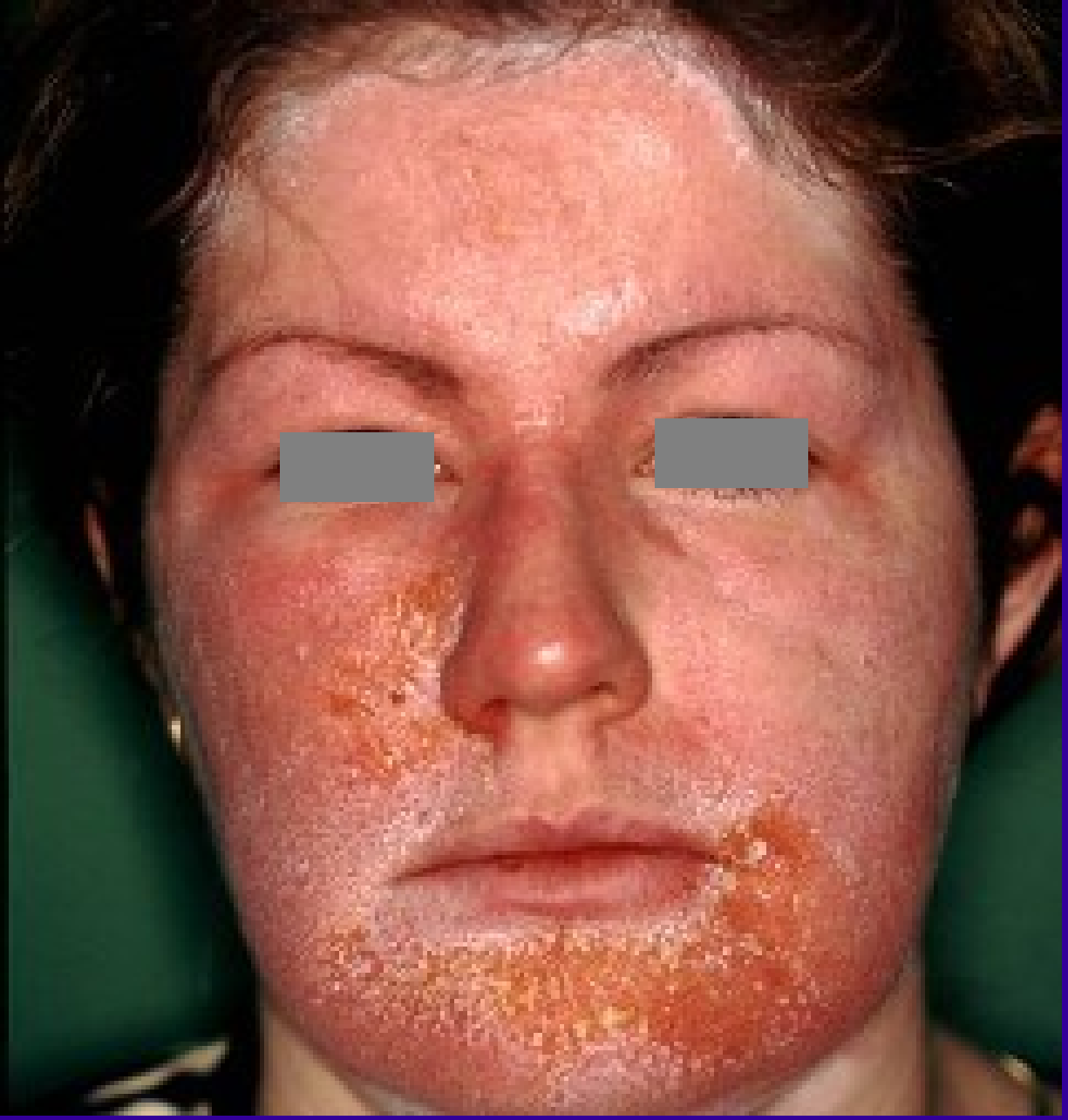
- source: leaves of the tea tree (Melaleuca alternifolia)

- occurence: Australia, Spain, Portugal
- use: folk /traditional/ medicine
- effects: antiseptic
  antifungal
  antibacterial

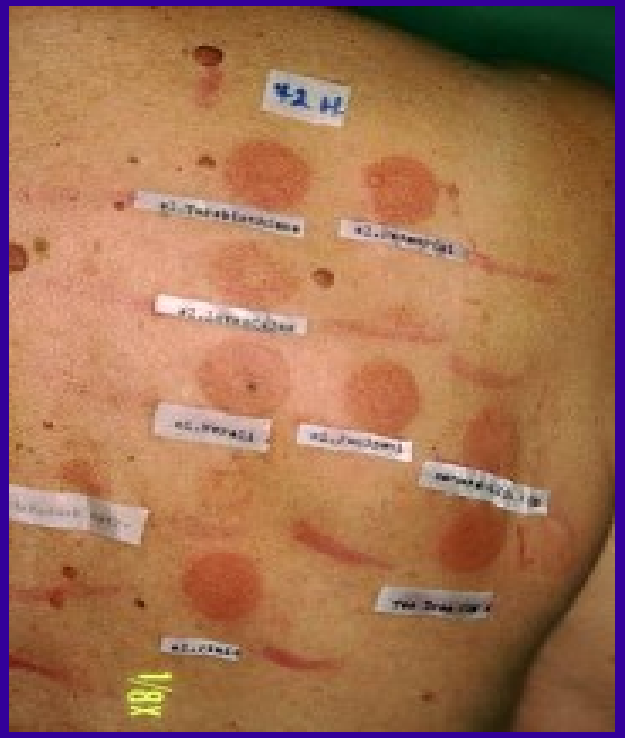
Components of Tea Tree Oil

Mixture of mono and sesquiterpens

- Terpinen-4-ol 30-45%
- Terpinen 10-28%
- Terpinen 5-13%
- Terpineol 1,5-8%
- Terpinolen 1,5-5%
- Pinen 1-6%
- Cymene 0,5-12%
- d-Limonen 0,5-4%
- 1,8 Cineol 0-15%
- Cadinen stopa-8%
- Aromadendren stopa-7%
- Sabinen stopa-3,5%
- Globulol stopa-3%
- Viridiflorol stopa-1,5%
- -Caren stopa-0,2%



Allergic contact dermatitis – tea tree oil
(cosmetic preparations)



Patch tests – contact allergy to tea tree oil and other etheric oils

Plant extracts
family of Compositae

main allergens - sesquiterpenolaktons

Extr. Chamomillae - chamomile

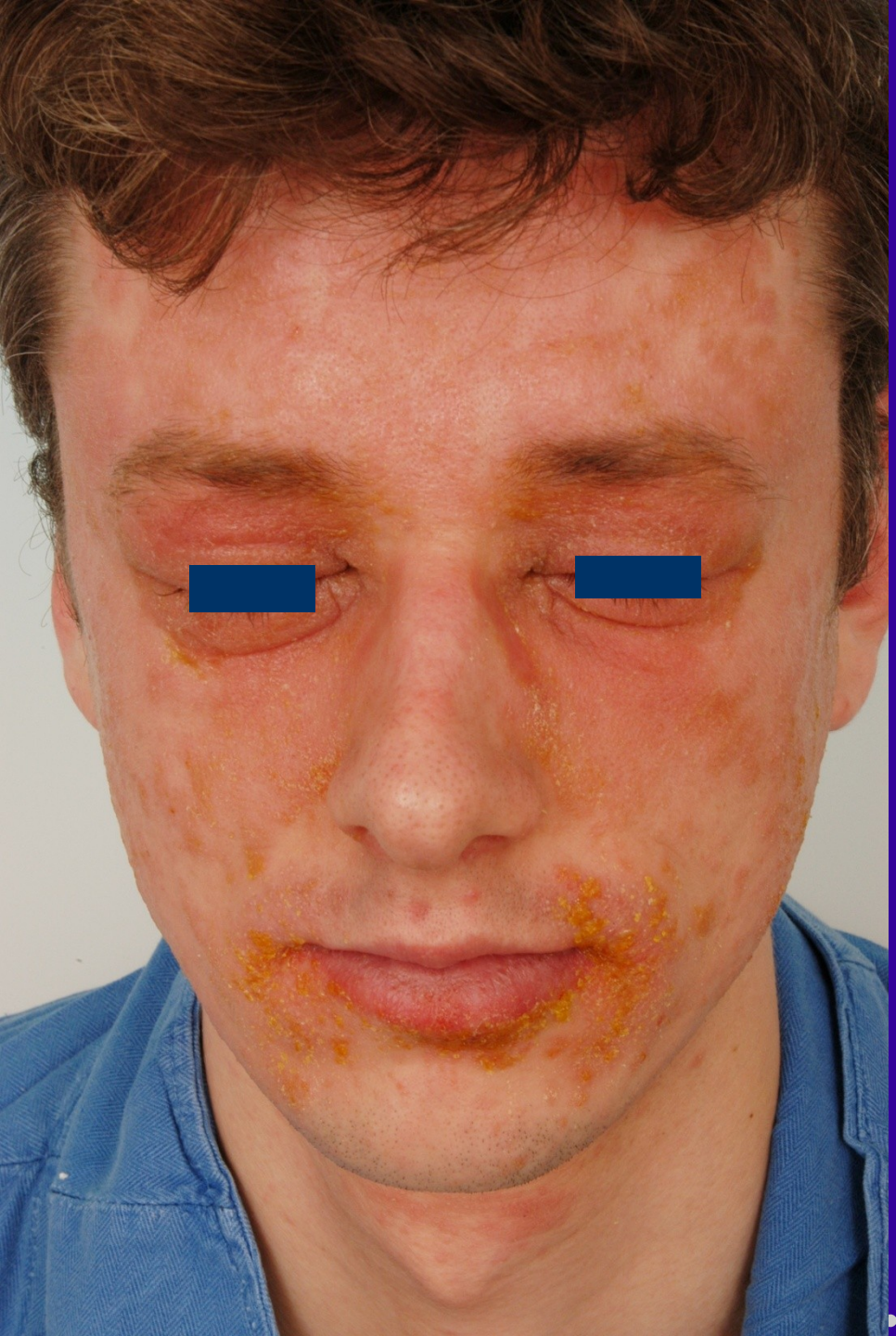
Extr. Calendulae - marigold

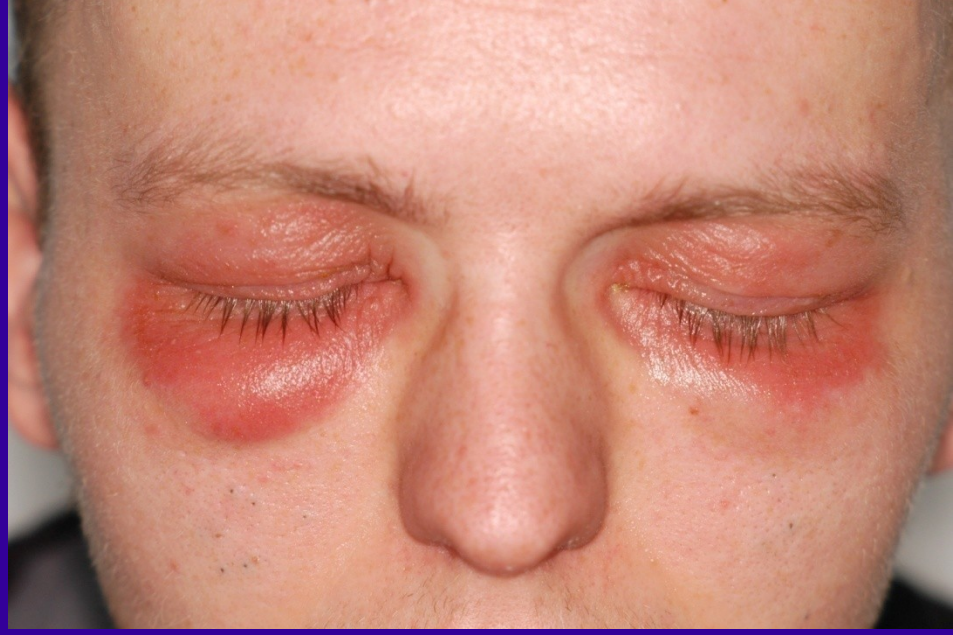
Extr. Arnicae - arnica

others:

Sunflower - Heliantus annuus,

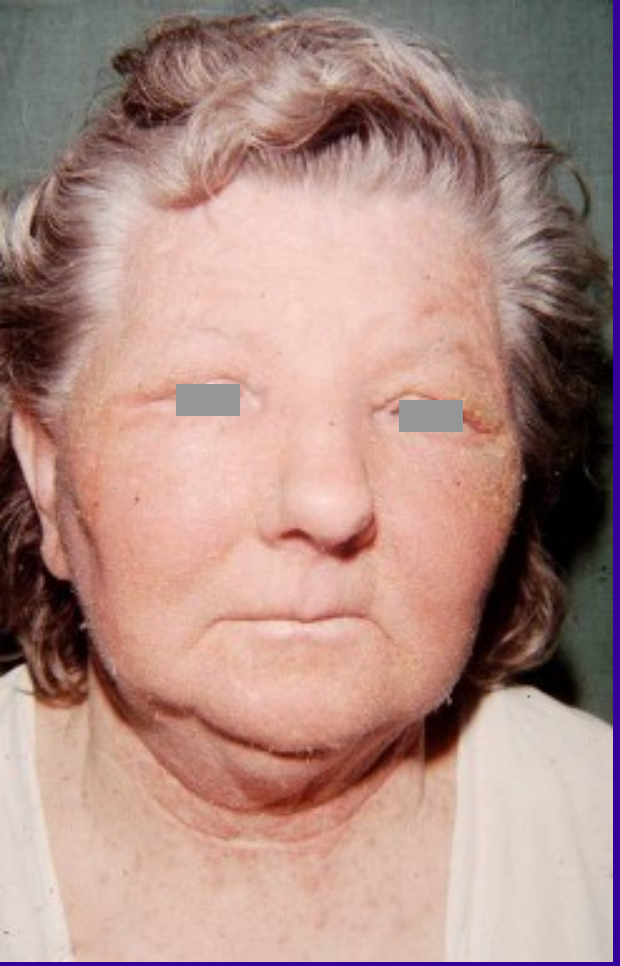
Chrysanthemum, Cynia, Astra etc.



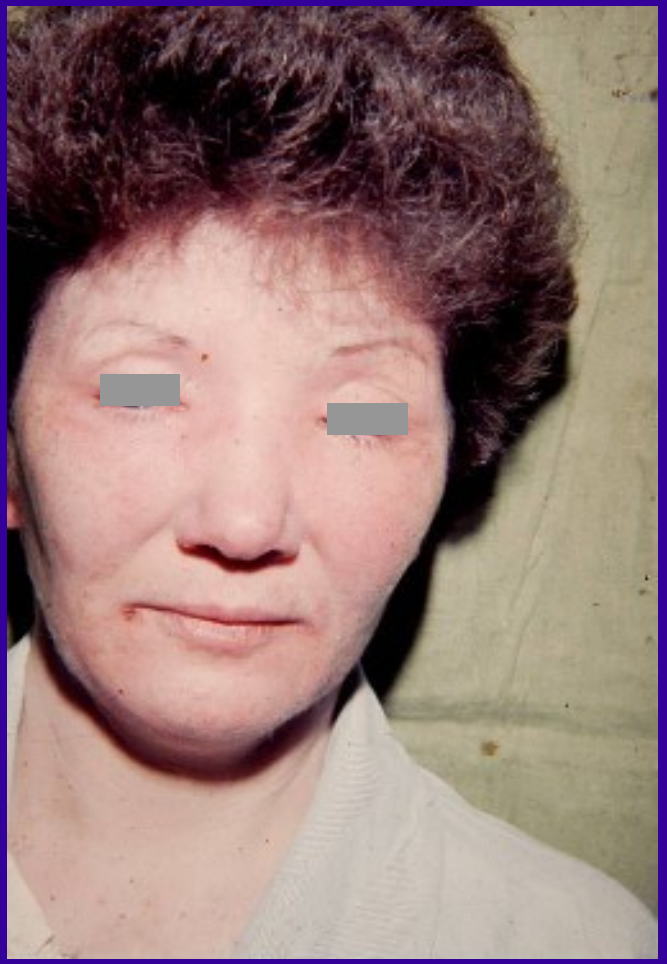


ACD to marigold (extr. Calendulae)

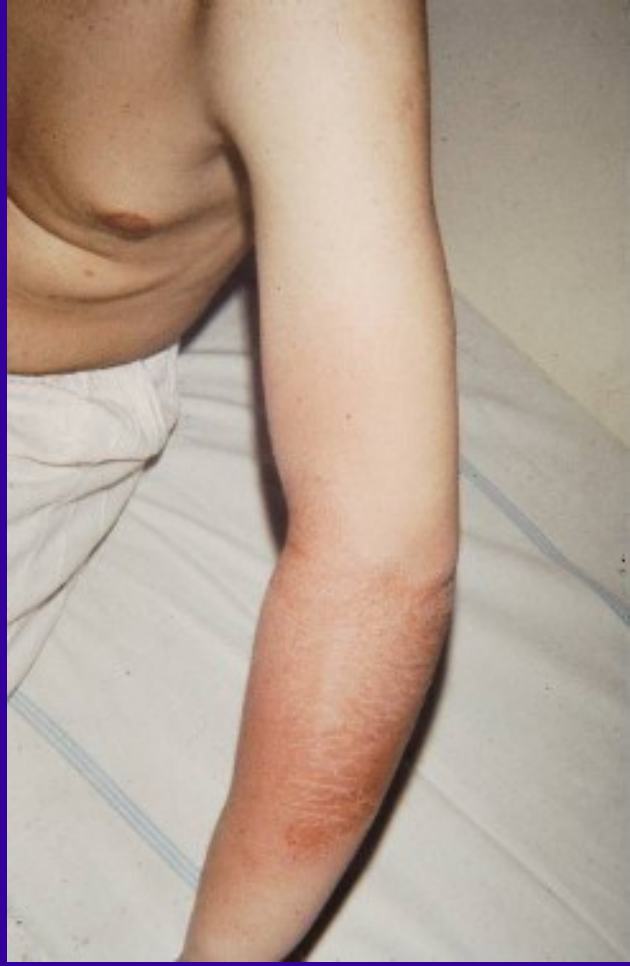
ACD to marigold in the terrain of atopic dermatitis



Eczema contactum - chloramphenicol, extr. Chamomillae



Eczema atopicum et contactum - extr. Chamomillae



Eczema contactum - Neomycin, extr. Chamomillae

Irritant contact dermatitis

- Nonallergic reaction
- Dose dependent
- Exposition to exogenous more or less toxic agent
- More common than allergic contact dermatitis

Irritant contact dermatitis

Causes:

- chemical agents:
- alkaline & acid solutions
- Organic solvents (toluene…)
- Detergents
- Disinfectants
- Food stuffs (fruit acids, mustard…)
- Even water

