Dermatologic Emergencies

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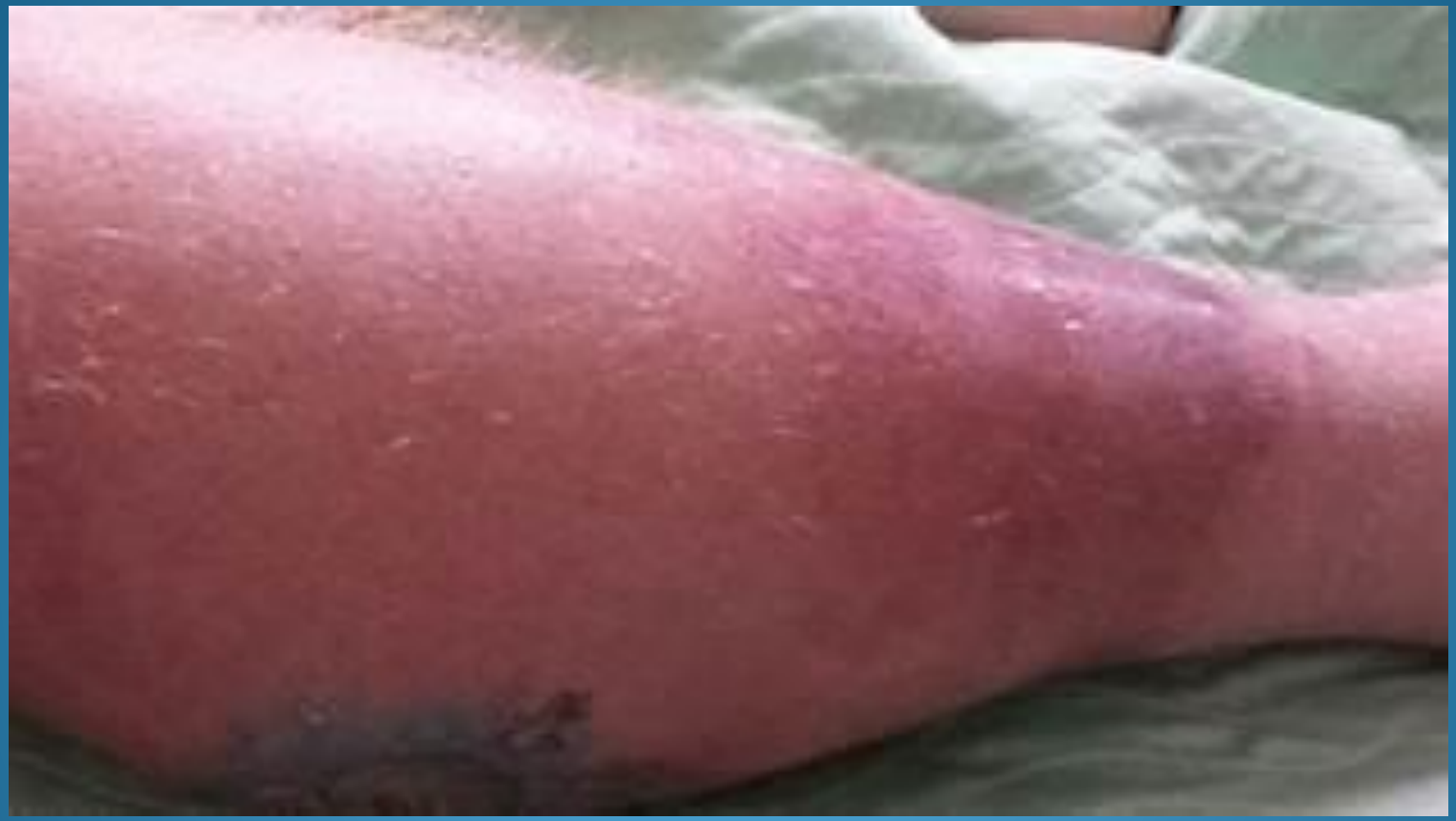
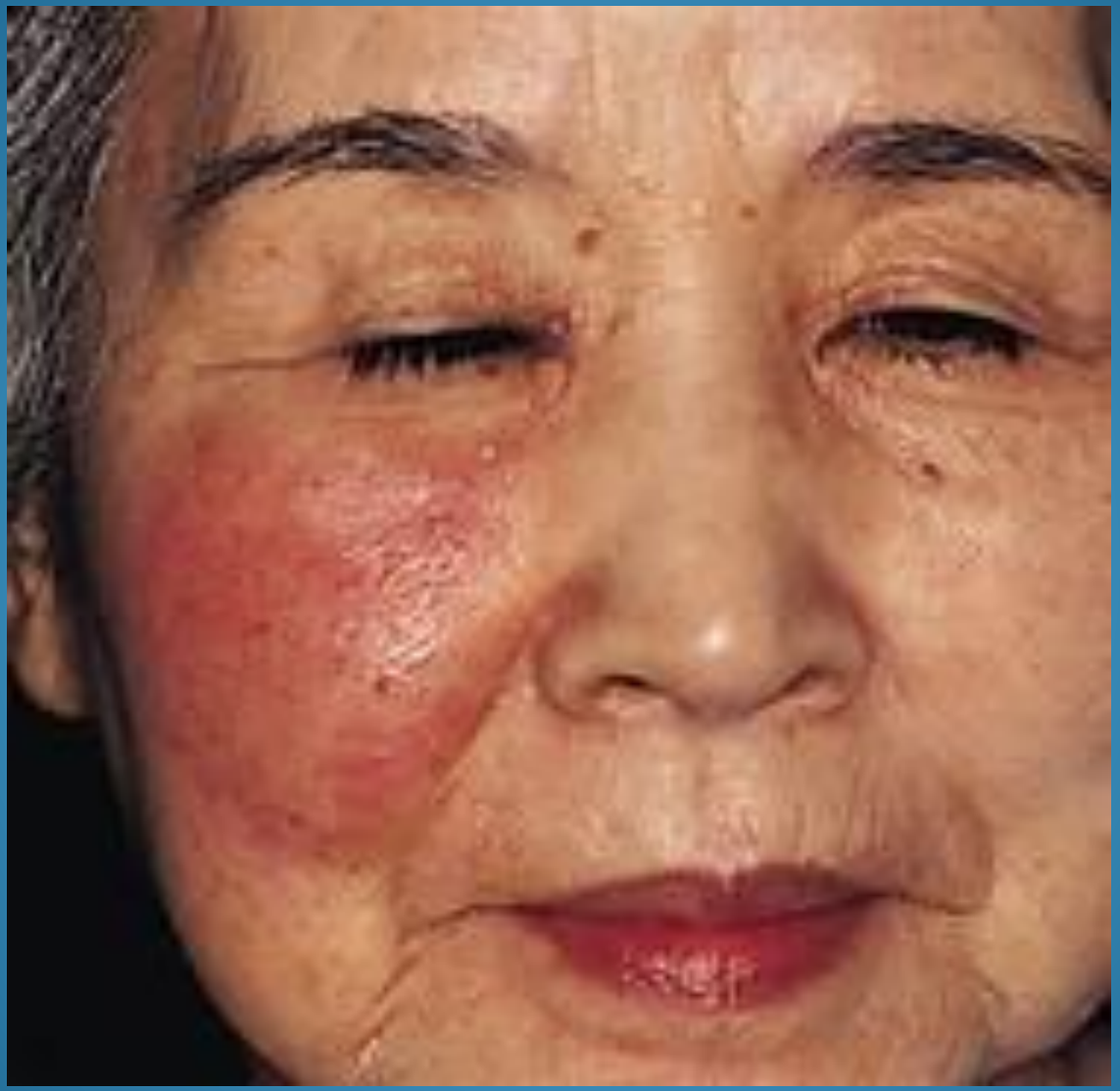
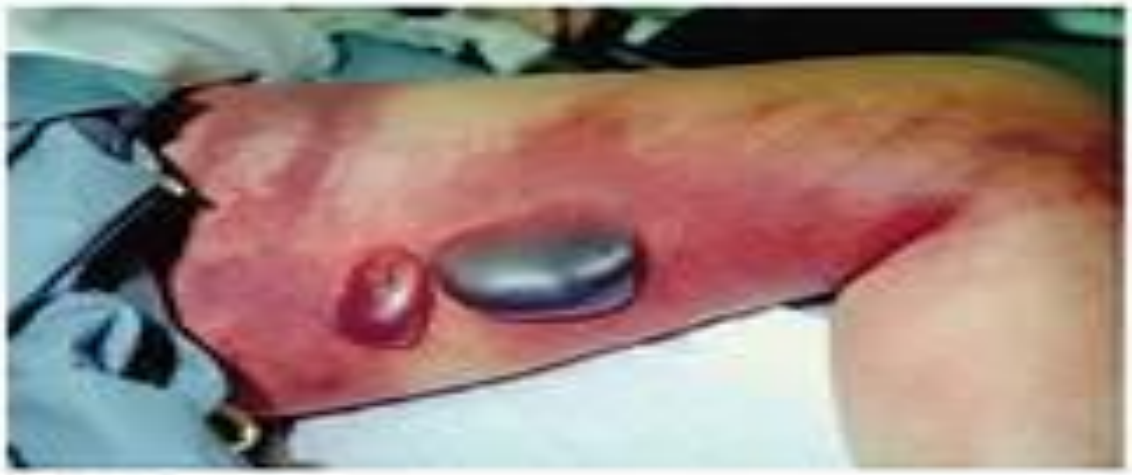
What are the major groups of dermatologic emergencies?

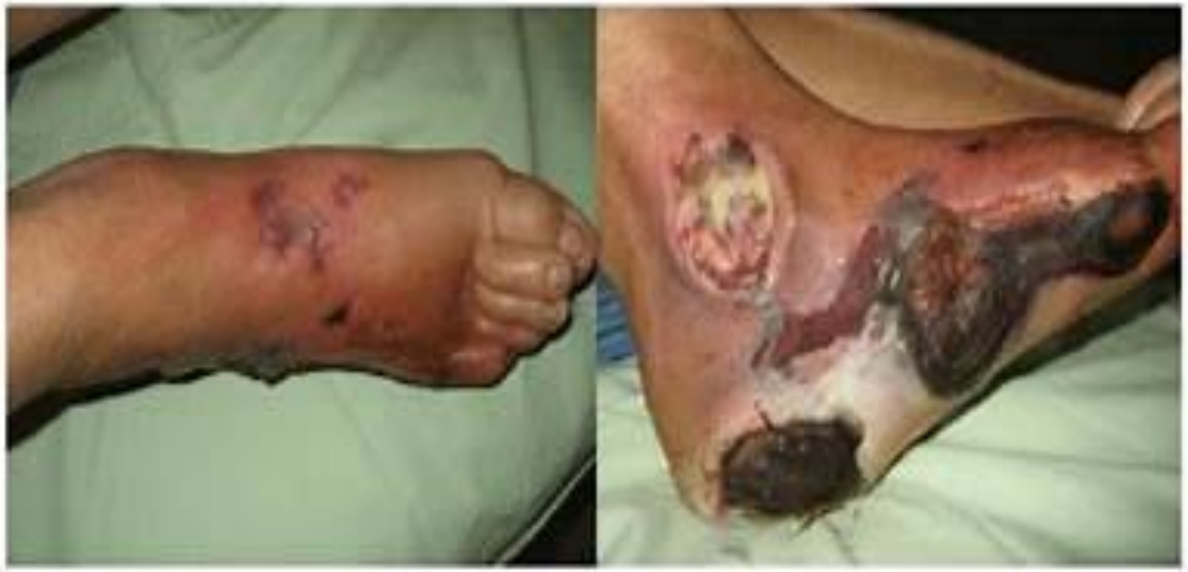
Infections

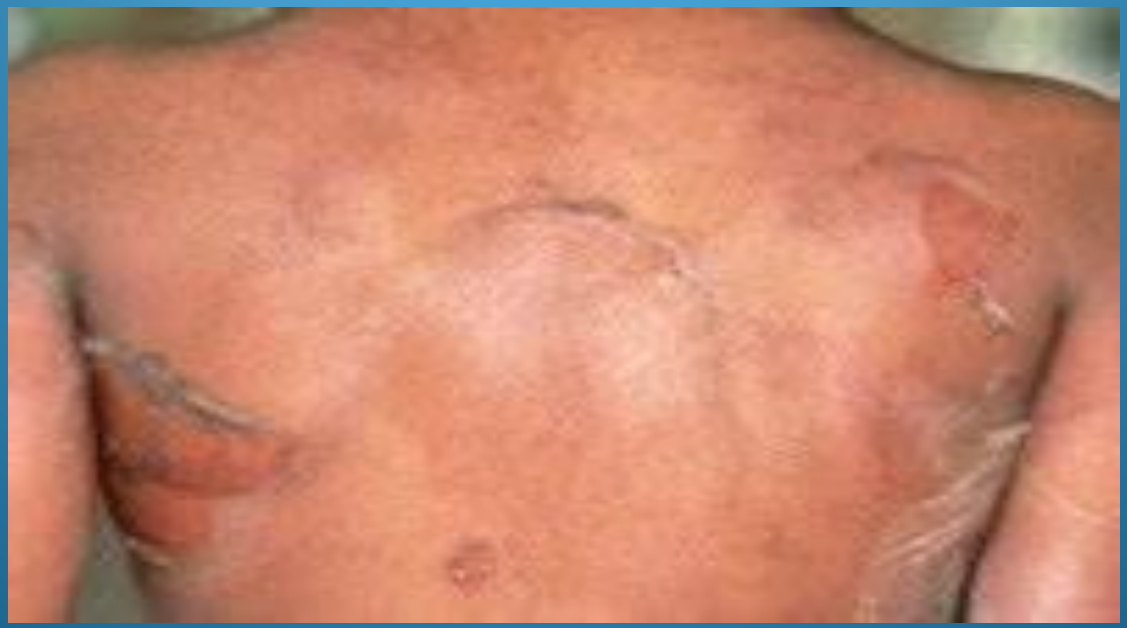
cellulitis
erysipelas
necrotizing fascitis
SSSS
neonatal herpes
neonatal candidiasis
mucormycosis

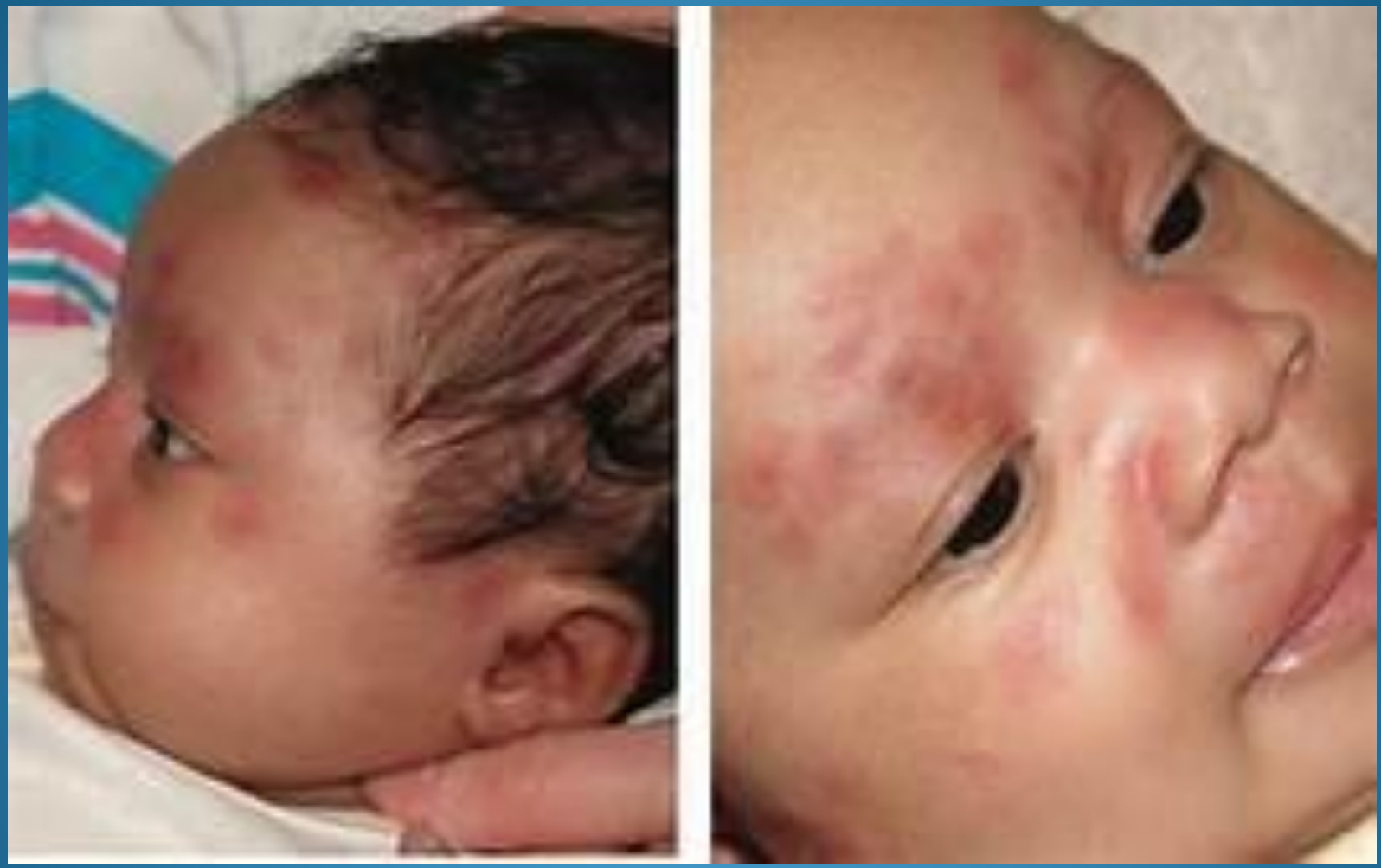


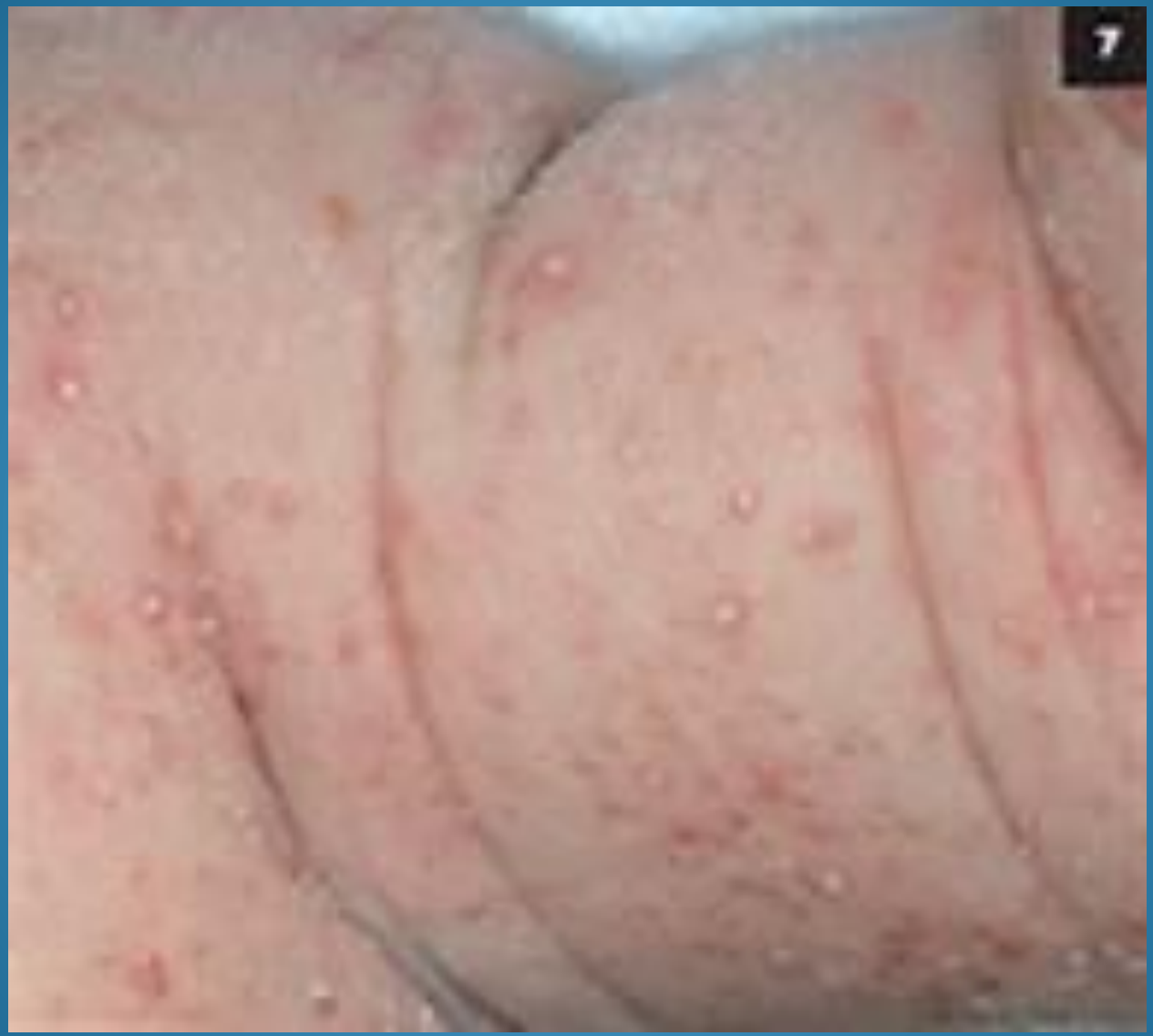








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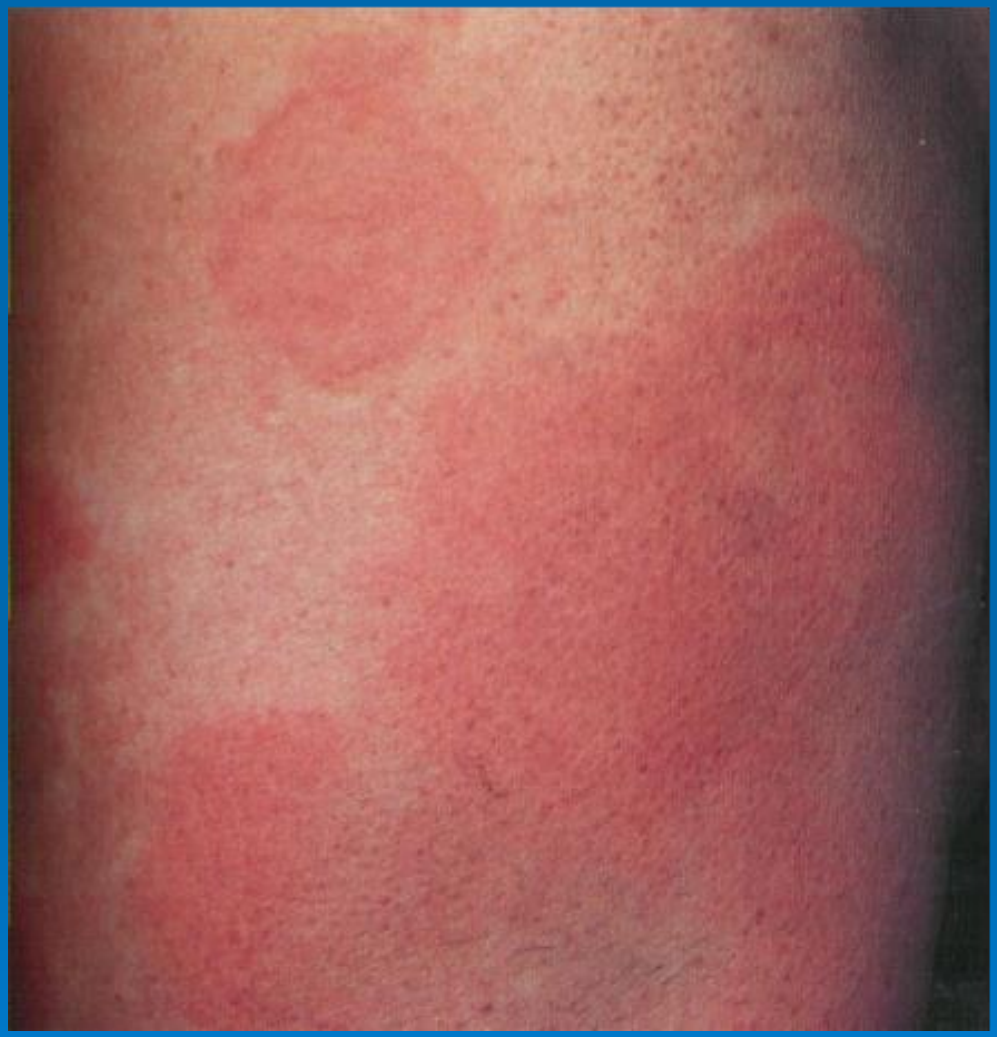
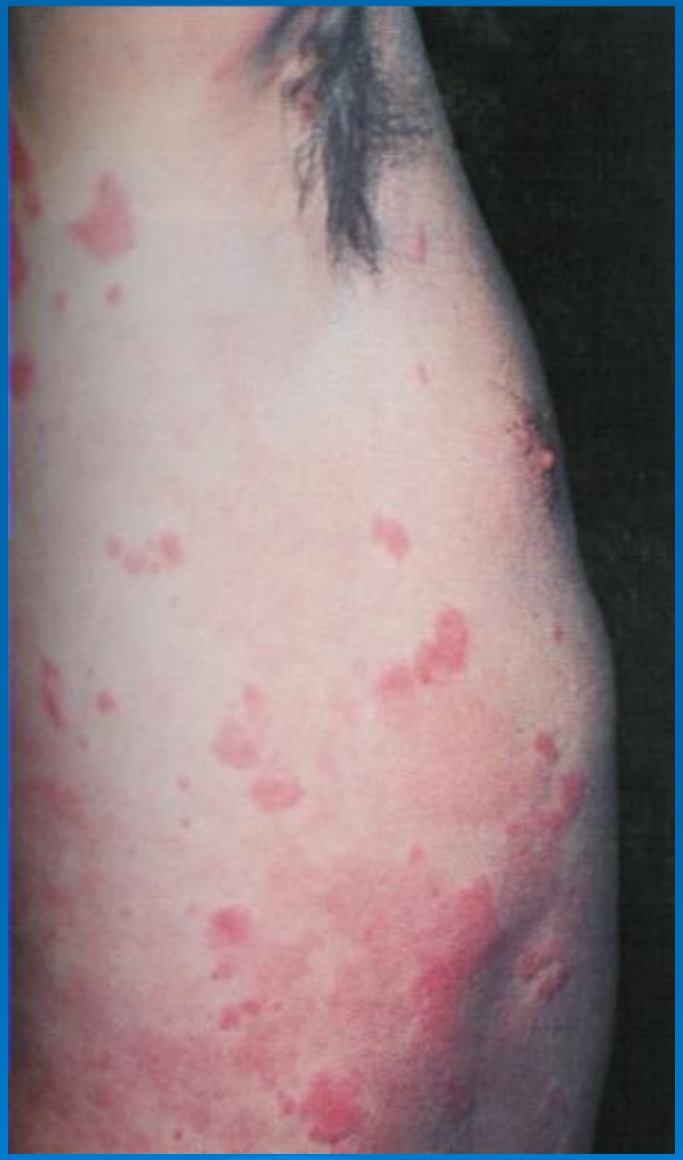
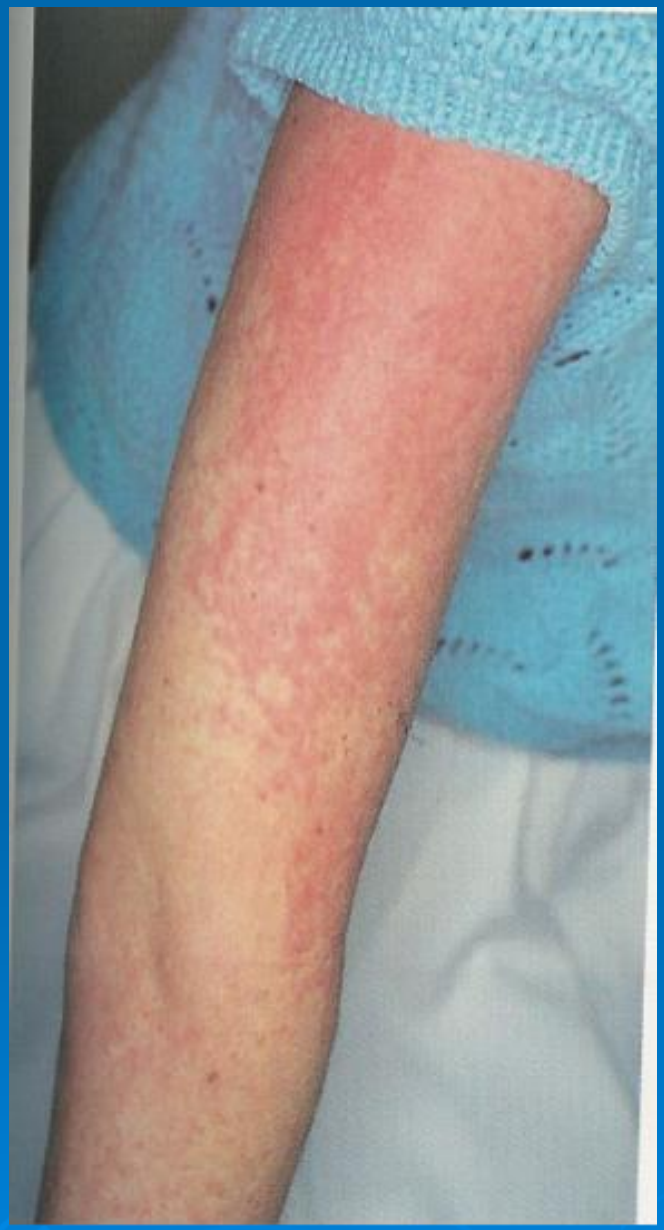
- Urticaria and angioedema, drug eruptions, kawasaki syndrome, exfoliative dermatitis, pustular psoriasis
- Drug reactions: EM, SJS, TEN
- Vesiculobullous disorders
- Autoimmune disorders
- miscellaneous

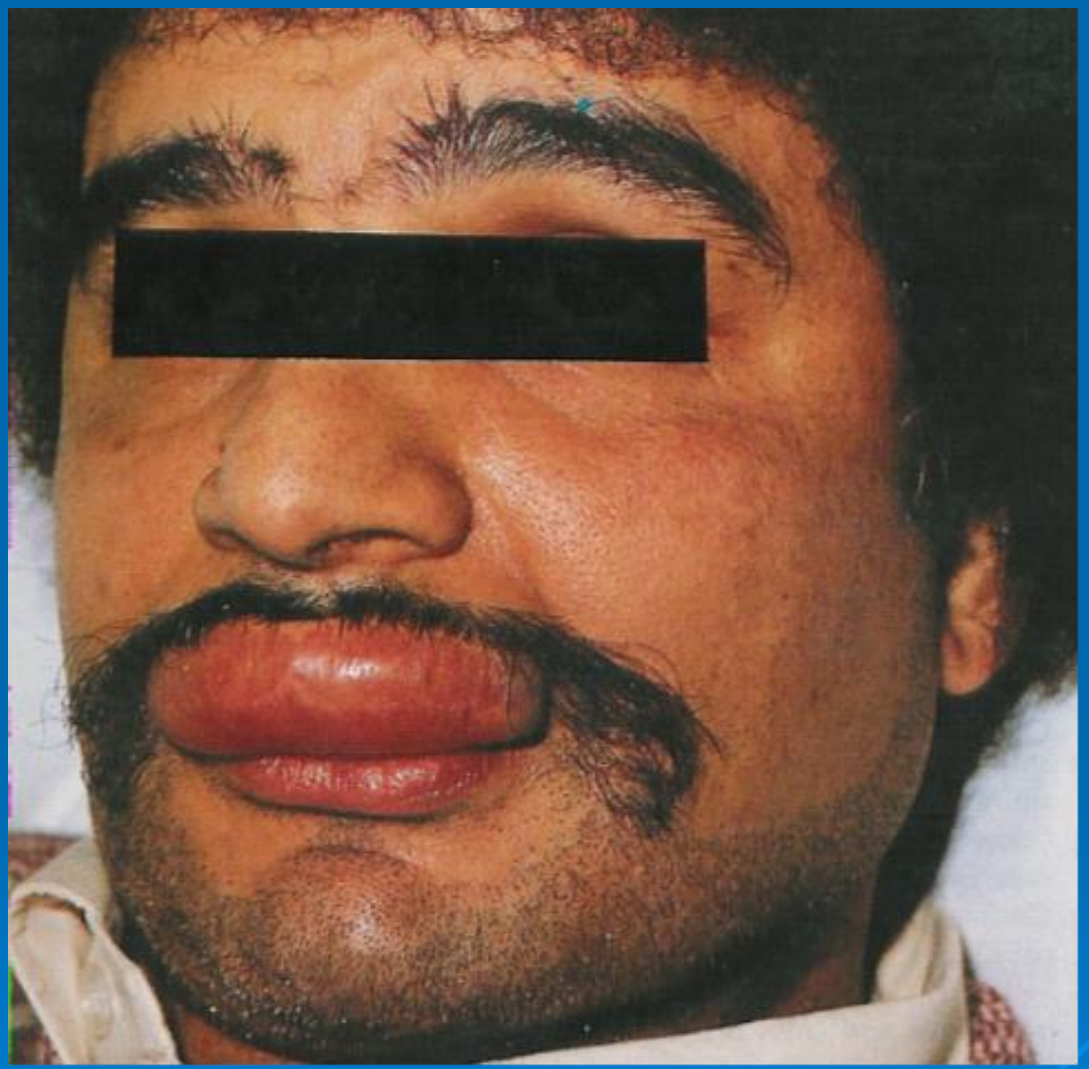
Urticaria and Angioedma

- **Essentials of Diagnosis:-**
  - Wheals or hives
  - Evanescent: individual wheals disappear within 24 hours
    and often within minutes.
  - Changing of configuration.
- **What is the major mediator of Urticaria?**
- **Pathogenic mechanism:**
  - Immunologic Type I (IgE mediated) or Type III.
  - Non immunologic (activation of the alternative complement pathway and direct release of histamine) from basophils or tissue mast cells by drugs or chemicals.



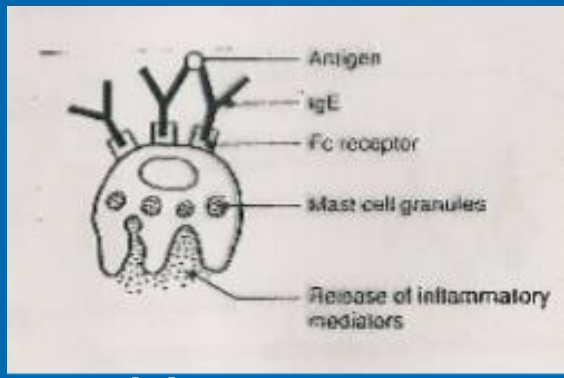






Classification of Hypersensitivity Reactions

Hypersensitivity is a state in which the immune responses frequently take place in such a way that cell damage occurs and harmful pathological lesions may occur.



5 types of hypersensitivity are recognized:

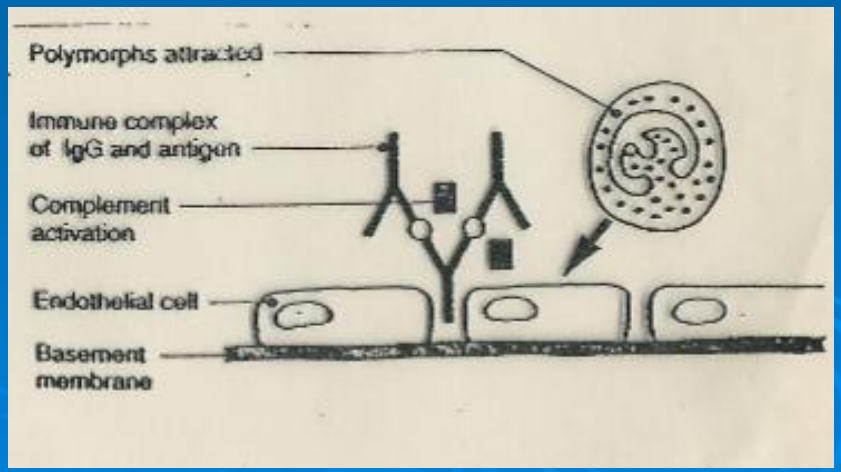
- Immediate (Antibody mediated):

Type I: Anaphylaxis

It is IgE-mediated. An antigen (allergen) reacting with specifically sensitized IgE that is fixed to mast cells through its FC portion. Degranulation of mast cells release of their mediators e.g. histamine, leukotrienes, ECF and NCF. The offending antigen is identified by intradermal prick tests giving immediate wheal and erythema reations or by provocation testing. There is a strong familial predisposition and a tendency to produce high levels of IgE. e.g. anaphylaxis, utricaria, atopy

Type III: Immune-Complex Reactions (arthus phenomenon)

The antigen reacts with specific circulating antibodies antigen-antibody complexes that act through : (a) complement activation and PMNL attraction inflammatory response. (b) platelet aggregation microthrombi and vasoactive amine release. E.g. serum sickness, nephritis in SLE, Allergic vasculitis, urticarial vasculitis, ? Bullous pemphigoid.



Acute Urticaria:

Chronic Urticaria:

Urticaria may accompany systemic Anaphylaxis or serum sickness reaction.

Serum sickness:

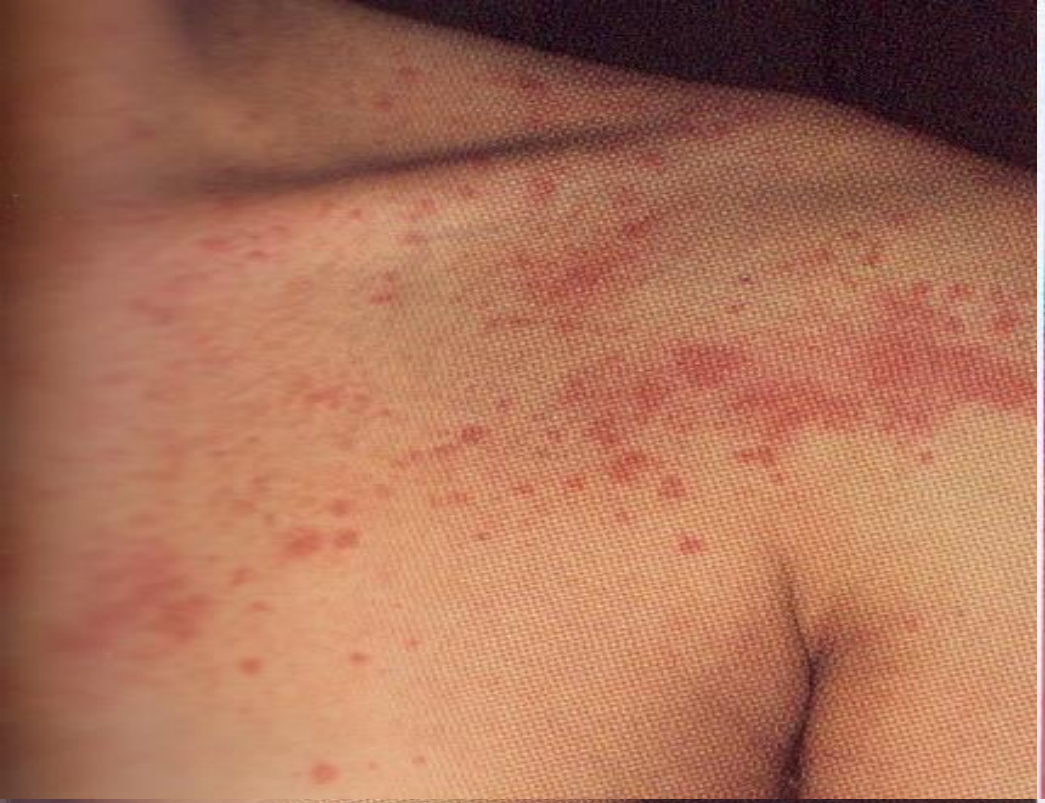
- 5d 3weeks after initial exposure (type III) immune complex mediated reaction.
- Fever, Urticaria, Angioedema, Joint pain and swelling, lymphadenopathy, occas: nephritis or endocarditis with eosinophilia.
- Minor form of Serum sickness: fever, Urticaria, transitory joint tenderness.

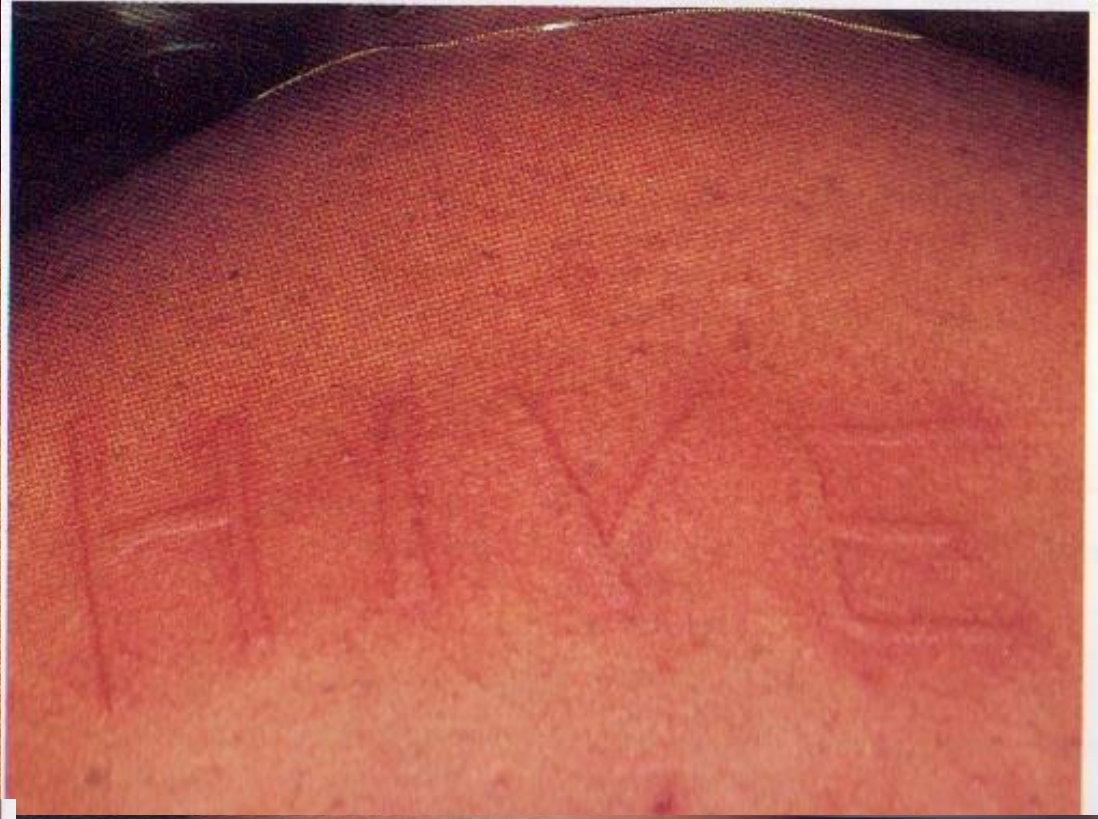
Angioedema: Involving oedema of the deep dermis or subcutaneous and submucosal areas.

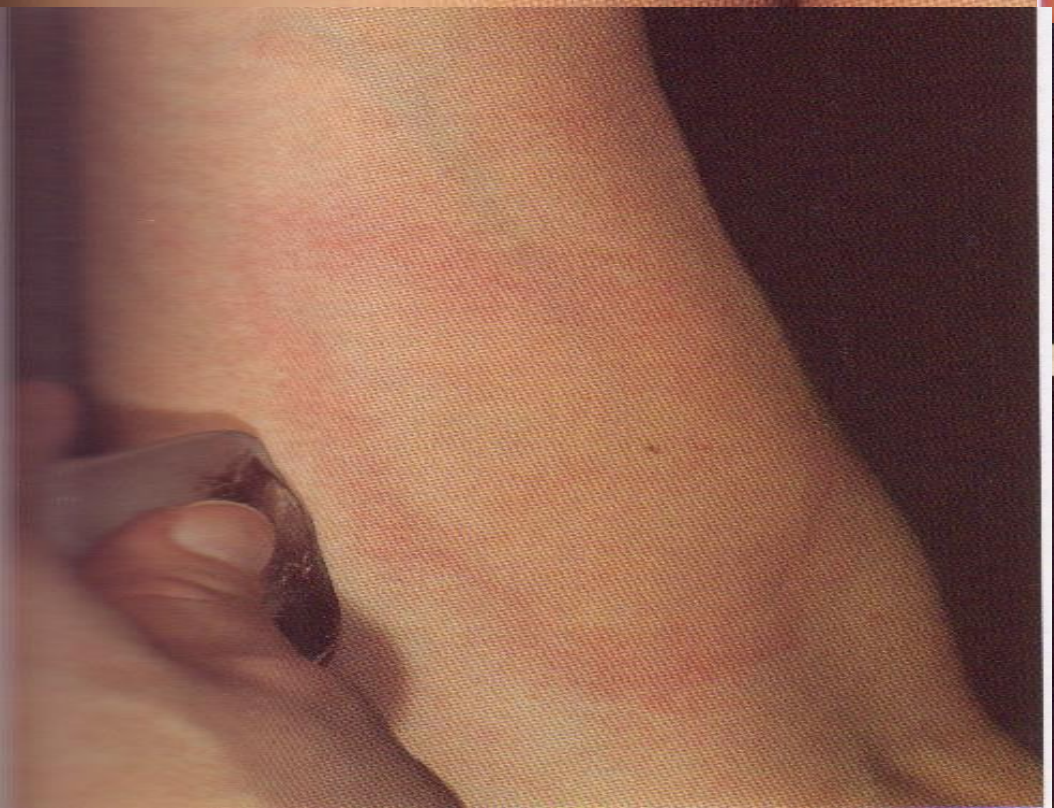
Causes of Urticaria and angioedema

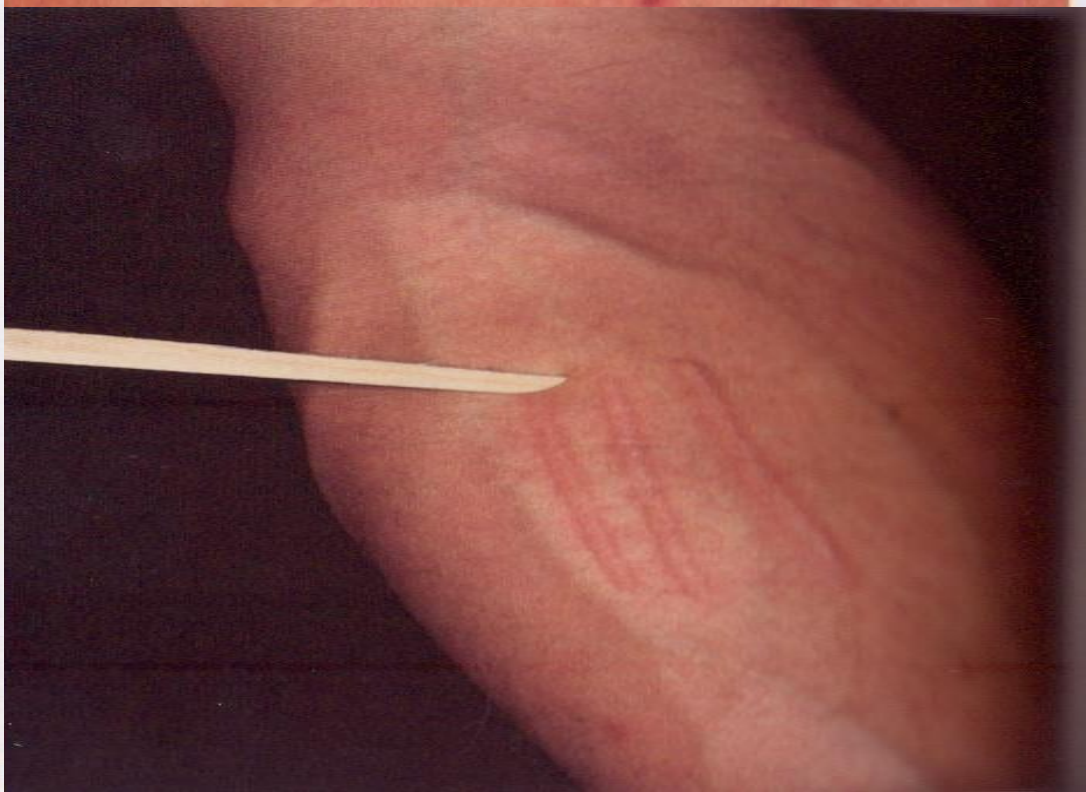
Drugs- Animal sera, vaccines containing egg protein, desensitizing agents, Antibiotics, ACEI, radiocontrast media, cylogenase inhibitors.

- Foods
- Inhalants
- Infections
- Autoimmune disease
- Insect bites
- Physical causes
- Hereditary causes, hereditary Angioedema
- Vasculitis
- Contactants
- Idiopathic
- SLE, thyroid dis
- Neoplasm (iymphoma, ca of lung and colon)









Approach to the patient with chronic Urticaria.

Treatment:

- Elimination or avoidance of the causative agent.
- Nonsedating H_1 antagonists.
- Sedating H_1 antagonists
- Addition of H_2 antagonists
- Corticosteroids
- Epinephrine
- Montelukast
- Thyroxine
- Colchicine
- Sulfasalazine
- Immunosuppressive therapy
- Omalizumab (humanized anti igE monoclonal antibody)
- C1-esterase inhibitor deficiency : oral danazole,oral tranexamic acid ,for emergency : C1 inhibitor concentrate , fresh frozen plasma , oestrogen & OCP avoidance .

Treatment (Continued):

- In acute emergency episodes
  - Secure the airway
  - I.V. line
  - Adrenaline subcutaneous or I.M., repeated every 10 min.
  - Diphenhydramine I.M. or I.V.
  - Hydrcortisone I.V.
  - Patients with severe angioedema should be admitted for at least 24 hours observation particularly where laryngeal edema has occurred.

Anaphylaxis

Essentials of Diagnosis:-

- Laryngeal edema or bronchospasm or both.
- Erythema, puritus, urtiaria or angioedma (any or all)
- Vomitting, cramps, diarrhea.
- Hypotension, cardiac arrythmia or shocks.

