Irritant Contact Dermatitis

- There are two major forms of contact dermatitis: irritant and allergic.
- Irritant contact dermatitis (ICD) is a cutaneous inflammatory disorder resulting from activation of the innate immune system by direct cytotoxic effect of a chemical or physical agent, whereas allergic contact dermatitis is a delayed-type hypersensitivity immune reaction mediated by hapten-specific T cells.
- ICD is the most common form of occupational skin disease, estimated to constitute between 70% and 80% of all occupational skin disorders.

- Clinical manifestations of ICD are determined by the properties of the irritating substance as well as host and environmental factors.
- These include concentration, pH, mechanical pressure, temperature, humidity, and duration of contact.
- Low ambient humidity and cold are important factors in decreasing the water content of the stratum corneum and, consequently, increasing the permeability to irritants.
- such as soaps, detergents, acids, bases, and solvents.

- Occlusion, excessive humidity, and maceration increase the water content of the stratum corneum, with consequent enhanced percutaneous absorption of water-soluble substances .
- In addition, irritated skin may become more susceptible to superimposed allergic sensitization.
- Important predisposing characteristics of the individual include age,sex, pre-existing skin disease, anatomic region exposed, and sebaceous activity.

- There are age-associated changes in the skin that can alter the skin’s response to irritants.
- Both infants and the elderly are more often affected by ICD because of their less robust epidermal barrier, and they also develop more severe symptoms.
- While skin irritation may be seen more often on the upper extremities of women than men, this higher prevalence of ICD may be due to increased frequency of exposure rather than inherent gender differences.

- Genetic factors also play a role.
- Patients with a history of atopic dermatitis have a 13.5 times greater risk of developing occupational dermatitis.
- Lastly, the most commonly affected sites are exposed areas such as the hands and the face, with hand involvement seen in ~80% of patients and facial involvement in 10%.
- Excessive exposure to water, soaps and detergents, common causes of ICD, play a critical role given that wet work (immersion in water for >2 hours, occlusive protective gear, and/or hand washing >20 times/day) represents one of the most important risk factors for developing irritant dermatitis.
- ...

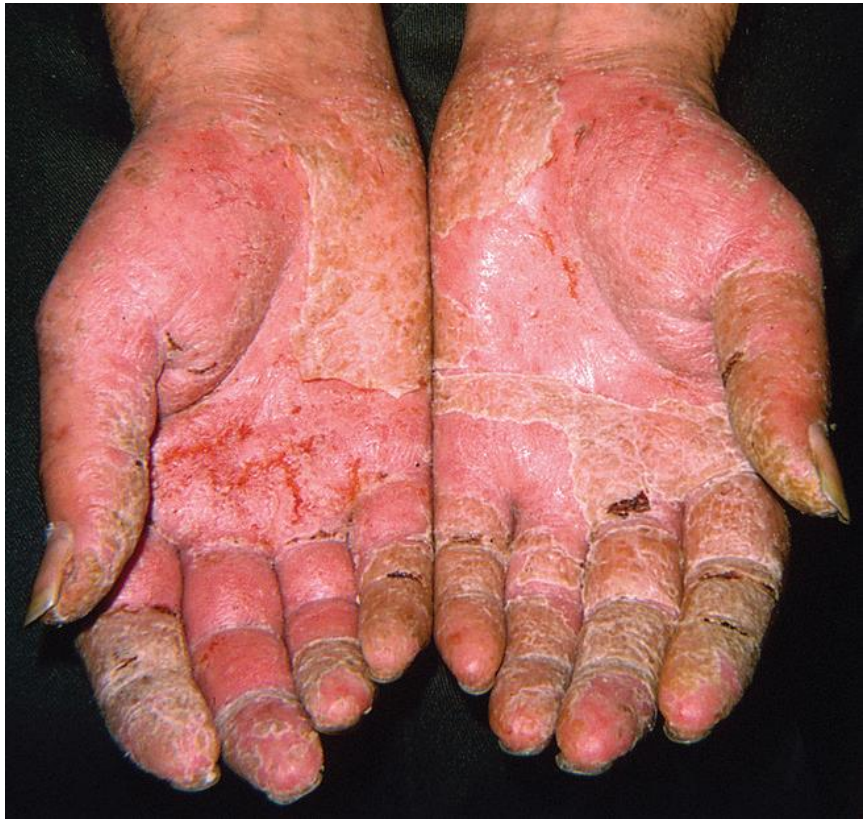
PATHOGENESIS

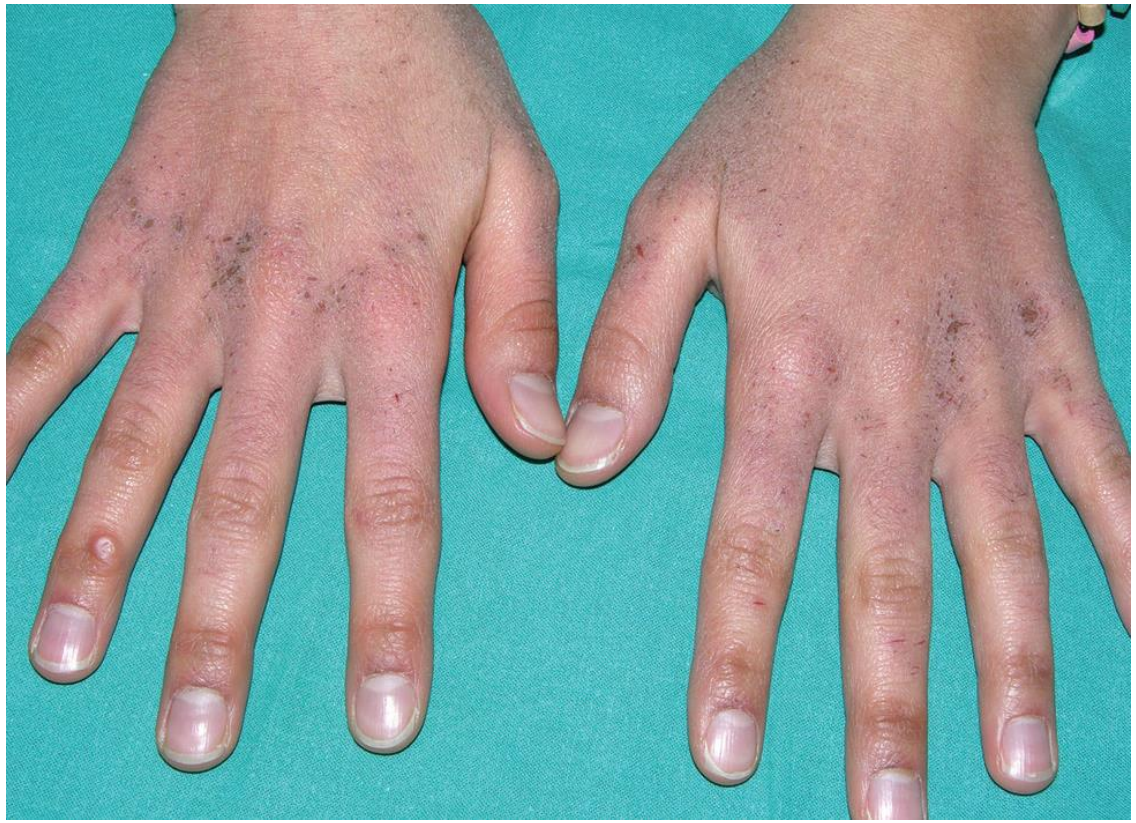
- Although the cellular mechanisms of ICD remain elusive, increasing evidence suggests that activated keratinocytes act as signal transducers in the control of host homeostatic responses to exogenous stimuli and they serve as key immunoregulators.
- While other mediators such as prostaglandins, leukotrienes, and neuropeptides may possibly play a role, cytokines carry the most interest in ICD as they are the central mediators in T-cell recruitment and inflammation.

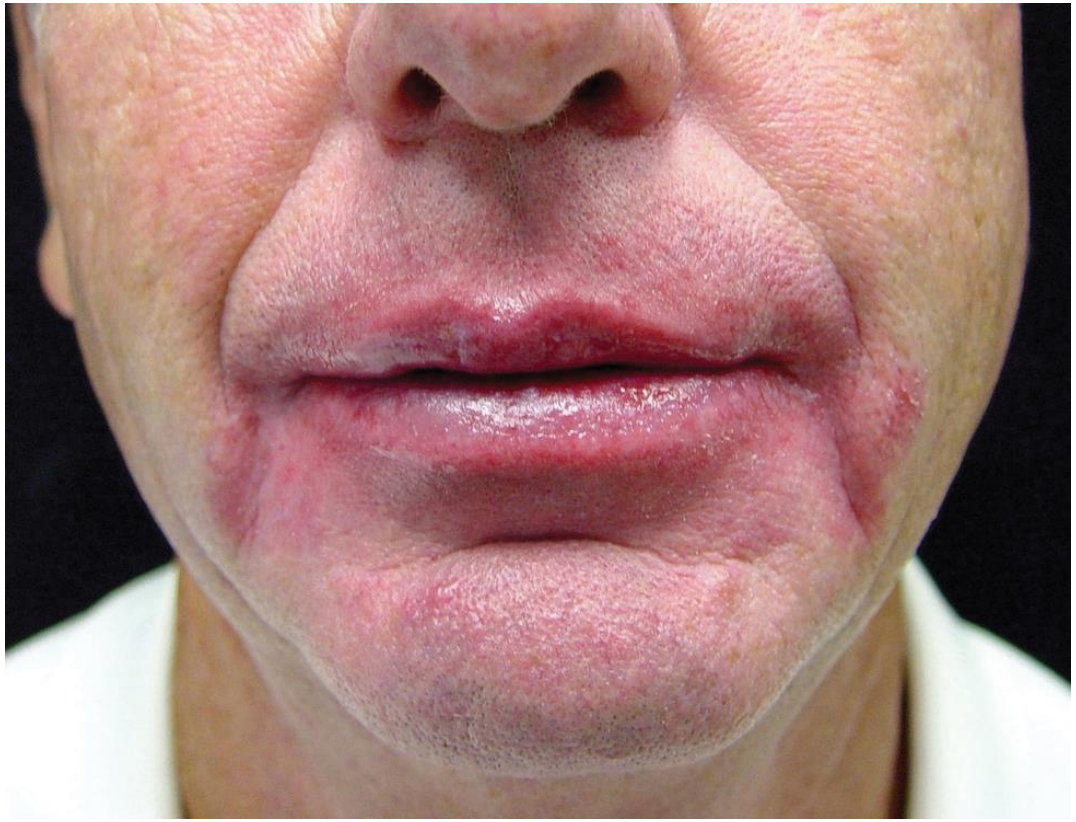
- IRRITANTS AND MECHANISMS OF TOXICITY
- Detergents Barrier disruption, protein denaturation, membrane Toxicity
- Acids Protein denaturation, cytotoxicity
- Alcohols Protein denaturation
- Alkalis Barrier lipid denaturation, cytotoxicity through cellular swelling
- Oils Disorganization of barrier lipids

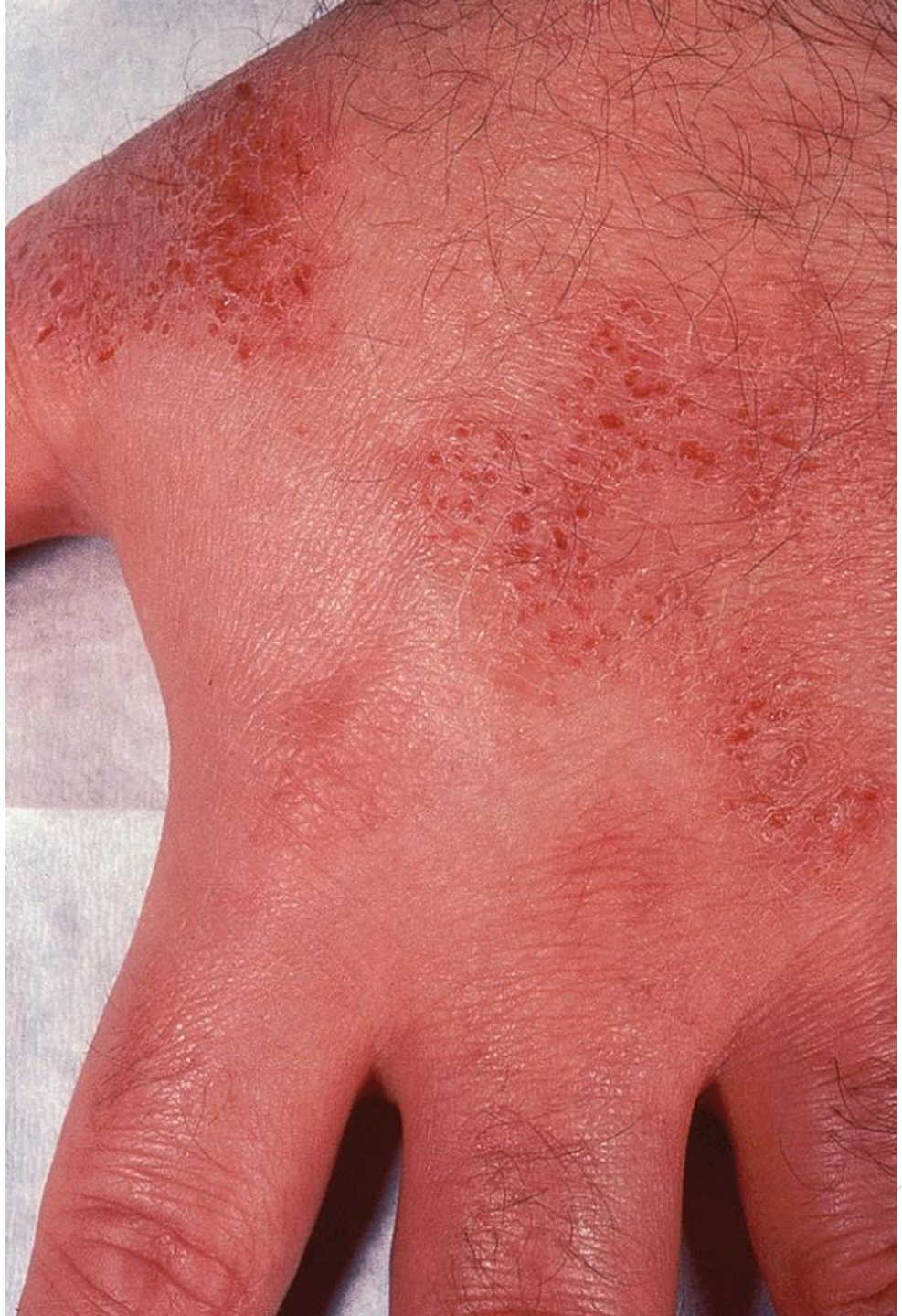
- Organic solvents Solubilization of membrane lipids, membrane toxicity
- Oxidants Cytotoxicity
- Water If barrier is disrupted, cytotoxicity through swelling of viable epidermal cells.

- Symptoms of acute ICD include burning, stinging, and soreness of the directly affected sites.
- Physical signs include erythema, edema, bullae, and possibly necrosis.
- These lesions are restricted to the area where the irritant or toxicant damaged the tissue, with sharply demarcated borders and asymmetry pointing to an exogenous cause.
- If there is no dermal injury, healing should be complete. The potent irritants that most frequently lead to ICD are acids and alkalis, resulting in chemical burns.









Asteatotic Dermatitis

- *Asteatotic dermatitis*, also referred to as asteatotic eczema, eczema craquelé, is a special variant seen primarily during dry winter months.
- Elderly individuals who frequently bathe without remoisturizing are at particular risk of developing asteatotic dermatitis.
- Intense pruritus is common, with the skin appearing dry with ichthyosiform scale and characteristic patches of superficially cracked skin.

Traumatic Irritant Contact Dermatitis

- *Traumatic* ICD may develop after acute skin trauma, such as from burns, lacerations, or acute ICD. Patients should be asked whether they have cleansed the skin with strong soaps or detergents.
- It is characterized by eczematous lesions, most commonly on the hands, that last for weeks to months with persistent redness, infiltration, scale, and fissuring in the affected areas.

Pustular and Acneiform Irritant Contact Dermatitis

- *Pustular and acneiform* ICD results from exposure to certain irritants, such as metals, mineral oils, tars, greases, and naphthalenes .
- This syndrome should be considered in conditions in which folliculitis or acneiform lesions develop in settings outside of typical acne, particularly in patients with atopic dermatitis, seborrheic dermatitis, or prior acne vulgaris.
- The pustules are “sterile” and transient.

Bodily fluids

- Urine, feces (especially in the setting of diarrhea), and saliva can lead to ICD.
- In babies, irritant diaper dermatitis is a common problem and is often characterized by glazed erythema of convex surfaces and at the diaper margins, with sparing of the skin folds; edema, scaling, and superficial erosions may be observed .
- Incontinence can lead to similar problems in the elderly.

TREATMENT

- Avoidance of causative irritants in the home or the workplace is the primary treatment for ICD.
- Strategies in the prevention of ICD include the identification of irritants with appropriate substitution, the establishment of engineering controls to reduce exposure, the utilization of personal protective equipment such as gloves and special clothing, and barriers such as ointments, emollients, or creams.

- The goal of treatment is to restore normal epidermal barrier function.
- Topical corticosteroids are frequently used, but their efficacy has been controversial, as experimental studies have provided conflicting results.
- Systemic corticosteroids, although potentially helpful in reducing acute inflammation, are not useful in the treatment of chronic ICD unless corrective measures are taken to avoid the offending contactants.

- Narrowband ultraviolet B or photochemotherapy
- (PUVA) irradiation may be considered for chronic dermatitis that does not respond to any other form of therapy.
- Hyperkeratotic palmoplantar dermatitis from frictional or chronic ICD or a combination of dermatitis and psoriasis may benefit from the adjunctive use of systemic retinoids such as acitretin and alitretinoin or systemic immunomodulators such as methotrexate, cyclosporine, and possiblytargeted (biologic) therapy.

- Physical skin protection
- Emolient
- Barrier creams
- Topical corticosteroids
- Topical calicineurin inhibitors
- Cyclosporine
- UVB therapy
- PUVA therapy
- Superfacial radiotherapy
- Acitretin

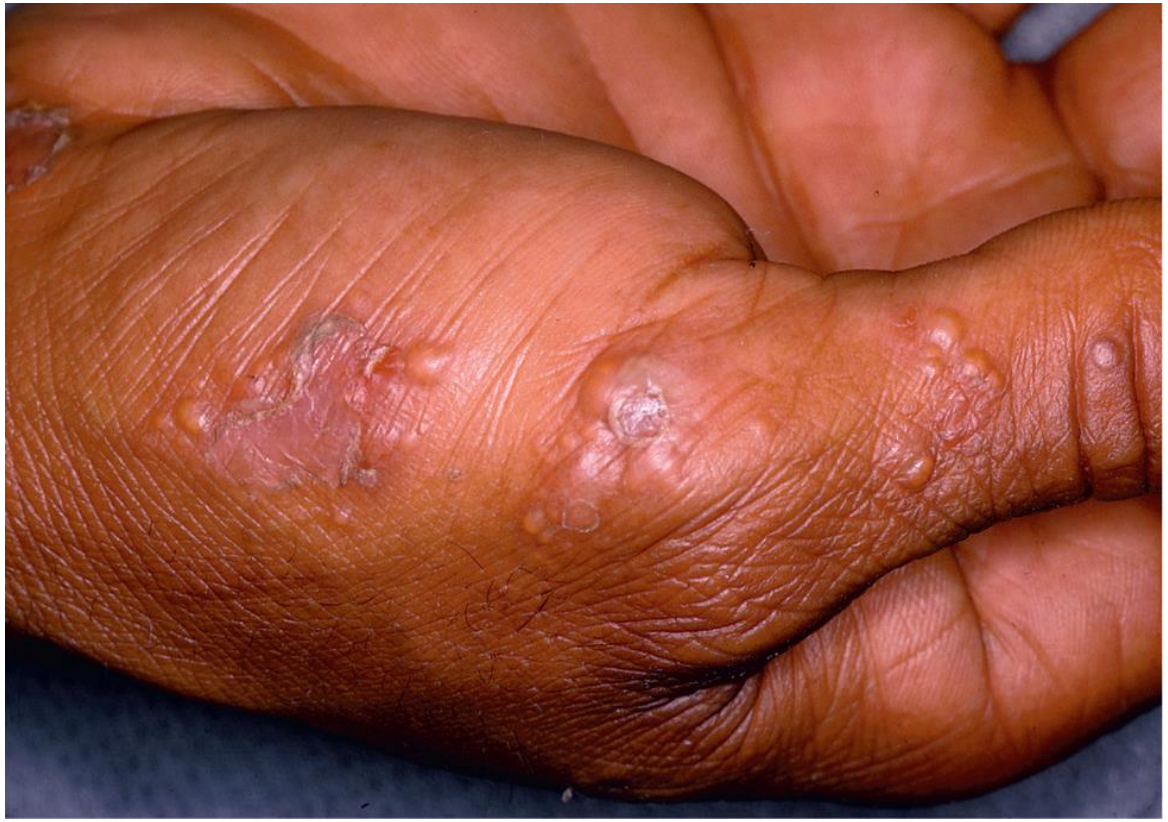
Dyshidrotic Eczema

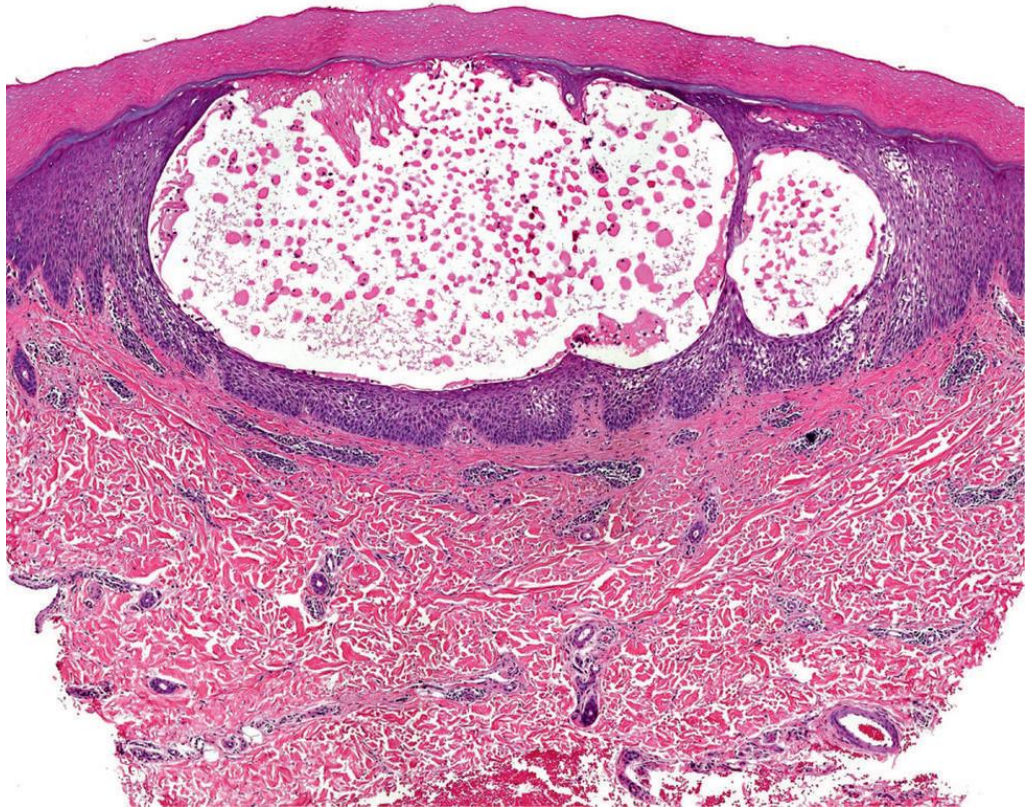
- Synonyms: ■ Pompholyx (larger bullae) ■ Acute and recurrent
- vesicular hand dermatitis.
- Key features
- ■ Firm, pruritic vesicles of the palms, soles, and lateral and medial
- aspects of the fingers and toes.
- ■ Association with atopic dermatitis and contact dermatitis (allergic
- and irritant).
- ■ No disturbance of sweat gland function.

- *Clinical features*
- Dyshidrotic eczema is characterized by symmetric, firm, deep-seated vesicles of the palms, the lateral and medial aspects of the fingers, and less often the soles and toes.
- The size of the vesicles may vary from pinhead-sized to several centimeters (“pompholyx”).
- While the markedly pruritic vesicles initially contain clear fluid, they

have a tendency for purulent superinfection.

Dyshidrotic eczema resolves via desquamation of characteristically thick scales.





- *Pathogenesis*
- Although the formation of vesicles is not linked to sweat gland dysfunction or trapping of sweat within the epidermis (as the term may suggest), hyperhidrosis can be an aggravating factor in some patients.
- Notably, treatment of hyperhidrosis with botulinumtoxin A may ameliorate dyshidrotic eczema.

- Dyshidrotic eczema is frequently an expression of atopic dermatitis,
- particularly as a late-stage manifestation.
- Occasionally, administration of IVIg is followed by acute episodes of

dyshidrotic eczema.

- The role of ingestants, in particular nickel and cobalt, has been debated, but improvement with a low-nickel diet (in nickel-sensitive patients with a positive oral provocation test) has been observed.

- Lastly, flares can also follow periods of emotional stress as well as exposure to hot climates and rarely sunlight.

- *Differential diagnosis*
- Inflammatory tinea manuum and pedis, scabies, and palmoplantar pustular psoriasis need to be considered as well as dyshidrosiform pemphigoid, dyshidrotic cutaneous T-cell lymphoma, erythema multiforme, fixed drug eruption, and, in children, infantile acropustulosis.

- *Treatment*
- Topical and systemic corticosteroids are the mainstay of treatment.

Topical calcineurin inhibitors may be helpful.

Bath PUVA has been shown to be more effective than oral PUVA or UVB.

An underlying allergic or irritant contact dermatitis needs to be considered and addressed.

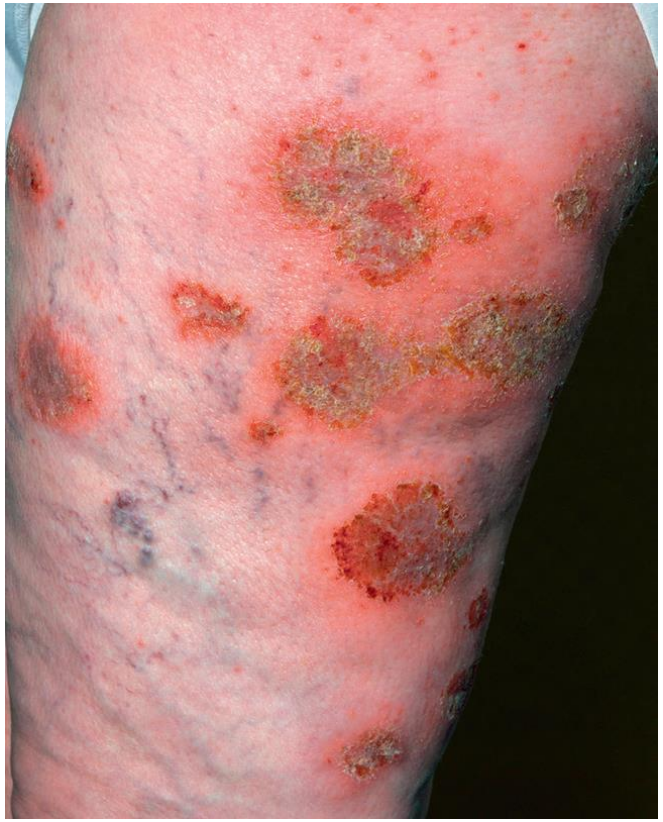
NUMMULAR DERMATITIS

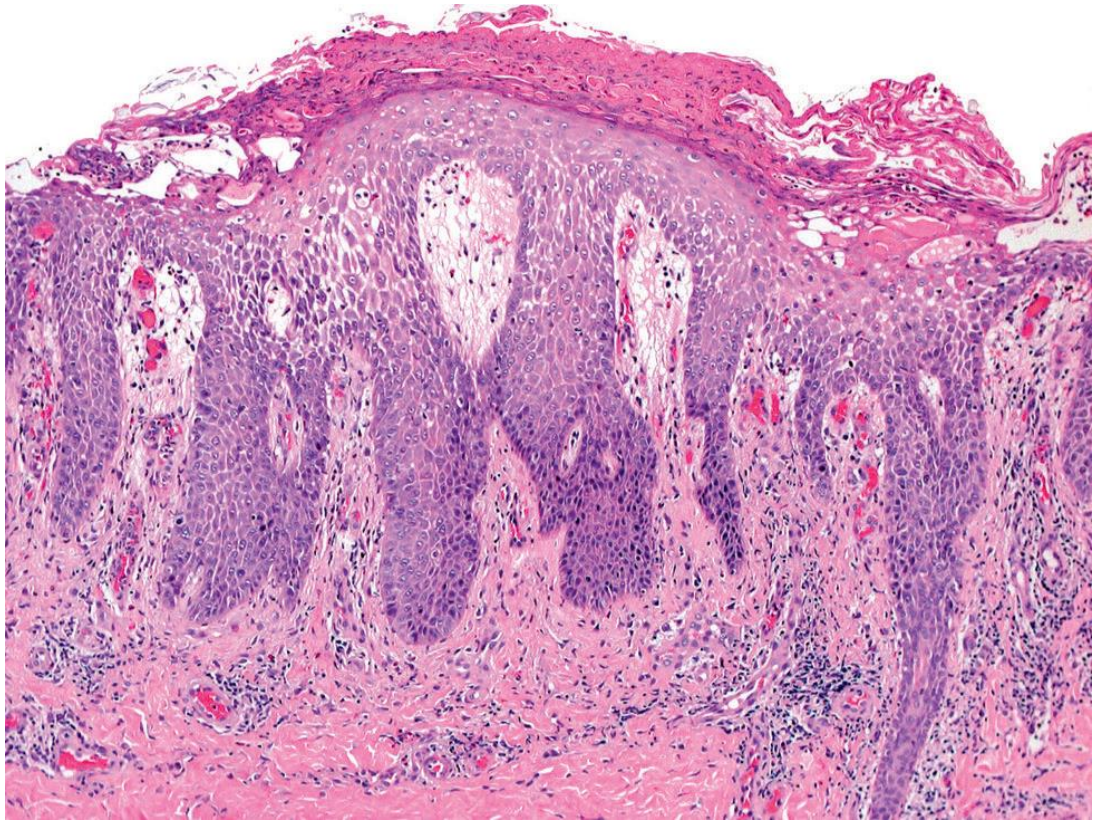
- Clinical Features
- Nummular dermatitis is defined as an eruption of round (discoid)

eczematous patches almost exclusively of the extremities, often the lower legs in men and the forearms and dorsal aspects of the hands in women.

