Dermatiti psoriasiformi e spongiotiche

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In Memory of Raffaele Gianotti

Prof. Raffaele Gianotti ("Raf" for friends) was born on July 7th,1959 in Milan (Italy). His father, Prof. Ferdinando Gianotti (1920-1984), was a famous pediatric dermatologist that gave his name to the "papular acrodermatitis of childhood" (Gianotti-Crosti Syndrome).

Raffaele graduated cum laude in Medicine in 1985 at the University of Milan. In 1988 he completed the residency in Dermatology and in 1992 he got also his second specialization in Surgical Pathology. Soon after in 1993, he made a fellowship in Dermatopathology by Bernie Ackerman in New York. Back again in Milan, he started the work of his life of in Dermatopathology with a full researcher position at Istituto di Scienze Dermatologiche Fondazione Ospedale Maggiore Policlinico, Mangiagalli e Regina Elena. Between 2000-2005 he collaborated with H. Kutzner in Friedricshafen (Germany) on Lampyris101, an automatized software for the diagnosis of inflammatory skin diseases. In 2005, he also got the International Board Certification in Dermatopathology.

As academic, he published over 135 scientific papers on various topics on inflammatory and neoplastic skin diseases and reached an H-index of 27 (source: Scopus, last visited April 5, 2021). Particularly, he was recently very active in describing the histological features of COVID-19 infection in the skin and became very popular reporting the first proved Italian patient affected by COVID-19.[1-4]

Raf was also author of Dermosprint (https://www.dermosprint.com/), a freely available Dermatopathology collection with contributions by worldwide best dermatopathologists.

He passed away on March 27, 2021 at the age of 61.

In his memory, we decided to publish posthumous in Dermatology Reports an editorial that Raf wrote for Dermosprint.

Ciao Raf!
Rest in peace.

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[2]Consultant Pathologist at Lifebrain s.r.l. Limena, Padua (Italy)

Raf Gianotti with both his work & hobby: dermatopathology and motorbike.

References

1. Crosti A, Gianotti F. Dermatose éruptive acro-située d'origine probablement virosique [Eruptive dermatosis of probable viral origin situated on the acra]. Dermatologica 1957;115:671-7.
2. Gianotti R. SARS-CoV-2 and the skin, a hidden treasure. Dermatol Rep 2020;12: 8881.
3. Gianotti R, Barberis M, Fellegara G, et al. COVID-19-related dermatosis in November 2019: could this case be Italy's patient zero? Br J Dermatol 2021 Jan 7. doi: 10.1111/bjd.19804.
4. Gianotti R, Recalcati S, Fantini F, et al. Histopathological Study of a Broad Spectrum of Skin Dermatoses in Patients Affected or Highly Suspected of Infection by COVID-19 in the Northern Part of Italy: Analysis of the Many Faces of the Viral-Induced Skin Diseases in Previous and New Reported Cases. Am J Dermatopathol 2020;42:

Patterns istopatologici

Pattern= modello, struttura

Hermann Pinkus

Hermann K. B. Pinkus, M.D.
1905–1985

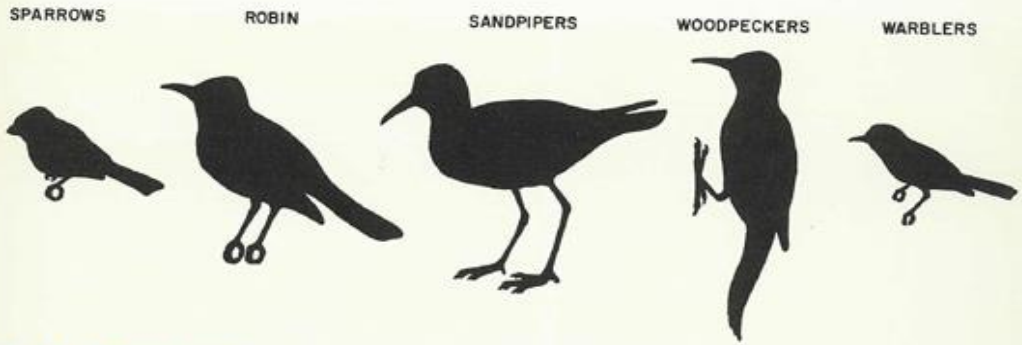


FIG. 5-1. Patterns of major families of birds. Note variation in size and shape of beak, body, wing, and tail.

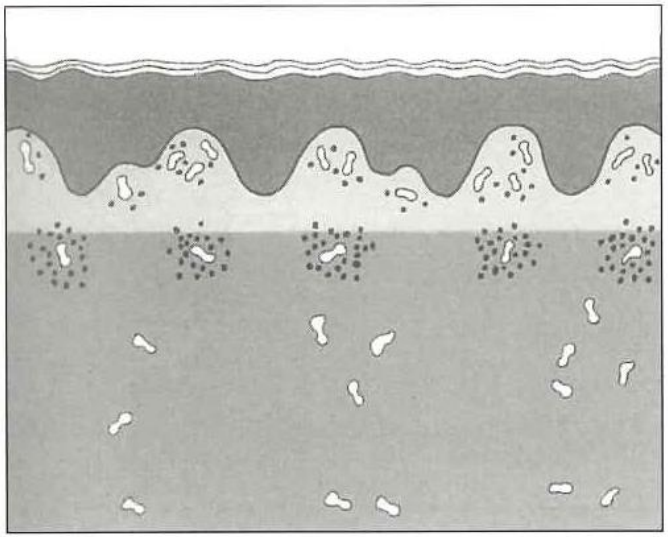
scalp. In addition, other diseases favor particular sites, e.g., necrobiosis lipoidica and erythema nodosum, the skin over the anterior tibiae; granuloma faciale and seborrheic dermatitis, the face; and dermatofibroma and nodular fasciitis, the limbs. Thus, by viewing the entire specimen and looking for the telltale topographic markers indicated in Chapter 1, one can make a reasonable judgment about the site from which the biopsy specimen was obtained, which in itself narrows diagnostic possibilities.

Continuing to use the scanning objective, the microscopist should next note the location of the pathologic changes. Are they in the epidermis, dermis, subcutaneous fat, or in a combination of these? When studying a particular histologic section, he must routinely ask himself: "Where is the dominant pathologic change and what is the pathologic pattern?" The histologic expressions of inflammatory skin diseases have been conveniently divided into nine strikingly different major patterns, usually discernible with the scanning objective.

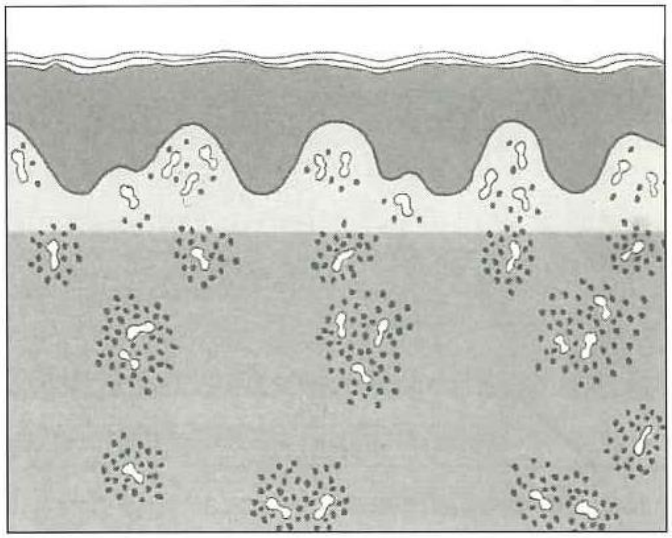
FIG. 5-2. White-throated sparrow and song sparrow, members of the same species. By noting subtle characteristic differences, separation can be made.

THE NEW SCHEMA (2004) IS AS FOLLOWS:

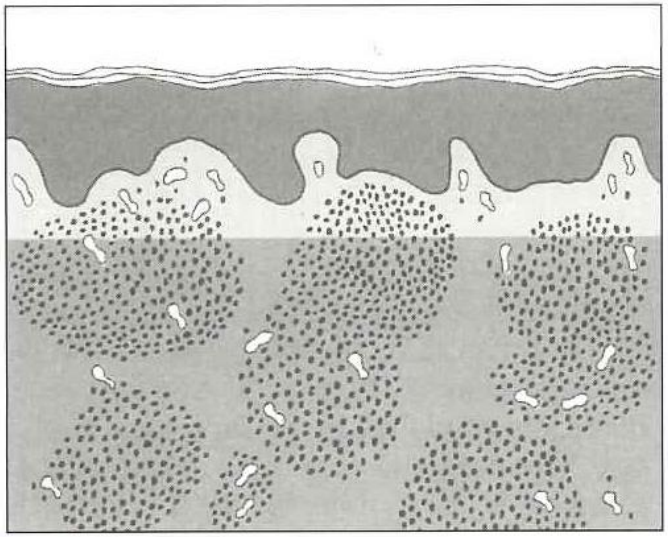
SKIN



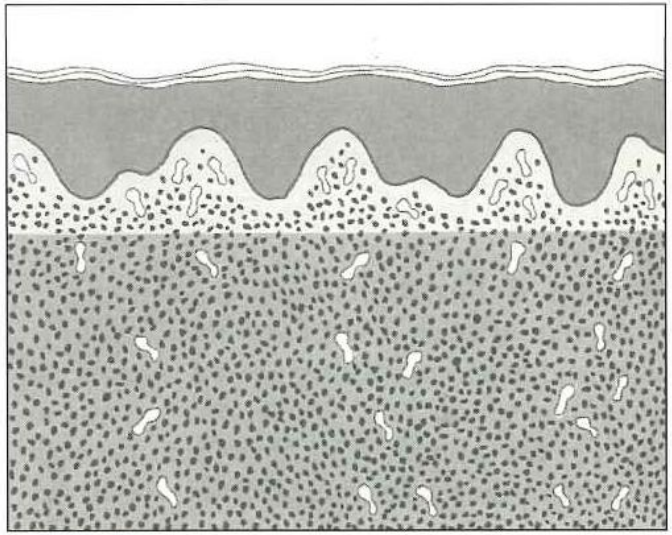
5.10A *Perivascular dermatitis, superficial*



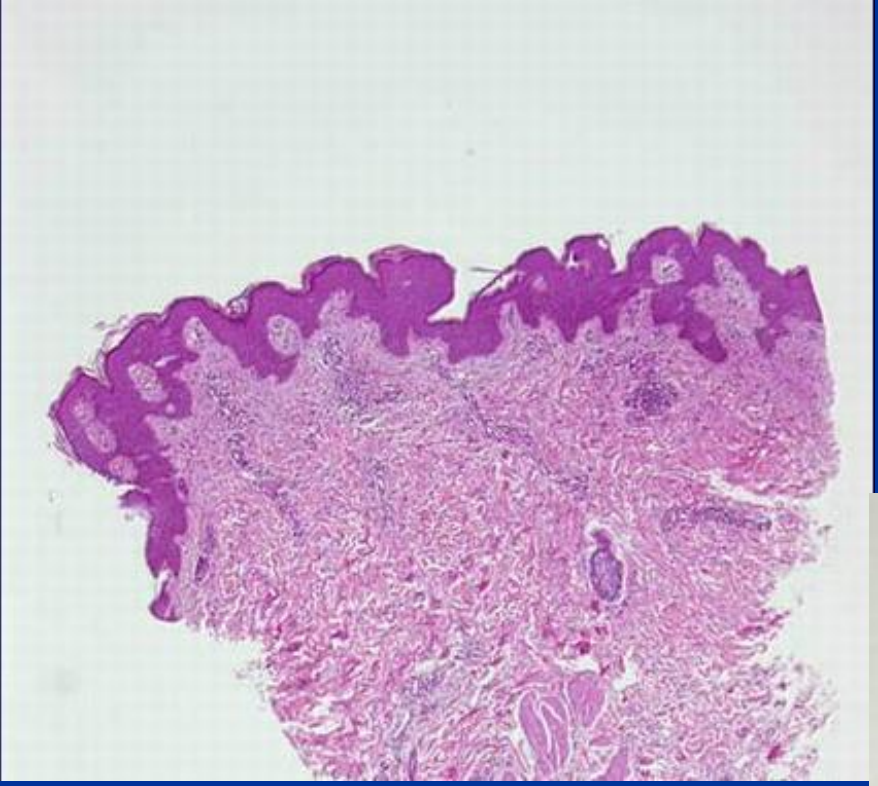
5.10B *Perivascular dermatitis, superficial and deep*