General considerations:

- Within minutes to hours, severe may be fatal.
- IgE mediated
- Chemical mediators are released
- Anaphylactoid reactions: clinically similar reactions that involve the nonimmunologic (non antigen-antibody) release of similar mediators e.g (reactions to radiographic contrast media, aspirin, local anesthetics)

Causative agents:

- Drugs
- Foods
- Vaccines and Antisera
- Insects – bees, wasps
- Immunotherapy of allergic rhinitis, asthma or stinging insect sensitivity.
- other cases:
  - Iodinated contrast media, aspirin, local anesthetics – anaphylactoid reactions.
  - Treatment of anaphylaxis

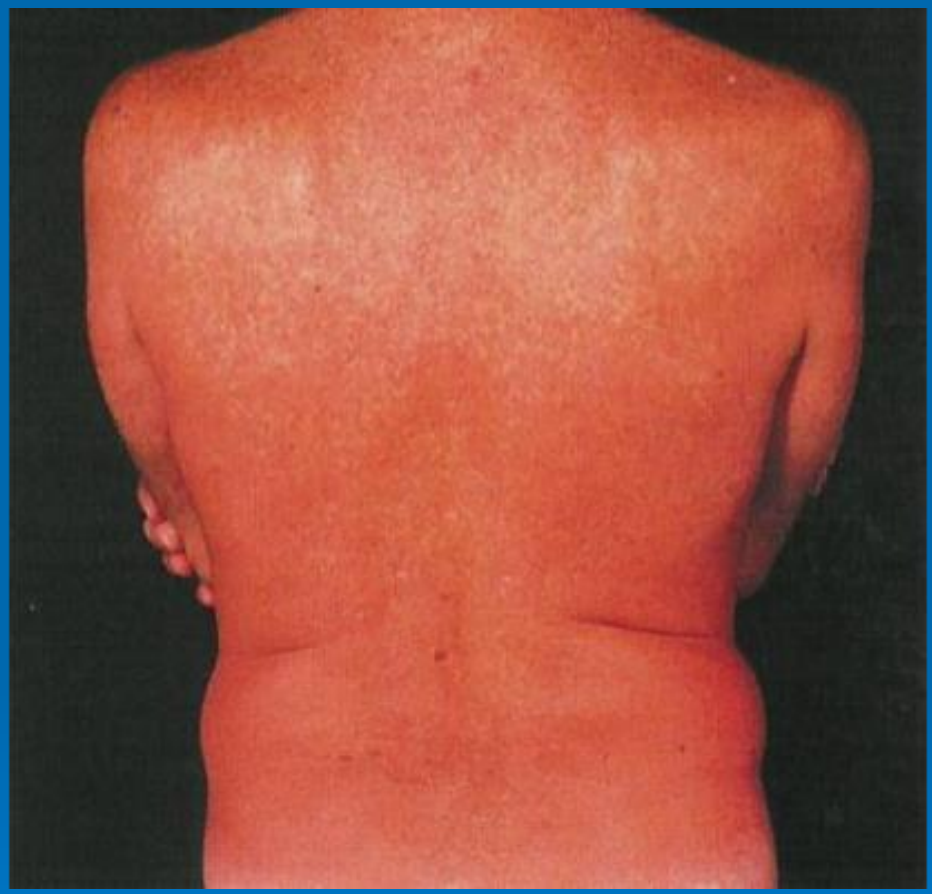
Erythroderma & Exfoliative Dermatitis

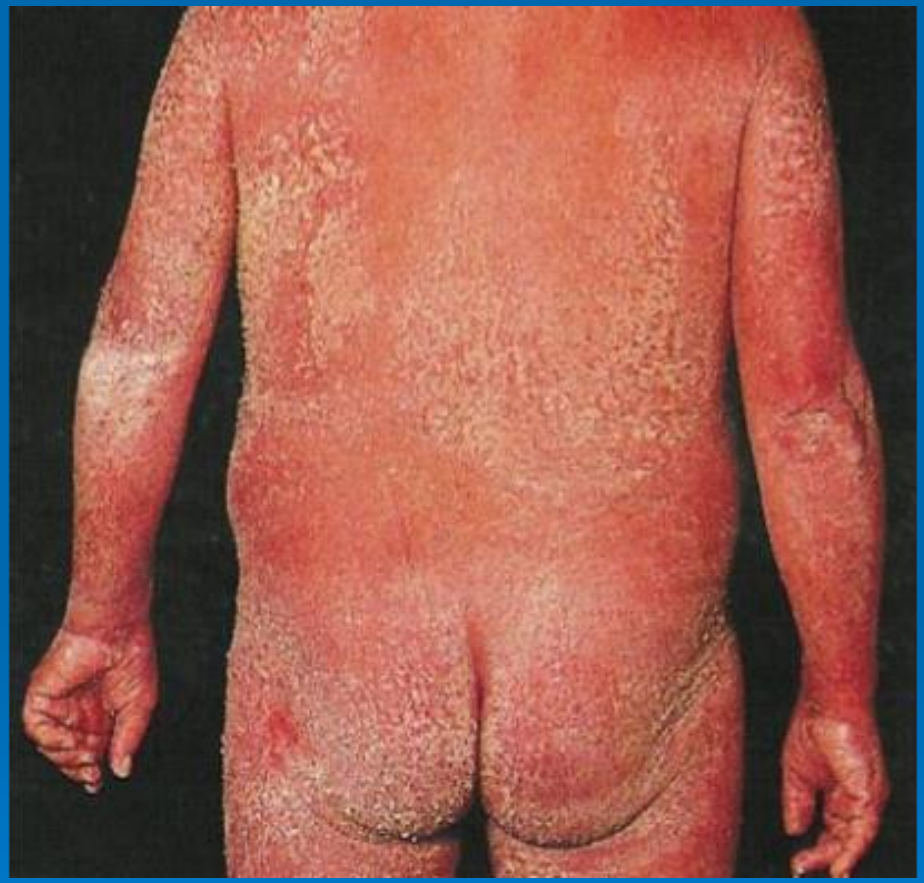
- Clinical features
- Complications: Hypothermia, fluid and electrolyte loss, infection,

HF., stress induced GIT ulcer, malabsorption., venous thrombosis.

Drug Etiology:

- Sulphonamides, antimalarias, penicillin, phenytoin.
- Other causes





Erythema Multiforme Major, SJS, Toxic Epidermal Necrolysis

- SJS , TEN May represent variants of the same disease process.
- Clinically: Mucous membrane erosions, target lesions
- epidermal necrosis with skin detachment.
- SJS < 10% BSA of epidermal detachment ,SJS –TEN Ooverlap 10-30%, TEN > 30%
- EM minor little or no mucosal involvment , Preceding herpes labialis in 50% of EM
- Systemic symptoms present in EM major & absent or limited in EM minor .

Suspected etiologic factors:

- Infections
- Drugs > 95% in TEN, anticonvulsants, sulphonamides, allopurinol, NSAI.
- Neoplasia
- Collagen disease
- immunizations

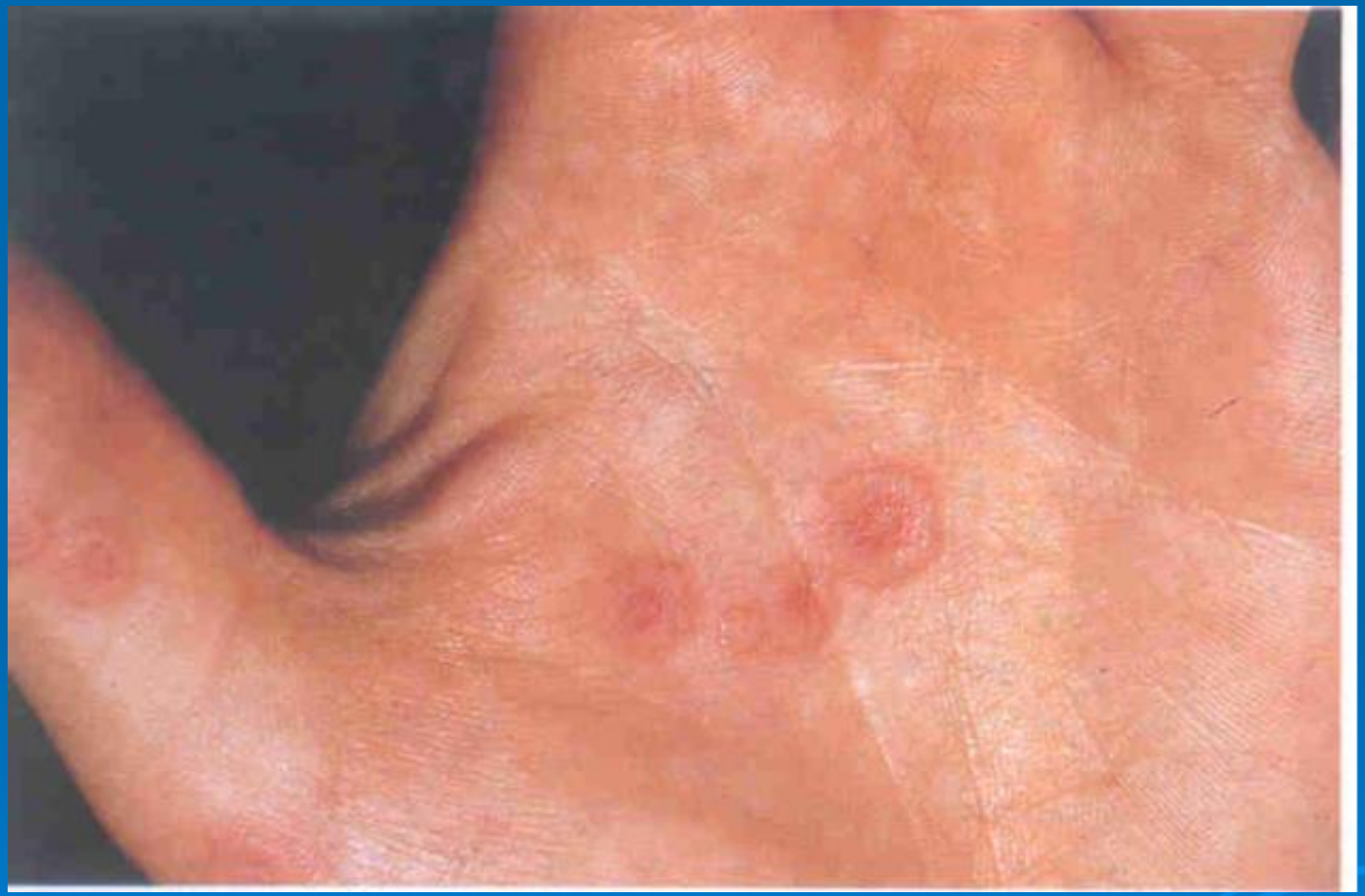
Risk factors for TEN

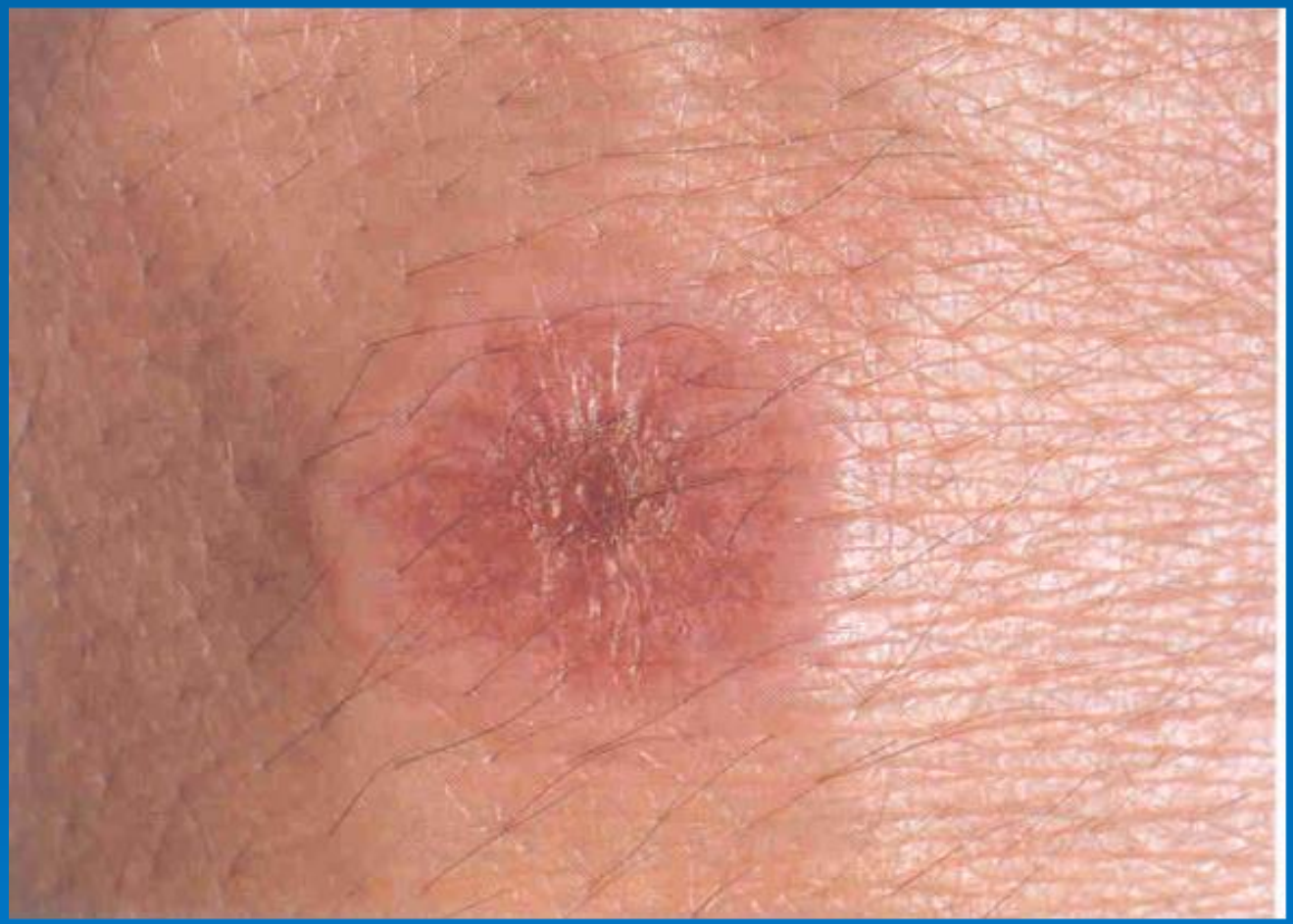
- slow acetylators
- Immunosuppression (HIV, Lymphoms)
- Radiotherapy & anticonvulsants
- Specific HLA

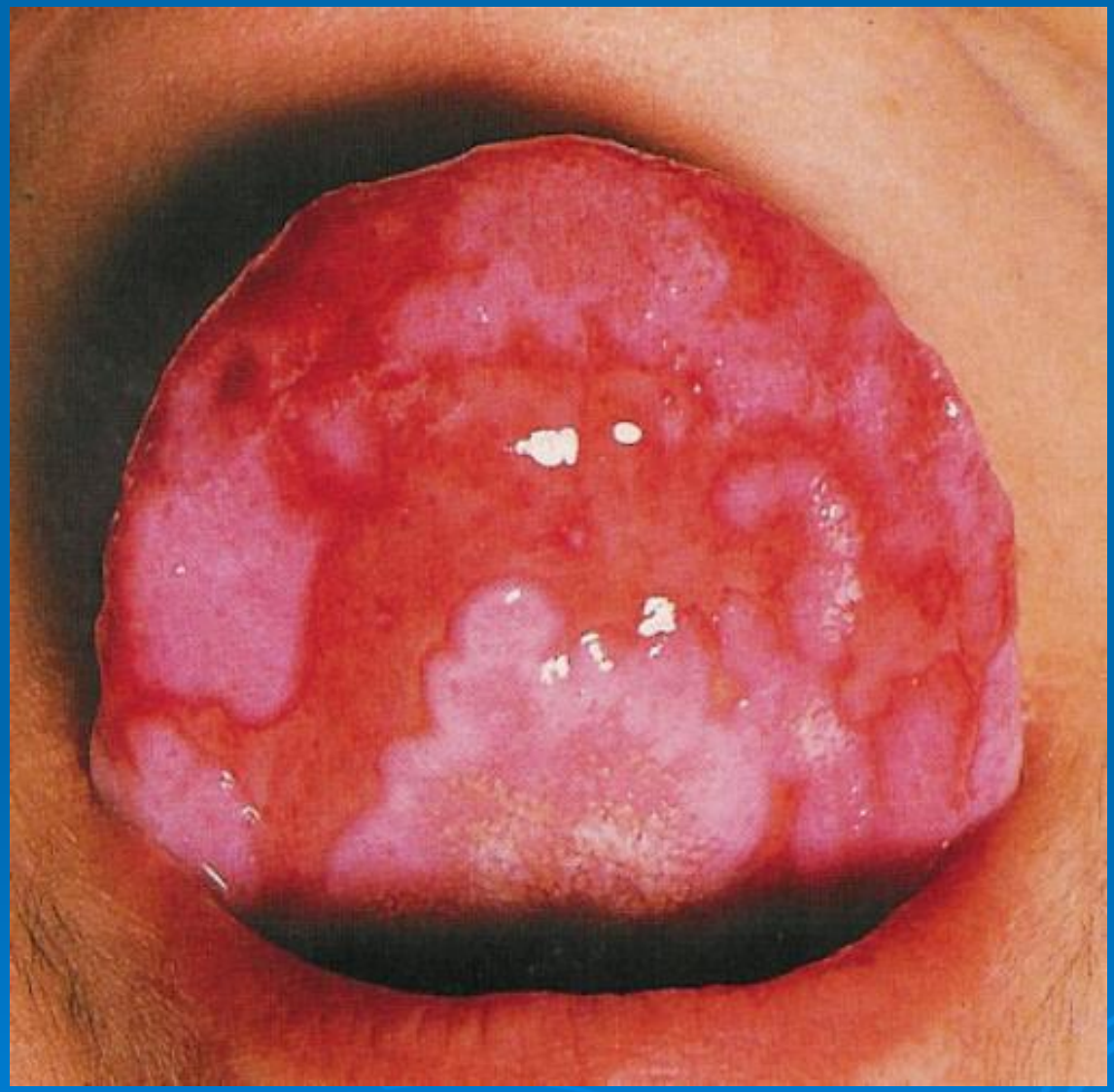
FDA recently recommended genotyping of all asians for the allele HLAB 1502 prior starting carbazepine

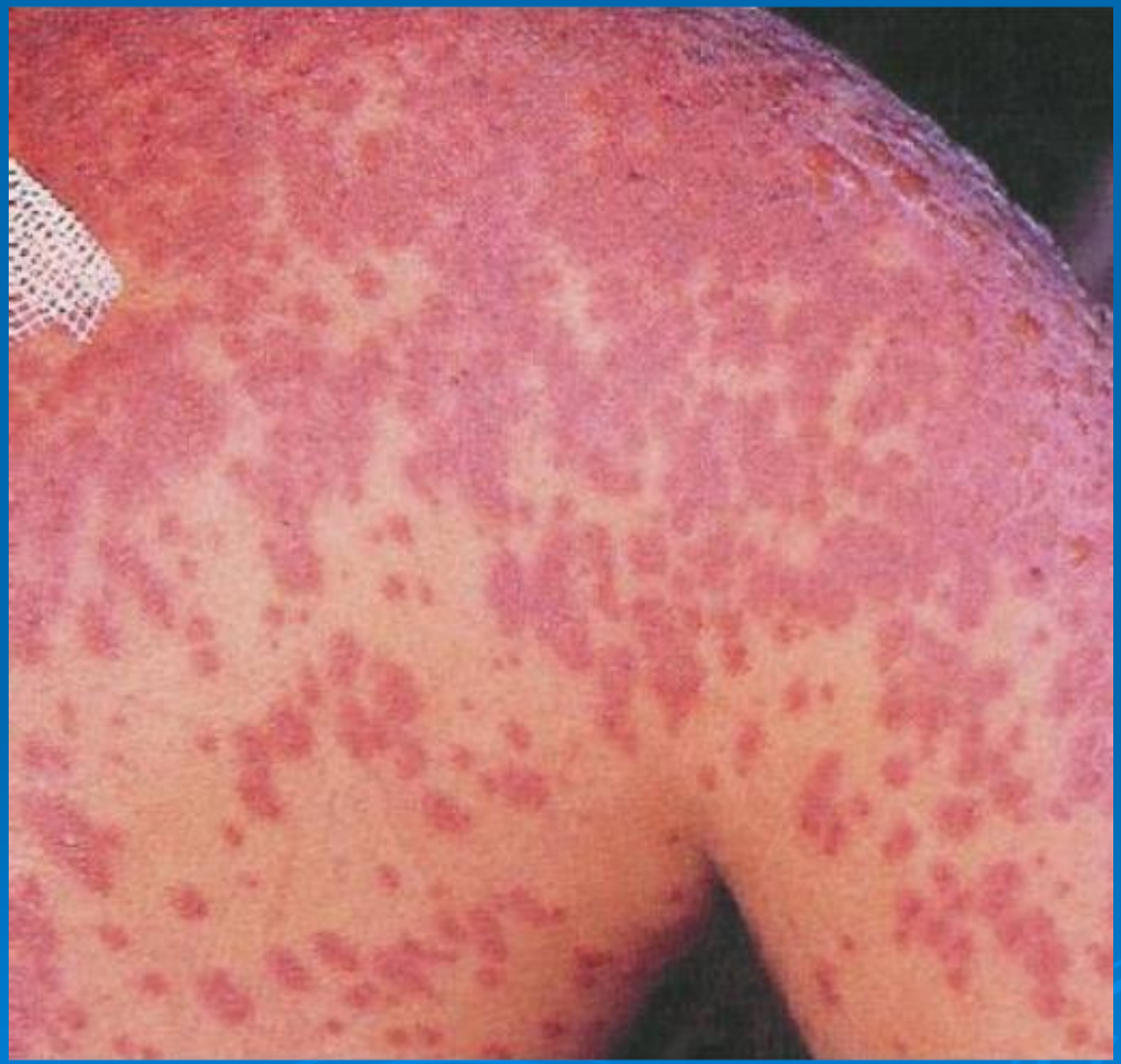
Prognostic factors in TEN

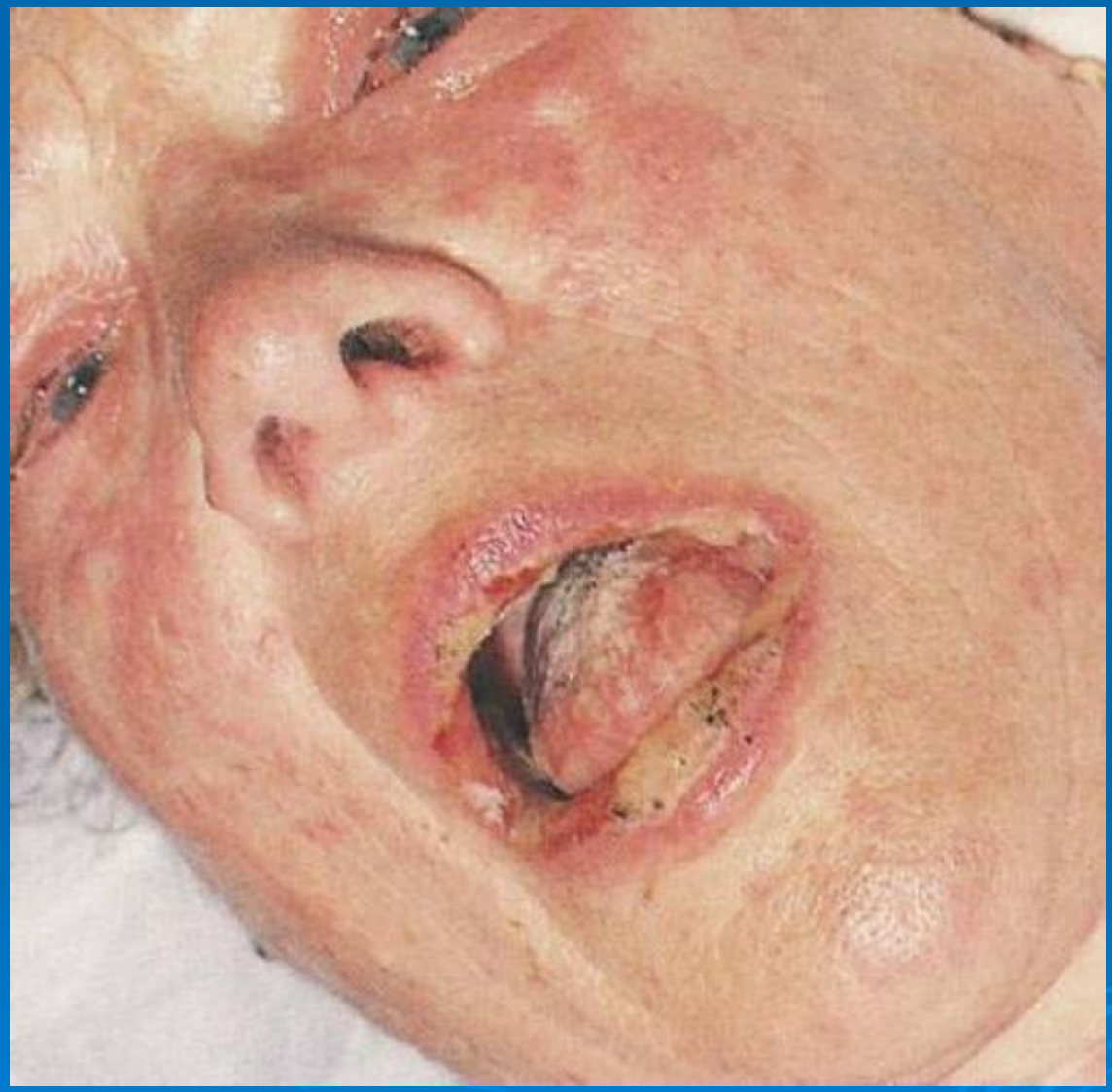
- Major cause of death
- Differentialdiagnosis

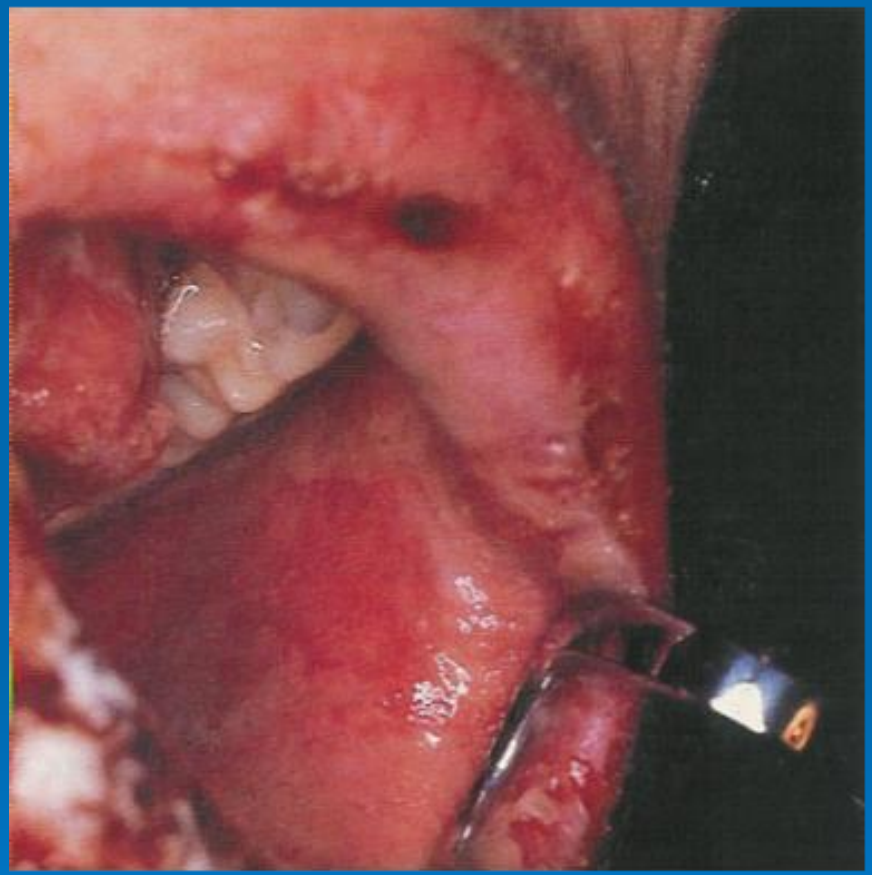


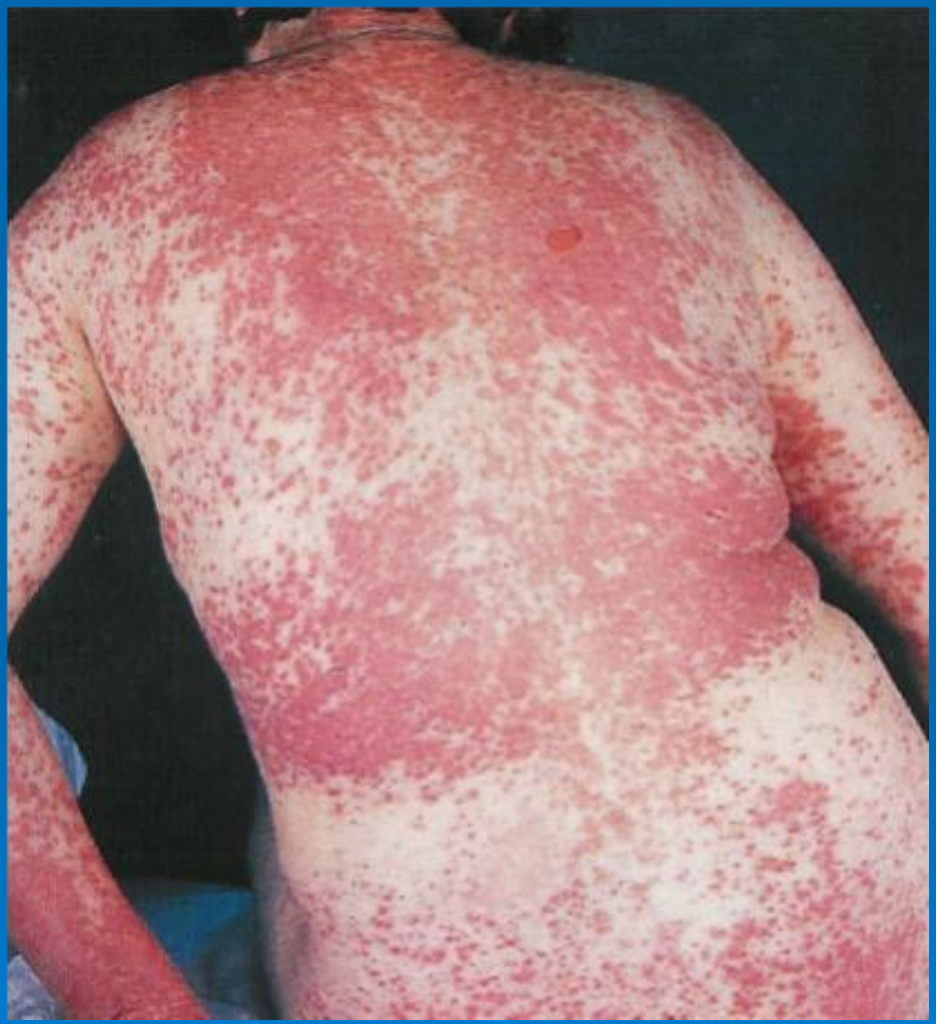


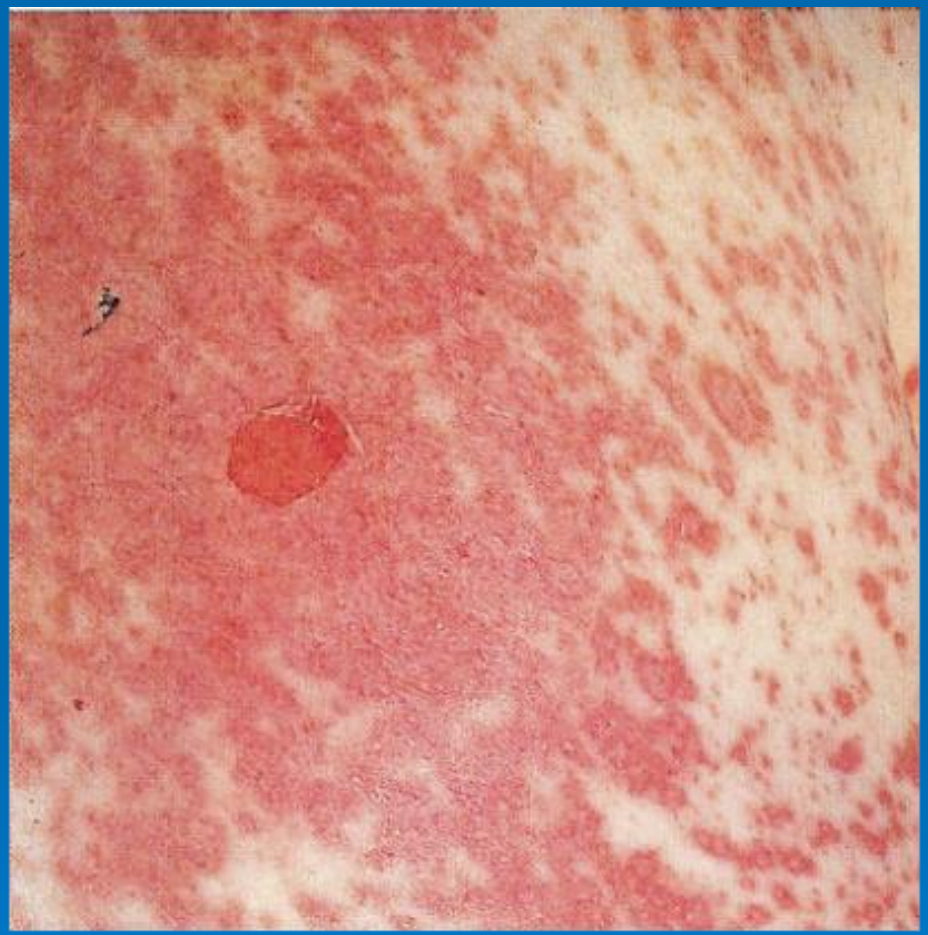


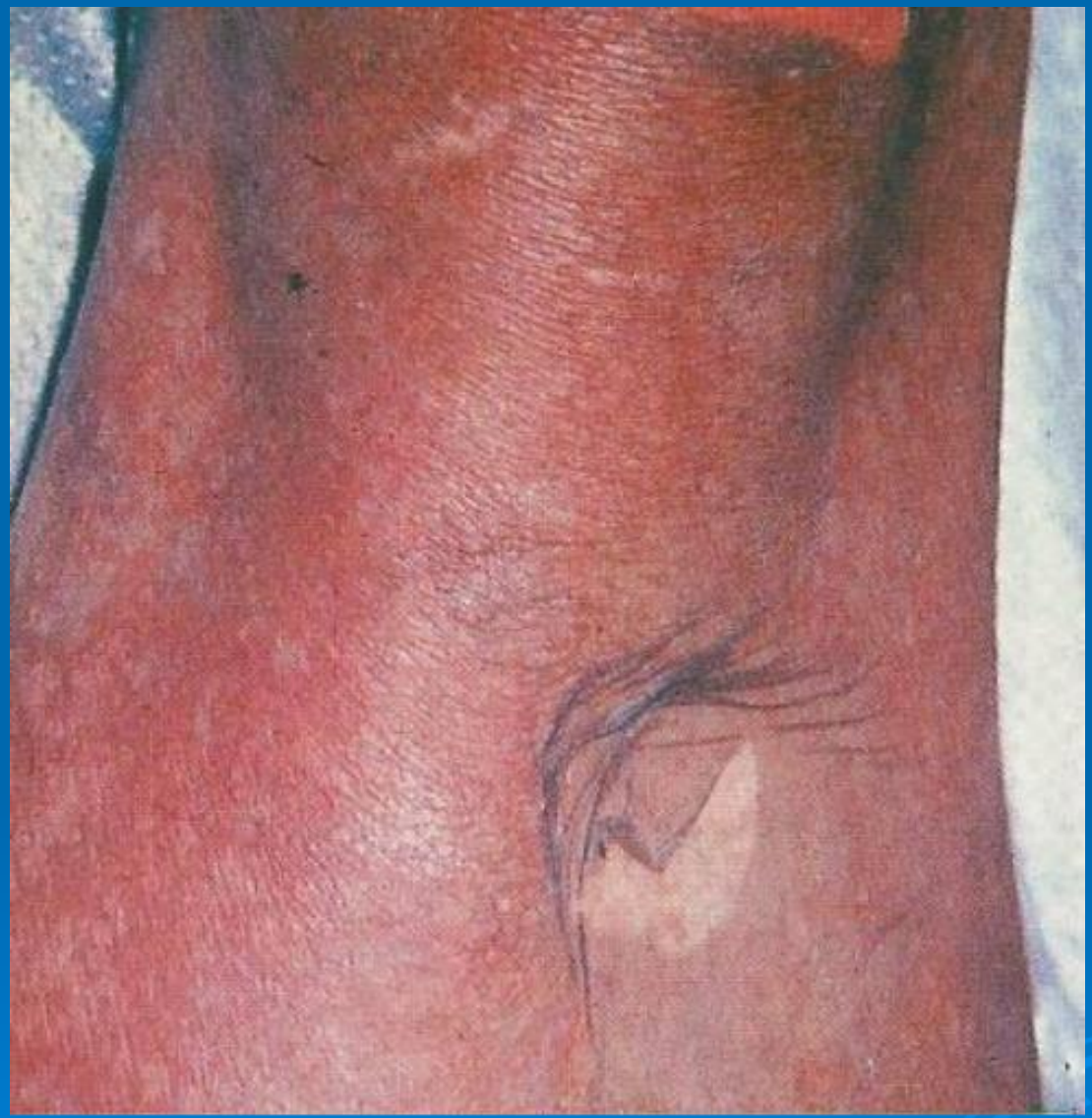


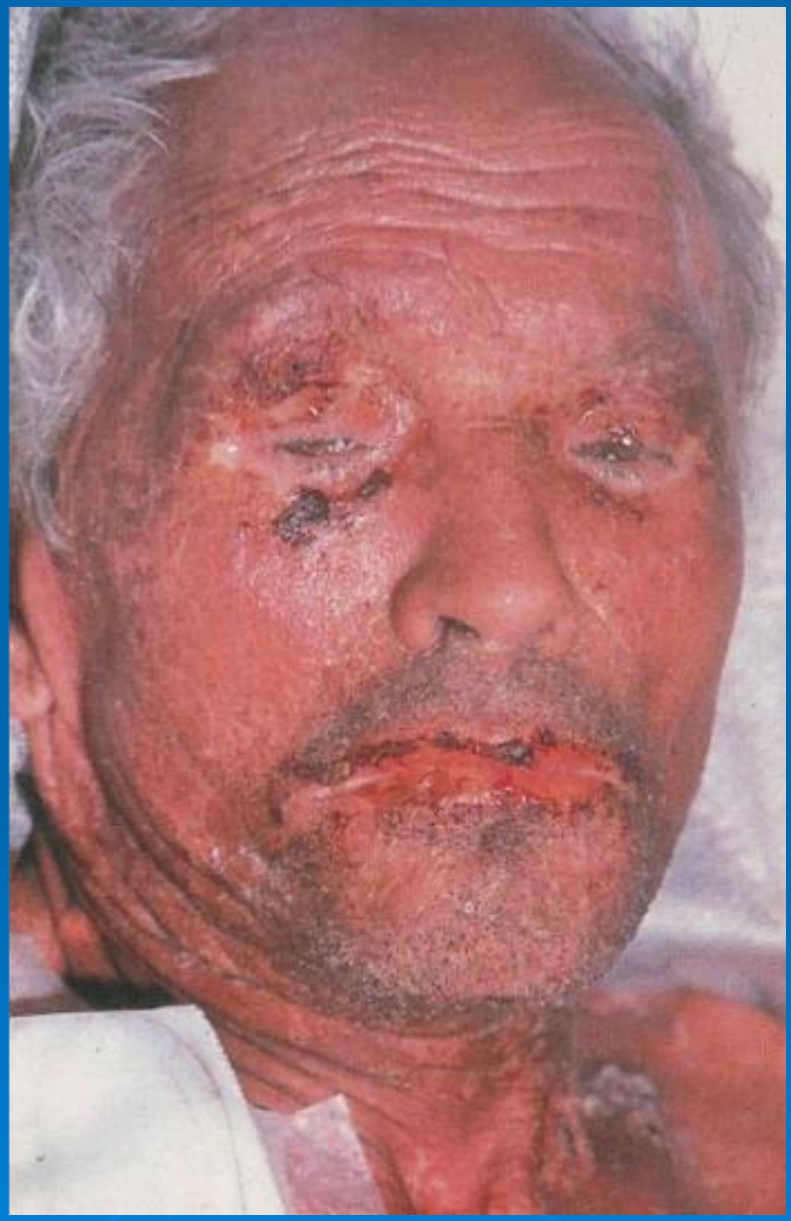












Pathogenesis:

- ? Metabolic base may trigger an immune response
- Prognosis: related to extent of skin involvement.
- Mortality for TEN: 30% related to sepsis, fluid and electrolyte imbalance.

Management

- Early diagnosis, withdrawal of suspected drug
- Patient best cared in a burn or I.C.U.
- Replacement of I.V. fluids and electrolytes
- Systemic corticosteroids-controversial, IVIg.
- CSA, cyclophosphamide, plasmapheresis, N-acetylcysteine, TNF alpha antagonist (etanercept, infliximab)
- Care for mucous membrane, eye involvement
- diagnose and treat complicating infections
- Prevention

TABLE 14-3 Kawasaki Syndrome CDC Diagnostic Criteria

Symptom	Percentage of occurrence
Fever lasting longer than 5 days plus at least four of the following:	100%
1. Bilateral conjunctival injection	92%
2. Mucous membrane changes (1 or more)	100%
Red or fissured lips	84%
Red pharynx	72%
"Strawberry" tongue	32%
3. Lower extremity changes (1 or more)	
Erythema of palms or soles	72%
Edema of hands or feet	48%
Desquamation (generalized or periungual)	56%
4. Rash—erythematous exanthem	100%
5. Cervical lymphadenopathy (At least 1 node larger than 1.5 cm)	72%

Data from Velez-Torres R, Callen JP: *Intern J Dermatol* 26:96-102, 1987.

TABLE 14-4 Kawasaki Syndrome Other Clinical Findings

Symptom	Percentage of occurrence
Arthralgias	24%
Cough	25%
Urethritis—sterile pyuria	75%
Aseptic meningitis and irritability	25%
Hepatitis—jaundice	20%
Diarrhea	28%
Hydrops of the gallbladder	5%
Vomiting	60%
Cardiac involvement	33%
Myocarditis	6%-24%
Pericarditis	4%
Coronary artery aneurysms	17%-31%

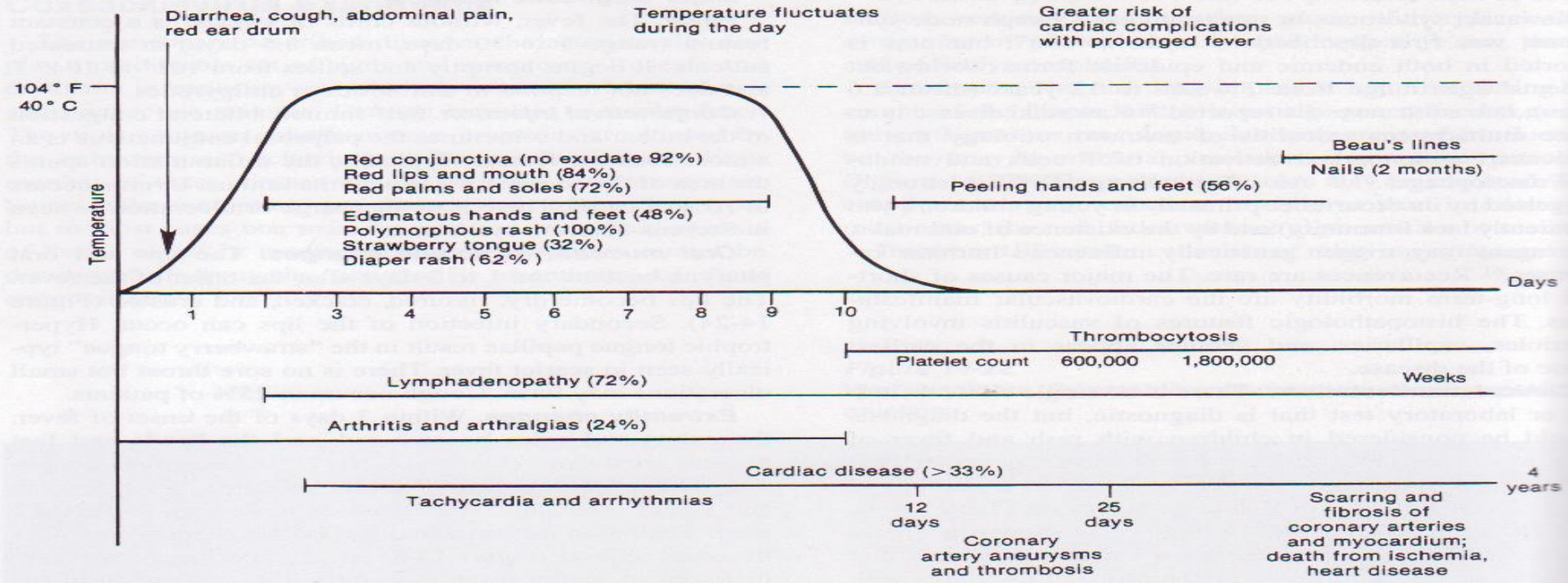


Figure 14-23
Kawasaki syndrome. Evolution of signs and symptoms.

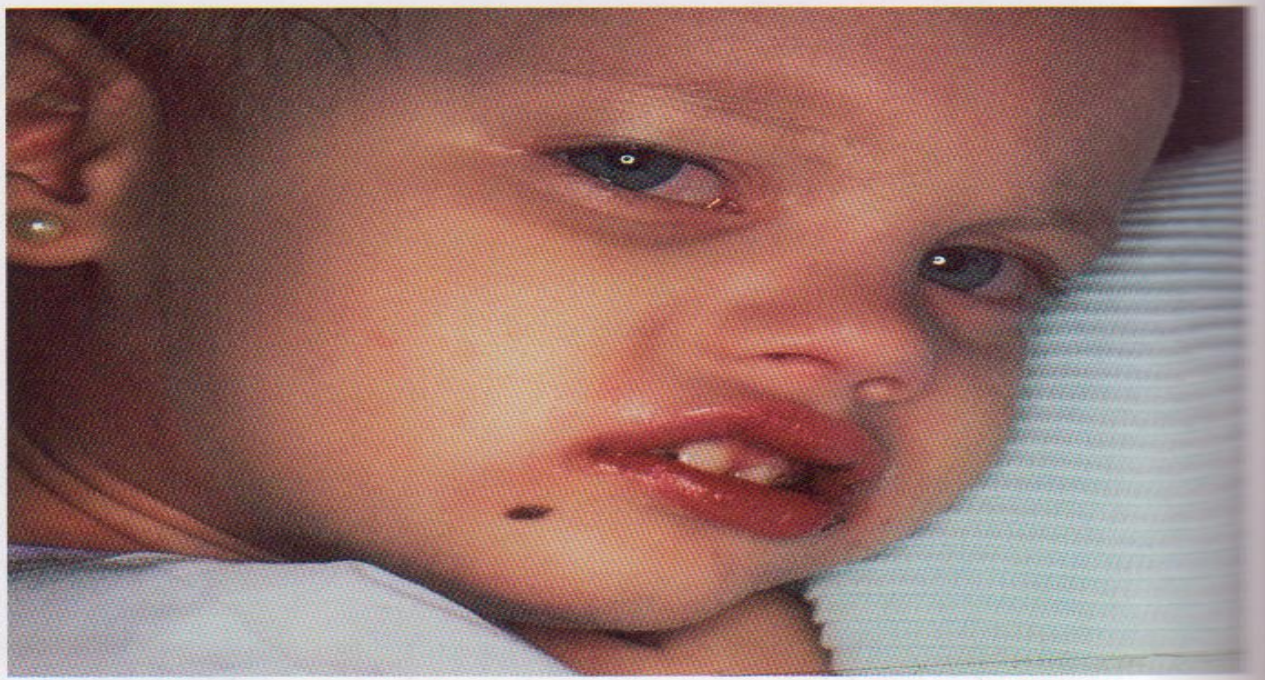


Figure 14-24
Kawasaki syndrome. Nonpurulent conjunctival injection and "cherry red" lips with fissuring and crusting are early signs of the disease. *(Courtesy Anne W. Lucky M.D.)*

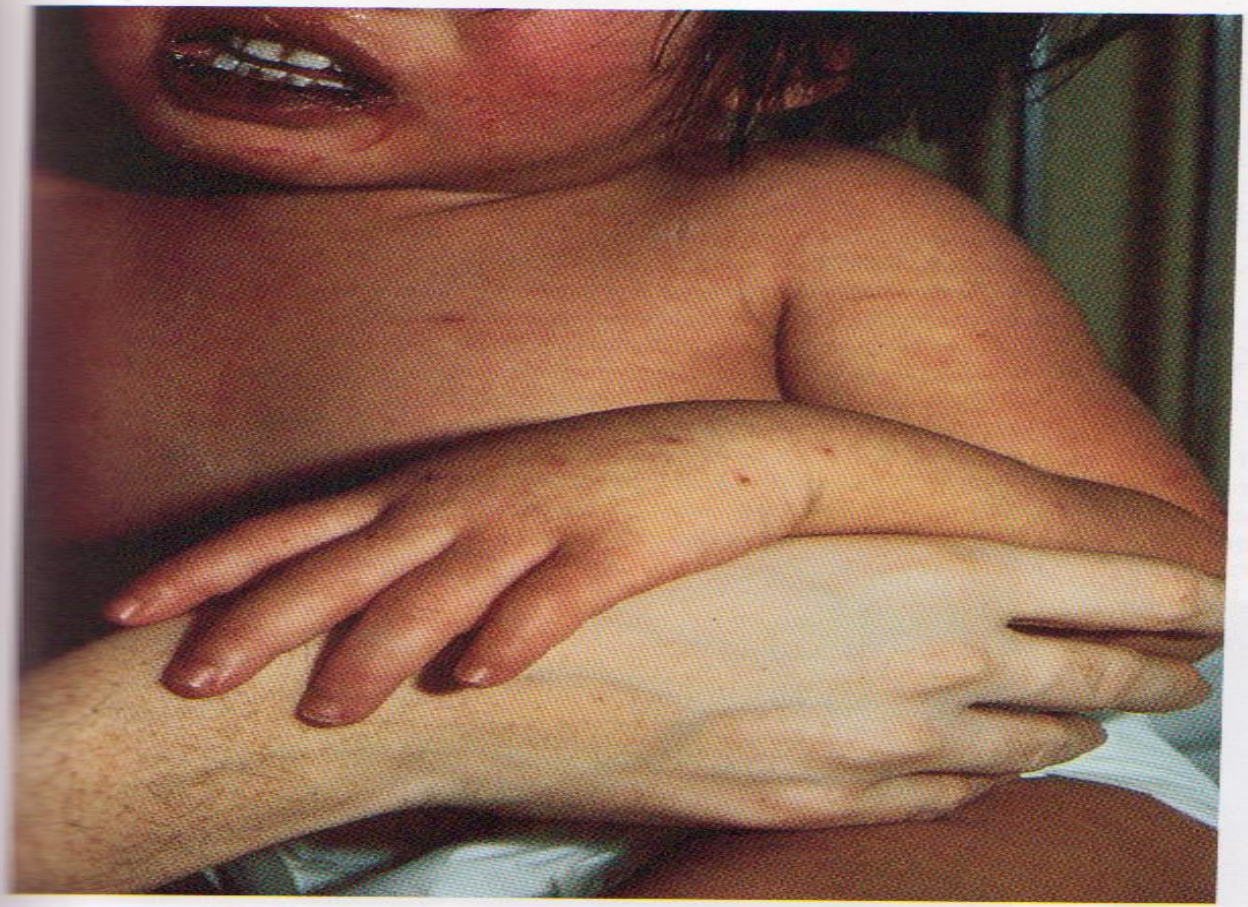
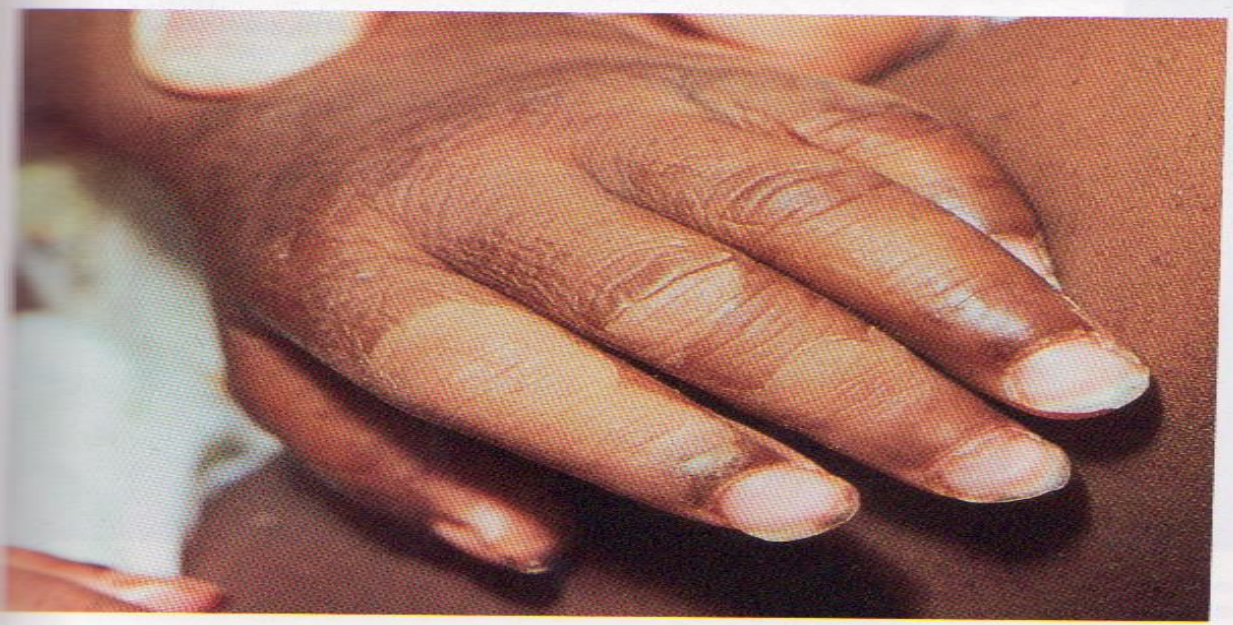


Figure 14-25 Kawasaki syndrome—Evolution of hand lesions. *(Courtesy Nancy B. Esterly M.D.)*

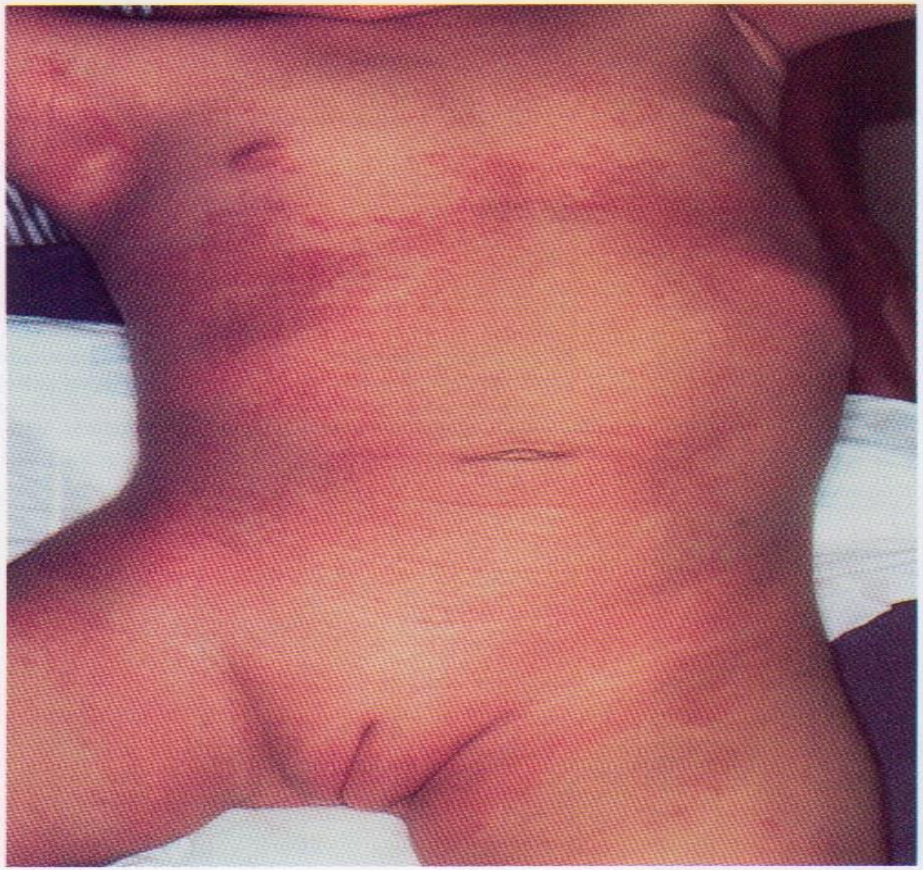
A, The hands become red and swollen.