- The lesions are well demarcated and measure 1–3 cm, only occasionally being larger.
- They may be acutely inflamed with vesicles and weeping, but are often lichenified and hyperkeratotic.

- Pruritus may be intense and excoriations are often prominent. Nummular dermatitis usually takes a very chronic course.
- A particularly therapy-resistant variety has been termed “oid-oid” disease, also referred to as exudative discoid and lichenoid chronic dermatitis or Sulzberger–Garbe syndrome.





- Differential Diagnosis
- Nummular dermatitis must be distinguished from nummular lesions of atopic dermatitis and dissemination secondary to contact dermatitis or stasis dermatitis.
- Other conditions to be considered are psoriasis, Bowen disease, mycosis fungoides, and tinea corporis.

- Treatment
- Options include medium- to high-potency topical corticosteroid ointments, topical tacrolimus or pimecrolimus, and emollients.
- phototherapy

Stasis Dermatitis

Stasis Dermatitis

- **Etiology.** Stasis dermatitis is an eczematous eruption that occurs on the lower legs in some patients with venous insufficiency.
- The dermatitis may be acute, subacute, or chronic and recurrent, and it may be accompanied by ulceration.
- Most patients with venous insufficiency do not develop dermatitis, which suggests that genetic or environmental factors may play a role.
- Some have speculated that it represents an allergic response to an epidermal protein antigen created through increased hydrostatic pressure, whereas others believe that the skin has been compromised and is more susceptible to irritation and trauma.

- **Allergy to Topical Agents.** Patients with stasis dermatitis have significantly more positive reactions when patch tested with components of previously used topical agents.
- Topical medications that contain potential sensitizers such as lanolin, benzocaine, parabens, and neomycin should be avoided by patients with stasis disease.
- Allergy to corticosteroids in topical medication is also possible.

Types of Eczematous Inflammation

- ***Subacute Inflammation***
- Subacute inflammation usually begins in the winter months when the legs become dry and scaly.
- Brown staining of the skin (hemosiderin) may have appeared slowly for months.
- The pigment is iron remaining after disintegration of red blood cells that leaked out of veins because of increased hydrostatic pressure.
- Scratching induces first subacute and then chronic eczematous inflammation.

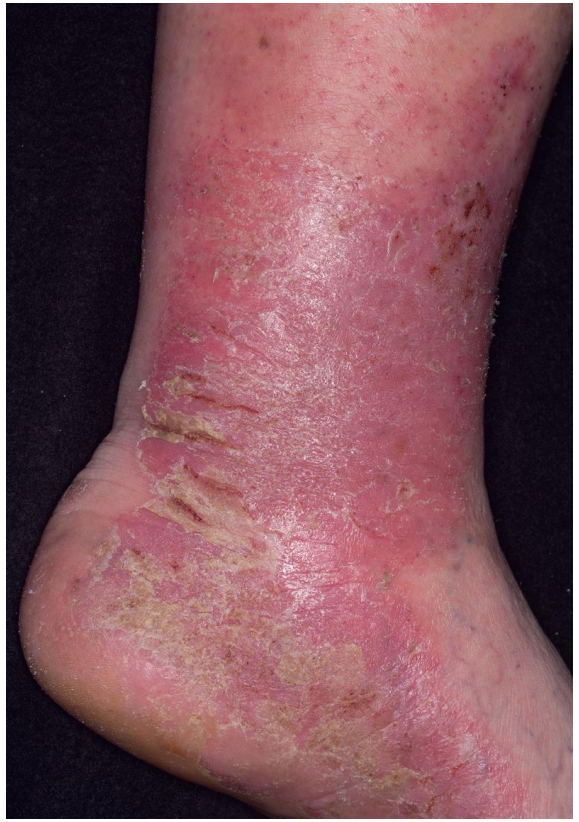
- ***Acute Inflammation***
- A red, superficial itchy plaque may suddenly appear on the lower leg. This acute process may be eczematous inflammation,cellulitis, or both.
- Weeping and crusts appear. A vesicular eruption (id reaction) on the palms, trunk, and/or extremities sometimes accompanies this acute inflammation.
- The inflammation responds to systemic antibiotics, wet compresses, and group III to V topical steroids.
- Wet compresses should be discontinued before excessive drying occurs.
- The id reaction resolves spontaneously as the primary site improves.

- ***Chronic Inflammation***
- Recurrent attacks of inflammation eventually compromise the poorly vascularized area, and the disease becomes chronic and recurrent.
- The typical presentation is a cyanotic red plaque over the medial malleolus.
- Fibrosis following chronic inflammation leads to permanent skin thickening.
- The skin surface in these irreversibly changed areas may have a bumpy, cobblestone appearance that results from fibrosis and venous and lymph stasis.
- The skin remains thickened and diffusely dark brown (postinflammatory hyperpigmentation) during quiescent periods.

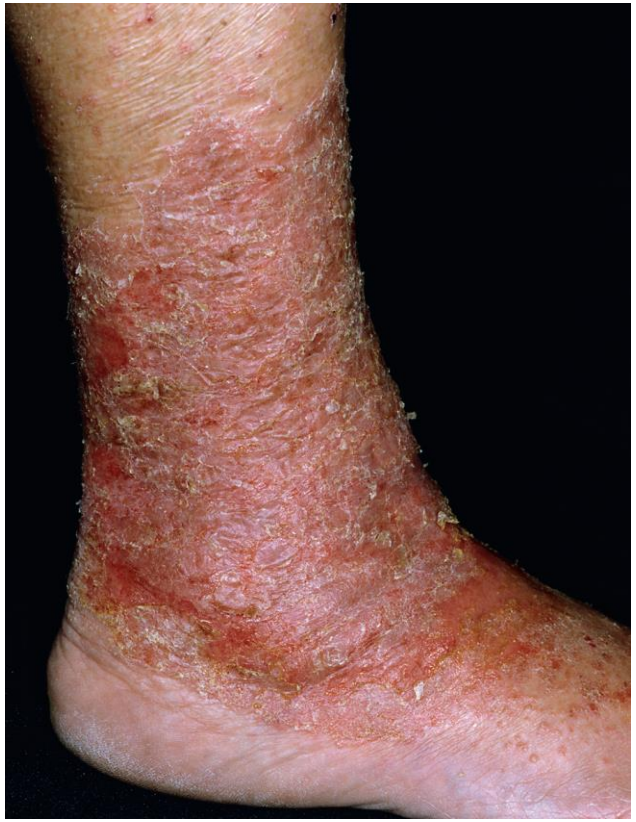
- ***Treatment of Stasis Dermatitis***
- **Topical Steroids and Wet Dressings.** The early, dry superficial stage is managed as subacute eczematous inflammation with group II to V topical steroid creams or ointmentsand lubricating creams or lotions.
- Oral antibiotics(usually those active against *Staphylococci,* e.g., cephalexin) hasten resolution if cellulitis is present.
- Moist exudative inflammation and moist ulcers respond to tepid wet compresses of Burow's solution or merely saline or water for 30 to 60 minutes several times a day. Wet dressings suppres inflammation while debriding the ulcer.

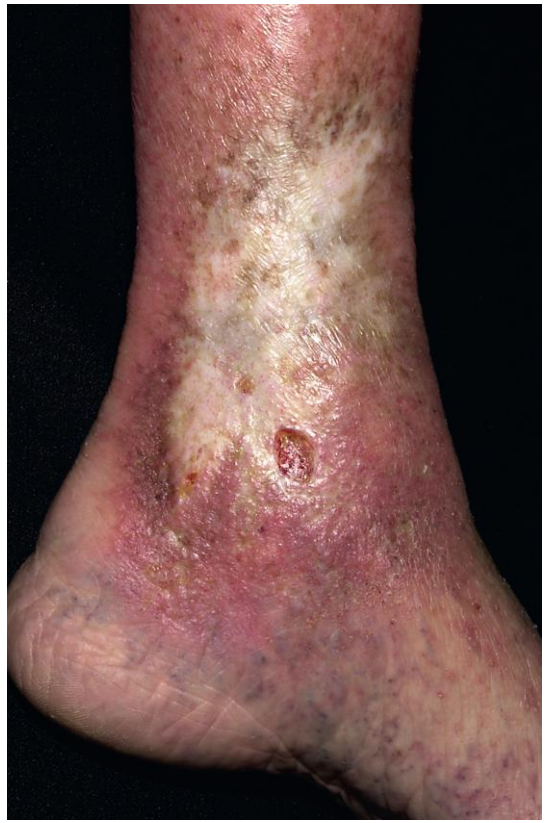
- Adherent crust may be carefully freed with blunt-tipped scissors.
- Group V topical steroids are applied to eczematous skin at the periphery of the ulcer. Patients must be warned that steroid creams placed on the ulcer stop the healing process.
- Elevation of the legs encourages healing.

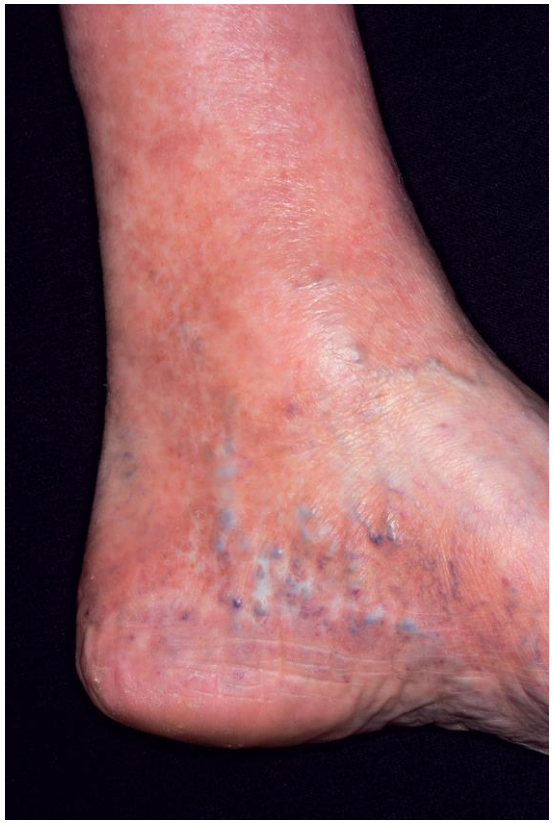
Severe, painful, exudative, weeping infected eczema with moist crust.



red, itchy plaque may suddenly develop acute inflammation







SEBORRHEIC DERMATITIS

- Seborrheic dermatitis is a common mild chronic eczema typically confined to skin regions with high sebum production and the large body folds.
- Although its pathogenesis is not fully elucidated, there is a link to sebum overproduction (seborrhea) and the commensal yeast *Malassezia*.

- There are infantile and adult forms, with the former being self-limited and confined to the first 3 months of life, while the latter is chronic with a peak in the fourth to sixth decades.
- The prevalence of seborrheic dermatitis is estimated to be 5%.
- Extensive and therapy-resistant seborrheic dermatitis is an important cutaneous sign of HIV infection. It is also more commonly observed in patients with Parkinson disease, cerebrovascular accidents, and mood disorders.
- In neonates and chidren consider acrodermatitis entropathica and zinc deficiency.

- There is no simple quantitative relationship between yeast number and severity of seborrheic dermatitis, and unaffected skin may carry a load of organisms similar to seborrheic dermatitis lesions.
- Seborrheic dermatitis occurs predominantly in areas of the skin with active sebaceous glands and is often associated with sebum overproduction.

- However, patients with seborrheic dermatitis may have normal sebum production and those with excessive sebum production are often free of seborrheic dermatitis. Thus, the amount of sebum produced alone does not appear to be the decisive risk factor.
- In patients with seborrheic dermatitis, triglycerides and cholesterol are elevated but squalene and free fatty acids are significantly decreased.
- Free fatty acids (which have a known antimicrobial effect) are formed from triglycerides by bacterial lipases, produced by the lipolytic *Propionibacterium* (*Corynebacterium*) *acnes*.
- A major constituent of the resident microbial skin flora, *P. acnes* has been found to be greatly reduced in seborrheic dermatitis.

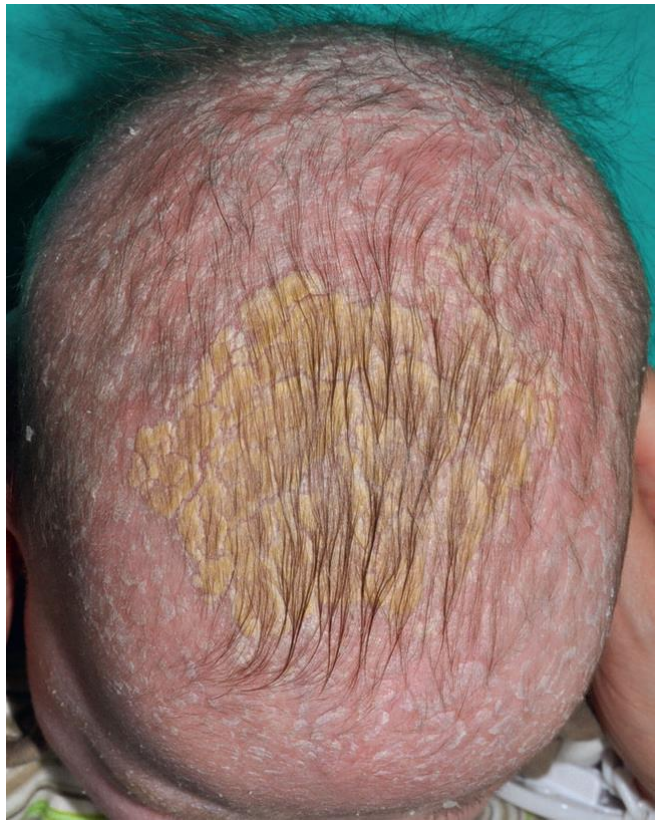
Clinical Features

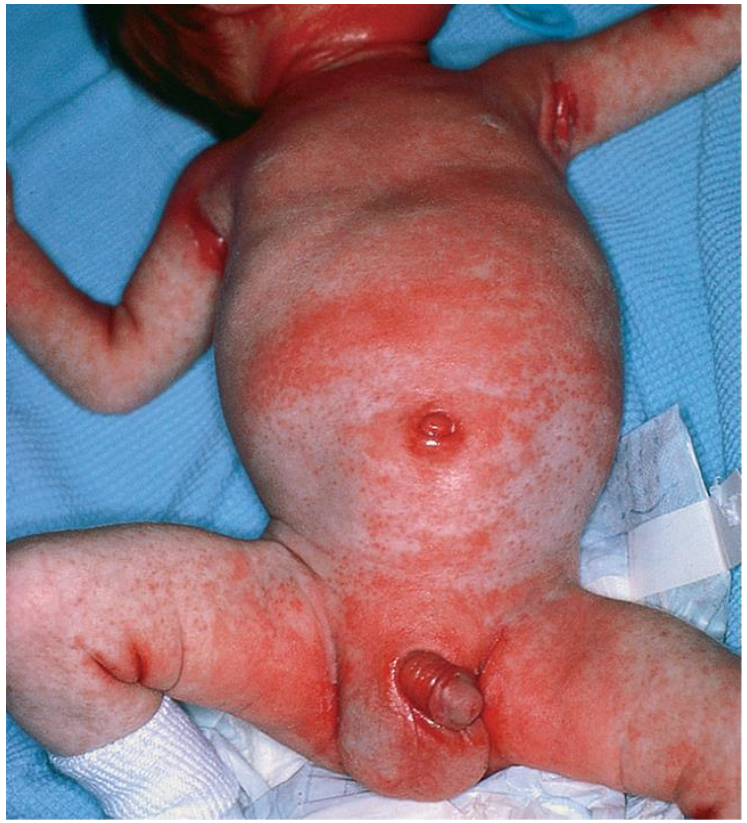
- Seborrheic dermatitis is defined by clinical parameters, including: sharply demarcated patches or thin plaques that vary from pink–yellow to dull red to red–brown with scales; vesiculation and crusting may occur but are rare and mostly due to irritation.

- Predilection for areas rich in sebaceous glands – scalp, face, ears,presternal region – and, less often, the intertriginous areas.
- Mild course with little or moderate discomfort.
- Generalized and even erythrodermic forms can occur, albeit rarely.

Infantile seborrheic dermatitis

- This form usually begins about one week after birth and may persist for several months. Initially, mild greasy scales adherent to the vertex and anterior fontanelle regions arise which may later extend over theentire scalp.
- Inflammation and oozing may finally result in a coherent scaly and crusty mass covering most of the scalp cradle cap.
- Lesions of the axillae, inguinal creases, neck, and retroauricular folds are often acutely inflamed, oozing, sharply demarcated, and surrounded by satellite lesions.
- Superinfection with *Candida* spp. or occasionally bacteria (e.g. group A *Streptococcus*) can occur.



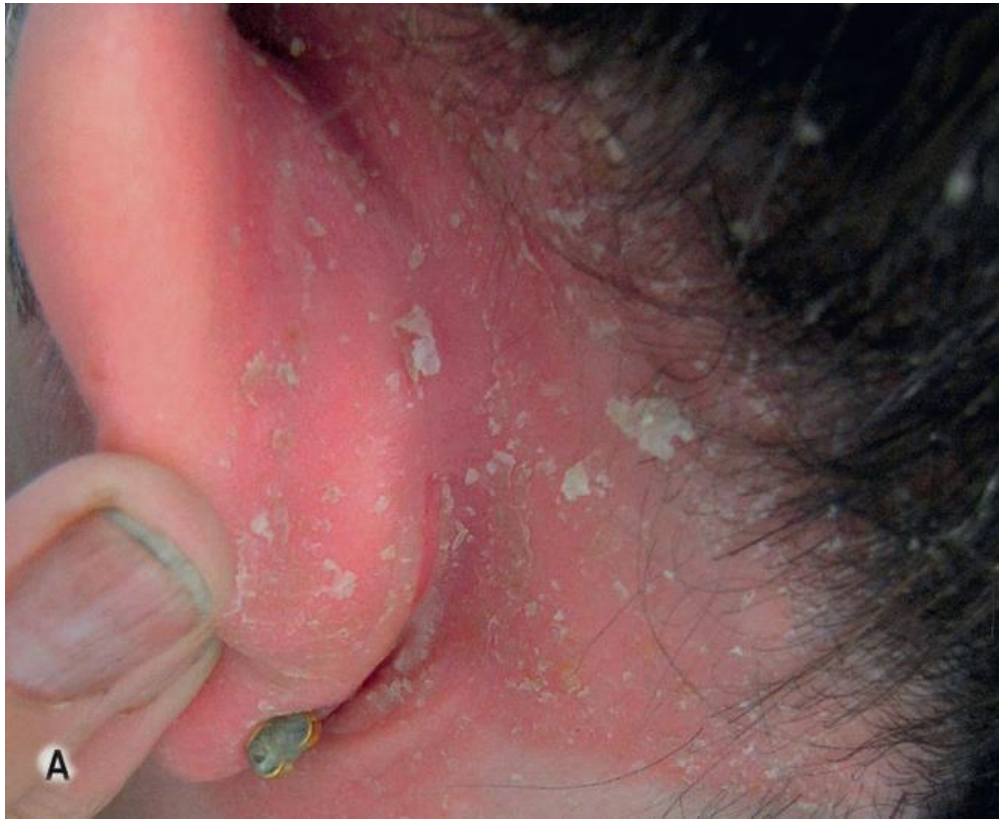


Adult seborrheic dermatitis

- In adults, seborrheic dermatitis is generally found on the scalp and,usually of milder intensity, on the face; less often, lesions occur on thecentral upper chest and the intertriginous areas. Erythrodermic seborrheic dermatitis has been described as a rarity.
- In seborrheic dermatitis of the scalp, there is inflammation and pruritus in addition to dandruff.
- The vertex and parietal regions are predominantly affected, but in a more diffuse pattern than the discrete plaques of psoriasis.

- Seborrheic dermatitis of the facial skin is often strikingly symmetric,affecting the forehead, medial portions of the eyebrows, upper eyelids,nasolabial folds and lateral aspects of the nose, retroauricular areas, and occasionally the occiput and neck.
- Seborrheic dermatitis, like inverse psoriasis, is a cause of intertrigo.
- In patients with seborrheic dermatitis, the skin is sensitive to irritation, and exposure to sun or heat, febrile illnesses, and overly aggressive topical therapy may precipitate flares and dissemination.

- *Malassezia*
- *(Pityrosporum)* folliculitis is another complication characterized by pruritic erythematous follicular papules, sometimes pustules, typically in sites rich in sebaceous glands.
- The facial immobility of patients with Parkinson disease might result in a greater accumulation of sebum on the skin, resulting in a permissive effect on the growth of *Malassezia.*
- Rebound flares of seborrheic dermatitis can follow tapers of systemic corticosteroids.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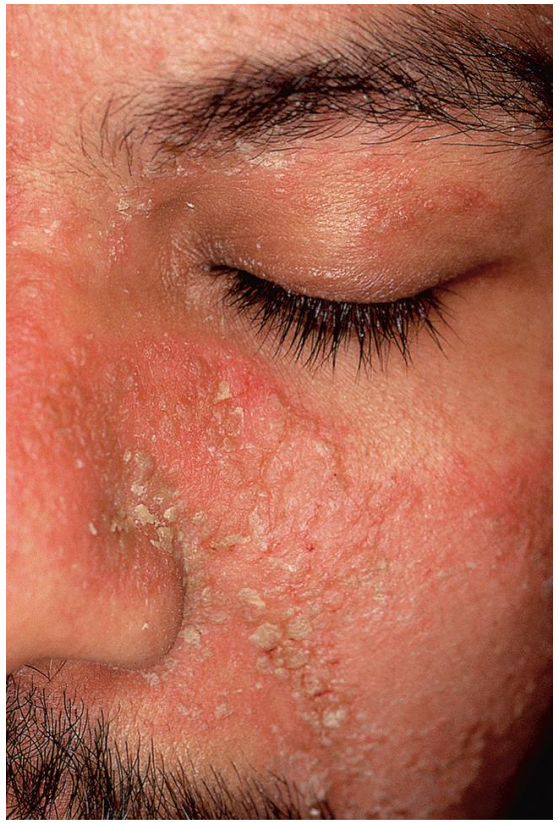
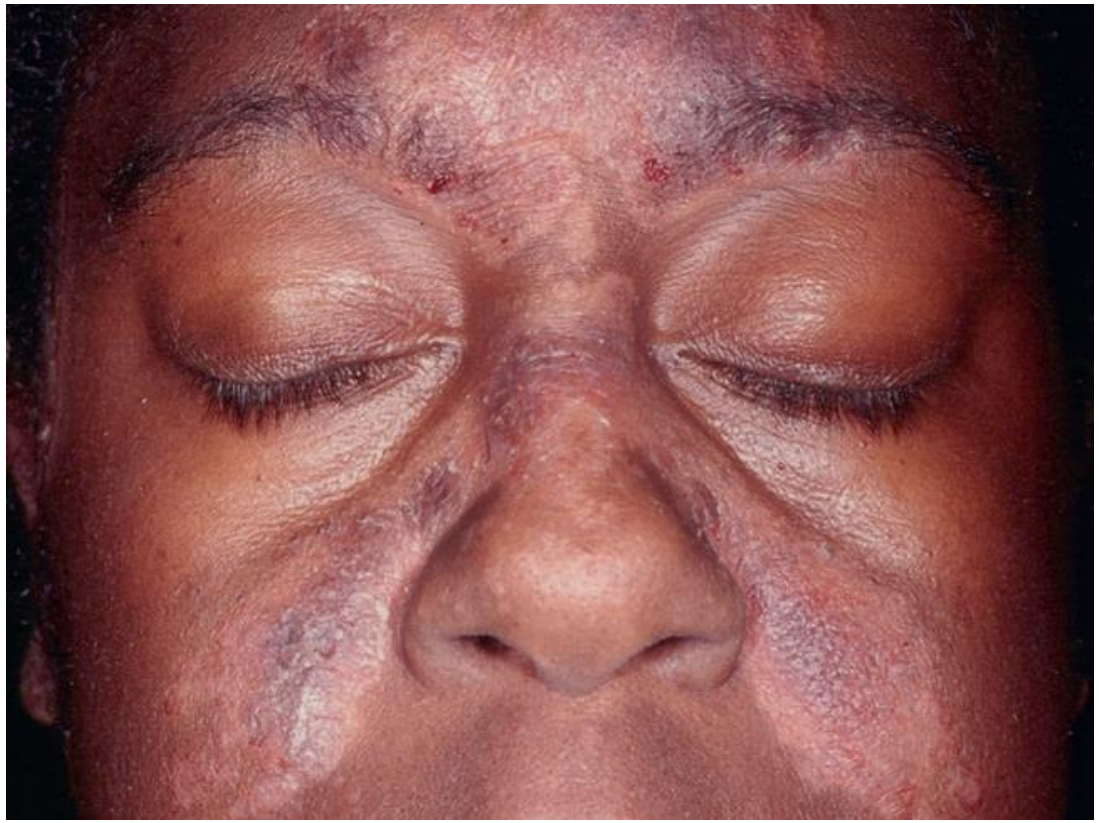




Treatment

Infantile seborrheic dermatitis

- Infantile seborrheic dermatitis usually responds satisfactorily to bathing and application of emollients. Ketoconazole cream (2%) is indicated in more extensive or persistent cases.
- Short courses of low-potency topical corticosteroids may be used initially to suppress inflammation.

Adult seborrheic dermatitis

- The mainstay of therapy is the use of topical azoles (e.g. ketoconazole),either as shampoos (scalp) or as creams (body).
- Ciclopirox olamine has antifungal and anti-inflammatory activities and has also been shown to be effective as a shampoo or cream in double-blind, randomized trials.
- Additional measures, particularly in the initial stages of treatment,include emollients and low-potency topical corticosteroids.
- Second-line treatment options include zinc pyrithione, selenium sulfide, and tar shampoos as well as topical calcineurin inhibitors.

- Lithium gluconate 8%
- Ciclopirox olamine cream
- Miconazole- clotrimazole
- Oral terbinafine
- Oral itraconasole
- Phototherapy
- Benzoyl peroxide
- Topical terbinafin

Lichen Simplex Chronicus

Lichen Simplex Chronicus

- Also known as circumscribed neurodermatitis, lichen simplex chronicus results from long-term chronic rubbing and scratching,

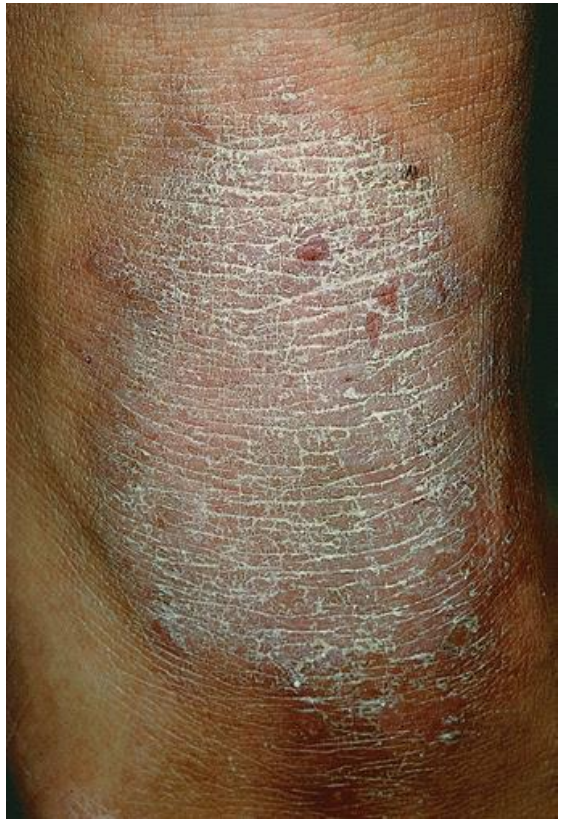
more vigorously than a normal pain threshold would permit, with

the skin becoming thickened and leathery.