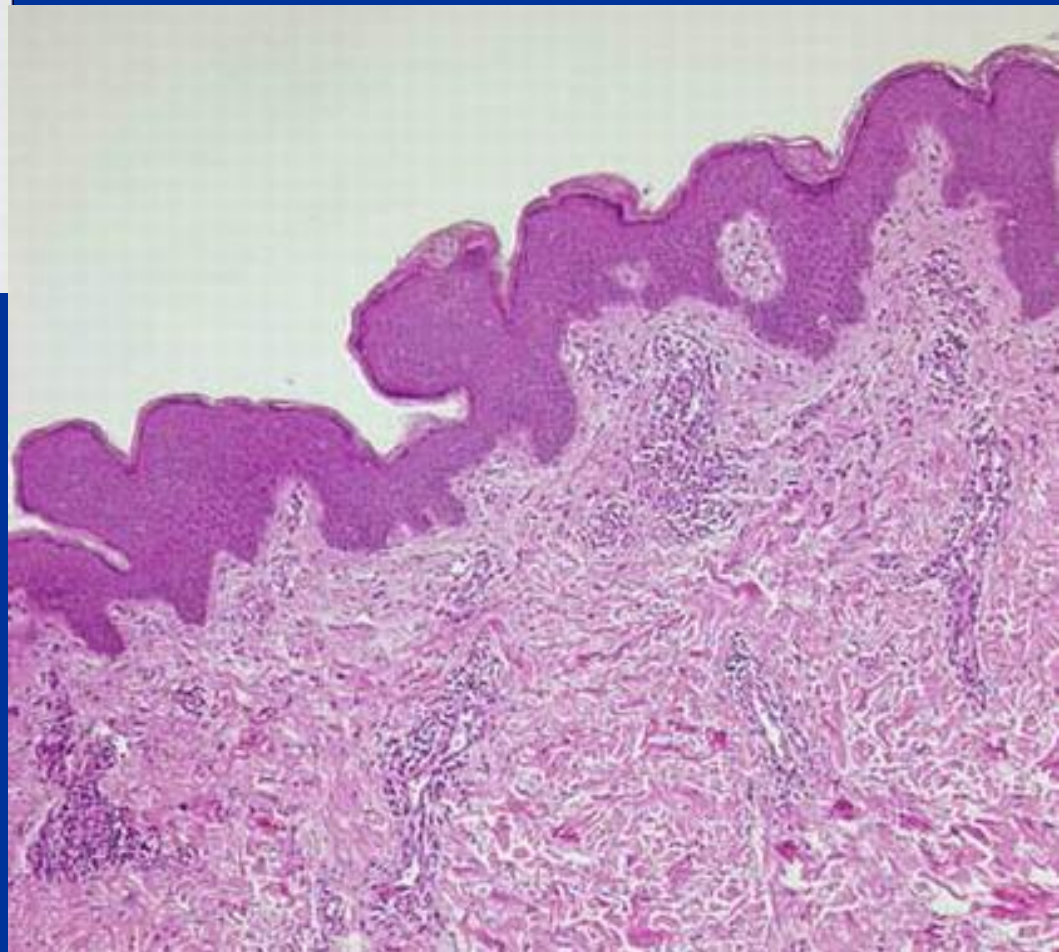


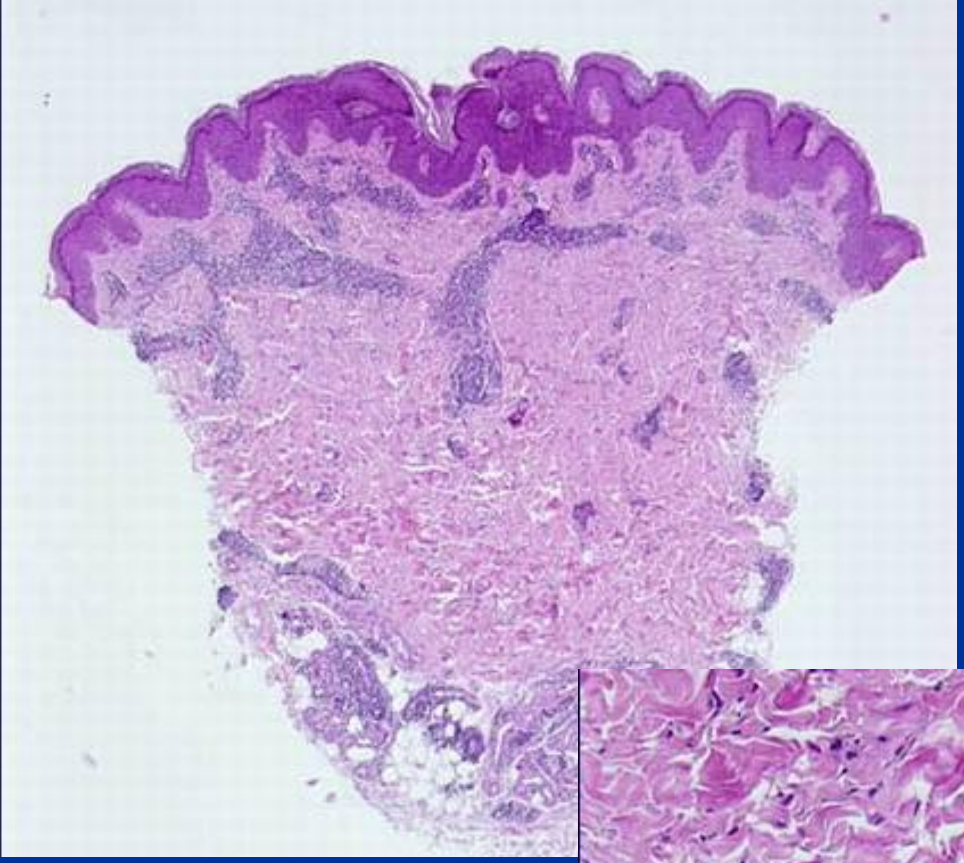
5.11A *Nodular dermatitis*

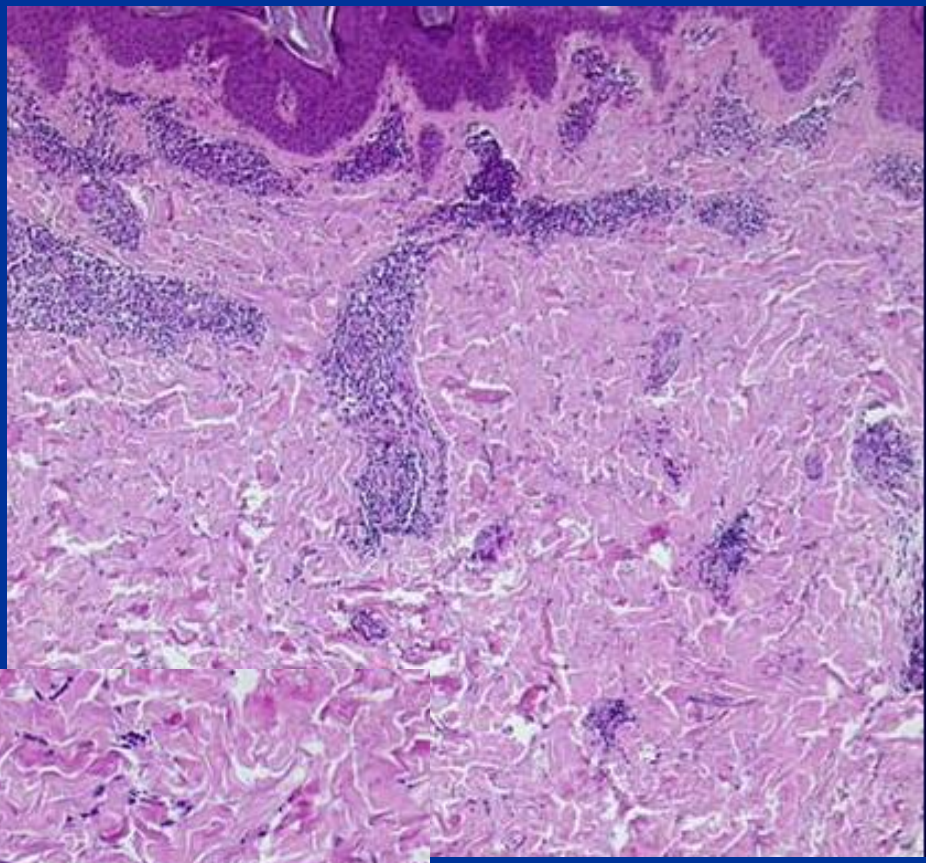


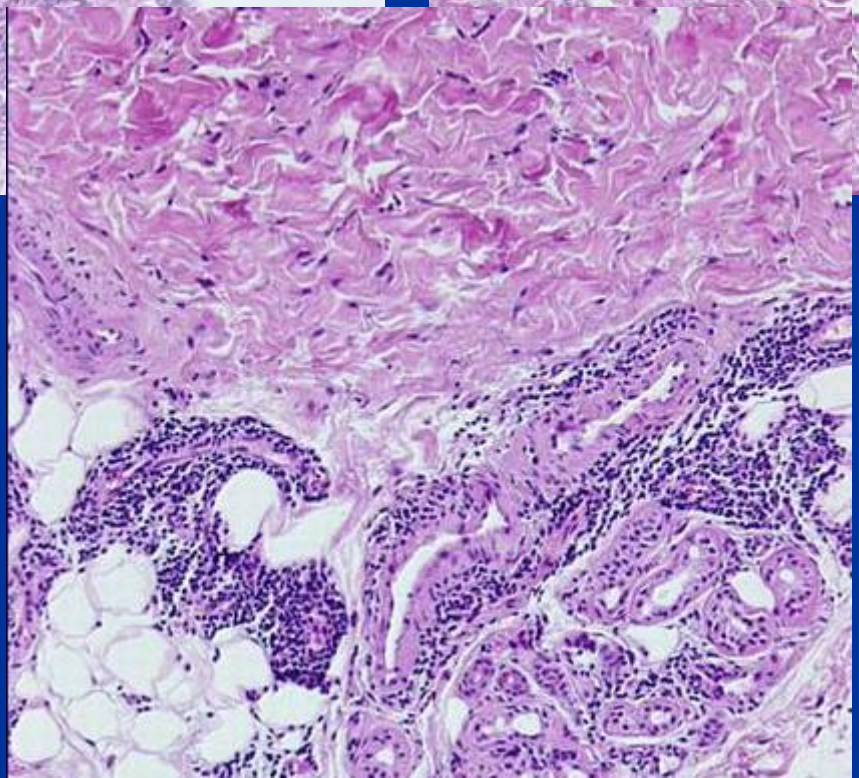
5.11B *Diffuse dermatitis*

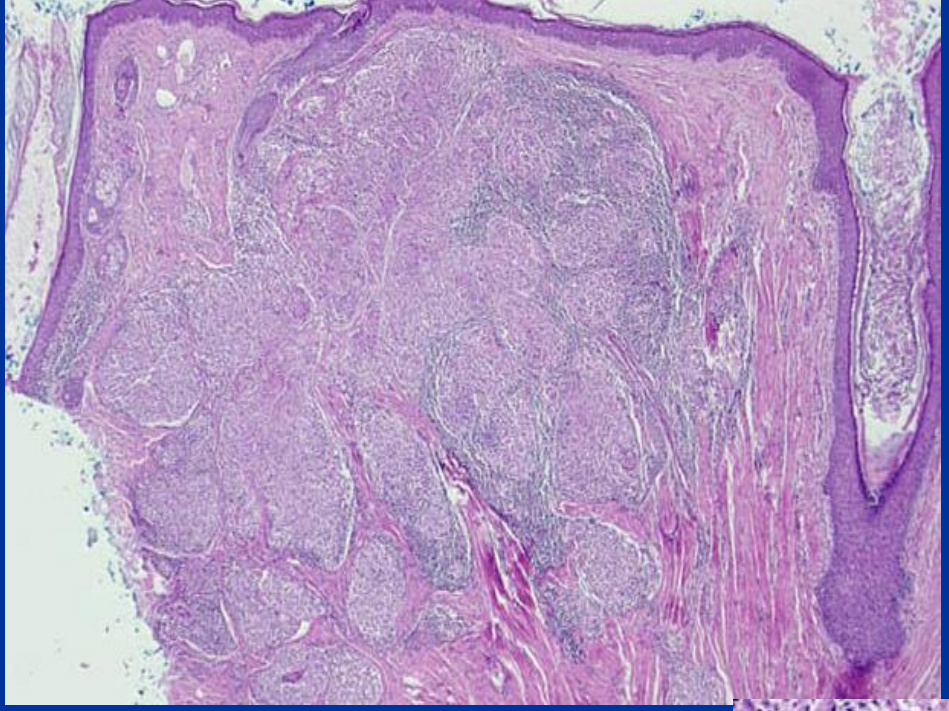


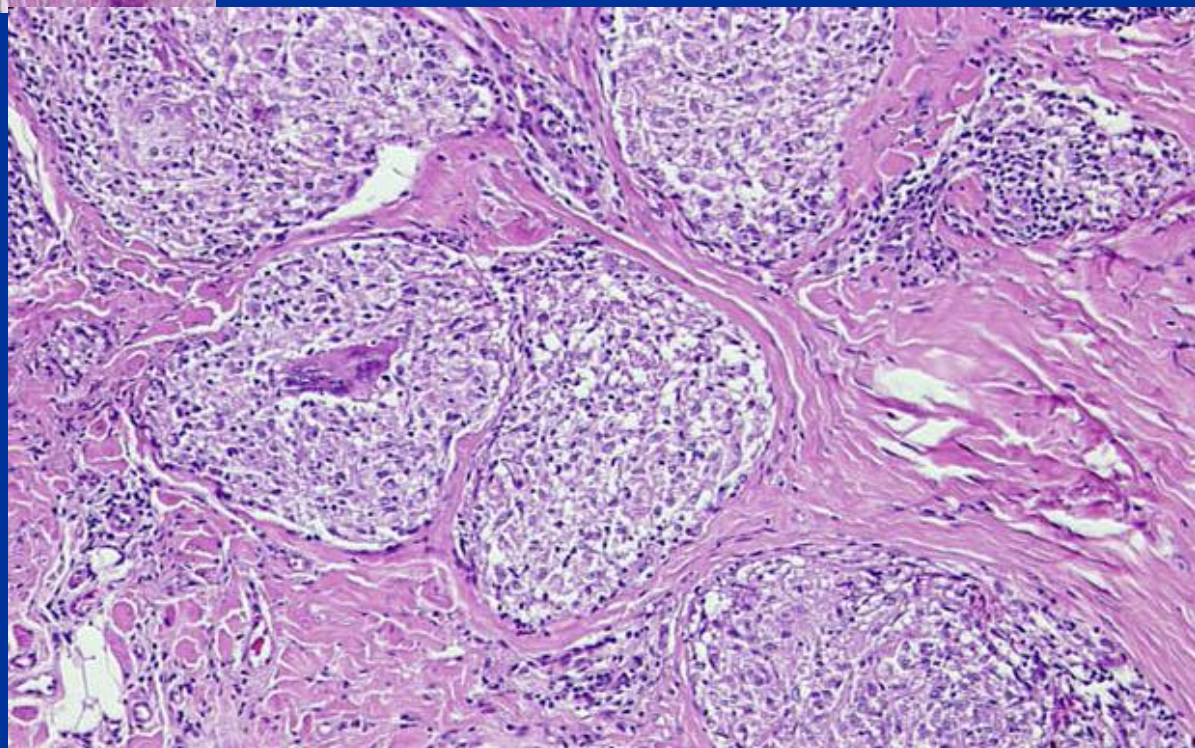


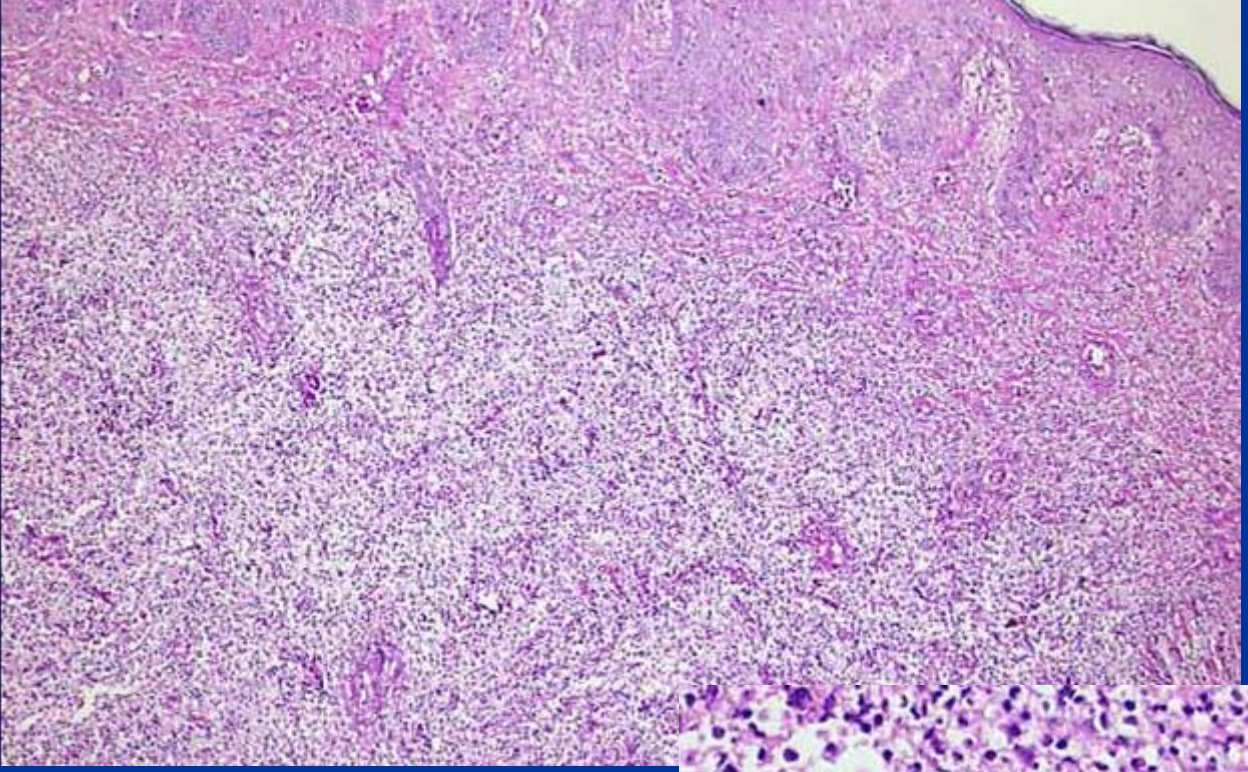


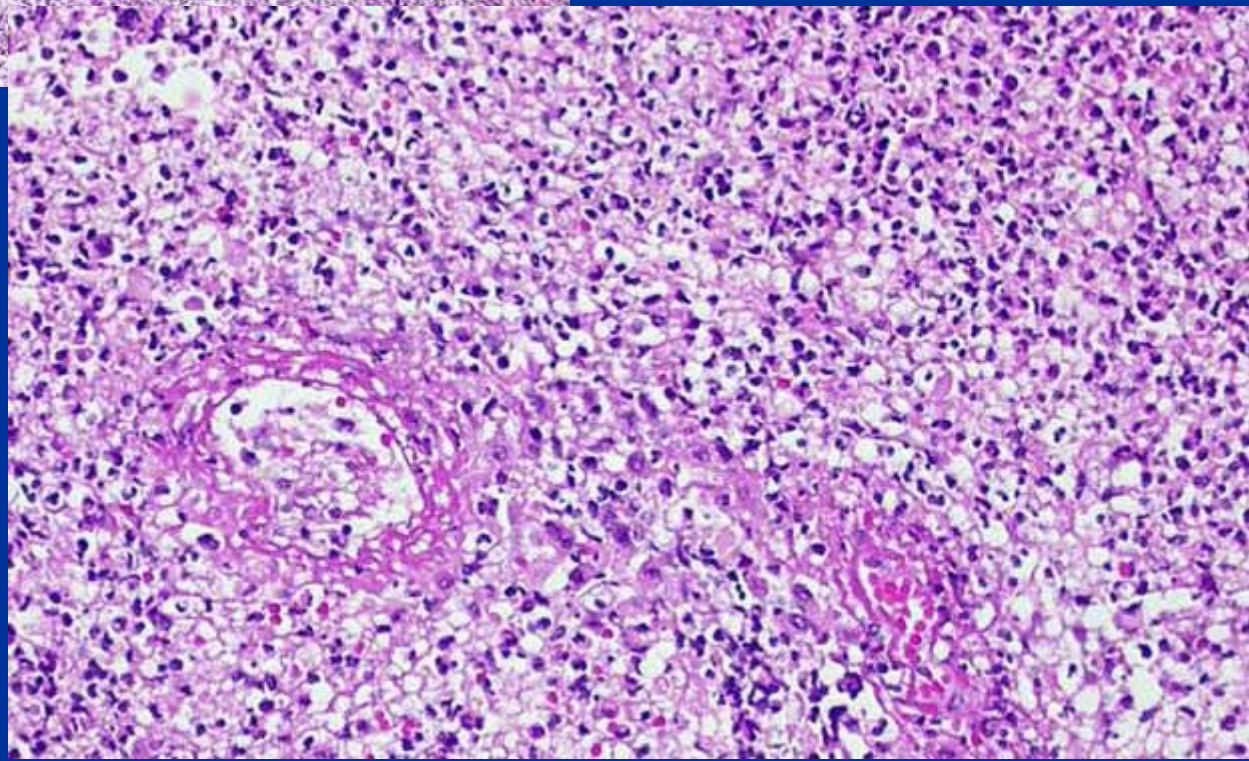


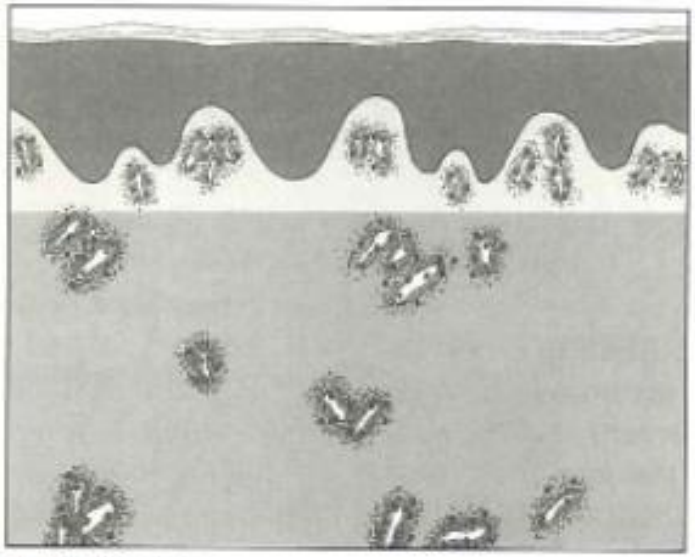




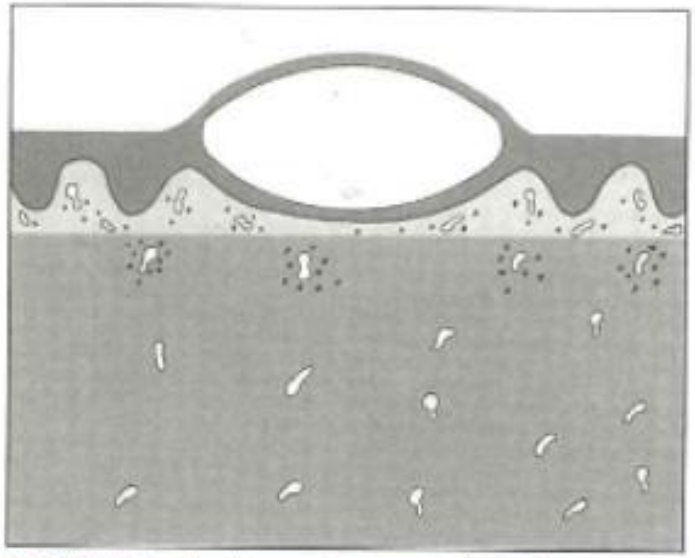




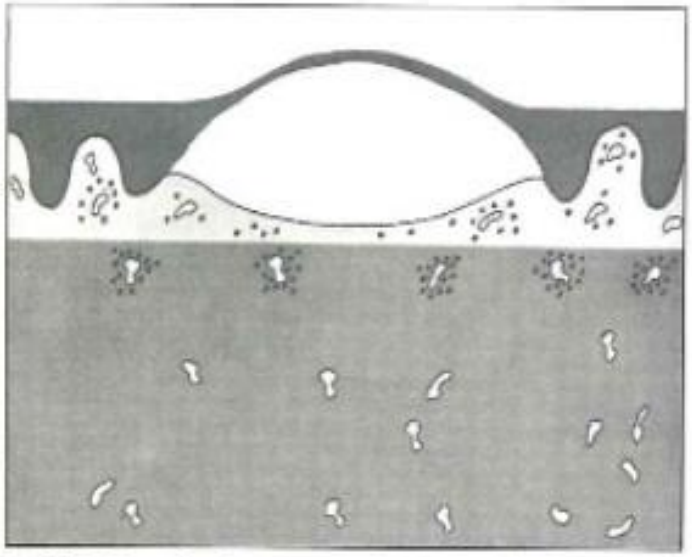