- physical agents: UV radiation, heat, cold, mechanical factors

Clinical picture

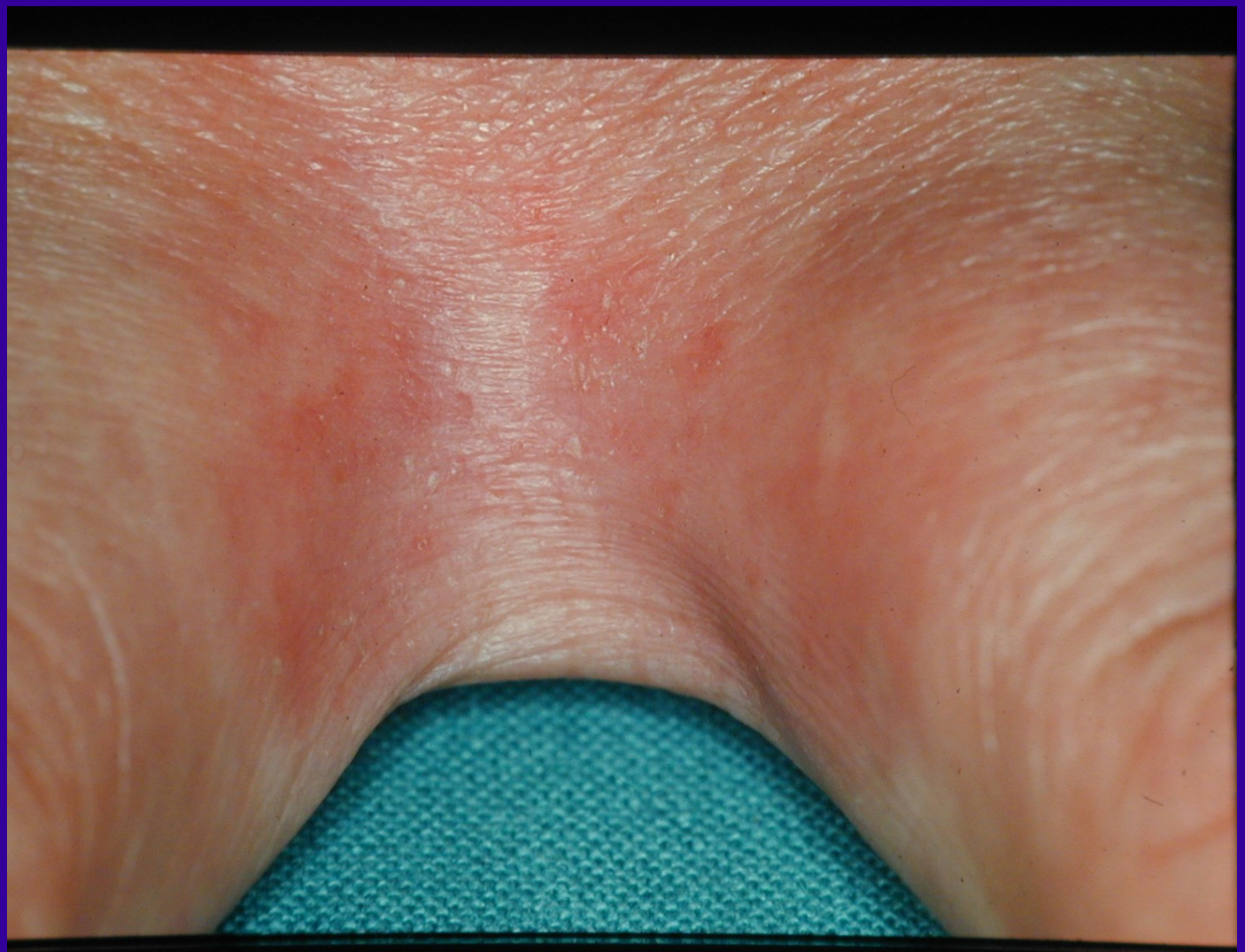
- Lesion sharply bordered
- Intensity depends on the toxicity of the subsance (more toxic.. more acute reaction)
- Toxic agents:

redness – swelling - blisters - necrosis

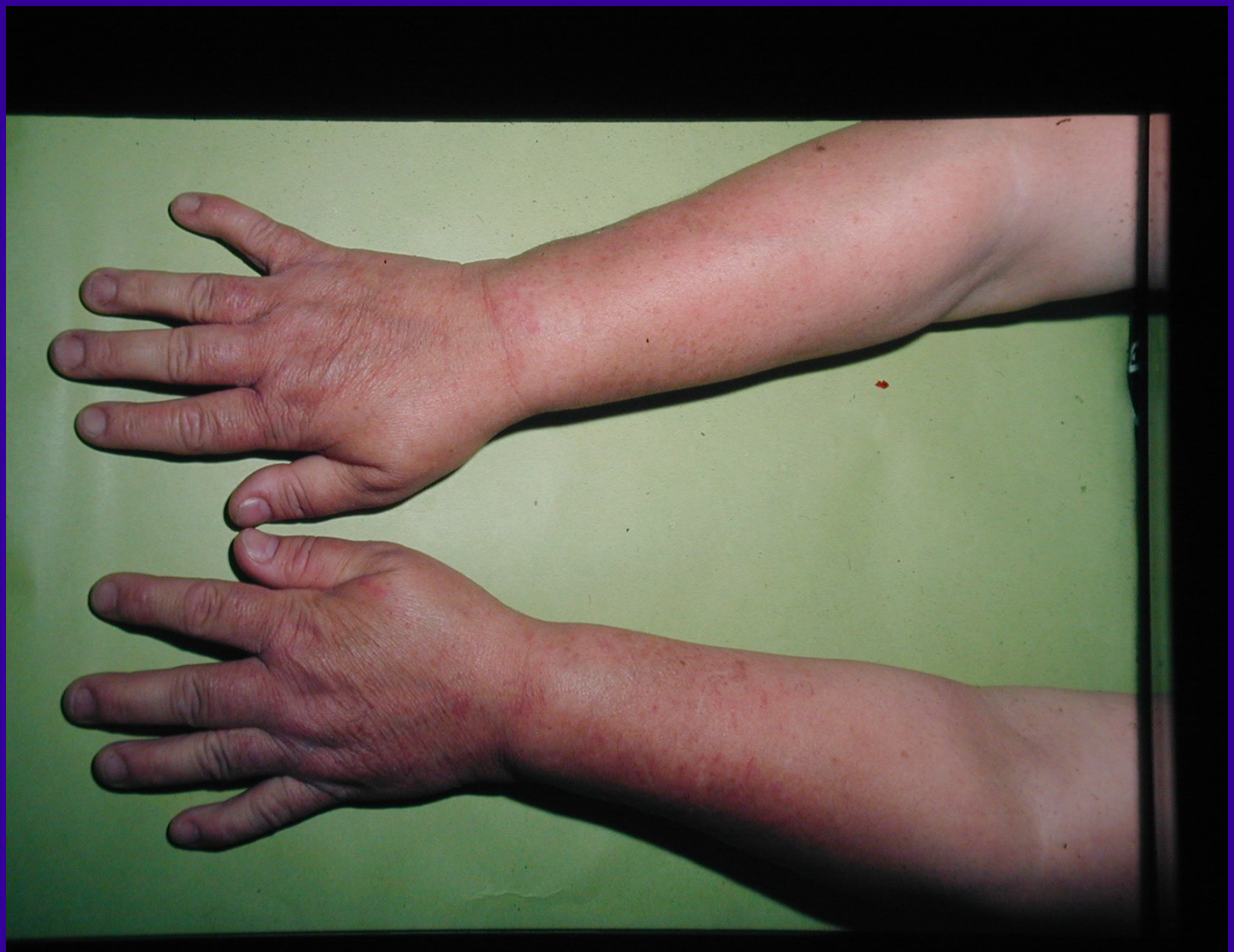
- Less toxic agents – chronic ICD

Redness, scales, lichenification, hyperkeratosis

Acute ICD



Chronic ICD



Treatment of ACD & ICD

Topical corticosteroids

Class I - low potency CS

HCT acetate (HCT ung.), DXM acetate (DXM crm.)

Class II mid-potent CS

HCT butyrate (Locoid crm.,lotio), TMC acetonid (TMC crm.), alclomethason (Afloderm crm, ung.)

prednikarbate (Dermatop crm., ung.)

methylprednisolon aceponate (Advantan crm.)

Class III - potent CS

betamethasone dipropionate (Beloderm,Diprosone crm.)

fluocinolone acetonide (Gelargin gel,ung.)

momethason furoate (Elocom crm., ung., lotio)

Class IV – very potent CS

clobethasol propionate (Dermovate crm., ung)

Antihistamines, systemic corticosreroids – short courses

Atopic dermatitis

- strongly pruritic chronic or chronically relapsing non-infectious dermatitis with variable morphology and clinical course, usually starting during early childhood

- often associated with positive personal or family history in terms of allergic rhinitis, conjunctivitis and bronchial asthma.
- genetic predisposition
- In about 80% associated with ↑ IgE levels

Atopic dermatitis - epidemiology

Incidence in population: 0,5 - 5%

(higher incidence – scandinavian countries)

infants	**16%**
children under 2 y	**14%**
children under 14 y	**12%**
adults	**2%**

Atopic dermatitis

two forms, same clinical picture

extrinsic 80% — **elevated IgE**

sensitization to airborne and/or food allergens (sIgE)

- association with allergic rhinoconjunctivitis and/or allergic asthma

intrinsic 20% — **normal levels of IgE**

skin barrier disturbace

Etiology of AD: unknown

basis = genetic predisposition

1) skin barrier disturbance
2) hyperreactivity of the skin

environmental triggers:

1) irritant substances, allergens
2) stress
3) many others ….

I. skin barrier disturbance

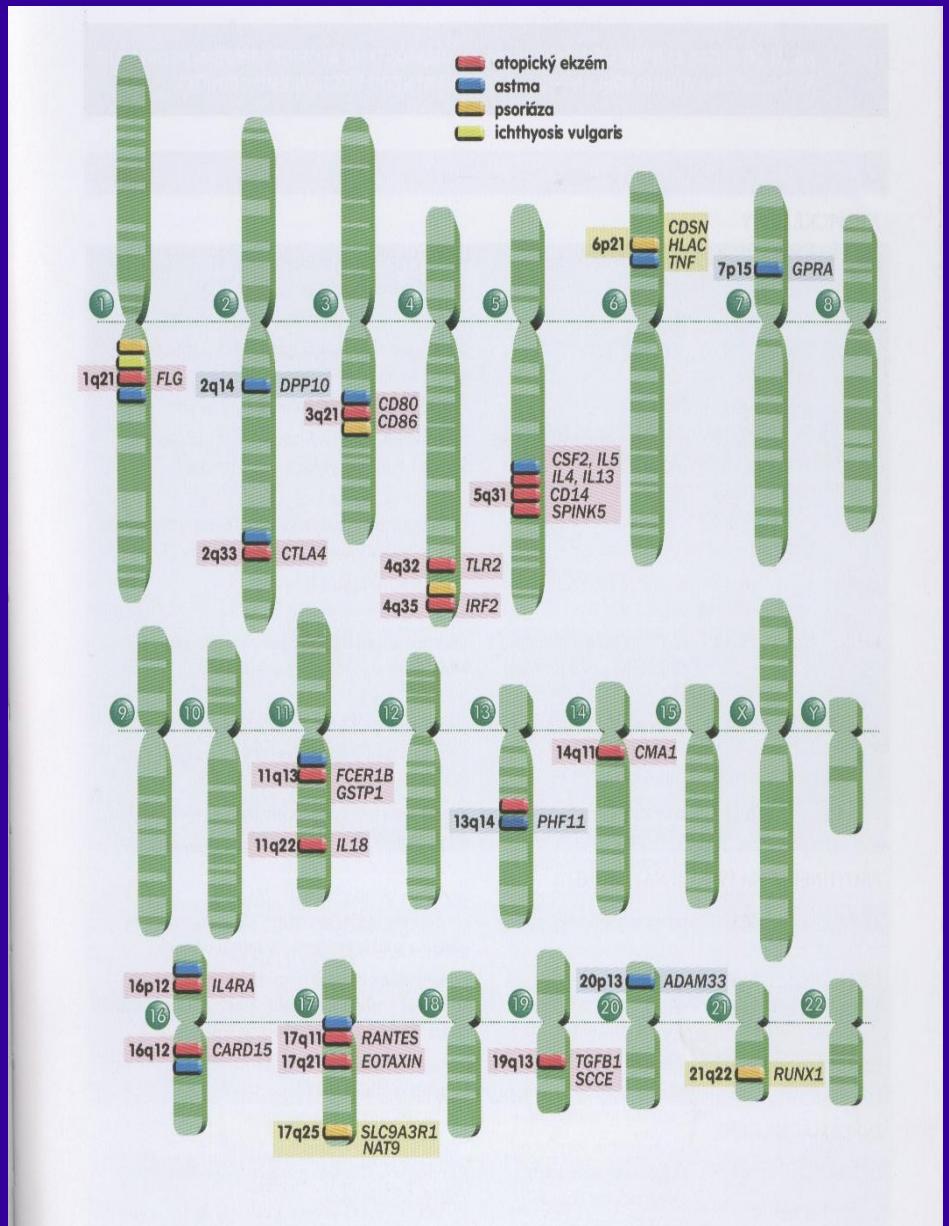
Genetically conditioned:

Filaggrin: null mutation of FLG R501X and 2282del4 alleles lead to increased permeability of skin barrier and they are associated with AD (in about 50% cases), as well as with ichtyosis vulgaris

Claudin- 1, corneodesmosin

Increased activity of serin proteases

Genes involved in AD



skin barrier disturbance

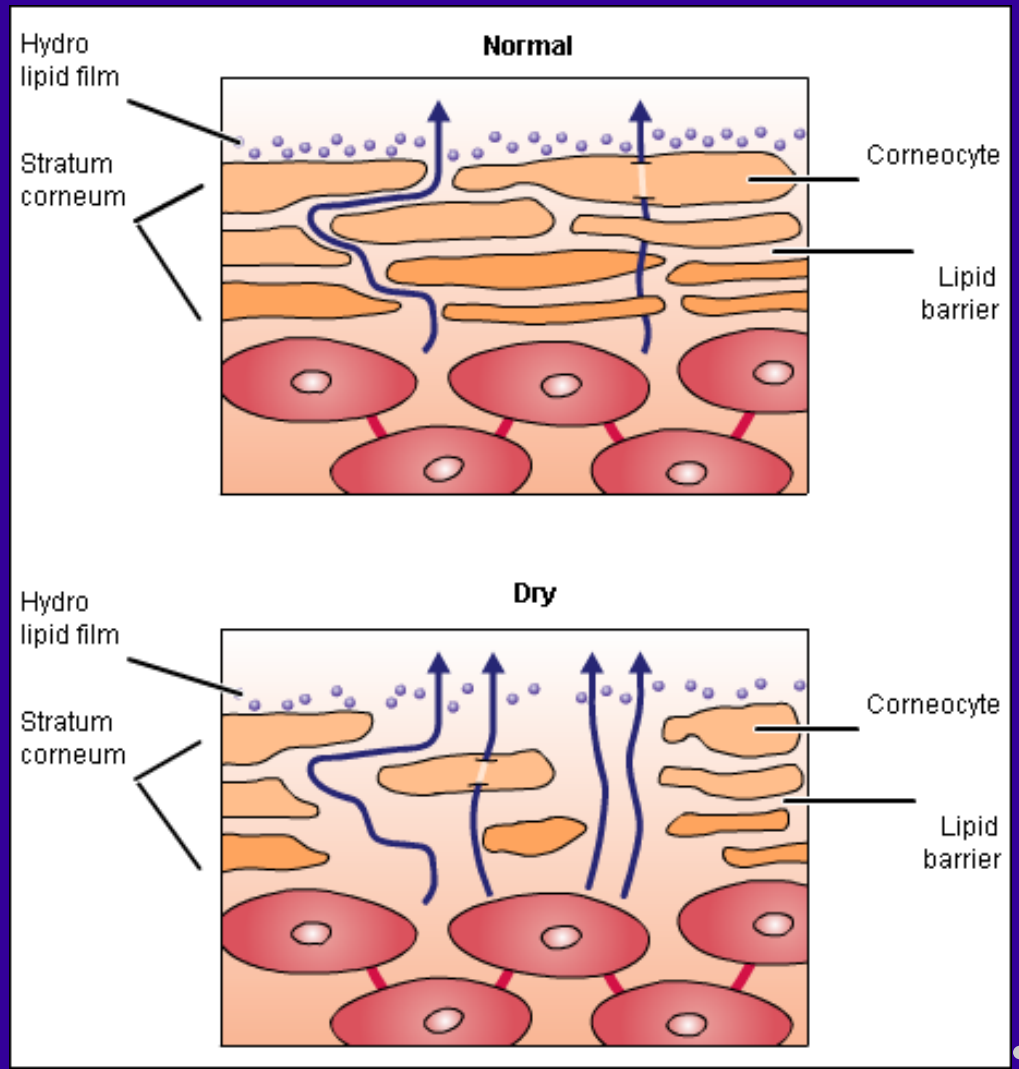
- Defective synthesis of ceramides

  (takes place in lamellar bodies in granular layer of epidermis)

  ↓

  decreased ability to bind water in the skin

skin barrier disturbance



AD and skin barrier

Defective structure and function of skin barrier

↓

insufficient hydration (TEWL ↑)

↓

dryness - xerosis

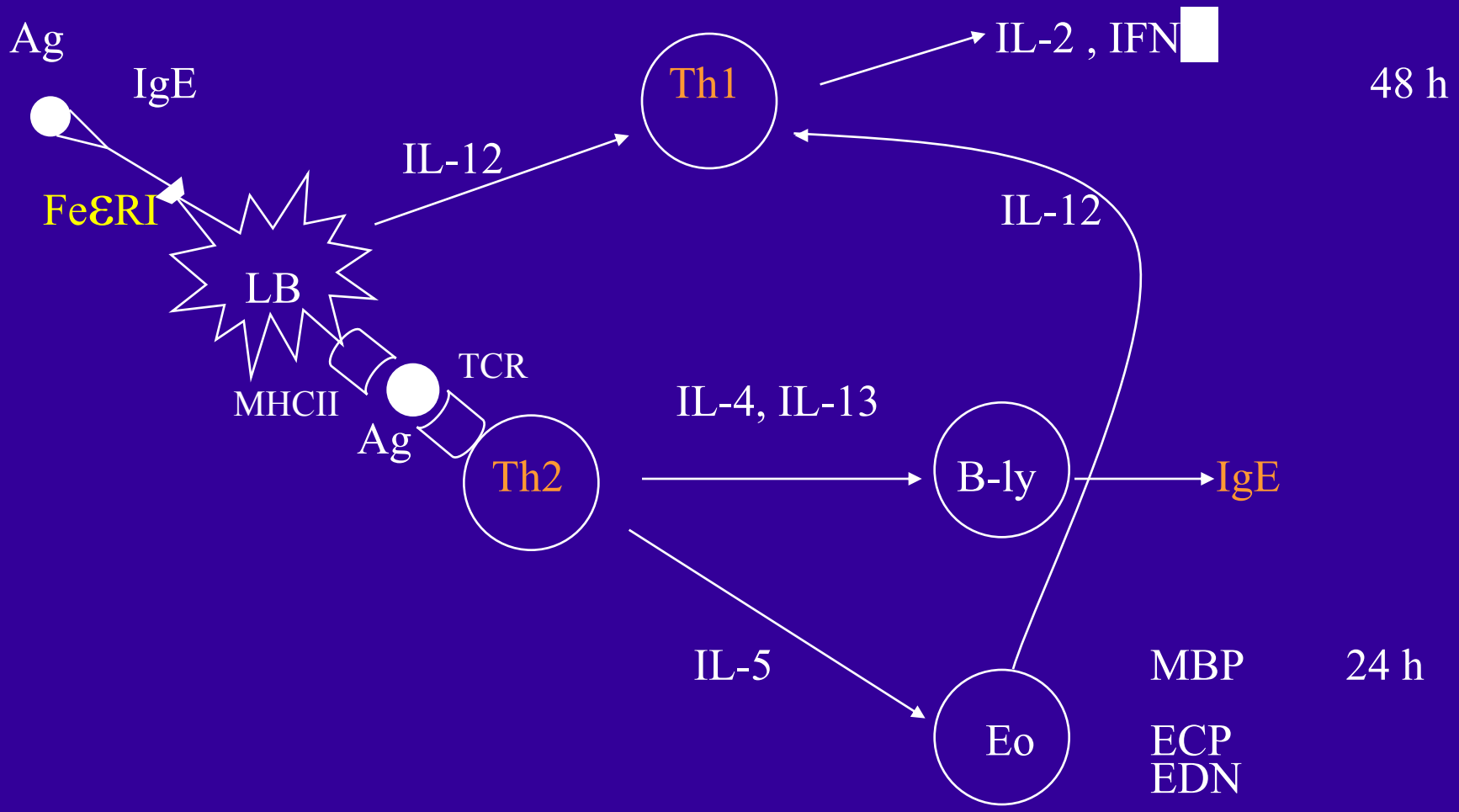
↓

increased irritability of the skin

possibility of contact sensitization

II: Immunological abnormalities in AD

biphasic model of AD (Th2 → Th1 shift)



III. Staphyloccus aureus and AD

- colonization of AD lesions in 74 - 96% atopic patients, 30 - 56% even on „healthy“ skin

Mechanisms:

- Defective skin barrier with „naked“ laminin and fibronectin enables SA binding the skin

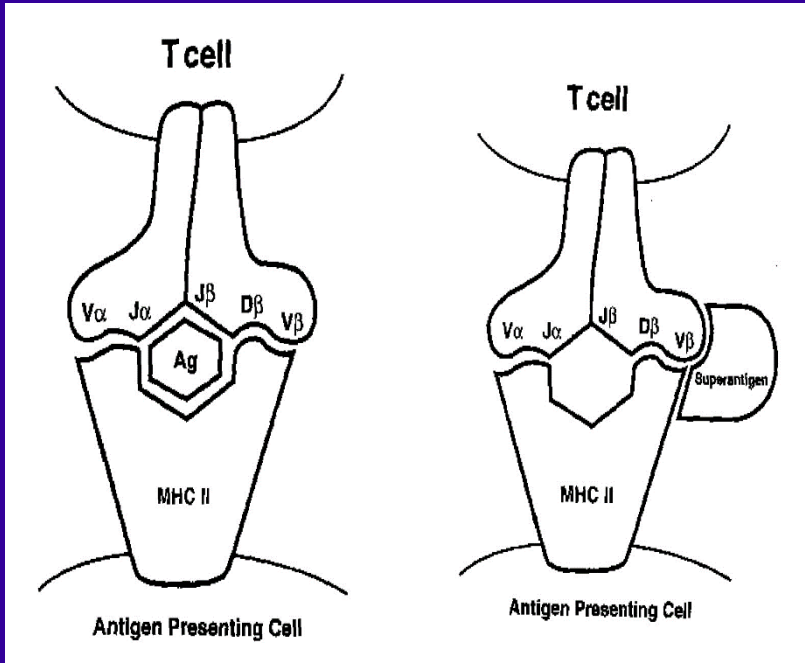
- Decreased defensive mechanisms: defective signallization via TLR 2

[redacted] defensine a kathelicidine

[redacted]roduction of IFN [redacted]

Staphyloccus aureus and AD

1) Toxic effect: staphylococcal exfoliatine
2) Stimulation of sIgE production (sIgE → stimulation of basophils → histamine)
3) superantigens: SEA- SEE a TSST-1



- without previous processing by LC
- able to bridge V chain of TC Receptor,
- not necessary exact conformity of all 5 subunits of the receptor

  1000x stimulation
- non-specific but huge stimulation of Tly

  (1 SA even 20% of circulating lymph.)