- This change, known as lichenification, may originate on seemingly normal skin or may develop on skin that is the site of another disease, such as atopic or allergic contact dermatitis or ringworm.

- Circumscribed, lichenified pruritic patches may develop on any part of the body.
- predilection for the back and sides of the neck, the scalp, the upper eyelid, the orifice of one or both ears, the palm, soles, or often the wrist and ankle flexures.
- The vulva, scrotum, and anal areas are common sites, although the genital and anal areas are seldom involved at the same time.

- Persistent rubbing of the shins or upper back may result in dermal deposits of amyloid and the subsequent development of lichen or macular amyloidosis, respectively.
- It may be associated with anxiety disorders and in depressed patients.



- **Treatment**
- A high-potency steroid cream or ointment should be used

initially but not indefinitely because of the potential for steroidinduced

atrophy.

- Topical doxepin, capsaicin, or pimecrolimus cream or tacrolimus ointment provides significant antipruritic effects and is a good adjunctive therapy.
- Intralesional injections of triamcinolone suspension, using a

concentration of 2.5–5 mg/mL, may be required.

- Botulinum toxin A injection may be curative.

- Prurigo Nodularis
- Prurigo nodularis is a disease with multiple itchy nodules mainly

on the extremities , especially on the anterior surfaces of the thighs and legs. A linear arrangement is common.

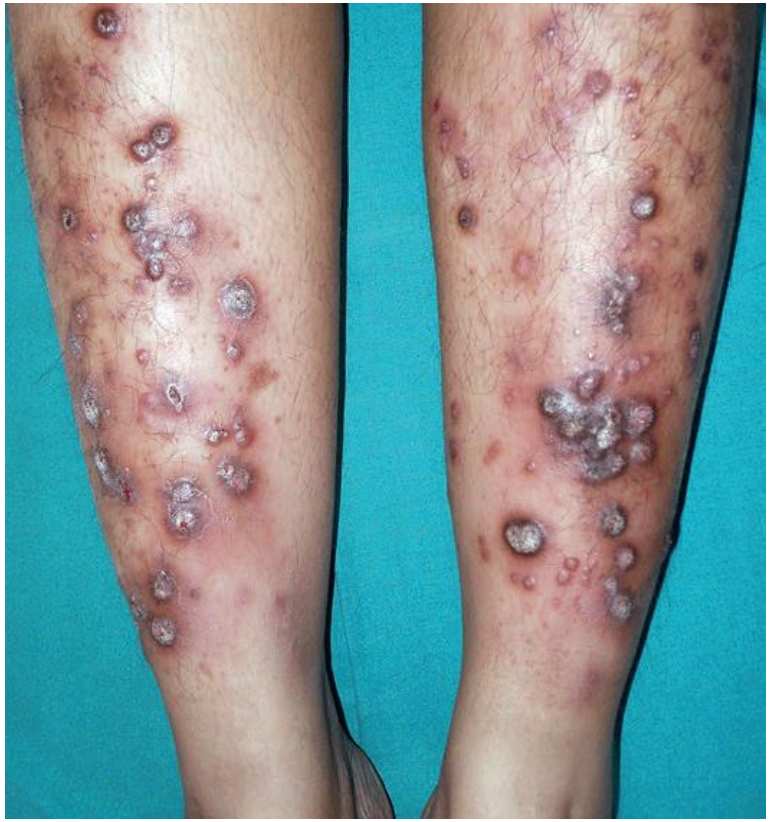
- The cause of prurigo nodularis is unknown; multiple factors

may contribute, including atopic dermatitis, hepatic diseases

(including hepatitis C), HIV disease, pregnancy, renal failure,

lymphoproliferative disease, stress, and insect bites.

Pemphigoid nodularis may be confused with prurigo nodularis clinically.



- **Treatment.** The initial treatment of choice for prurigo nodularis

is intralesional or topical administration of steroids.

- PUVA, NBUVB, and UVA alone have been shown to be effective in some
- patients.
- The combination product containing calcipotriene and betamethasone dipropionate ointment, calcitriol ointment, or tacrolimus ointment applied topically twice daily may be therapeutic and steroid sparing.
- Isotretinoin, 1 mg/kg/day for 2–5 months, may benefit some patients.
- Managing dry skin with emollients and avoidance of soap, with administration of antihistamines, antidepressants, or anxiolytics, is of moderate benefit in allaying symptoms.

- With thalidomide, onset may be rapid or slow, and sedation may occur; initial dose is 100 mg/day, tittered to the lowest dose required. Patients treated with thalidomide are at risk for developing a dose-dependent neuropathy at cumulative doses of 40–50 g.
- Lenalidomide, an analog of thalidomide, has less problems with neuropathy but may cause myelosuppression, venous thrombosis, and Stevens-Johnson syndrome.
- Methotrexate,
- 7.5–20 mg weekly produced improvement in 10 of 13 treated

patients.

- Pregabalin, 75 mg/day for 3 months, improved 23 of

30 patients in one study.

- Cyclosporine at doses of 3–4.5 mg/kg/ day has also been shown to be effective in treating recalcitrant disease.
- Cryotherapy may be used adjunctively.

Atopic Dermatitis

- Atopic dermatitis (AD) is a chronically relapsing skin disease that occurs most commonly during early infancy and childhood.
- It is frequently associated with elevated serum IgE levels and a personal or family history of AD, allergic rhinitis, and/or asthma.
- prevalence in children of 10 to 20 percent in the United States .
- The prevalence of AD in adults is approximately 1 to 3 percent.
- There is also a female preponderance for AD, with an overall female/male ratio of 1.3:1.

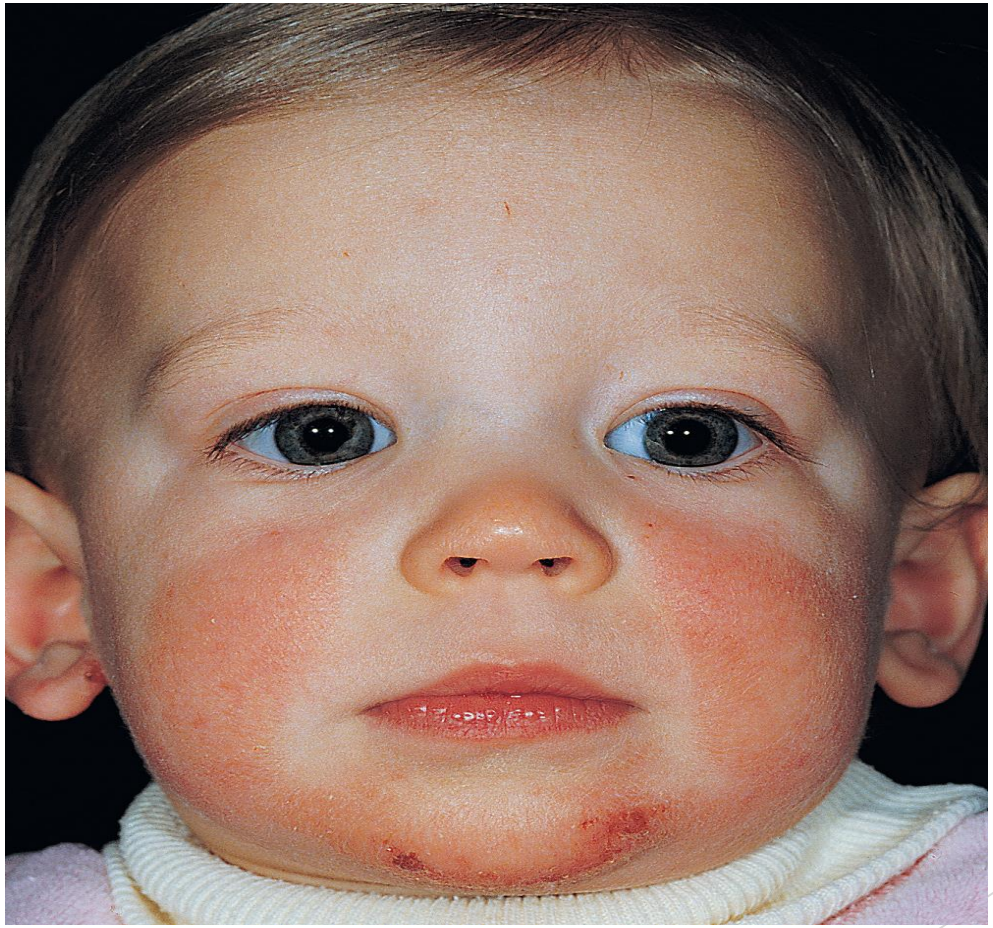
CLINICAL MANIFESTATIONS

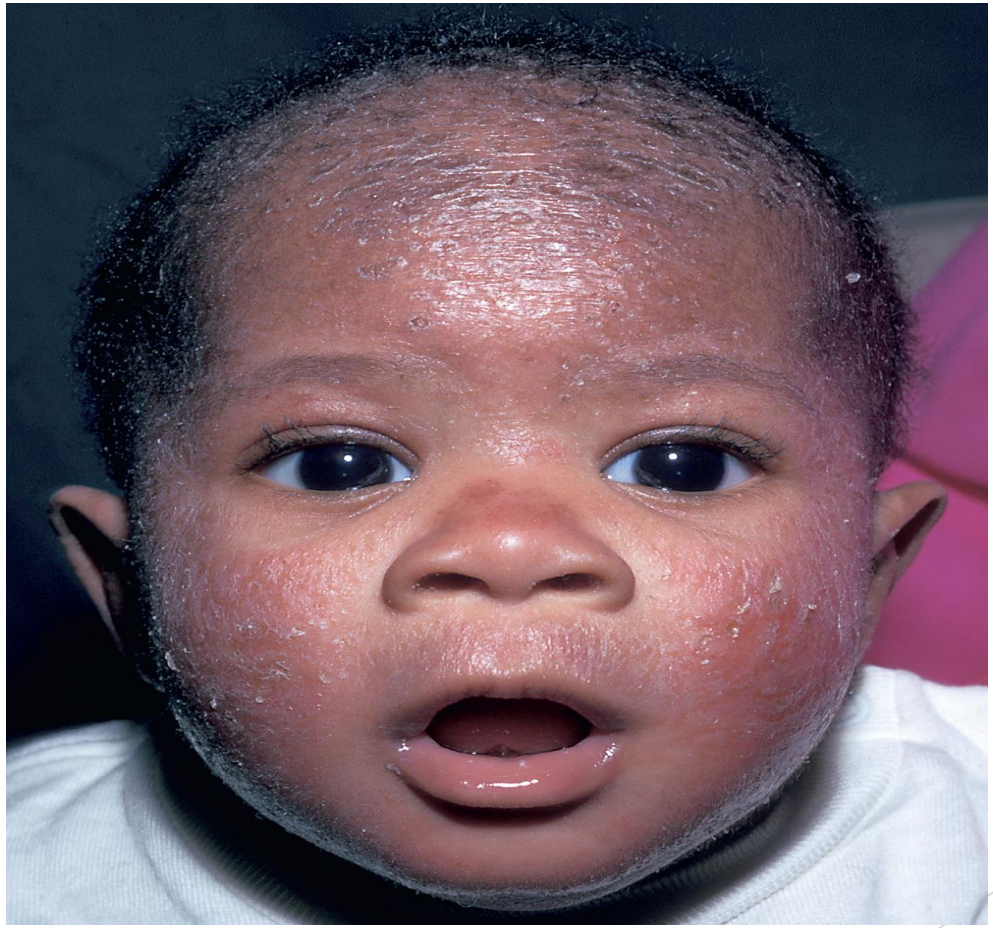
- AD typically begins during infancy.
- Approximately 50 percent of patients develop this illness by the first year of life and an additional 30 percent between the ages of 1 and 5 years.
- Nearly 80 percent of patients with AD eventually develop allergic rhinitis or asthma later in childhood.
- Intense pruritus and cutaneous reactivity are cardinal features of AD.
- Its consequences are scratching, prurigo papules , lichenification , and eczematous skin lesions.

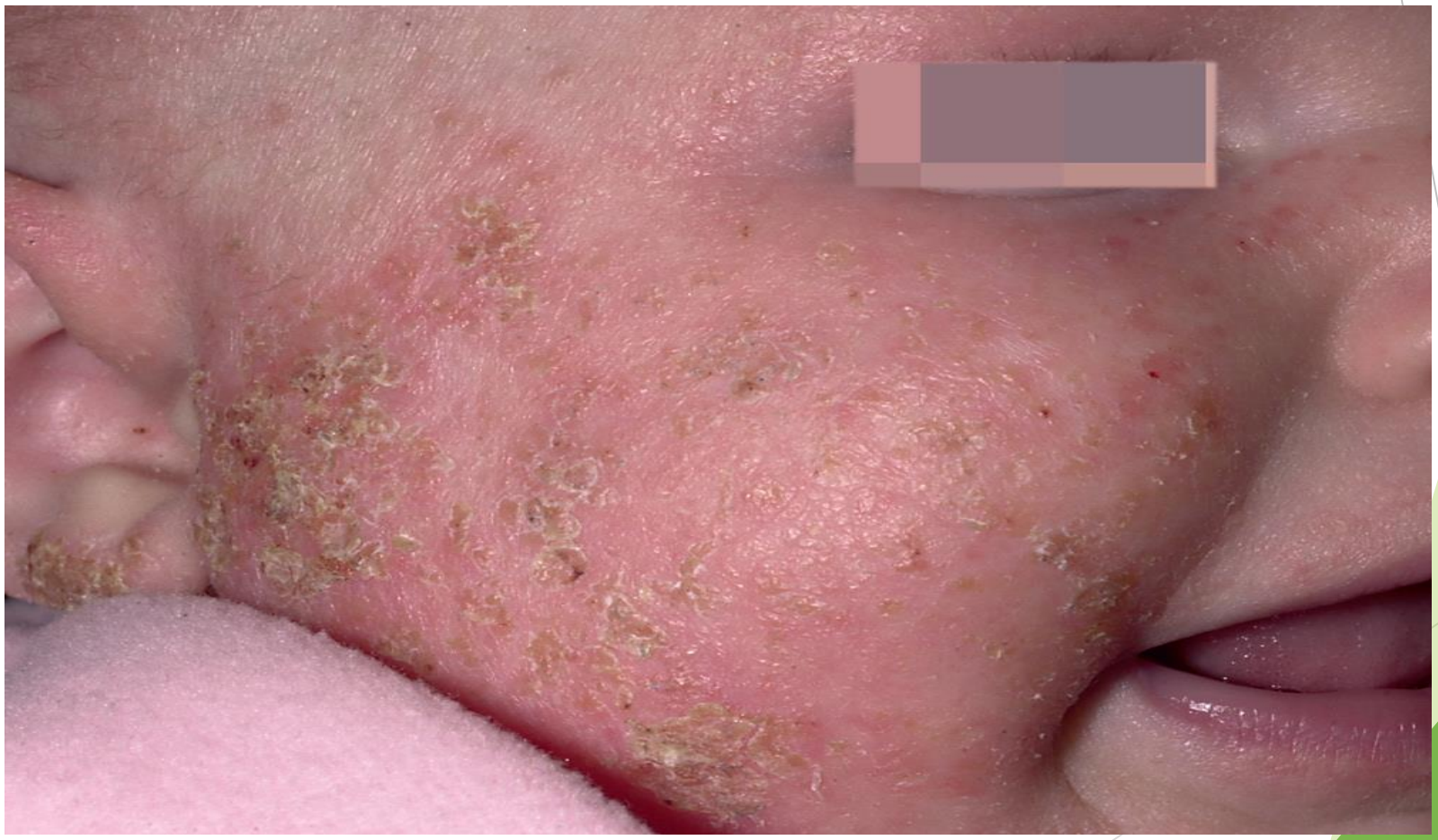
- Infantile phase (0-2 years)
- Majority start within 6 m and onset around 3 m most common but earlier onset unusual.
- Face, scalp, extensors,napkin area rarly affected.
- Chidhood phase (2-12 years)
- Flexural involvement ,antecubital and popliteal.
- Adolescent phase(12-18 years)
- Flexural and upper trunk and eyelids.

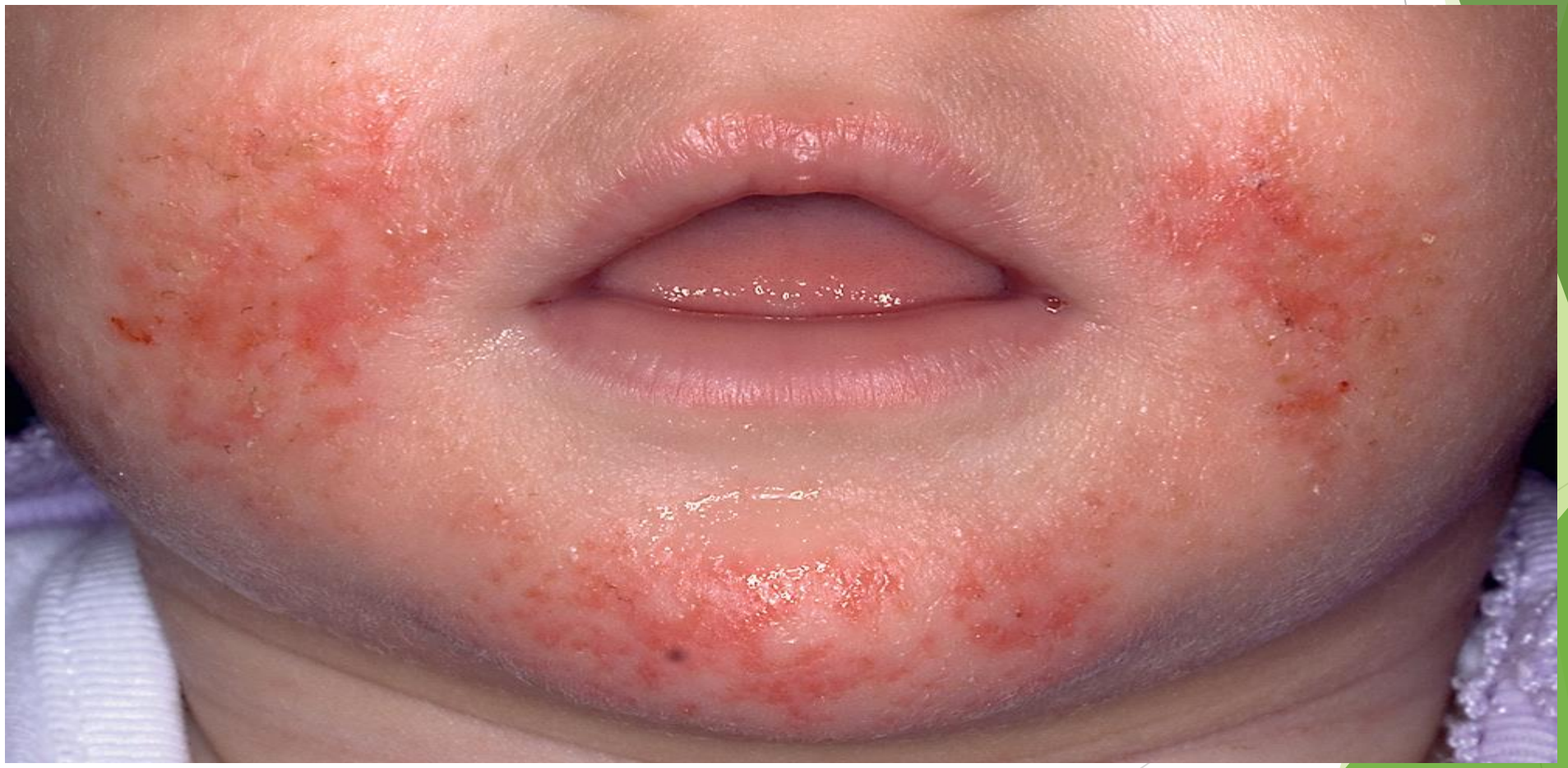
DIAGNOSIS

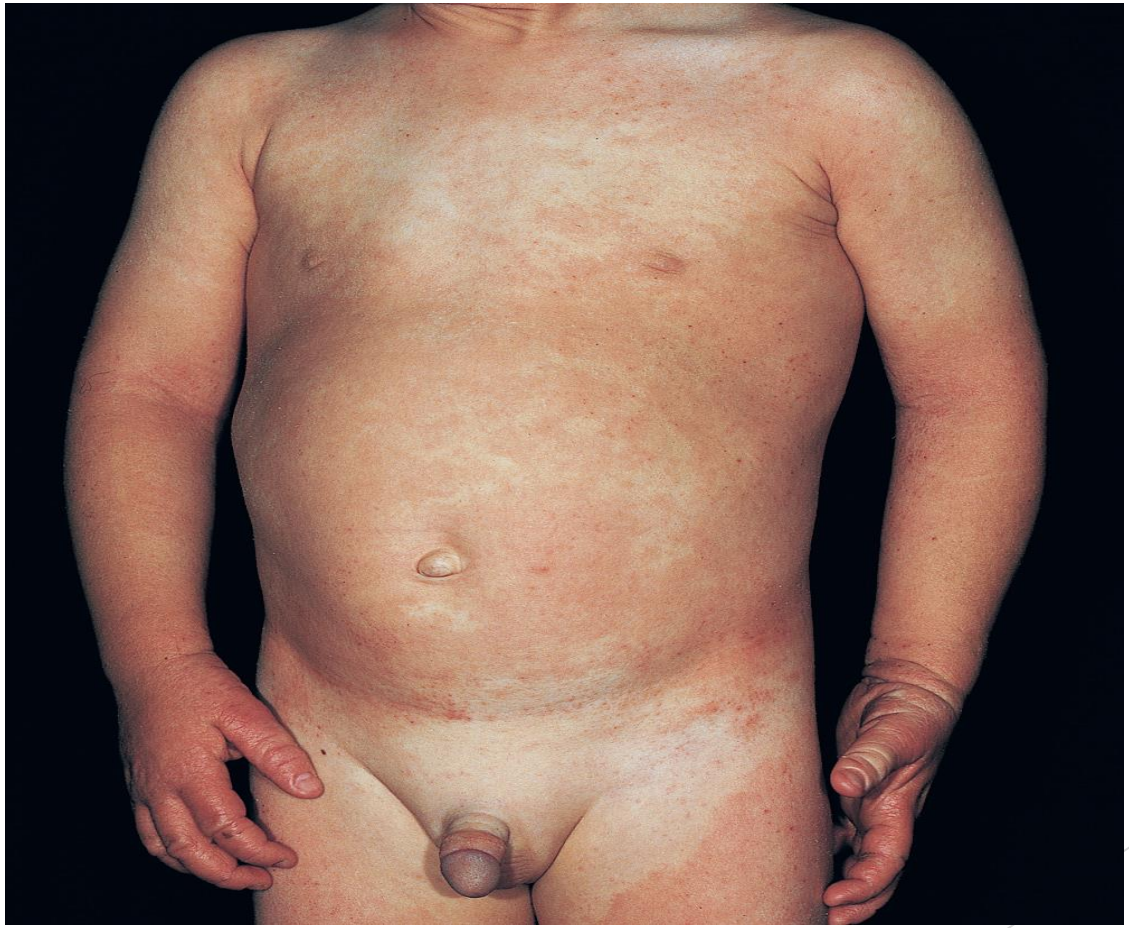
- Major criteria-all required
- Pruritus
- Typical morphology and distribution of rash.
- Common findings (at least two)
- Personal or family history of atopy
- Immediate skin test reactivity
- White dermographism
- Anterior subcapsular cataracts
- Associated findings (at least four)
- Ichthyosis’xerosis’hyperlinear palms’pityriasis alba’facial pallor’infraorbital darkening’dennie-morgan folds’keratoconus’hand dermatitis’repeated cutaneous infection.