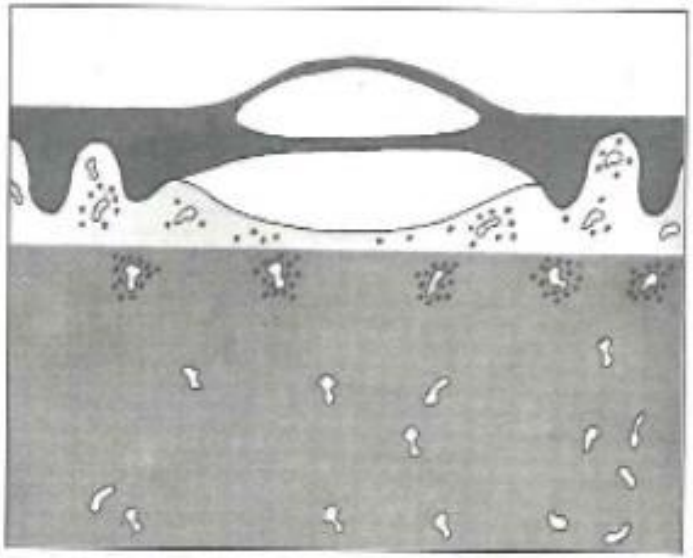
5.12 *Vasculitis*



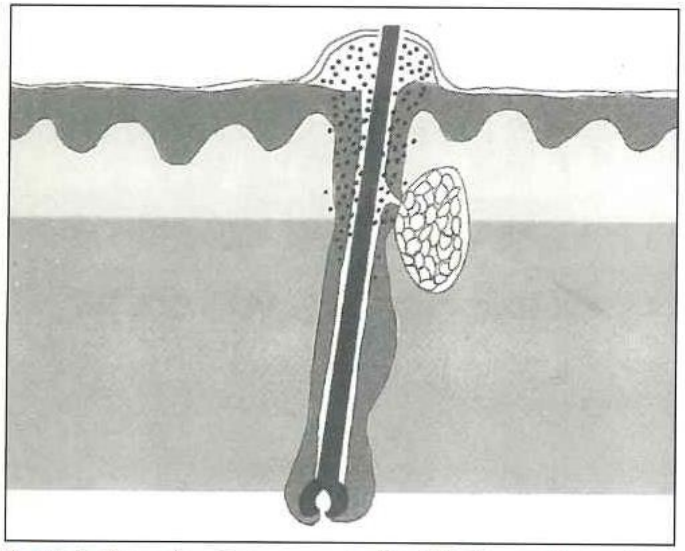
5.13A *Vesicular dermatitis, intraepidermal*



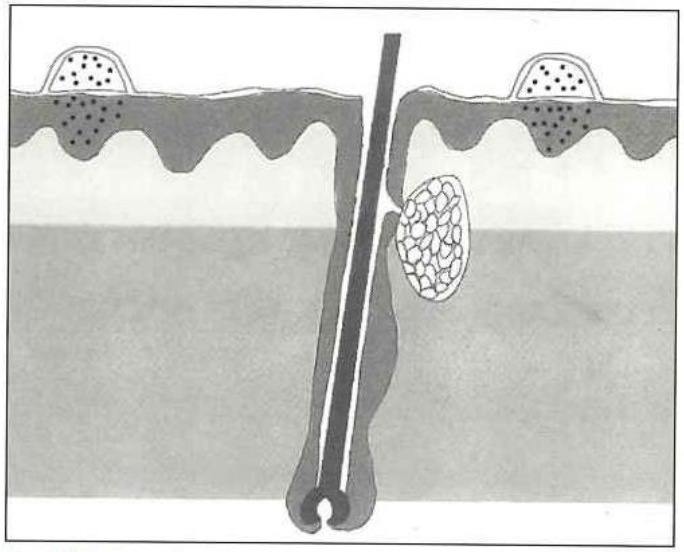
5.13B *Vesicular dermatitis, subepidermal*



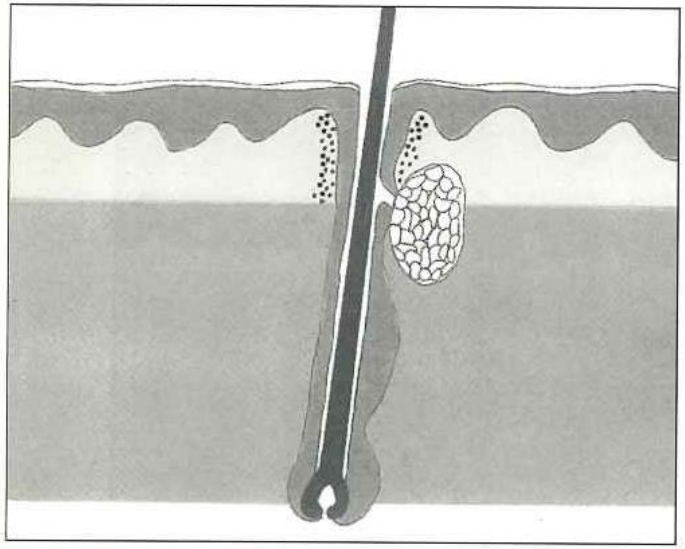
5.13C *Vesicular dermatitis, intra- and subepidermal*



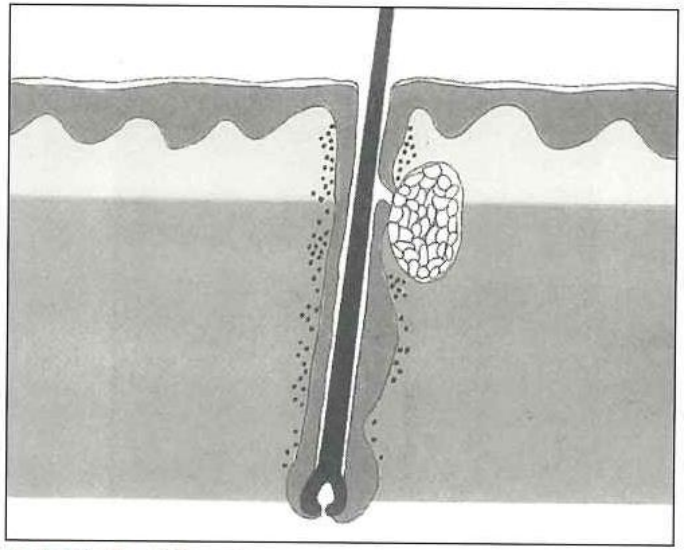
5.14A *Pustular dermatitis, infundibular epidermal*



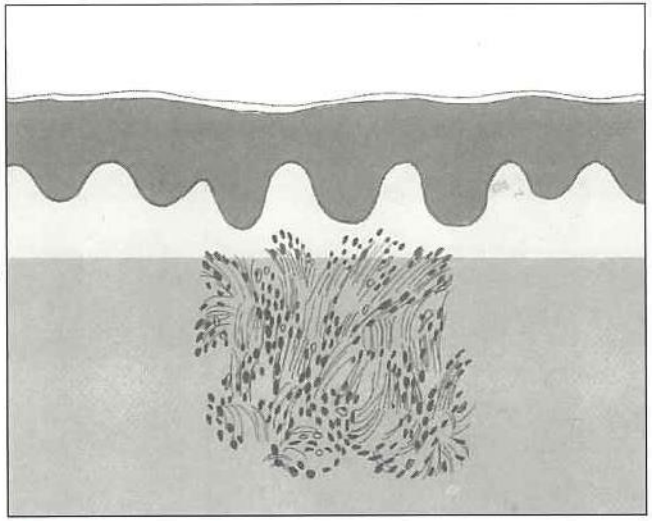
5.14B *Pustular dermatitis. surface epidermal*



5.15A *Peri-infundibulitis*