Triggering and maintaining factors of AD

Allergy (house dust mites, pollen, pets, molds, foods – milk, eggs, wheat, soya, nuts, fish)

Microbes – Staphylococcus aureus

Irritant substances (water,detergents etc.)

- climatic (temperature, wind, low humidity ..)

Psychological stress

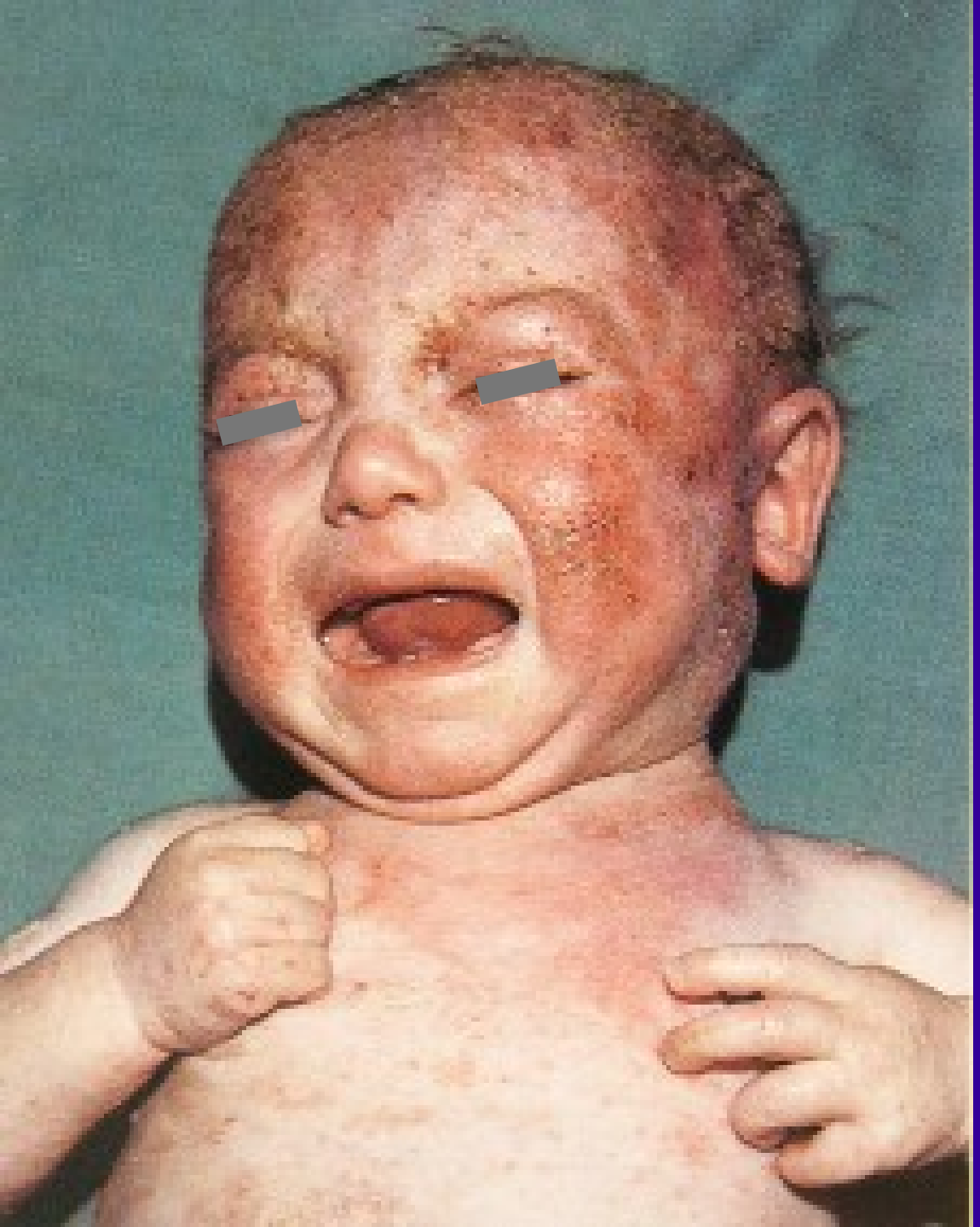
Clinical picture of AD

AD in infants

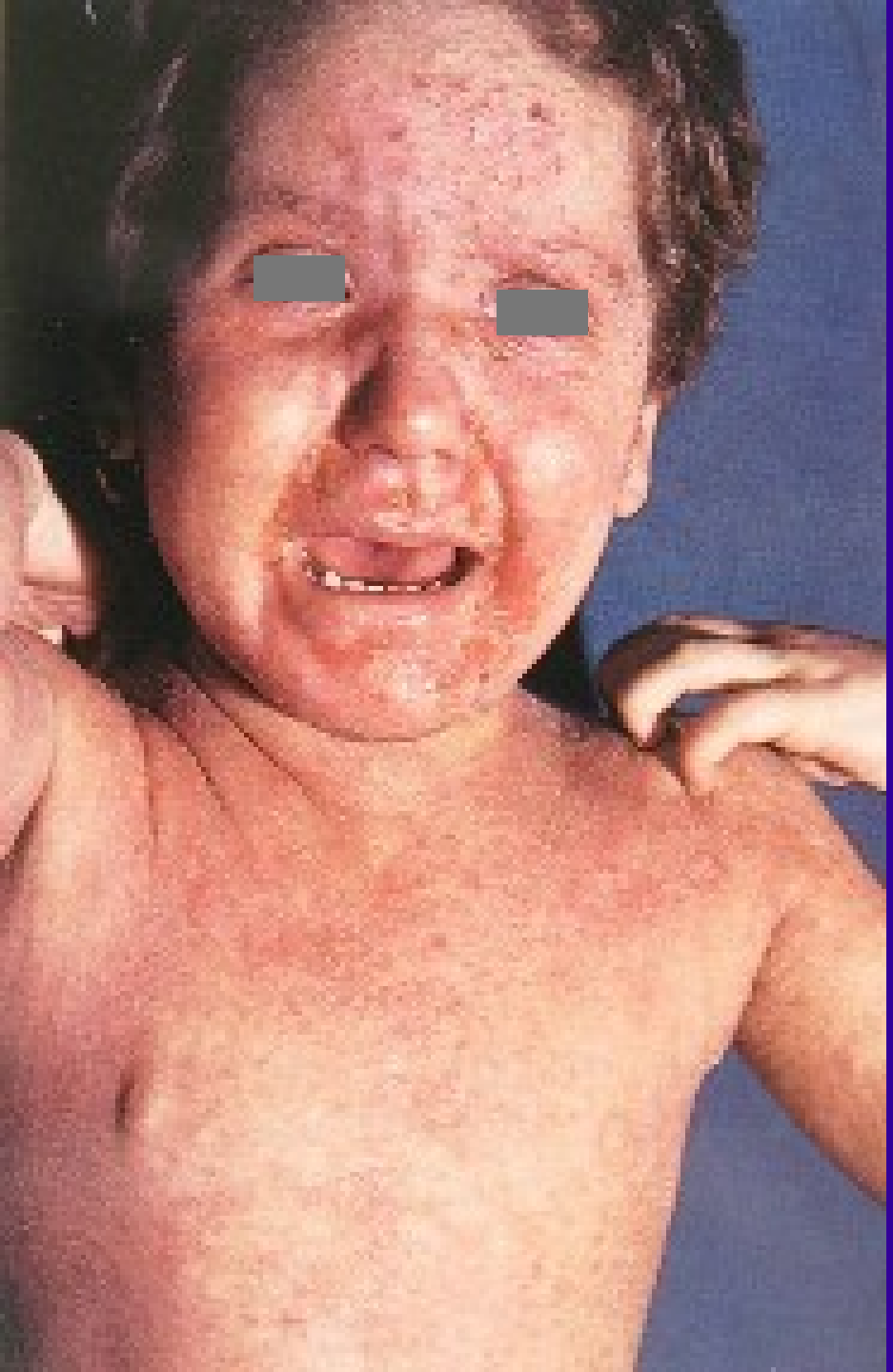
Exudative form – acute eczema (oozing, crusting)

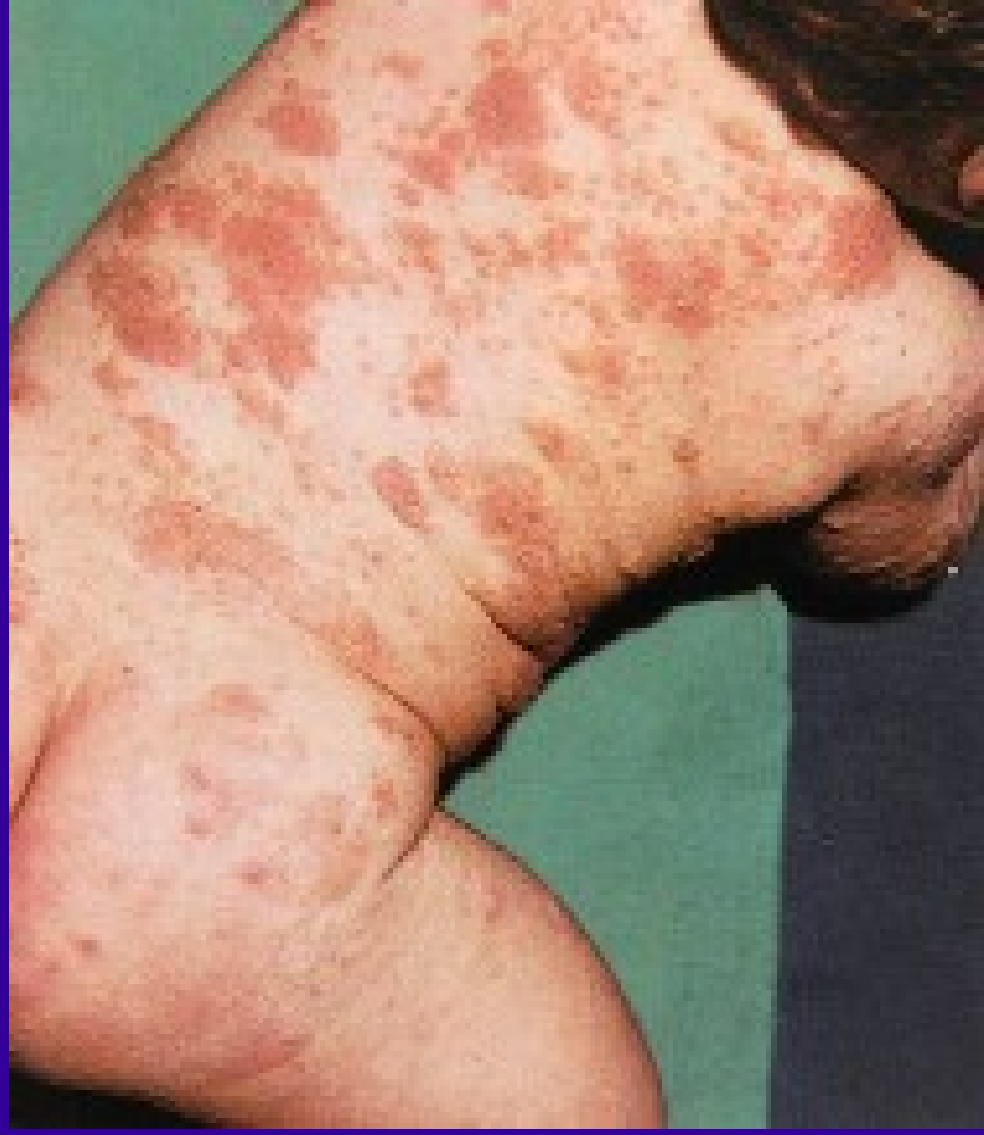
location
- **perioral**
- **periorbital**