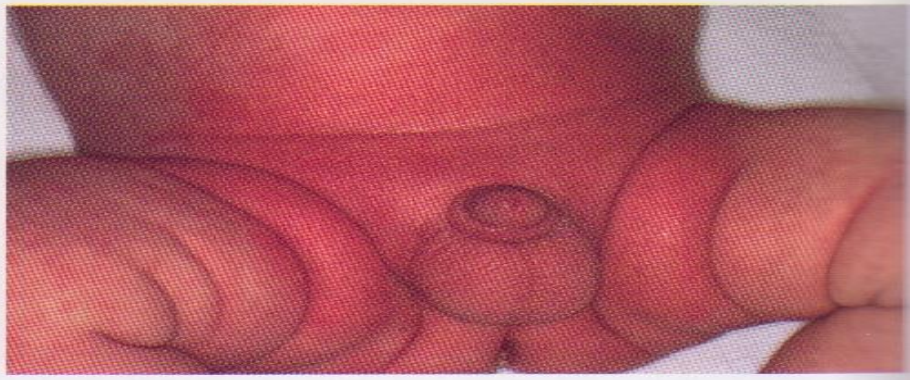
B, The hands peel approximately 2 weeks after the onset of fever.

Rash. A rash appears soon after the onset of fever. Several symptoms have been described. The most common forms are urticarial and a diffuse, deep red, maculopapular eruption (Figure 14-26, *A*). Less often the rash resembles erythema multiforme, scarlet fever, or the erythema marginatum seen in rheumatic fever. Dermatitis in the diaper area is common. The perineal rash usually occurs in the first week of the onset of symptoms. Red macules and papules become confluent (Figure 14-26, *B*). Desquamation occurs within 5 to 7 days. Perineal desquamation occurs 2 to 6 days before desquamation of the fingertips and toes (Figure 14-26, *C*). Vesiculopustules may develop over the elbows and knees.

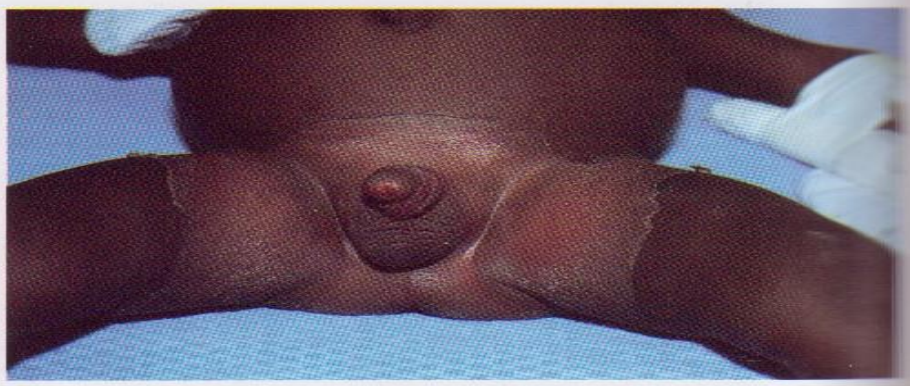
Figure 14-26 Kawasaki syndrome—erythematous exanthem rash.



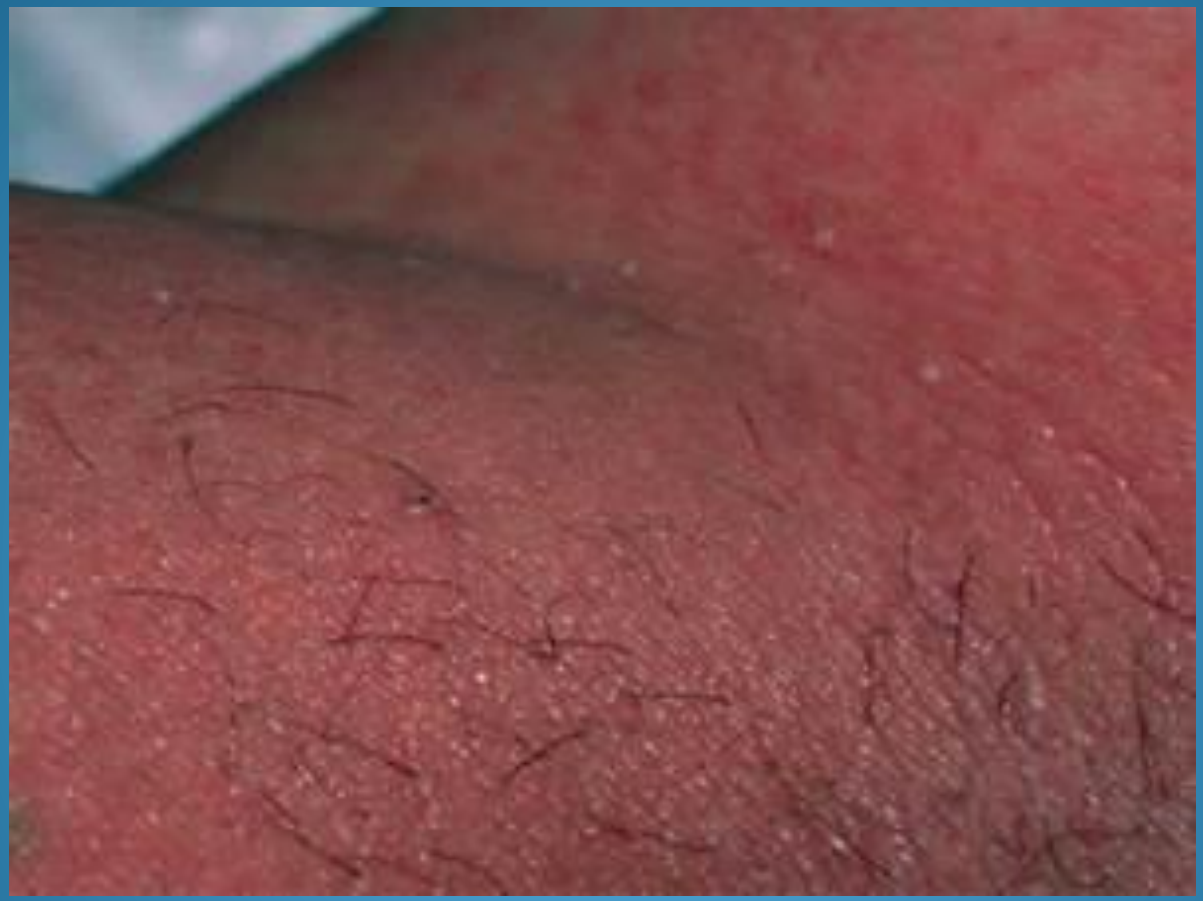
A, Diffuse, blanching, erythematous, macular exanthem. The eruption is frequently concentrated in the perineal area.



B, Red macules and papules appear in the perineal area 3 to 4 days after the onset of the illness. The rash becomes confluent and desquamates within 5 to 7 days. Desquamation of the fingertips and toes occurs 2 to 6 days later.



C, The skin of children with diaper-area inflammation peels at the margin of the rash. *(Courtesy Anne W. Lucky M.D.)*



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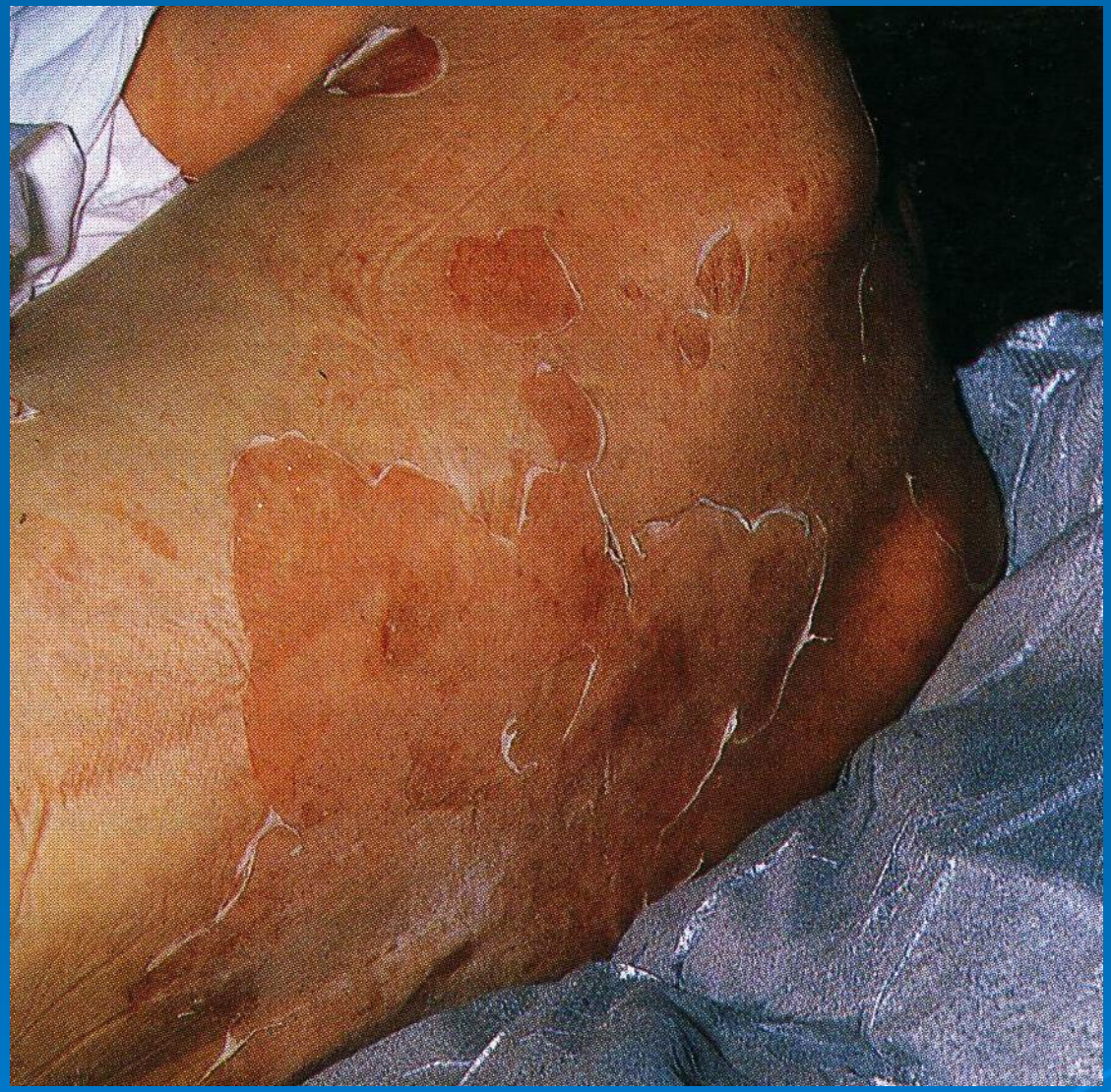
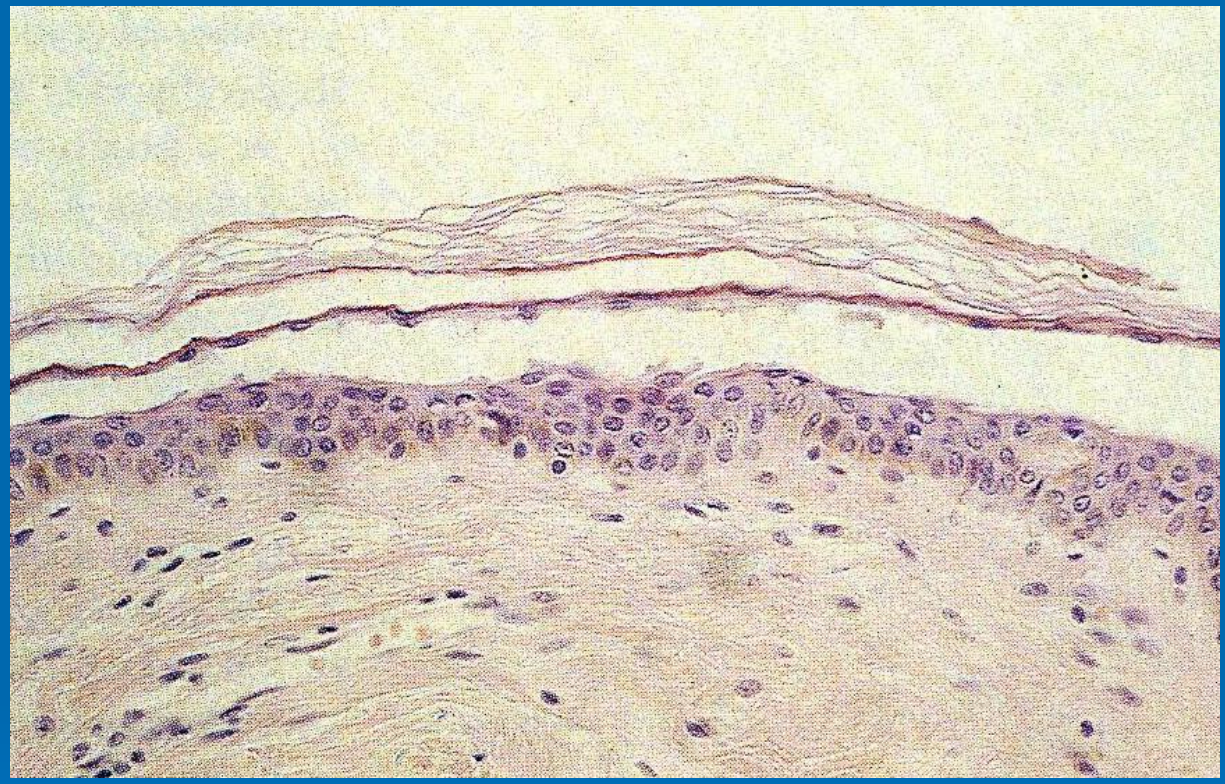




DRUG ERUPTION

Definition:

Incidence

Differential risk of adverse drug reactions amongst patient groups.

- Sex
- Age
- AIDS
- Sjogrenis Syndrome
- Route of administration

Classification and Mechanism of Drug Reactions

-Non immunological
- Predictable
- Unpredictable

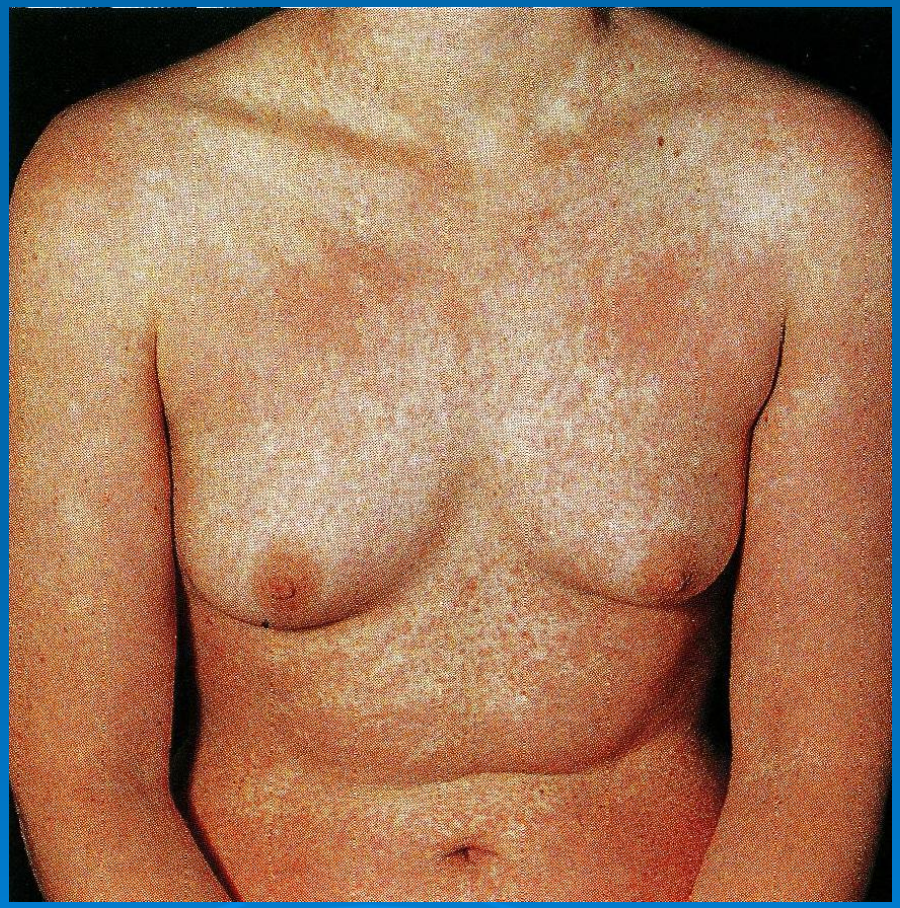
-Immunological
- Unpedictable

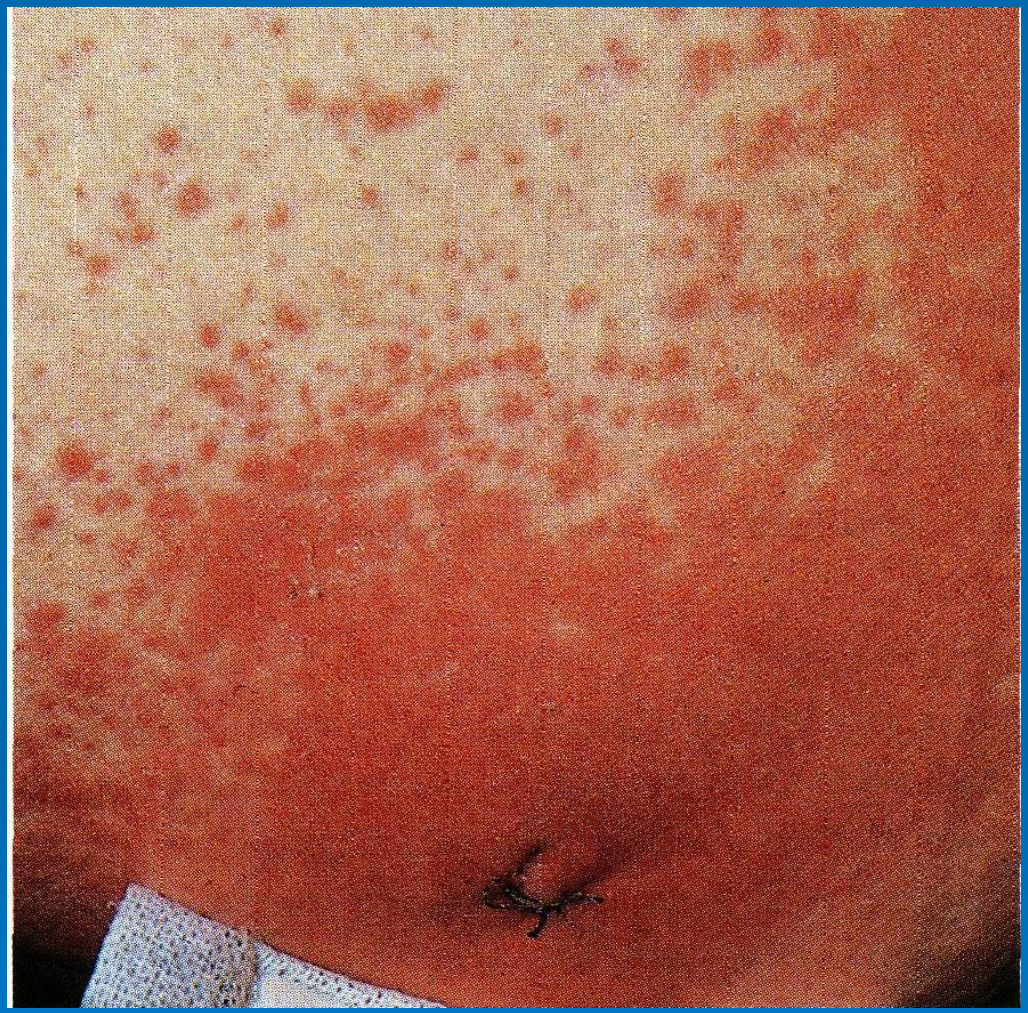
Steps in the Approach to a Suspected Adverse Drug reaction

1. Clinical Diagnosis
2. Analysis of Drug exposure
3. Differential diagnosis
4. Literature Search
5. Confirmation
6. Advice to the patient
7. Reporting to licensing authorities and/ or manufacturer

Types of Clinical Reaction

1. Exanthematous (Maculopapular) – the most frequent

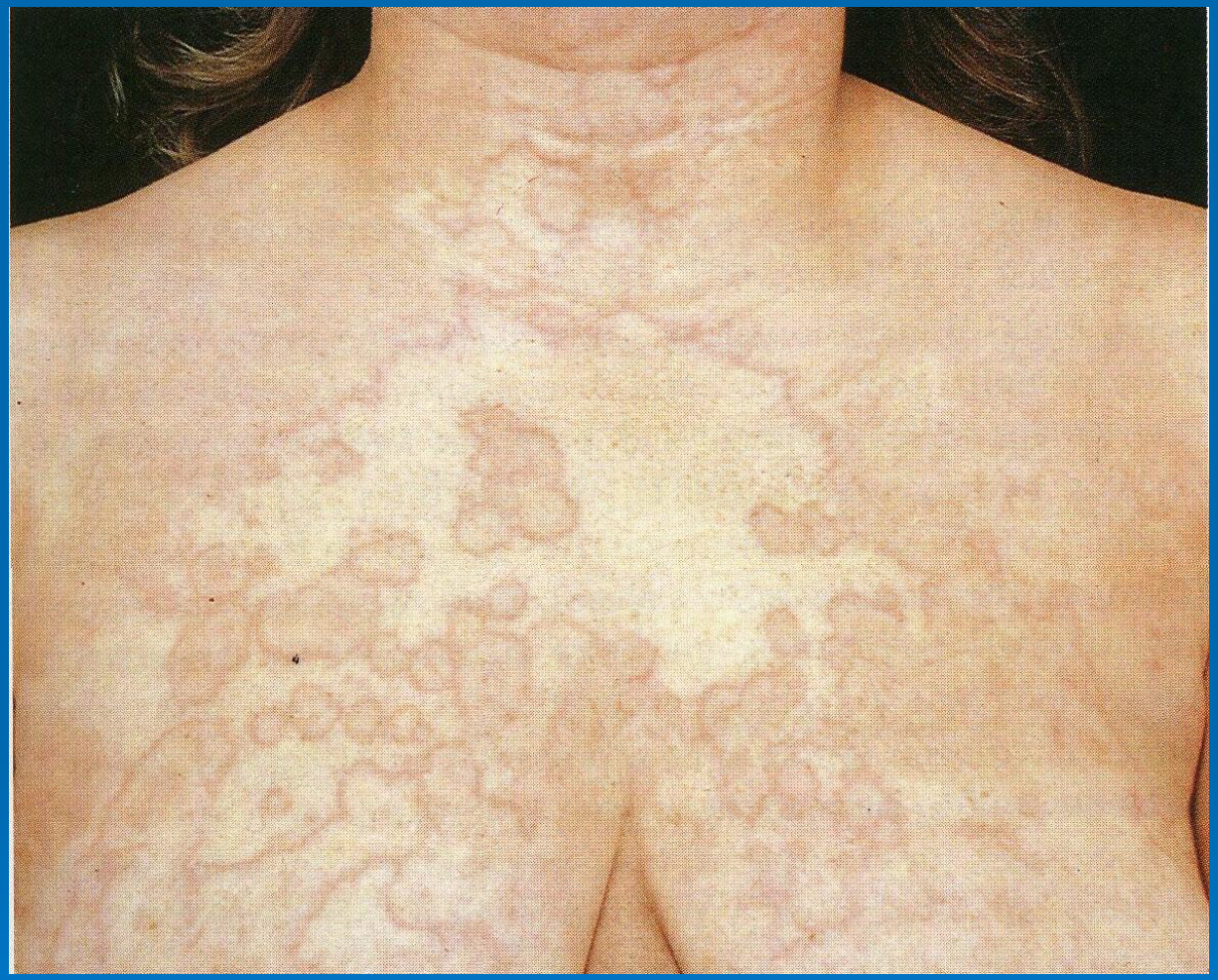


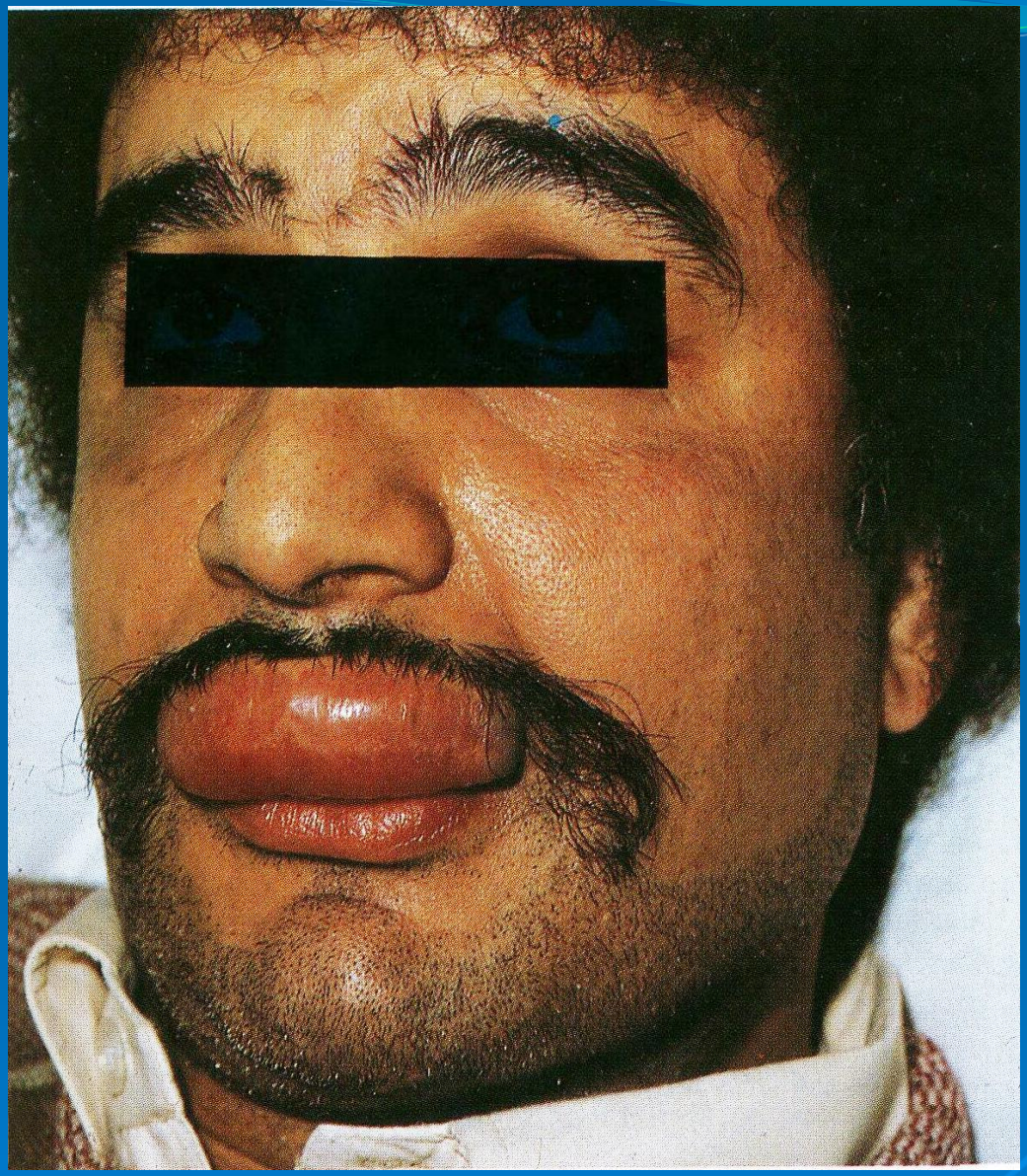


2. Hypersensitivity Syndrome Reaction

- Triad of fever, skin eruption and internal organ involvement.
- Potentially life threatening syndrome
- First exposure
- Anticonvulsants, sulfonamide, dapsone, allopurinol - most frequently associated with HSR
- Other Drugs : Azathioprine, Minocycline

3. Urticaria, Angioedema and Serum sickness





4. Latex Allergy

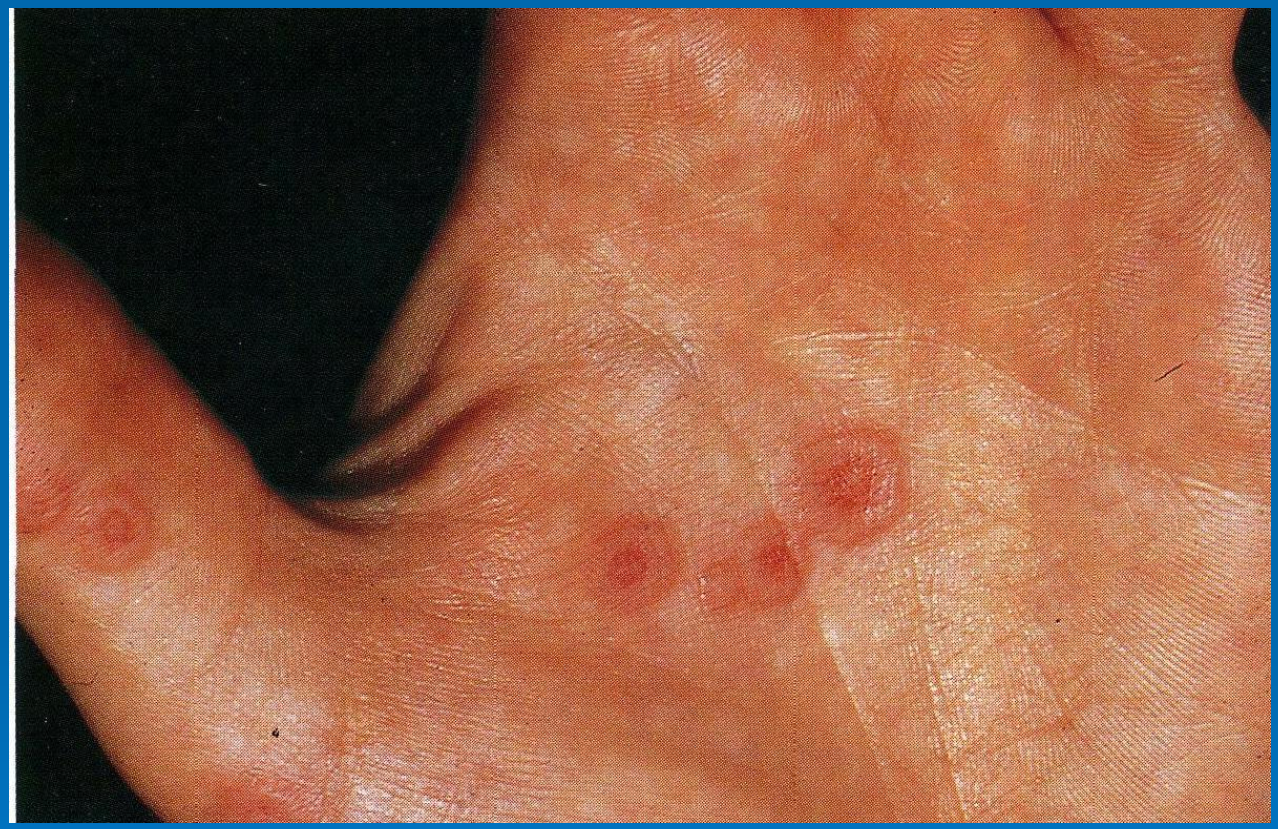
- Typely reaction to natural rubber latex proteins

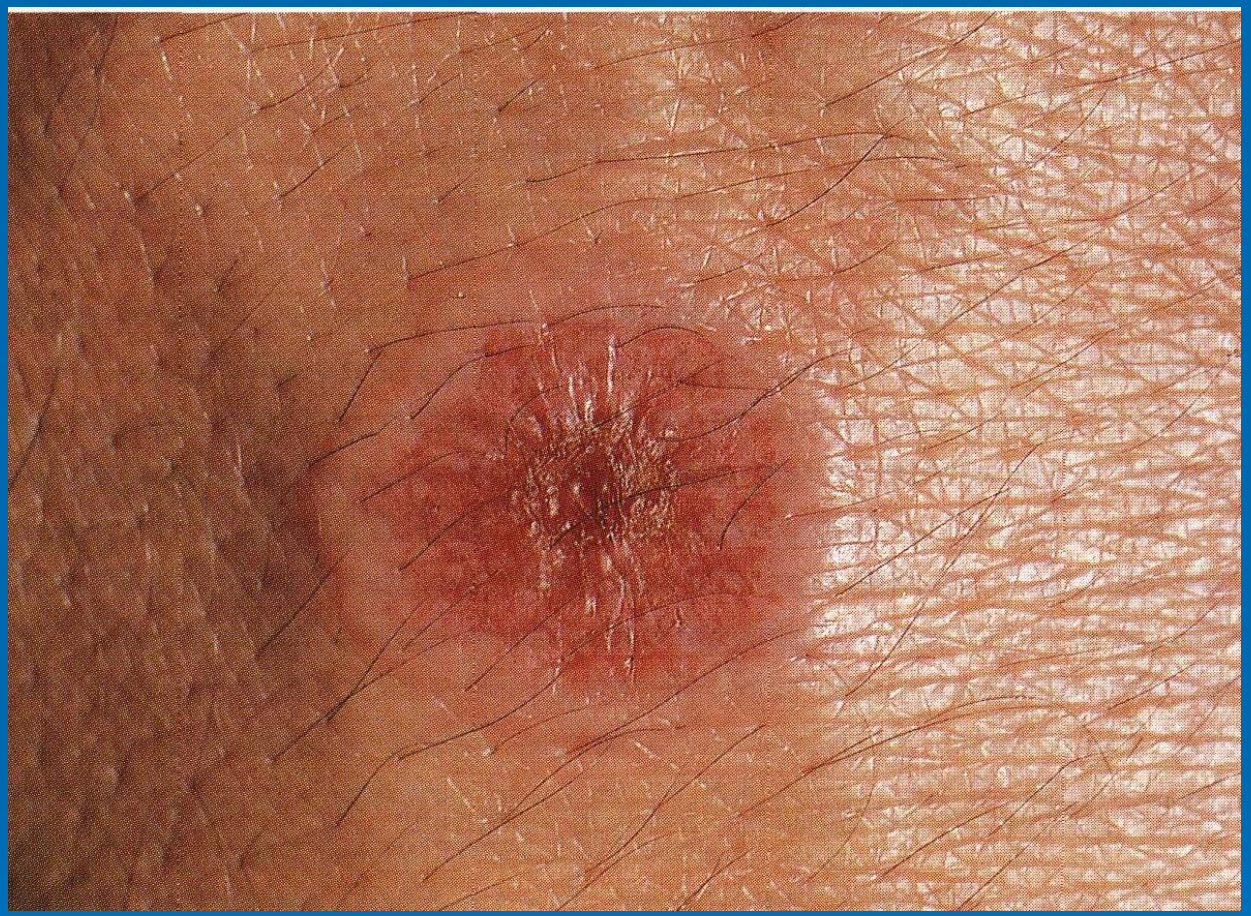
•Clinical Manifestations – Contact Urticaria, Fatal Anaphylaxis

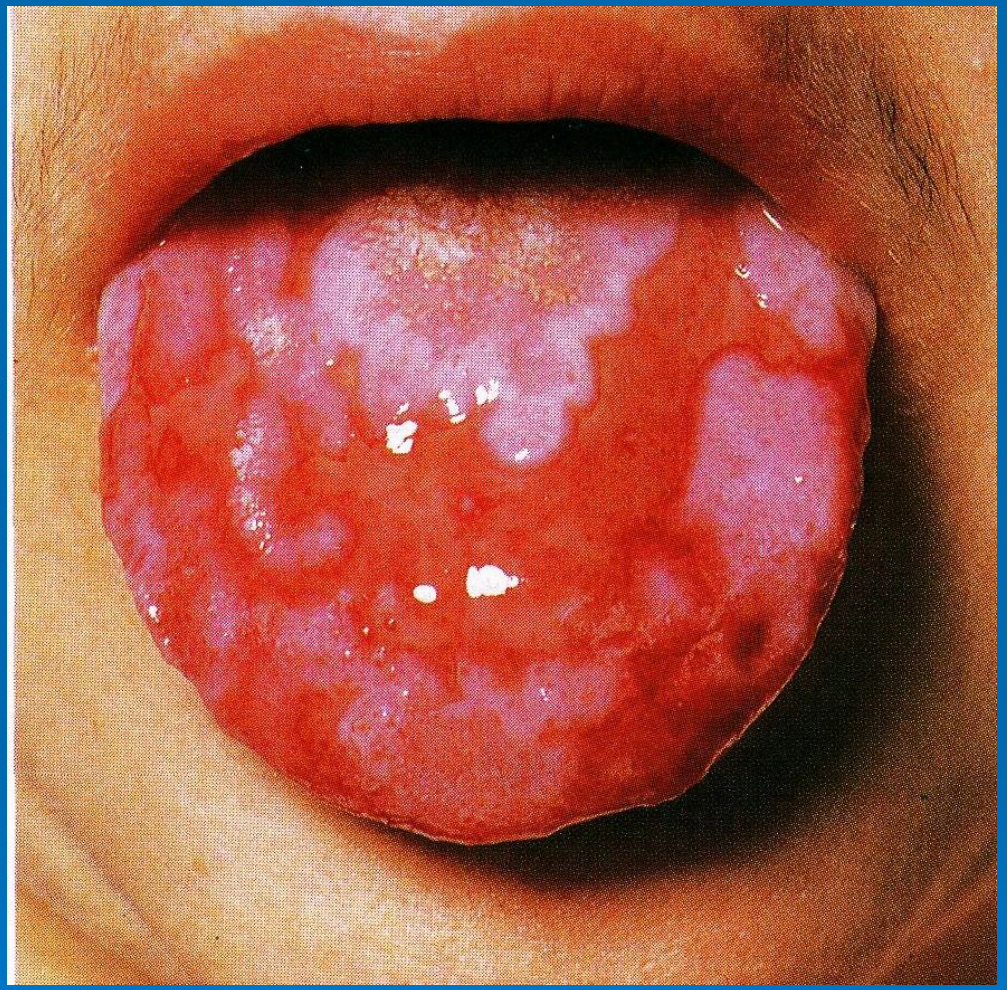
•Foods that cross react with latex proteins : Banana, Kiwi, Avocado, Chestnuts

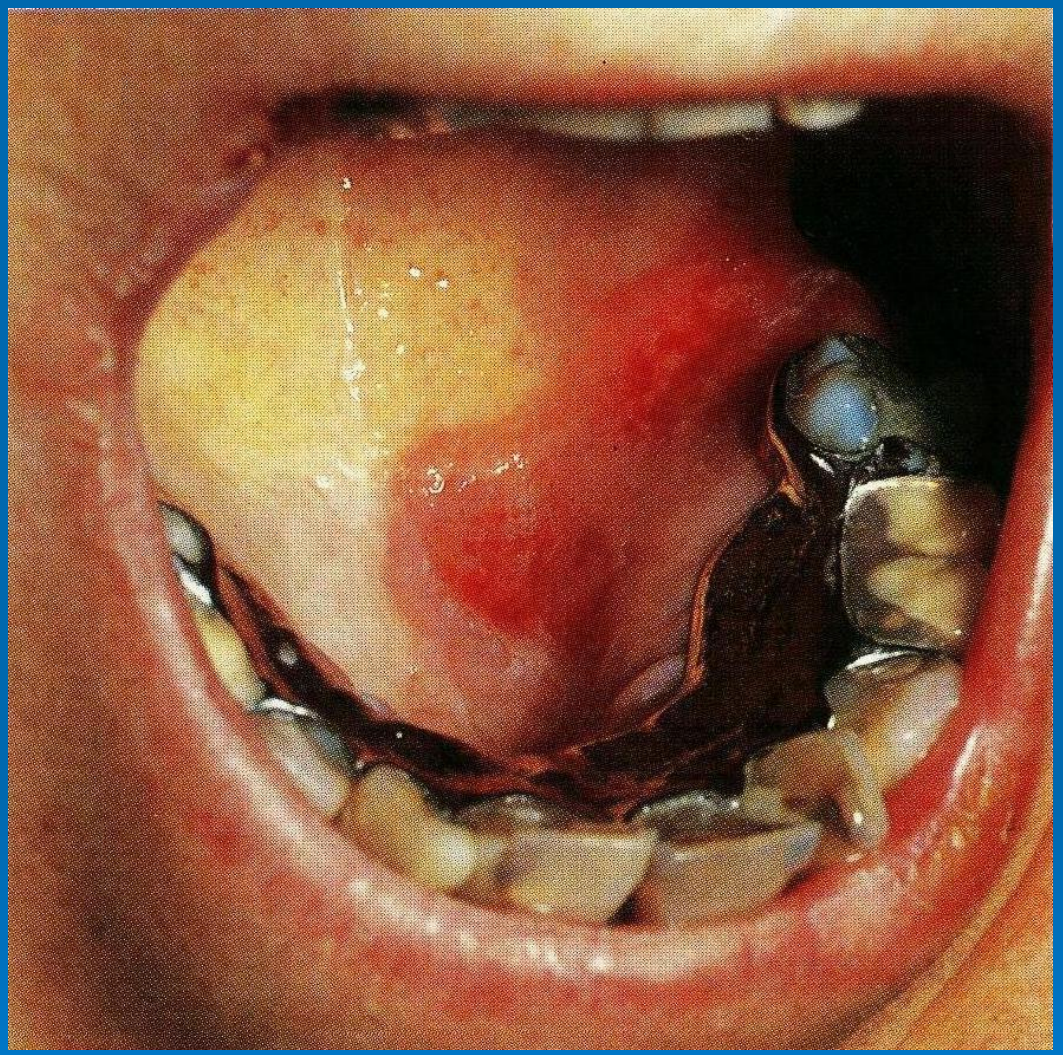
5. Anaphylaxis and Anaphylactoid reactions

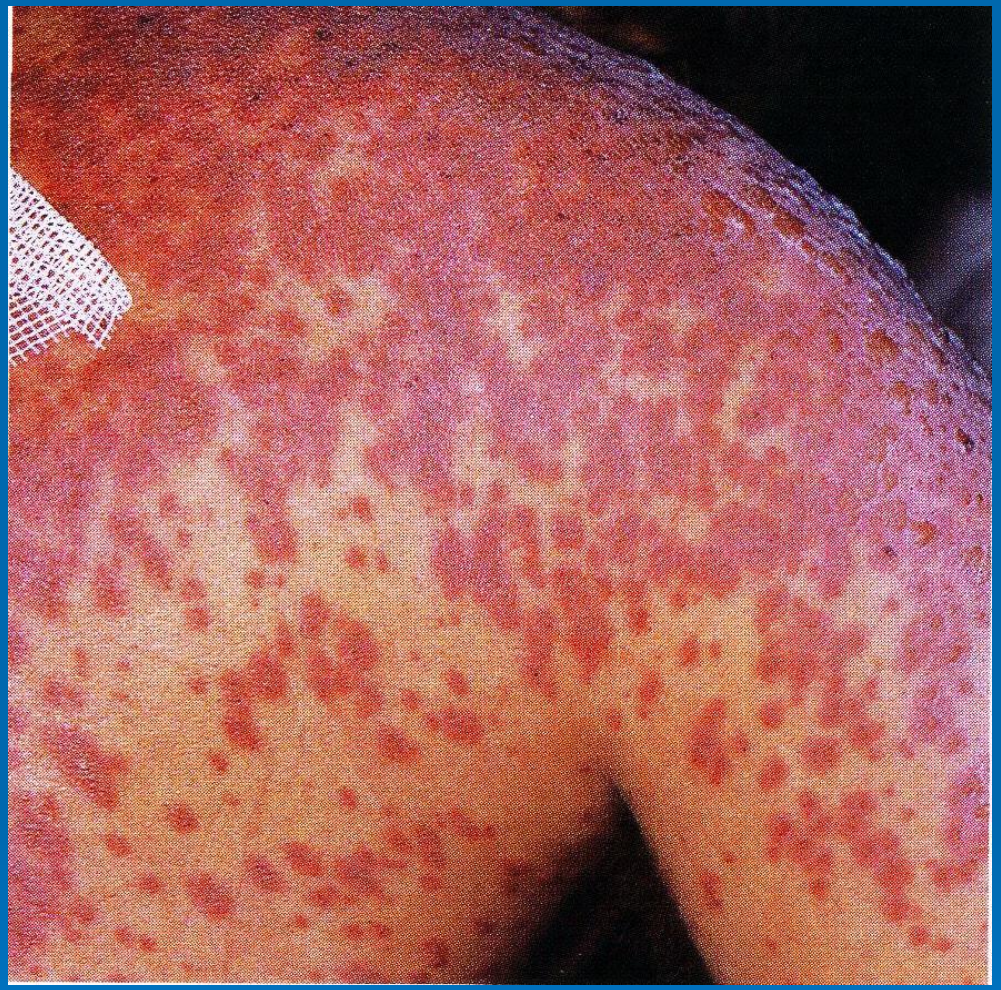
6. Erythema Multiforme and the Steven Johnson's Syndrome