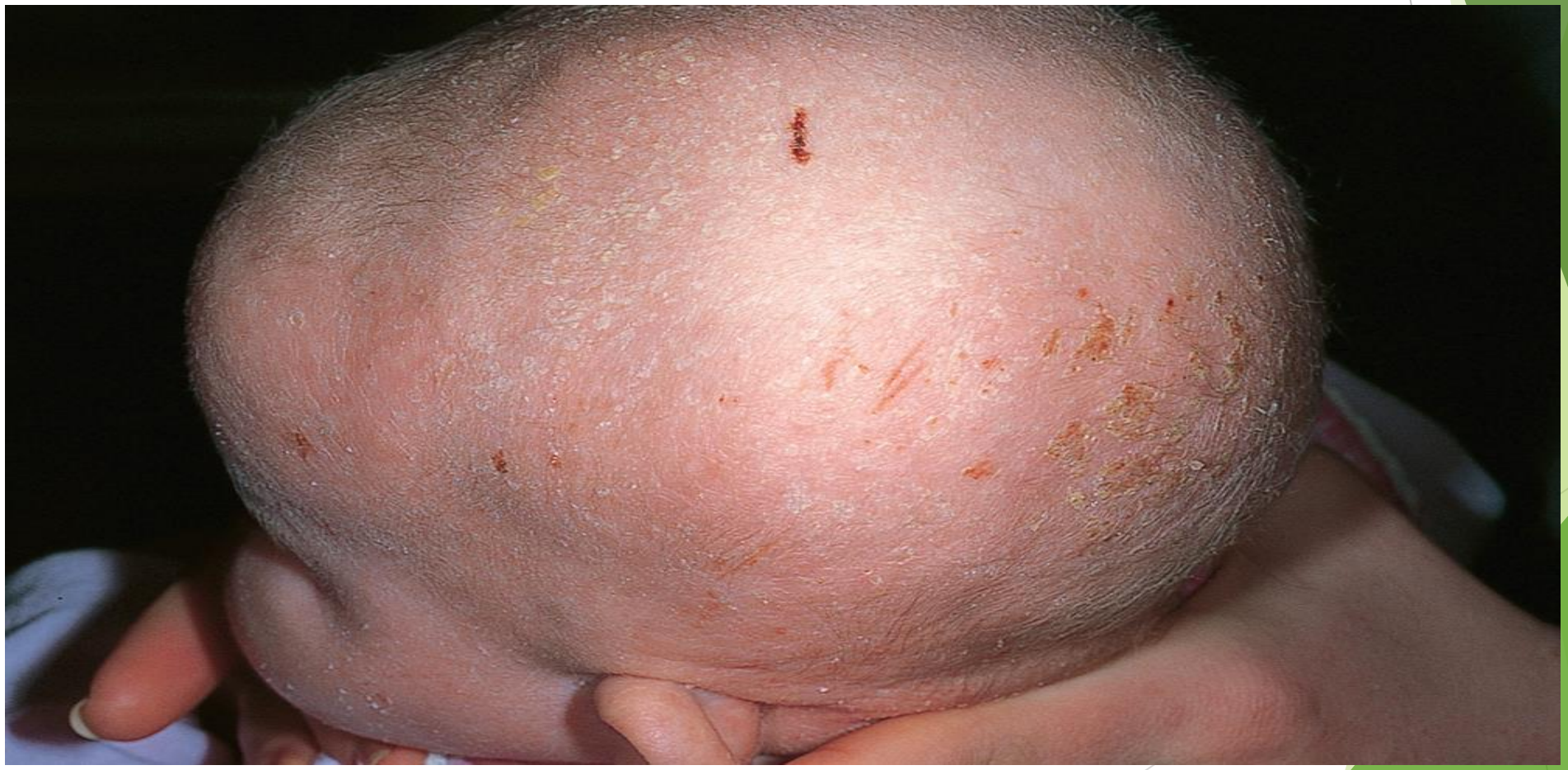


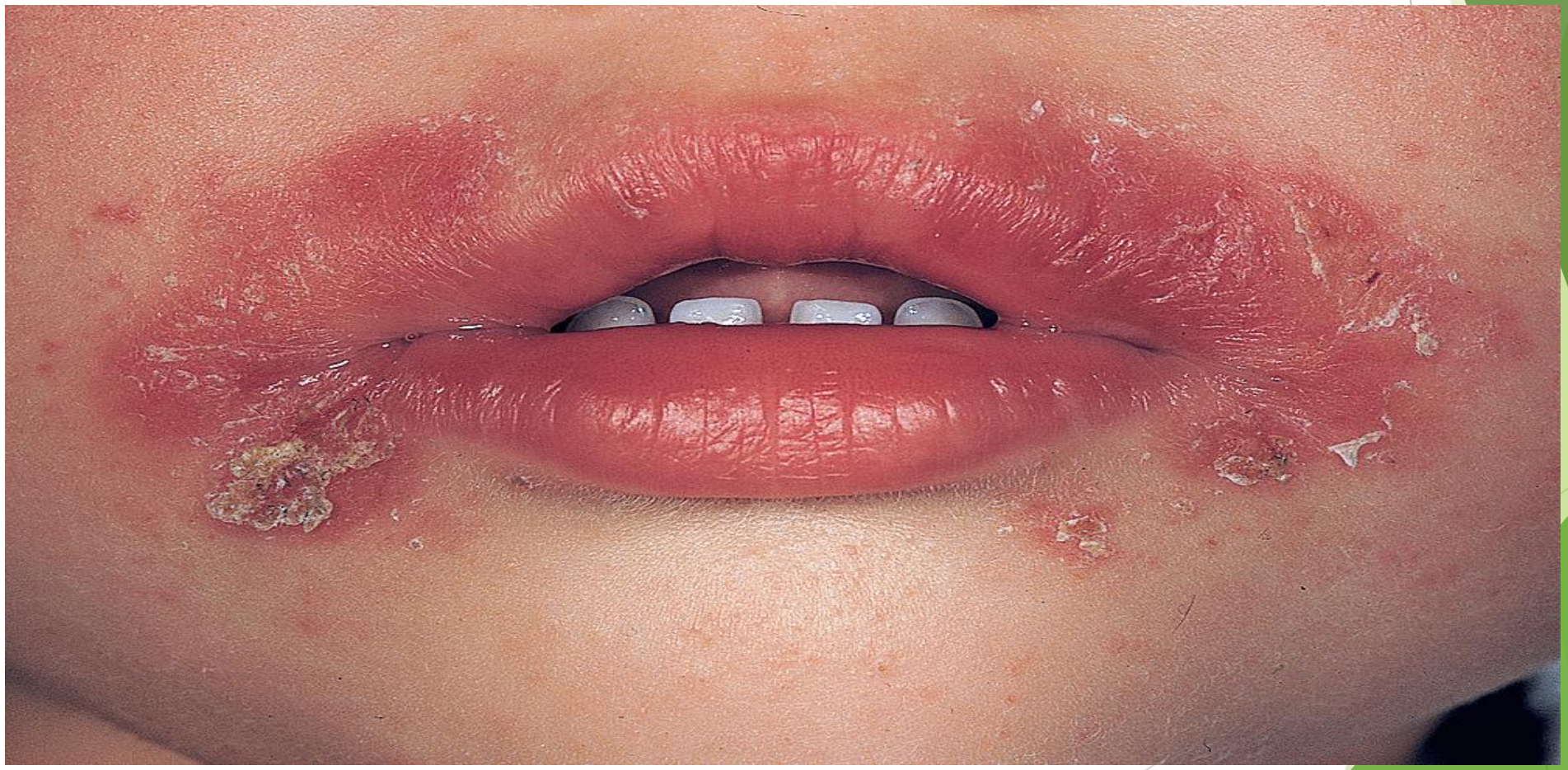


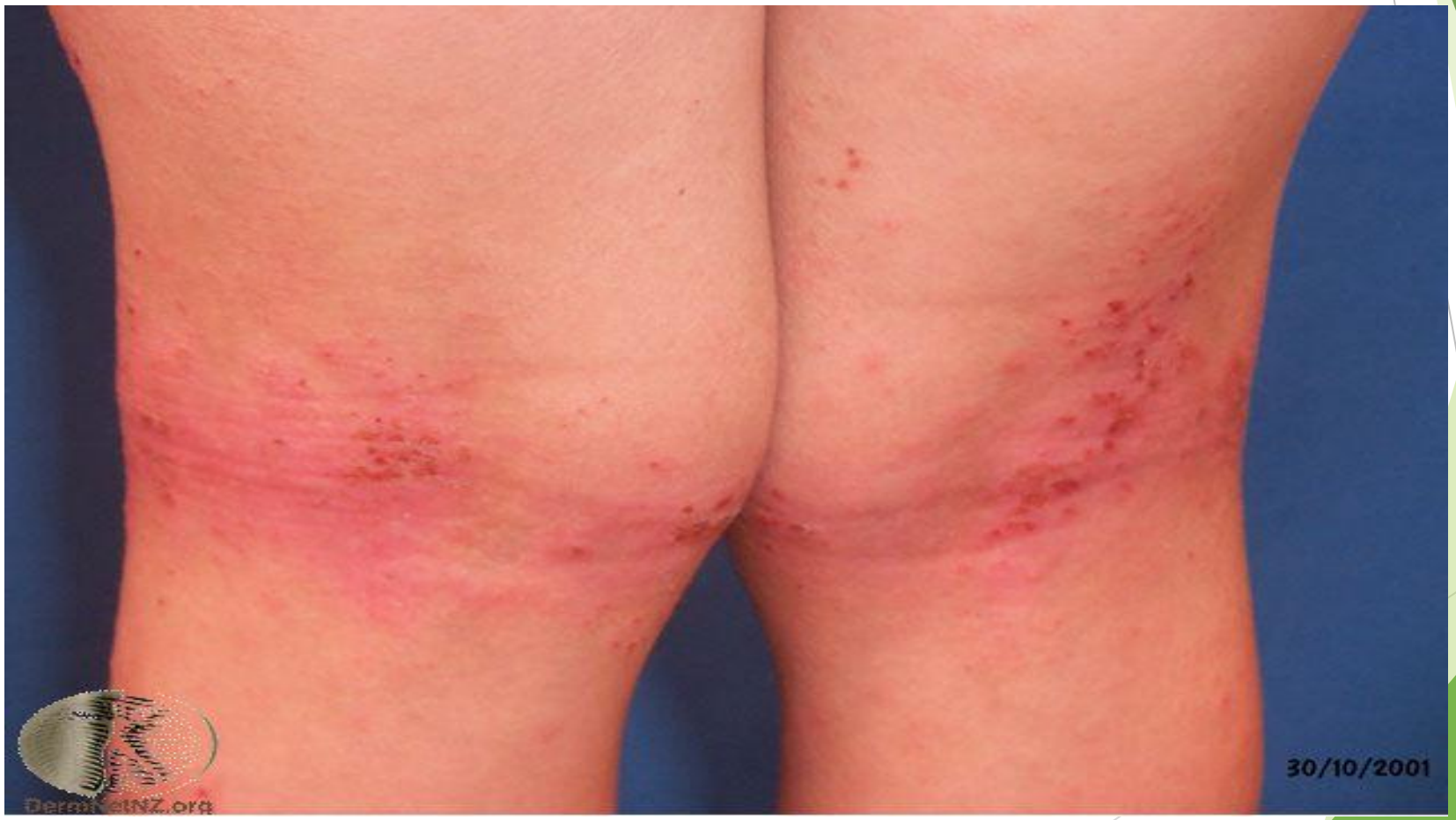








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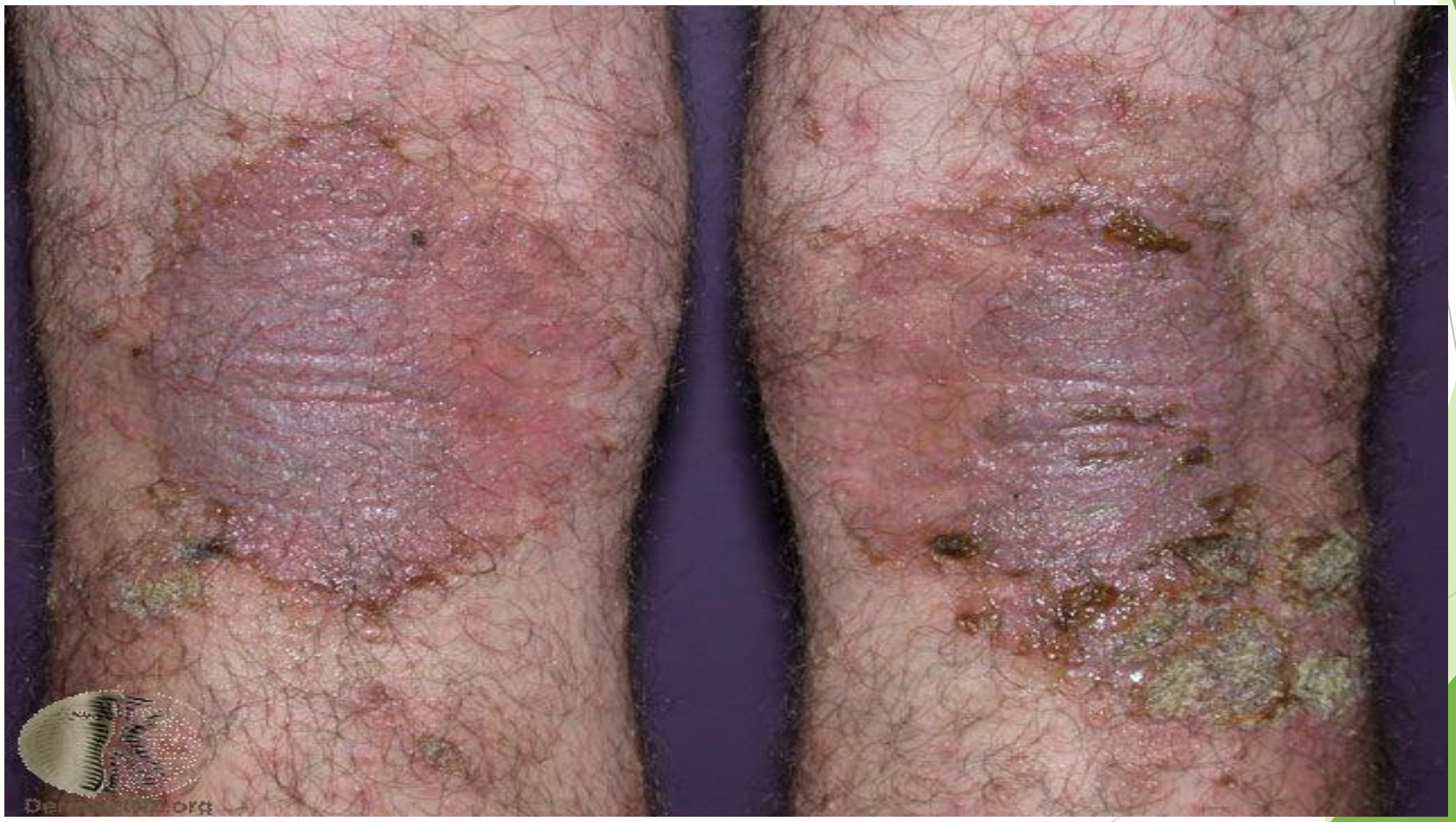

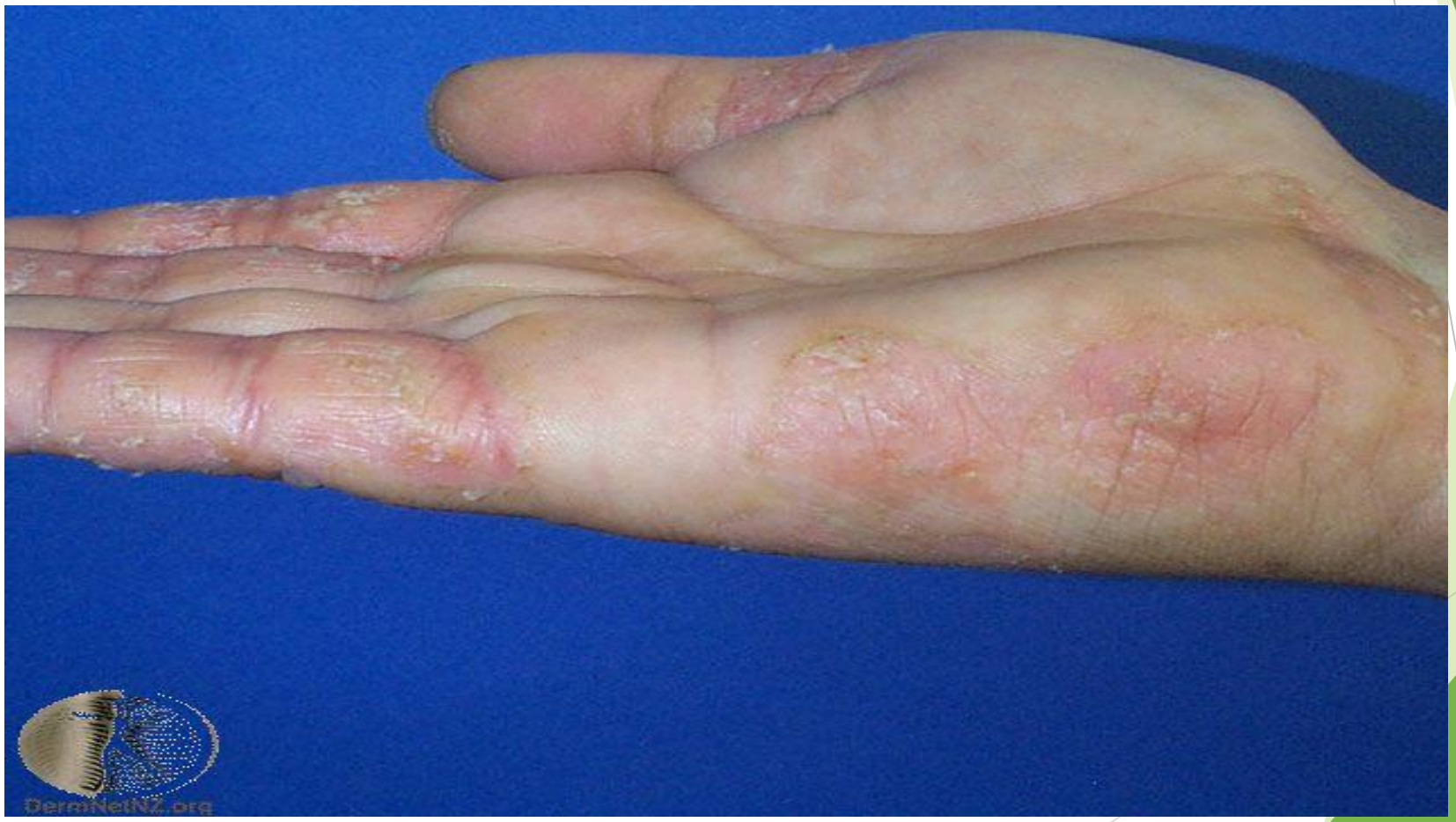
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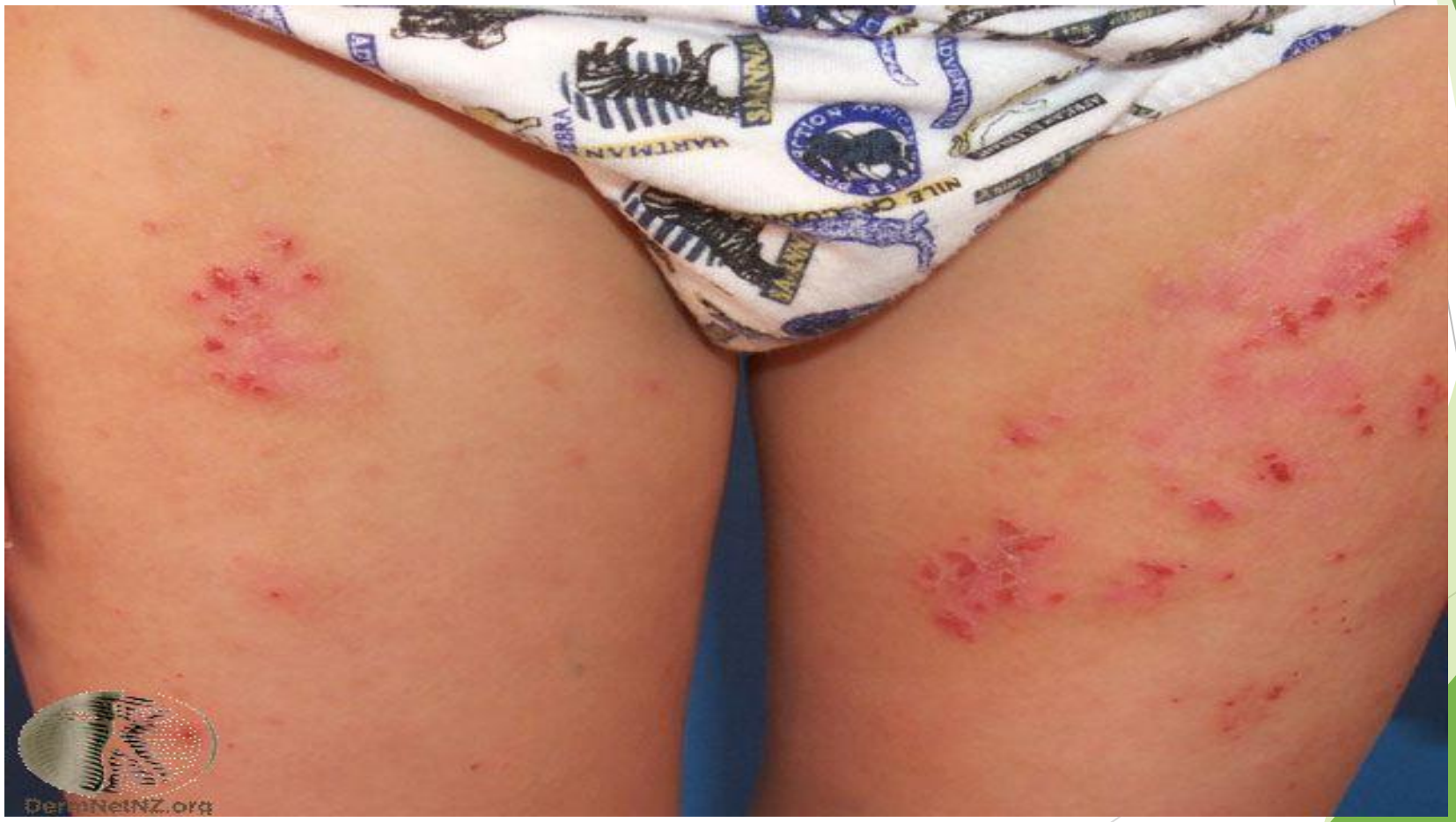
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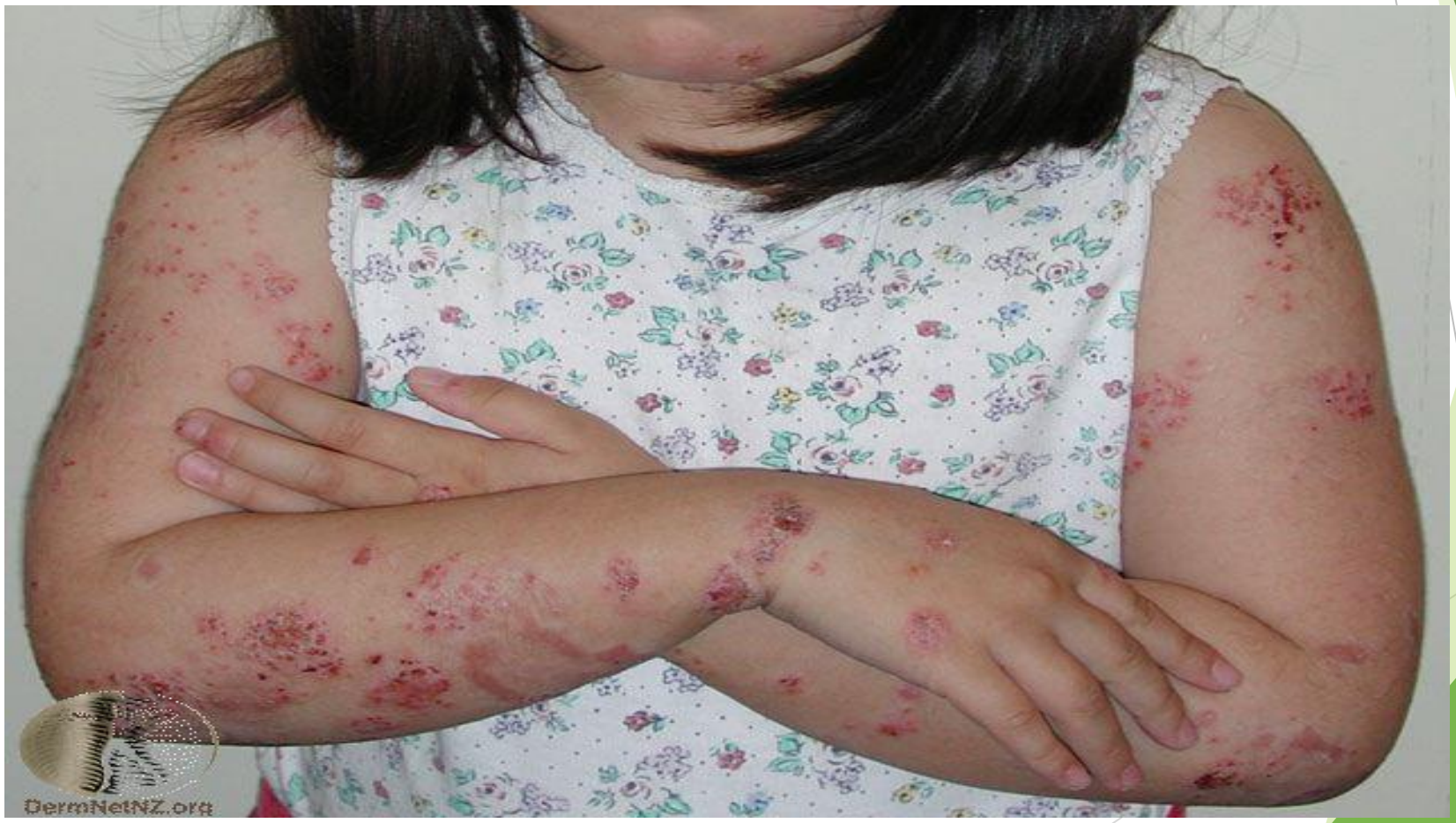
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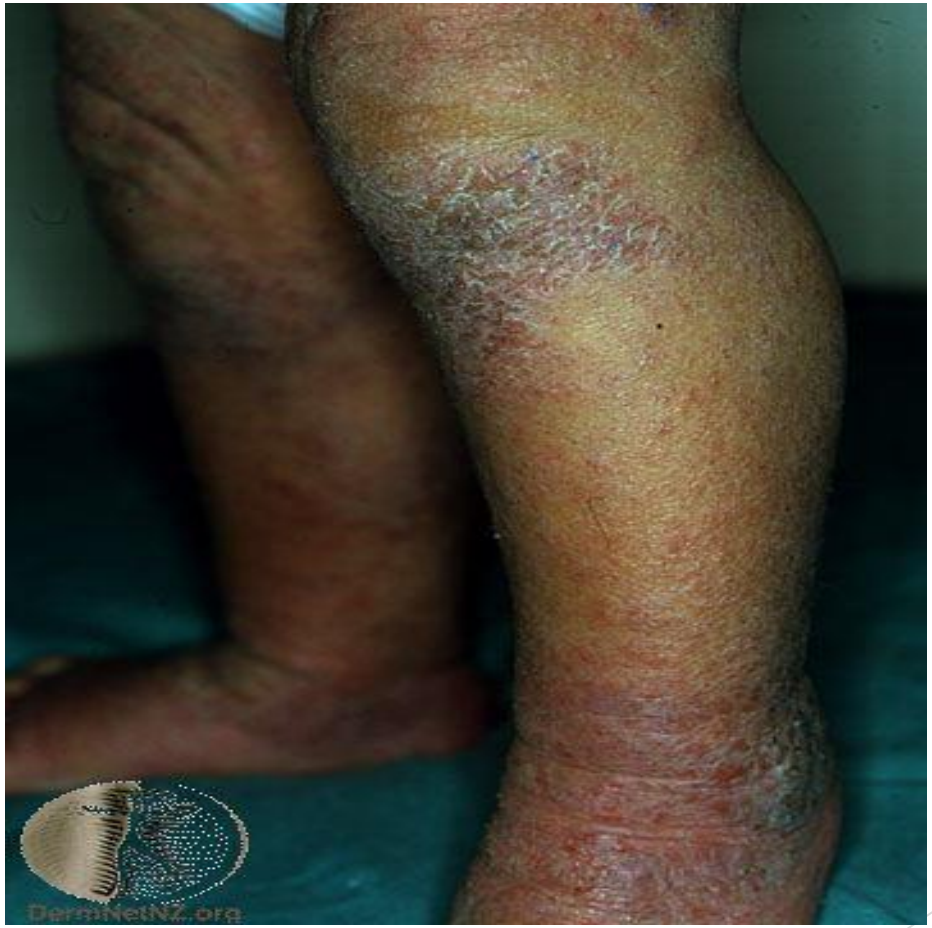
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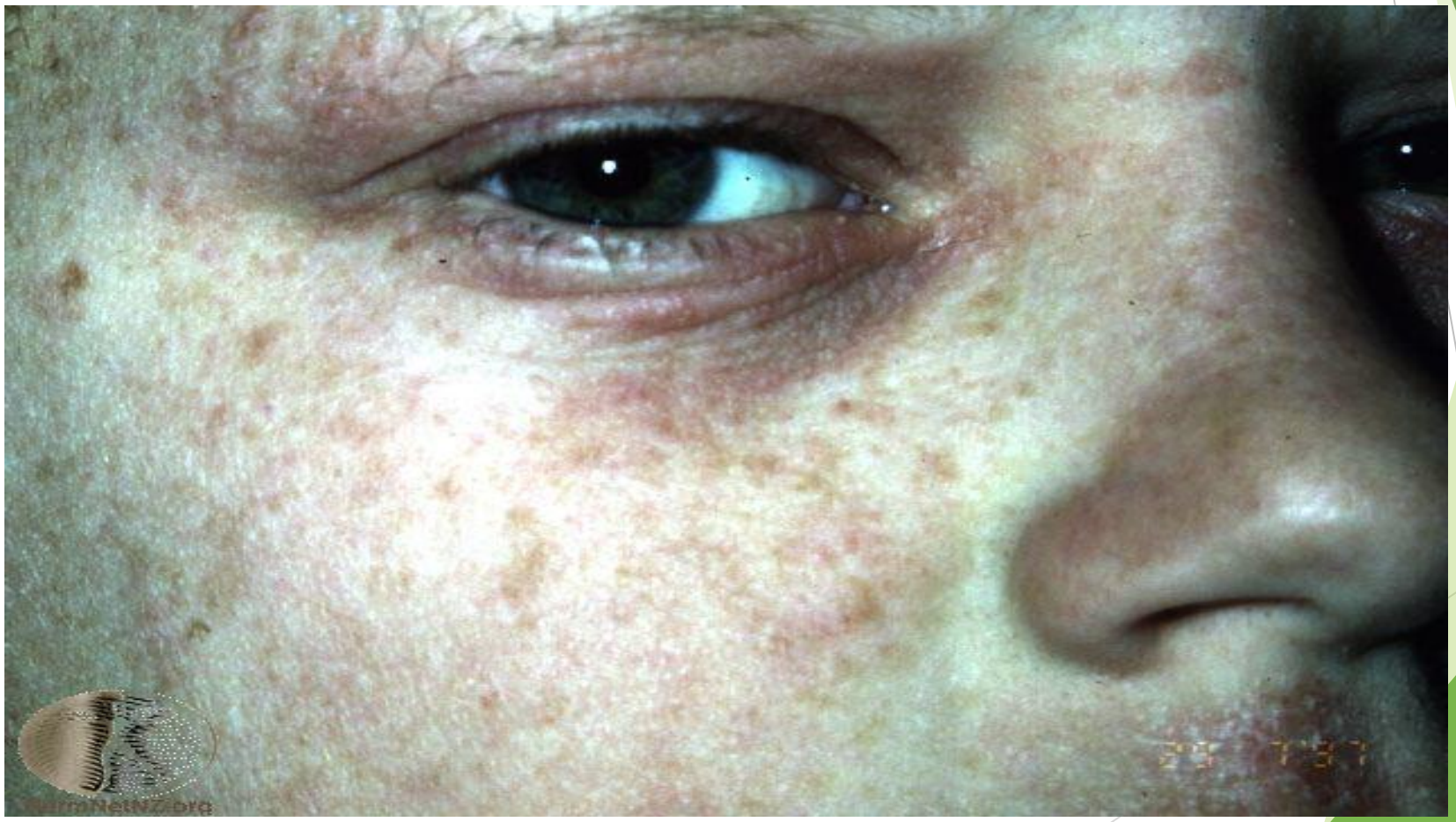