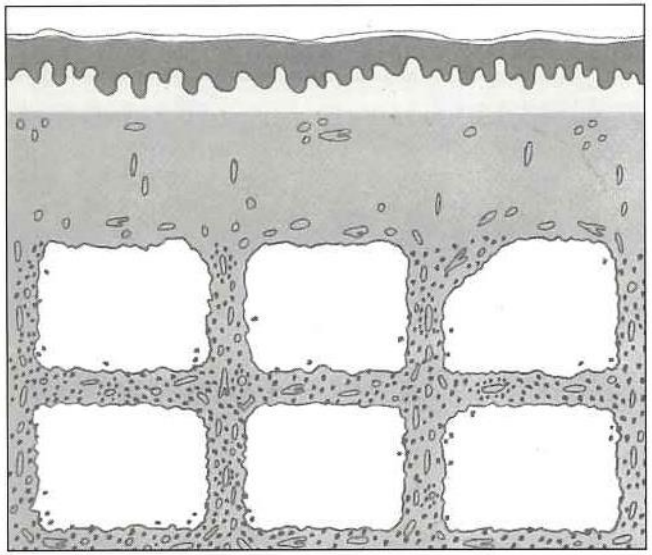


5.15B *Perifolliculitis*

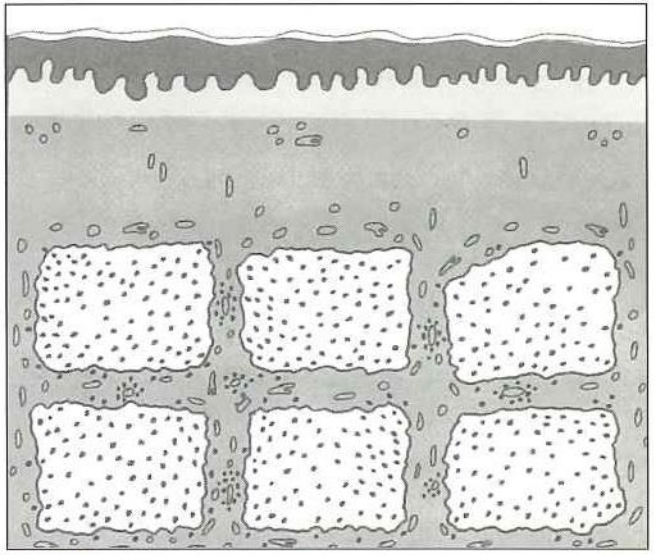


5.16 *Fibrosing dermatitis*

SUBCUTANEOUS FAT



5.17A *Panniculitis, mostly septal*



5.17B *Panniculitis, mostly lobular*

Perivascular superficial

- Perivascular only
- Interface lichenoid/Interface vacuolar
- Spongiotic
- Psoriasiform
- Spongiotic-psoriasiform
- Psoriasiform/spongiotic psoriasiform band-like
- Psoriasiform ballooning
- Ballooning band-like
- Ballooning
- Perivascular + interstitial