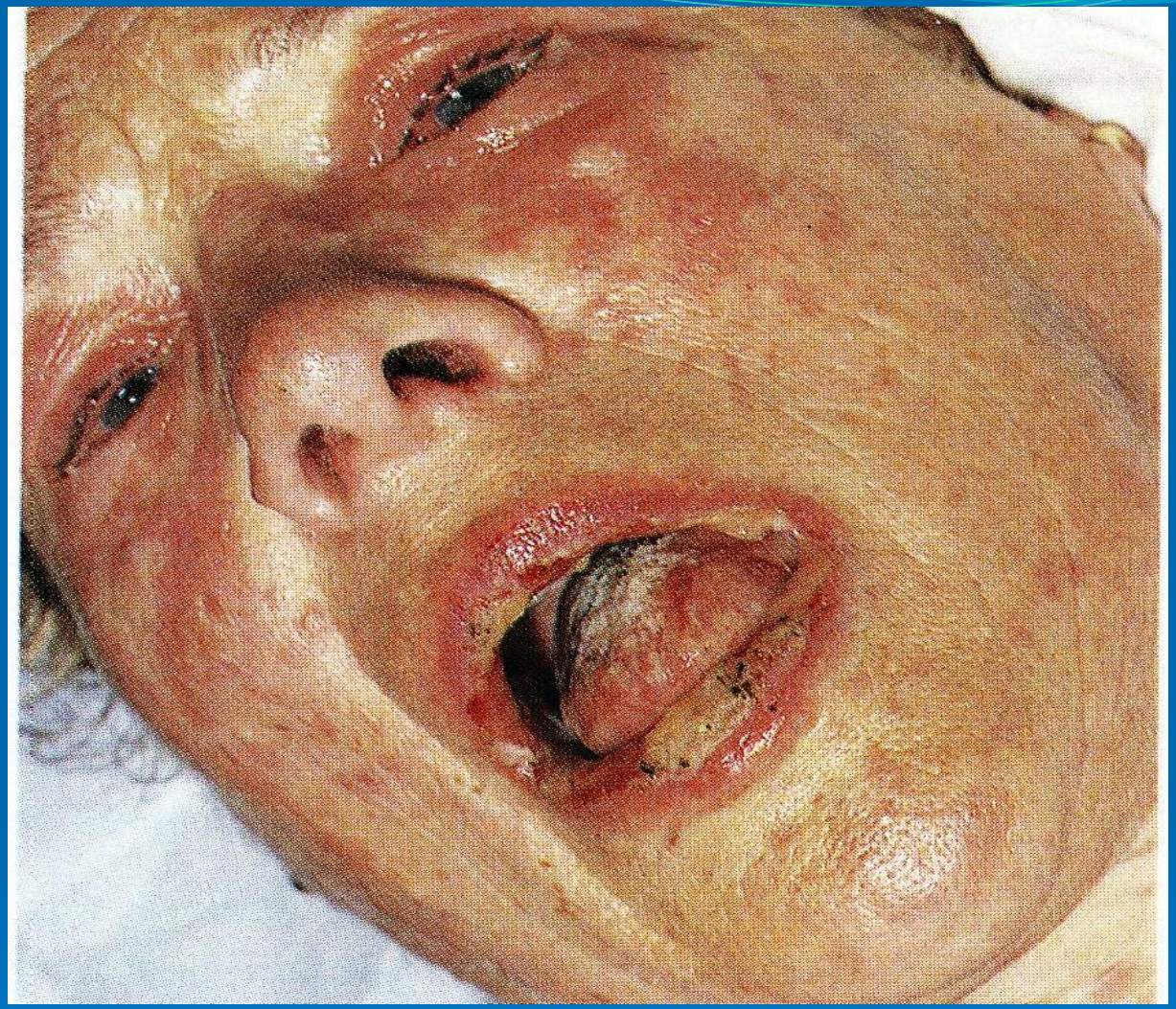


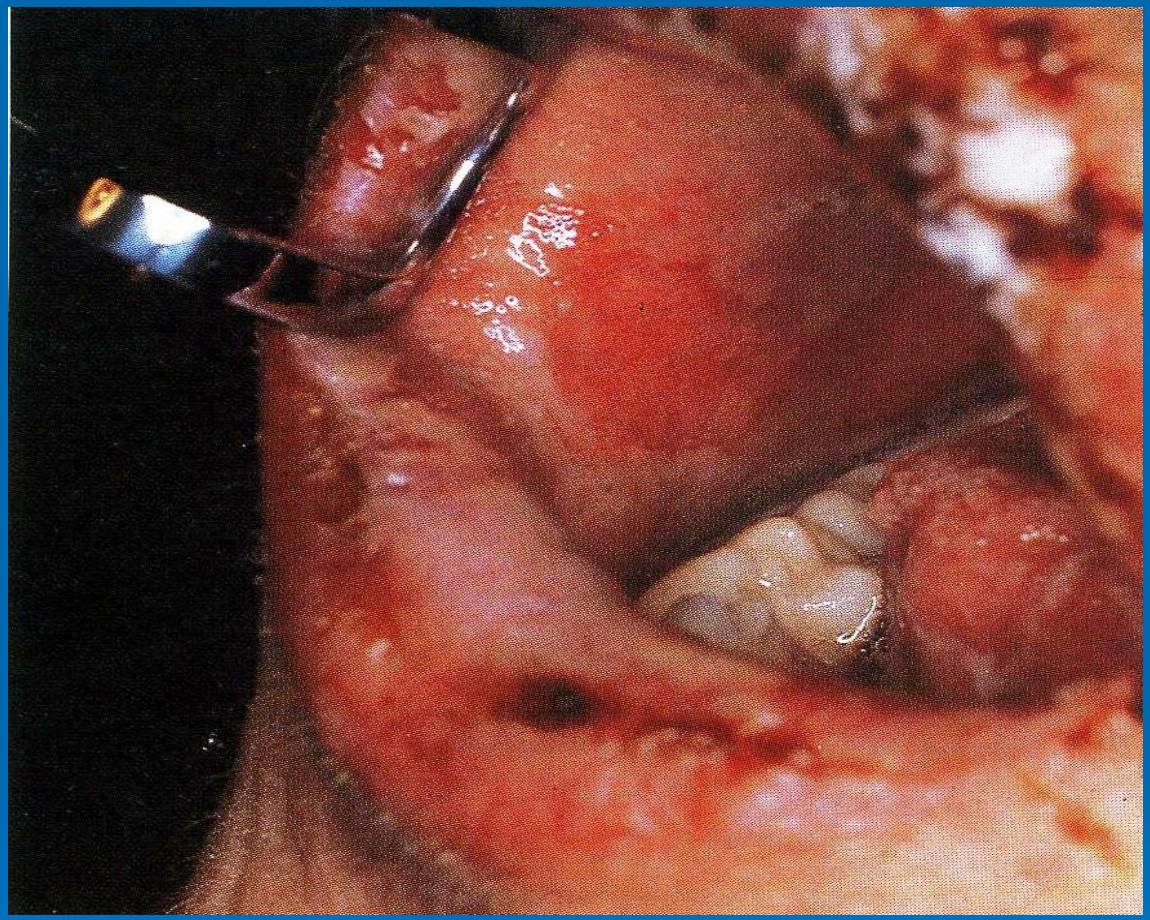




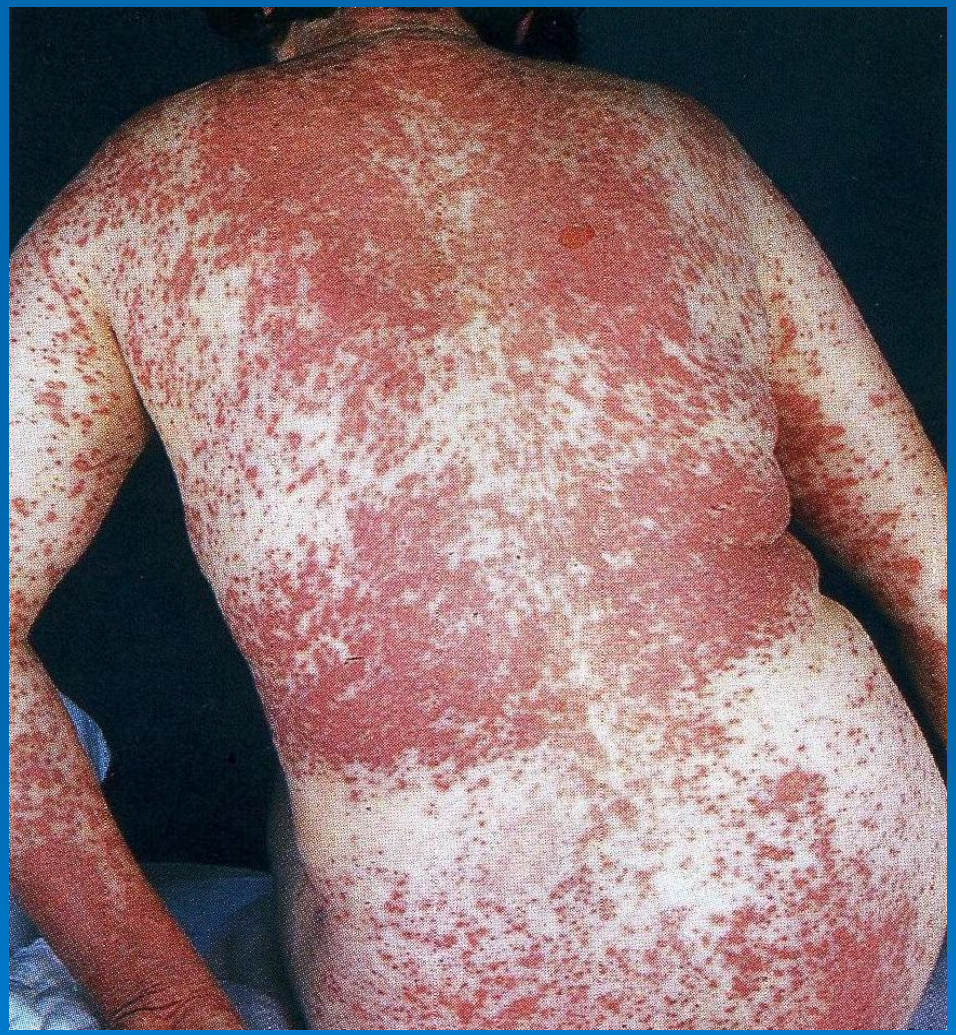


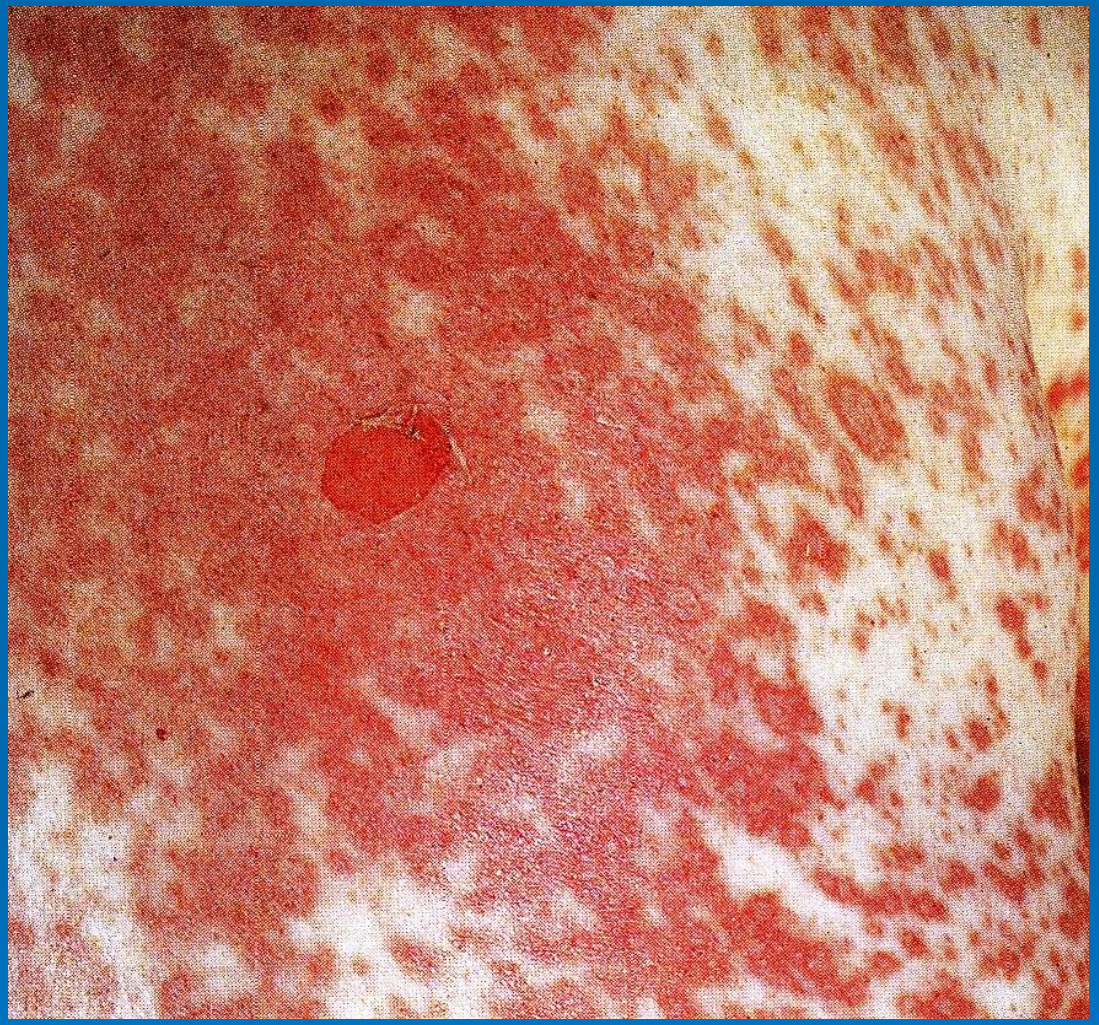


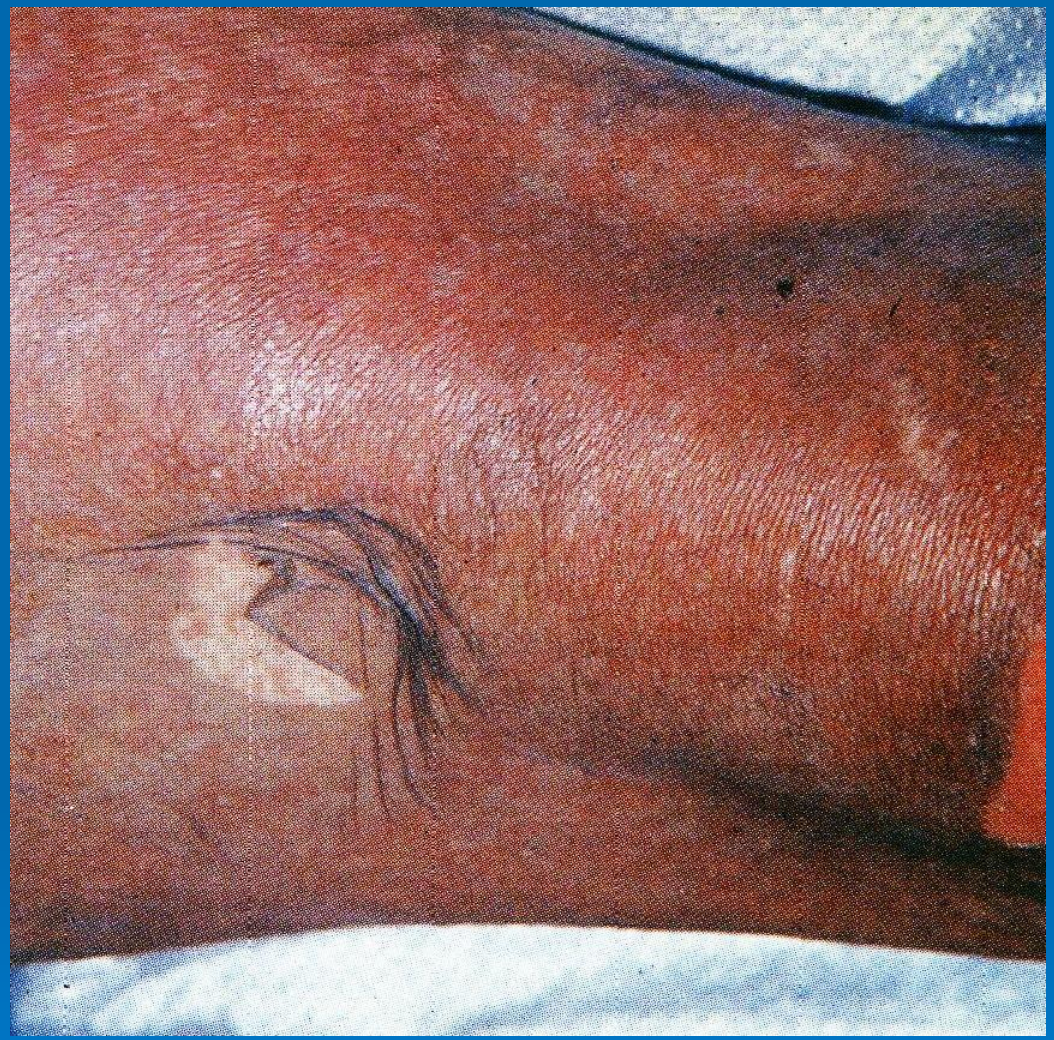


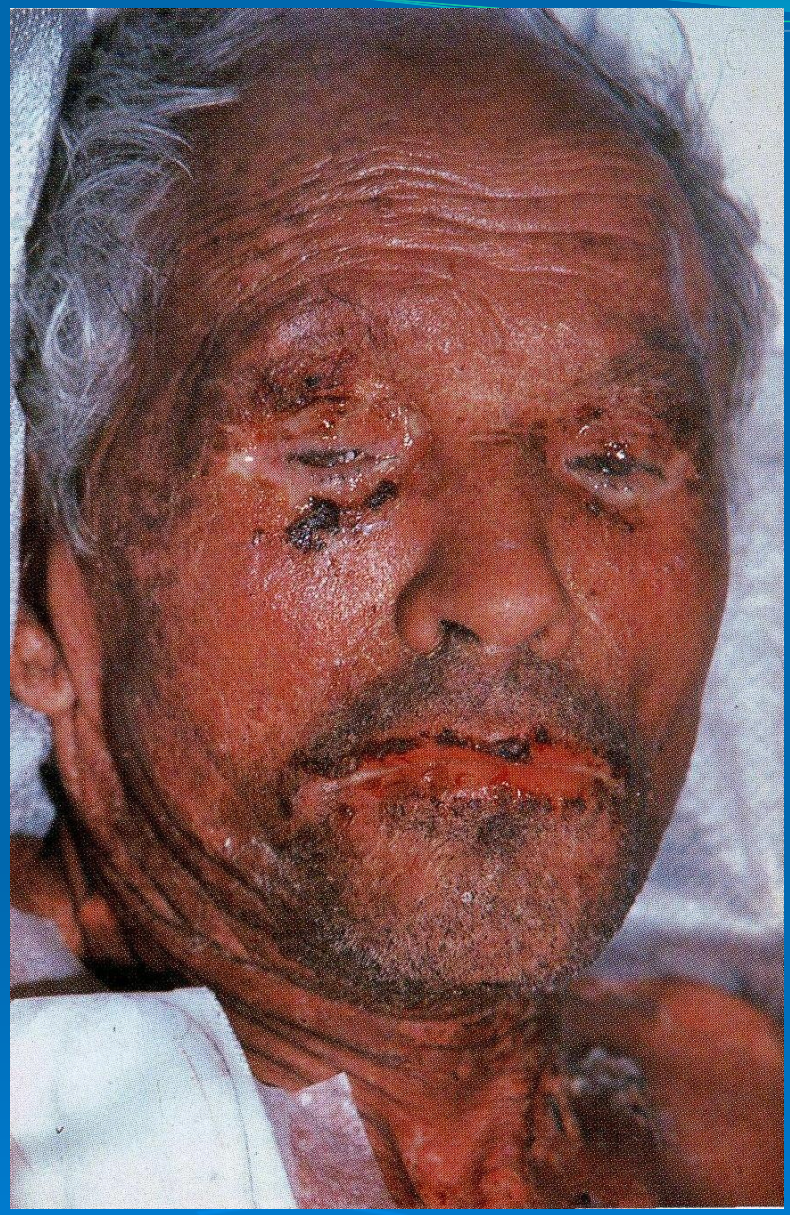


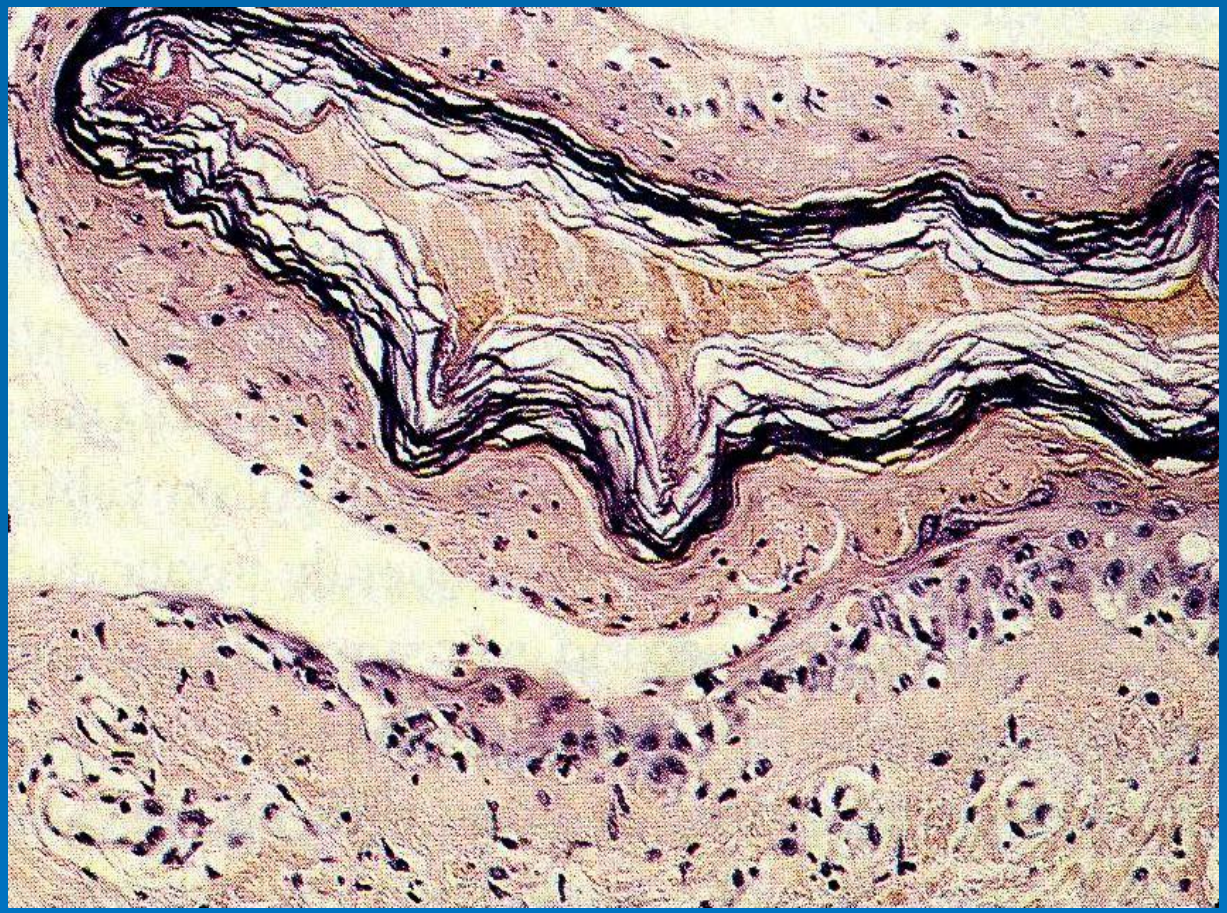
7. Toxic Epidermal Necrolysis



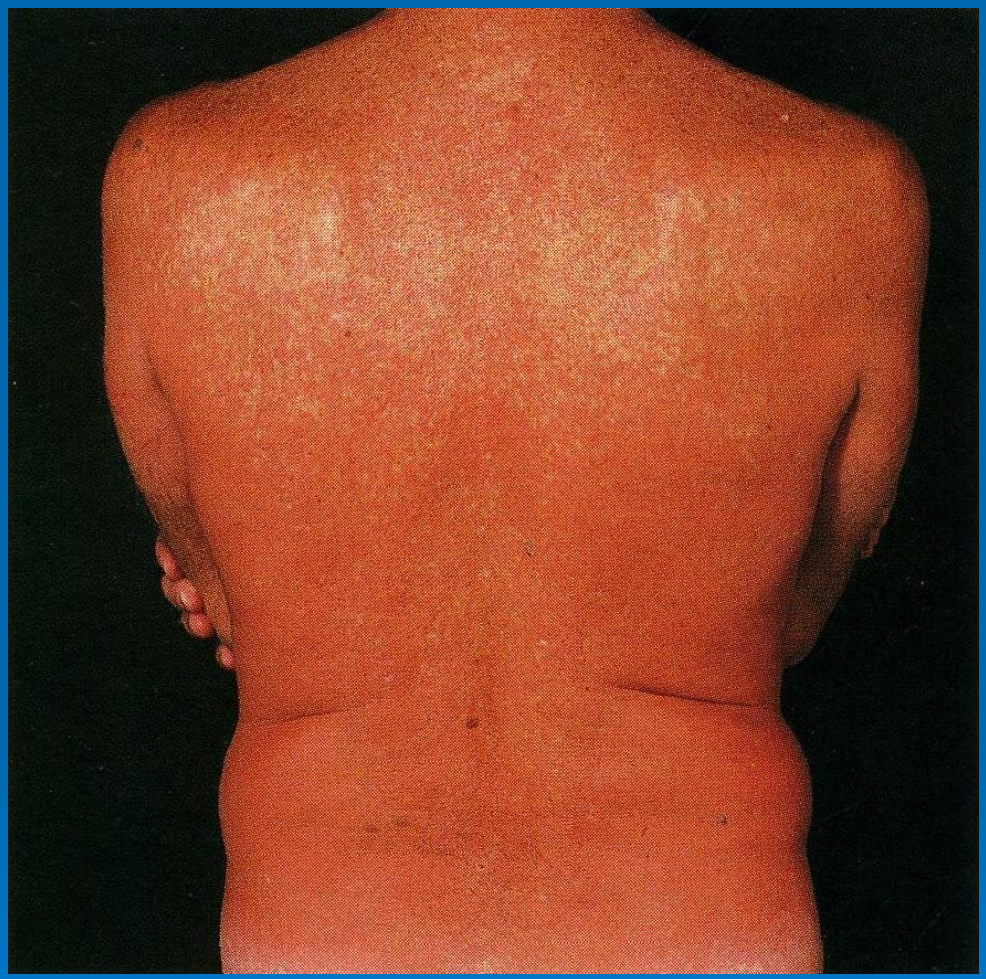


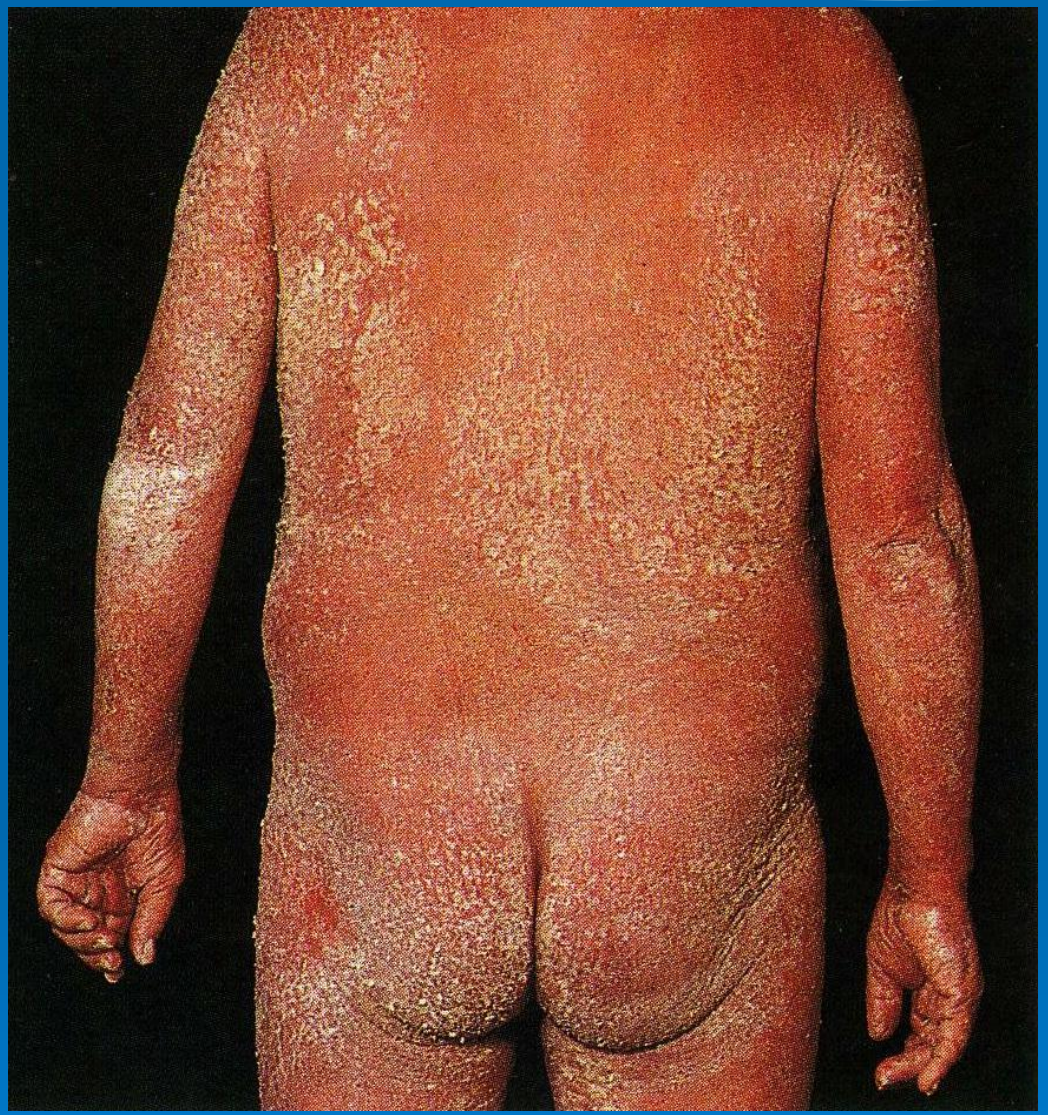




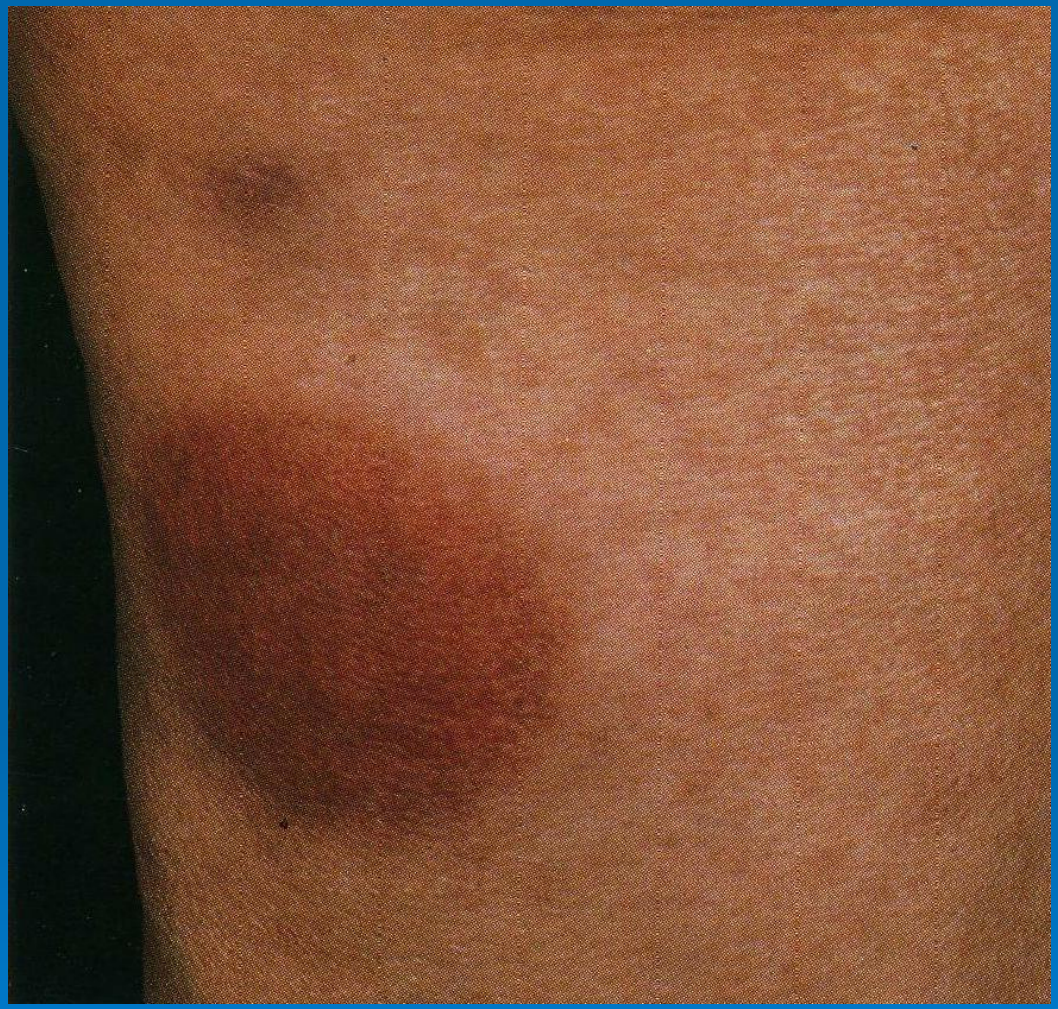


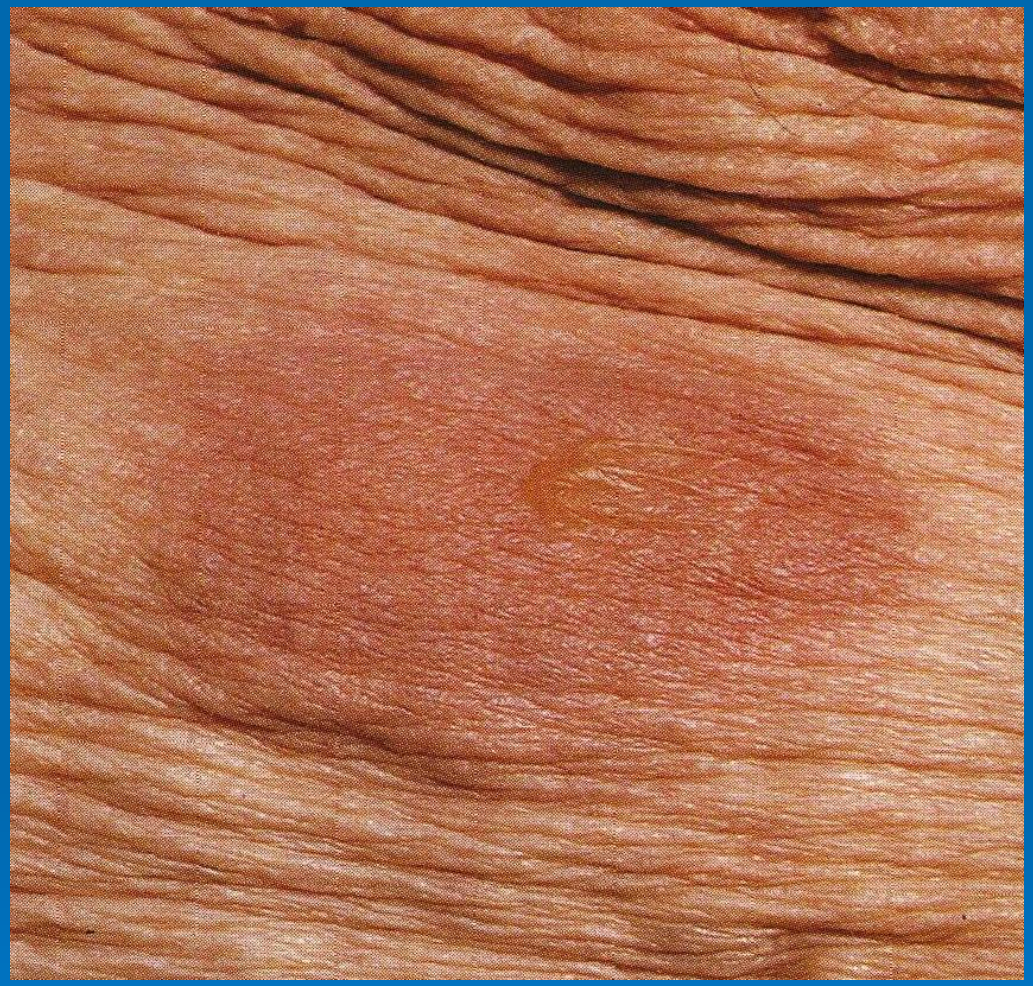
8. Erythroderma and Expoliative Dermatitis