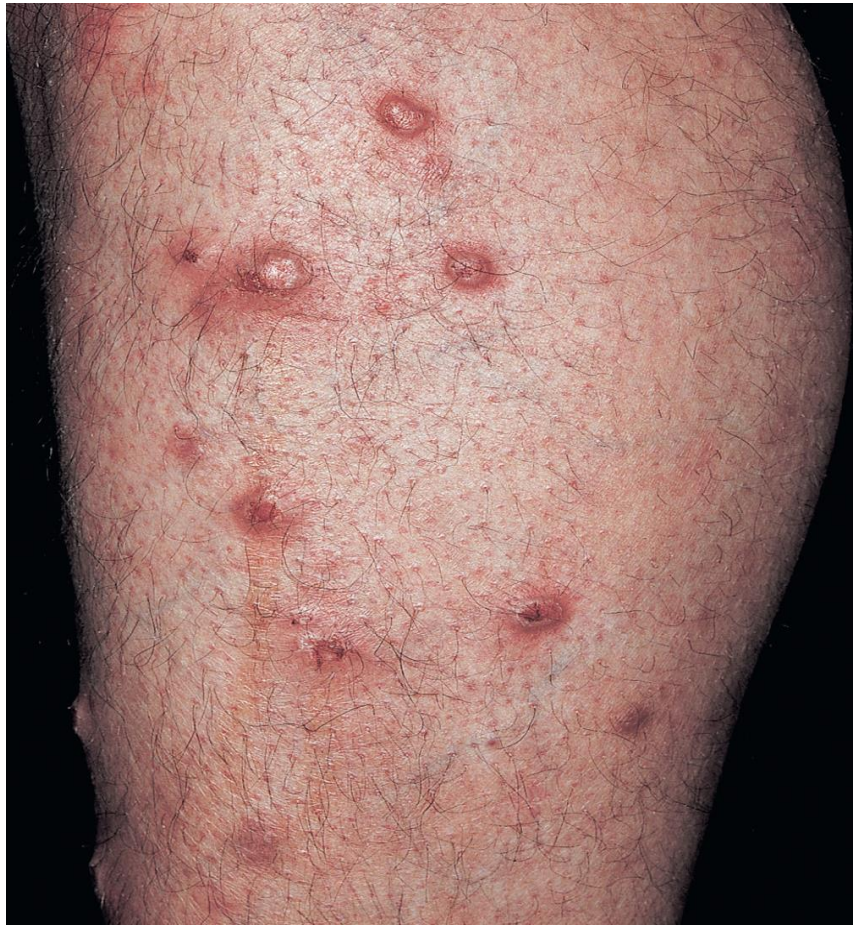

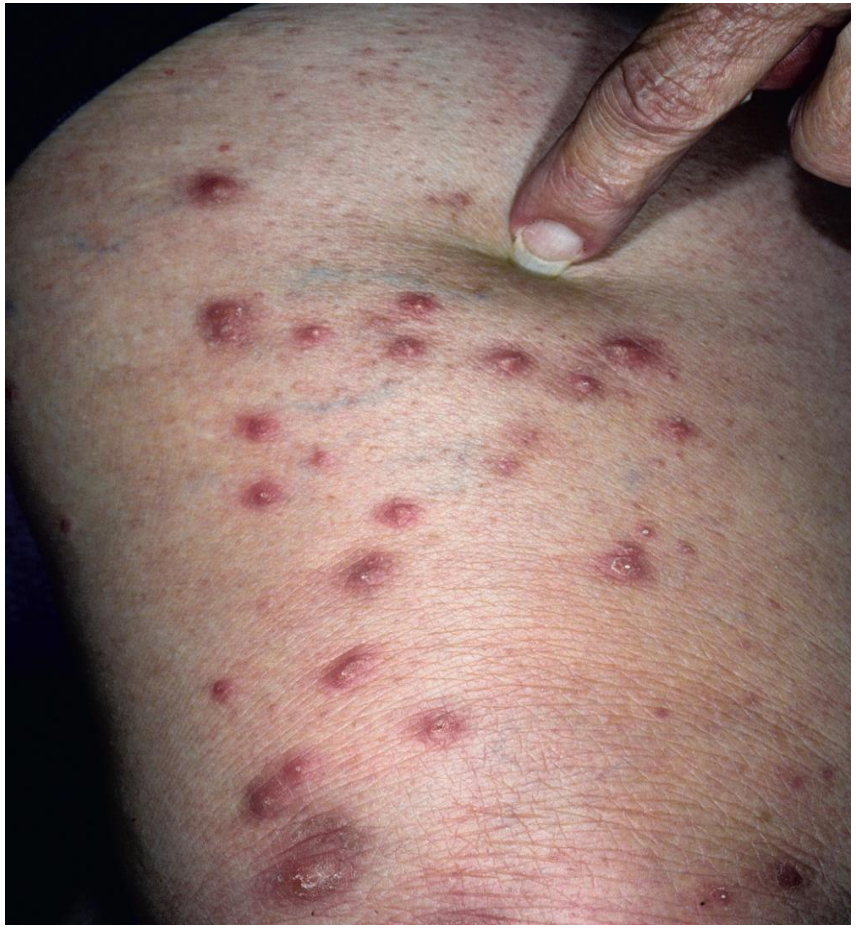

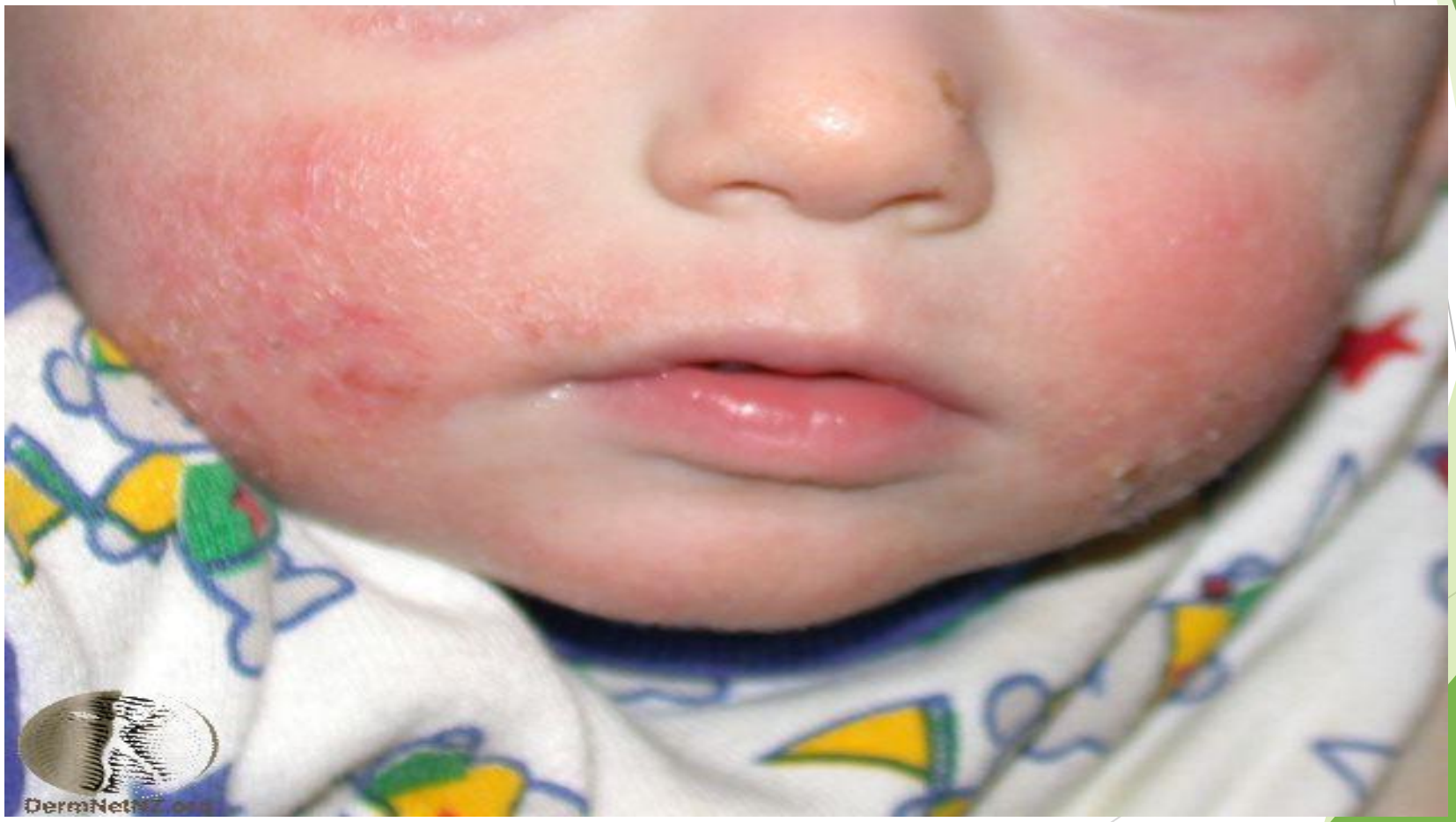

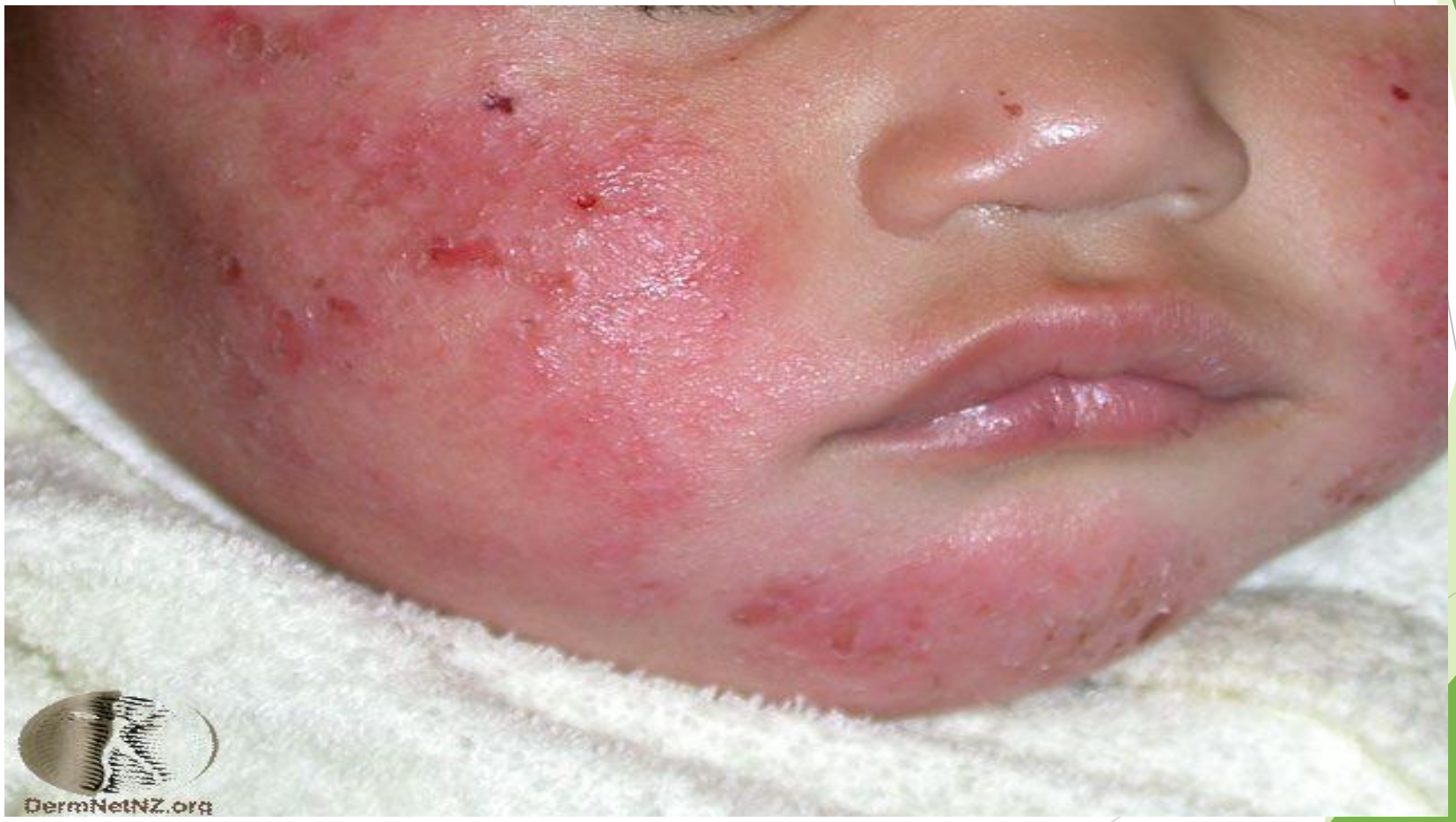
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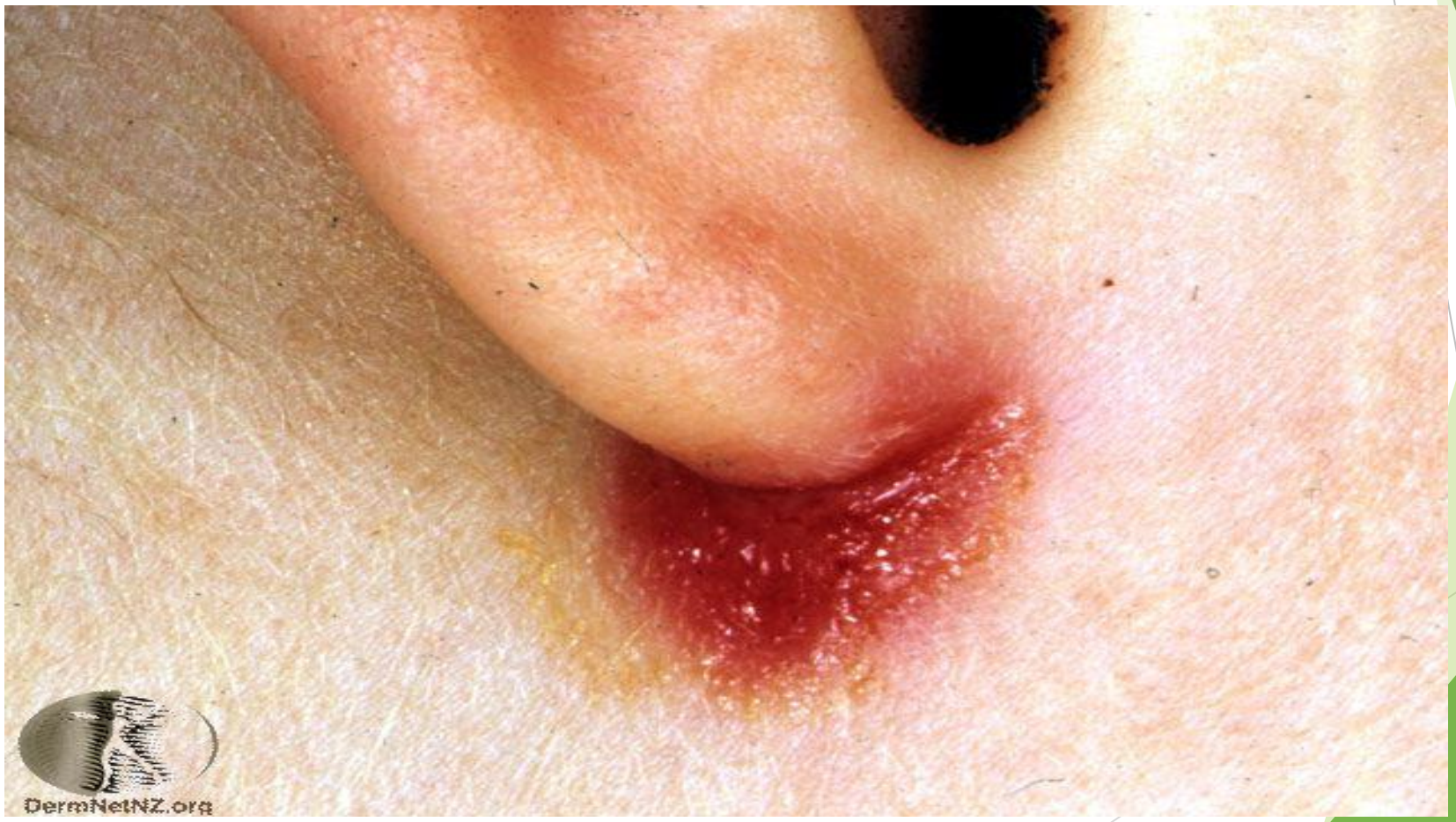
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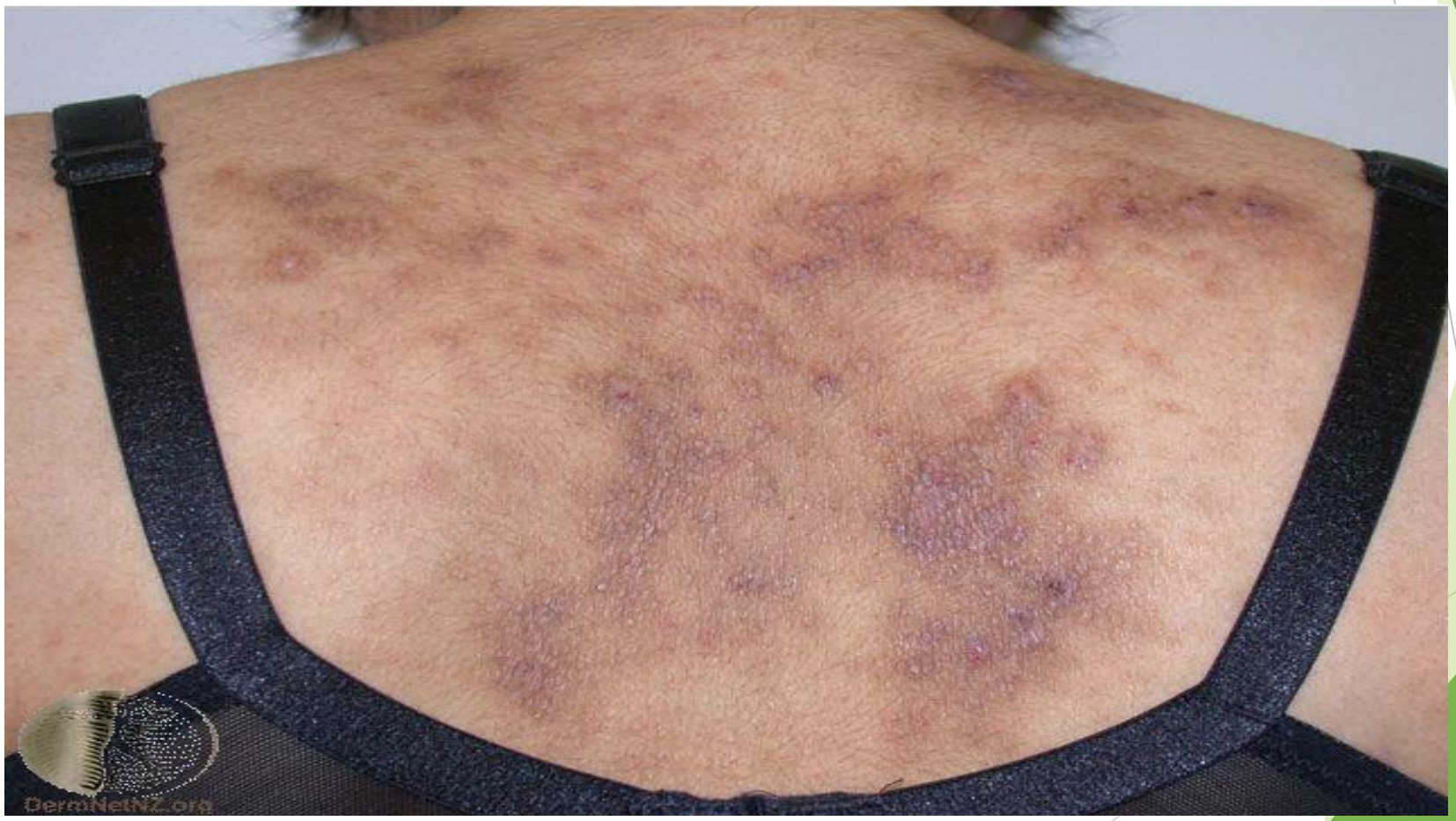
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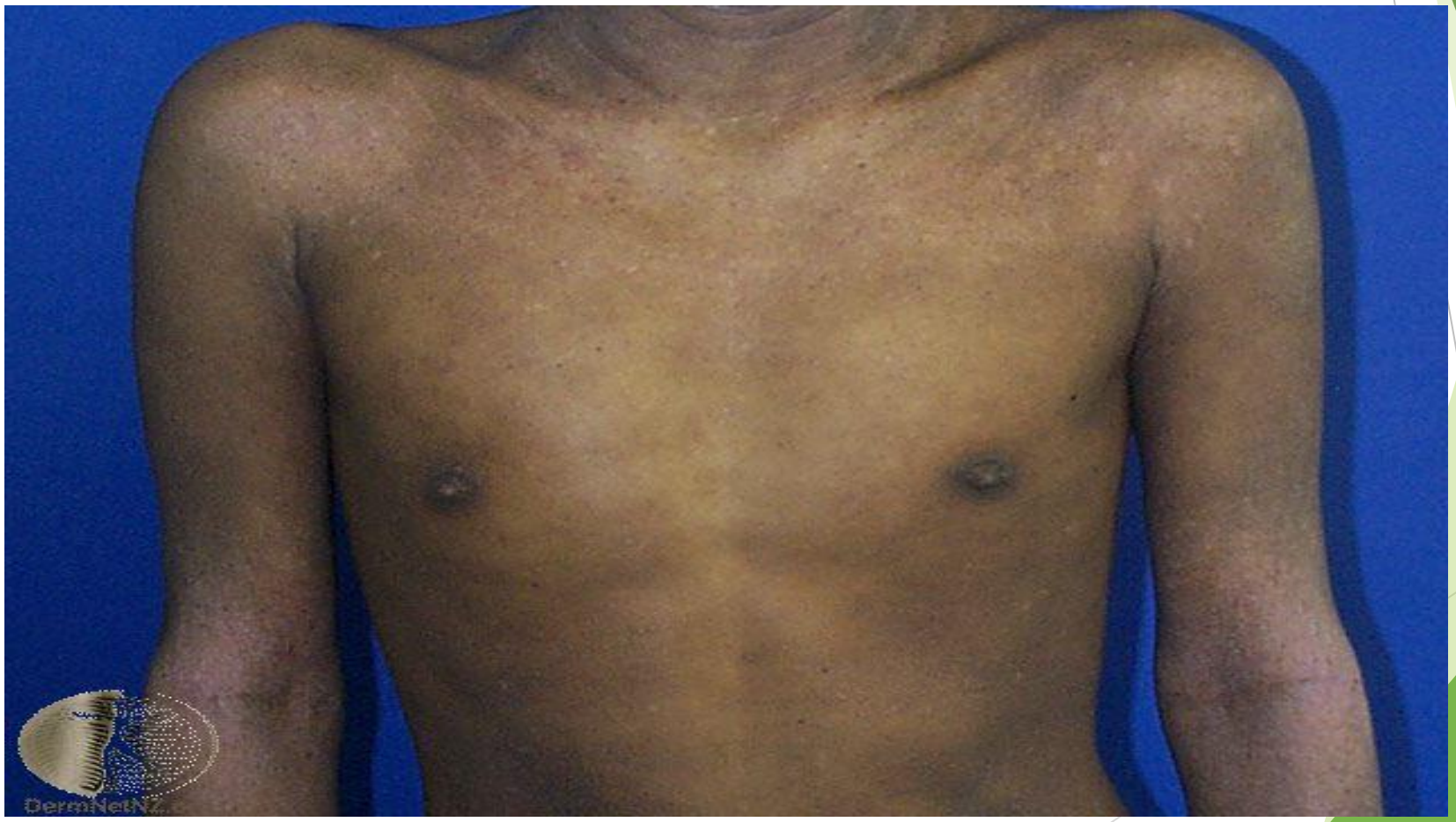
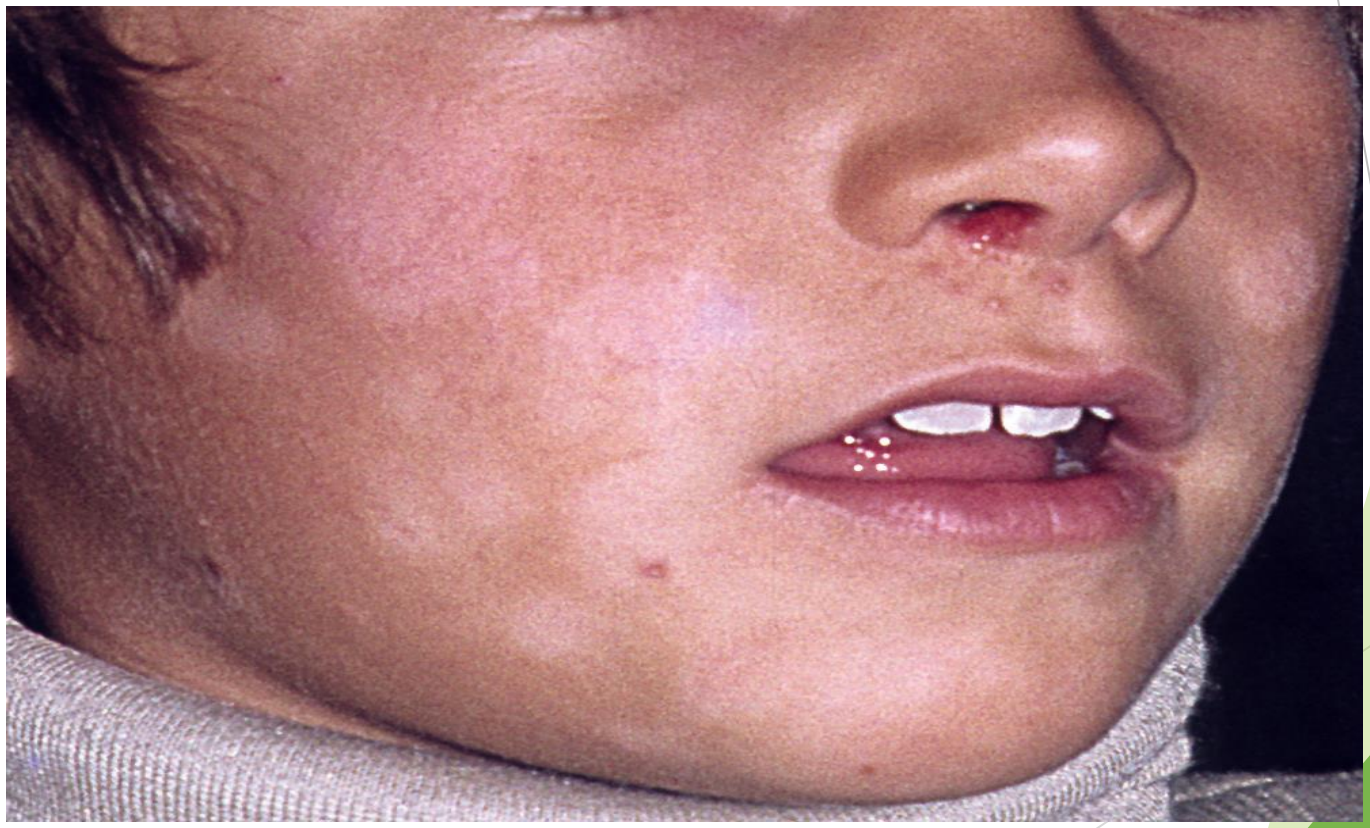
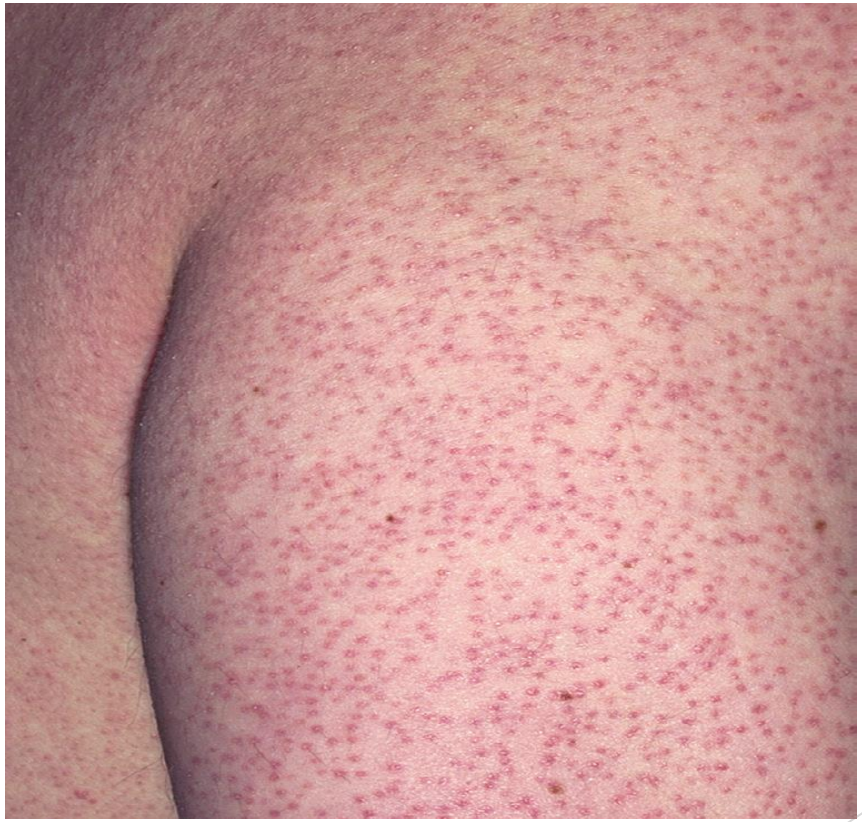
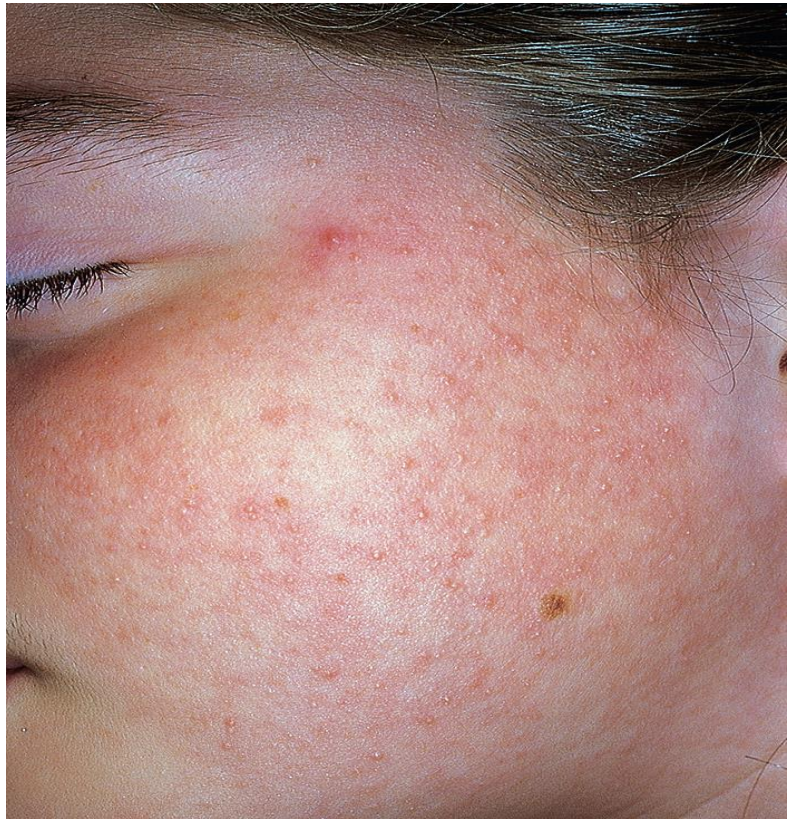
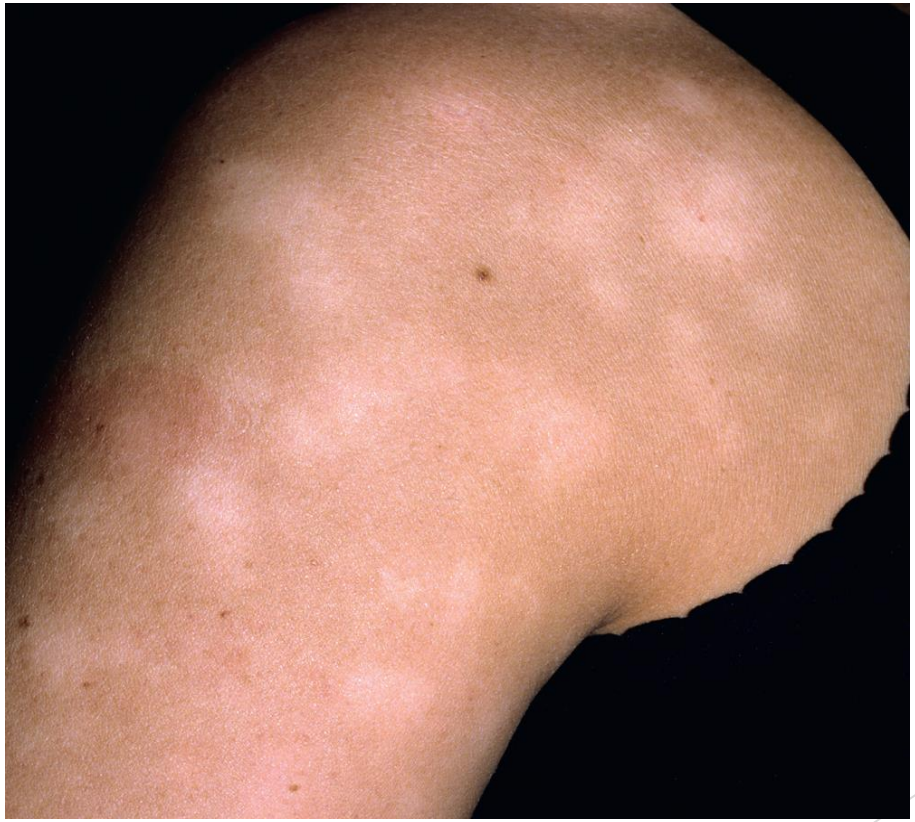