SPONGIOSIS

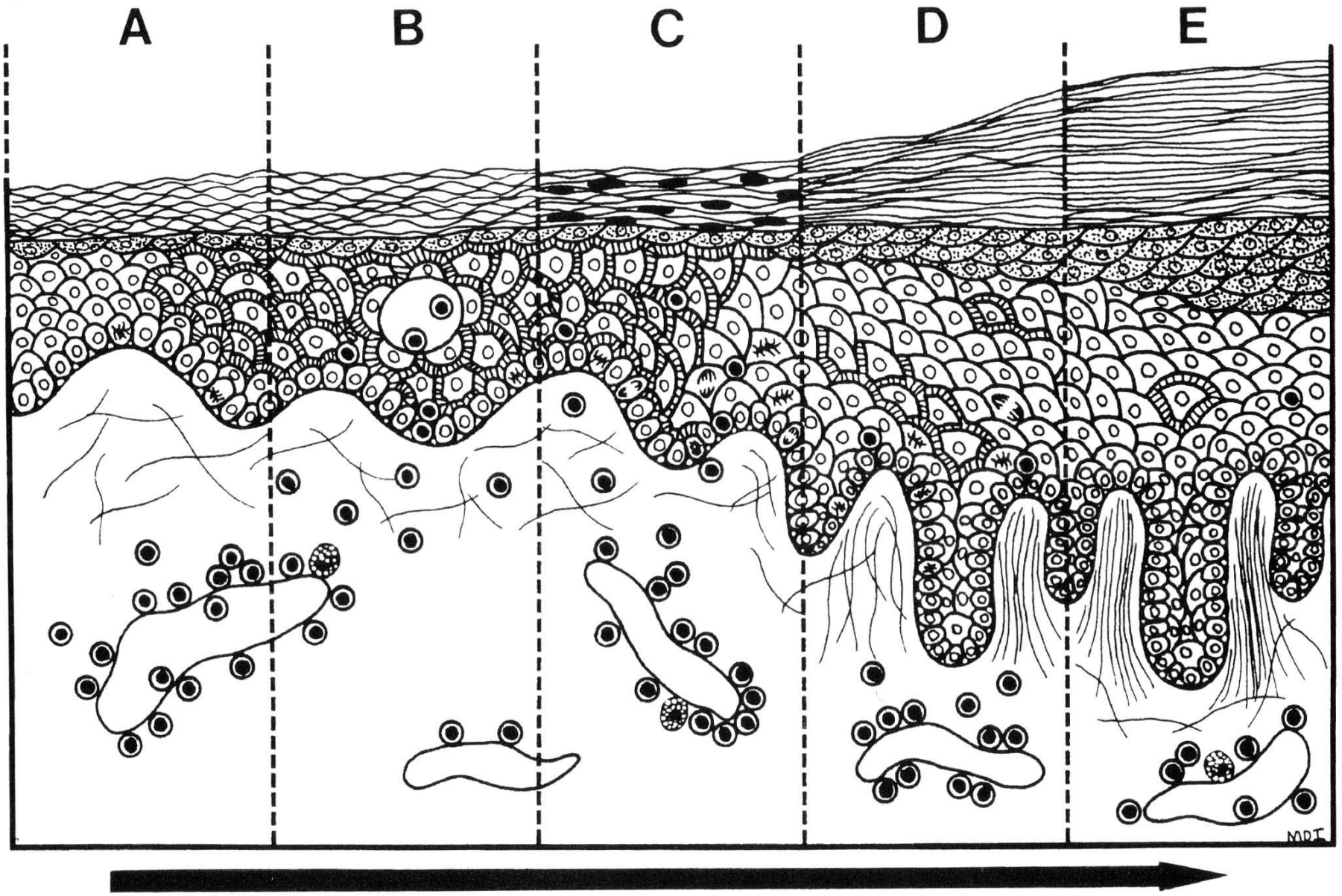
"Intercellular edema of the epidermis"

Absorbent Sponge:
Porous connective tissue skeleton of marine animals of the "Phylum Porifera"

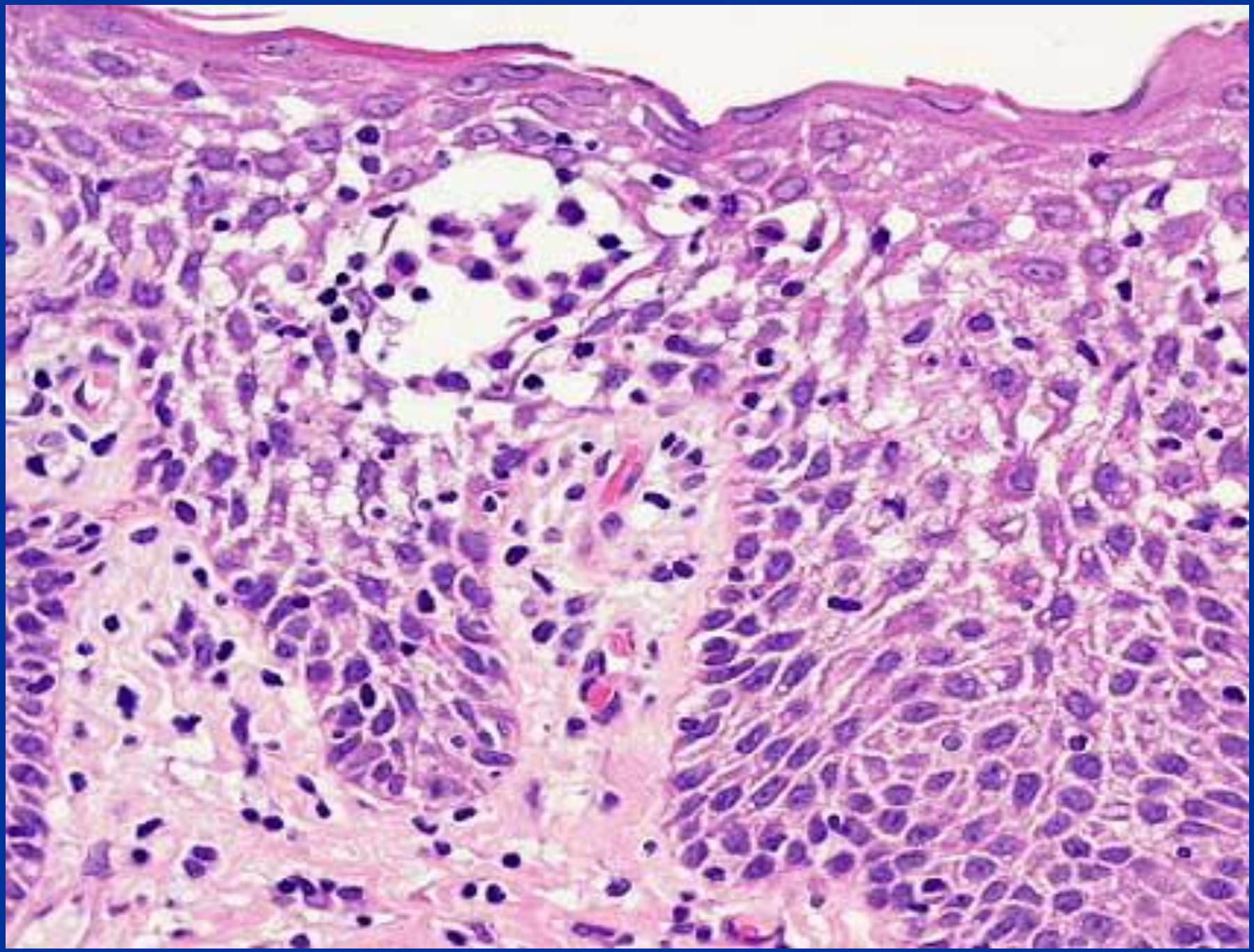
NATURAL EVOLUTION OF SPONGIOTIC DERMATITIS

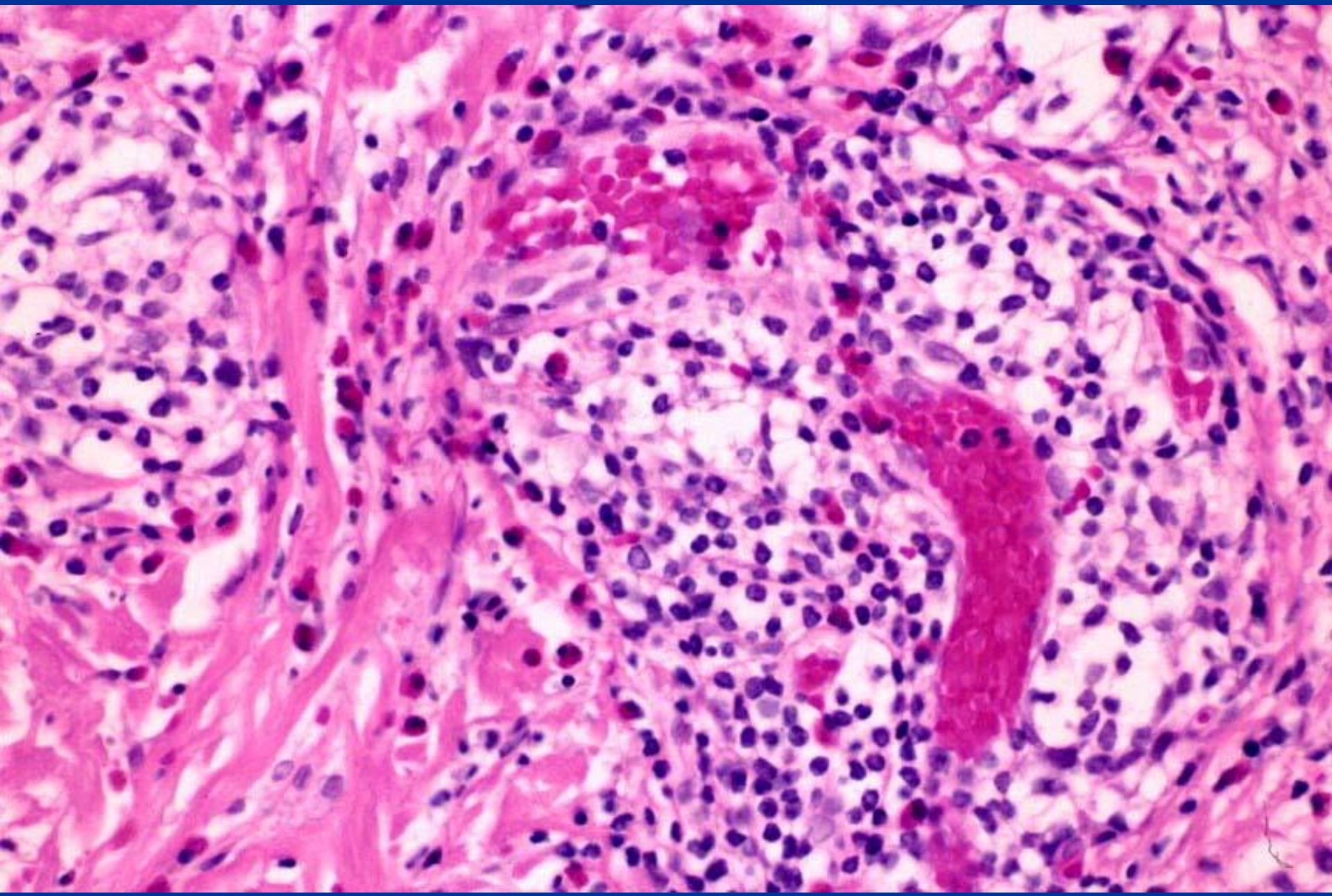
HISTOLOGIC ALTERATION	TEMPORAL PHASE		
	Acute	Subacute	Chronic
Spongiosis	+ + +	+ +	+
Vesiculation	+ to + + +	−	−
Epidermal hyperplasia	+	+ +	+ + +
Papillary dermal fibrosis	−	−	+ to + + +
Epidermotropism	+ to + + +	+	−
Endothelial activation*	+ + +	+ to + +	−
Hyperkeratosis and parakeratosis	−	− to +	+ + to + + +

*Defined as bulging of endothelial cells into venular lumina.
+ + +, marked; + +, moderate; +, slight; − absent.


A
B
C
D
E

Dermatite acuta spongiotica





StileAlpino
9 Maggio 2018 ·

SOS EDERA VELENOSA
Conosciuta anche come l'ortica del climber, cresce lungo rocce e falesie. Questa pianta è pericolosissima e provoca una dermatite allergica da contatto con bolle, prurito, arrossamenti.