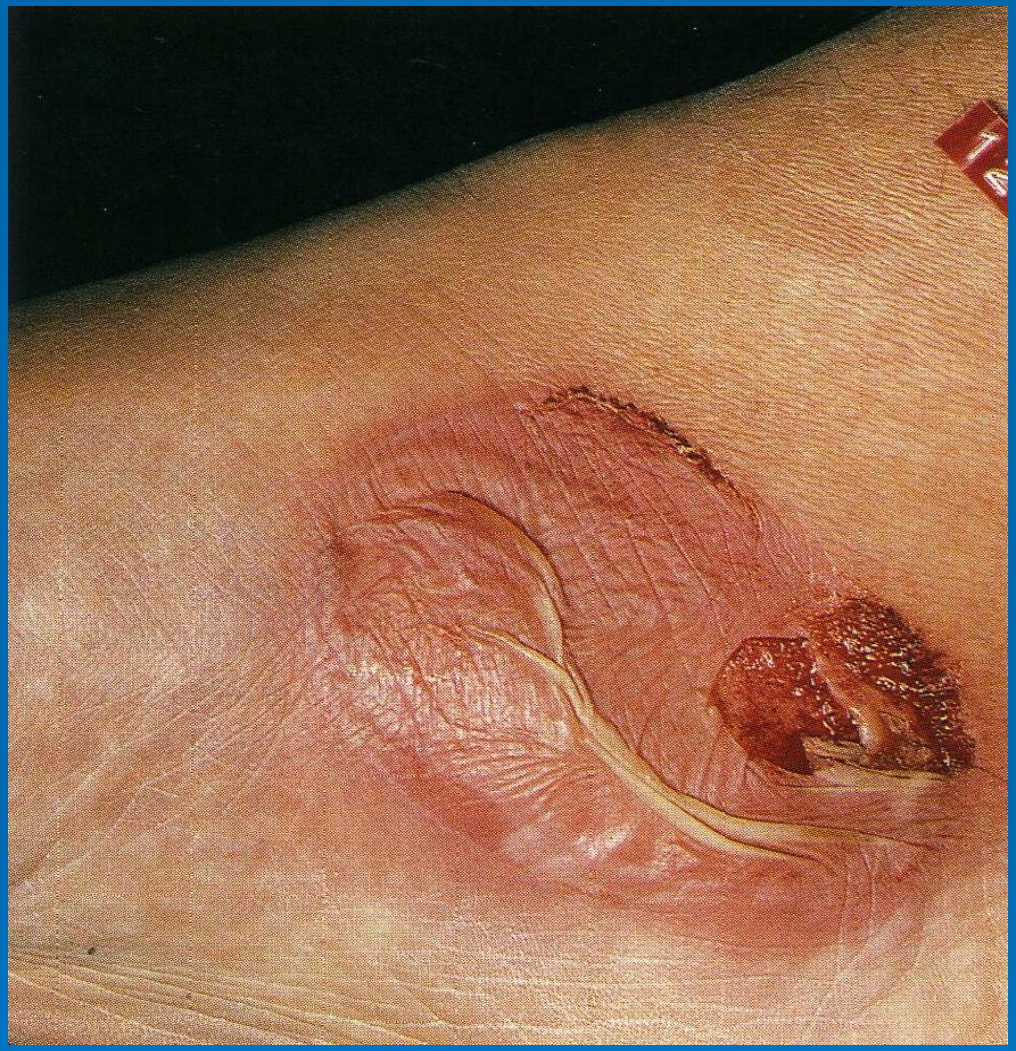


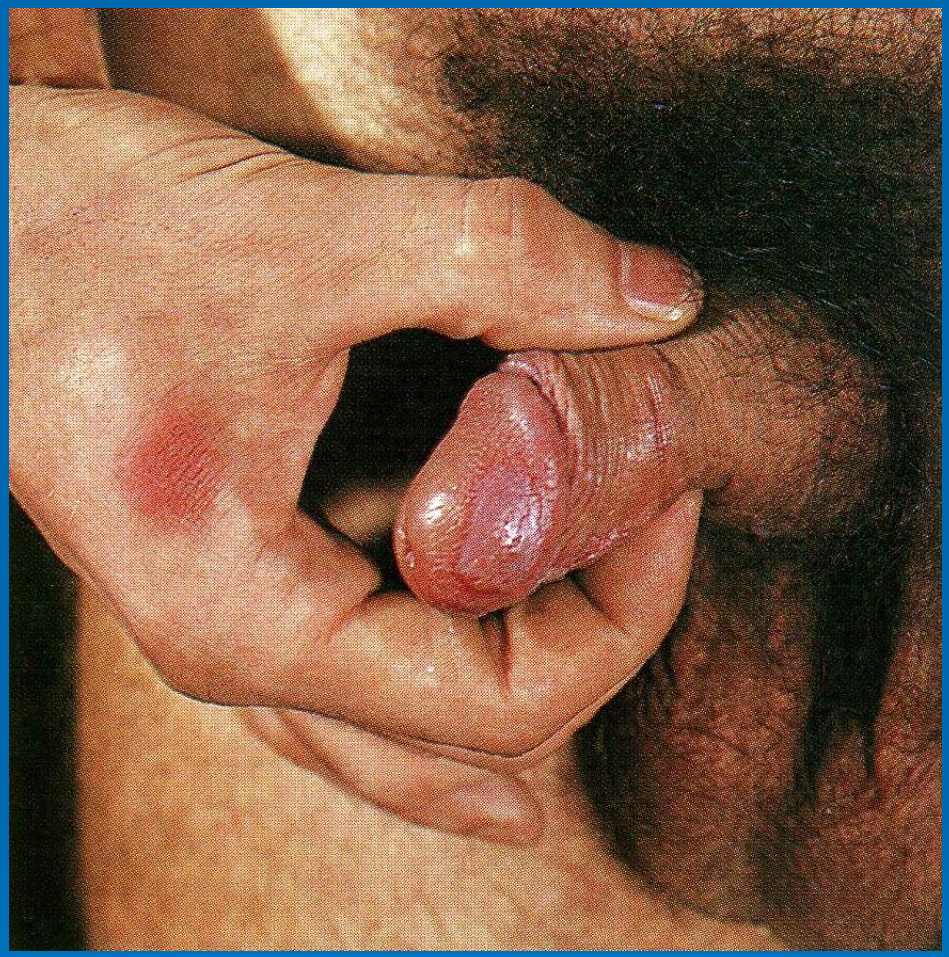


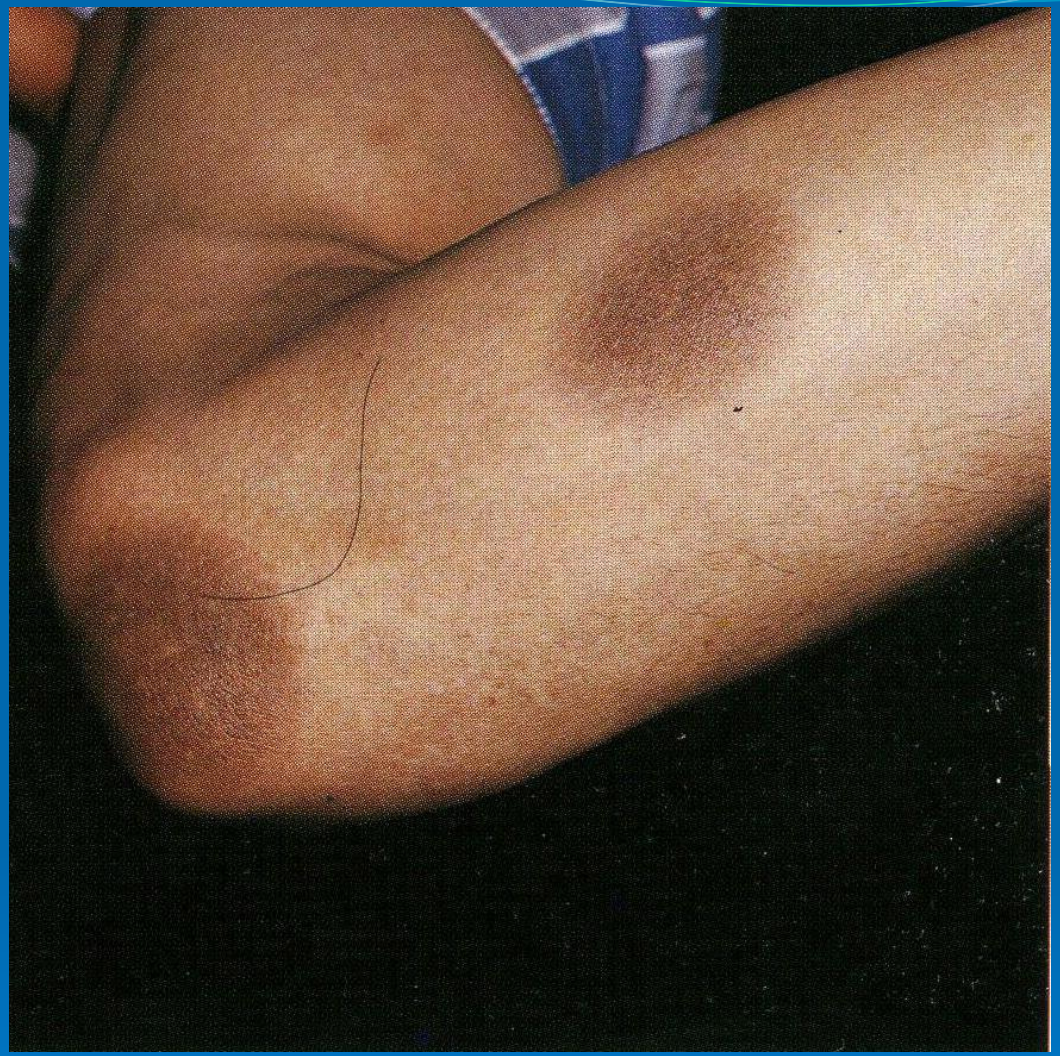
9. Fixed Drug Eruptions



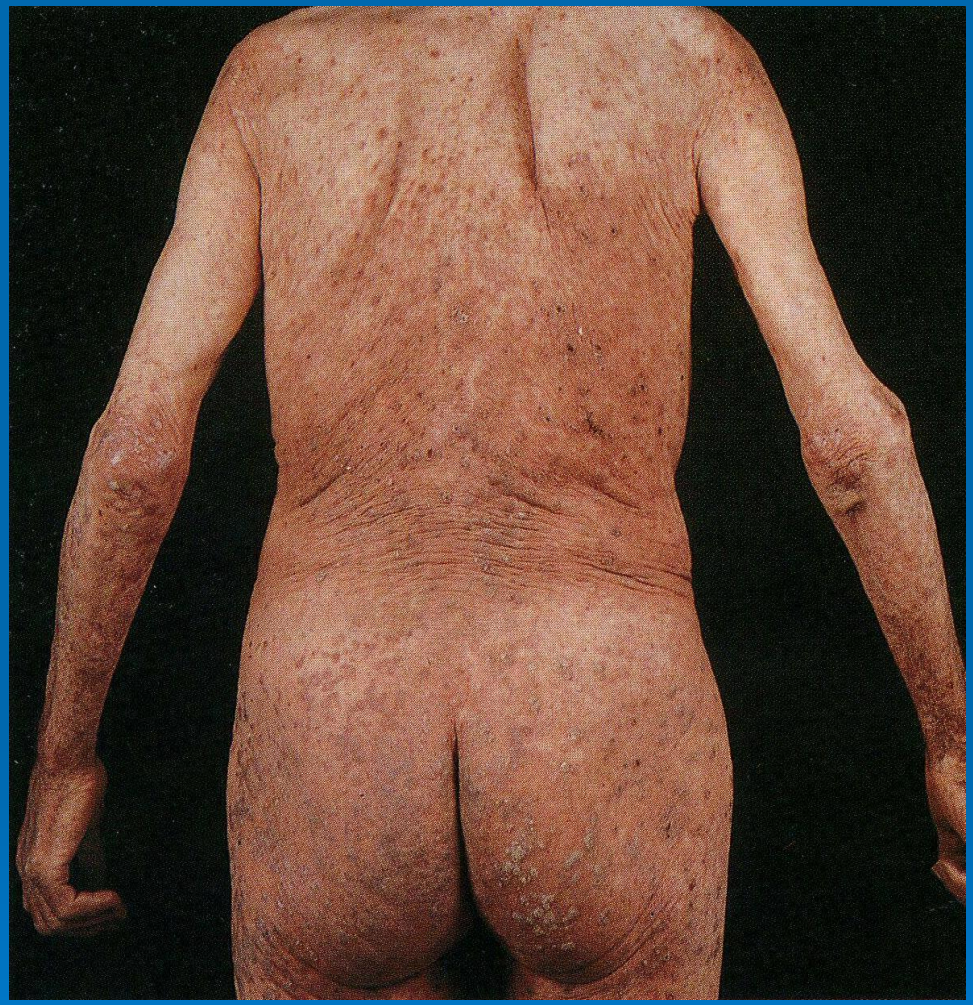


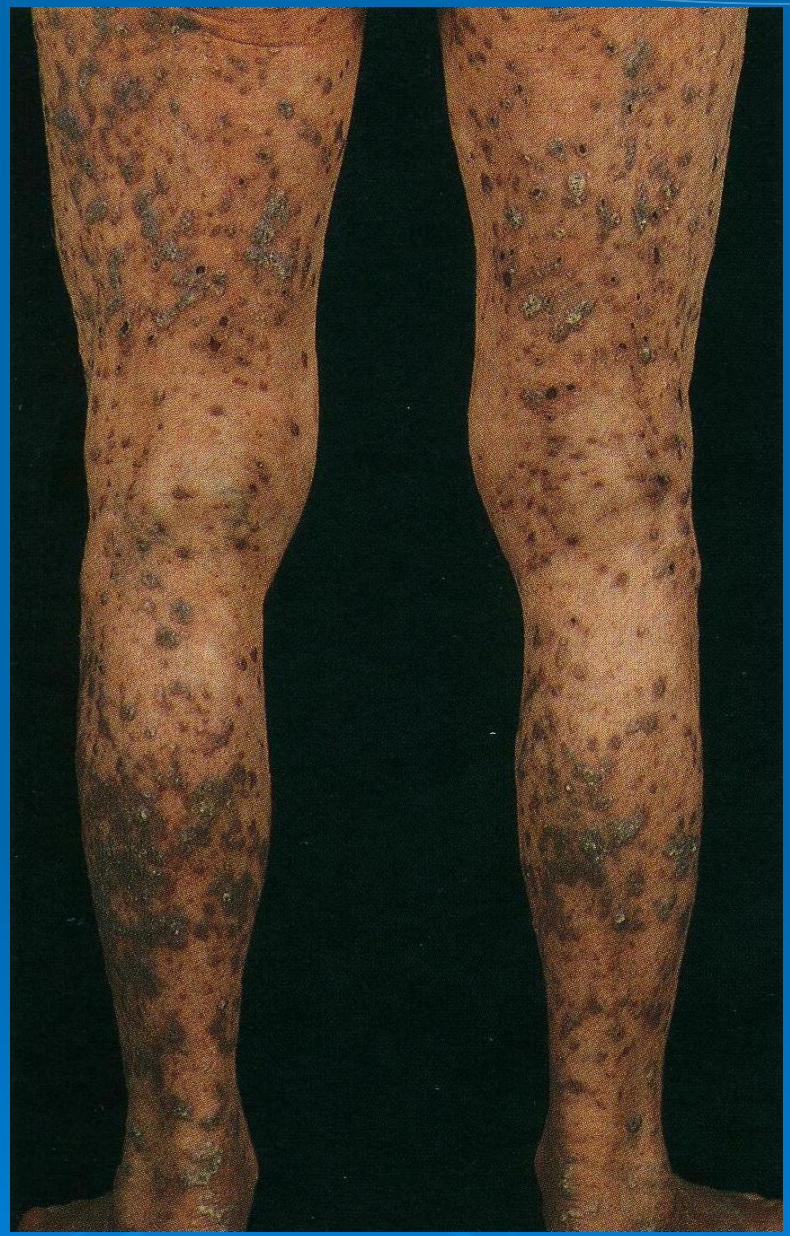


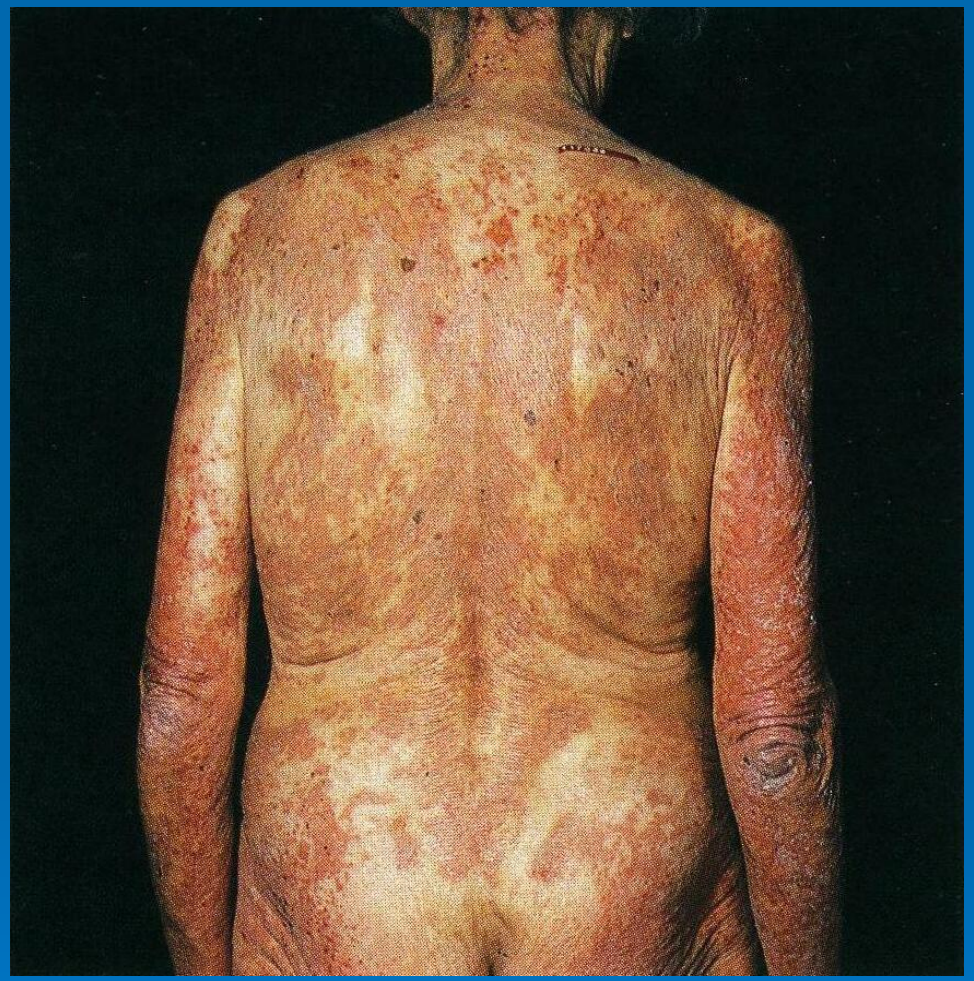


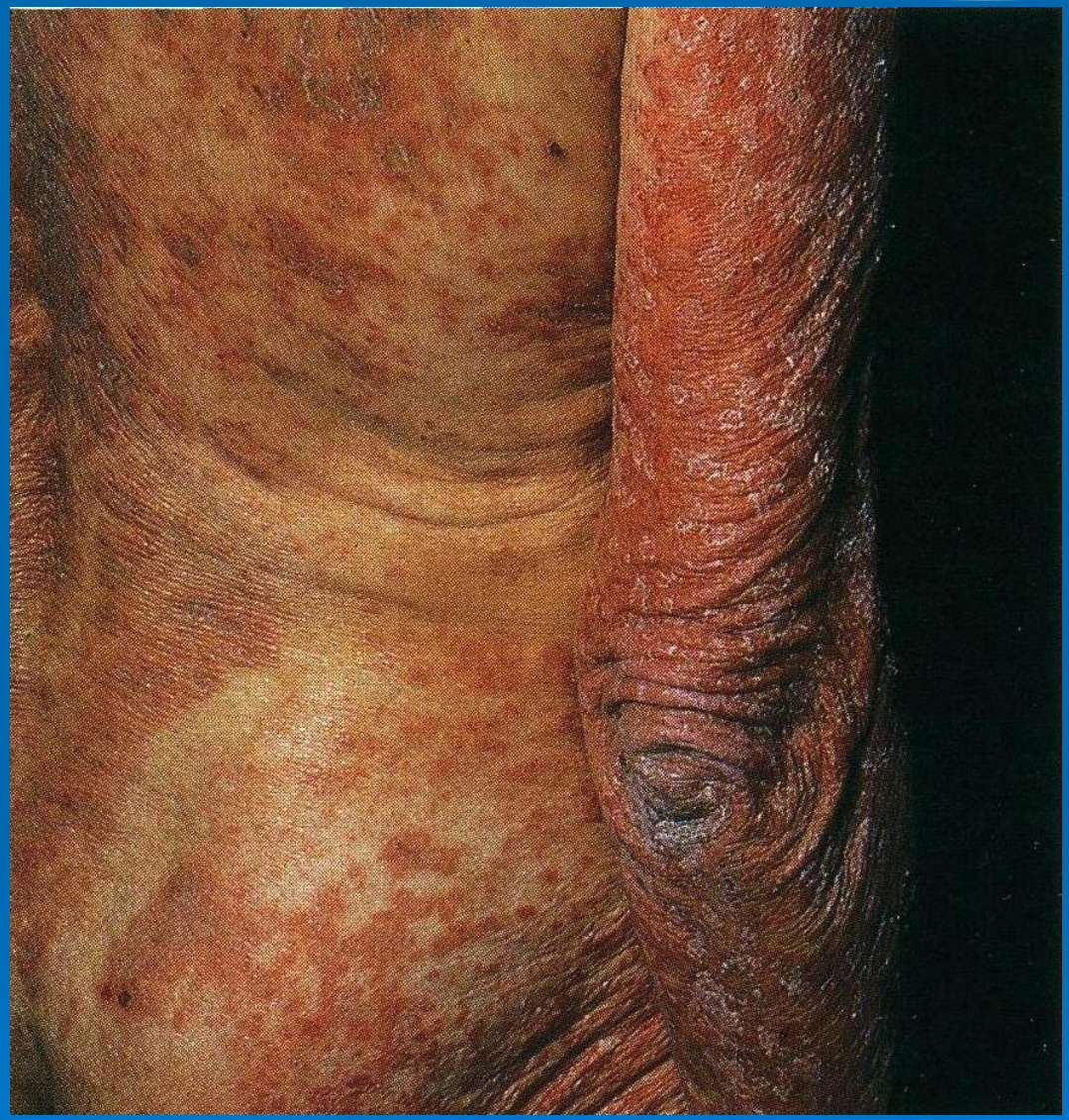


10. Lichenoid Eruptions

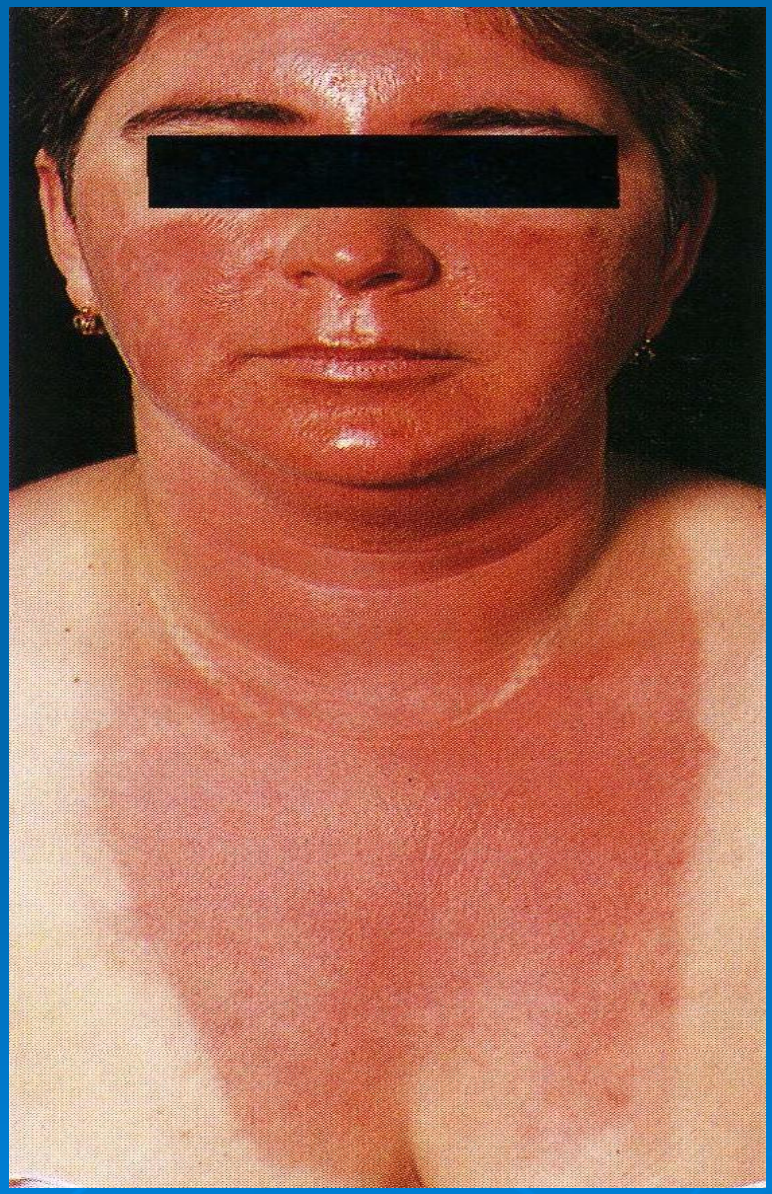




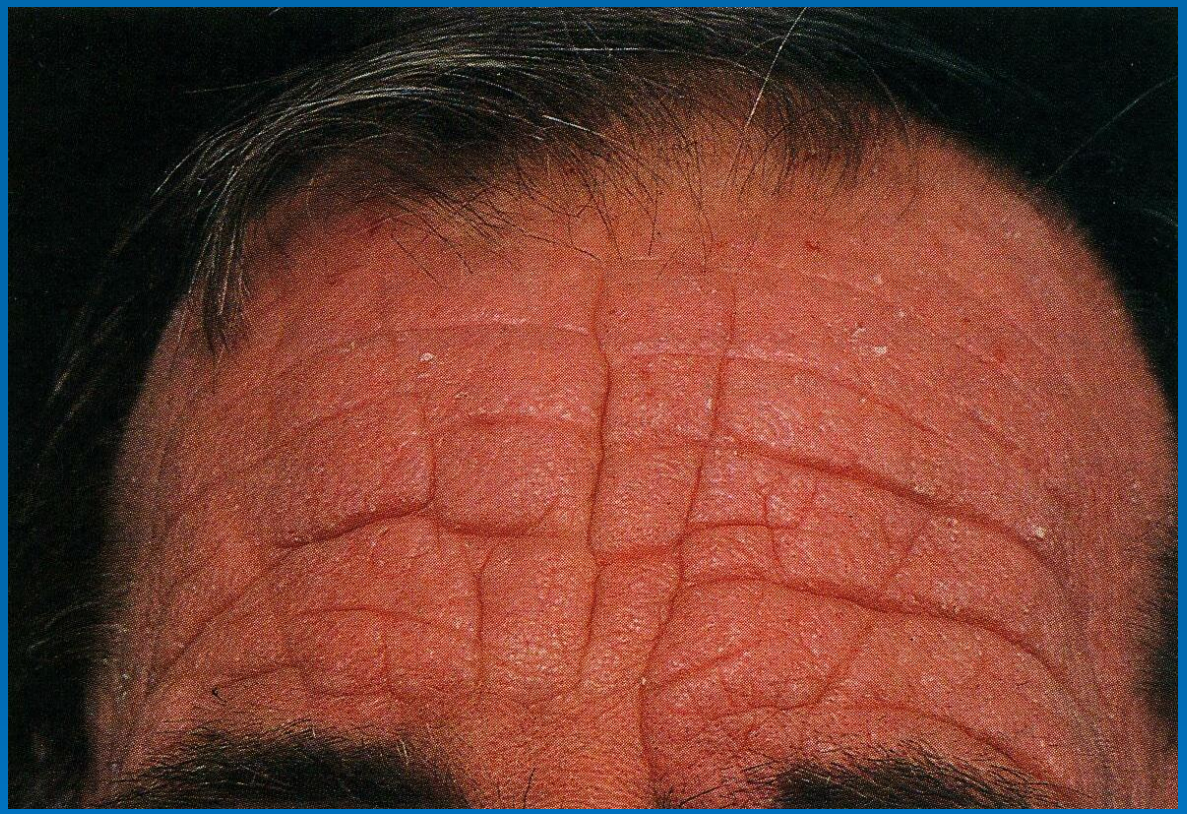




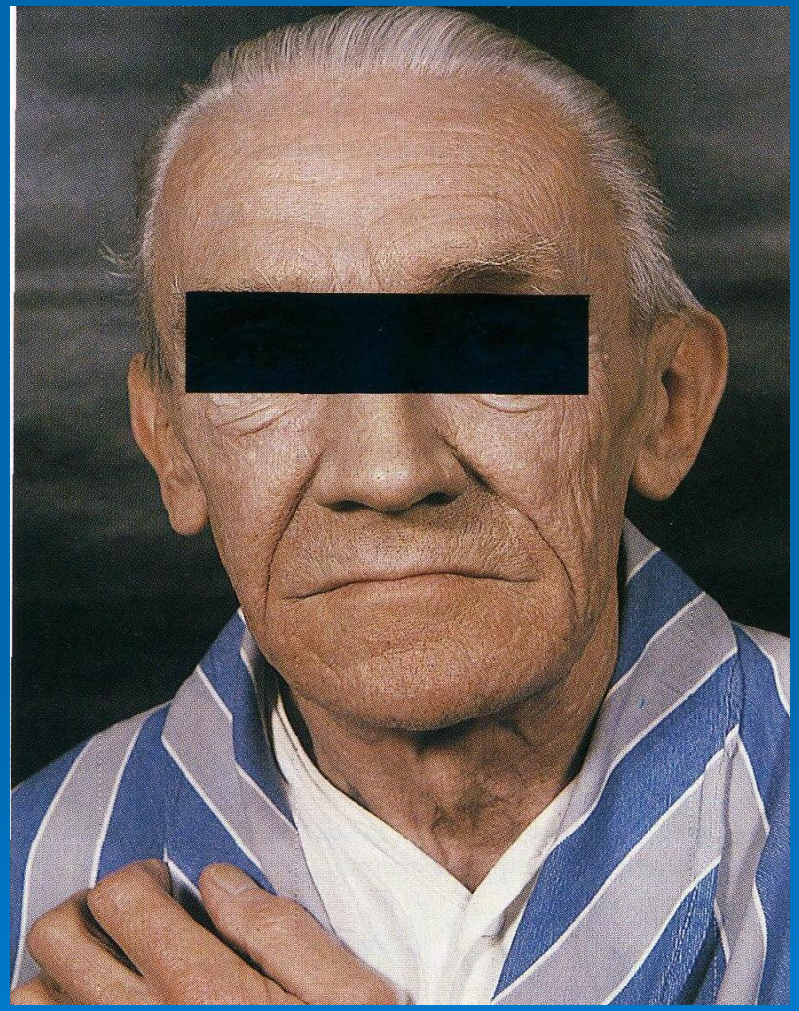
11. Photosensitivity



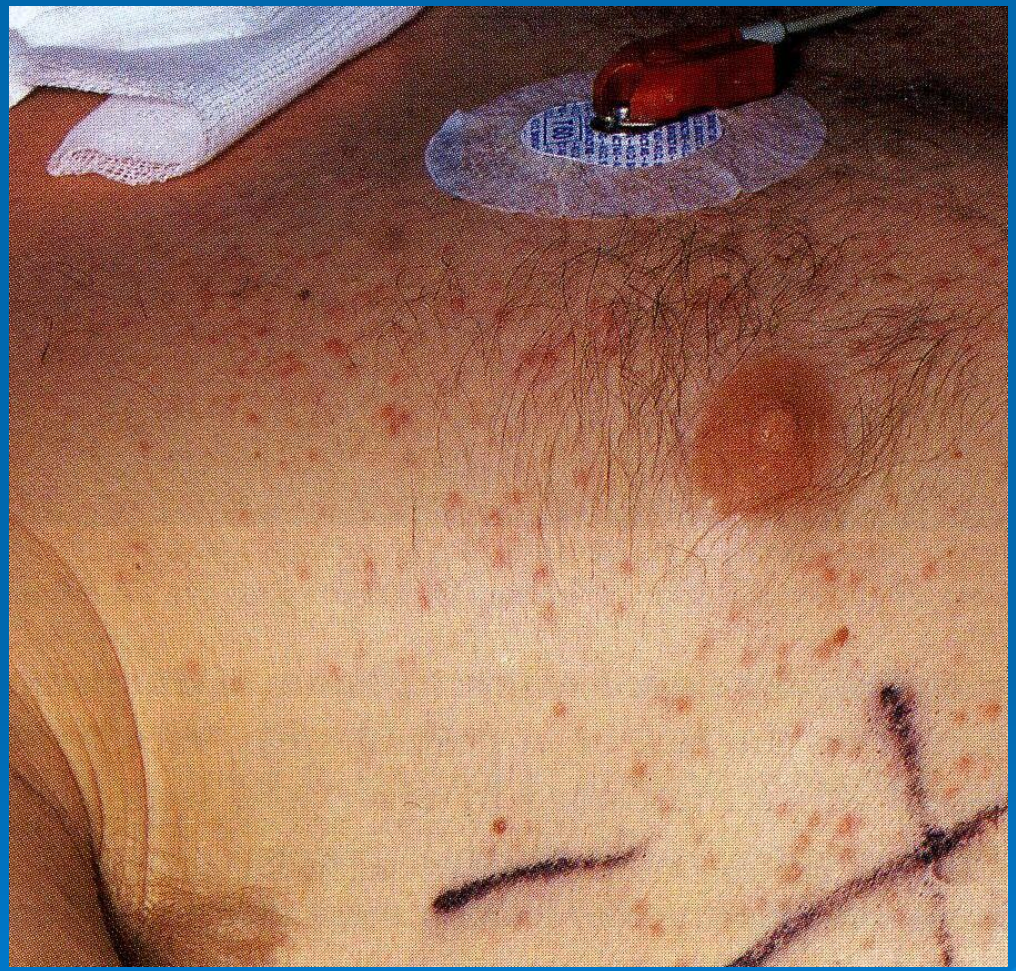
12. Chronic Antinic Dermatitis

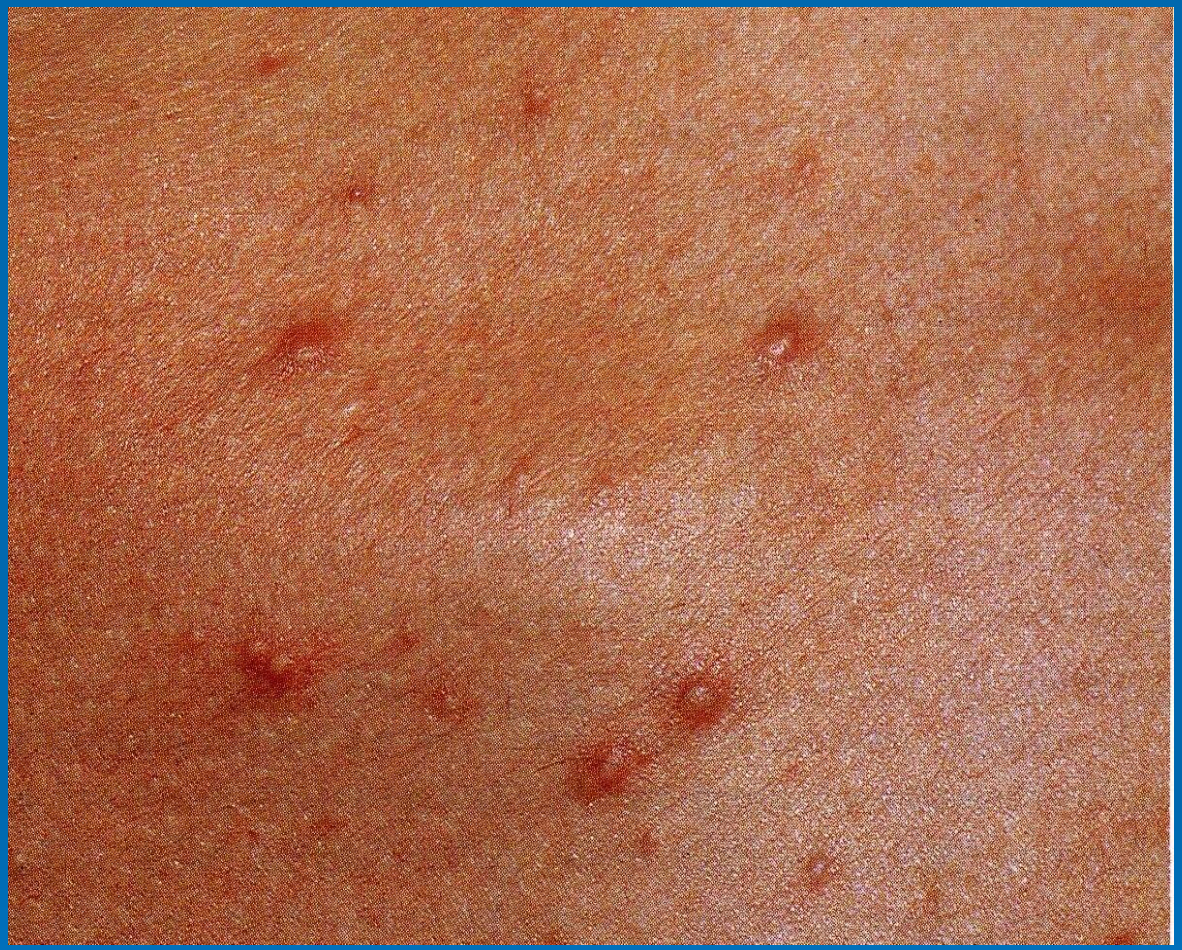


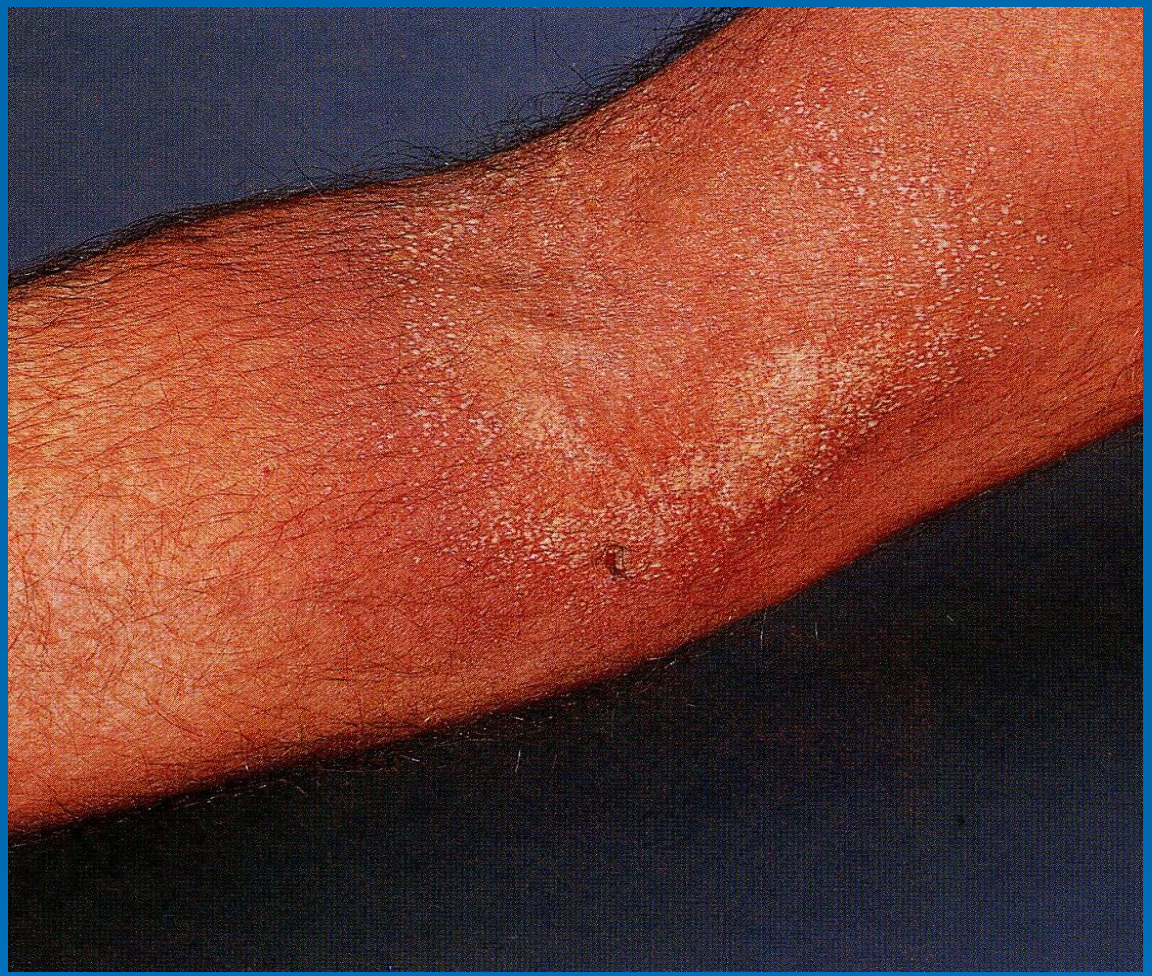
Pigmentary Abnormalities



13. Acneiform and Pustular Erupstions





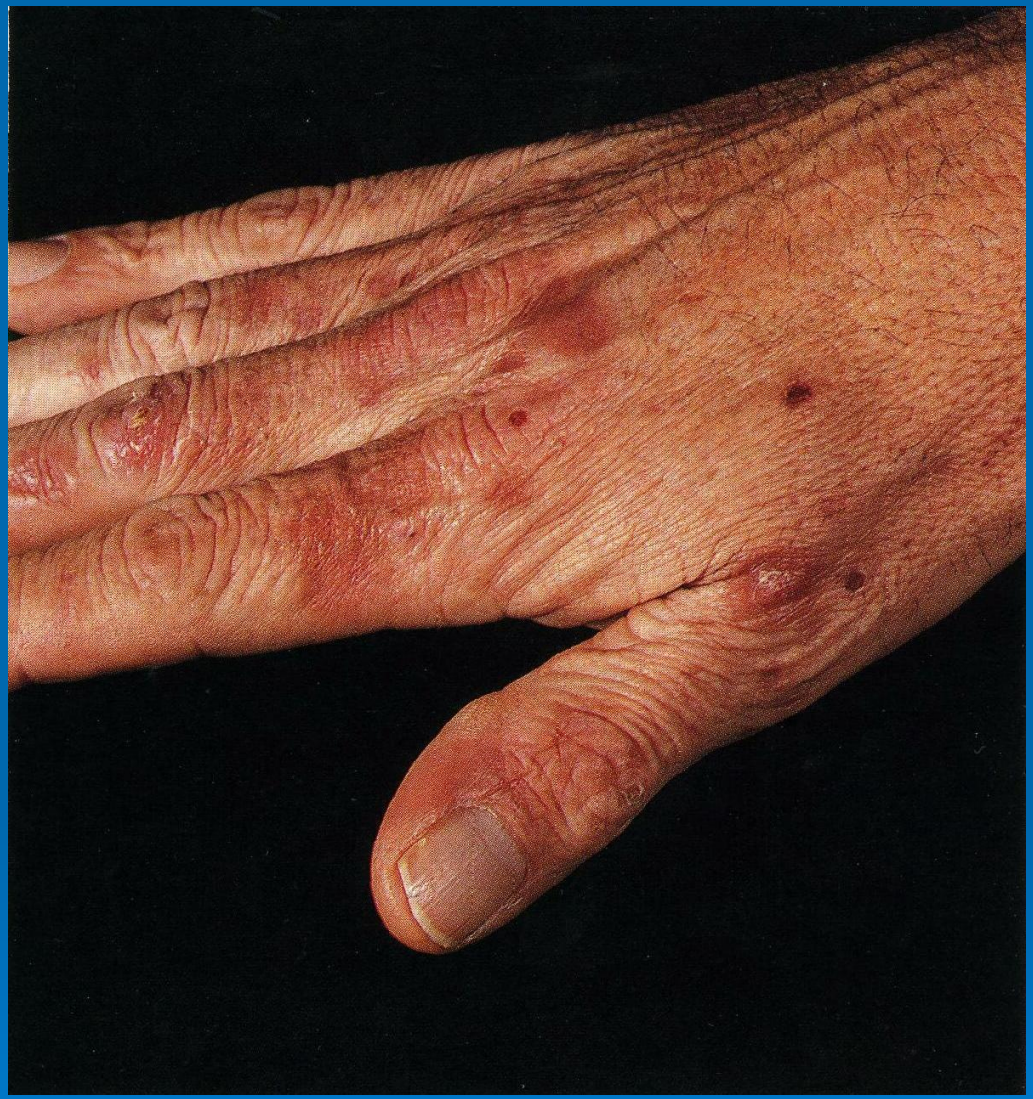


14. Bullous Eruptions

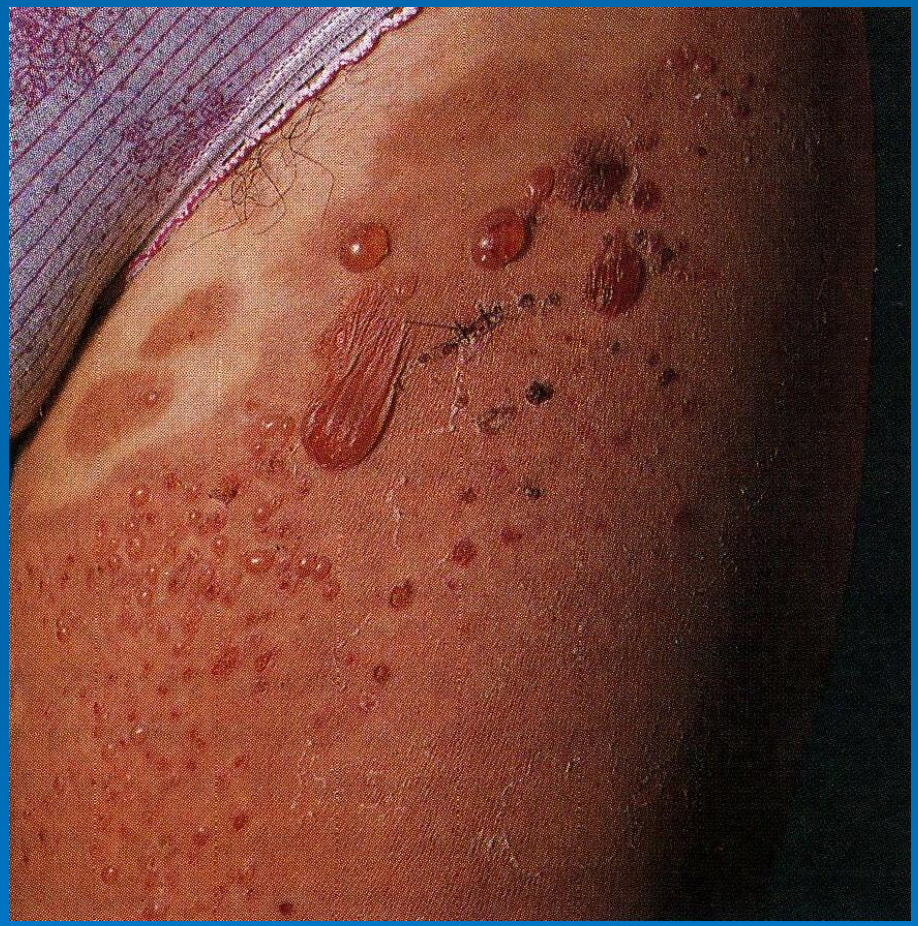
- Fixed drug eruptions
- Erythema Multiforme
- Drug induced Vasculitis

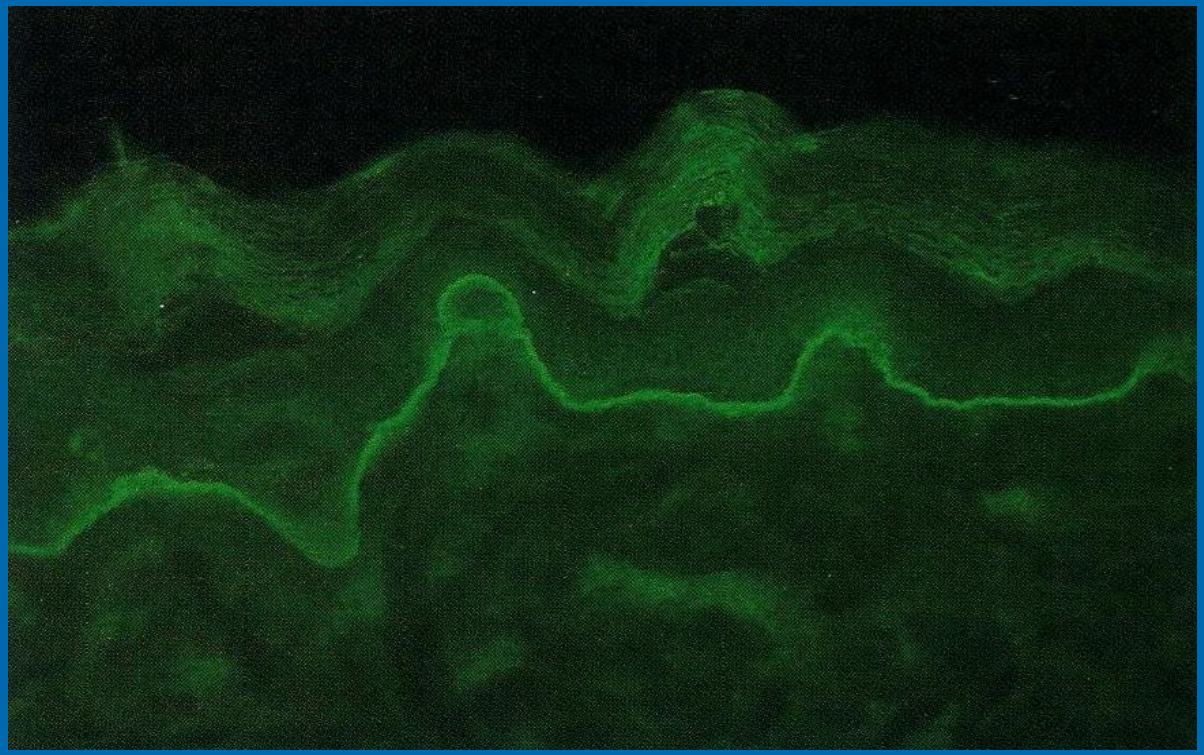
All may have a bullous component

- Drug induced TEN (Widespread blistering)
- Drug induced Porphyria and Pseudoporphyria