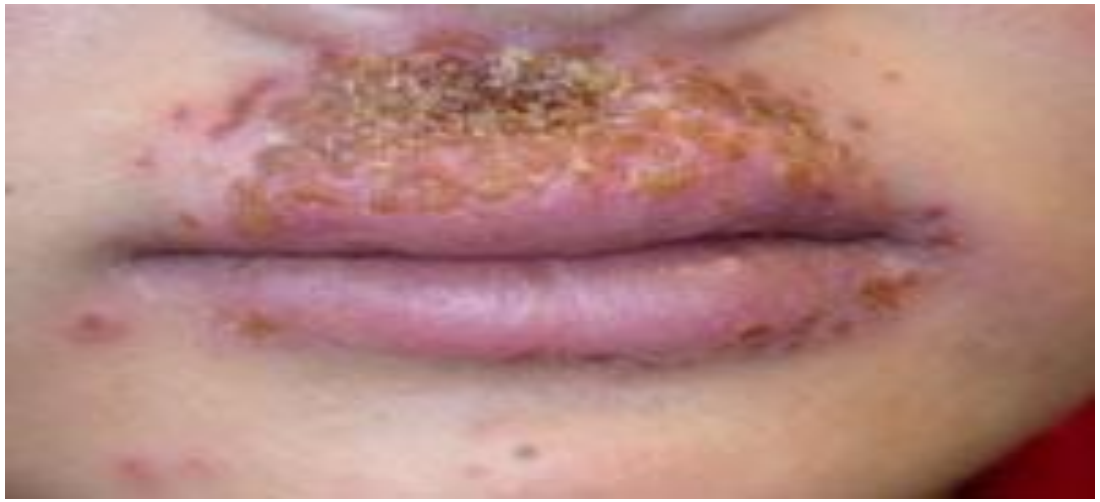
Complications

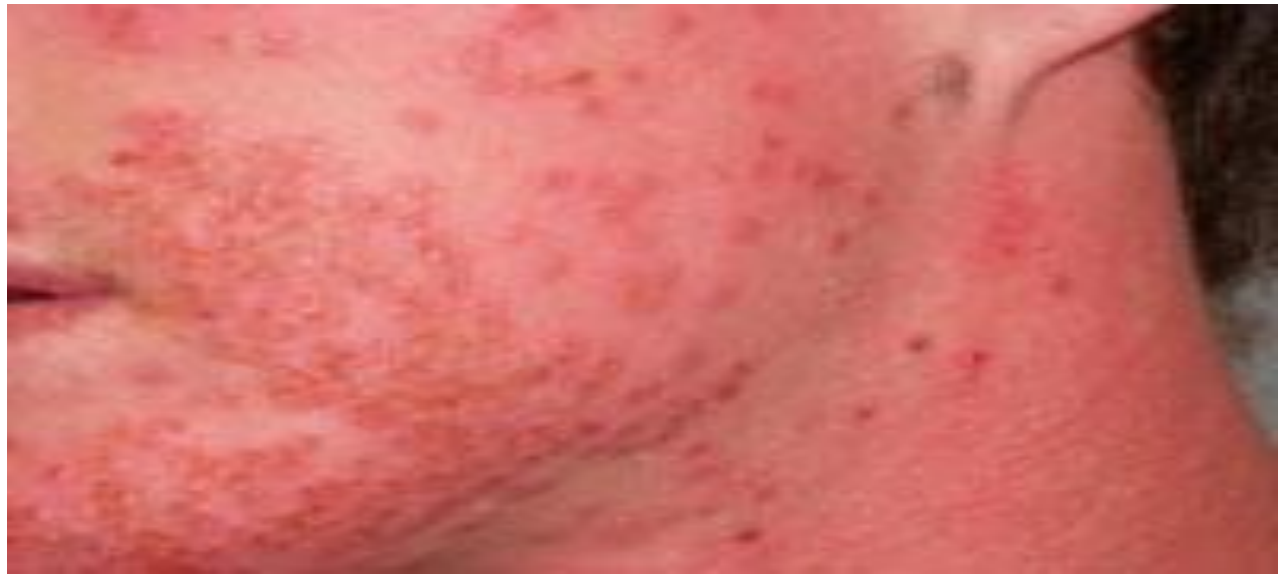
- **OCULAR PROBLEMS** Eyelid dermatitis and chronic blepharitis are commonly associated with AD and may result in visual impairment from corneal scarring.
- Atopic keratoconjunctivitis is usually bilateral and can have disabling symptoms that include itching, burning, tearing, and copious mucoid discharge.
- Keratoconus is a conical deformity of the cornea believed to result from chronic rubbing of the eyes in patients with AD and allergic rhinitis.
- Cataracts were reported in the early literature to occur in up to 21 percent of patients with severe AD.

INFECTIONS

- The most serious viral infection is herpes simplex, which can affect patients of all ages, resulting in Kaposi's varicelliform eruption or eczema herpeticum.
- After an incubation period of 5 to 12 days, multiple, itchy, vesiculopustular lesions erupt in a disseminated pattern; vesicular lesions are umbilicated, tend to crop, and often become hemorrhagic and crusted . Punched out and extremely painful erosions result. These lesions may coalesce to large, denuded and bleeding areas that can extend over the entire body.

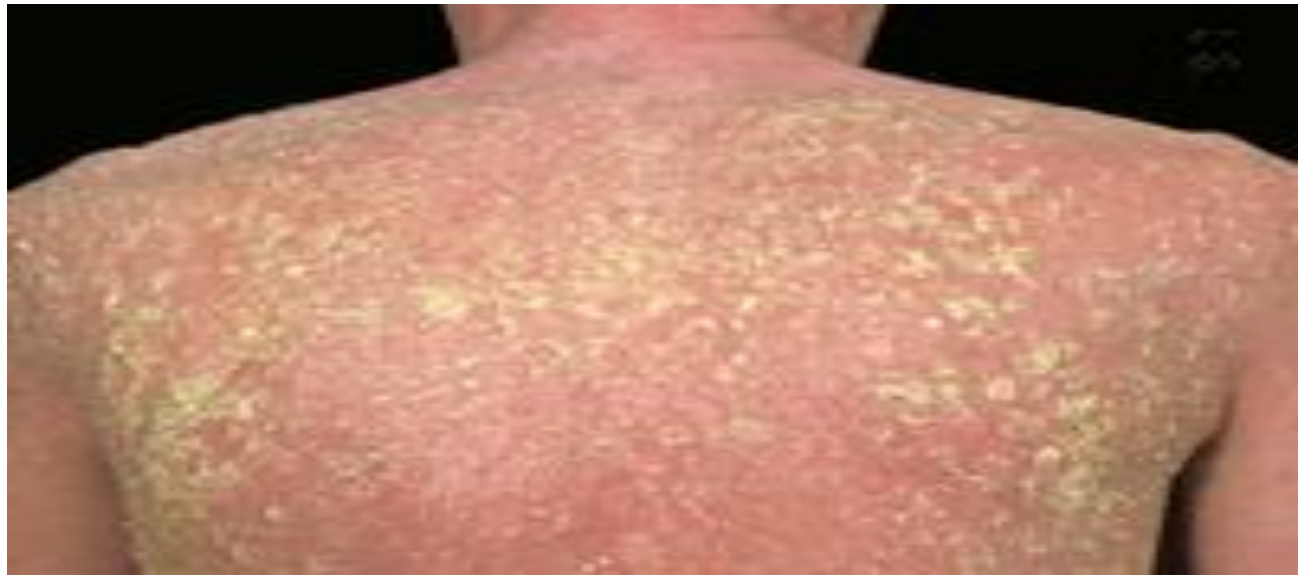
  Superficial fungal infections are also more common in atopic individuals and may contribute to the exacerbation of AD. Patients with AD have an increased prevalence of *Trichophyton rubrum* infections compared to nonatopic controls.
- As discussed earlier, *S. aureus* is found in more than 90 percent of AD skin lesions. Honey-colored crusting, folliculitis, and pyoderma are indicators of secondary bacterial skin infection, usually due to *S. aureus* that requires antibiotic therapy.

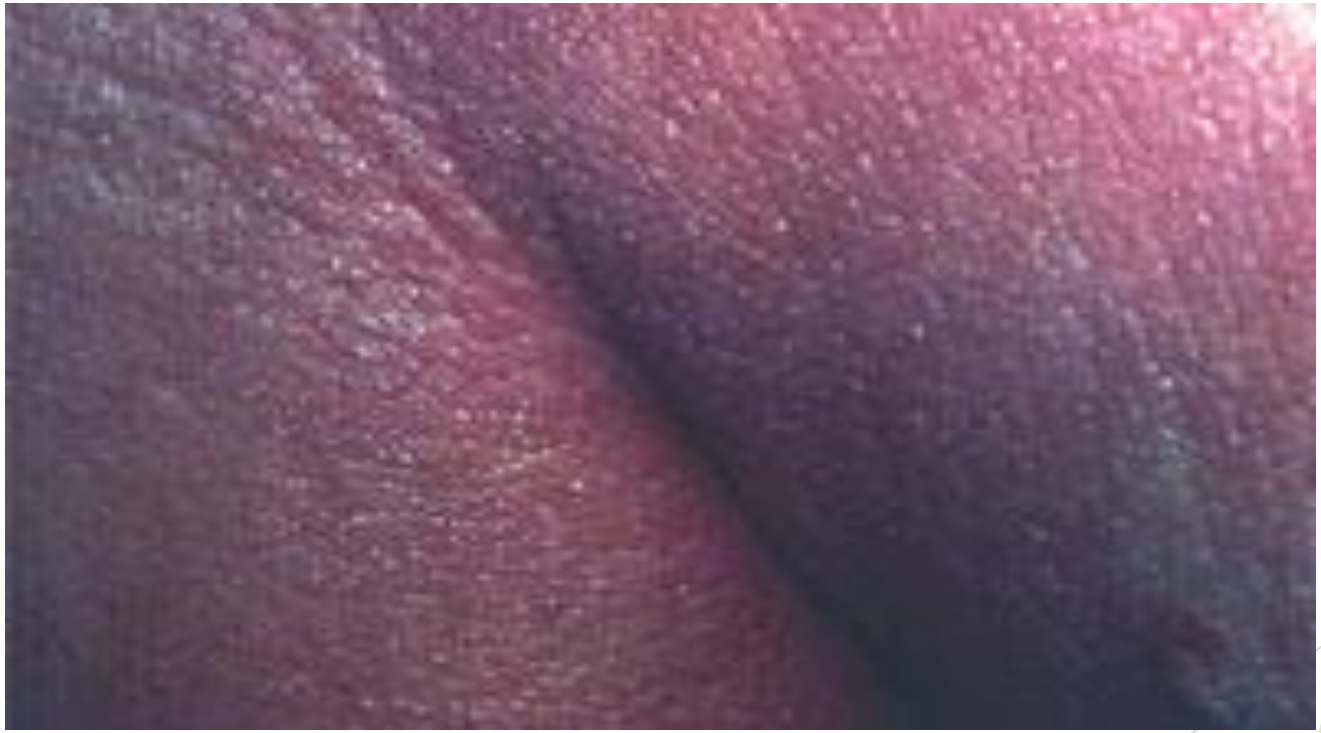


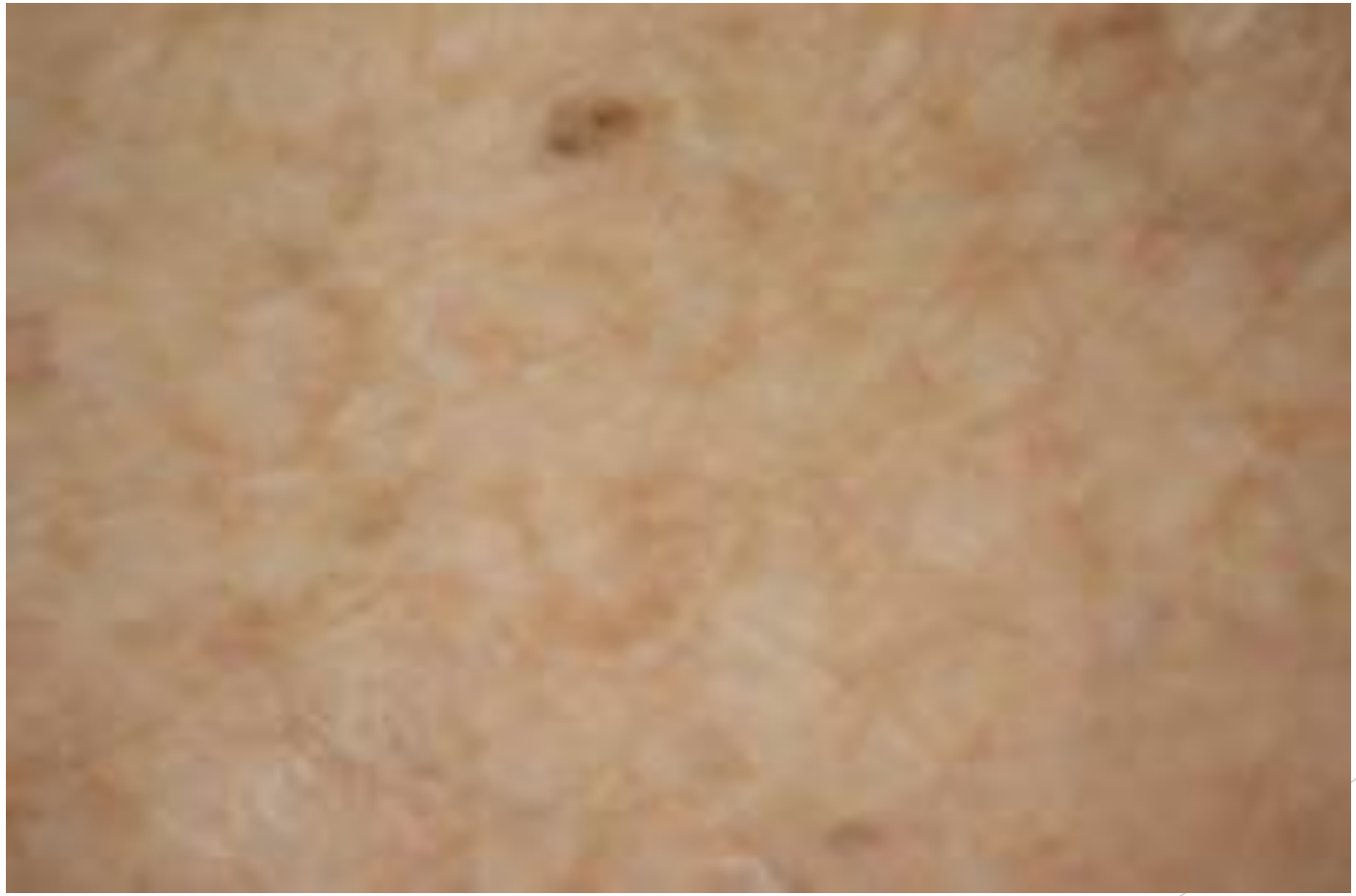


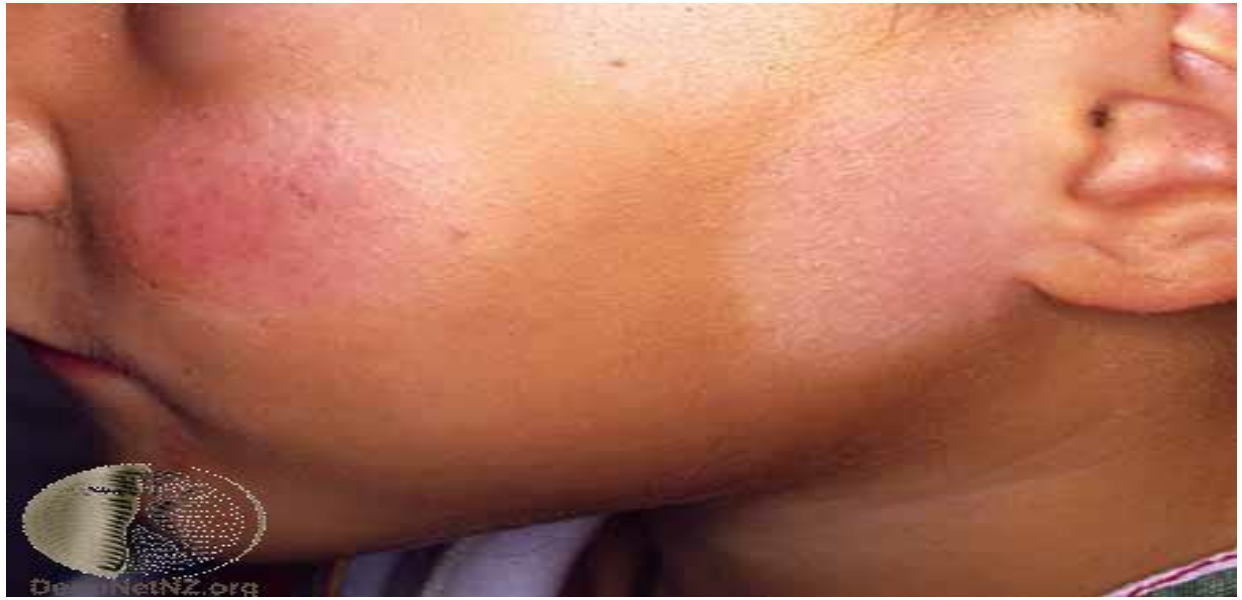
- **HAND DERMATITIS** Patients with AD often develop a nonspecific, irritant hand dermatitis.
- It is frequently aggravated by repeated wetting and by washing of the hands with harsh soaps, detergents, and disinfectants.
- Atopic individuals with occupations involving wet work are prone to develop an intractable hand dermatitis in the occupational setting. This is a common cause of occupational disability.

- **EXFOLIATIVE DERMATITIS** Patients with extensive skin involvement may develop exfoliative dermatitis.
- This is associated with generalized redness, scaling, weeping, crusting, systemic toxicity, lymphadenopathy, and fever .
- Although this complication is rare, it is potentially life-threatening.
- It is usually due to superinfection, for example, with toxin-producing *S. aureus* or herpes simplex infection, continued irritation of the skin, or inappropriate therapy.
- In some cases, the withdrawal of systemic glucocorticoids used to control severe AD may be a precipitating factor for exfoliative erythroderma.

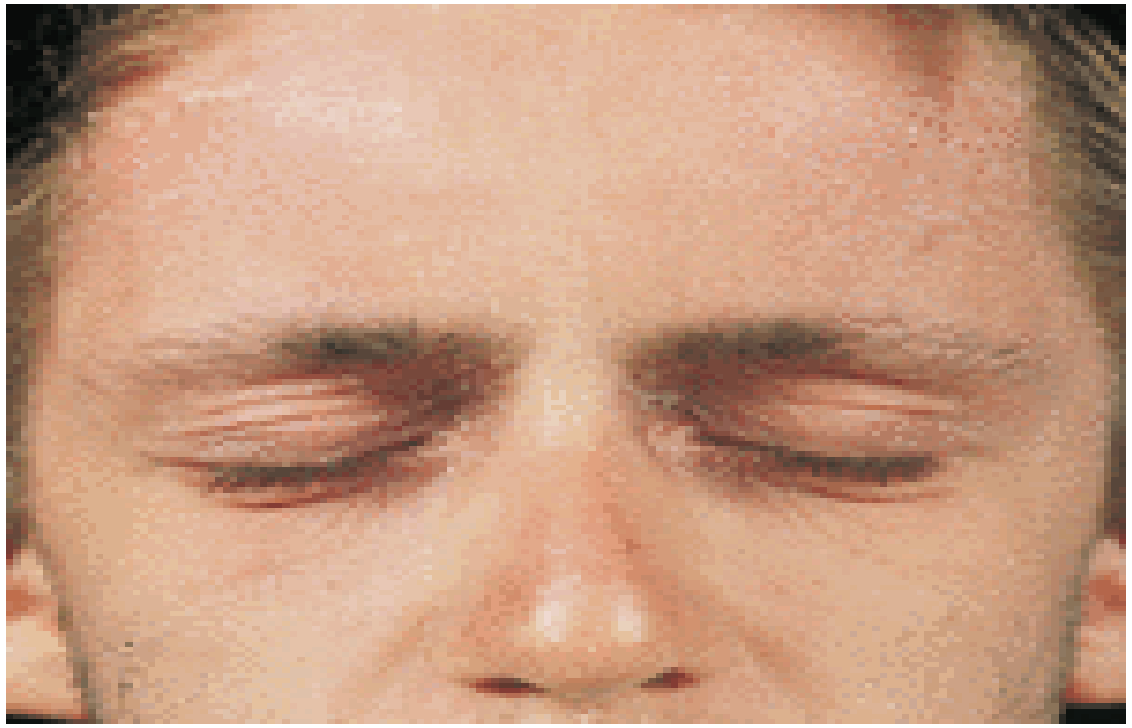








Infiltrated, erythematous facial lateral thinning of eyebrows and infraocular (Morgan's) fold