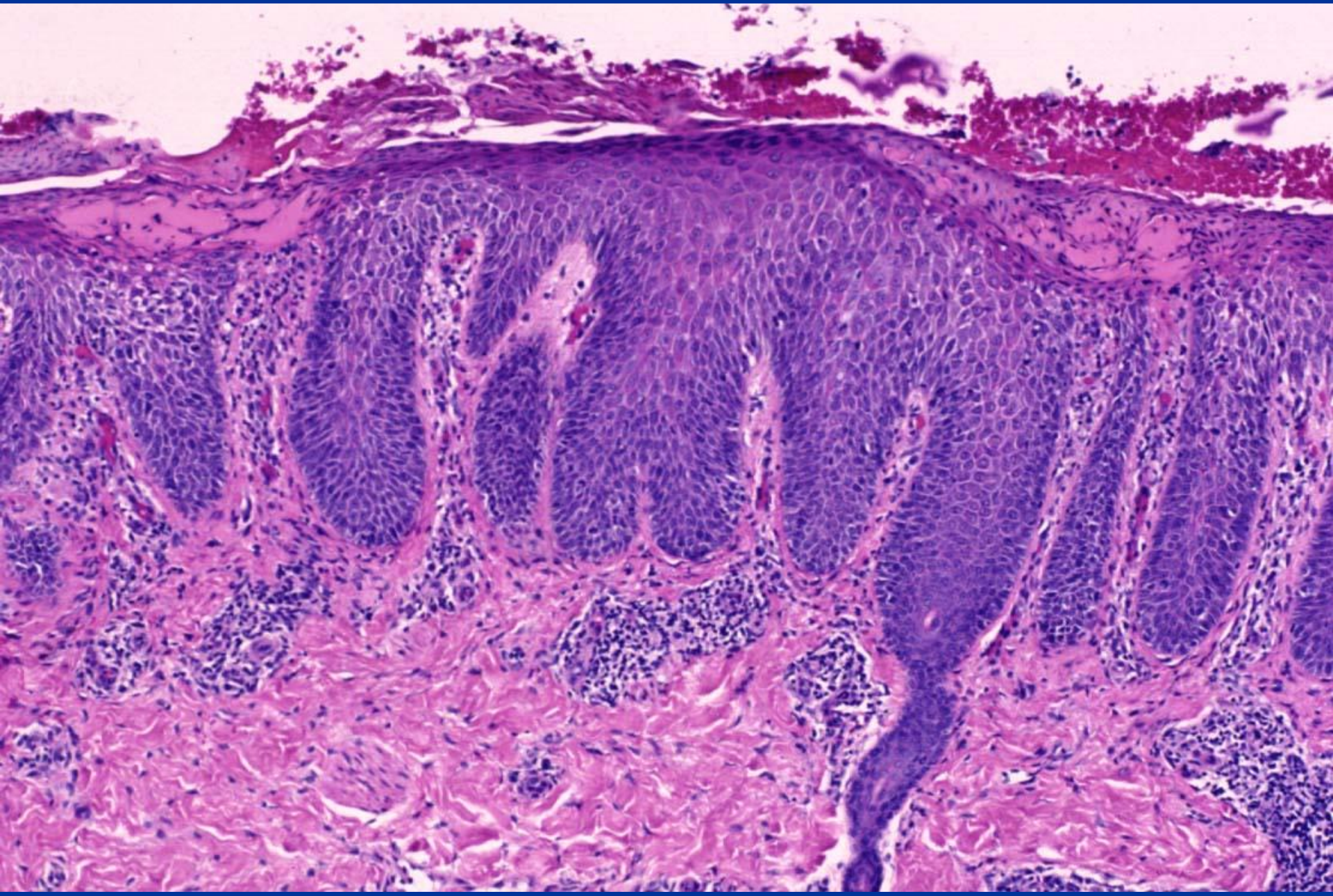
➡️Cosa devi fare subito dopo il contatto:
- la resina è idrosolubile: lava la parte con acqua e sapone;
- entro 30 minuti usa prodotti specifici che sciolgono la resina;
- non usare solventi alcolici, che aumentano la penetrazione della resina nella pelle;
- in caso di arro... **Altro...**

2 Condivisioni: 1

Mi piace Commenta Condividi

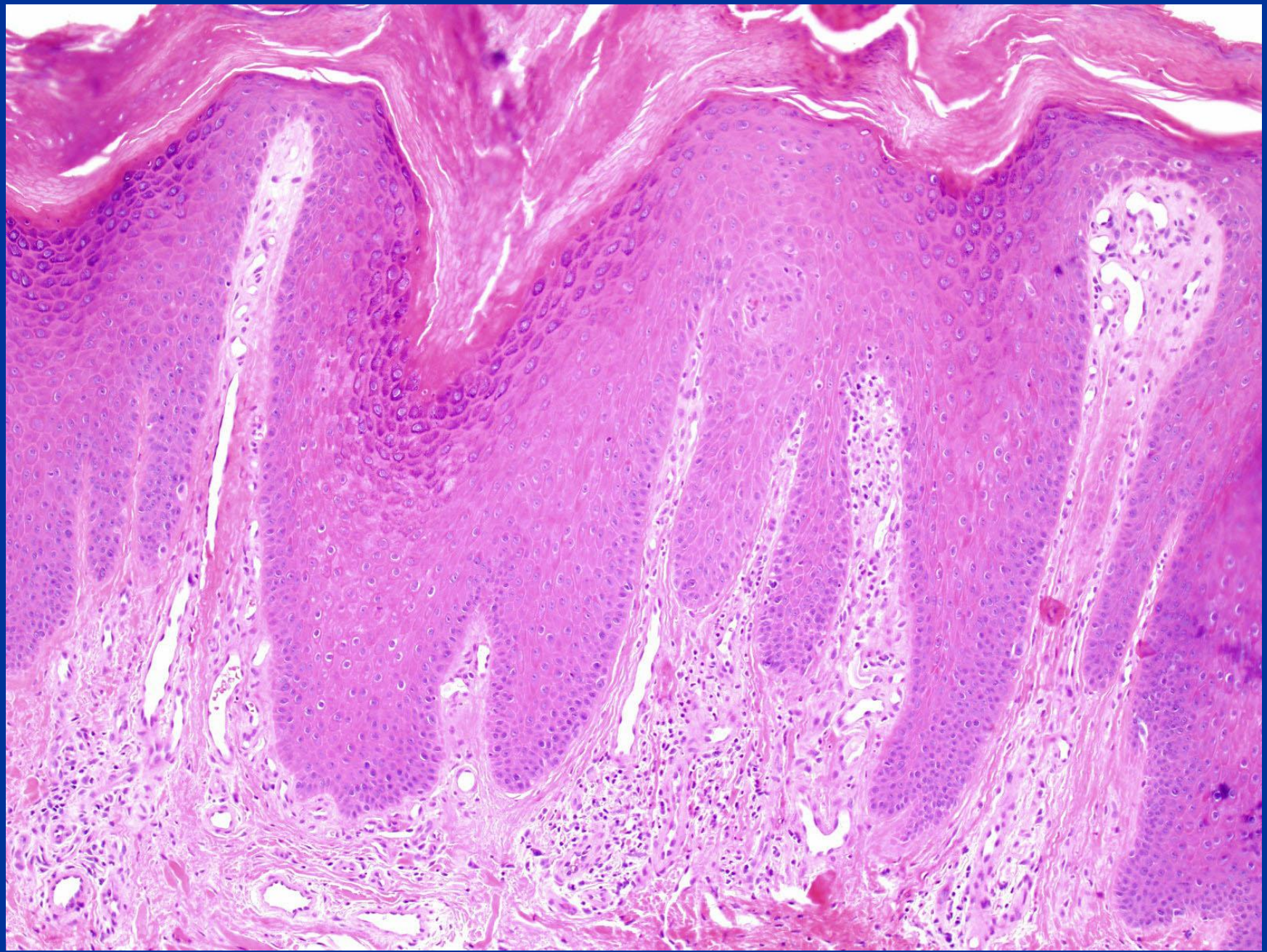
Vedi altri contenuti StileAlpino su Facebook

Dermatite subacuta spongiotica



Dermatite cronica spongiotica

Lichen simplex chronicus



Lichen simplex - Histopathology

- Compact orthokeratosis with focal parakeratosis
- Hypergranulosis
- Occasional pseudoepitheliomatous hyperplasia
- Prominent irregular acanthosis with curvilinear, blunt rete ridges
- Papillary dermal fibrosis, with vertically oriented collagen bundles
- Superficial chronic perivascular inflammatory infiltrate
- Rare mast cells and eosinophils
- As lichenification may be superimposed on other dermatoses, careful search for other disorders is advised

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Journal of
Cutaneous Pathology
ISSN 0303-6987

The periodic acid-Schiff stain in diagnosing tinea: should it be used routinely in inflammatory skin diseases?