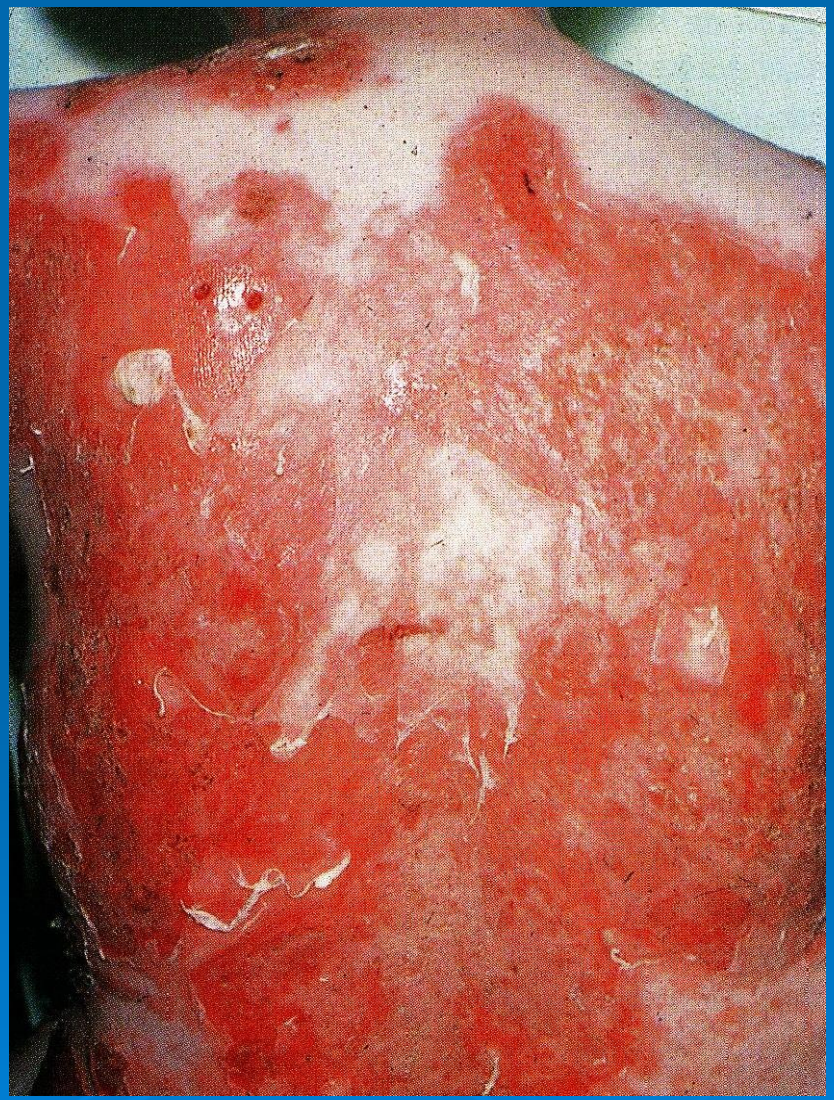


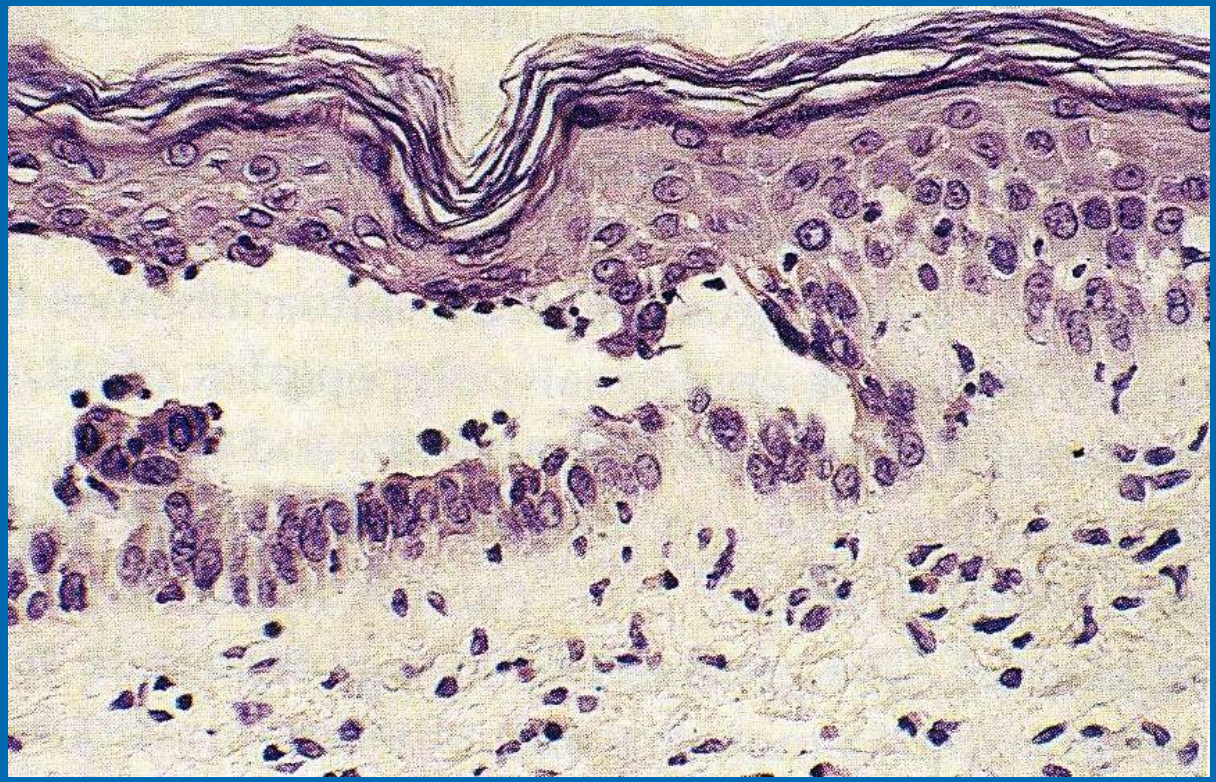
- Drug induced bullous pemphigoid



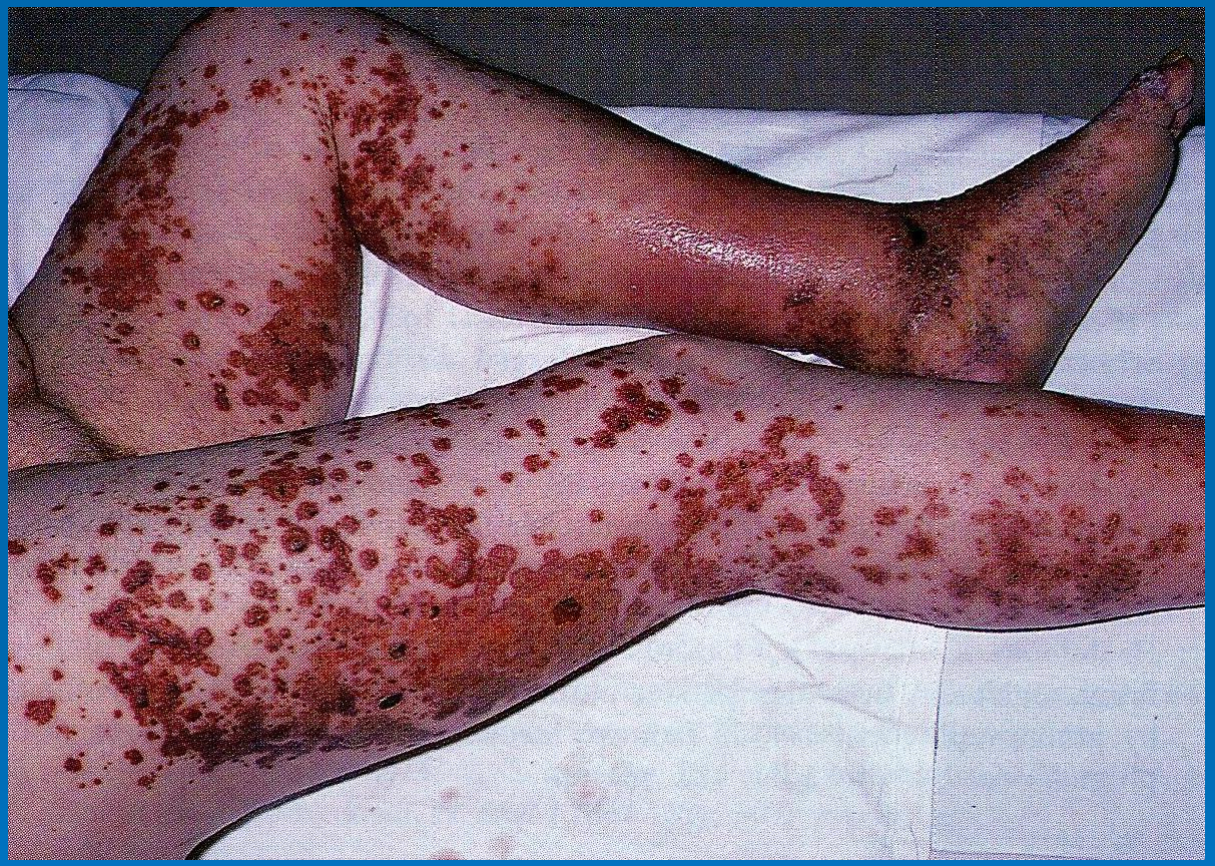


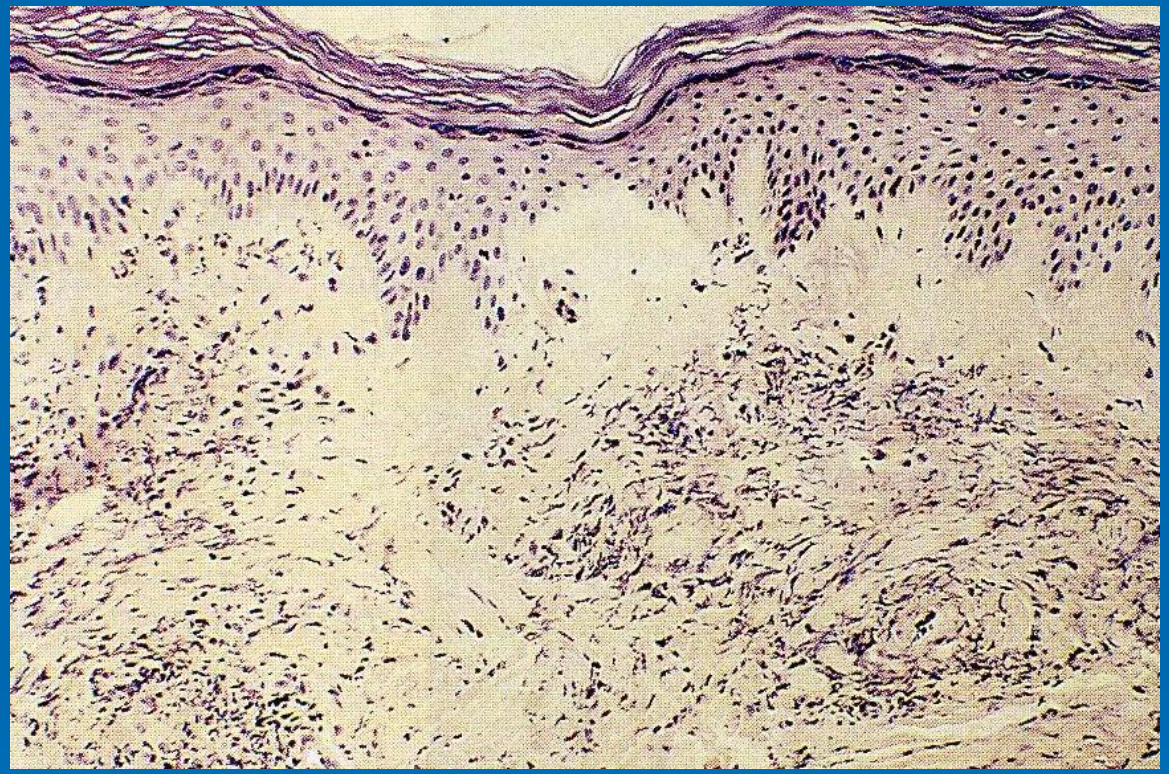
- Drug induced Pemphigus

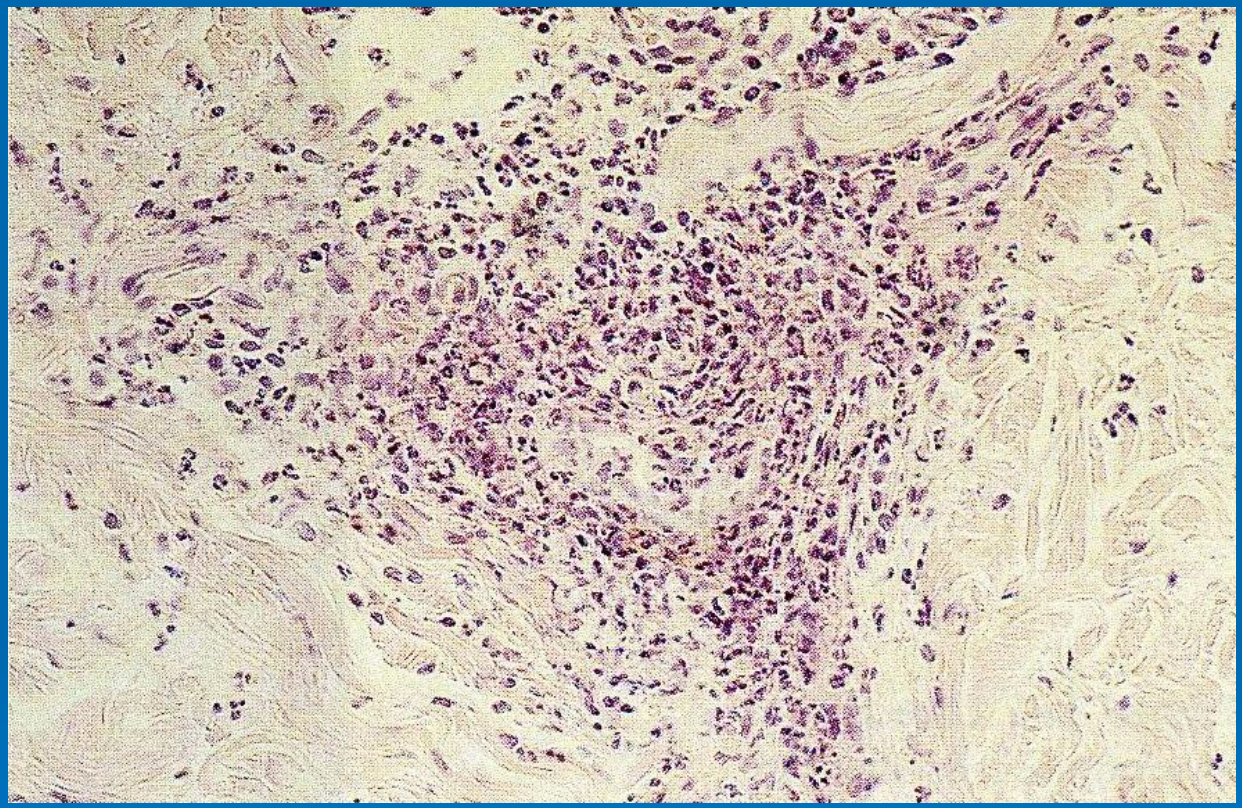


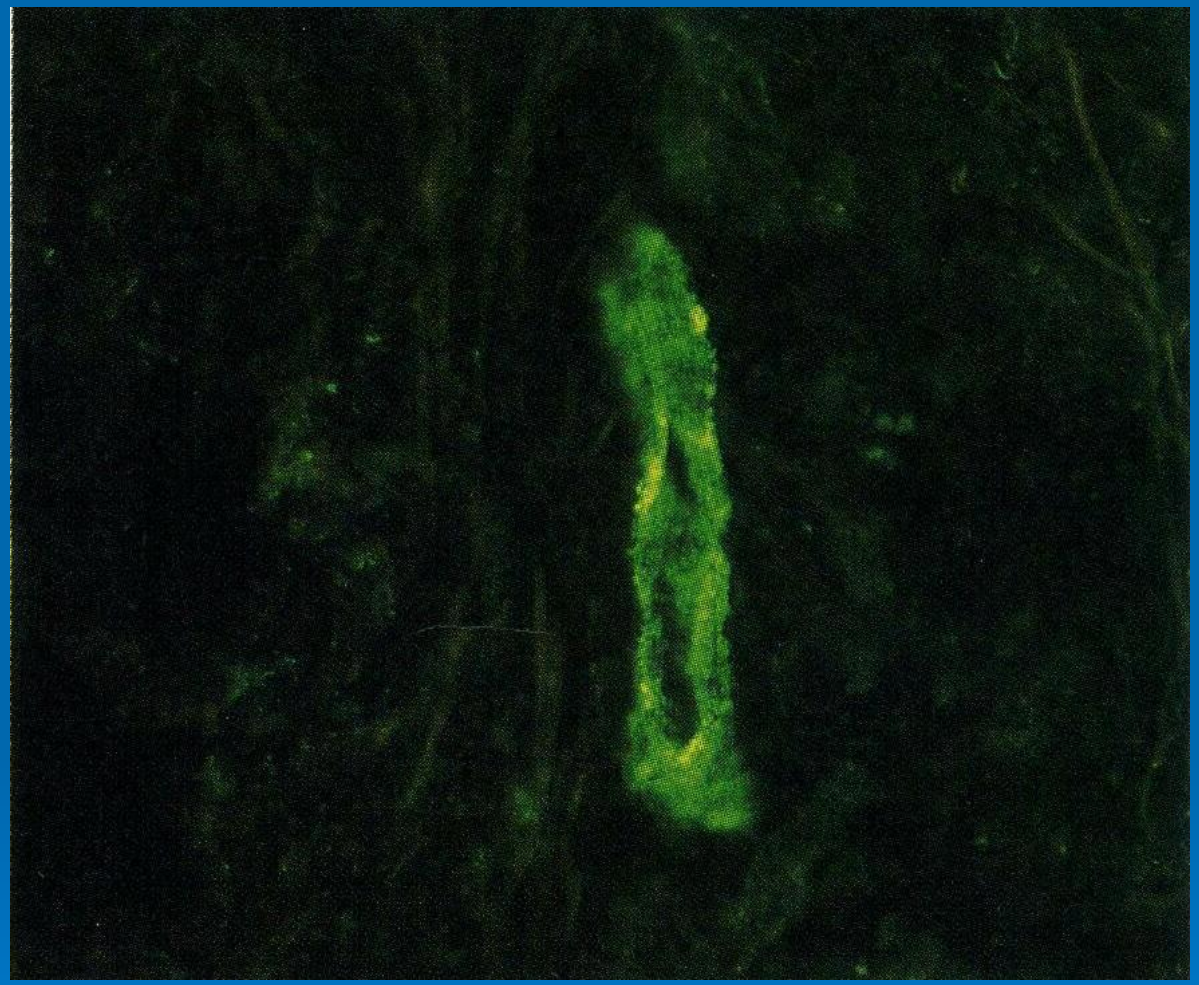


15. Vasculitis

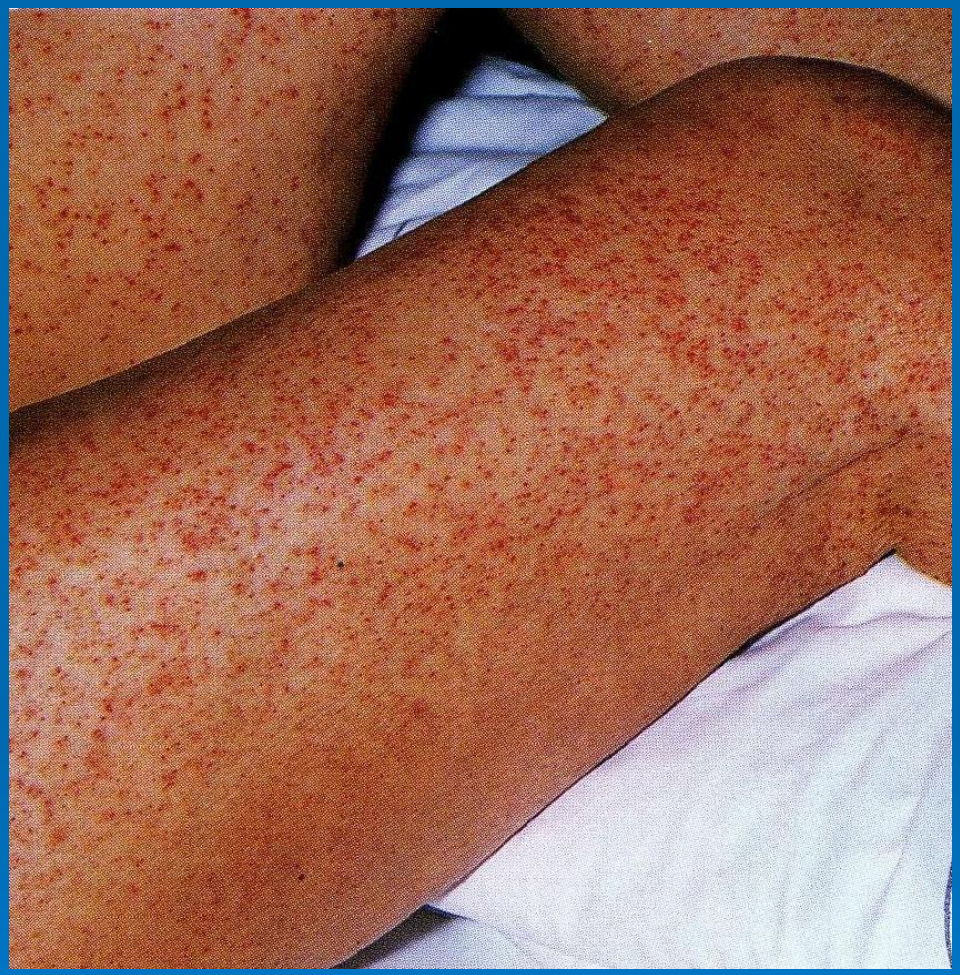


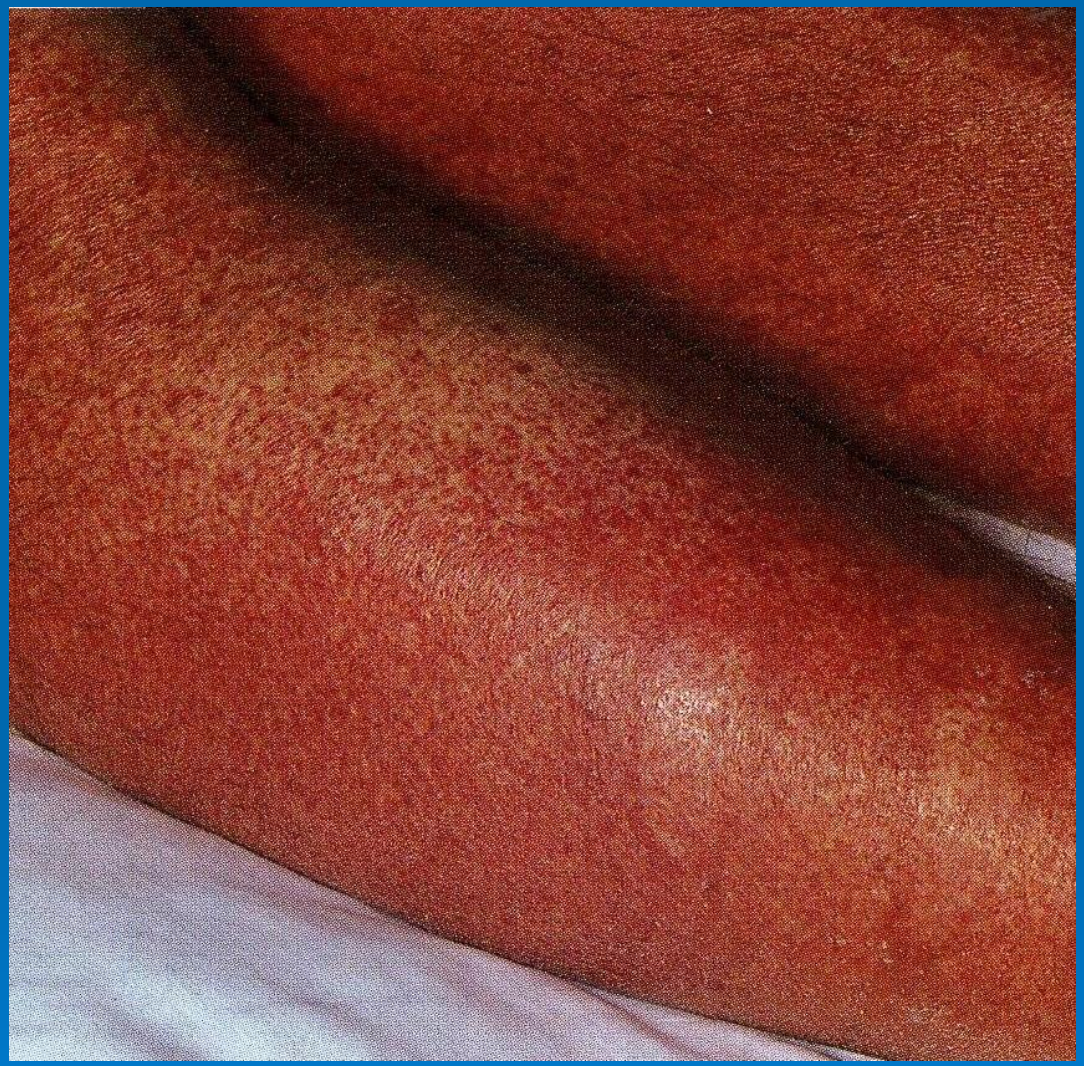


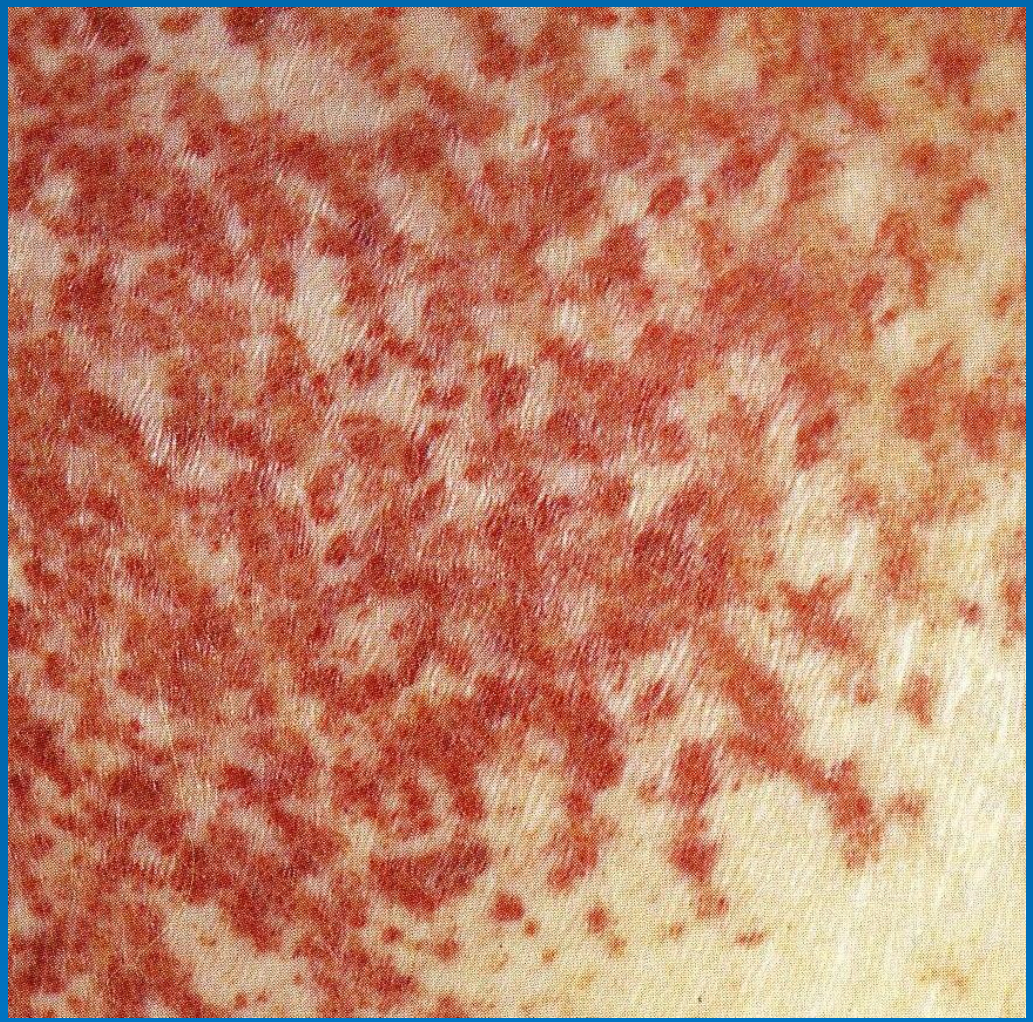




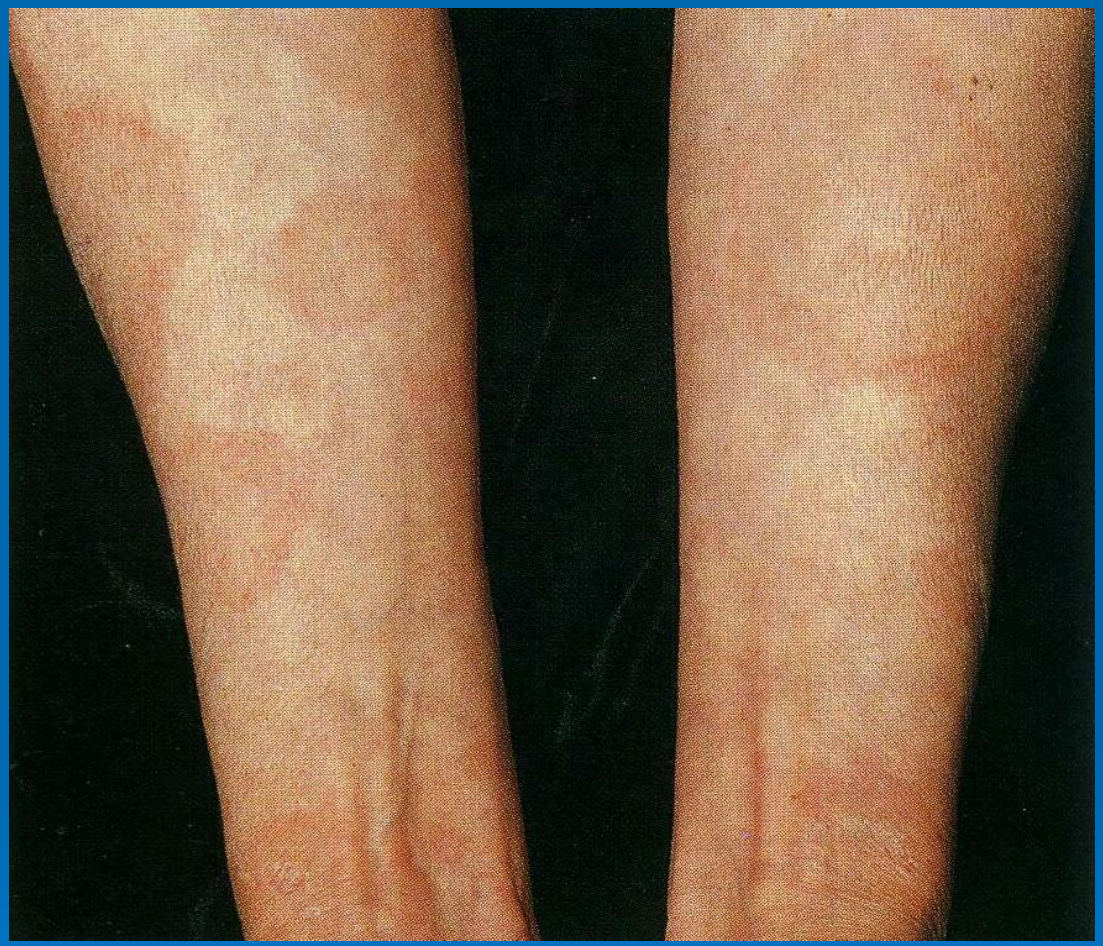
16. Purpura



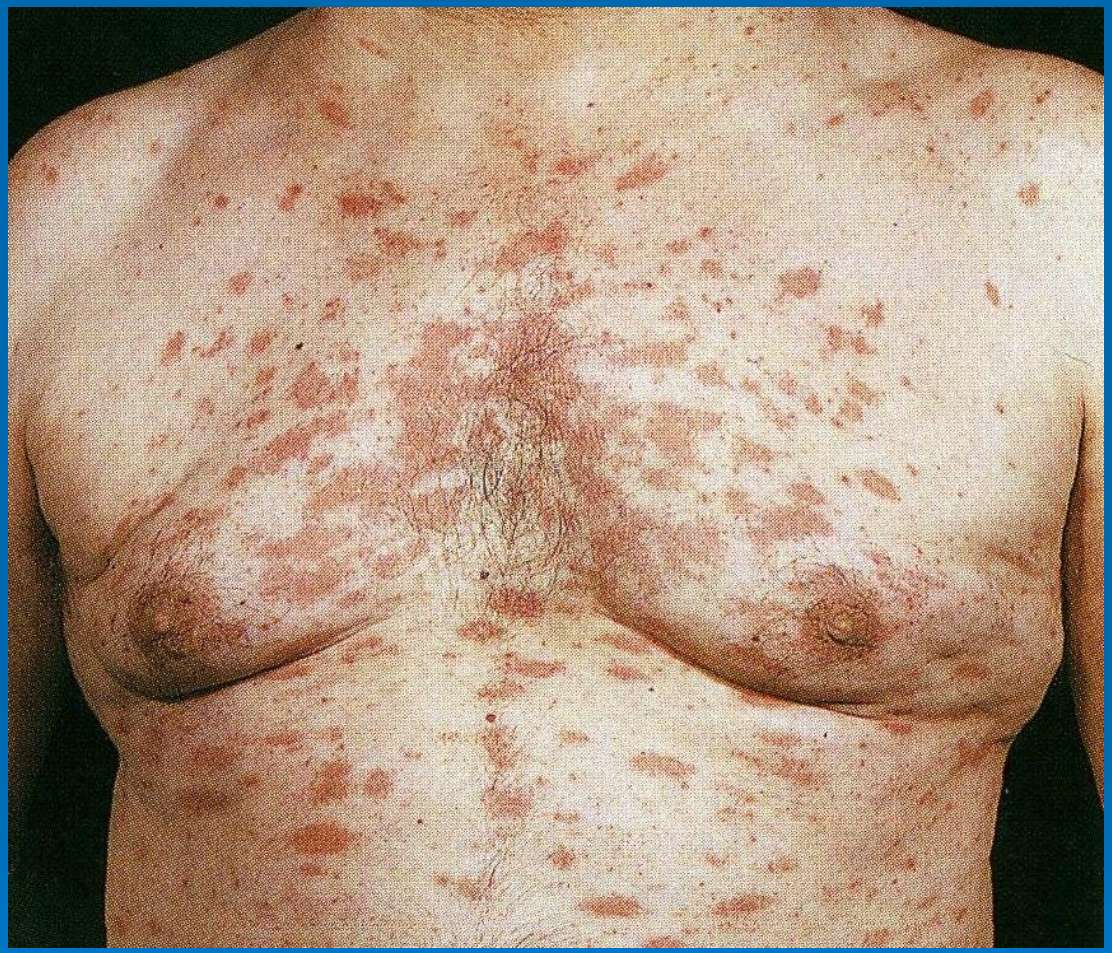




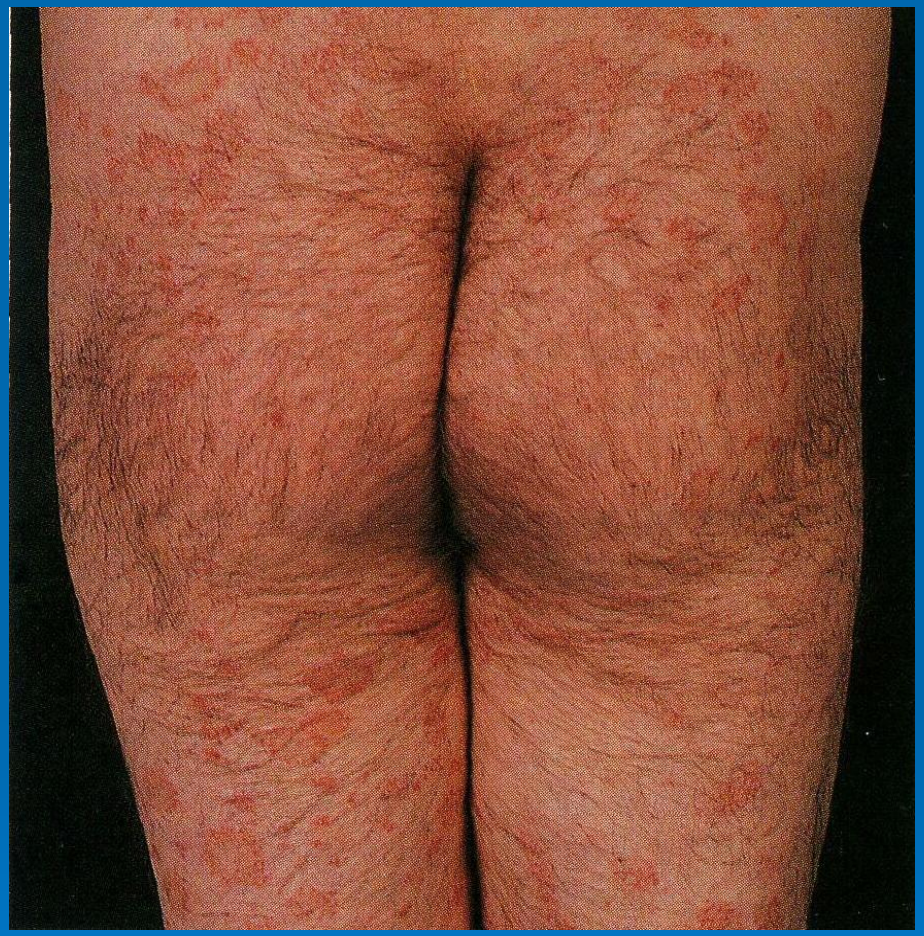
17. Annular Erythema



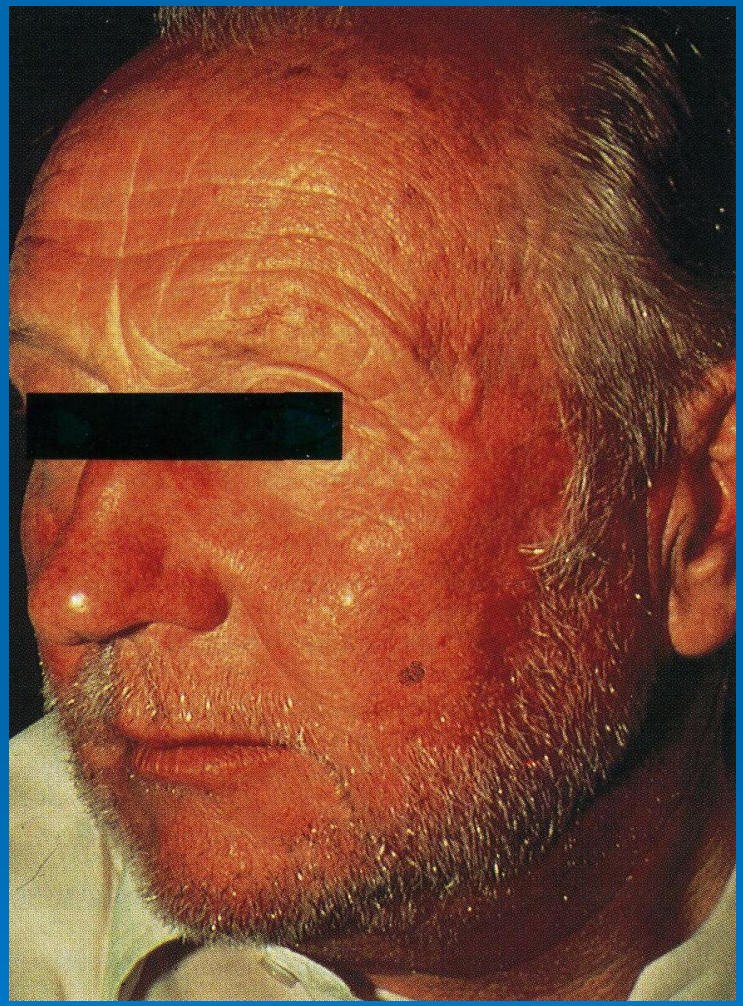
18. Pityriasis rosea like eruptions



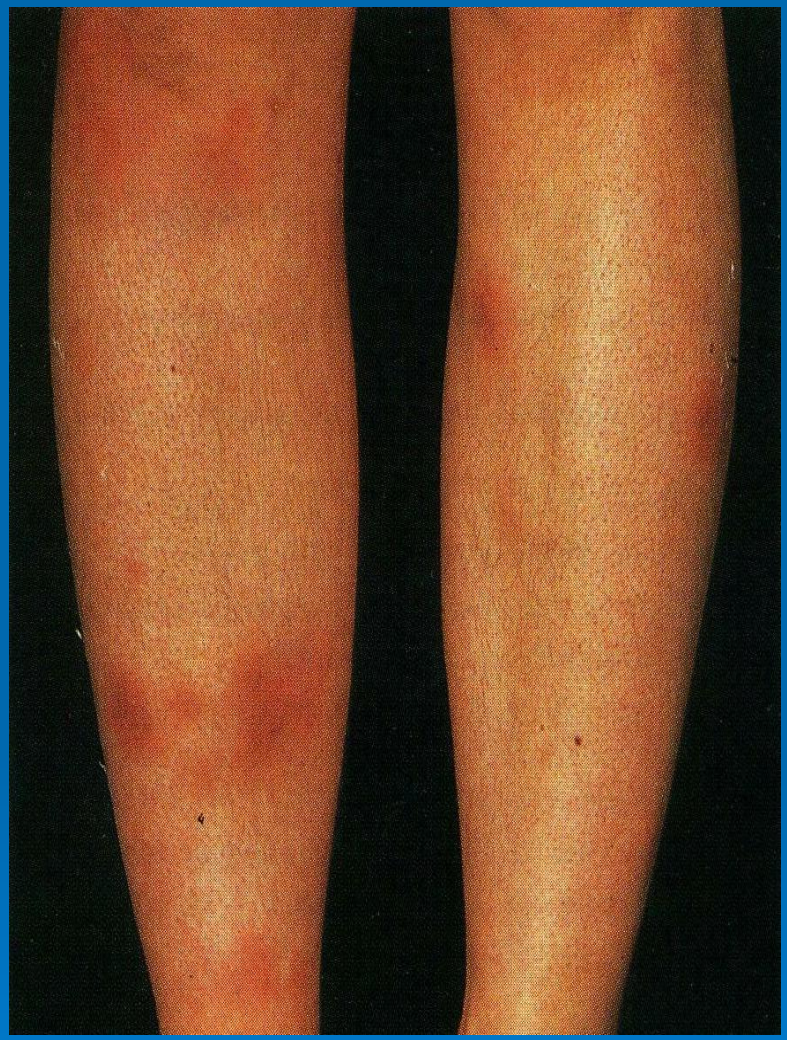
19. Psoriasiform drug eruption

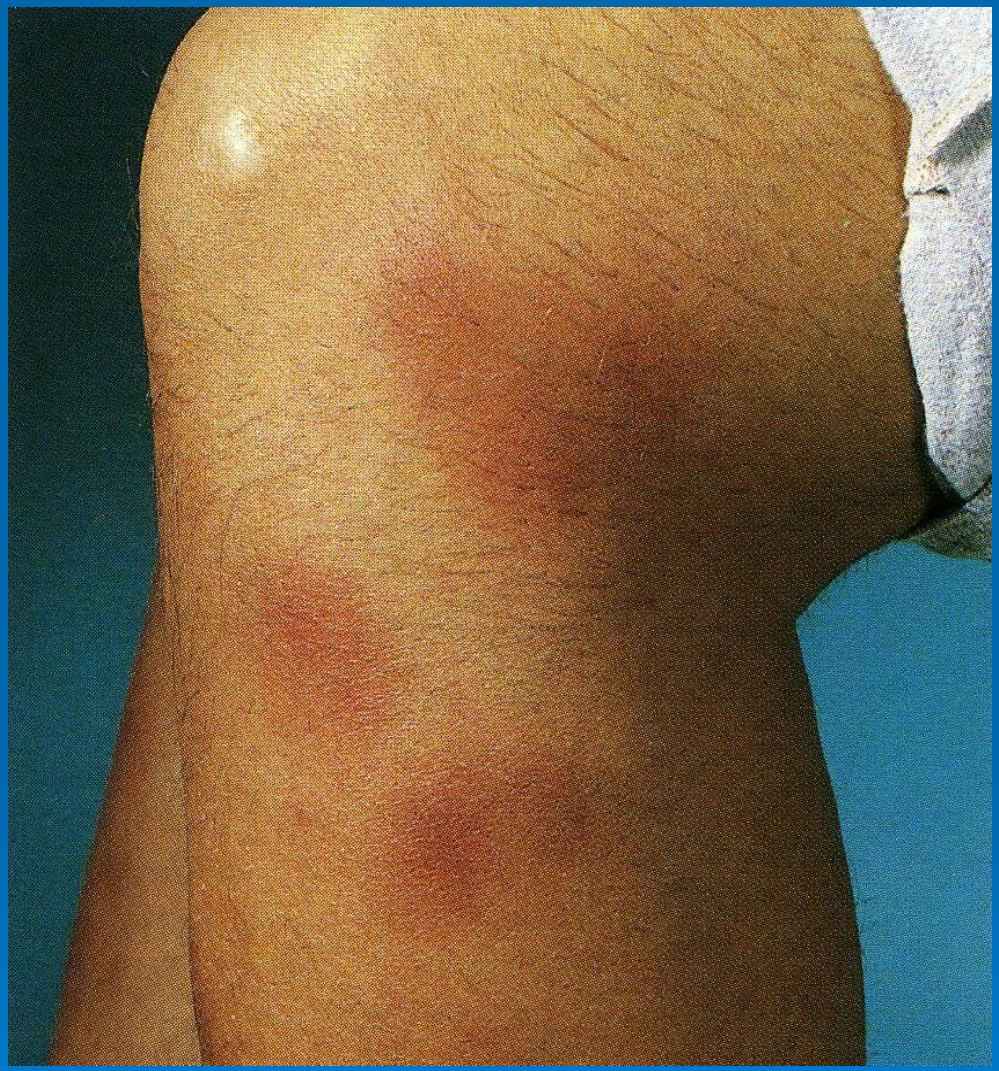


20. Lupus Erythematosus-like syndrome induced by drugs



21. Erythema Nodosum





Thank you

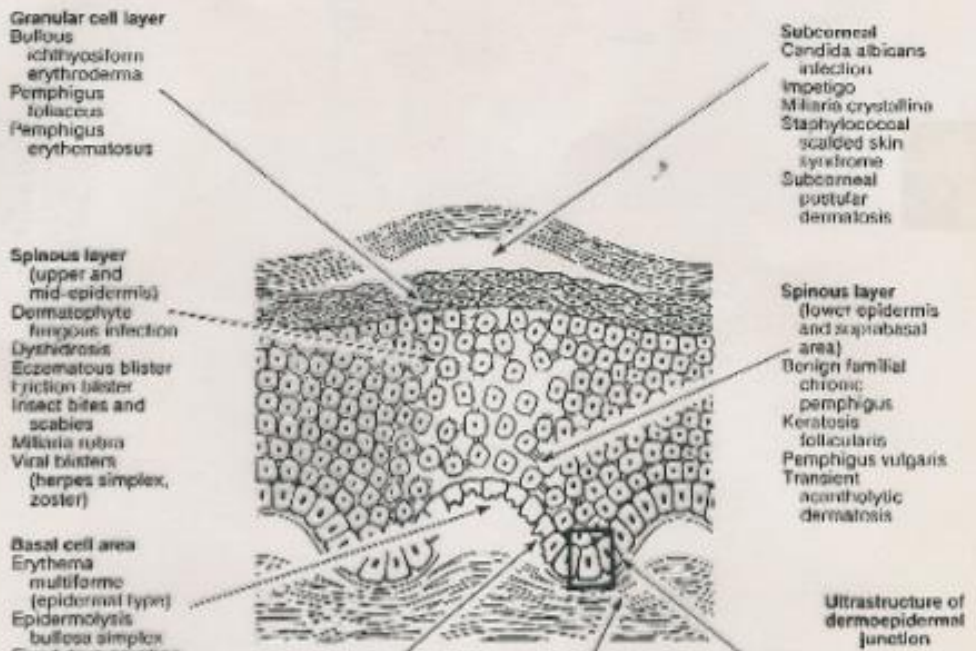
Vesiculobullous Disorders

Def: Circumscribed skin lesions containing fluid ≤ 5mm: vesicle.,>5mm:- bulla

Which skiing findings are helpful in evaluating a patient with blister?

- Distribution : Generalized skin and oral cavity
- Symmetry
- Associated lesions
- Additional types of skin lesions –urtic.
- Characteristic of blisters

Figure 16-1
Bullous diseases in the epidermis and dermoepidermal junction.



Which test are most useful in evaluating Vesiculobullous Dis.?

- Cultures (Bact, viruses, fungi)
- Smears from blisters (Bact, dermatophytes, multinucleate giant cells of H.S)
- Skin biopsy – non infectious cause

When are special tests necessary to diagnose blistering Dis. of the skin?

- Routine Hist.
- Dif- immunobullous Dis./IIF
- EM – EB.
- Urine porphyrin- PCT
- Zinc levels - ADE

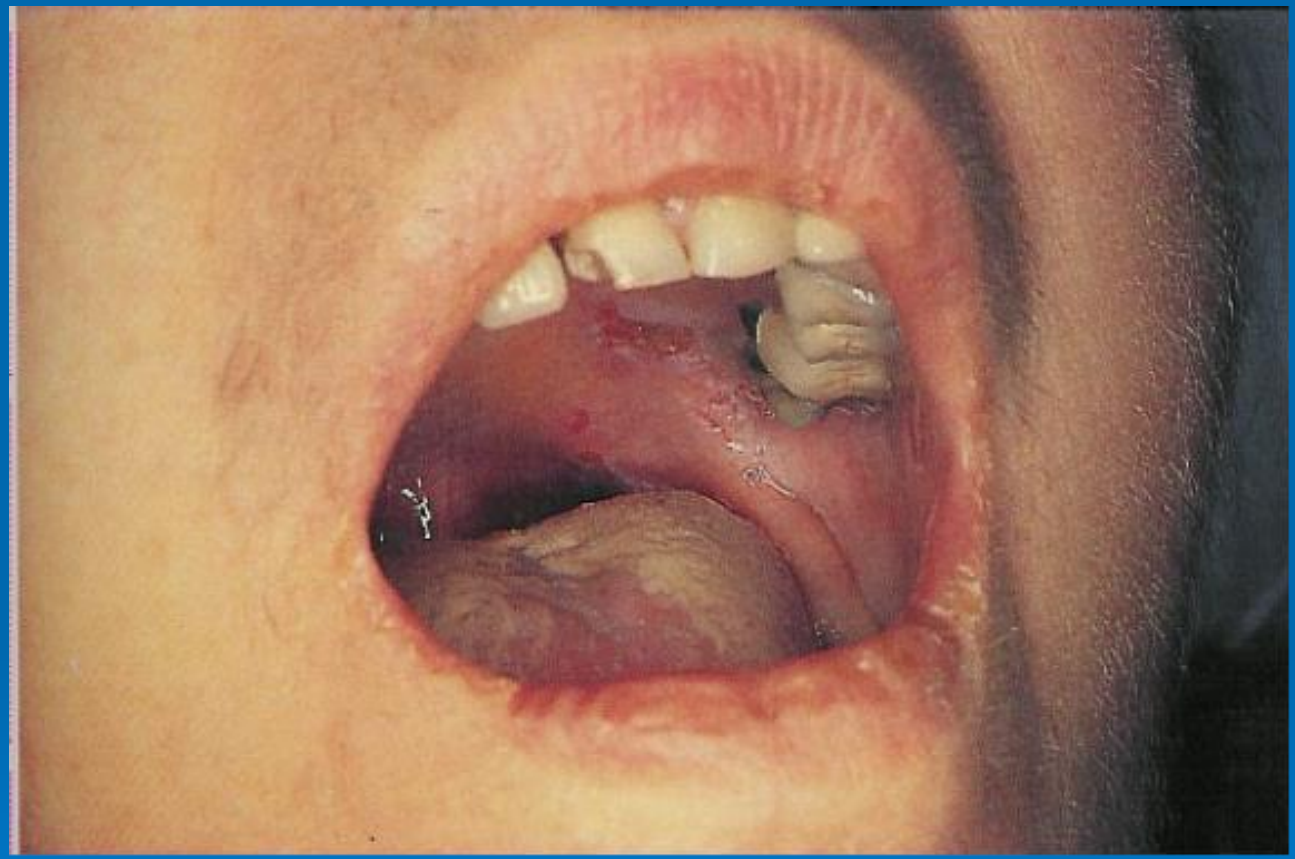
Pemphigus

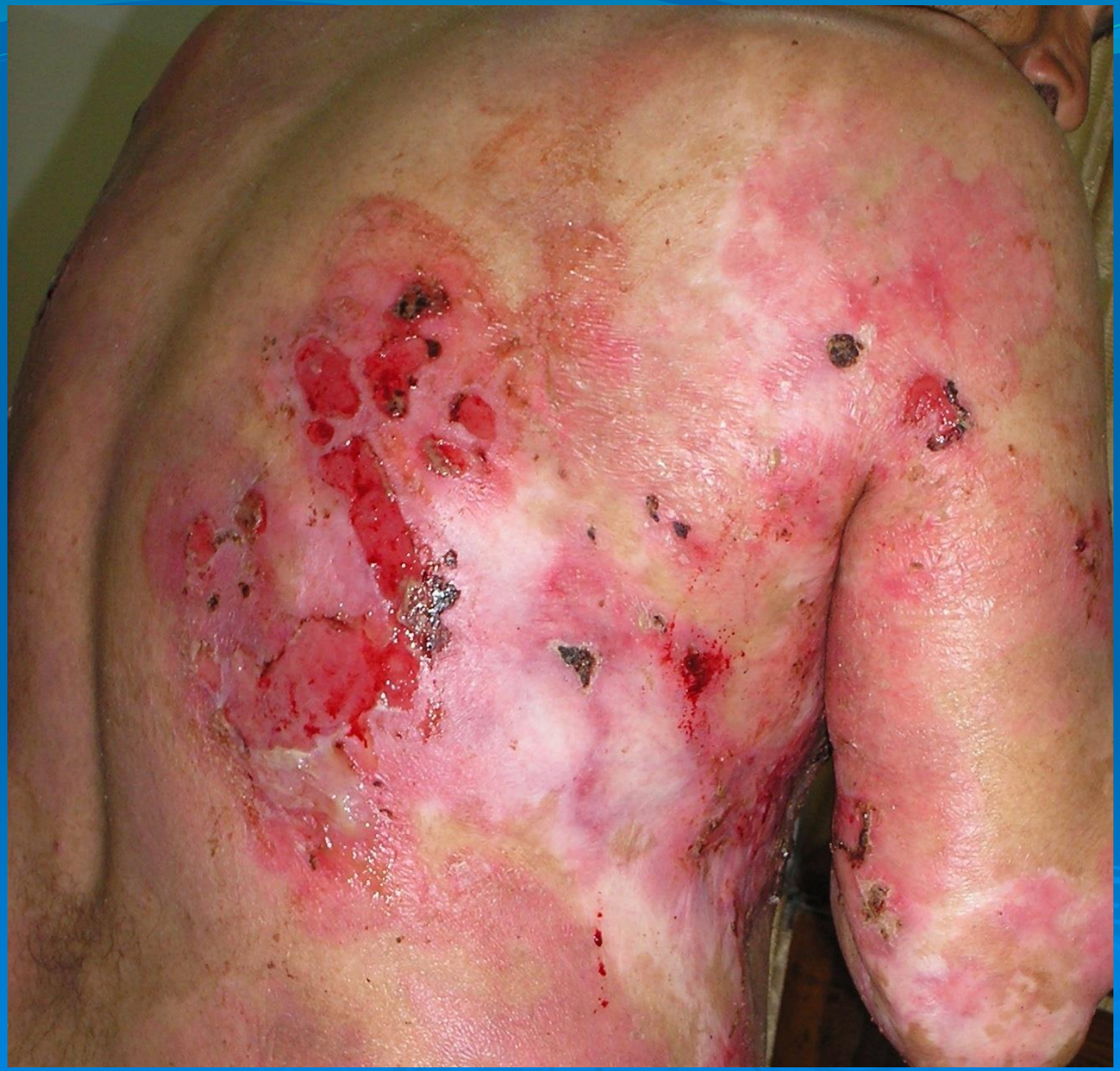
Essential of

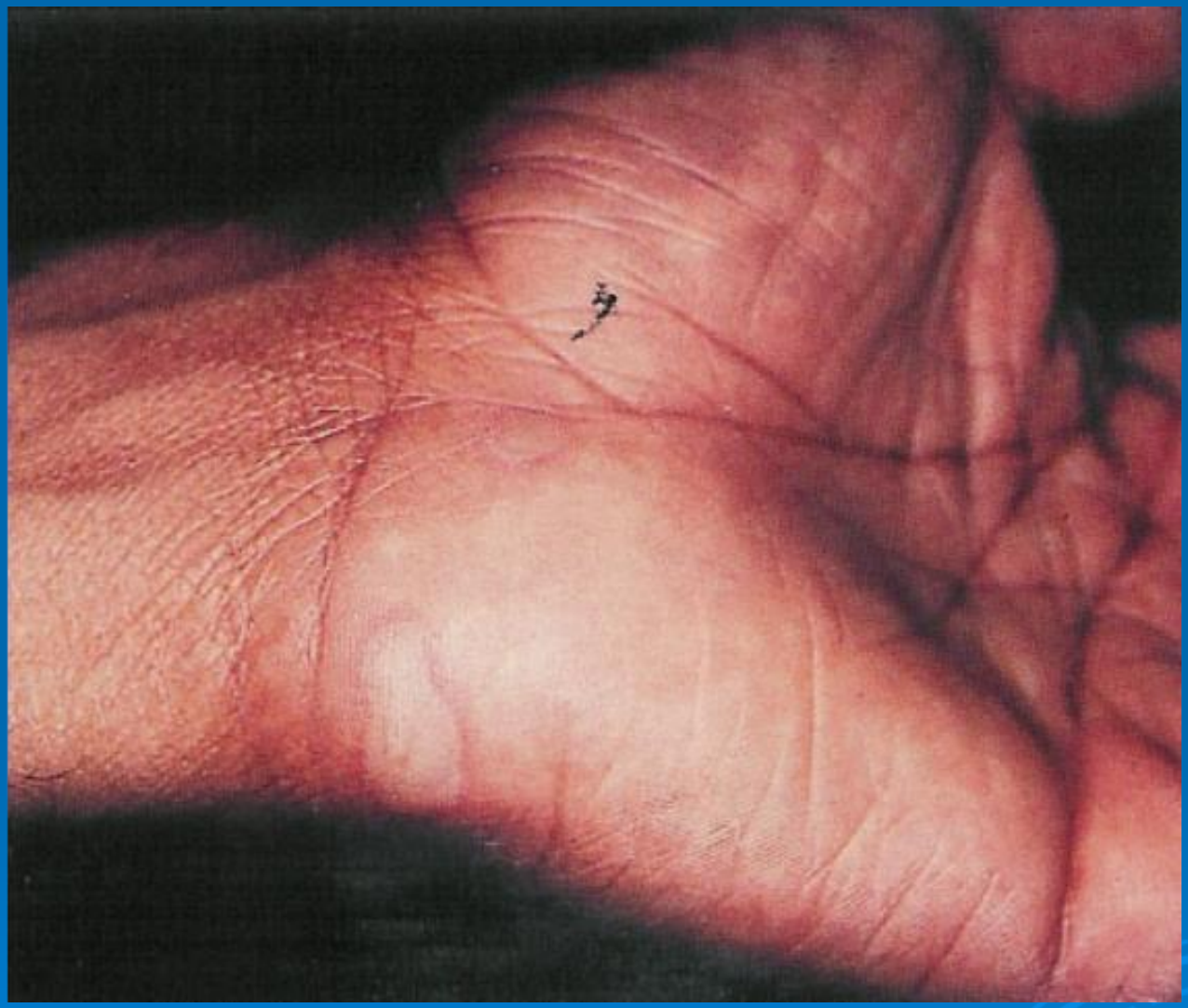
- Flaccid blisters, weeping painful erosions
- M.M. involv.
- Nikolosky's sign
- Asboe – Hansen sign or (bulla spread sign)
- Acantholysis and intra epidermal cleft
- IgG, C3 in intercellular space of epid.
- IF:- S.Abs to epidermal cell surface Ags – Titer reflects Dis. Activ.
- Rare, Lethal, Autoimmune blistering Dis.
- Age – sixth decade

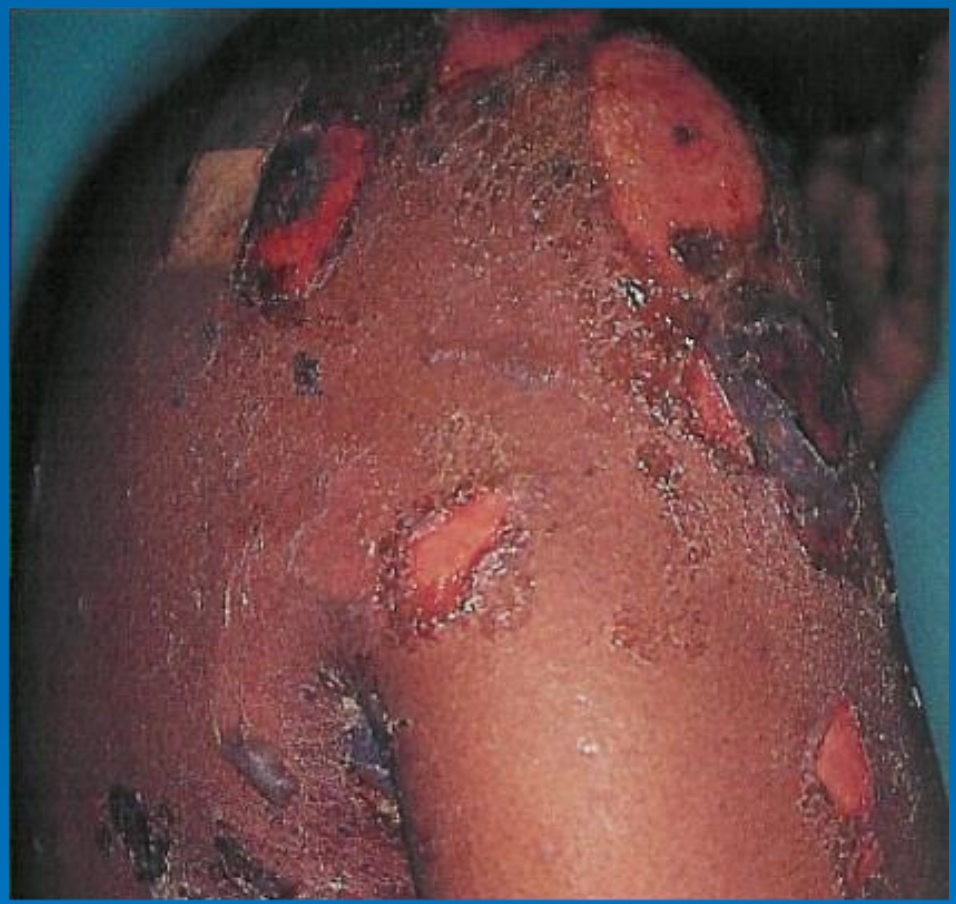
Classification of pemphigus :

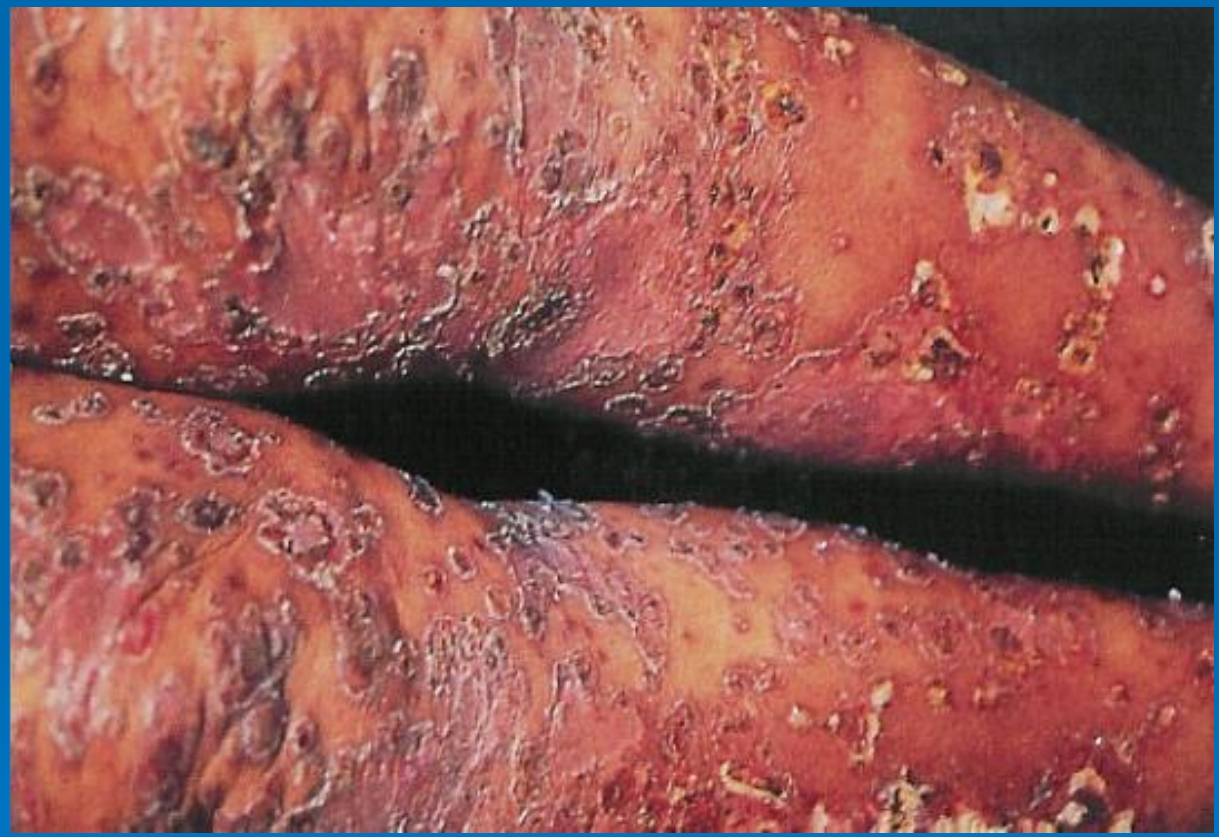
- Pemphigus vulgaris
  - Pemphigus vegetans
- Pemphigus foliaceus
  - Pemphigus erythematosus : localized
  - Fogoselvagem : endemic
- Drug induced pemphigus
- Paraneoplastic pemphigus
- IgA pemphigus