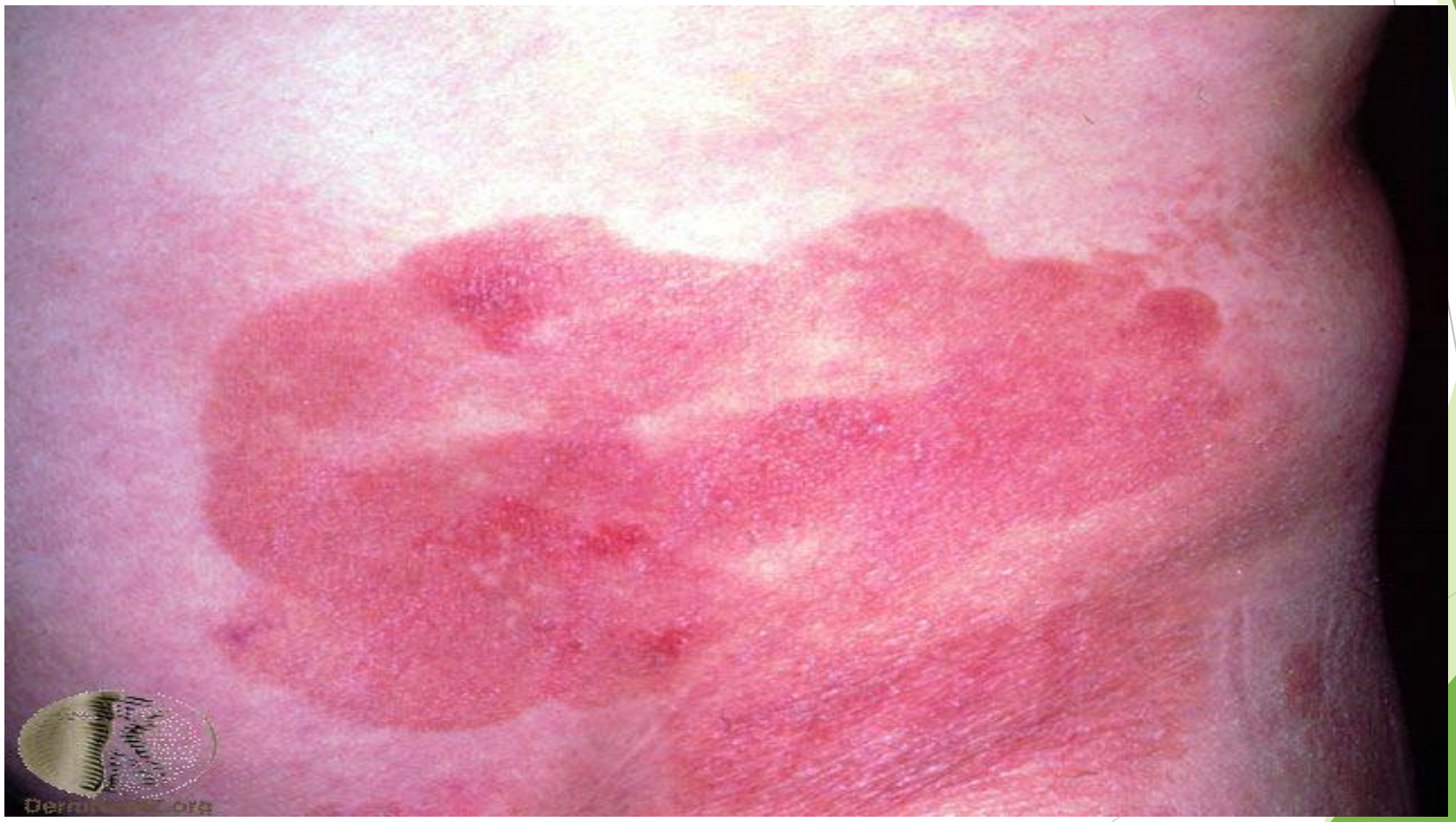


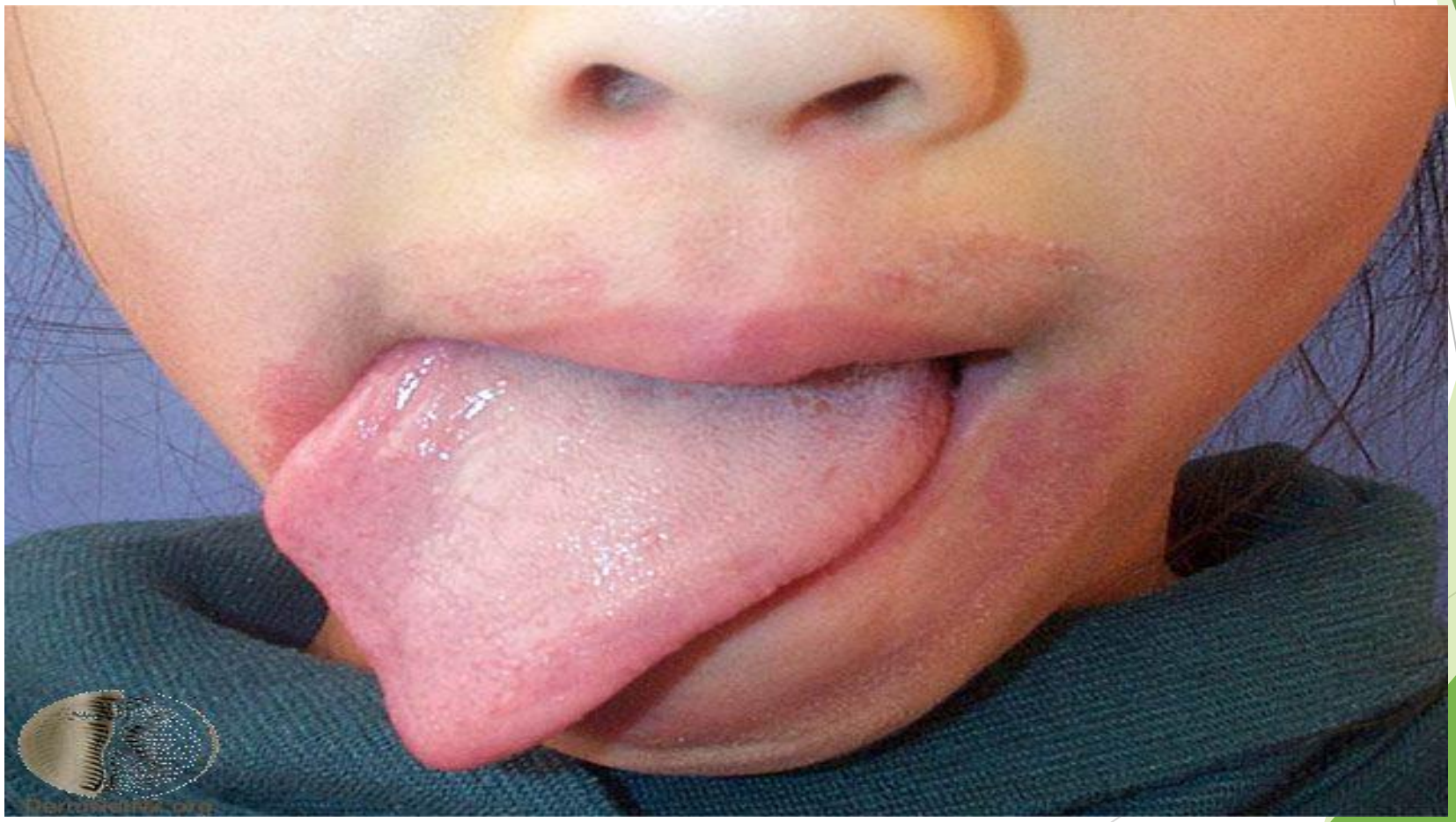
DIFFERENTIAL DIAGNOSIS

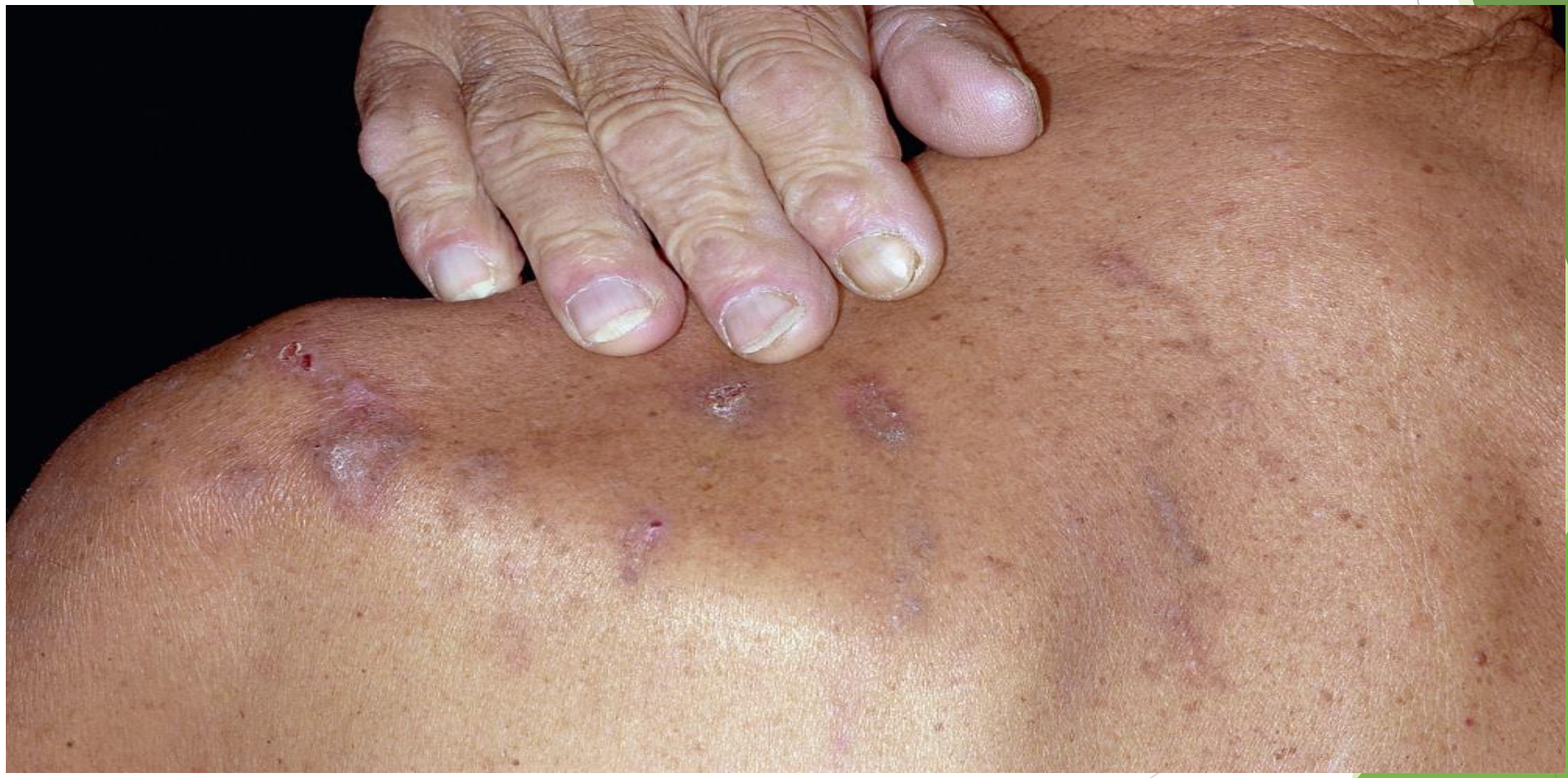
- Of the major features, pruritus and chronic or remitting eczematous dermatitis with typical morphology and distribution are essential for diagnosis.
- lists a number of inflammatory skin diseases, immunodeficiencies, skin malignancies, genetic disorders, infectious diseases, and infestations that share symptoms and signs with AD.

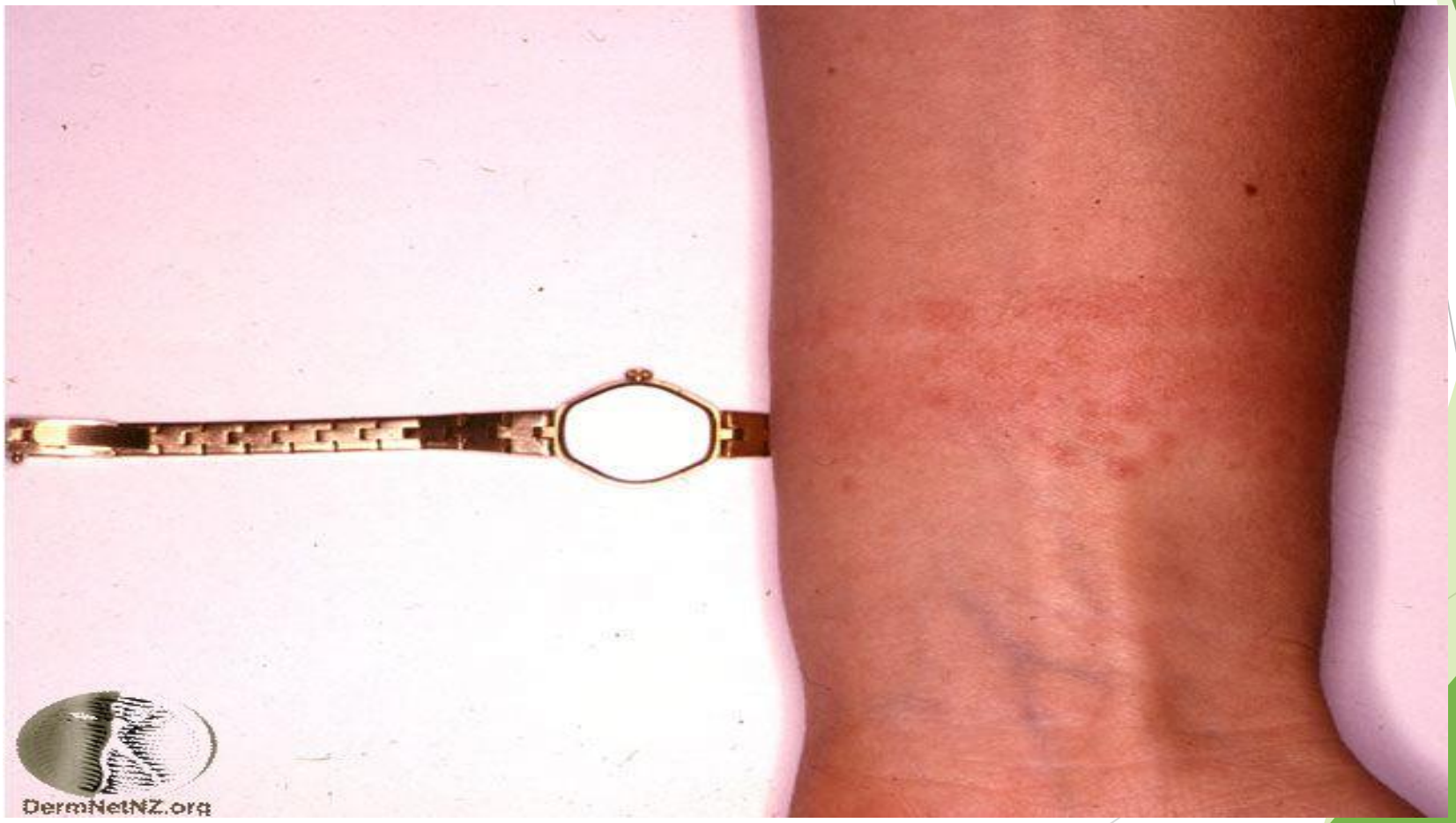
- Infants presenting in the first year of life with failure to thrive, diarrhea, a generalized scaling erythematous rash, and recurrent cutaneous and/or systemic infections should be evaluated for severe combined immunodeficiency syndrome .
- Wiskott-Aldrich syndrome is an X-linked recessive disorder characterized by cutaneous findings almost indistinguishable from AD. It is associated with thrombocytopenia, variable abnormalities in humoral and cellular immunity, and recurrent severe bacterial infections.
- Hyperimmunoglobulin-E syndrome is characterized by markedly elevated serum IgE levels, defective T cell function, recurrent deep-seated bacterial infections, including cutaneous abscesses due to *S. aureus* and/or pruritic skin disease due to *S. aureus* pustulosis, or by recalcitrant dermatophytosis.

- It is important to recognize that an adult who presents with an eczematous dermatitis with no history of childhood eczema, respiratory allergy, or atopic family history may have allergic contact dermatitis .
- A contact allergen should be considered in any patient whose AD does not respond to appropriate therapy.
- Of note, topical glucocorticoid contact allergy has been reported increasingly in patients with chronic dermatitis on topical corticosteroid therapy.
- Cutaneous T cell lymphoma must be ruled out in any adult presenting with chronic dermatitis poorly responsive to topical glucocorticoid therapy.





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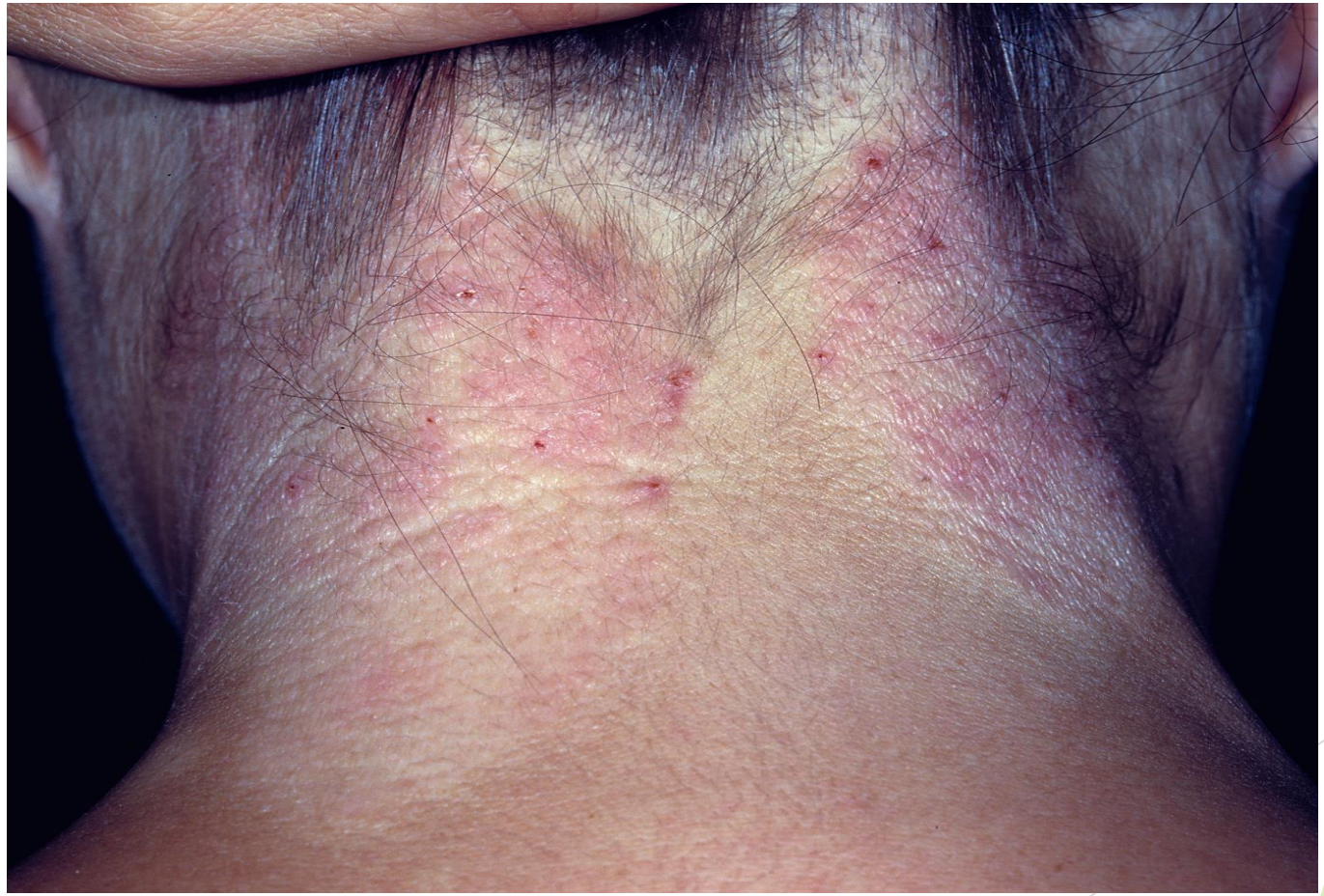
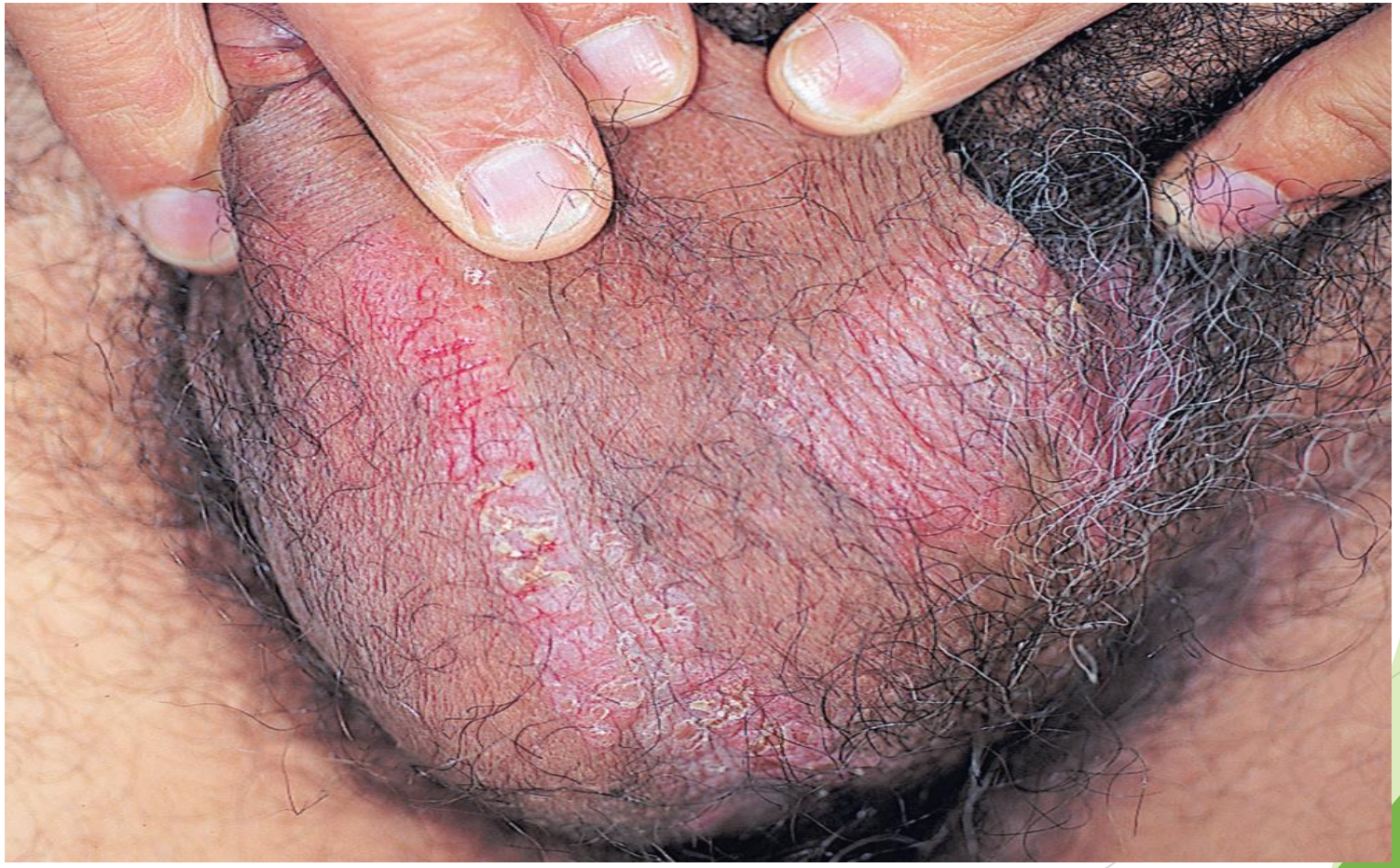
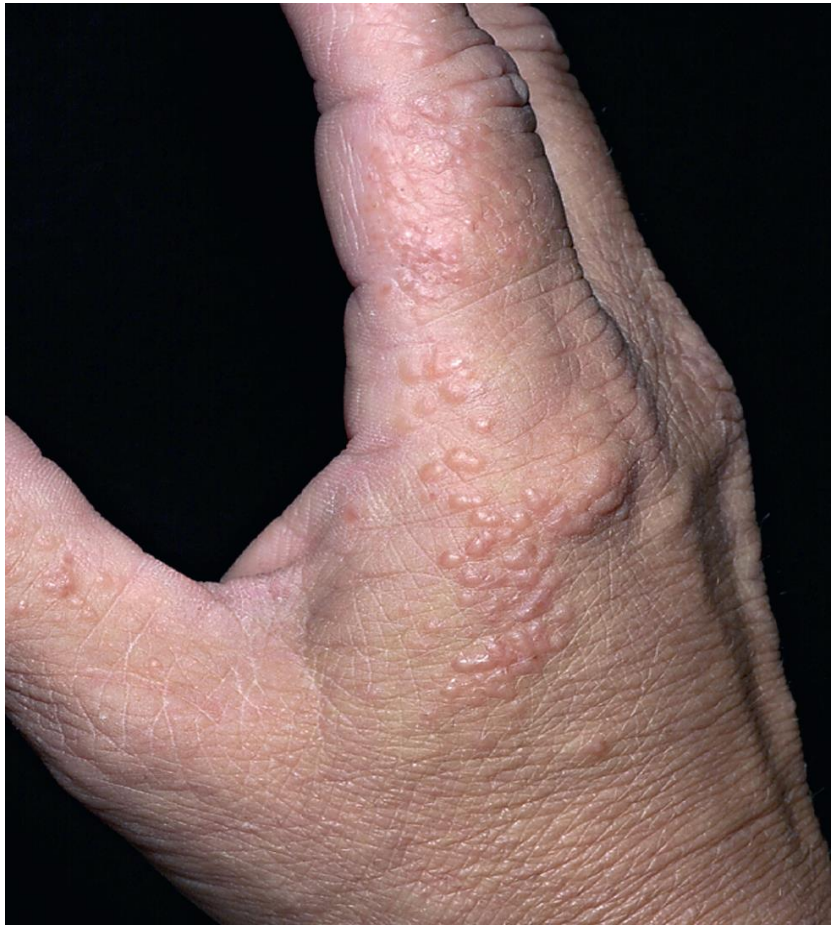
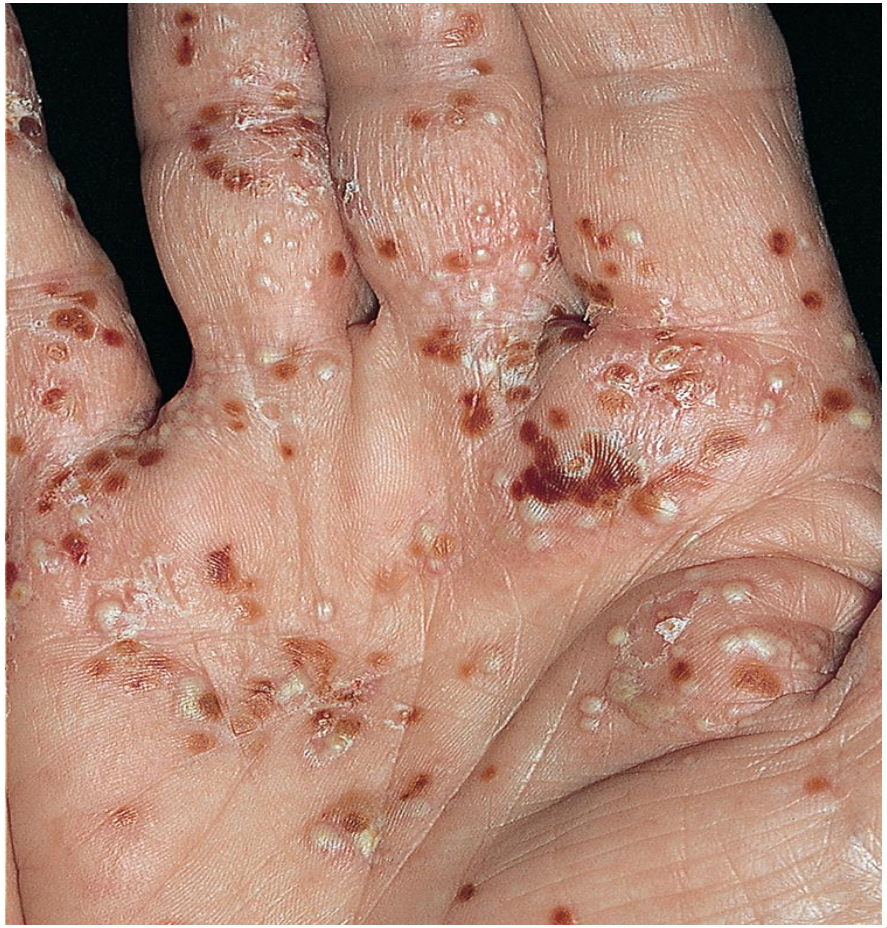


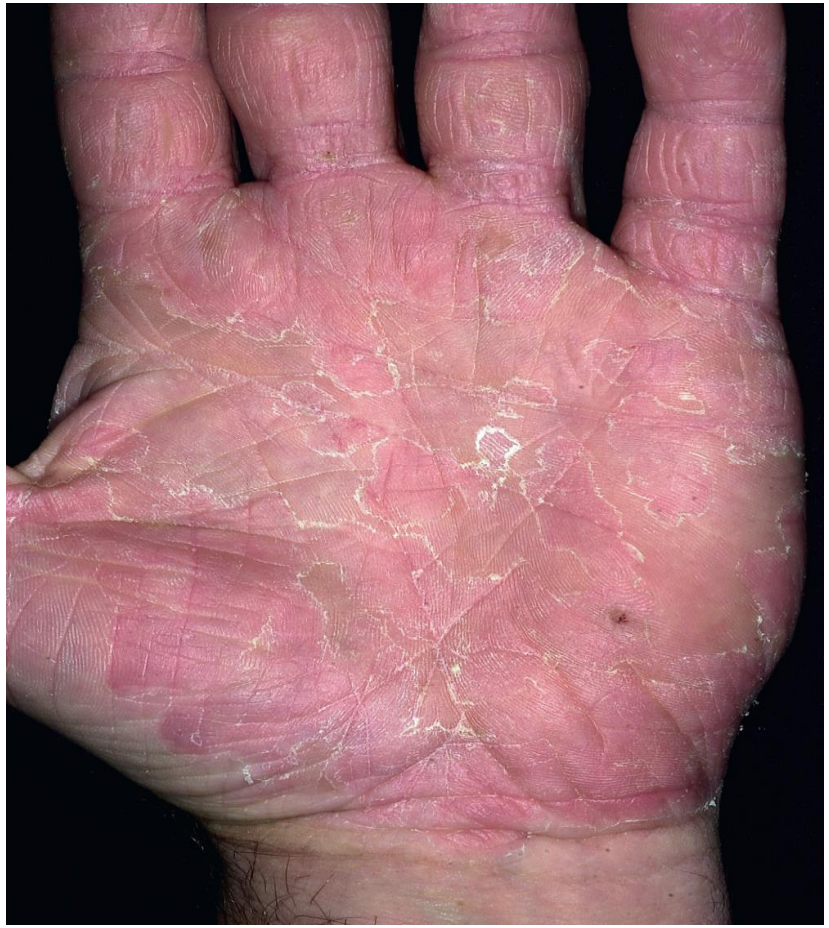


- Eczematous dermatitis has been also reported with HIV as well as with a variety of infestations such as scabies.
- Other conditions that can be confused with AD include psoriasis, ichthyoses, and seborrheic dermatitis.