Amina Al-Amiri*, Vandana Chatrath*, Jag Bhawan and Catherine M. Stefanato

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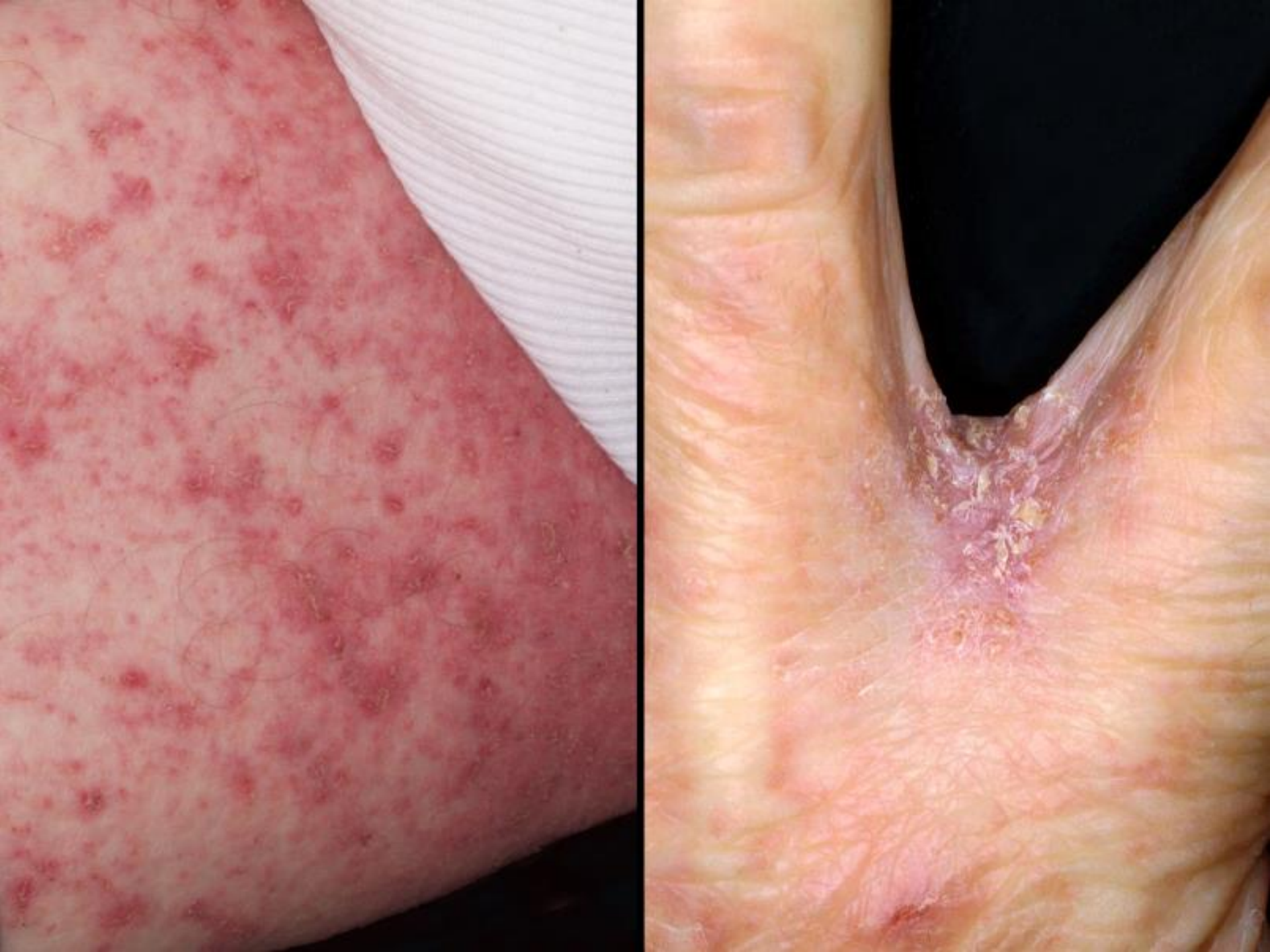
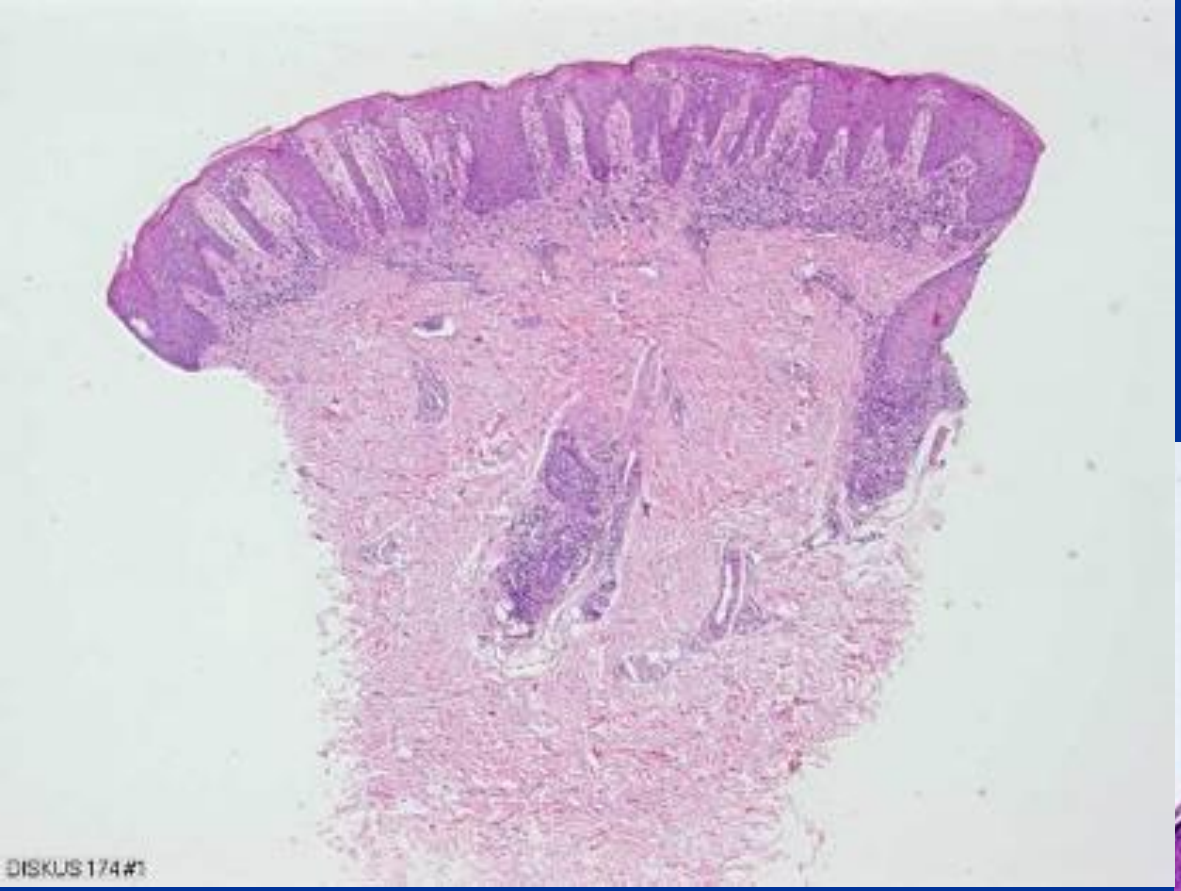
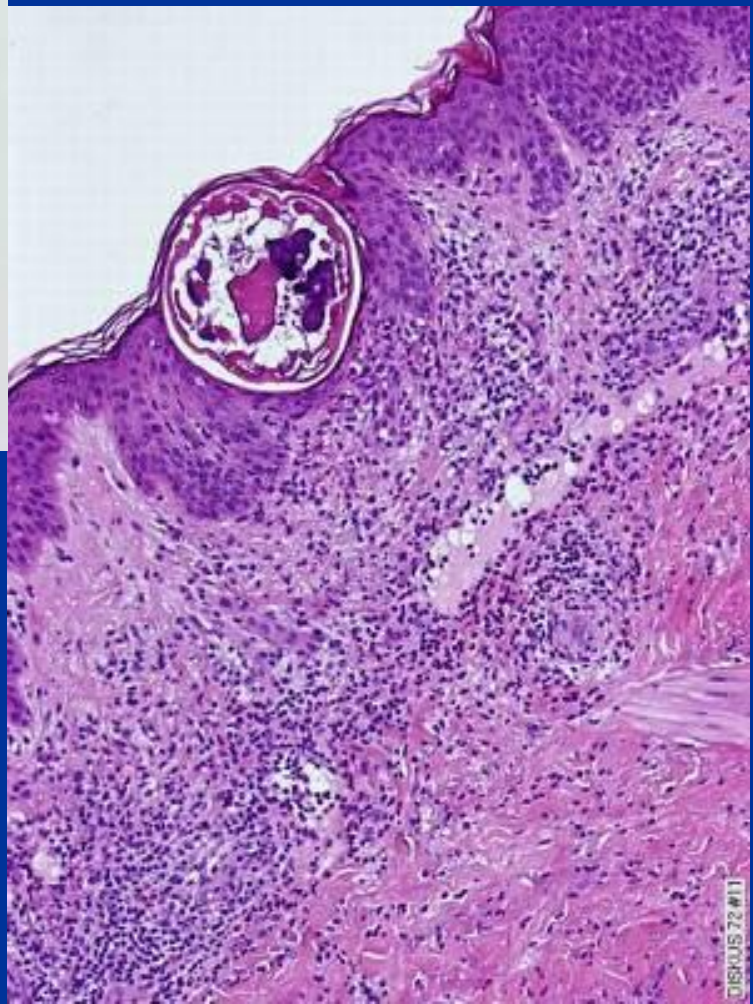
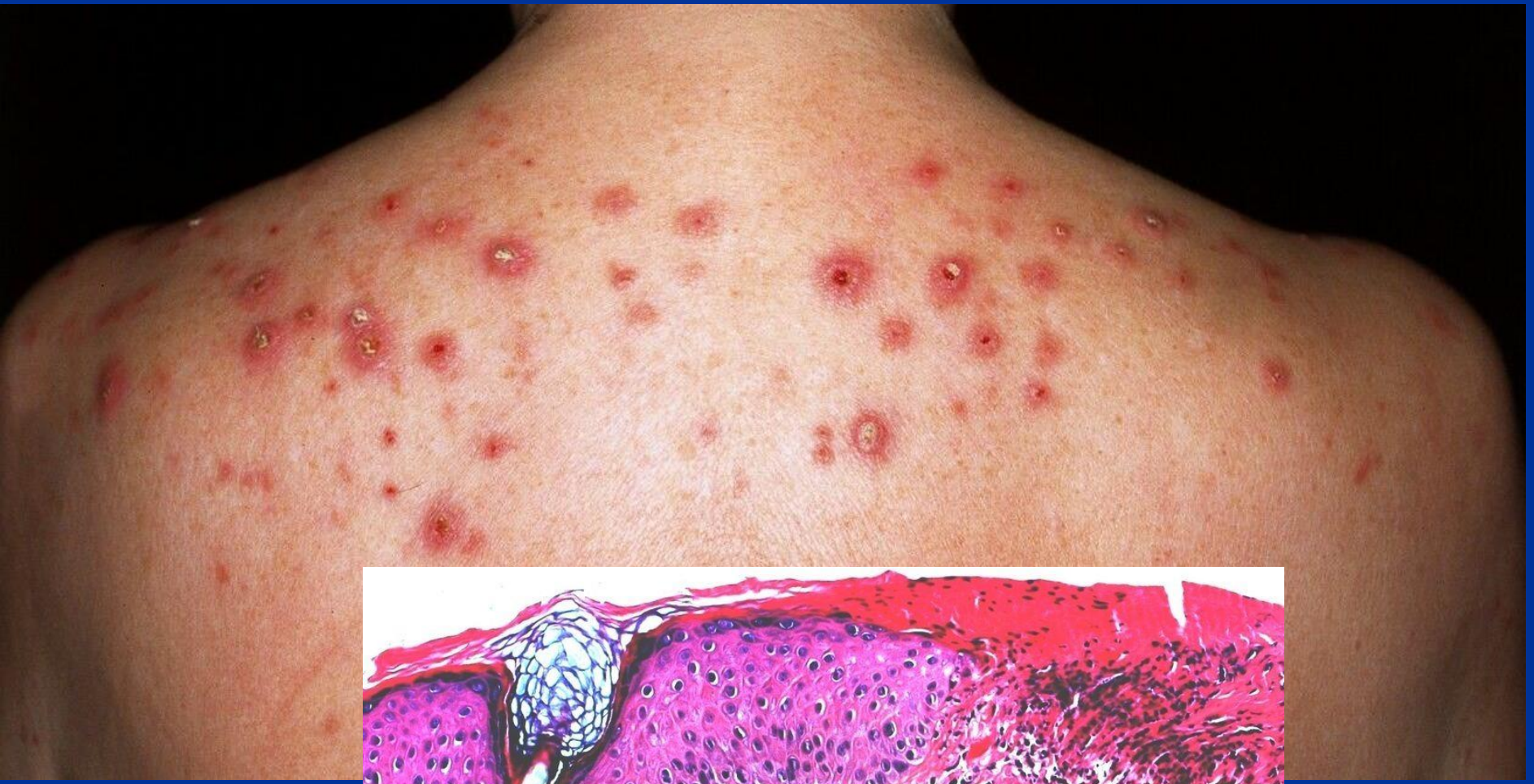