possibility of spreading - erythroderma



Atopic dermatitis – Infant AD





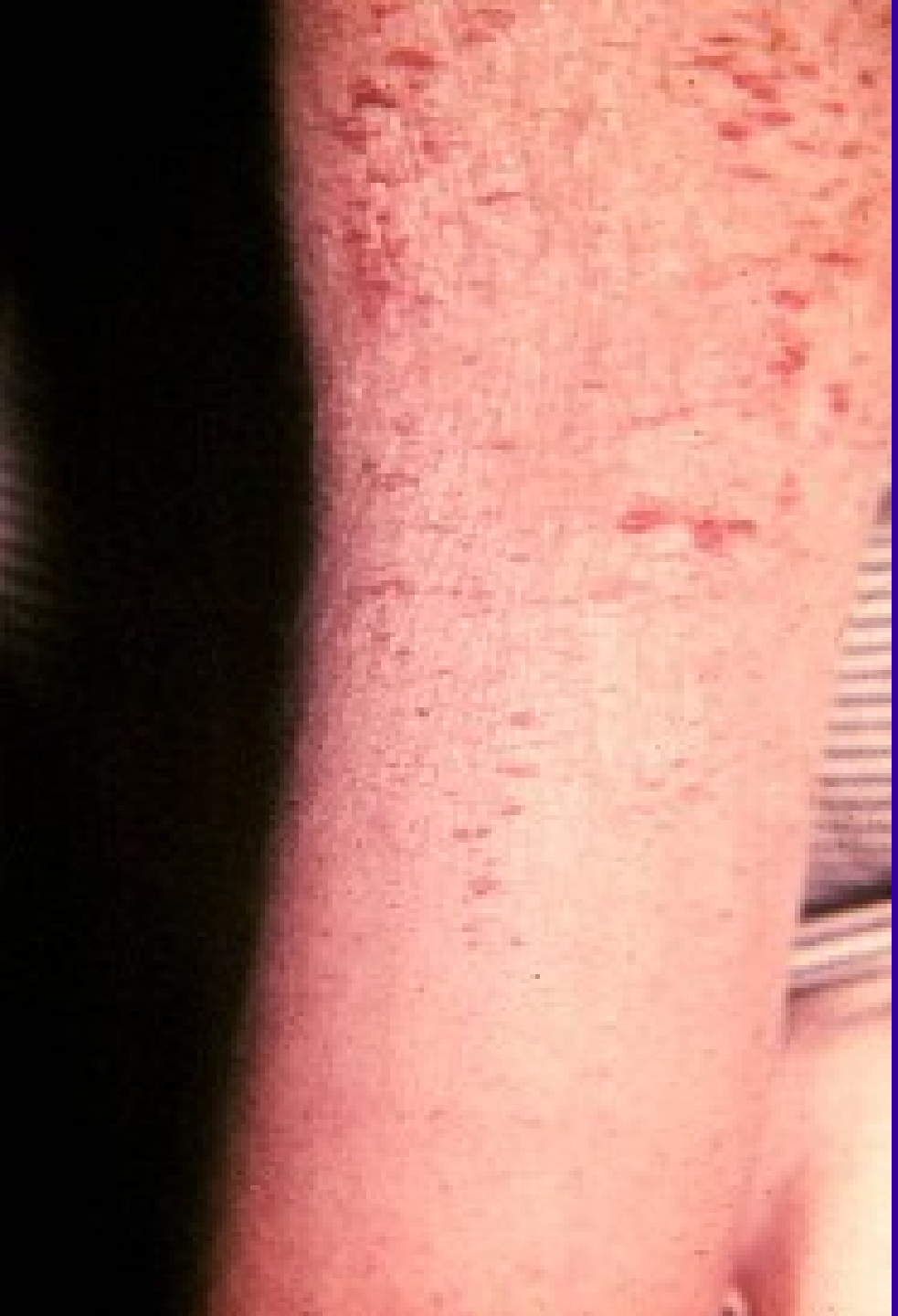
Infant AD

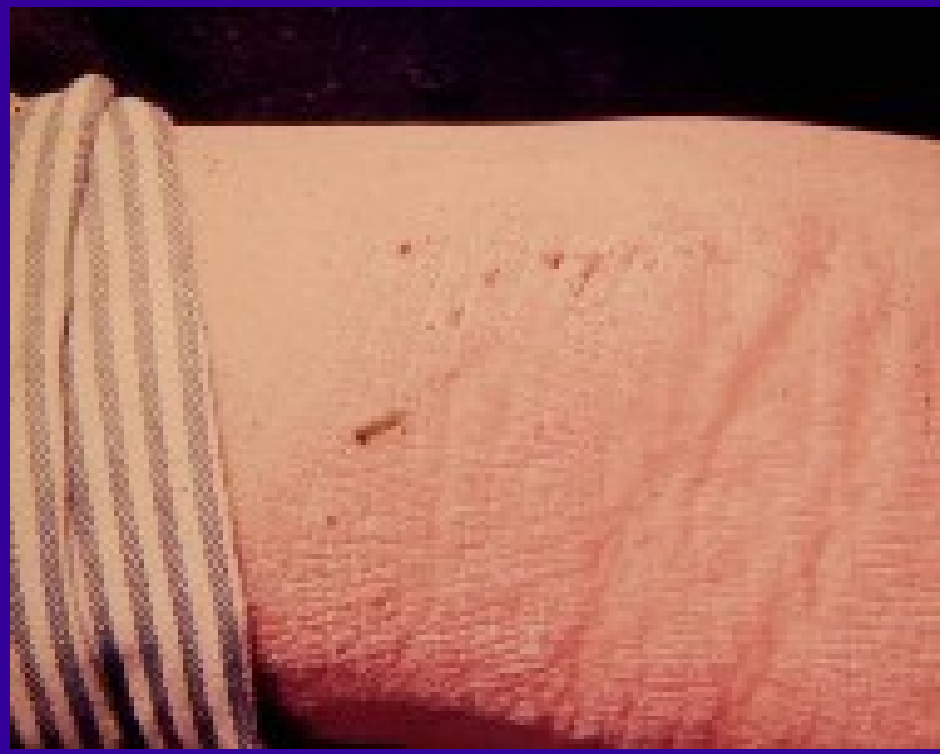
Clinical picture of AD

AD in children and adolescents

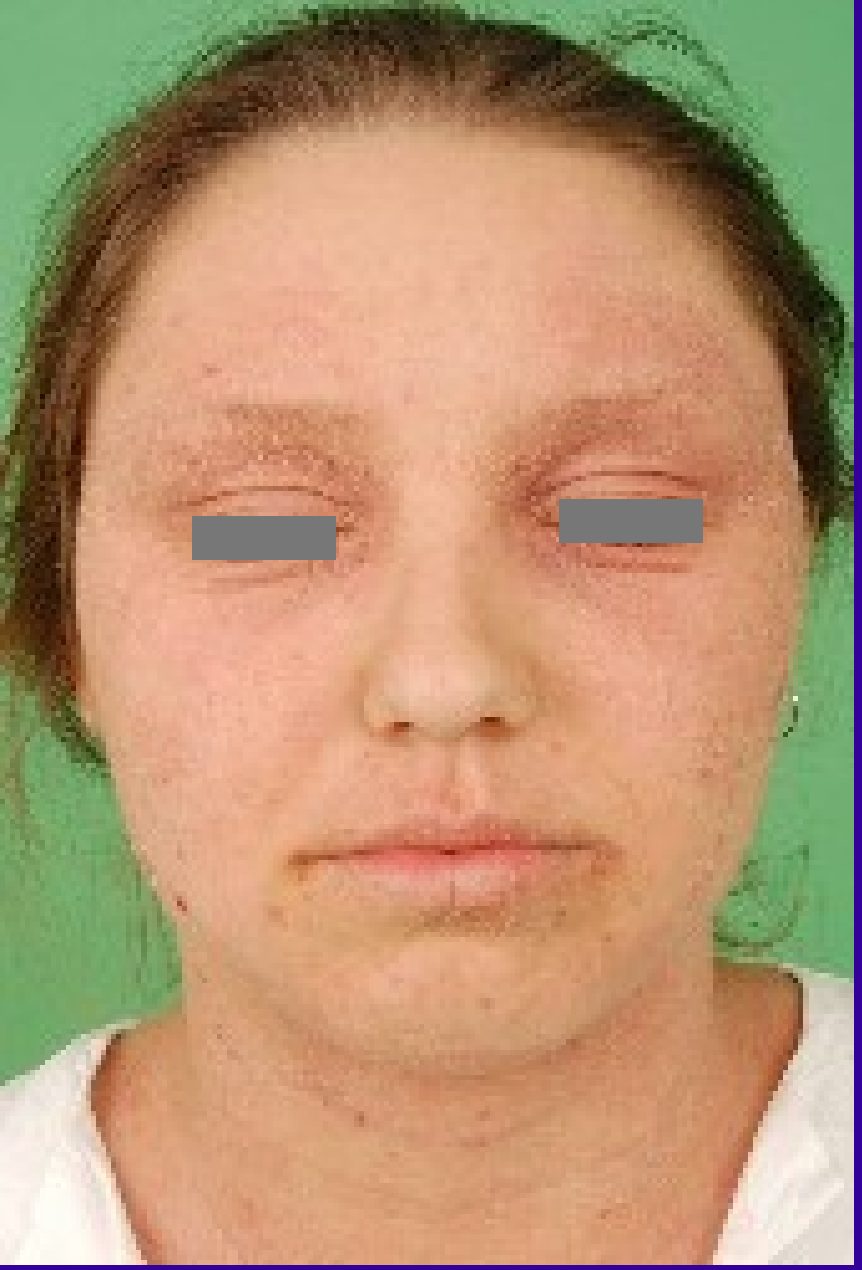
Decrease of exudation - lichenification

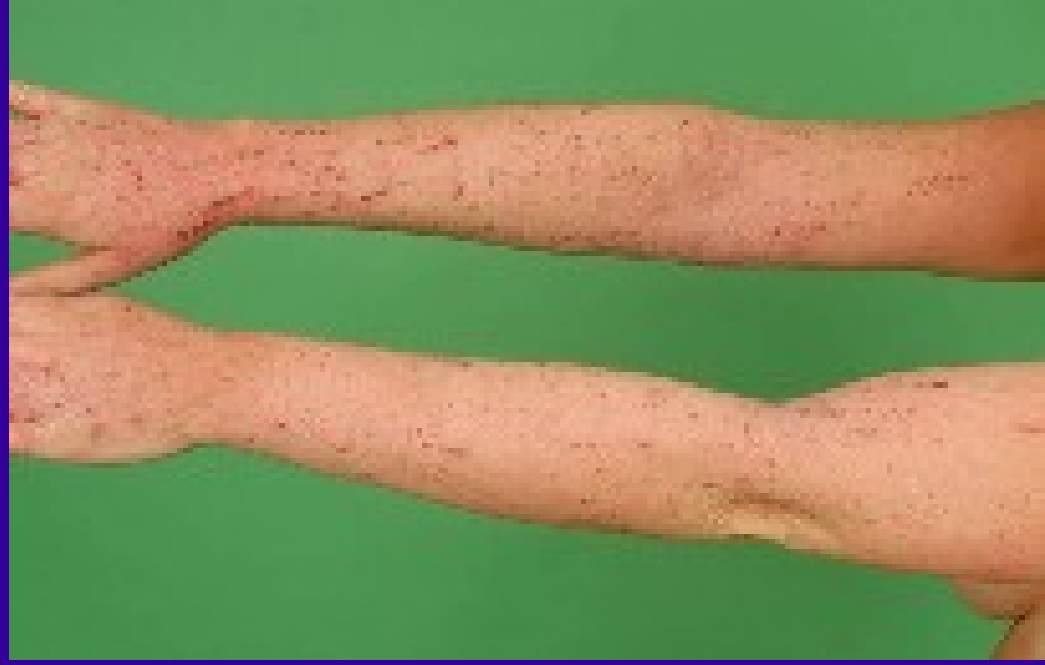
- most often – flexural eczema
  - facial eczema
- less often - erythroderma

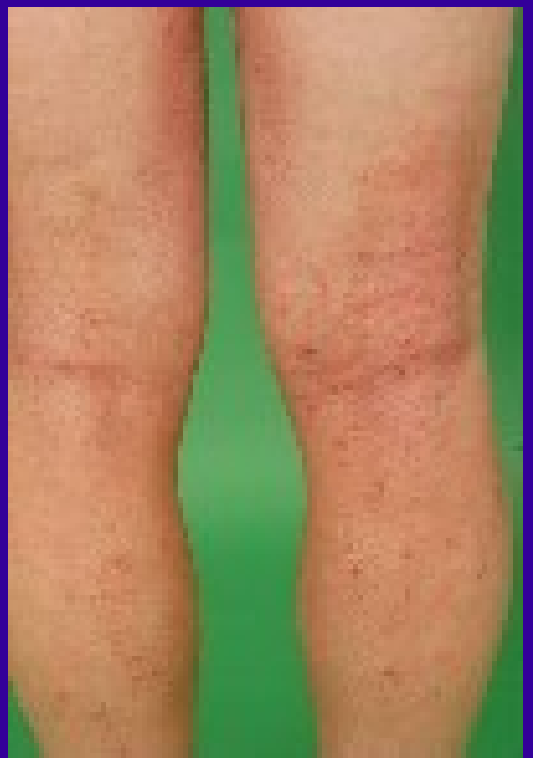


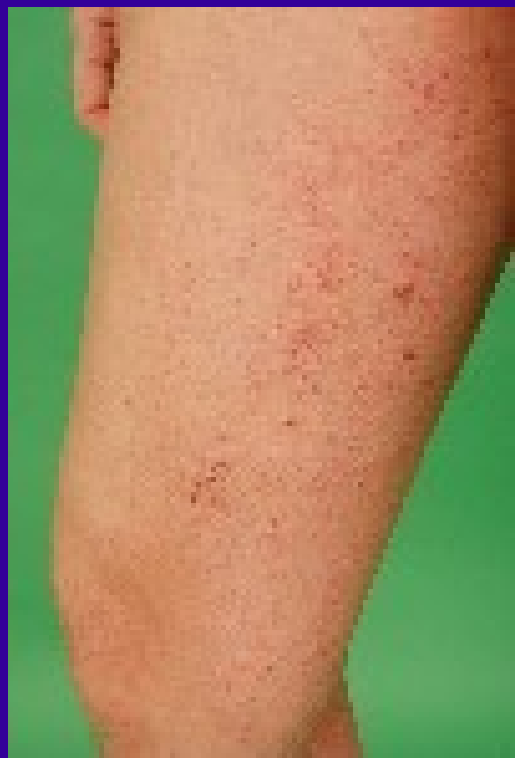


Atopic dermatitis – flexural eczema









Atopic dermatitis – erythrodermic form

Clinical picture of AD

AD in adults

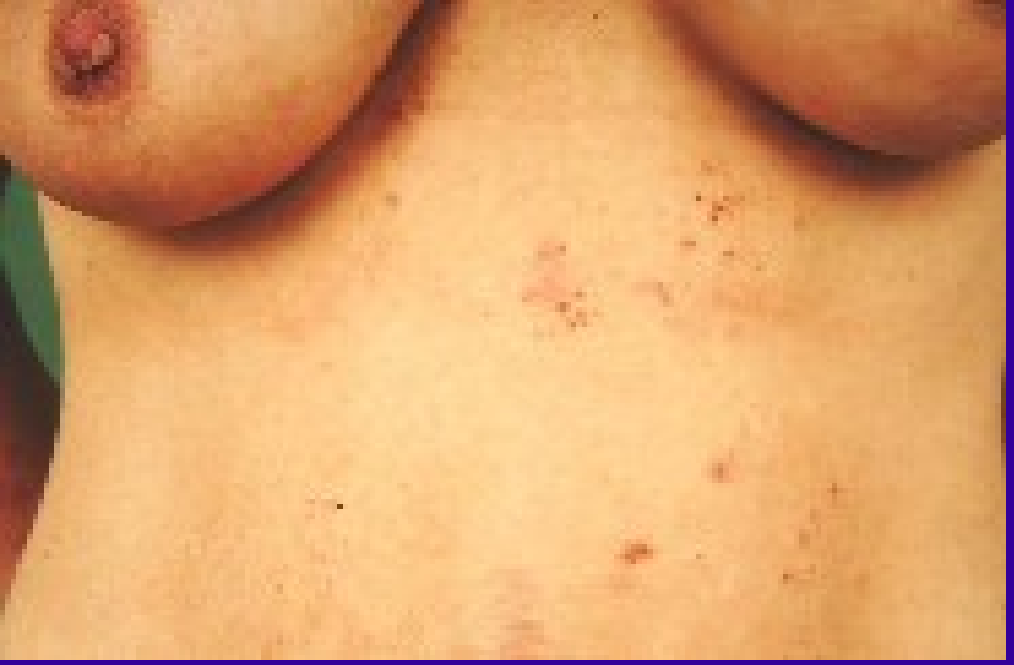
(about 15% of cases appear after puberty)

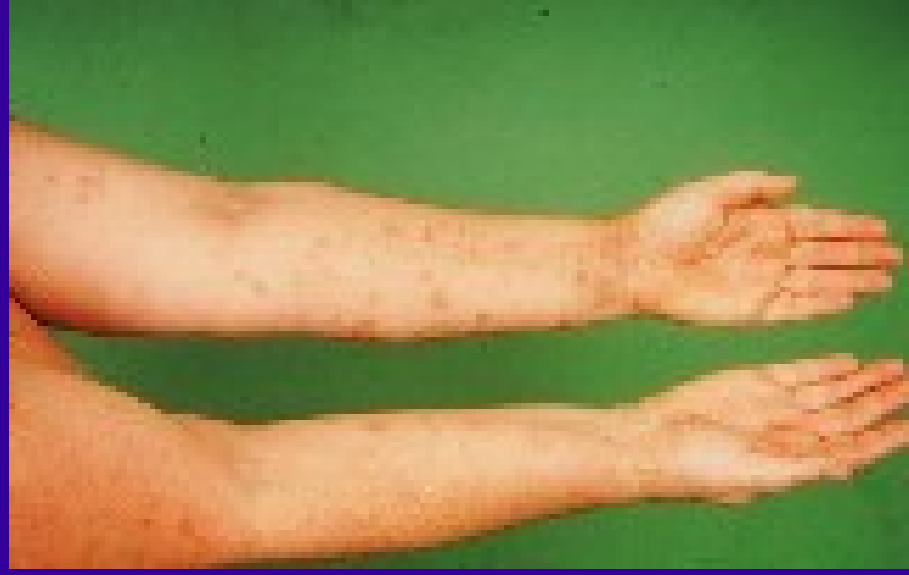
- **flexural**
- **pruriginous**
- **neurodermitic**
- **erythrodermic**