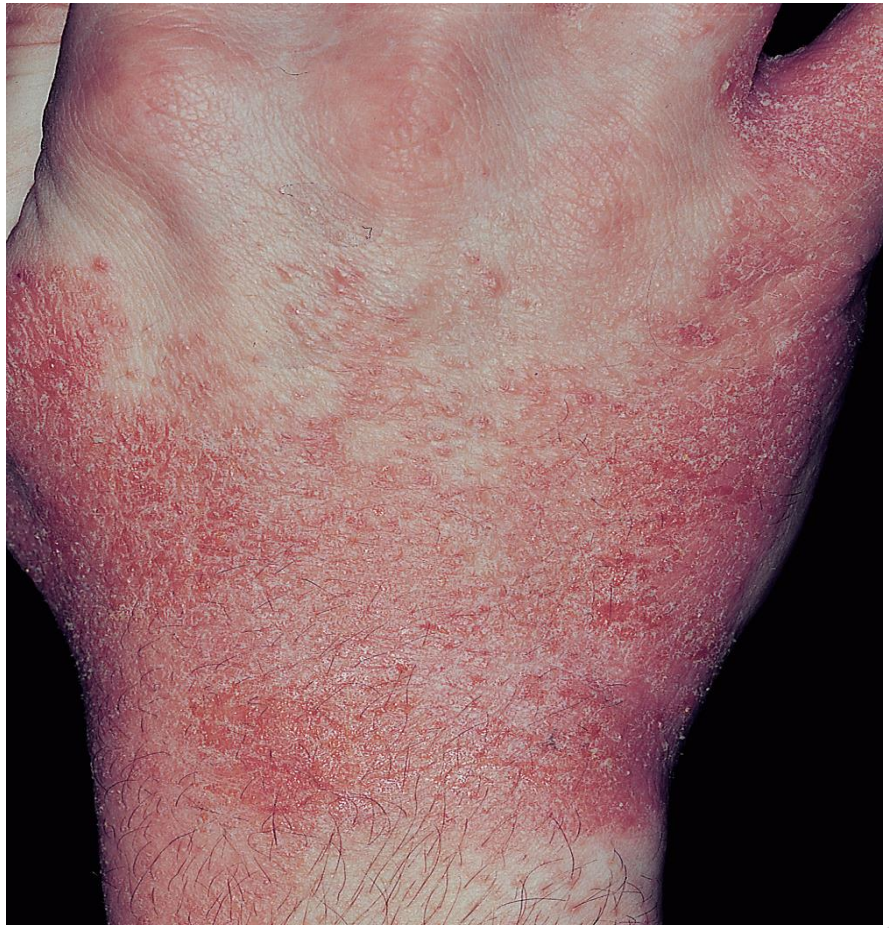




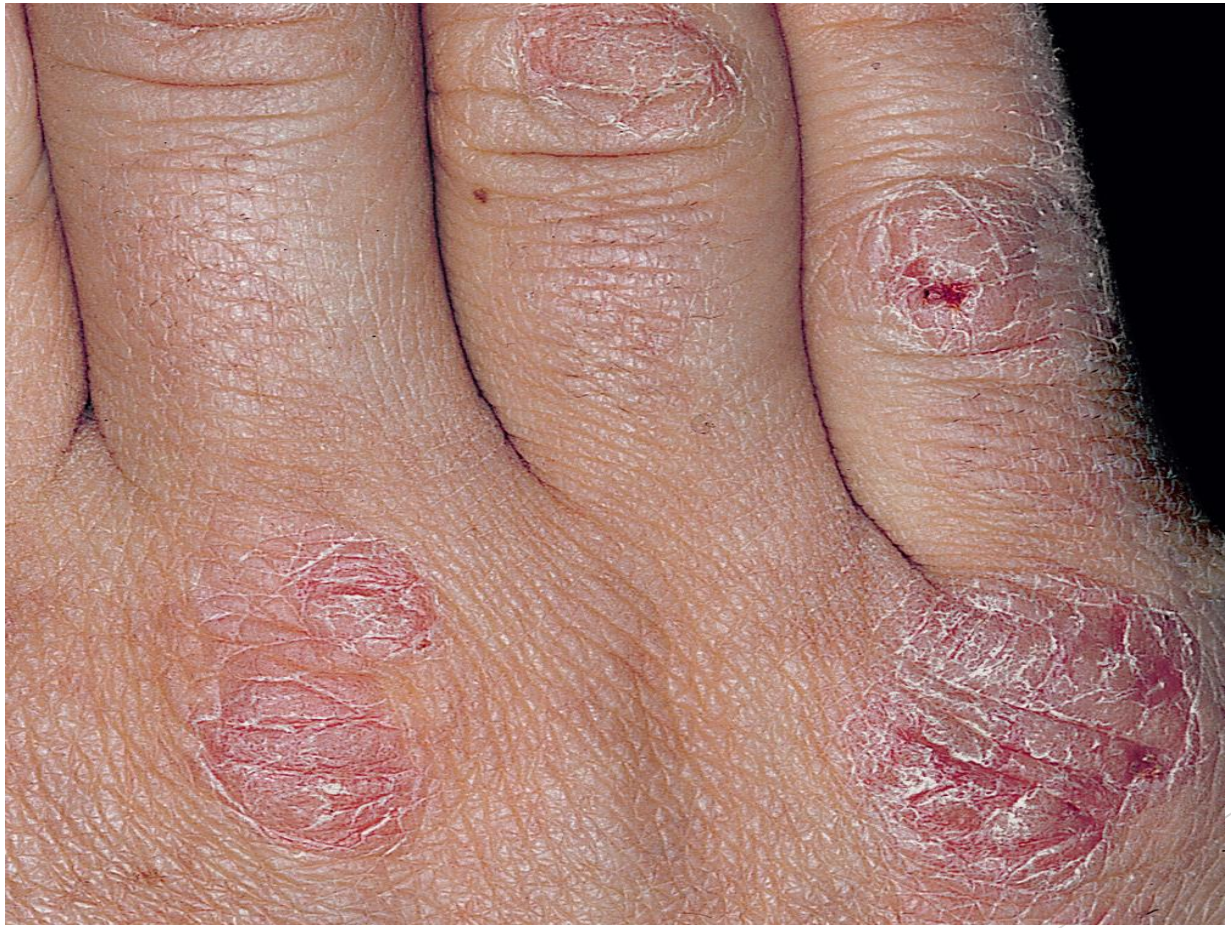
Keratolysis exfoliativa.

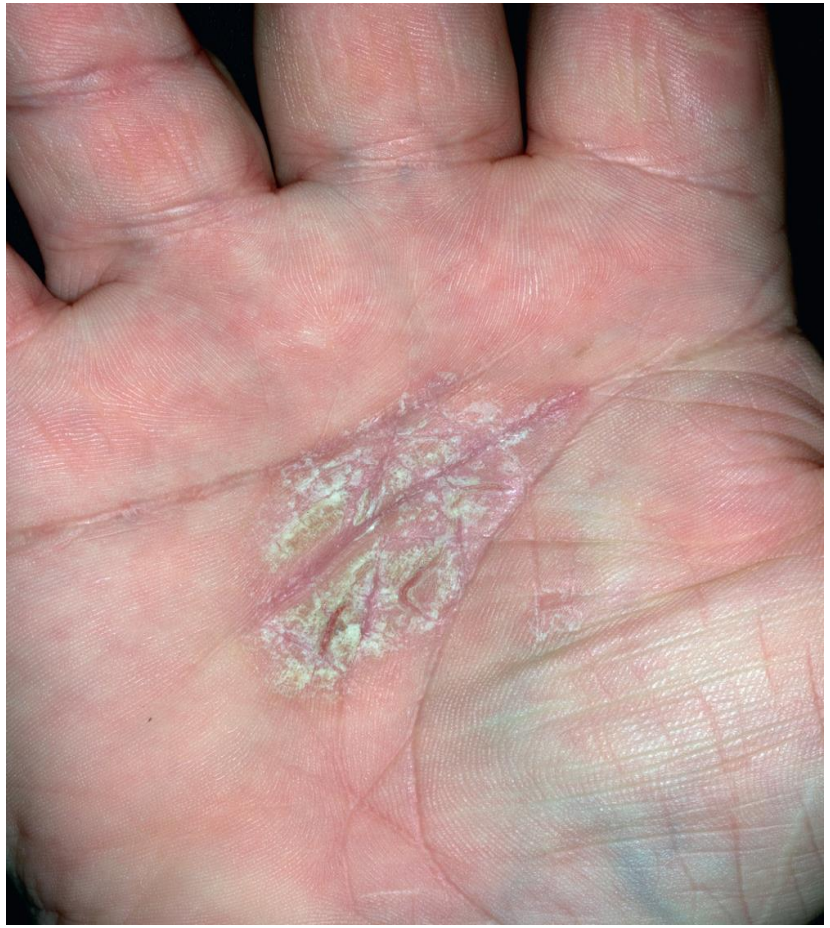


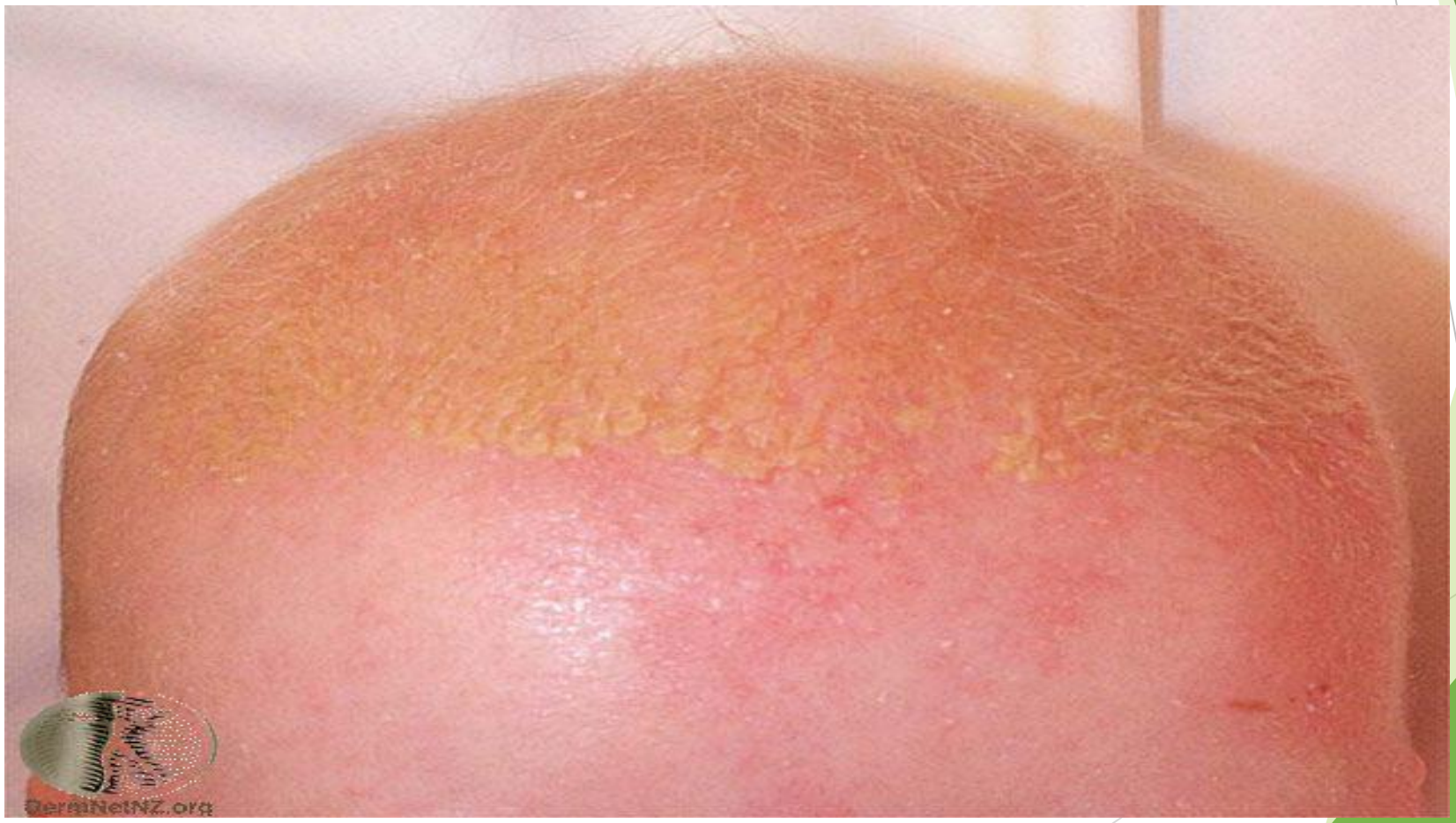
Irritant hand dermatitis



Nummular eczema.



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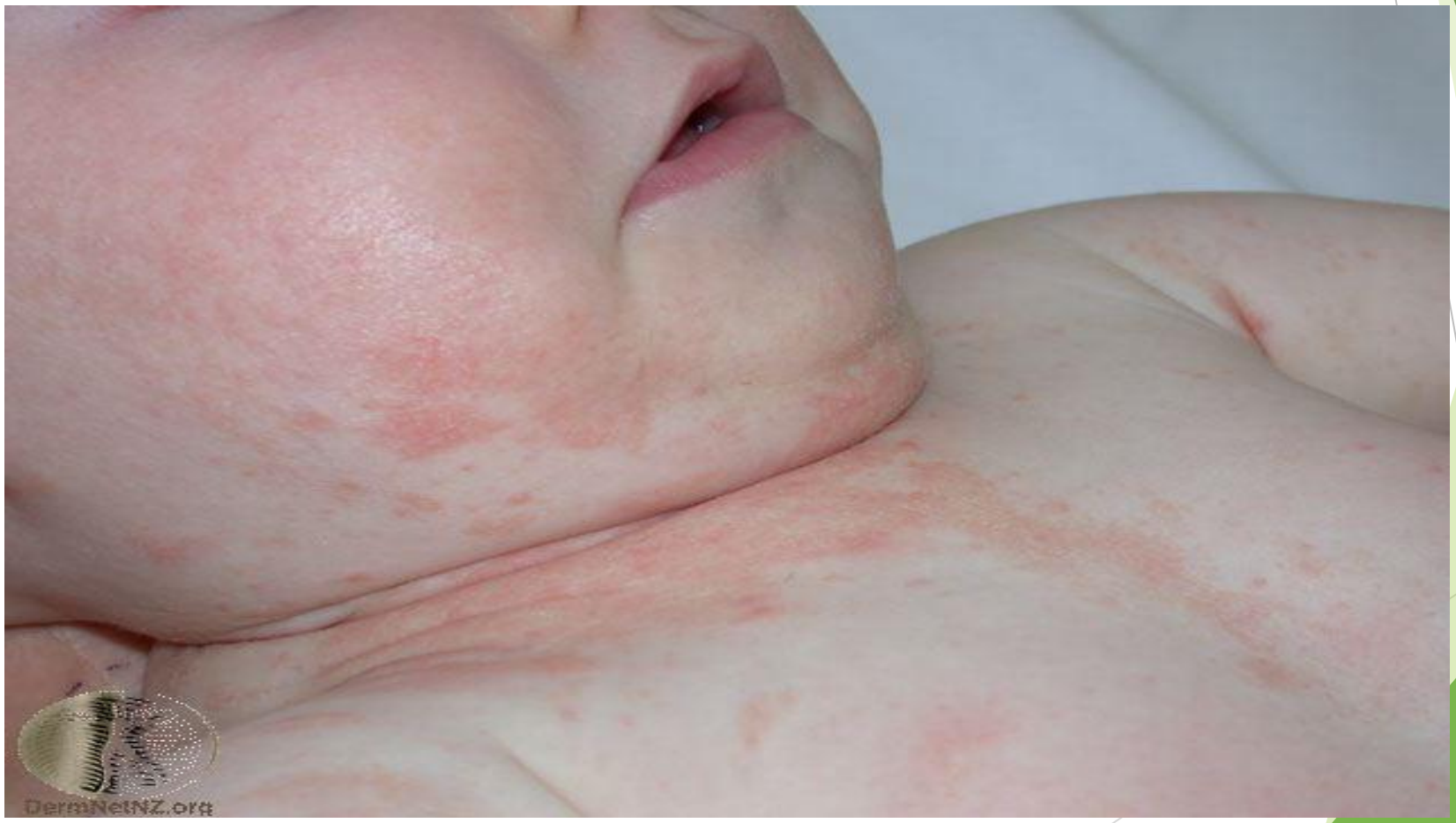
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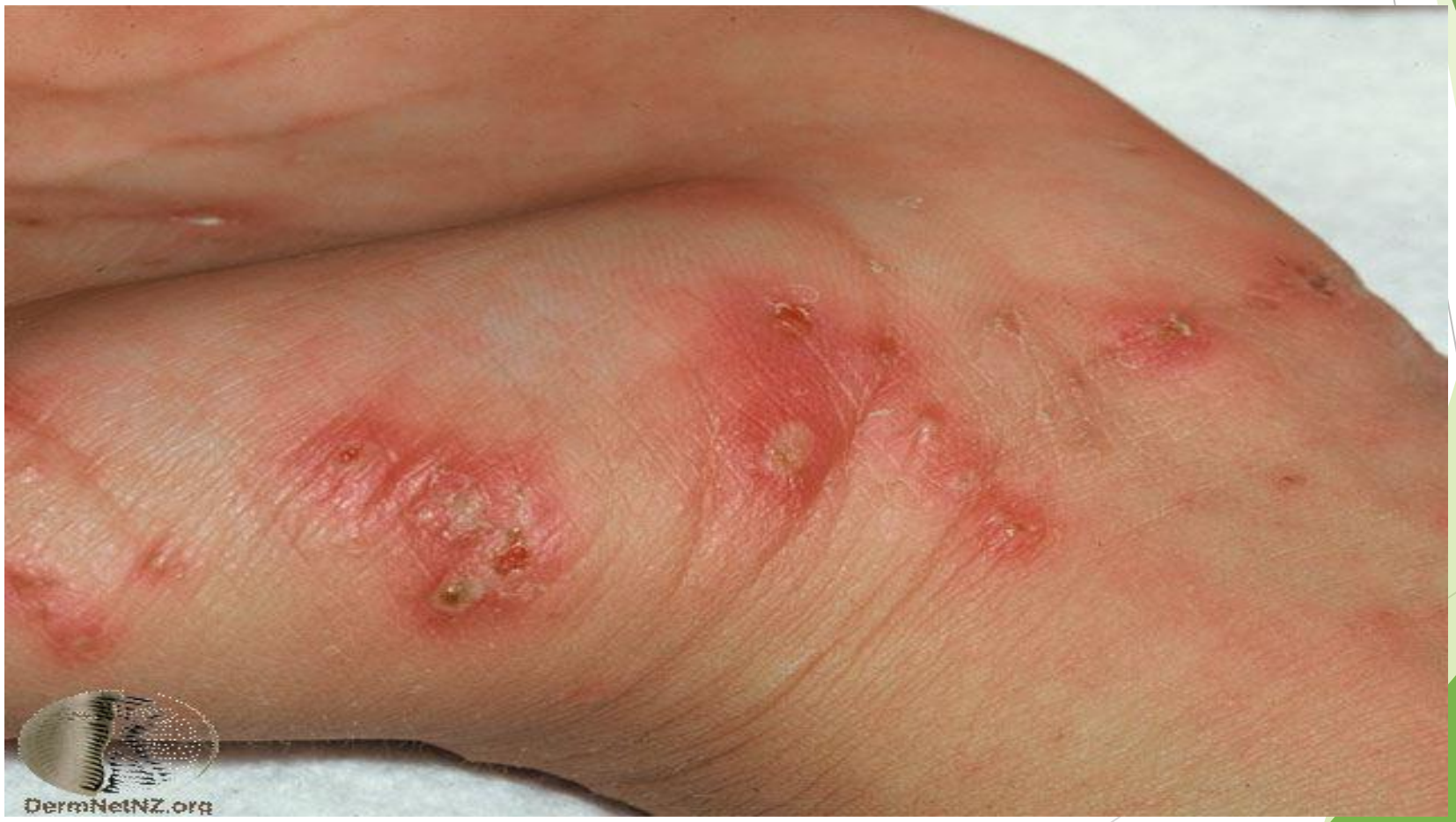
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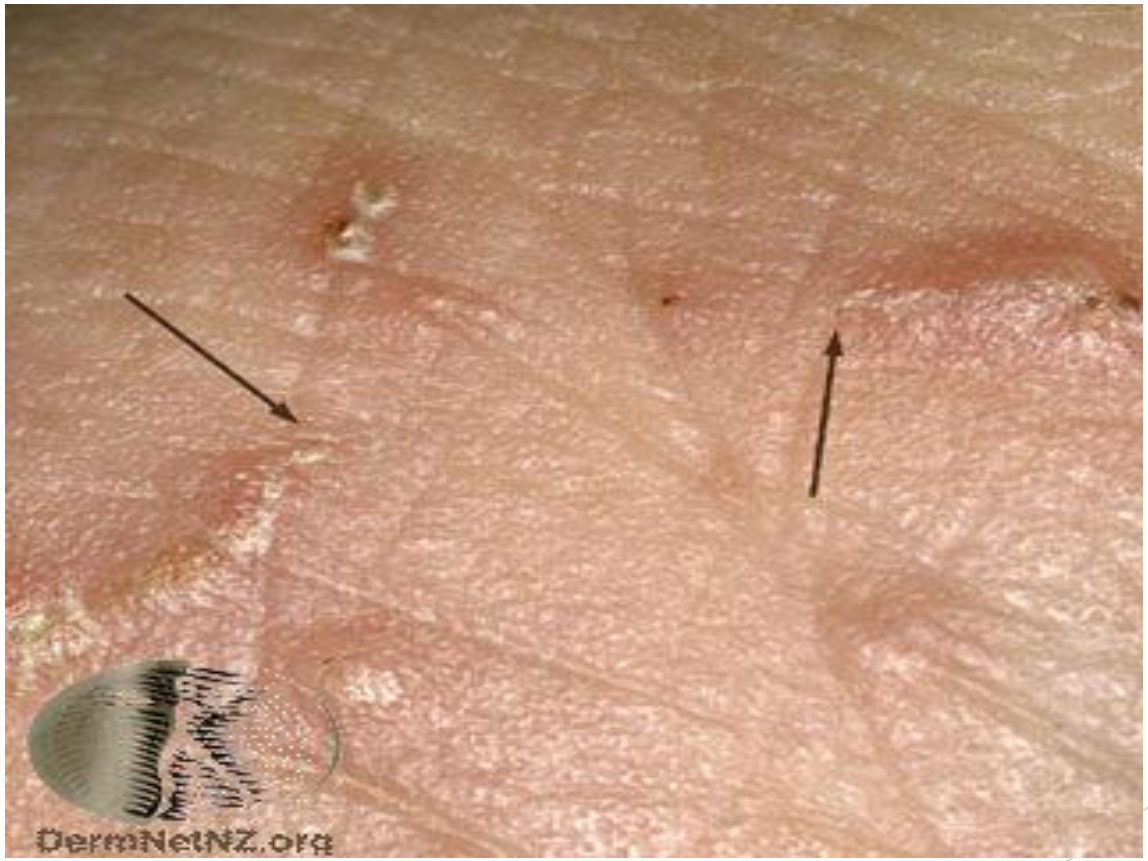
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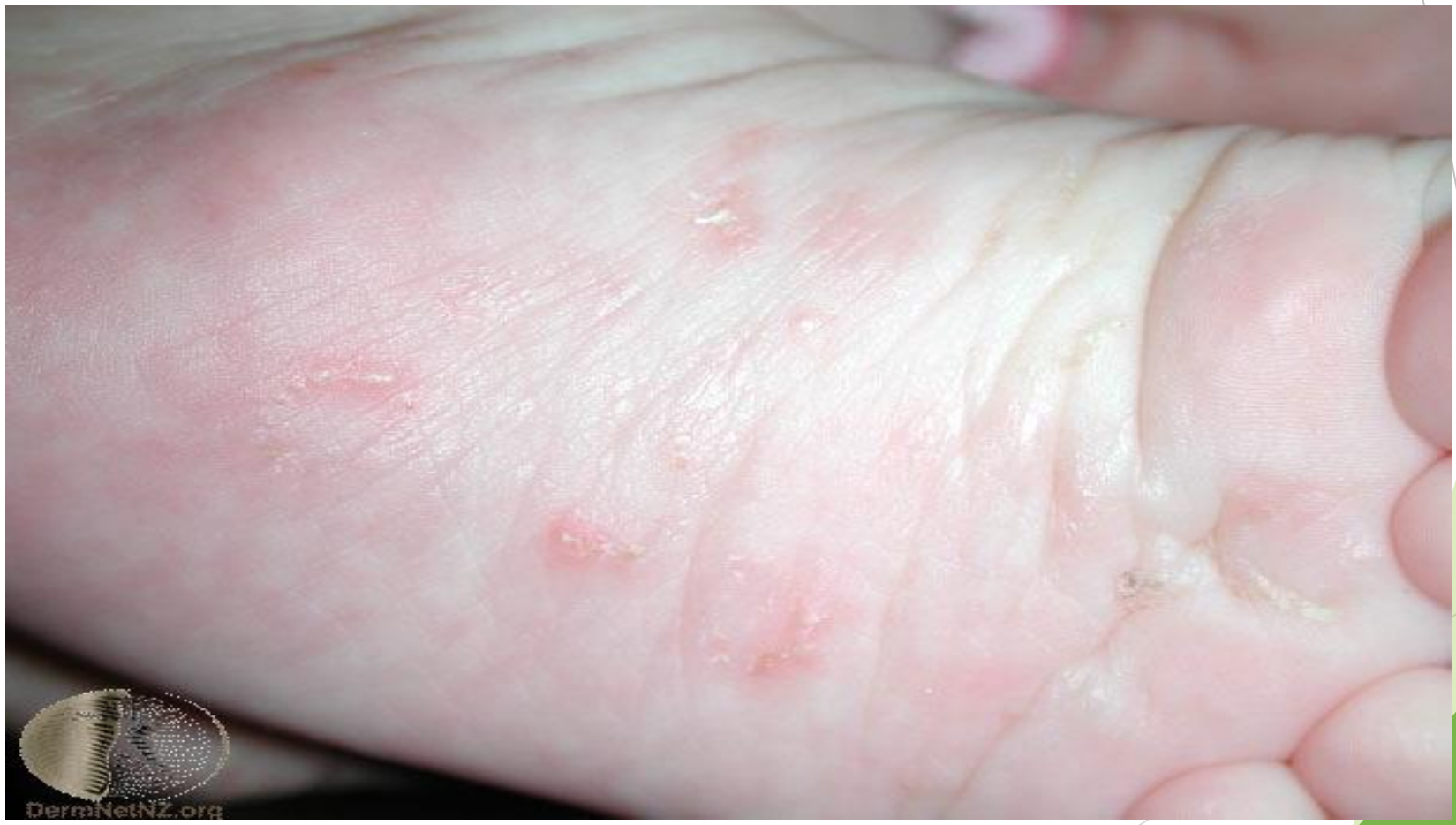
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TREATMENT AND PROGNOSIS

- **CUTANEOUS HYDRATION**
- **TOPICAL GLUCOCORTICOID TREATMENT**
- **Topical Immunomodulators**
- ***Tacrolimus***
- Tacrolimus inhibits the activation of a number of key cells involved in AD including T cells, Langerhans cells, mast cells, and keratinocytes.
- ***Pimecrolimus***
- **INFECTIOUS AGENTS**
- **Phototherapy**
- Ultraviolet B (311 nm), UVA-1 (340 to 400 nm), and combined UVAB phototherapy can be useful adjuncts in the treatment of AD.
- **CYCLOSPORINE**

- **ANTIMETABOLITES** Mycophenolate mofetil (MMF).
- Methotrexate.
- Azathioprine .
- **EXTRACORPOREAL PHOTOPHERESIS**
- **Dupilumab 600 mg sc then 300 mg every other week**
- **Interferon gama 50 microgram per metre**

- Following predictive factors correlate with a poor prognosis for AD: widespread AD in childhood; associated allergic rhinitis and asthma; family history of AD in parents or siblings; early age at onset of AD; being an only child; and very high serum IgE levels .