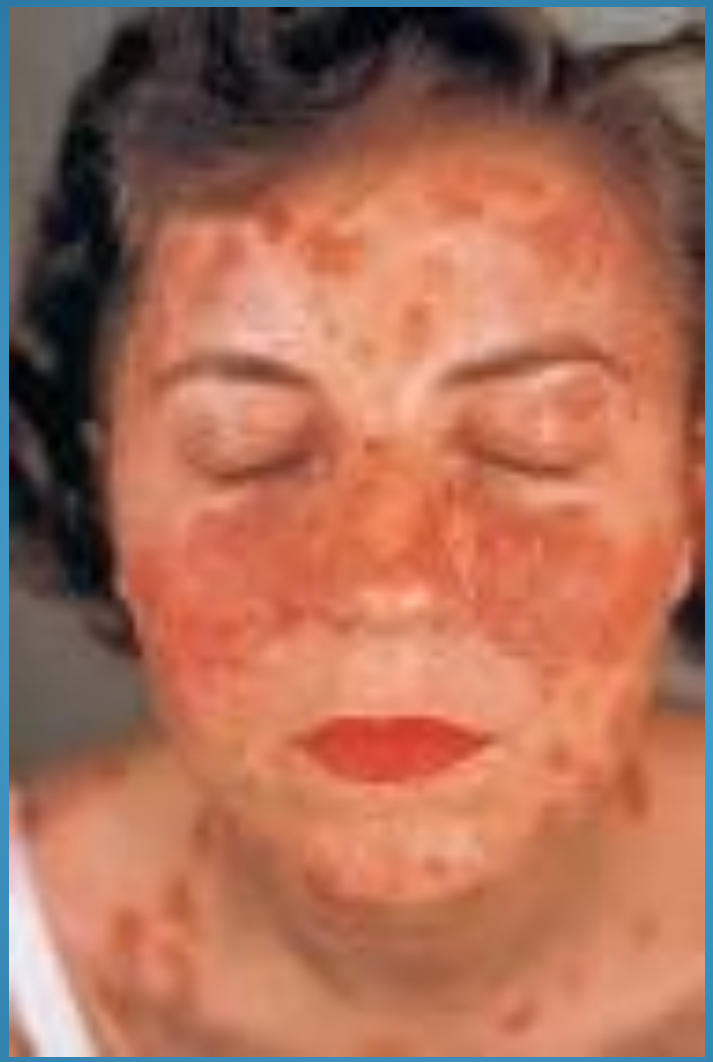


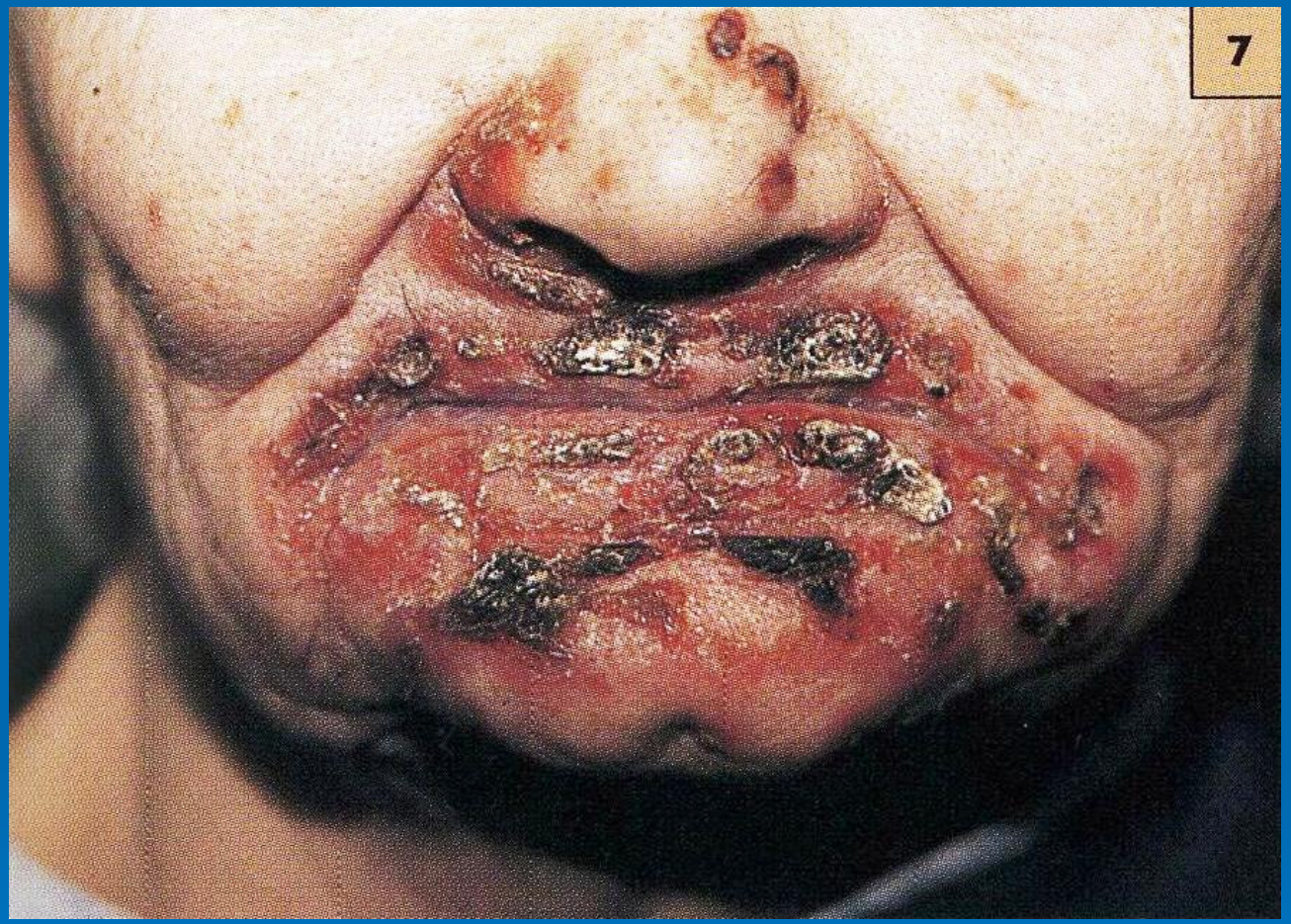








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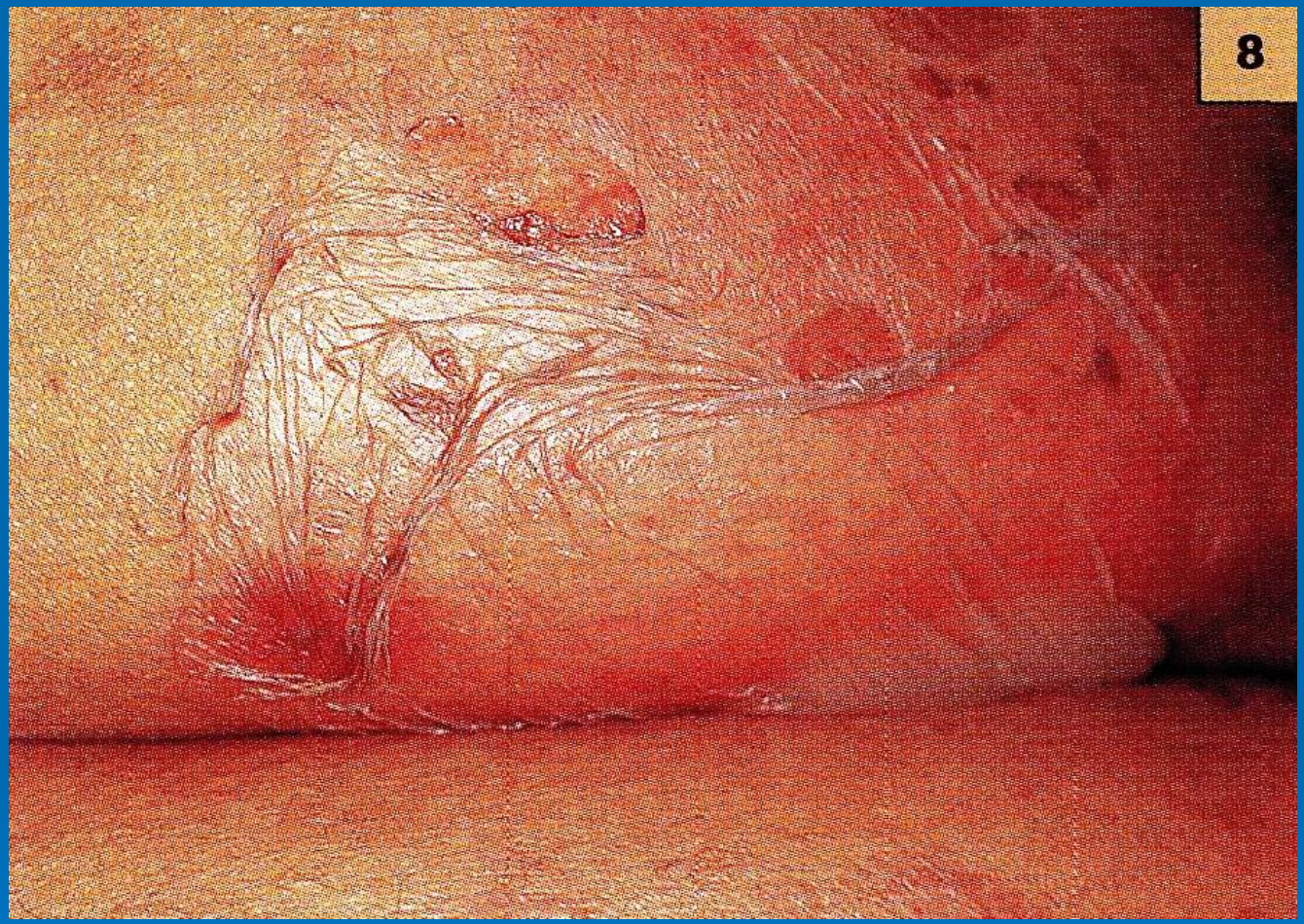
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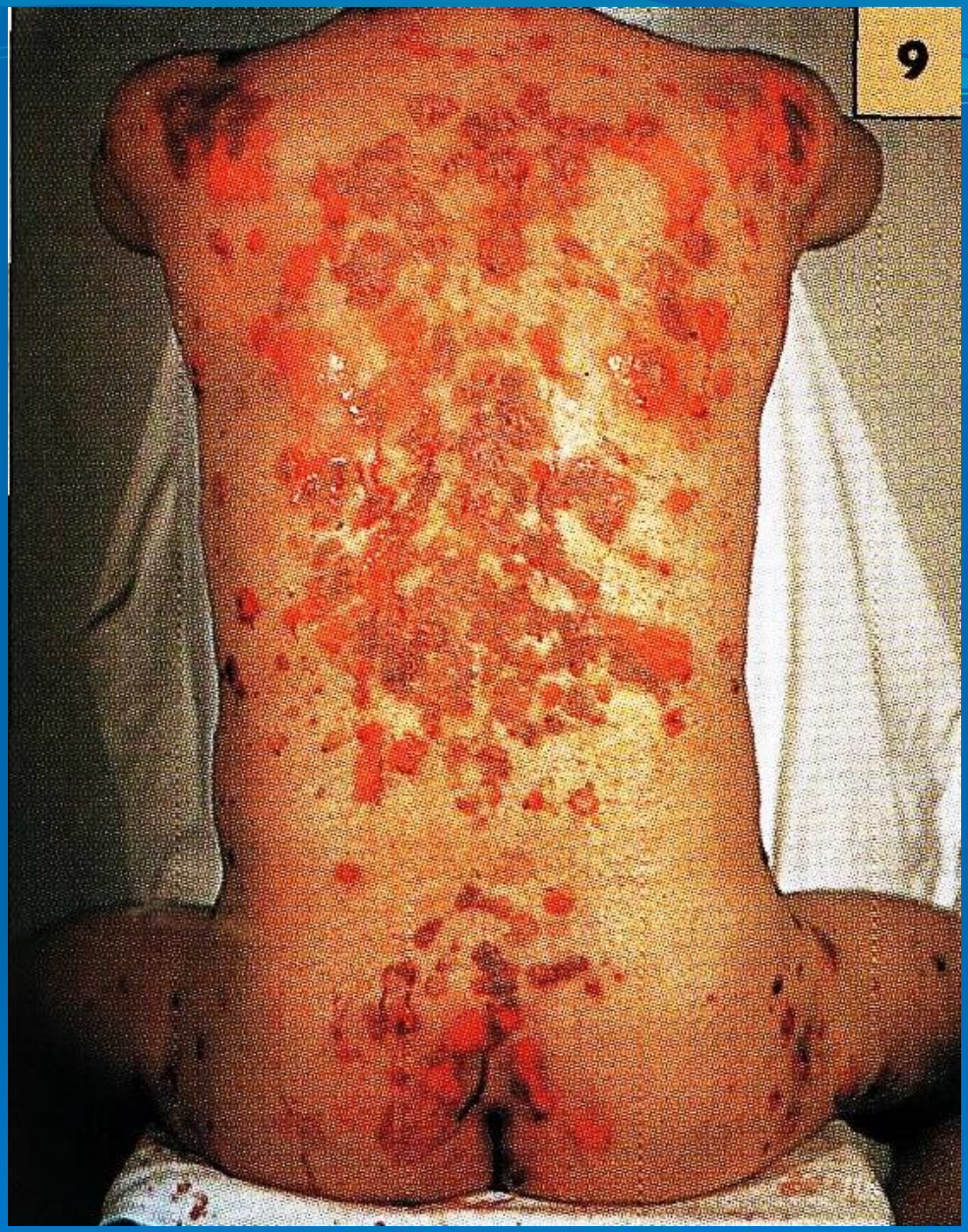
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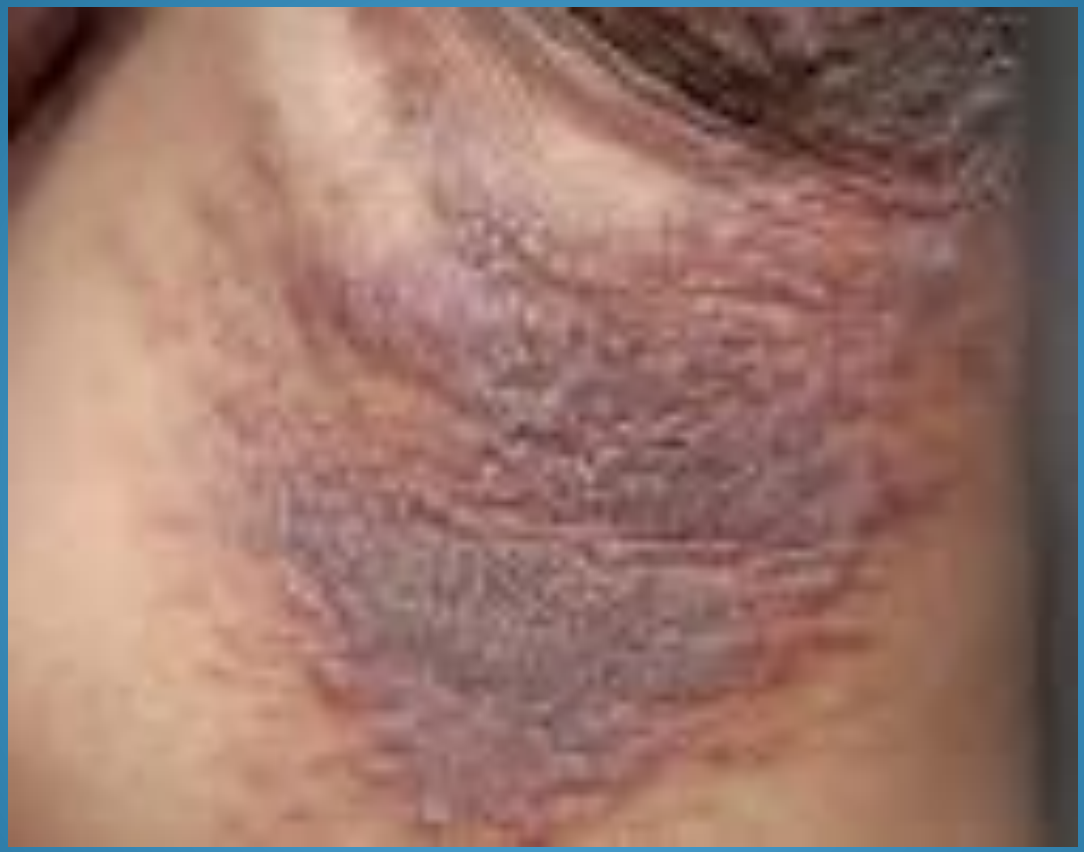

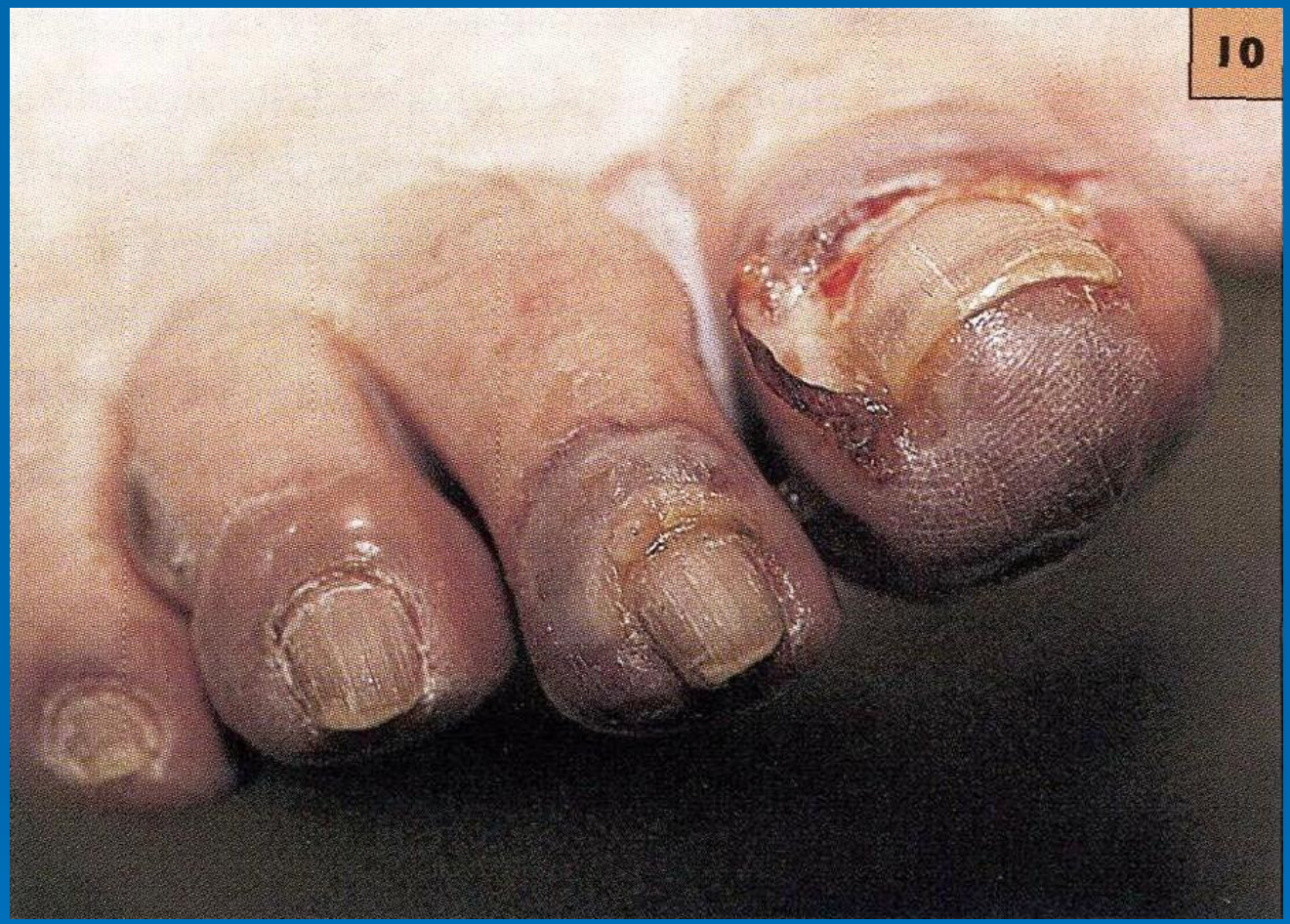
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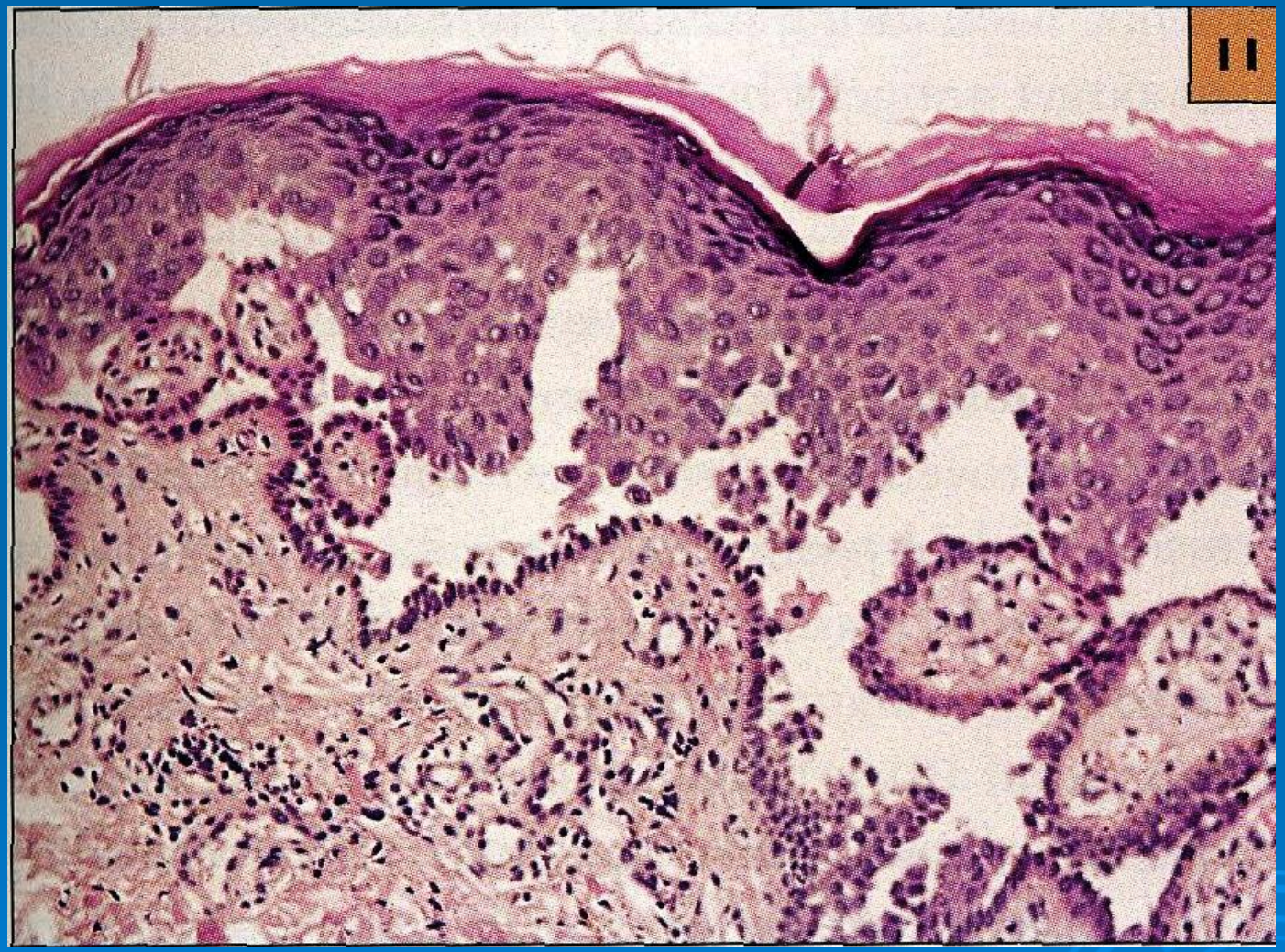
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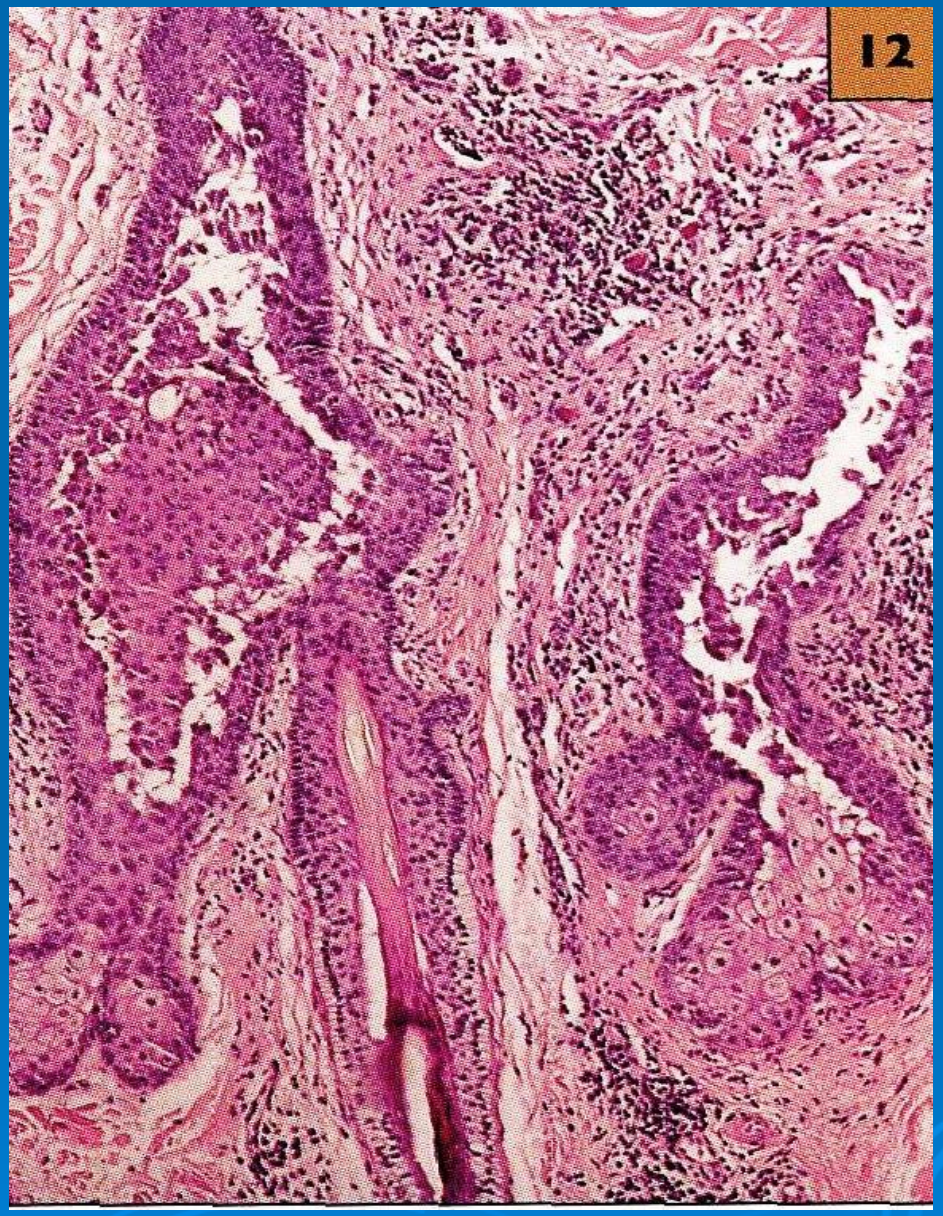
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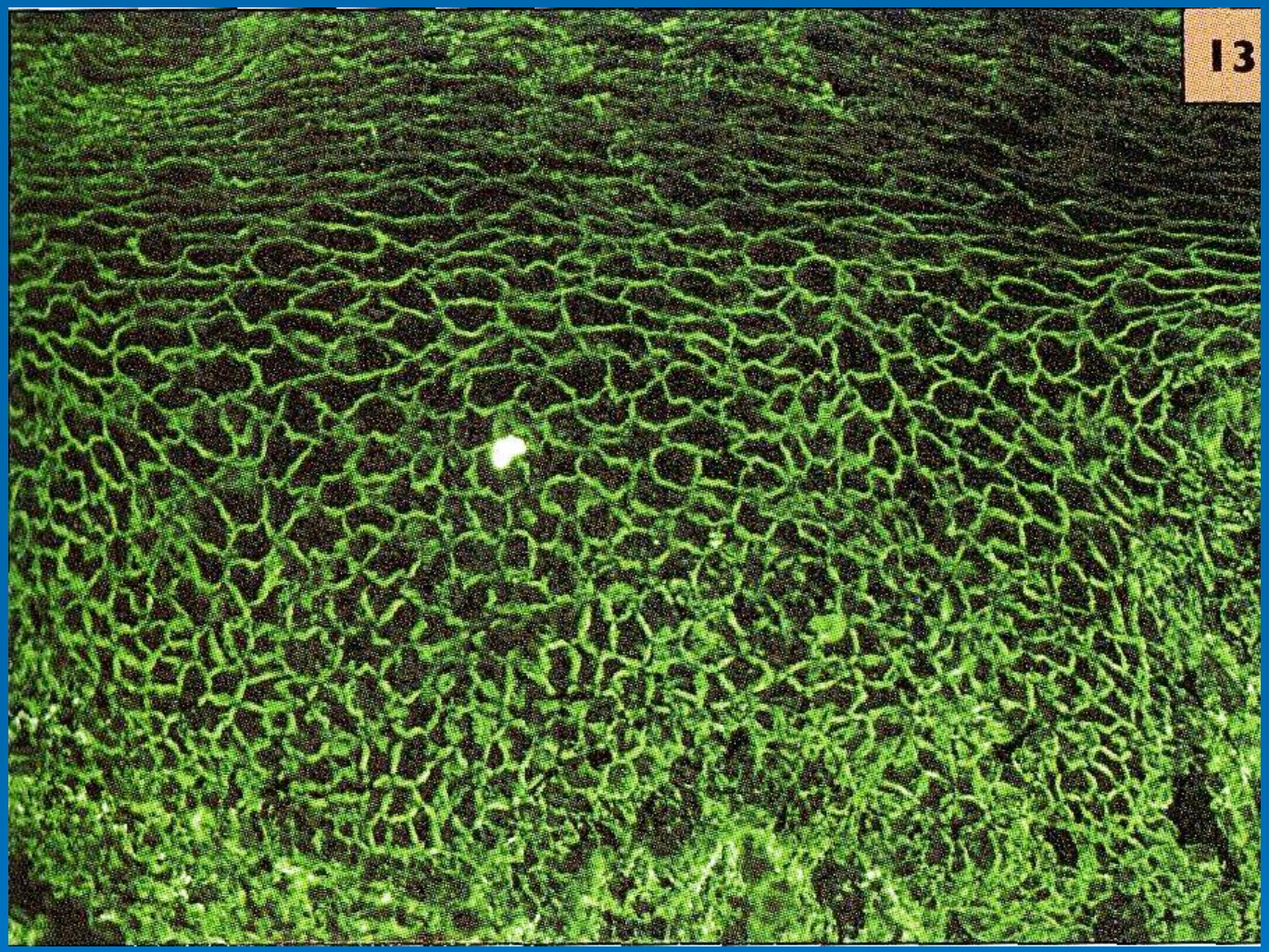
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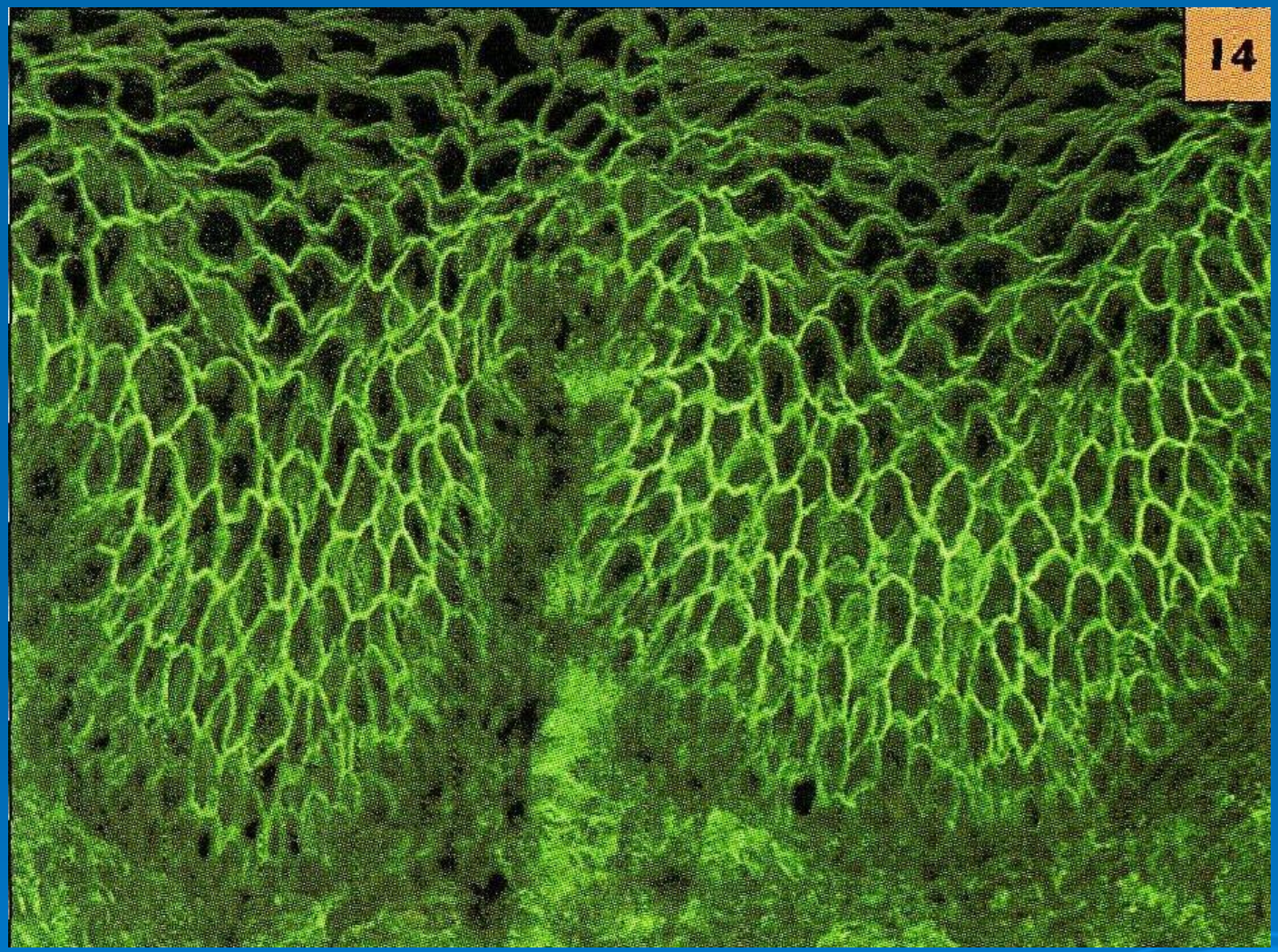
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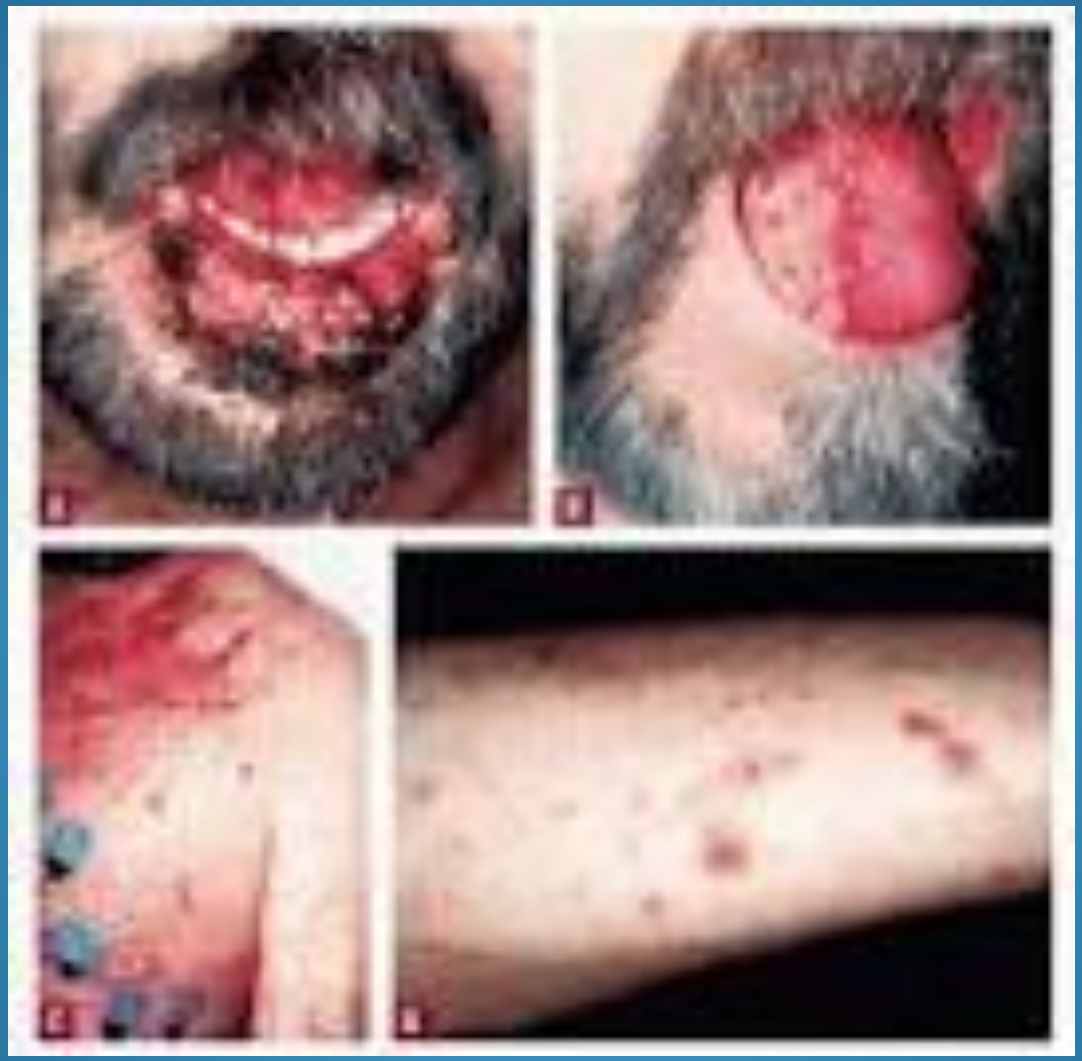


- Skin biopsy – light Mic.
- IF: IgG, C3 in intercellular substance of epid.
- IF: levels reflects the activity of Dis.

Rx

- Corticosteroids
- Immunosuppressive therapy (Azathioprine, mycophenolate, mofetil)
- IVIG
- Anti CD-20, Rituximab

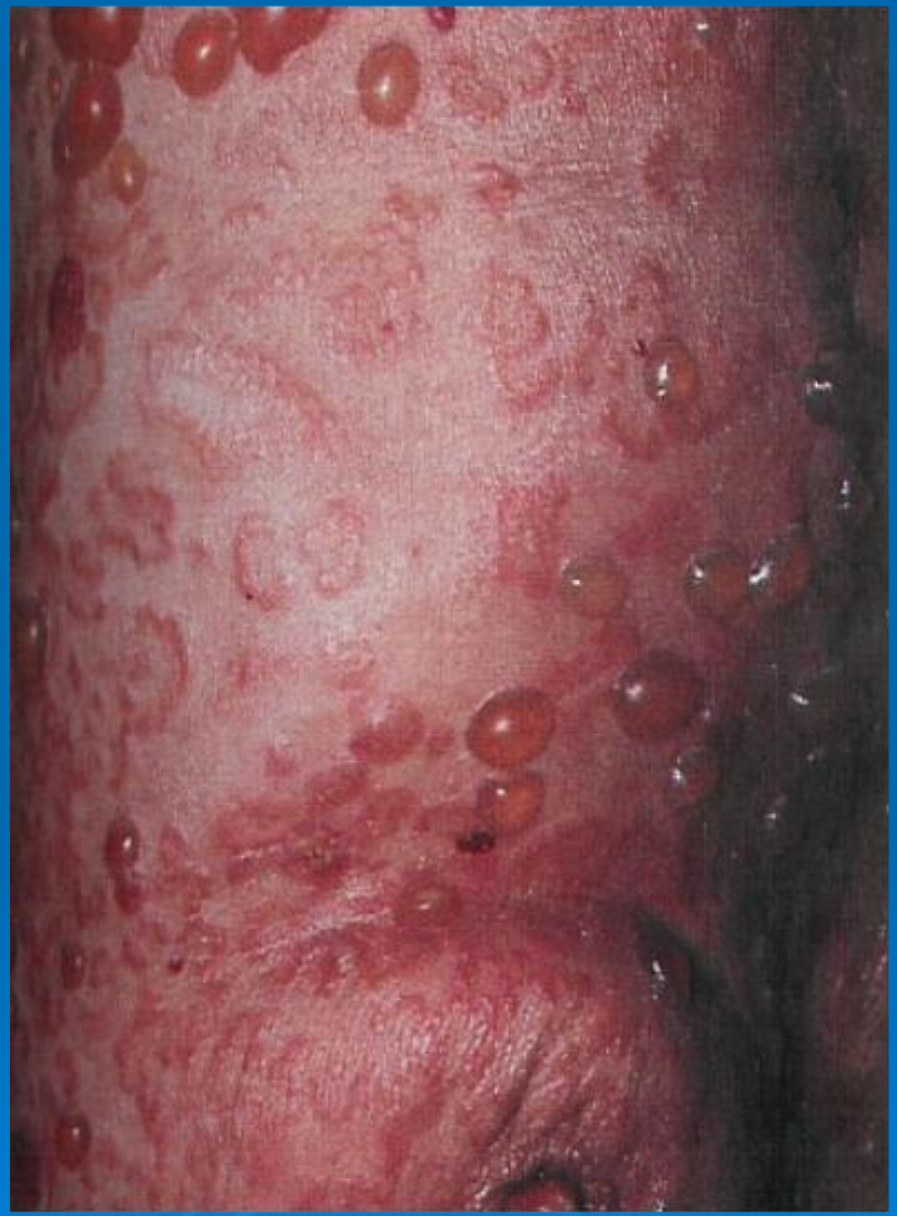
Pemphigoid Group of Dis.

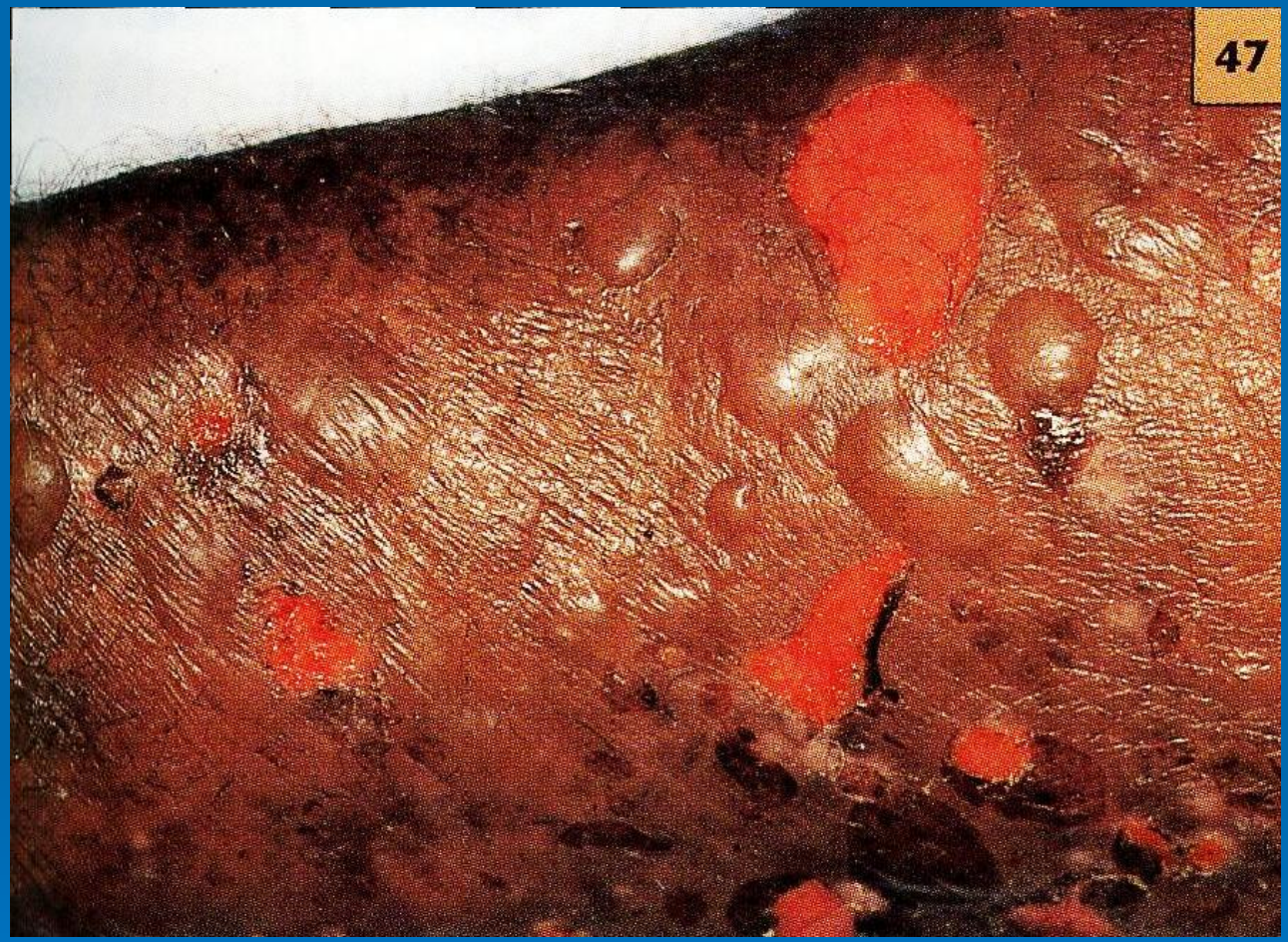
- Bullous pemphigoid, herpes gestations, cicatricial pemphigoid: auto immune sub epidermal blistering Dis. With circ.IgGand BMZ- bound IgG ABS α C_3.

Bullous Pemphigoid

Essentials of Diagnosis:

- Large bullae on erythematous plaques or normal appearing skin.
- No or only mild involv. of mucos.
- Sub epidermal blister
- IgG, C_3 at dermal epid. Junction.
- ABS. against BMZ in patients sera

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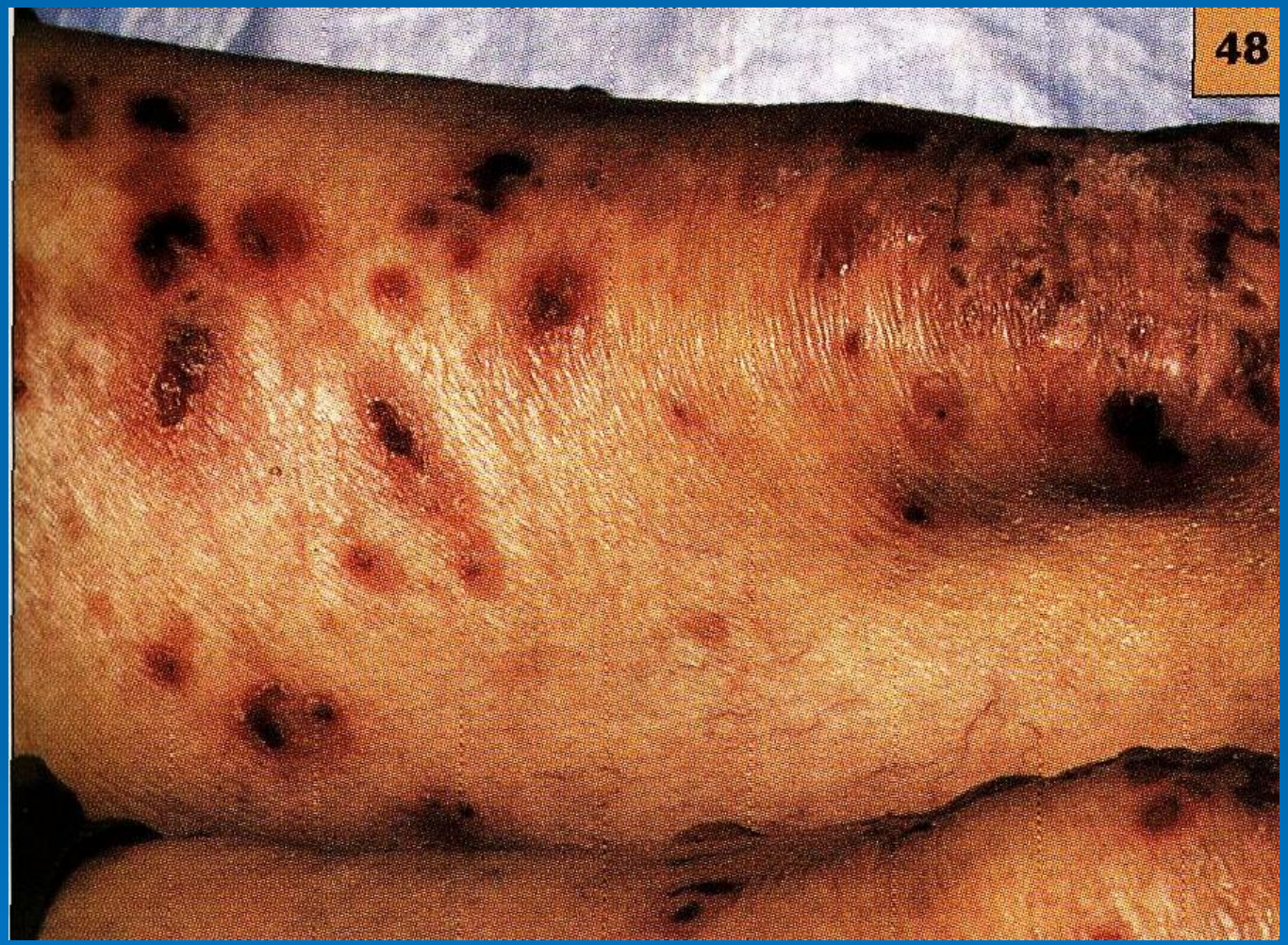
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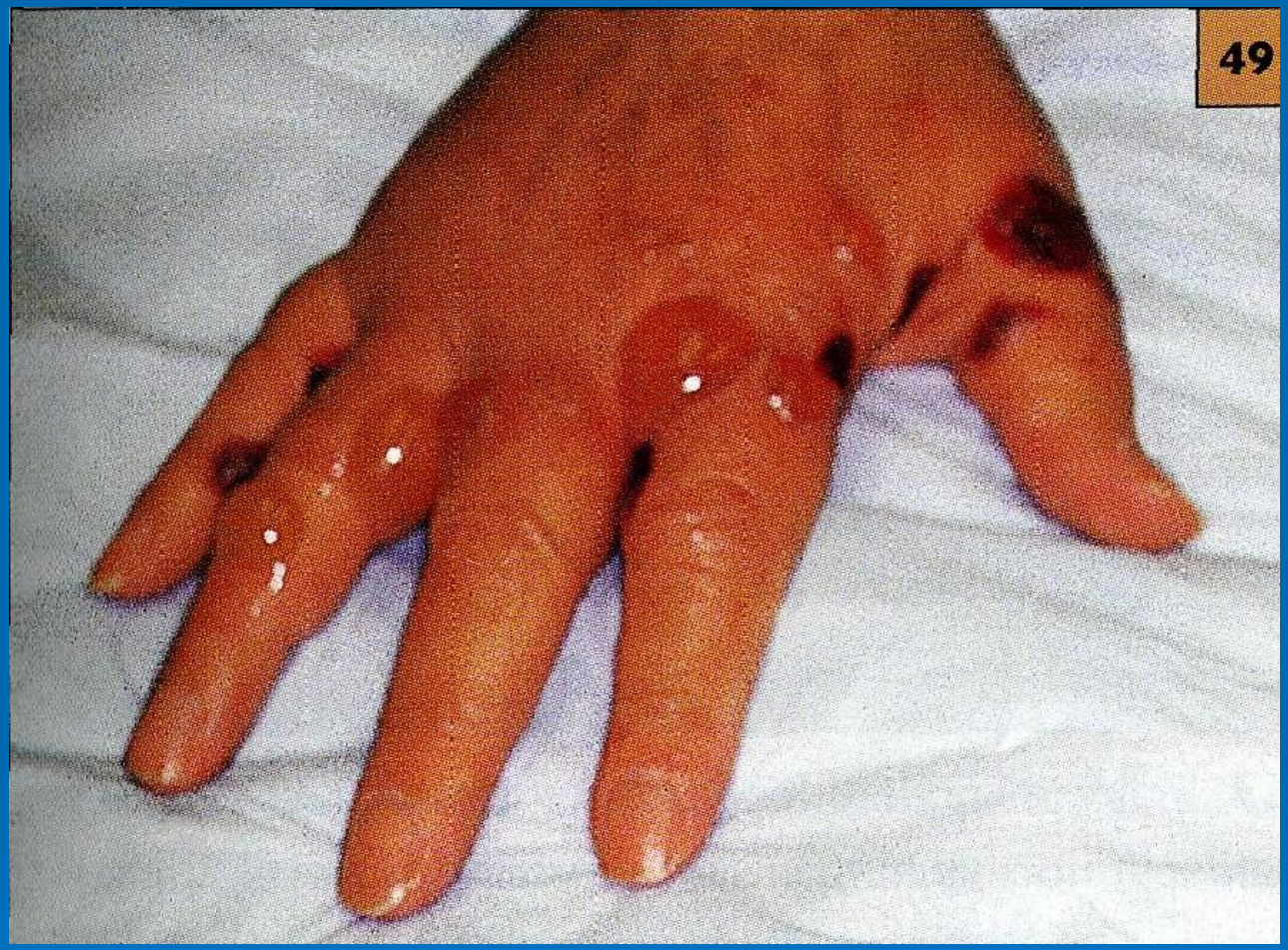
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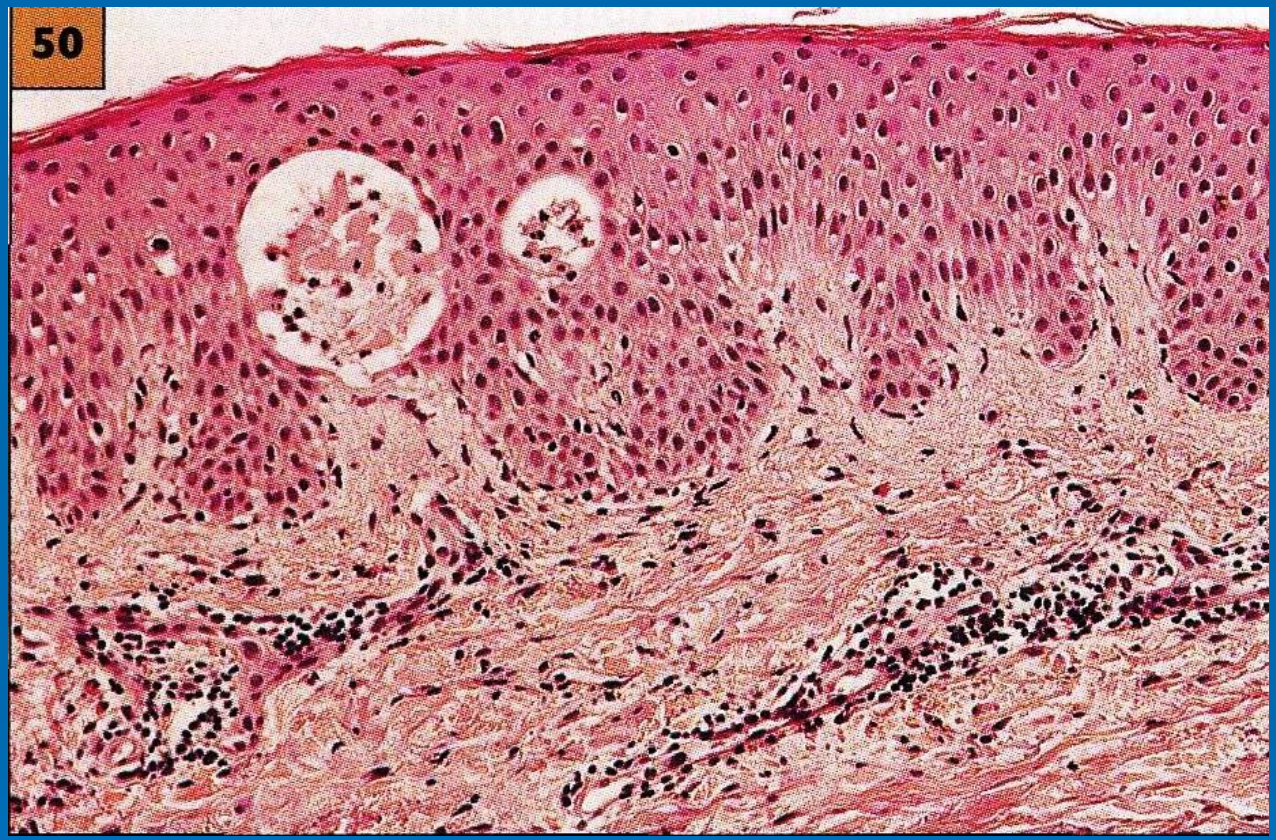
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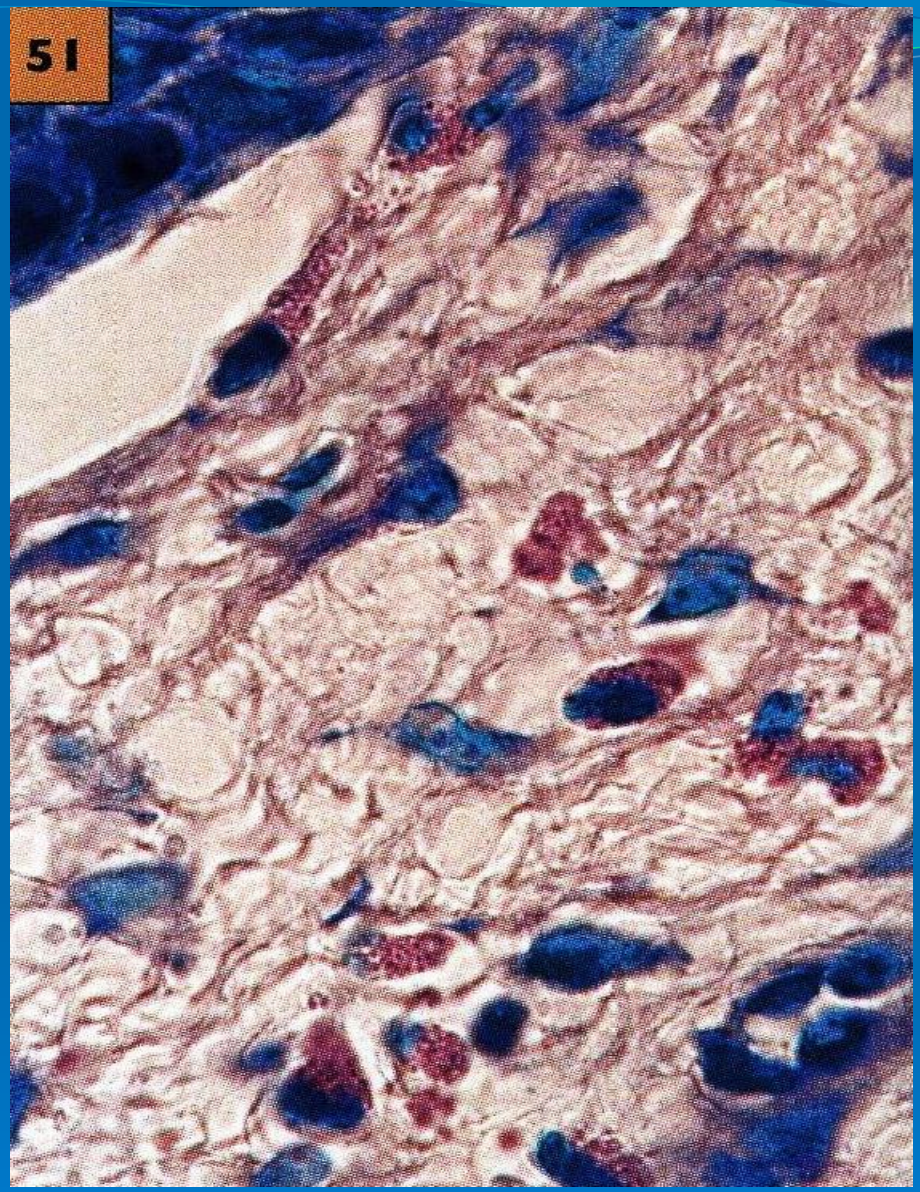
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Age: Elderly

- Relatively Benign

Clinical Manif.:

- Loc Erythema, Urticaria, Pruritus
- Tense blisters, lower part of abdomin, groin, flexors of Arms &

Legs.