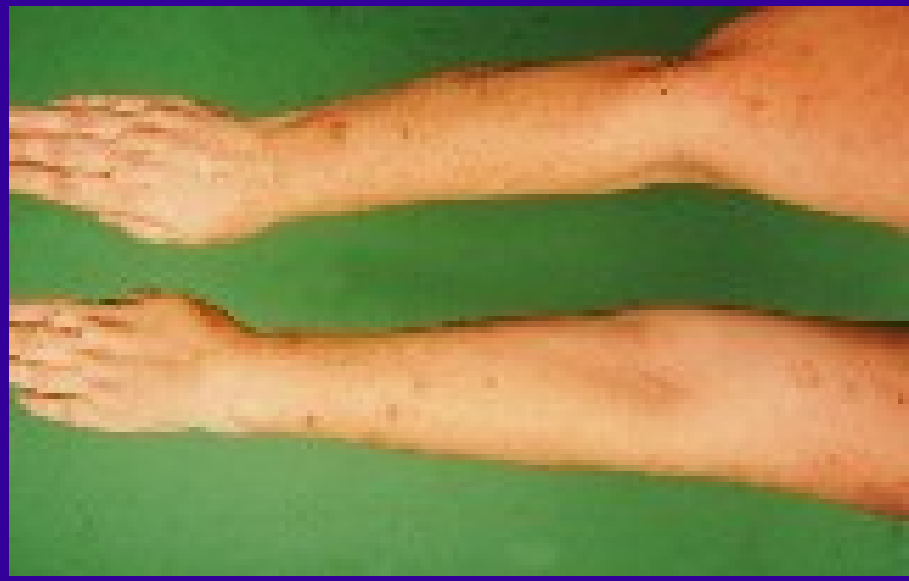
chronic course

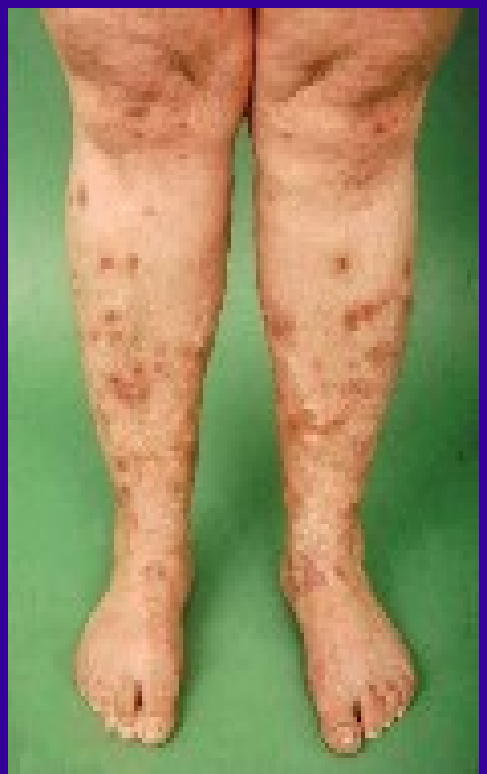
acute flares

possible

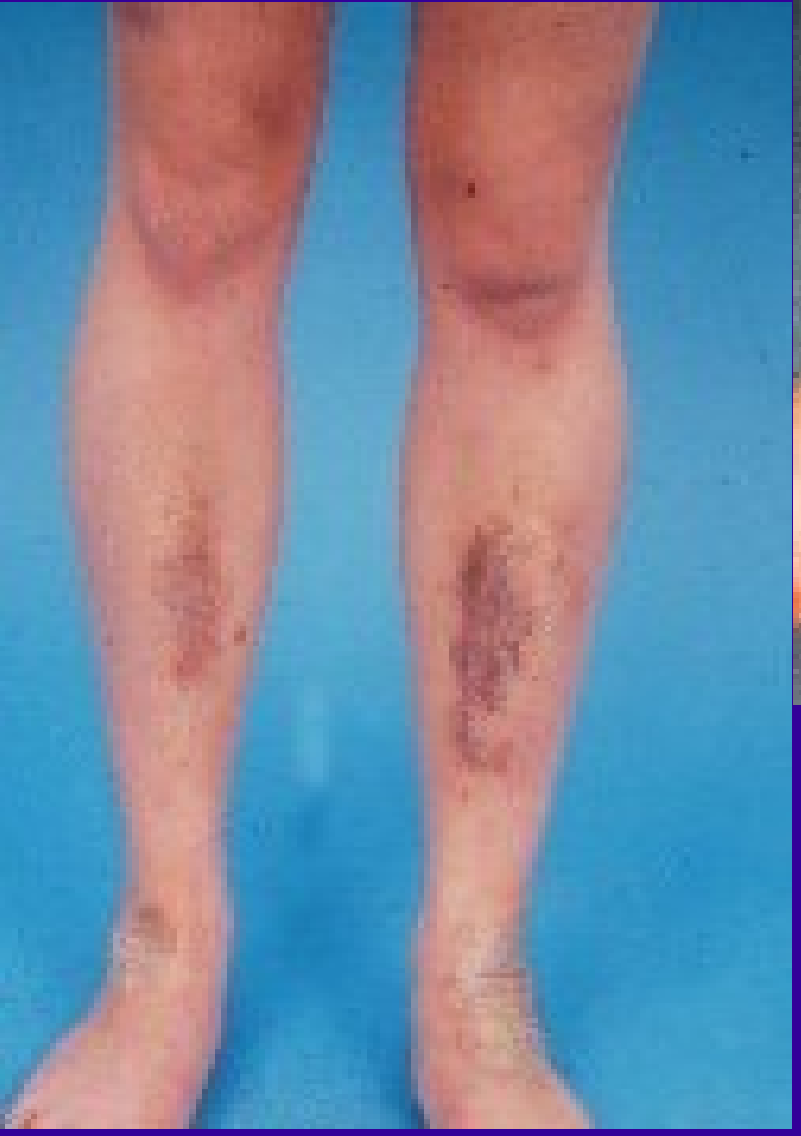


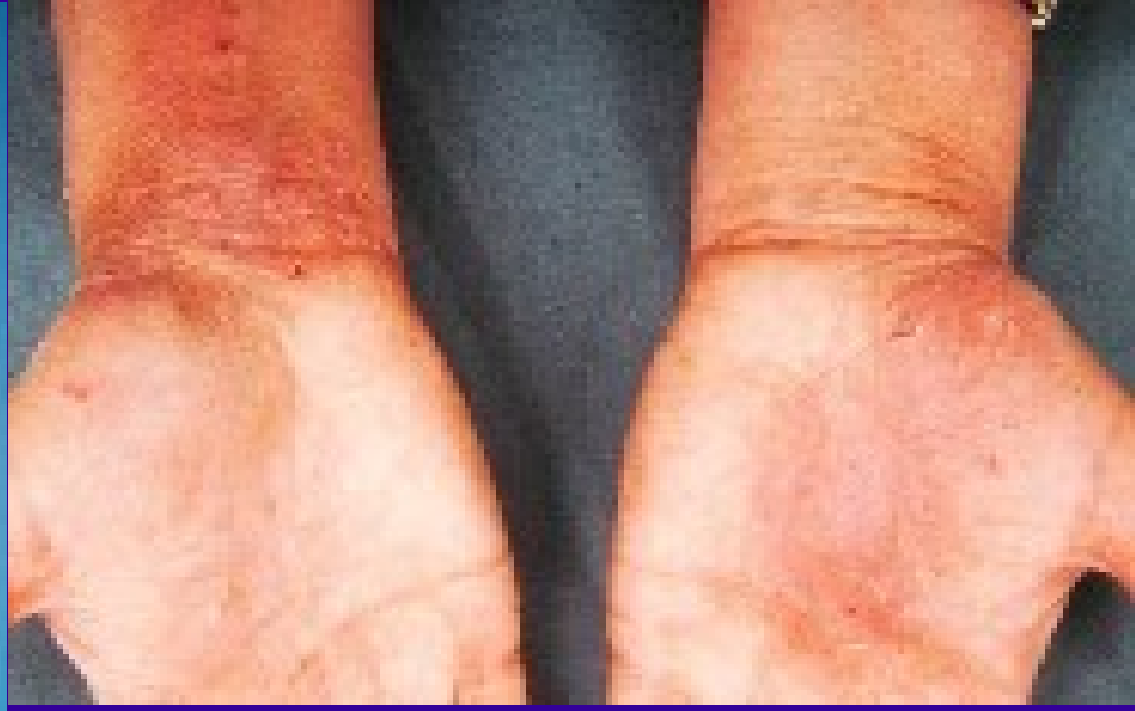


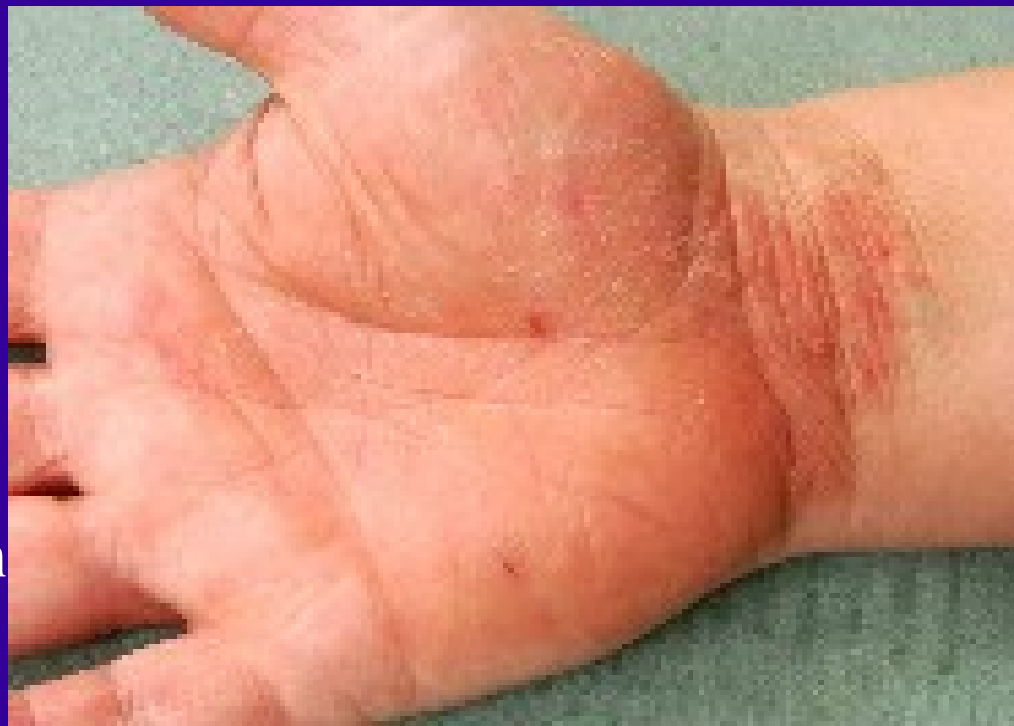




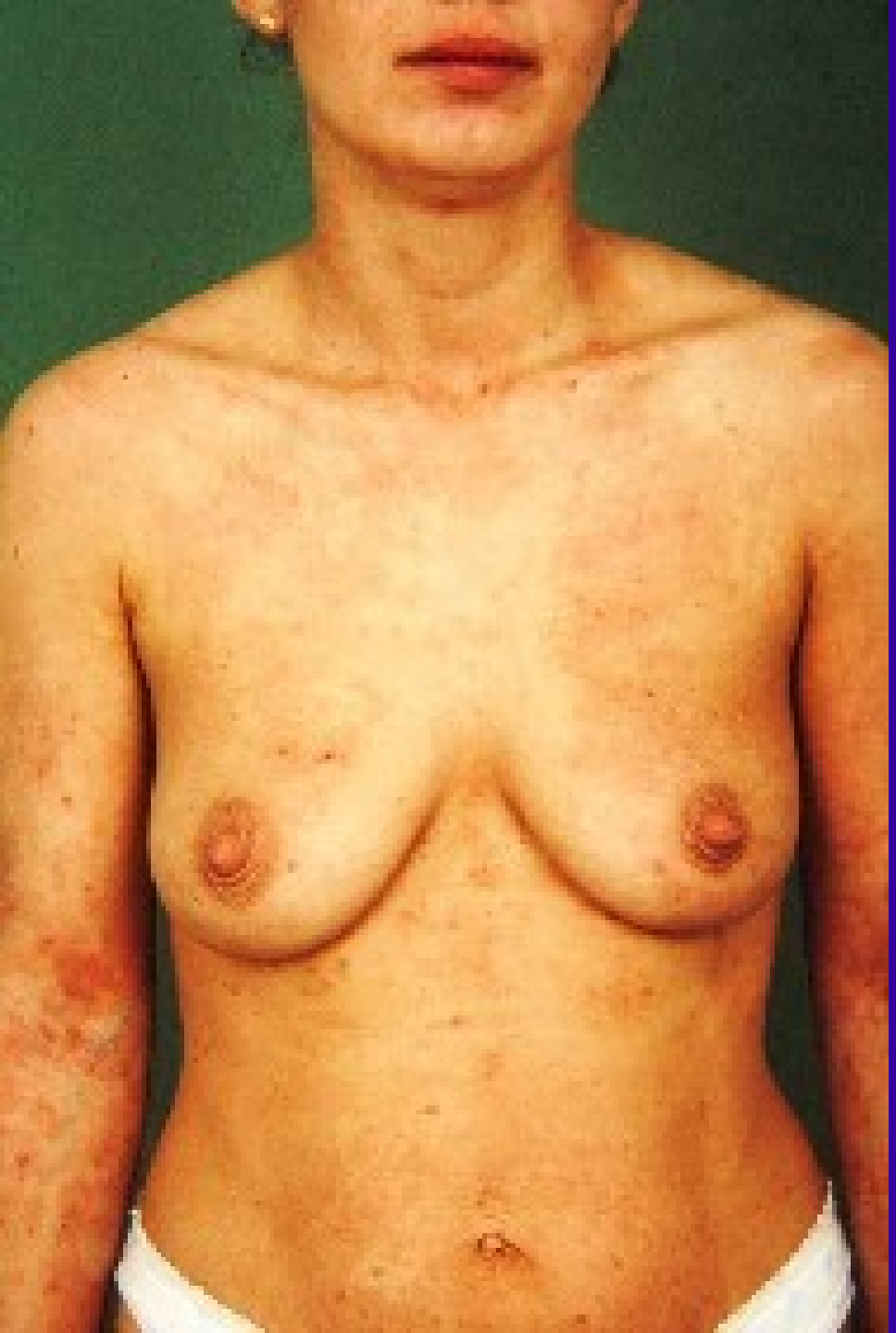
Adult AD – pruriginous form

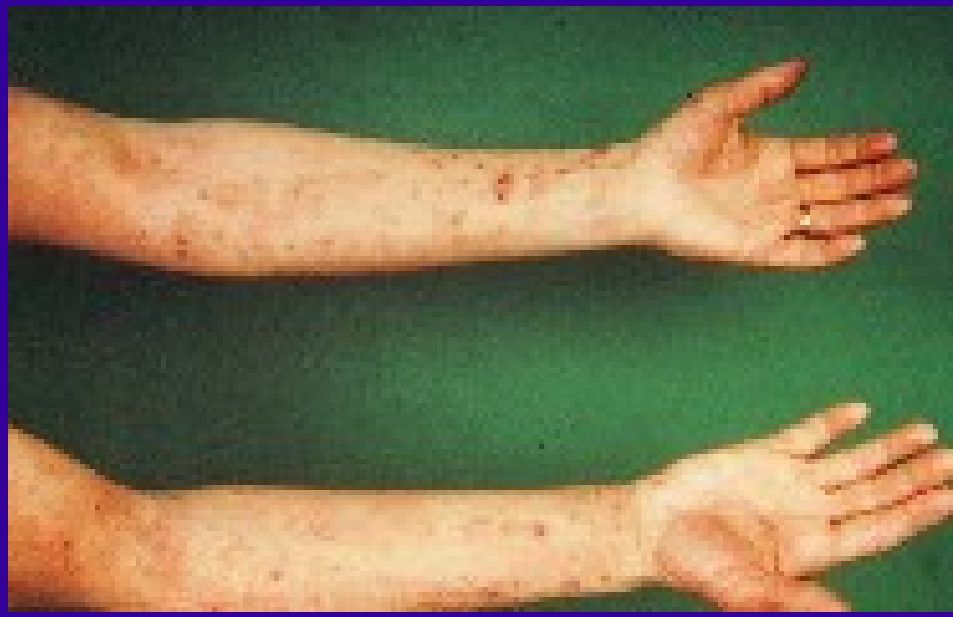






Adult AD – neurodermitic form



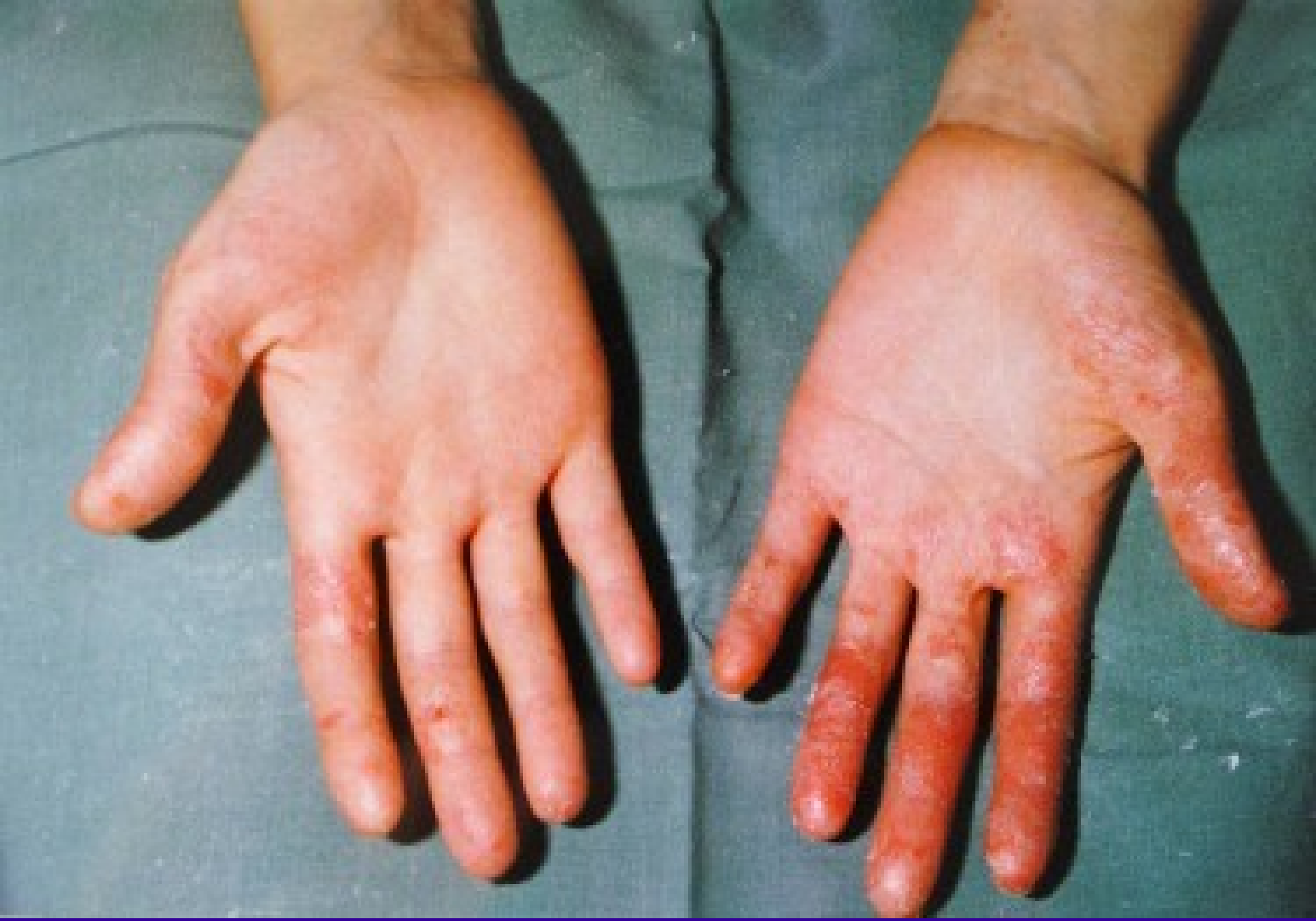


Adult AD – erythrodermic form

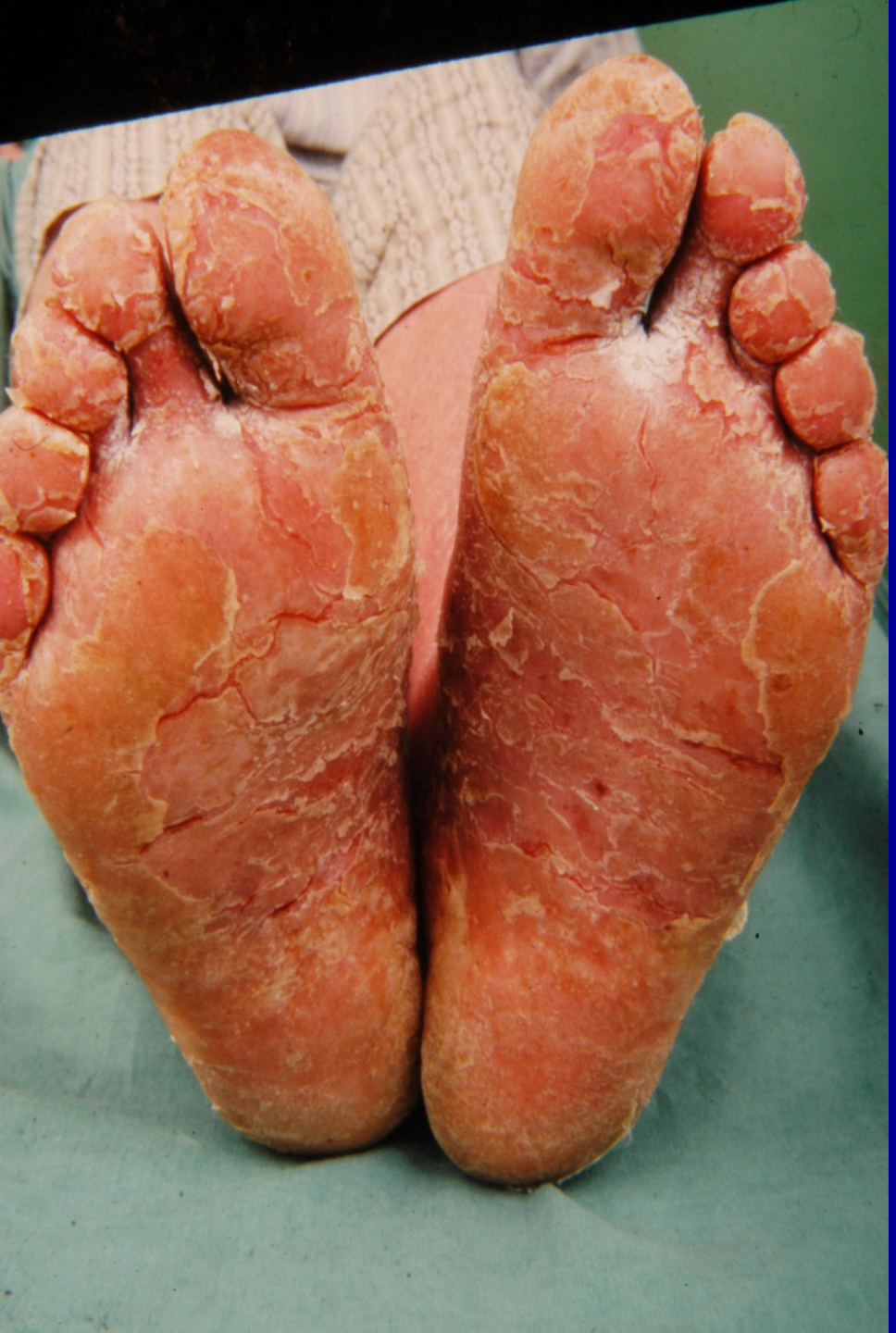
AD in adults

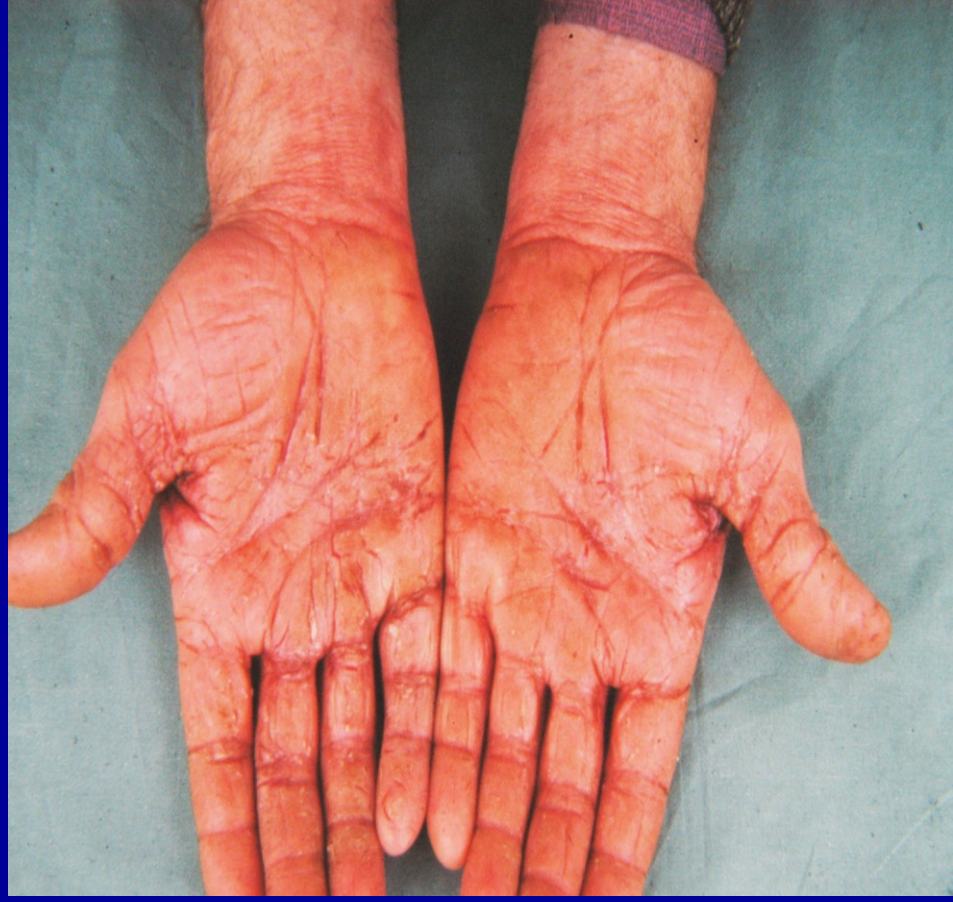
- **atypical forms** - nummular, dyshidrotic, hyperkeratotic forms

- **minimal forms** - cheilitis sicca, stomatitis angularis, pulpitis sicca, intertrigo retroauricularis, etc.

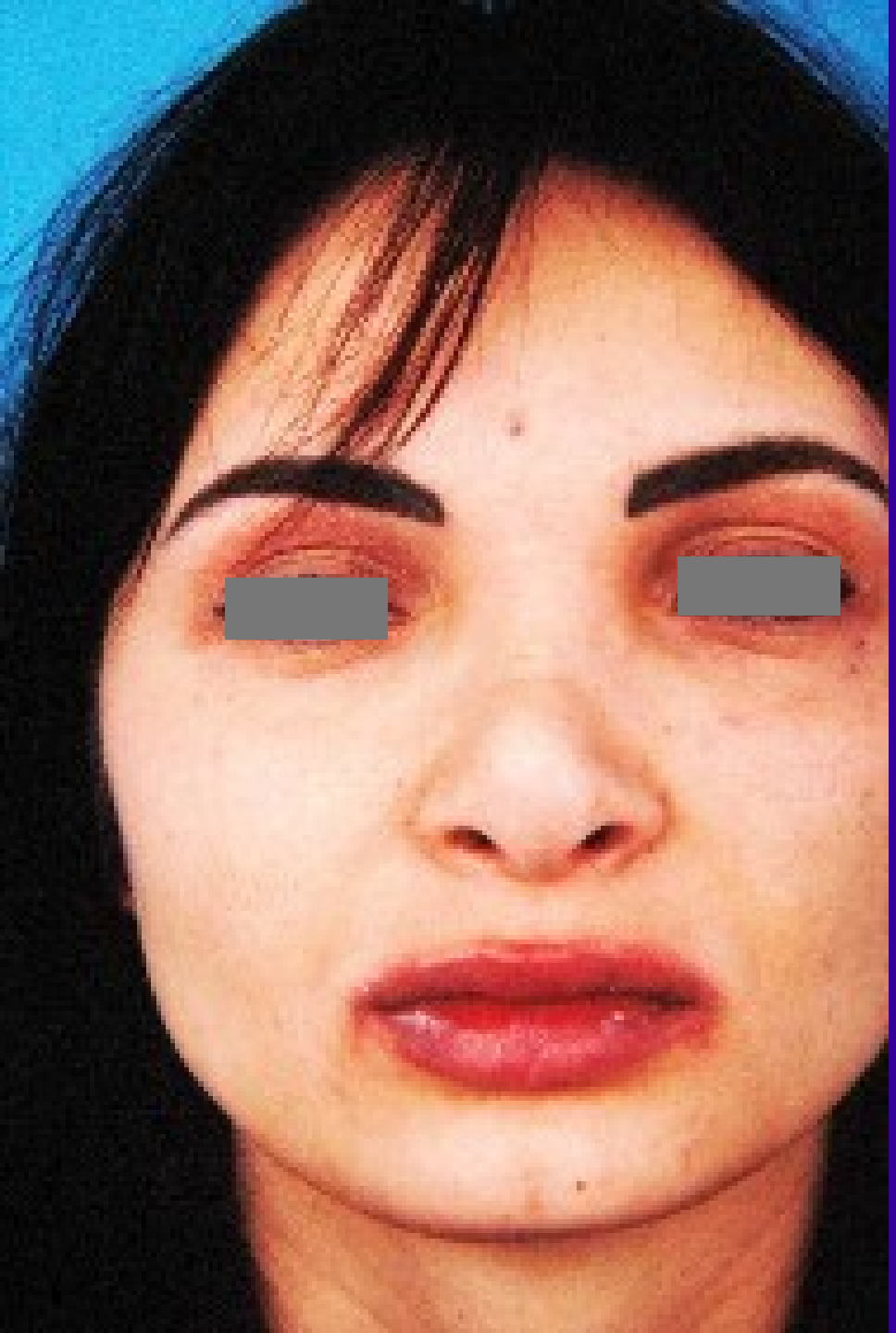


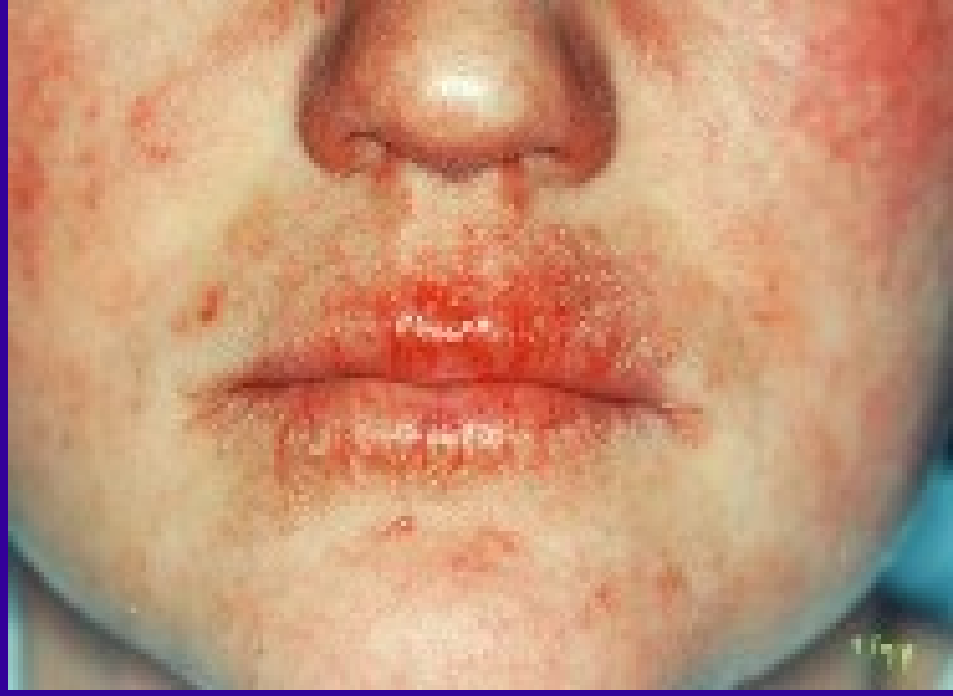
Adult AD - dyshidrotic form



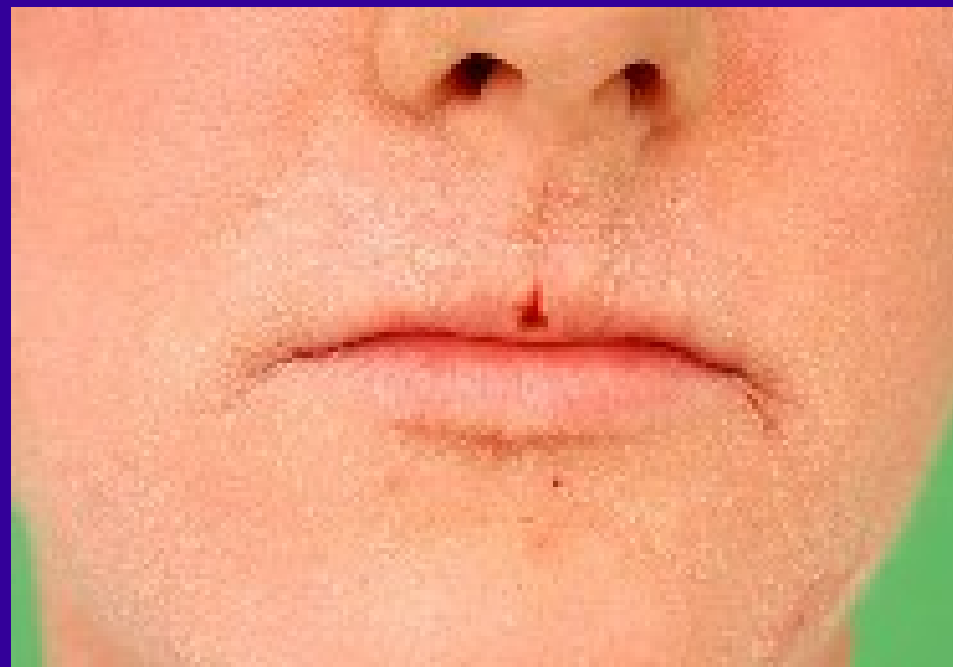


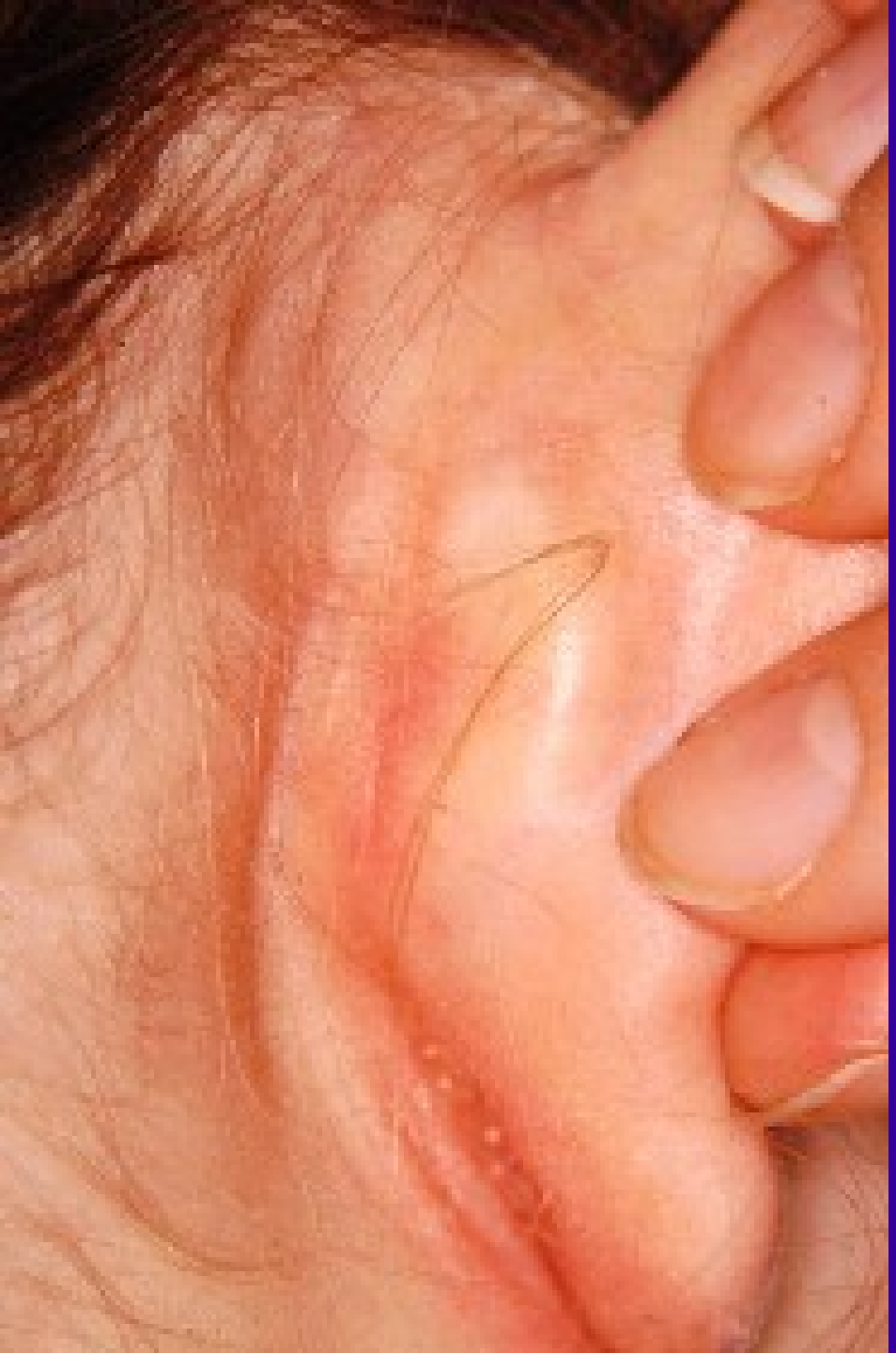
Eczema atopicum hyperkeratoticum

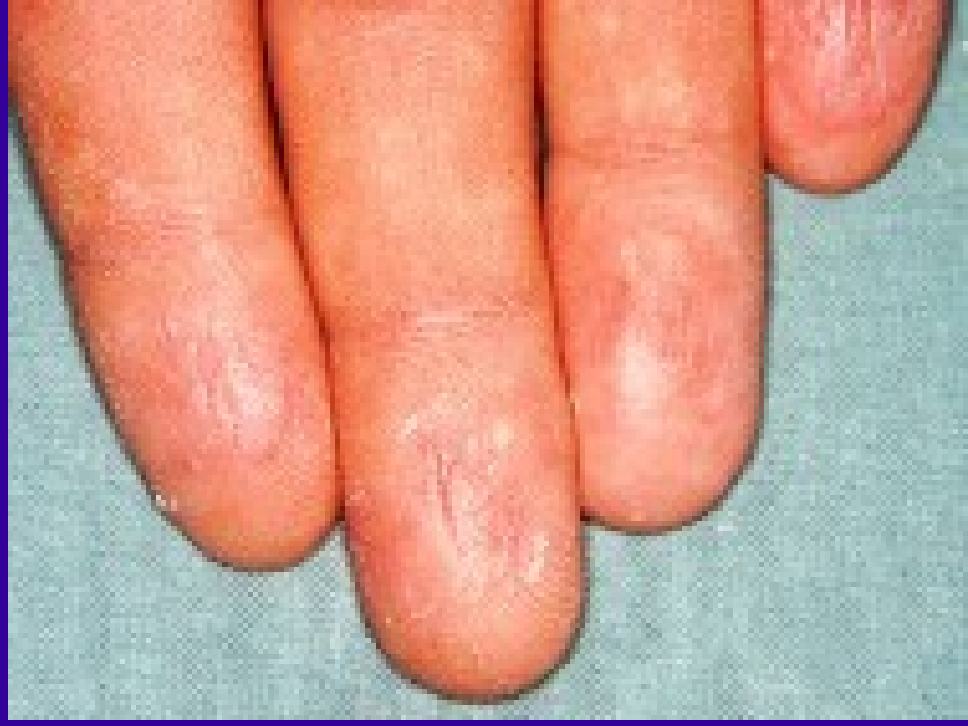




AD eyelid dermatitis, lip dermatitis







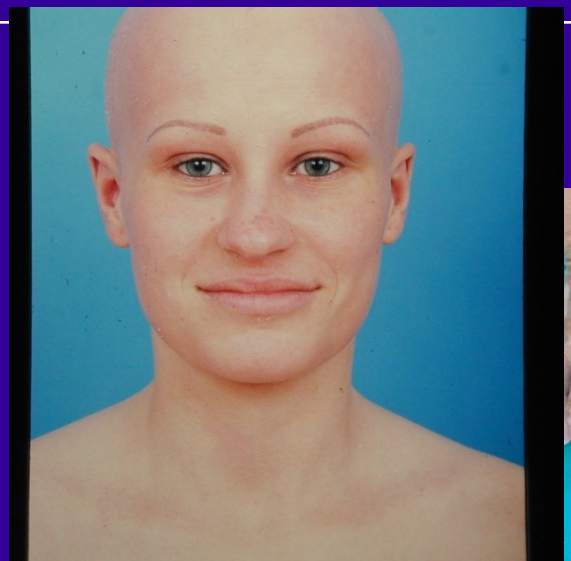
AD retroauricular dermatitis

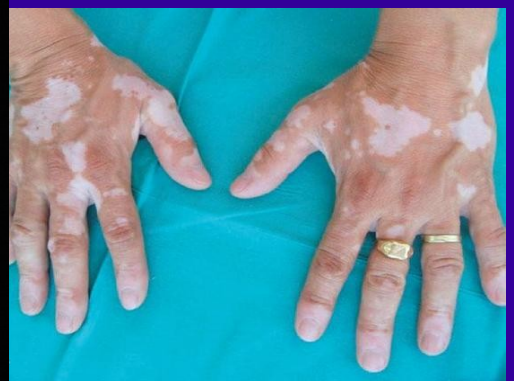
Complications of AD

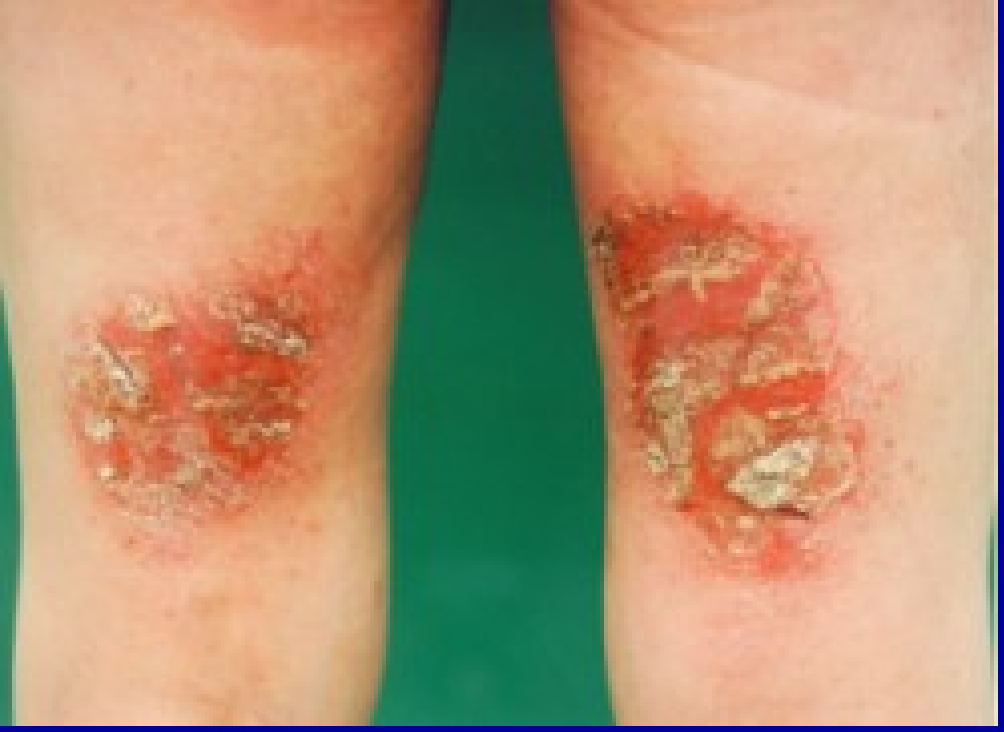
bacterial - impetiginization (St. aureus)
viral – herpetication-HSV, warts, mollusca
fungal (Tr. rubrum, Pityrosporum ovale)
contact sensitization (nickel, fragrances, KS…)

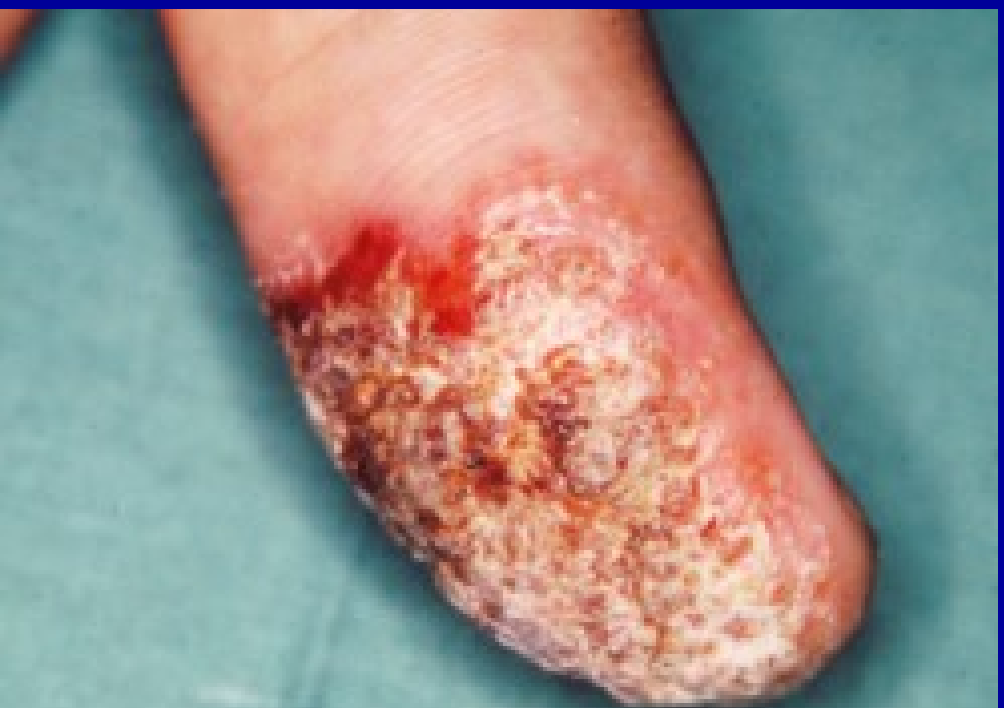
association:

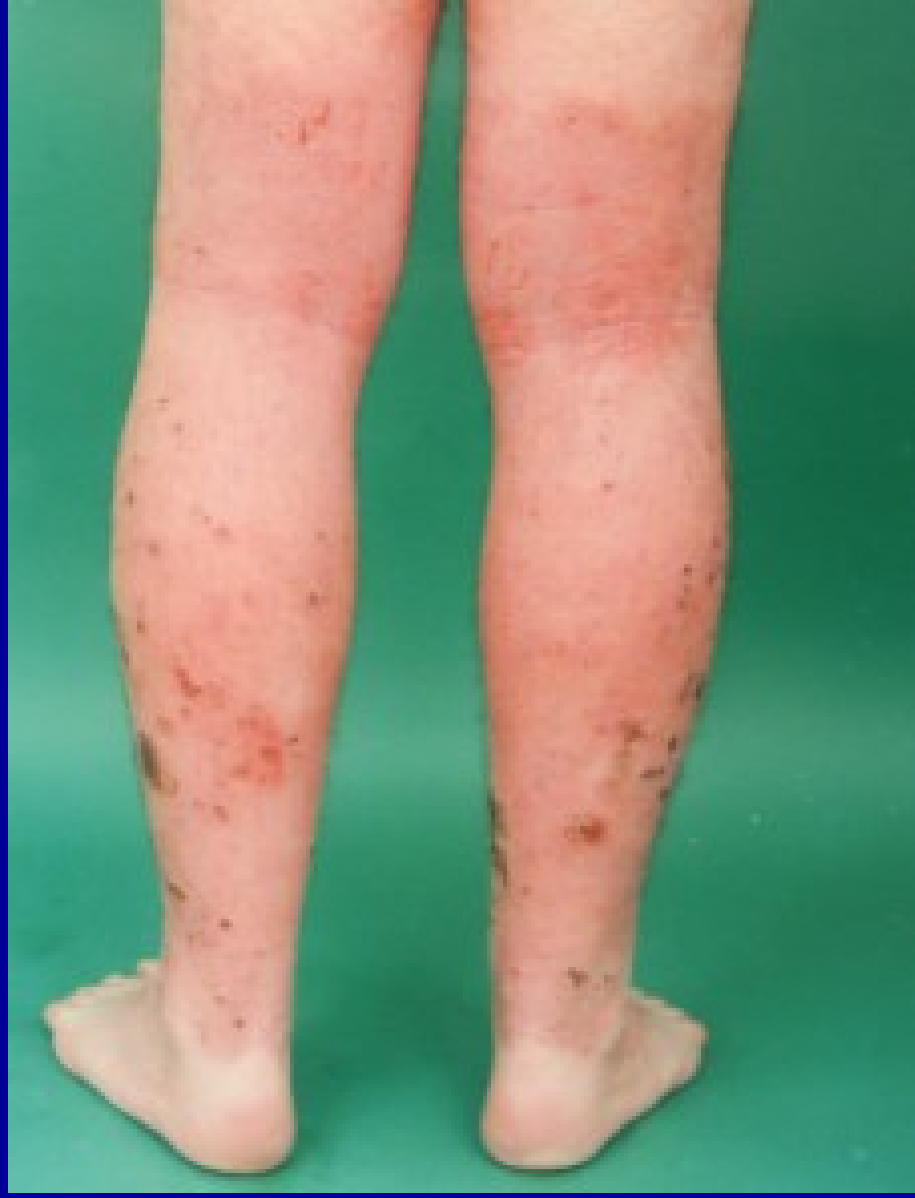
- **alopecia areata**
- **ichtyosis vulgaris**
- **vitiligo**



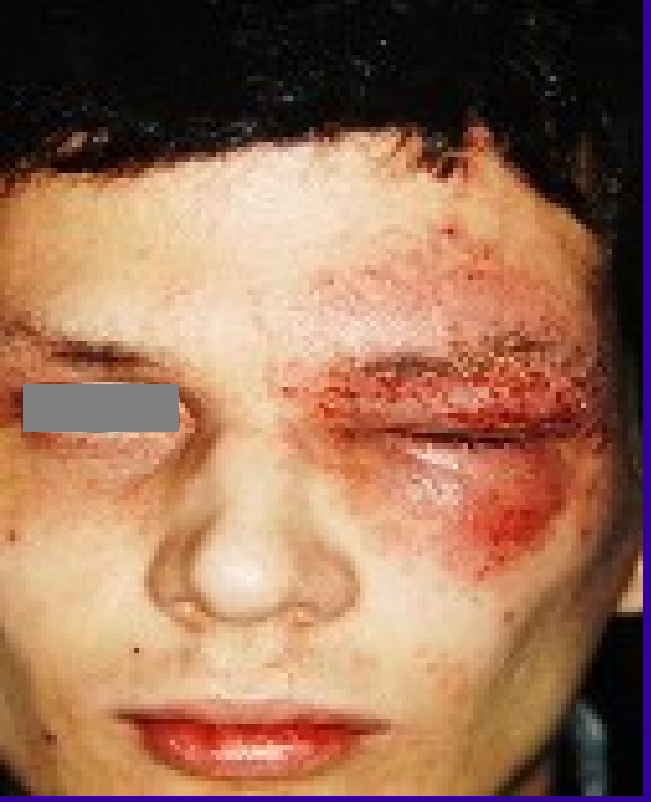
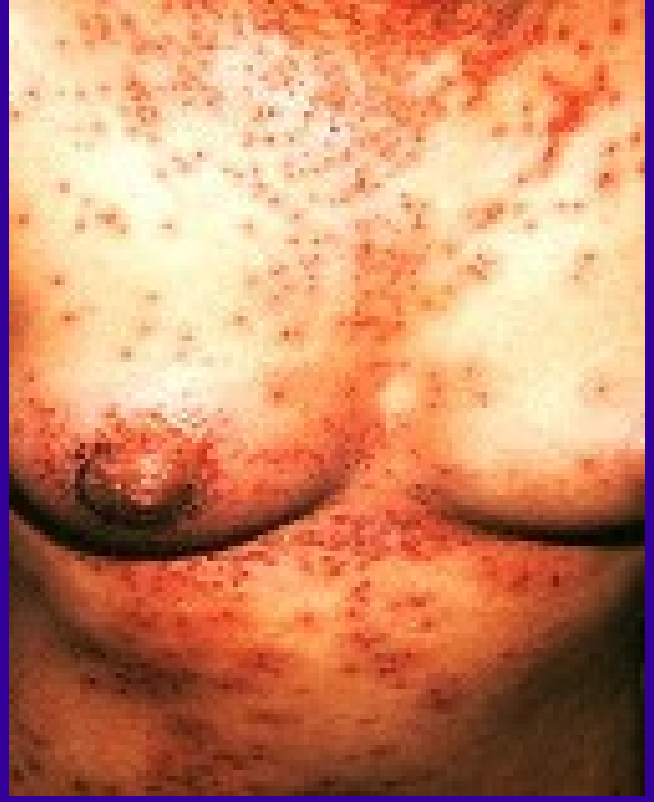
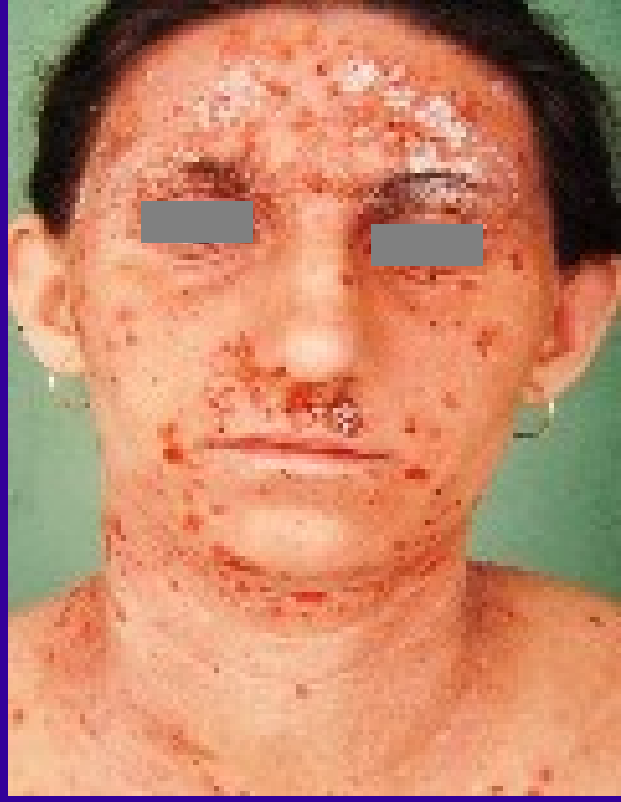
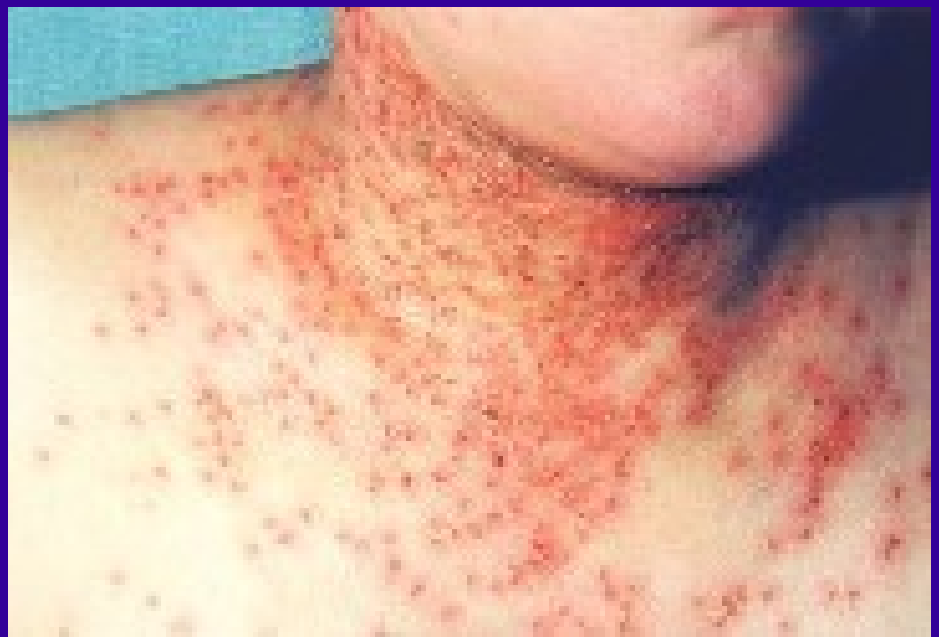








Eczema atopicum impetiginisatum

Eczema atopicum herpeticatum

Treatment of AD

mild form of AD (30-40% of patients):

education of pacient (or parents)

identification of triggering factor
and their elimination

emmolients and baths

topical corticosteroids

pimecrolimus

antihistamines during flares

Treatment of AD

- mid-severe form of AD (40-50% of patients):

  treatment similar as in mild form

  \+ tacrolimus

  or

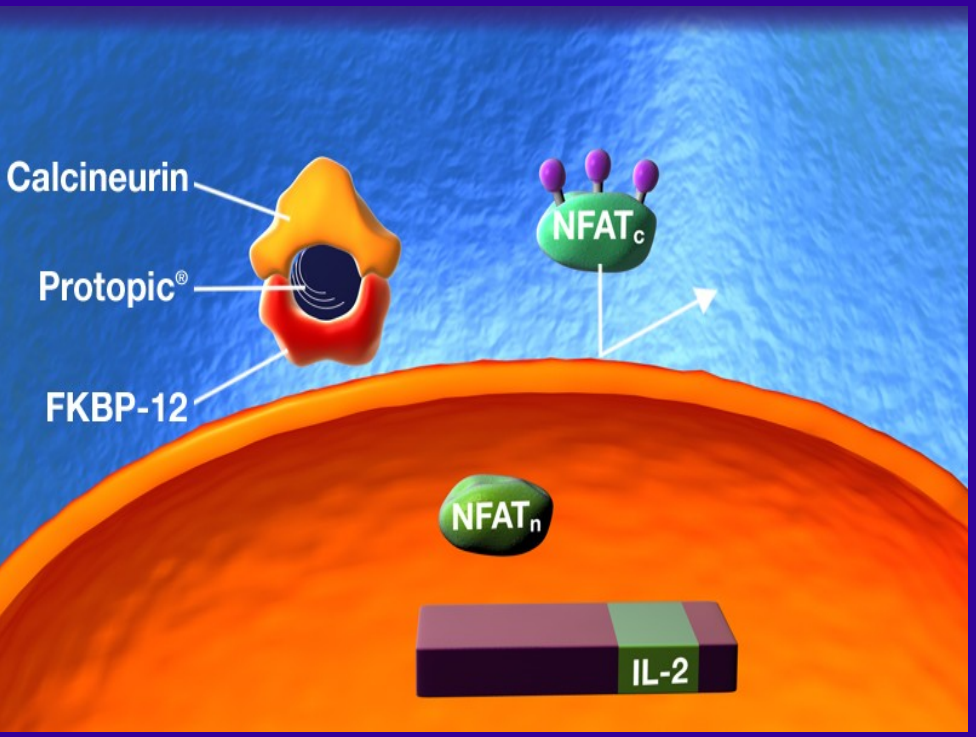
  hospitalization – lab. and clinical tests (triggers)

  traditional topical treatment /tar/

  or

  phototherapy (UVB 311nm, UVA-1)

Tacrolimus (PROTOPIC ointment)



- Topical Immunomodulator
- Blocks calcineurin
- antiinflammatory
- antipruritic
- Long - term treatment
- No skin atrophy

Treatment of AD

- severe form of AD (5-10% patients)

  phototherapy (PUVA, UVA-1)

  systemic corticosteroids (short courses)

  immunosupressives: cyclosporine A, MMF, AZT,MTX

  immunomodulants: IFN (?)

  experimental therapy: i.v. Ig

  JAK, PDE ihibitors

  biologicals (dupilumab....)

Microbial eczema

Allergy of IVth type to bacterial allergens – mostly to Staph. aureus

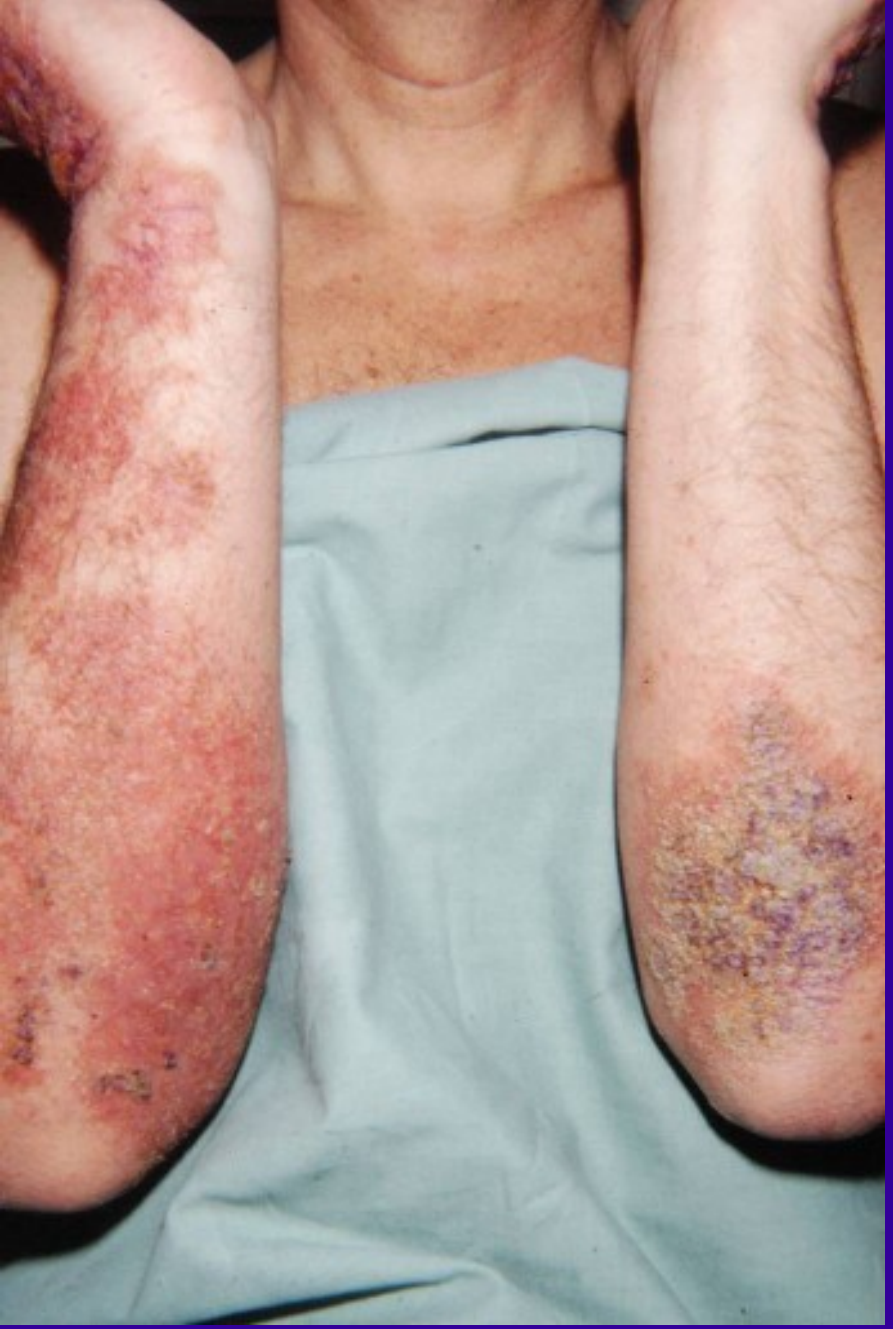
appears mostly secondary:

in pyodermas, scabies, atopic dermatitis, ICD

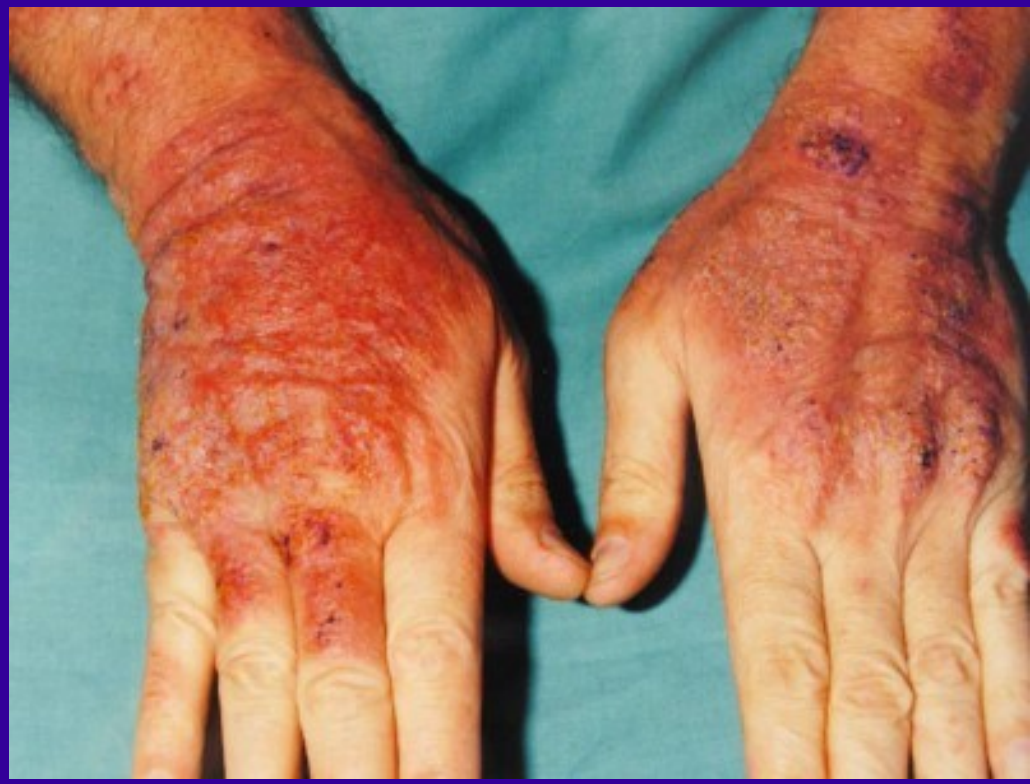
around fistulas, stomias, in varicous terrain on legs

around sites of inflamamtion (chronic rhinitis, otitis)