- Negative Nikolsky's sign
- Course: Variable, untreated B.P. Localized,Spont. Remission, generalized.

Diagnosis:

- CBC: Eosinophilia - 50%

IgE: - 70%

HIST

DIF

IIF

Rx

- Topical
- Systemic: Steroid
- Antibiotics: Tetracycline, Minocin
- Dapsone
- Immuno Suppressive (Azathioprine, Mycophenolate, Mofetil)
- In resistant cases : ivIg ,anti –CD20 immunotherapy (rituximab)

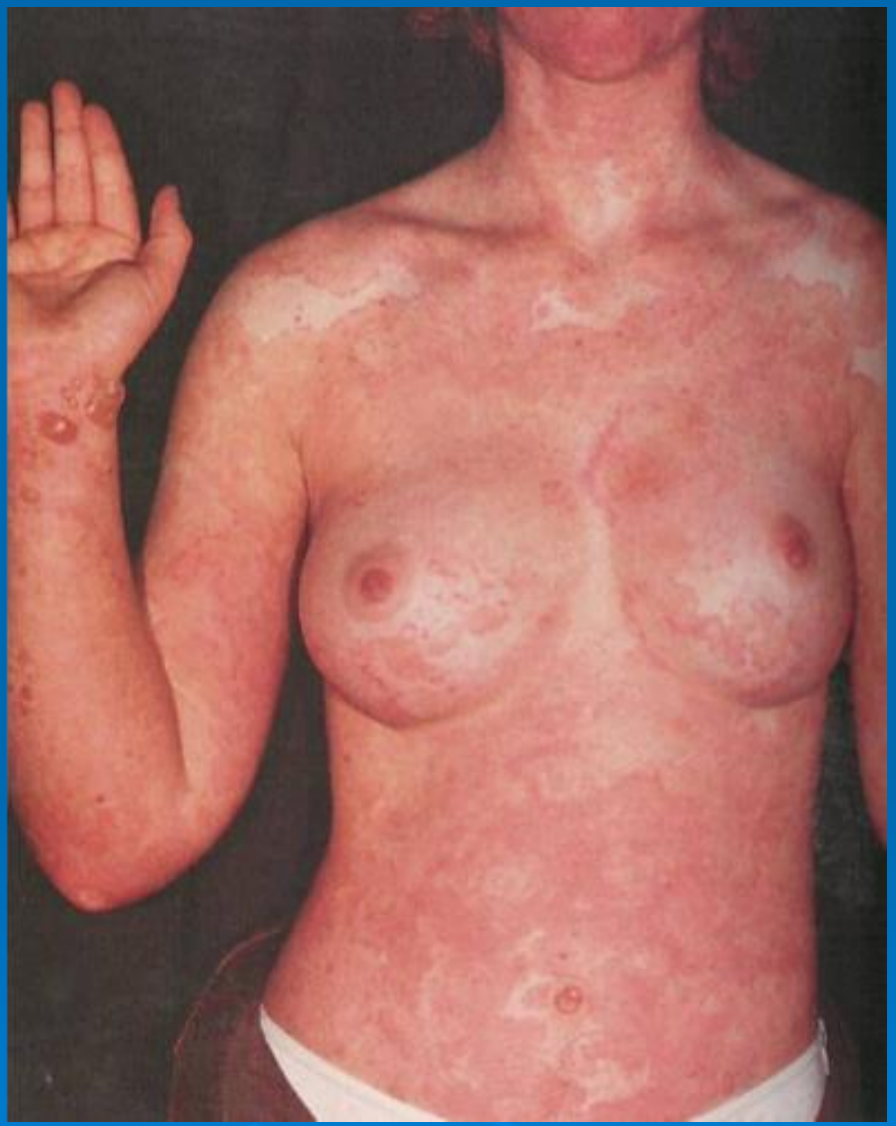
Pemphigoid Group of Dis.
(Pemphigoid Gestationis)

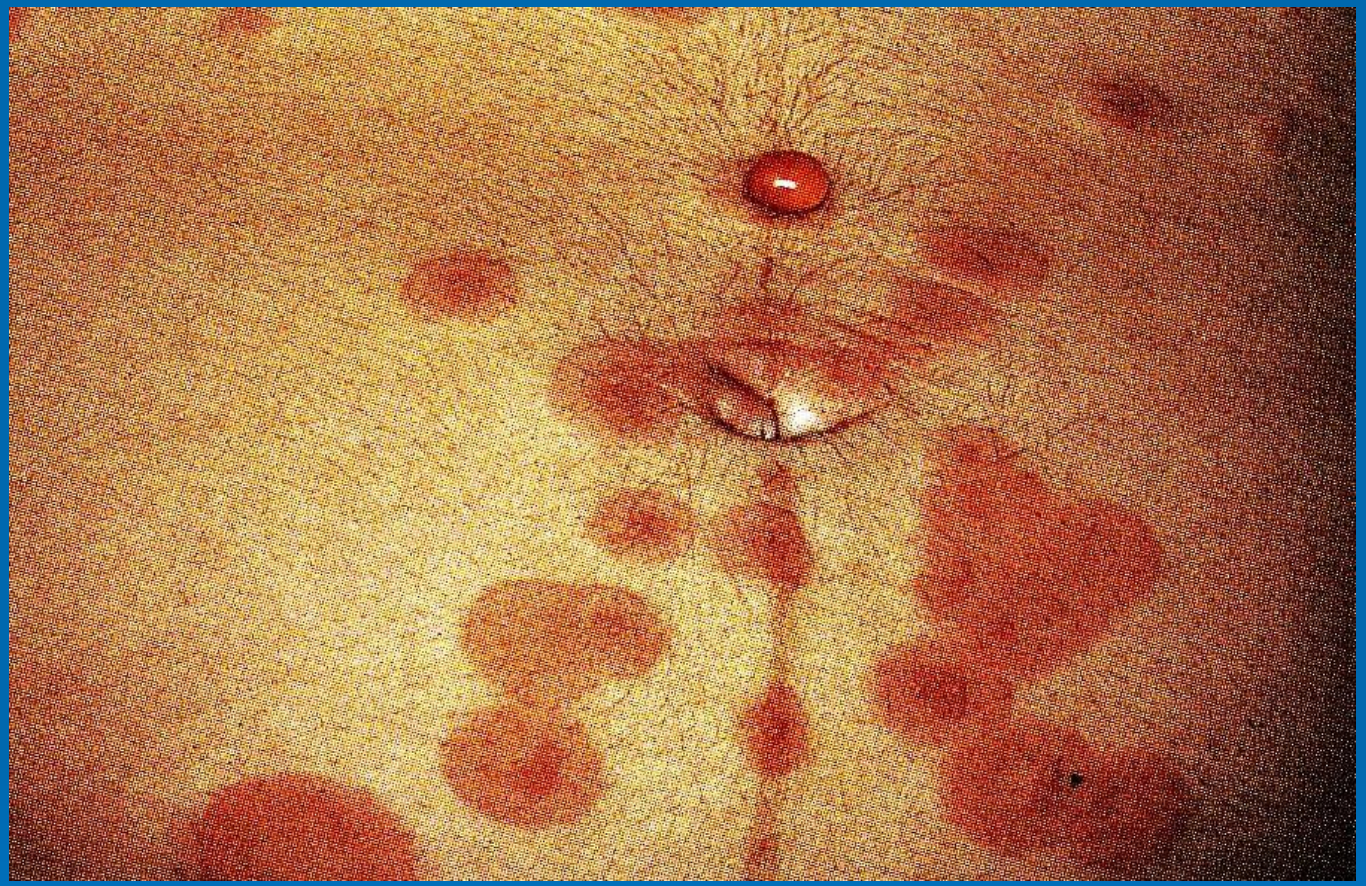
- Erythematous urticarial Plaques, alone or with papules, vesicles

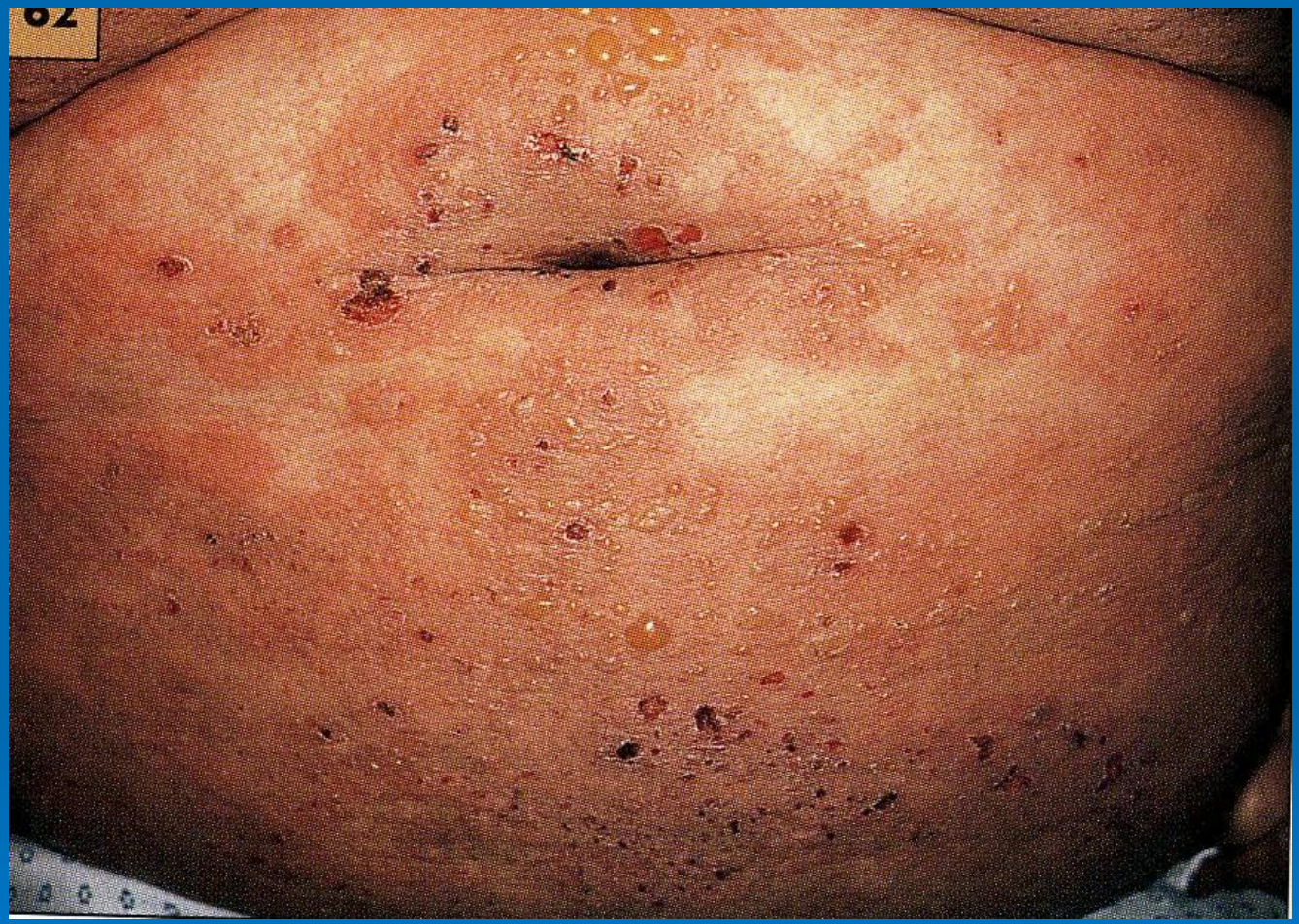
blisters, erosions, most on abdomen, proximal extr.

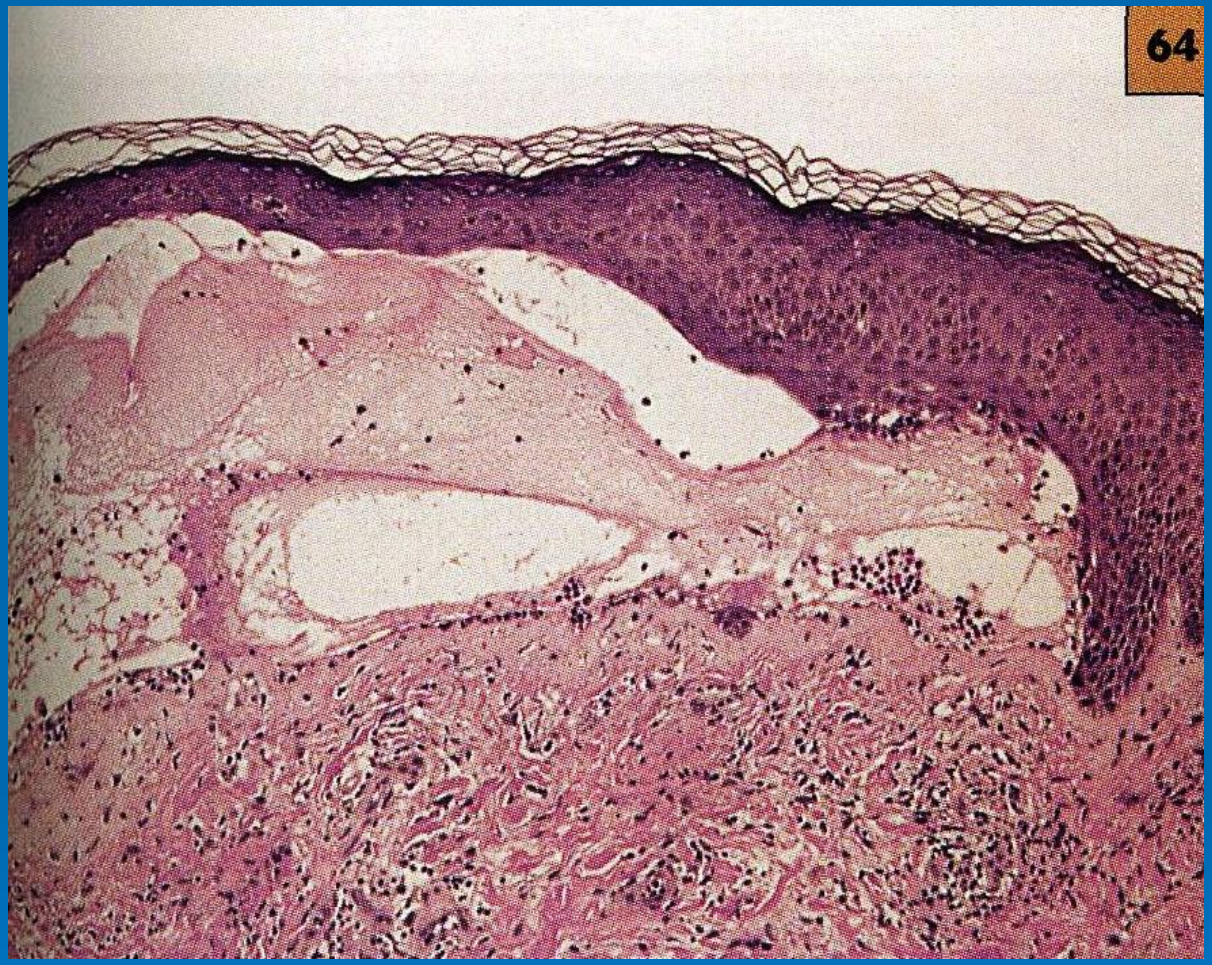
- Intense pruritus
- Sub epidermal blister
- C_3, IgG at BMZ, H.G. Factor in Patient's Sera

2^{nd}, 3^{rd} Trimester, Flares at post partum, OCP





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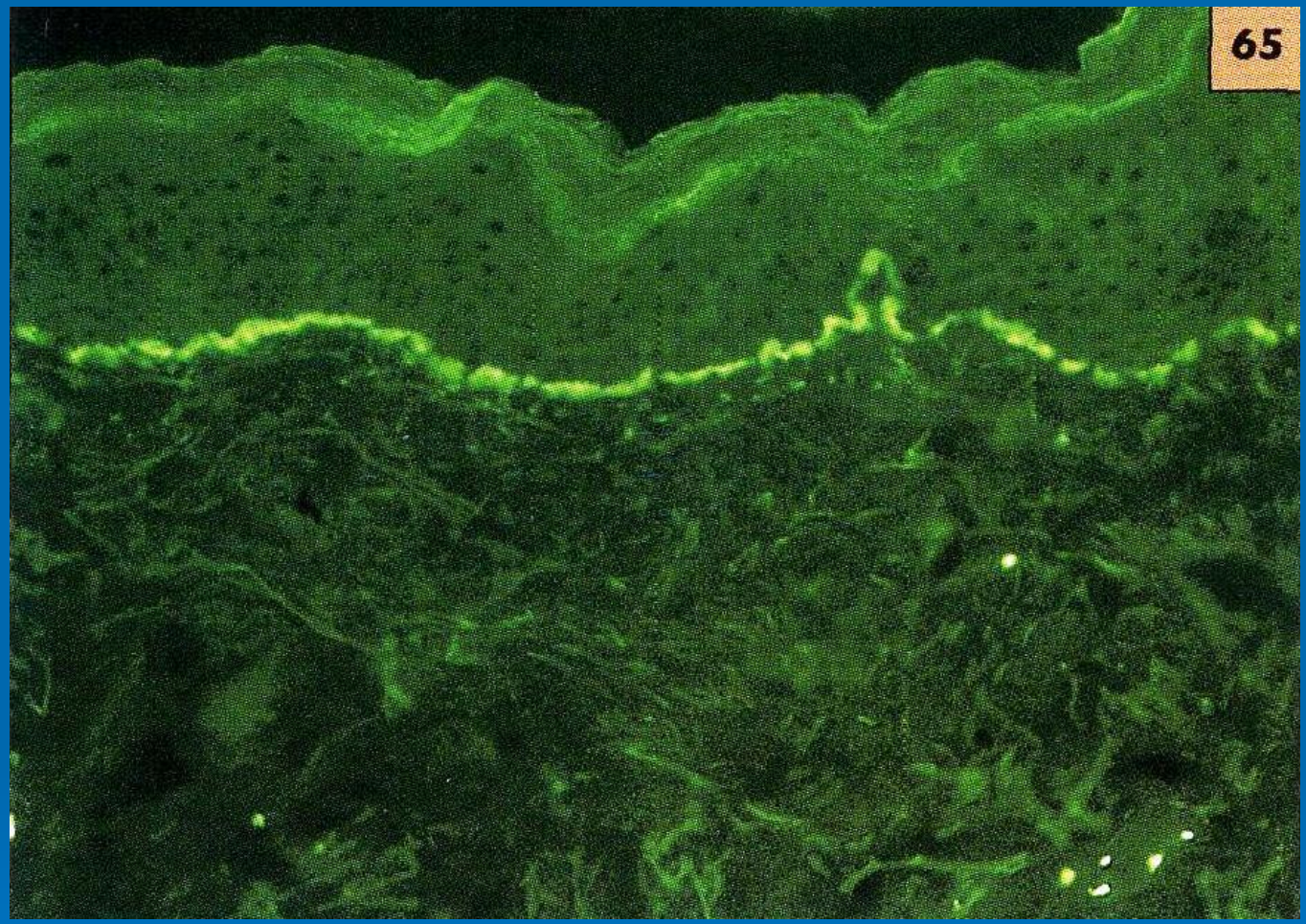
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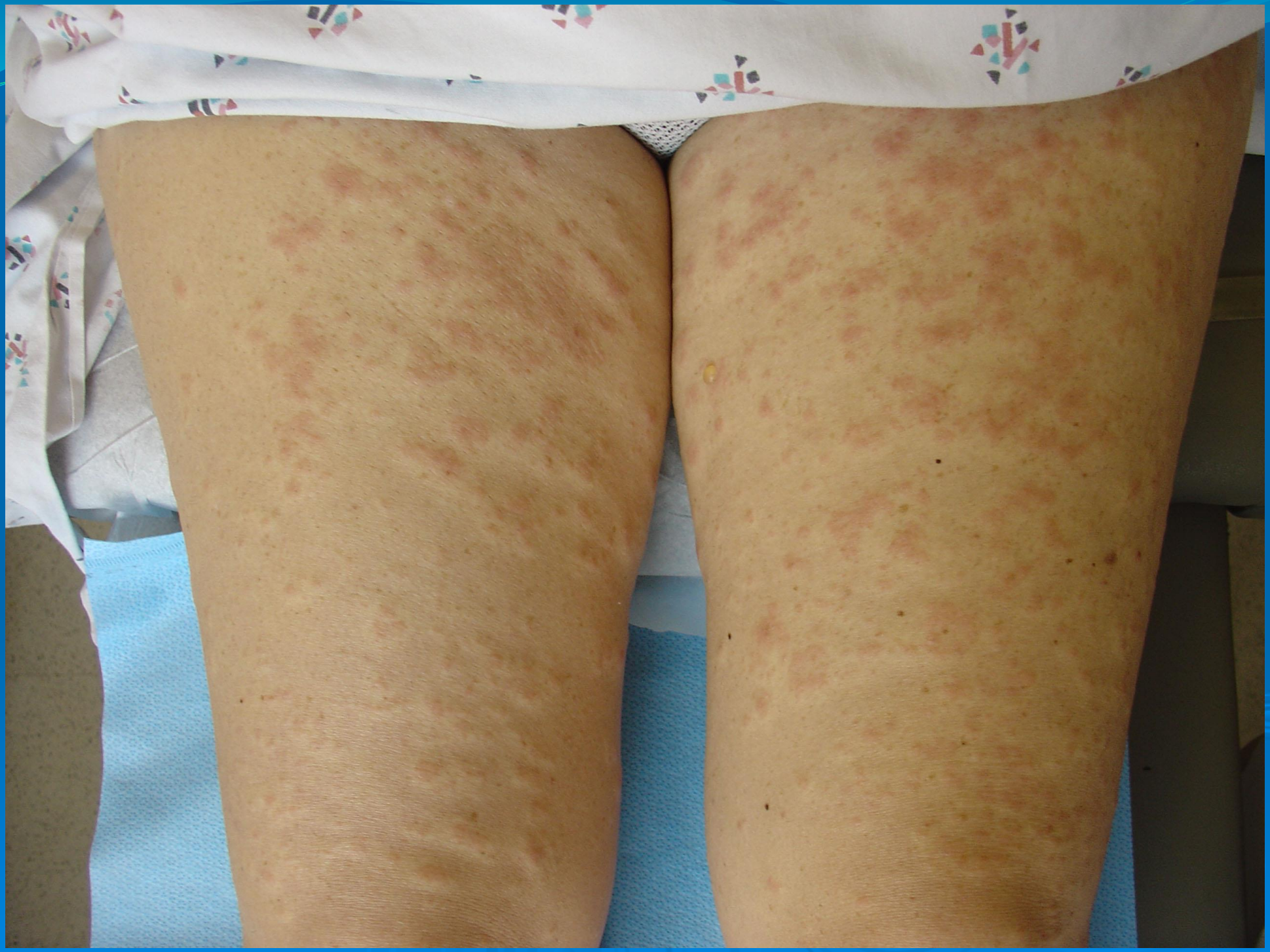
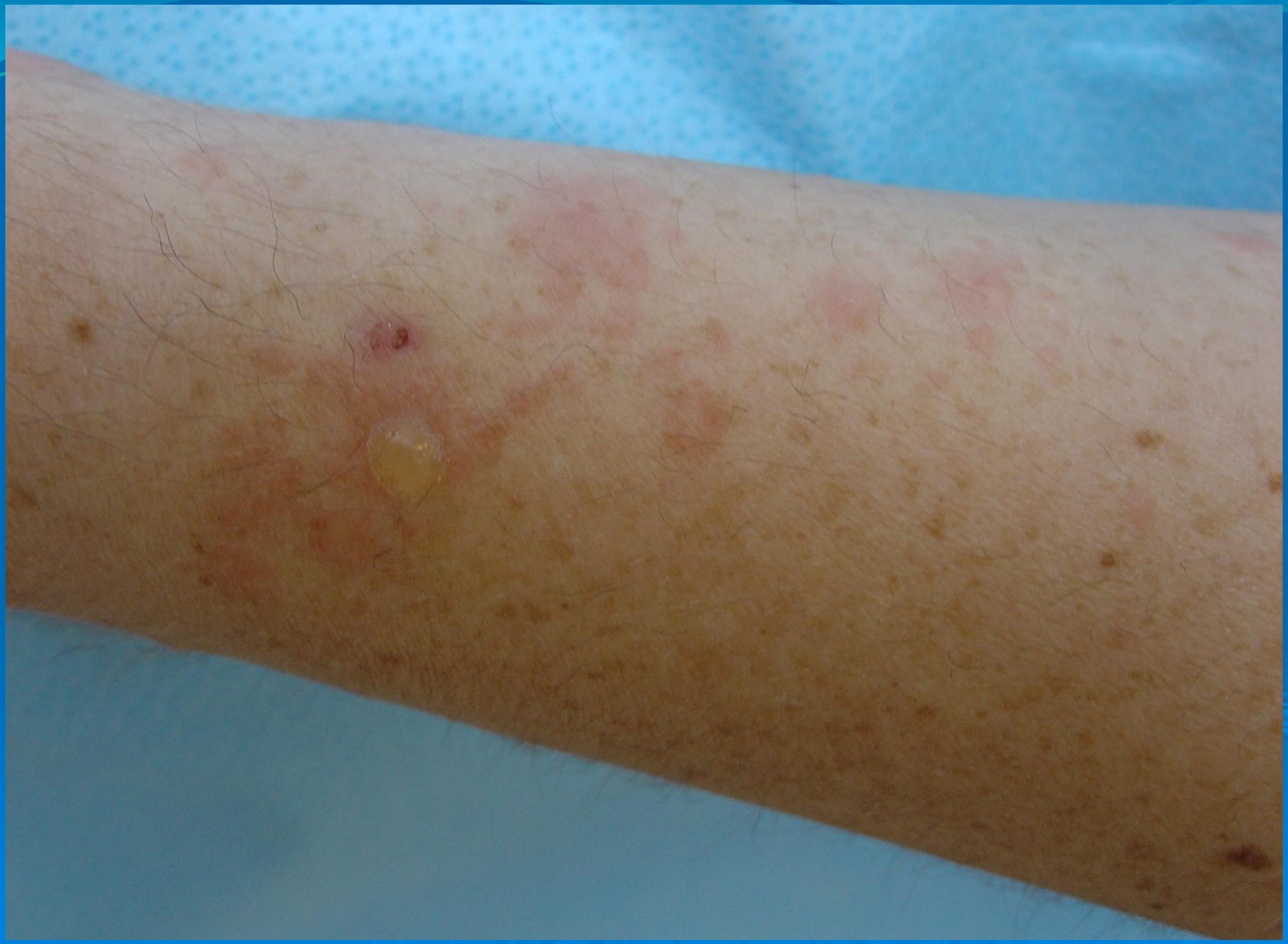
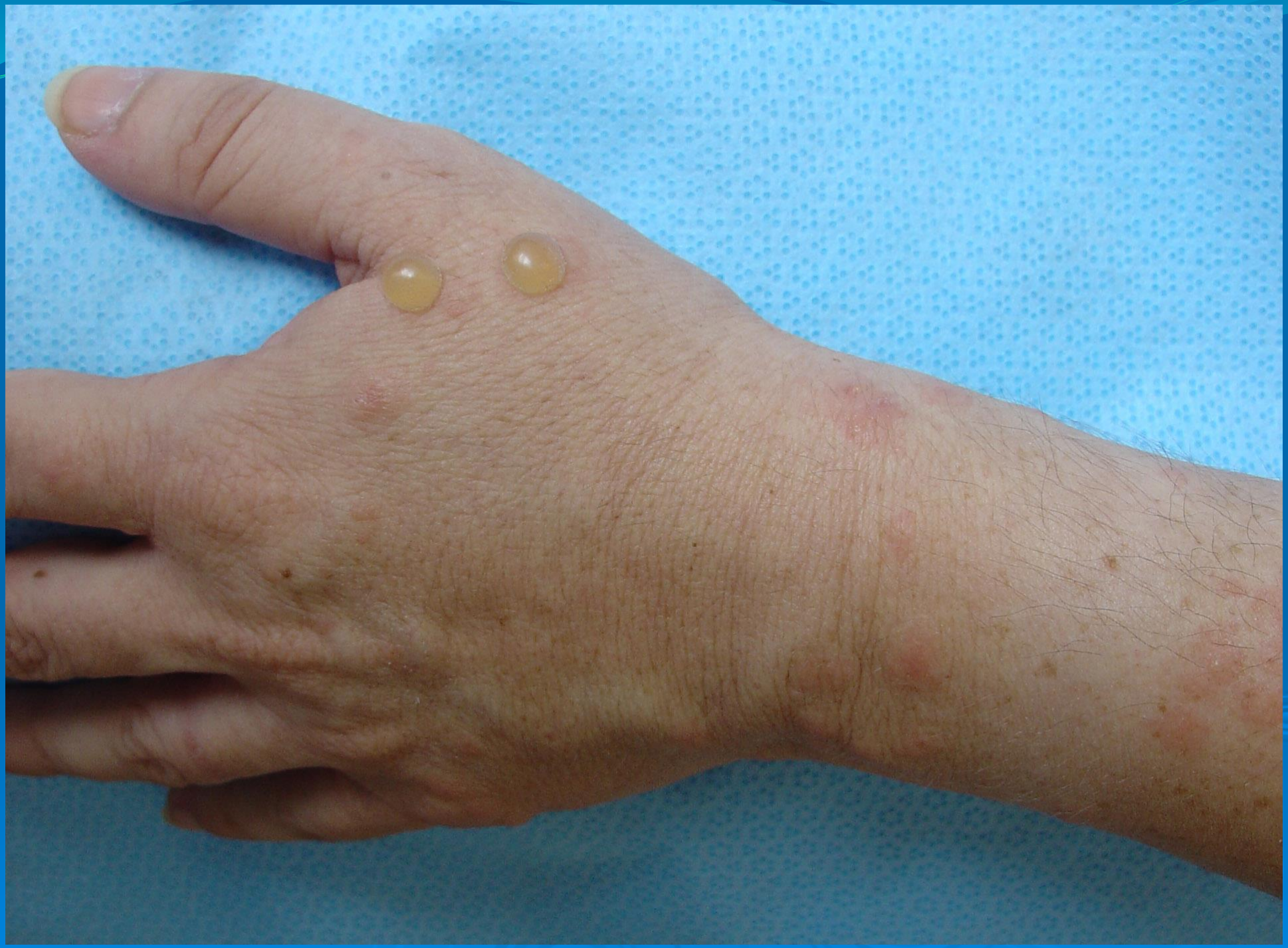
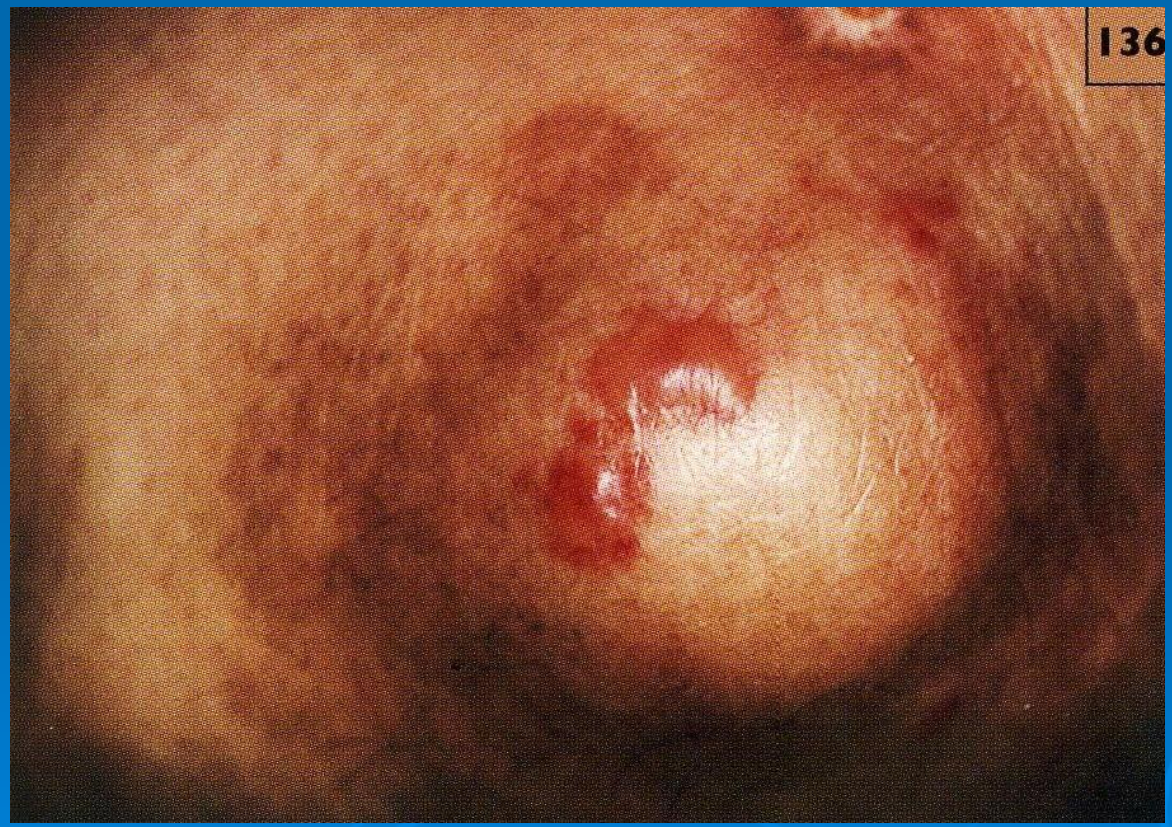


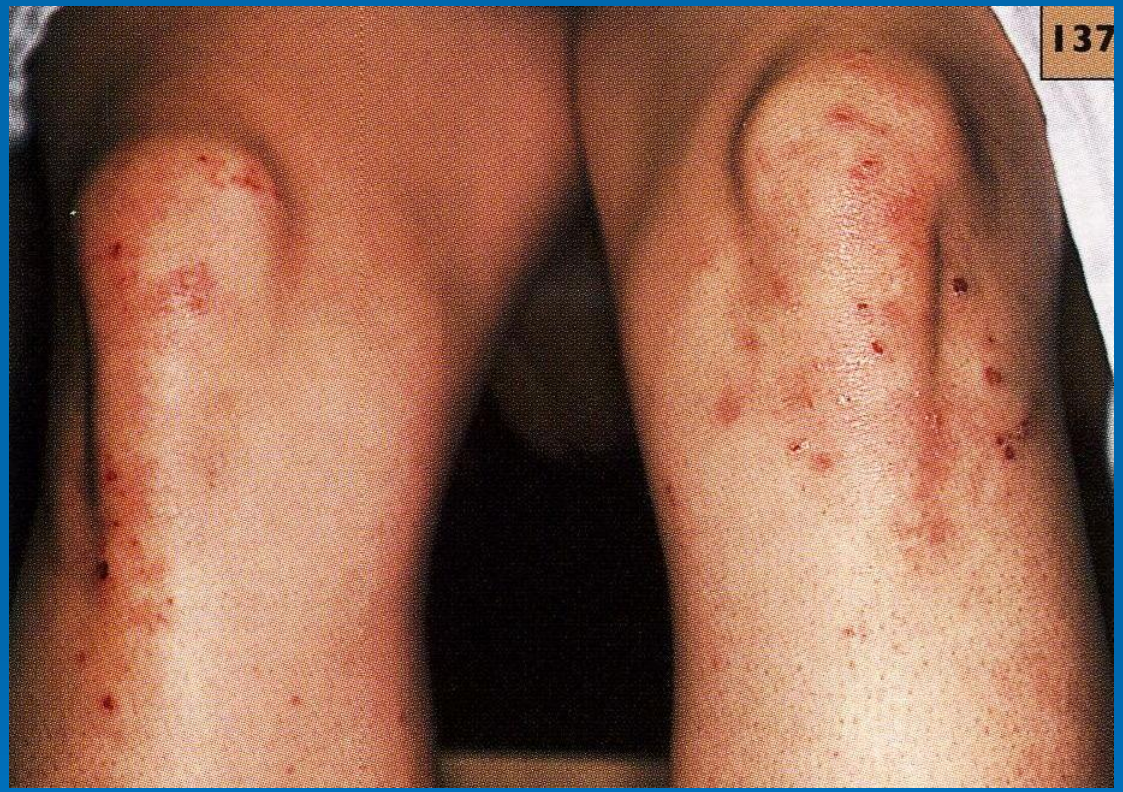




Dermatitis Herpetiformis

- Severe Pruritis
- Associated with gluten sensitive enteropathy
- Pruritic tense blisters on extensors

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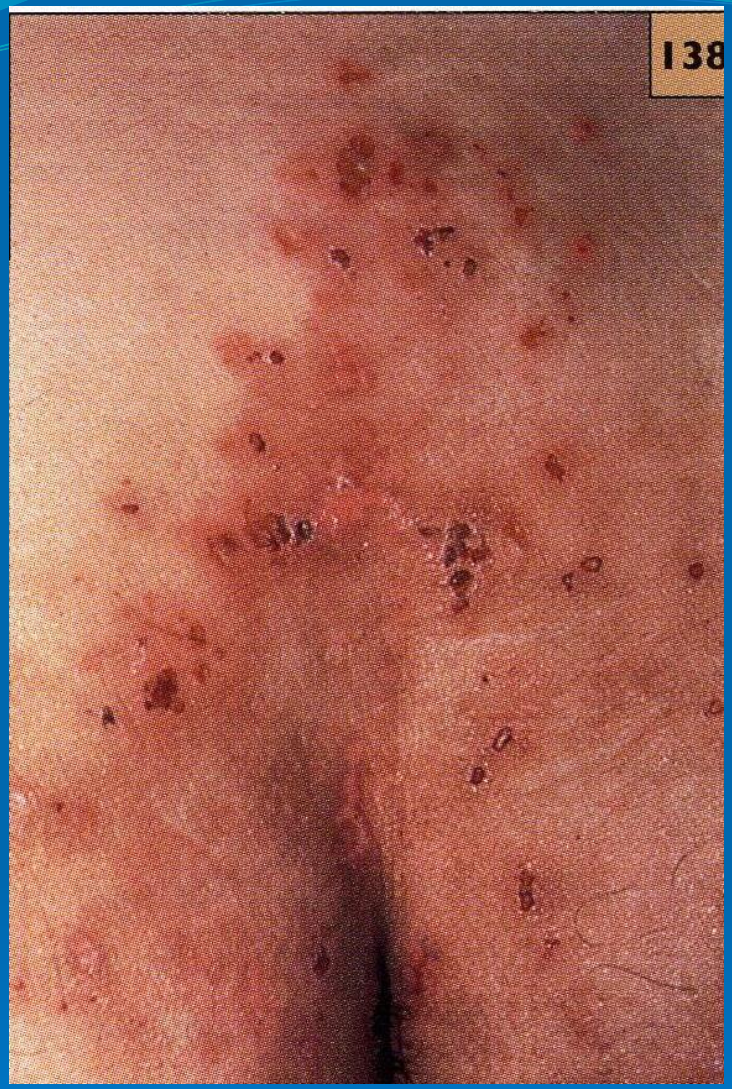
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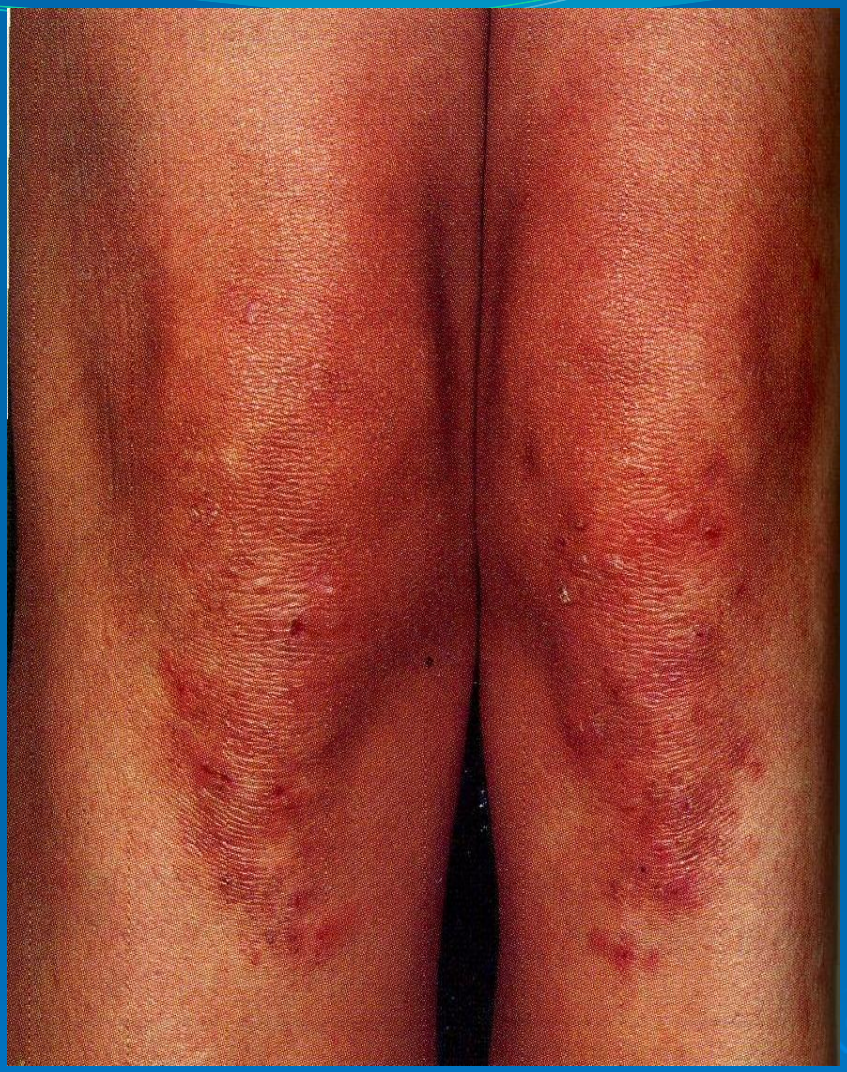
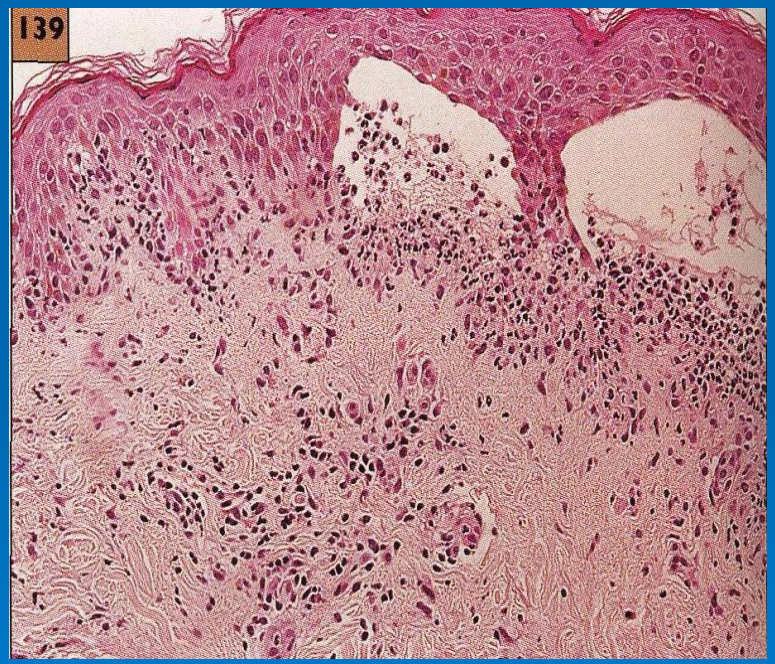
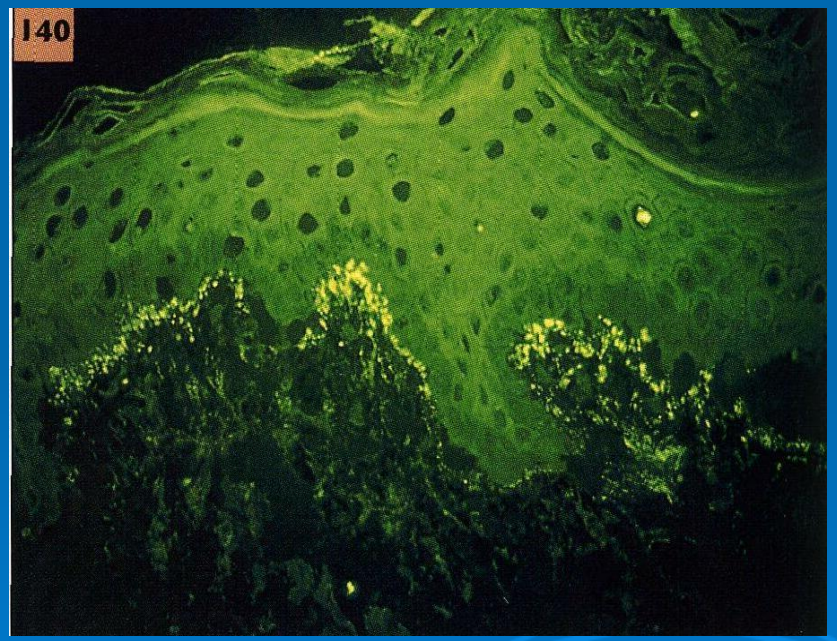


-Phatology

- Sub epidermal blister, prominent neutrophil infiltration
- Dif: granular IgA in dermal papillae





Treatment

- Gluten free diet
- Dapsone – 1st line of treatment
- Sulfa pyridine
- Tetracycline and Nicotinamide

THANK YOU