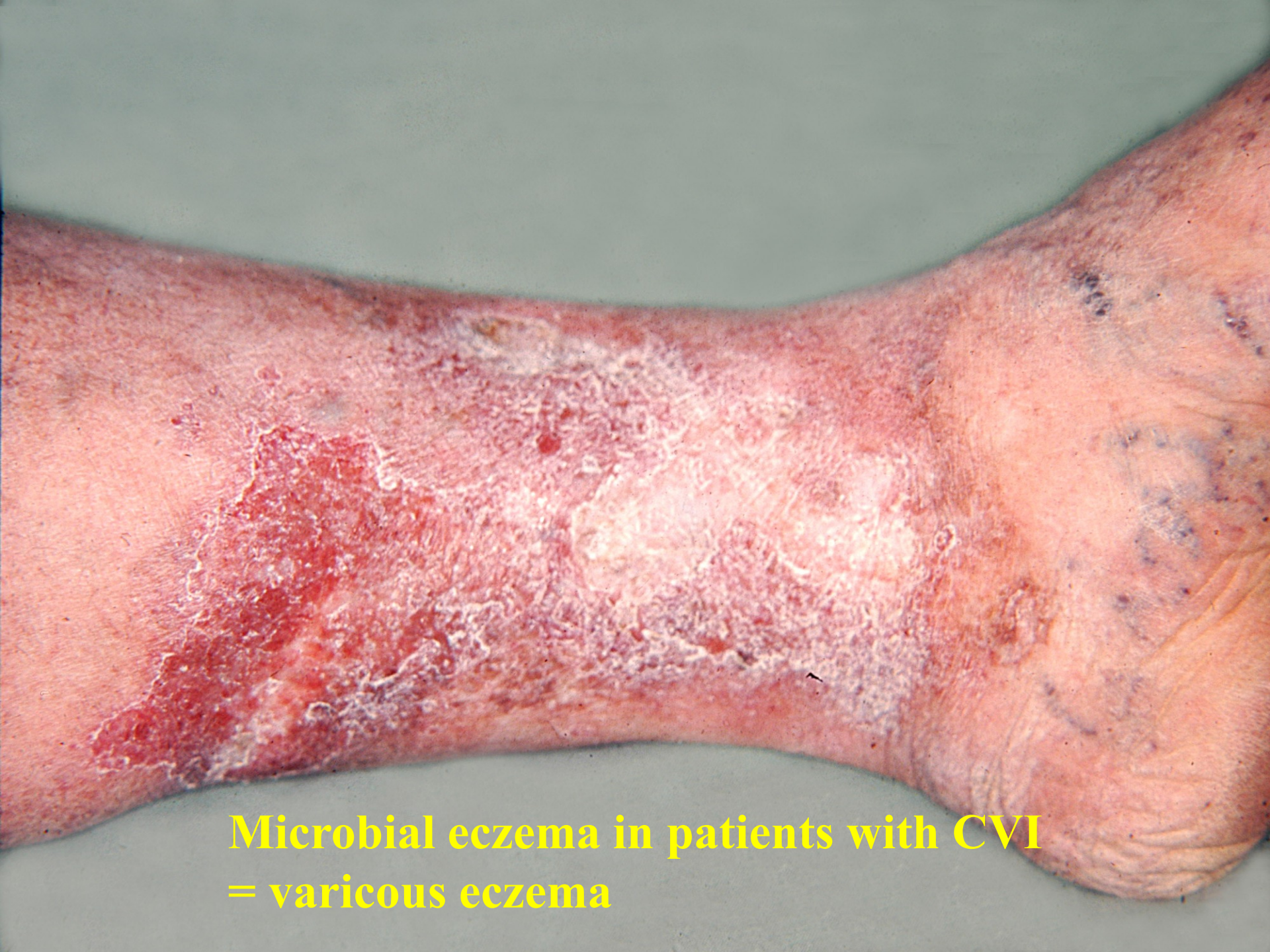
variant: nummular dermatitis (coin shaped patches and/or plaques) usually in patients with focal bacterial infection (tooth gfranuloma, chronic tonsillitis, chronic urogenital infections etc.

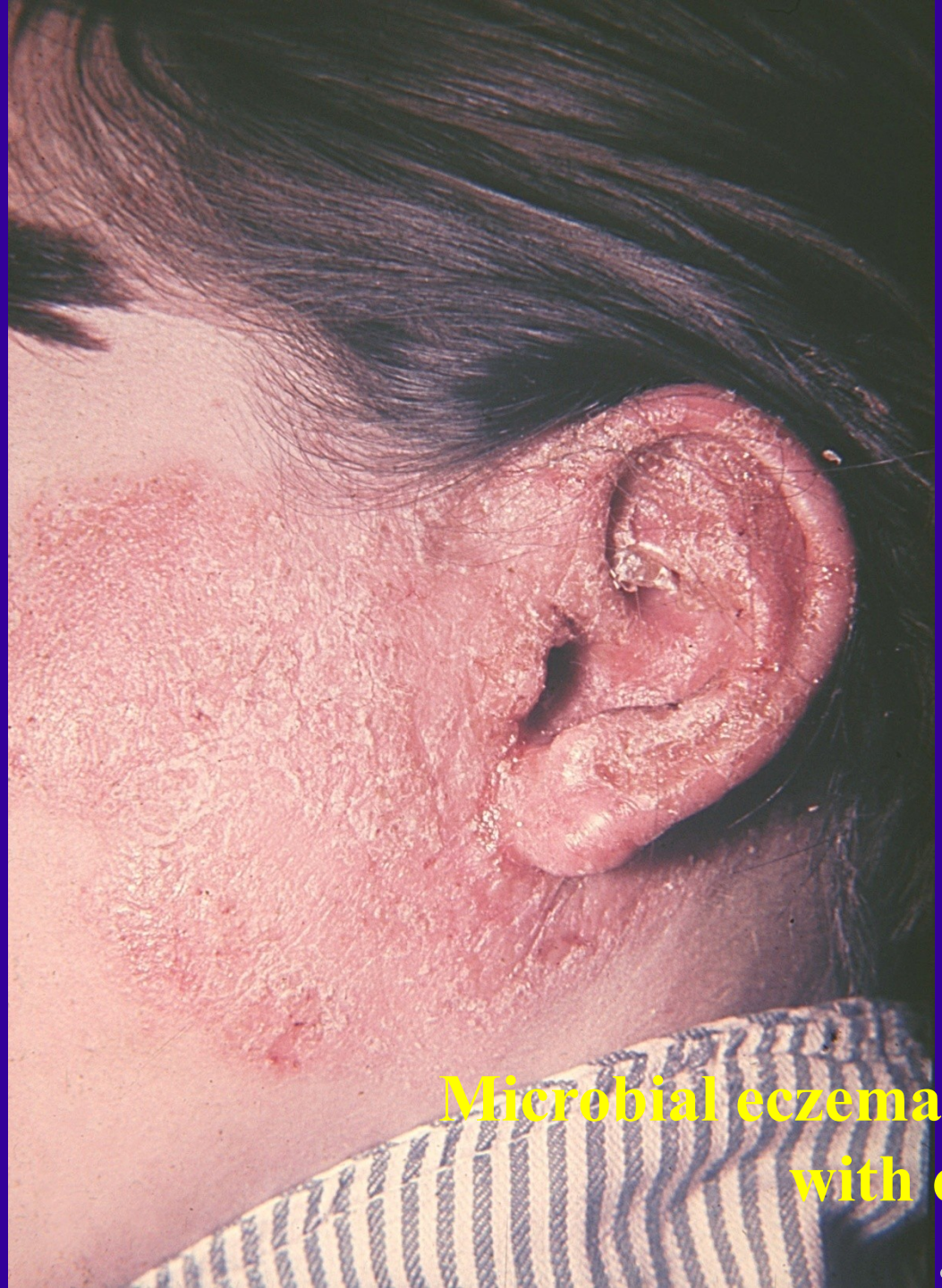


Microbial eczema

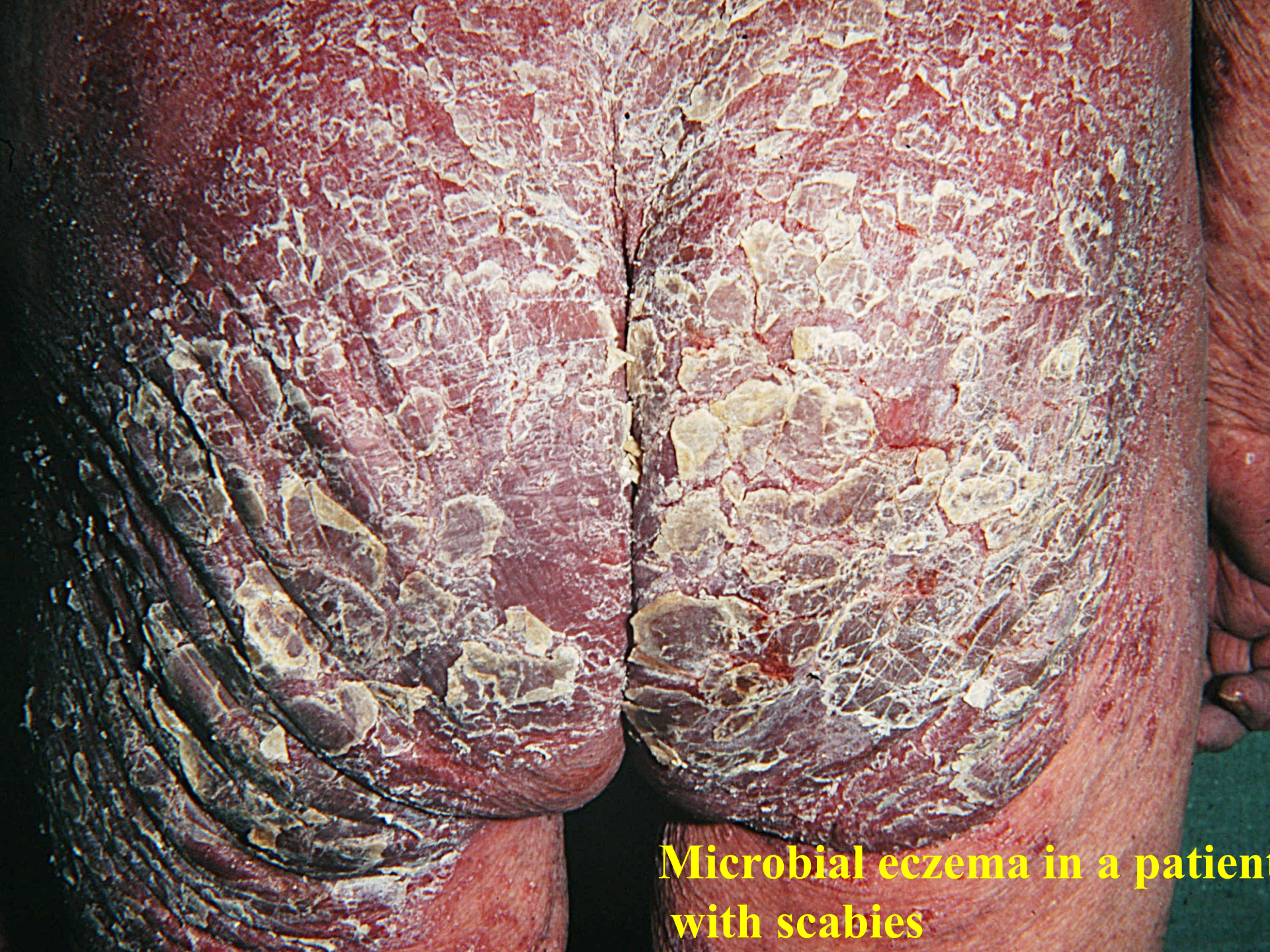


Microbial eczema


Microbial eczema in patients with CVI
= varicous eczema



Microbial eczema in a patient with chronic otitis


Microbial eczema in a patient
with scabies

Treatment of microbial eczema

Acute phase:

- Drying compresses
- Topical zinc preparations
- Topical corticosteroids in lotion base

Subacute and chronic phase:

- ATB paste, endiaron paste, tar preparations
- Combination with topical CS (TMC-E, Belogent, Fucicort

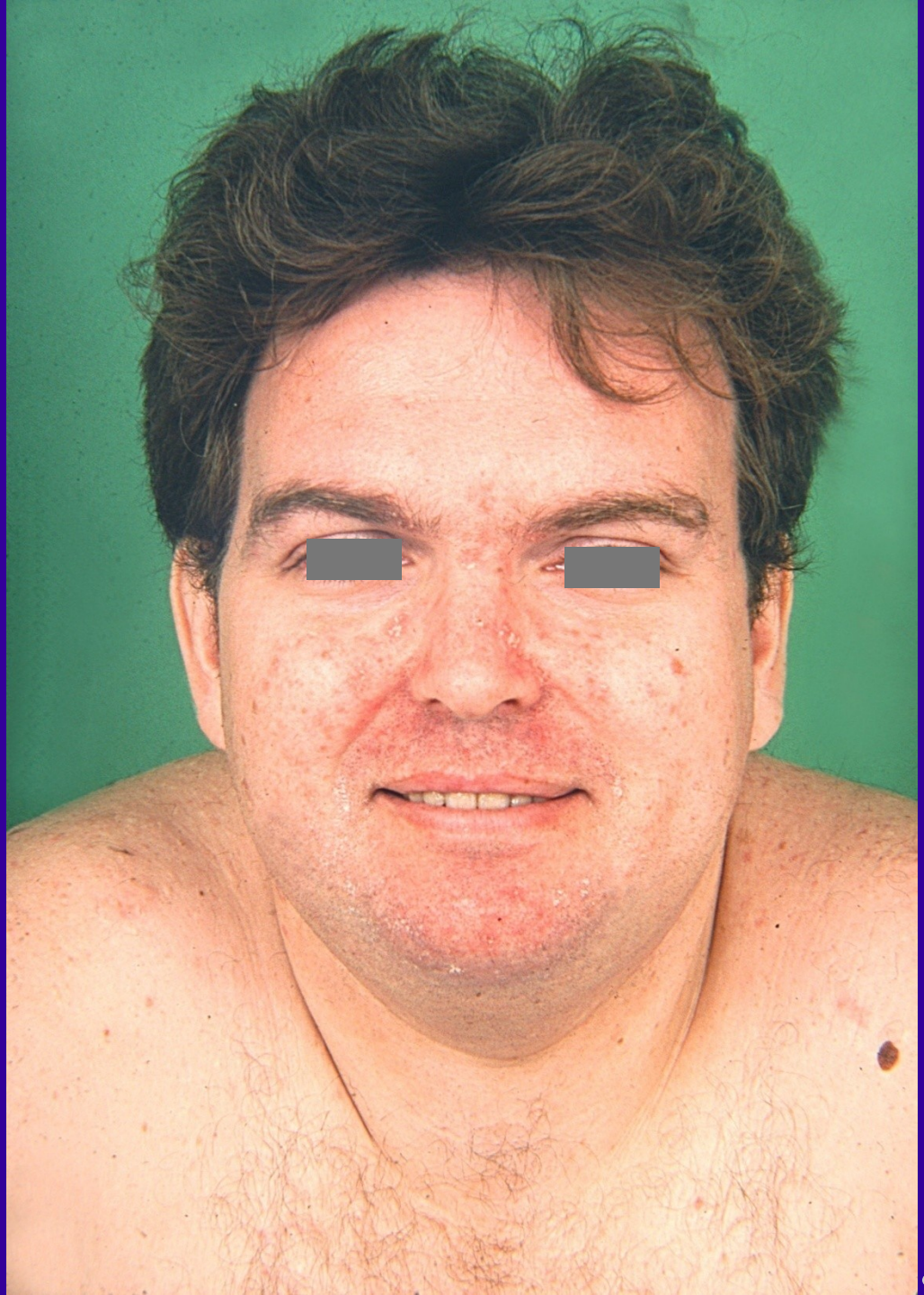
Systemic ATBs

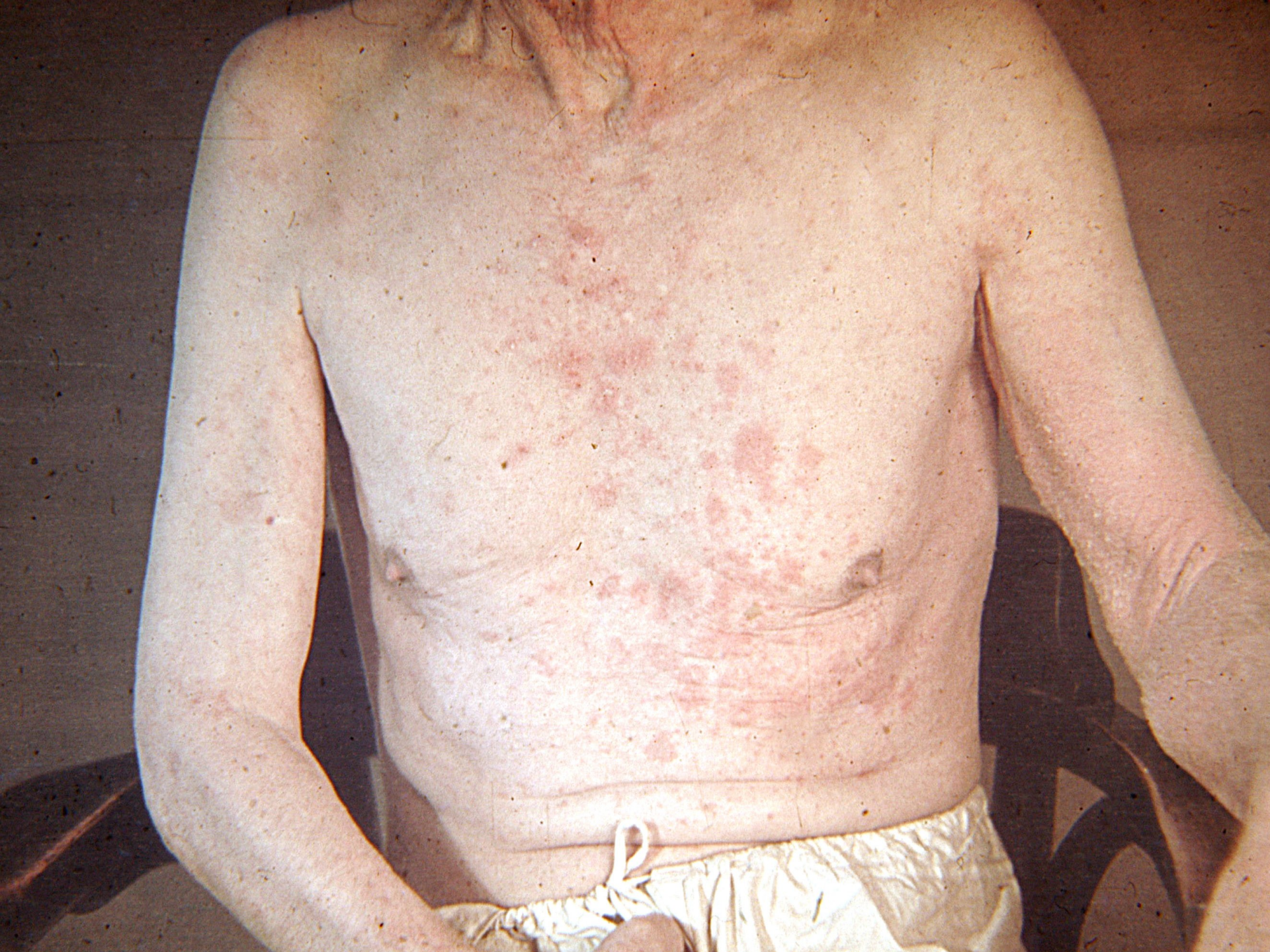
Seborrheic dermatitis

- localisation: seborrheic predilection sites
- etiology: genetic predisposition
  dysseborrhea – altered composition of sebum
  Malassezia furfur = pityrosporon ovale
  immunodeficiency - AIDS
  depletion of zinc

Clinical picture: erythematous scaly lesions
In typical sites: scalp, eyebrows, nasolabial folds, midchest region, around umbilicus, groins & axillae

- Subjective complaints: itching, burning





Treatment of seborrheic dermatitis

- Topical imidazole antifungals + topical corticosteroids
- Topical imidazole antifungals
- Topical preparation with zinc
- zinc supplementation
- (Systemic antifungals)