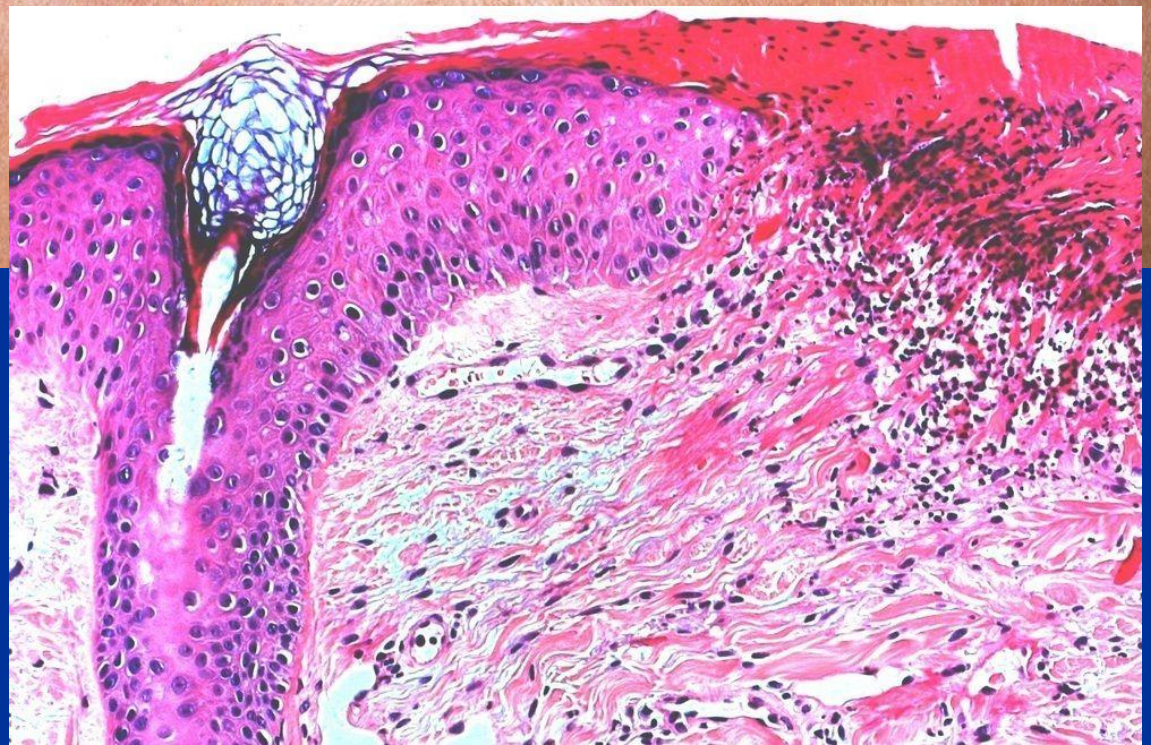

DISKUS 174#1

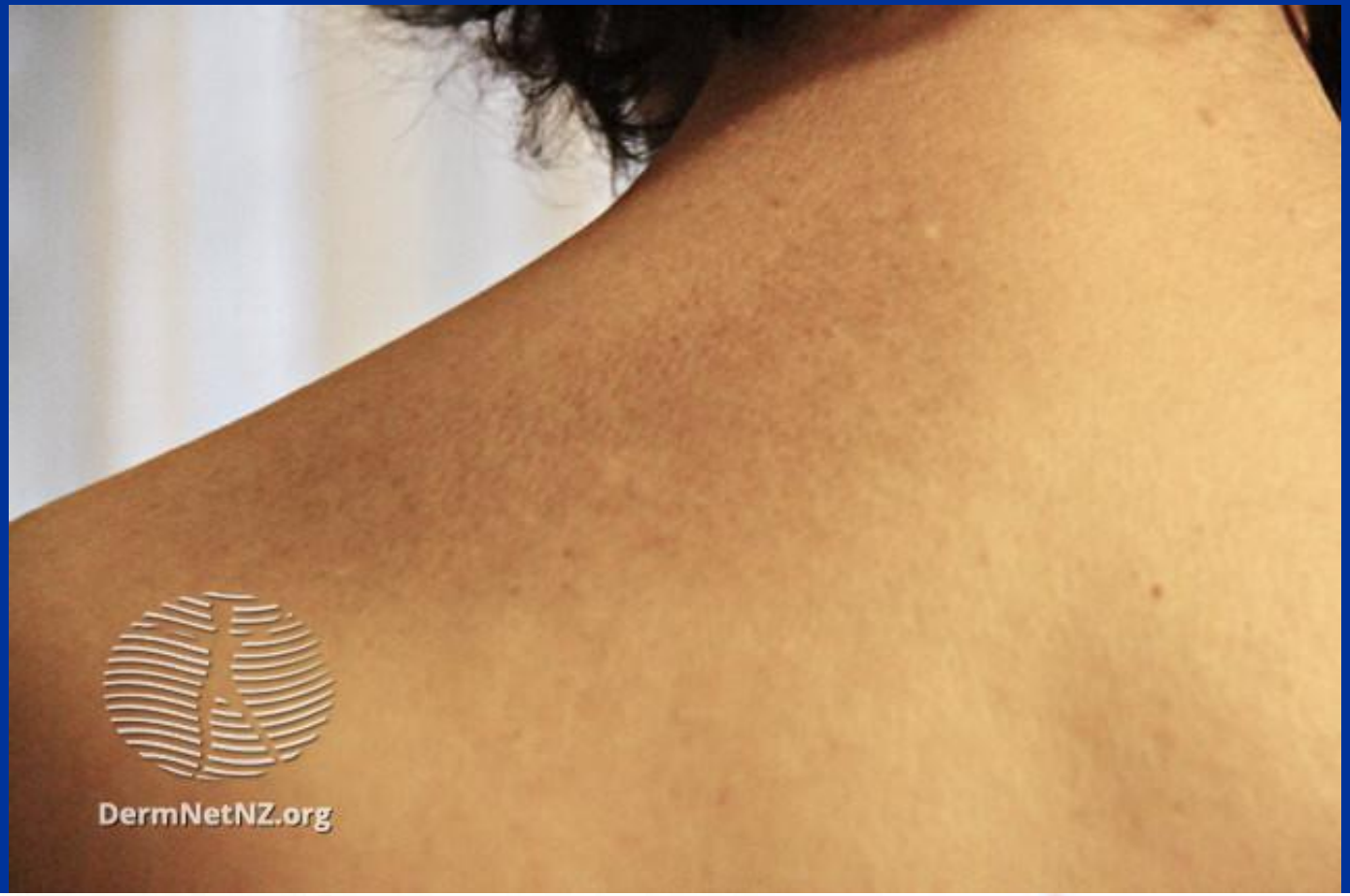

DISKUS 72#11

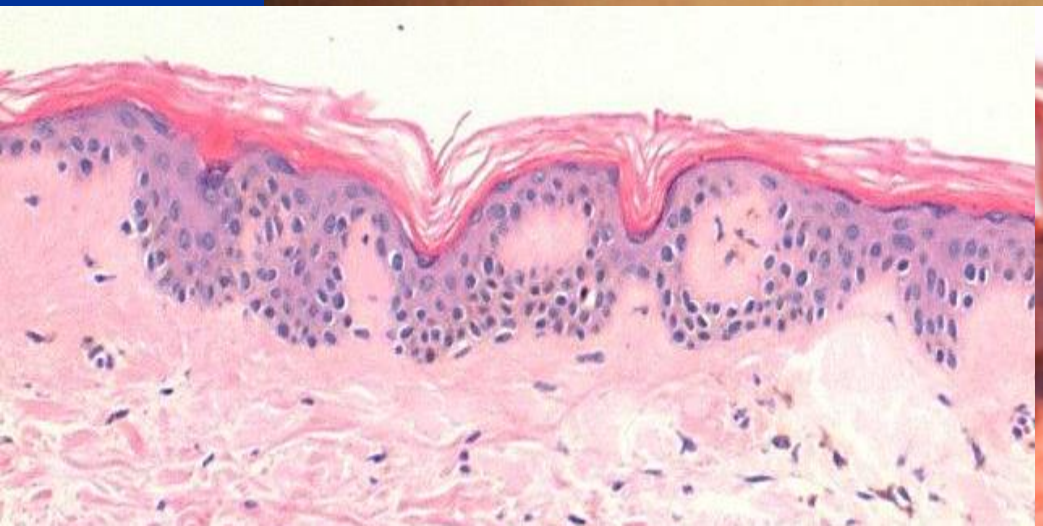
Entità anatomocliniche appartenenti allo spettro del lichen simplex

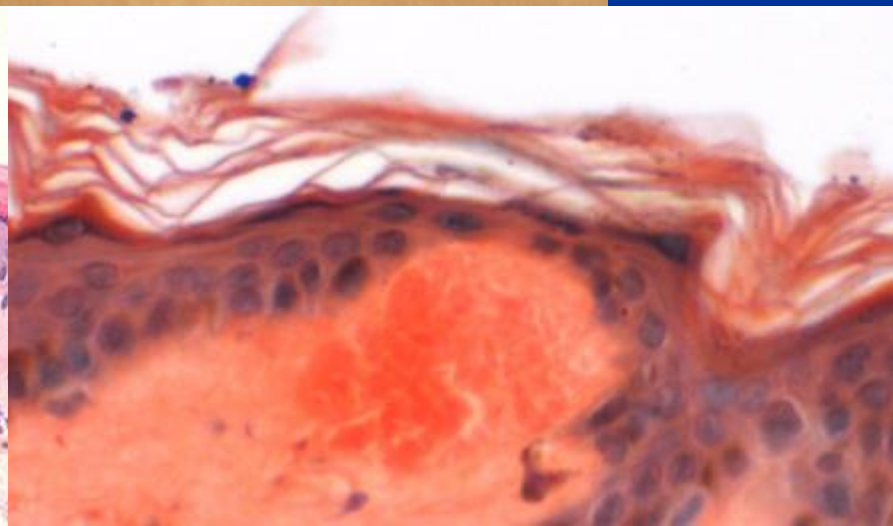
- Prurigine (prurigo simplex, prurigo subacuta)
- Amiloidosi maculare
- Lichen amiloidosico
- Prurigo nodulare

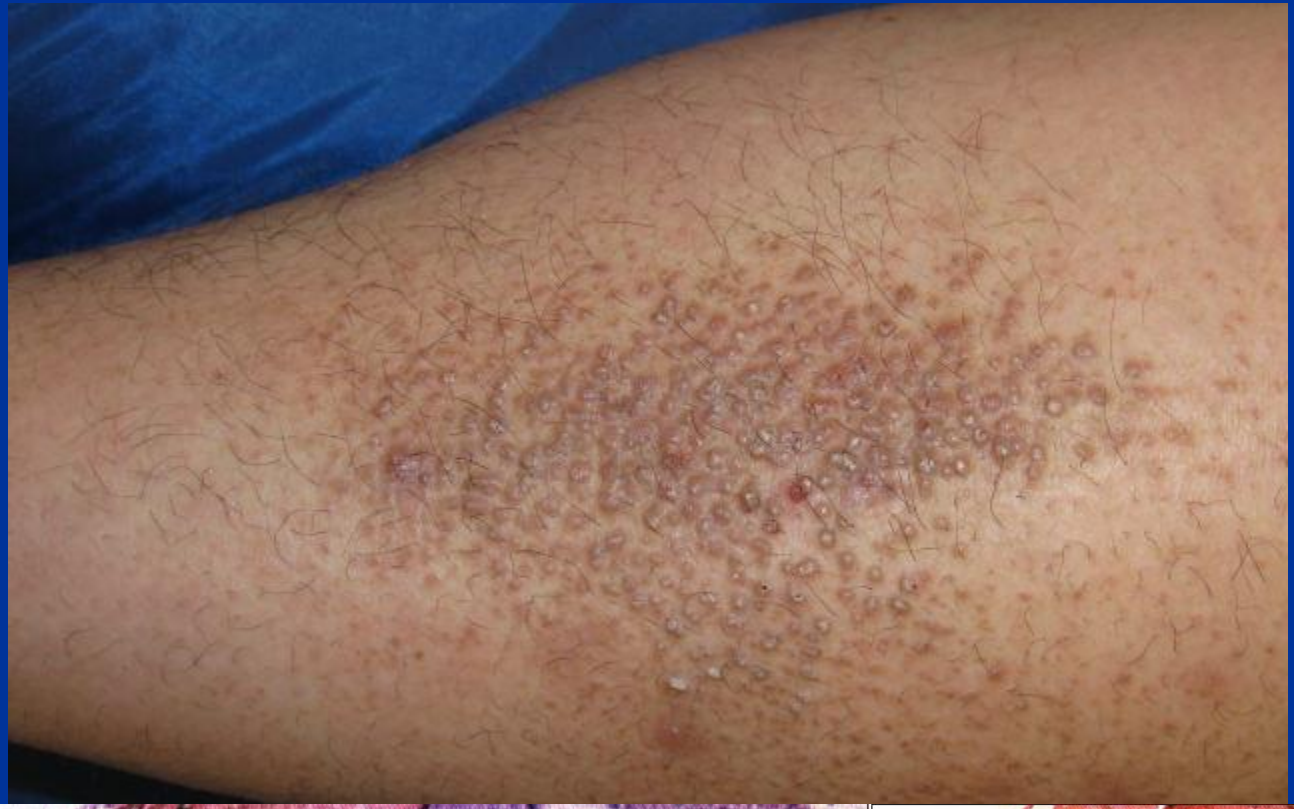


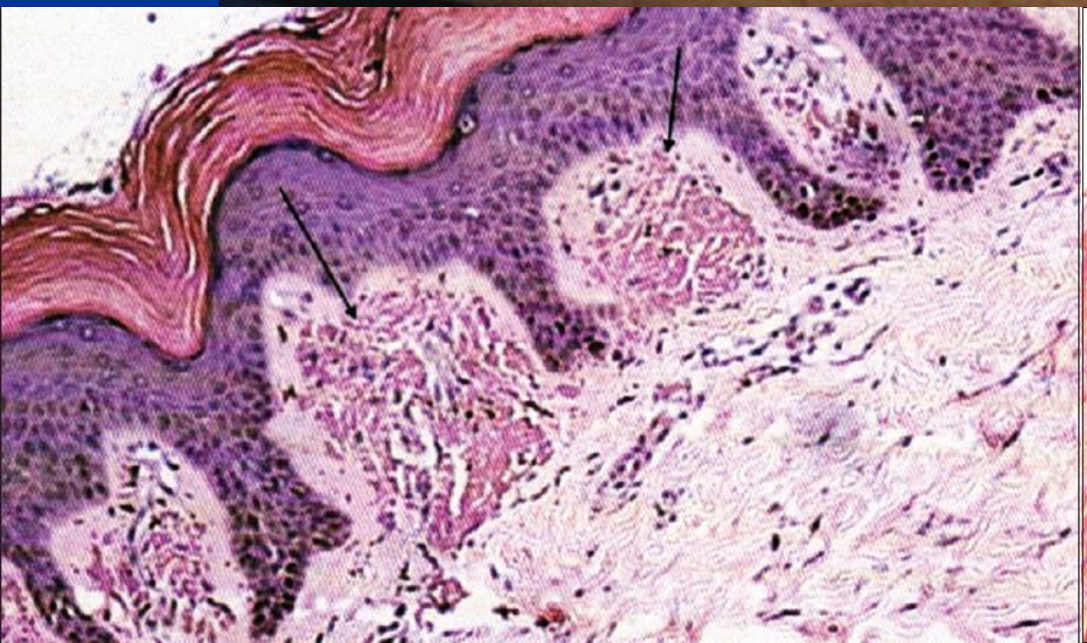


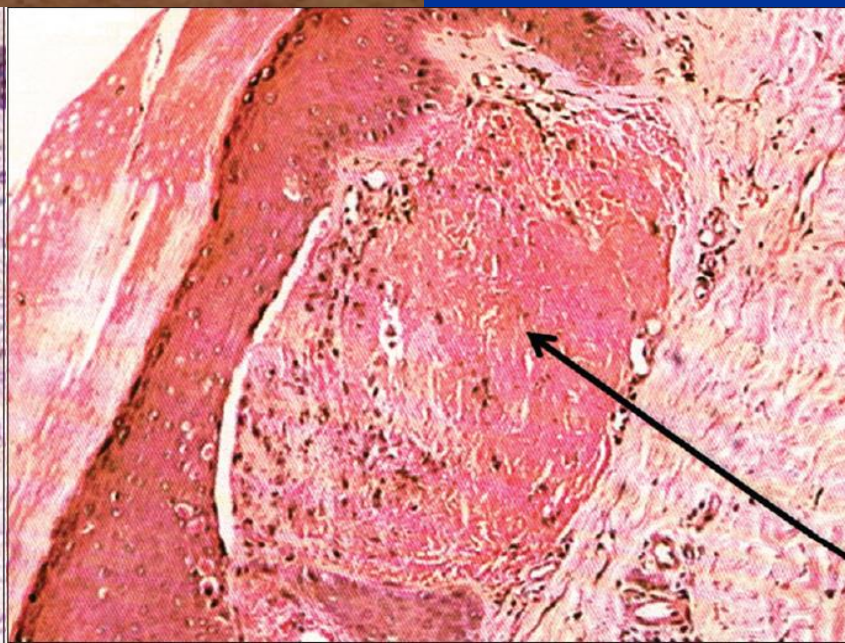

DermNetNZ.org

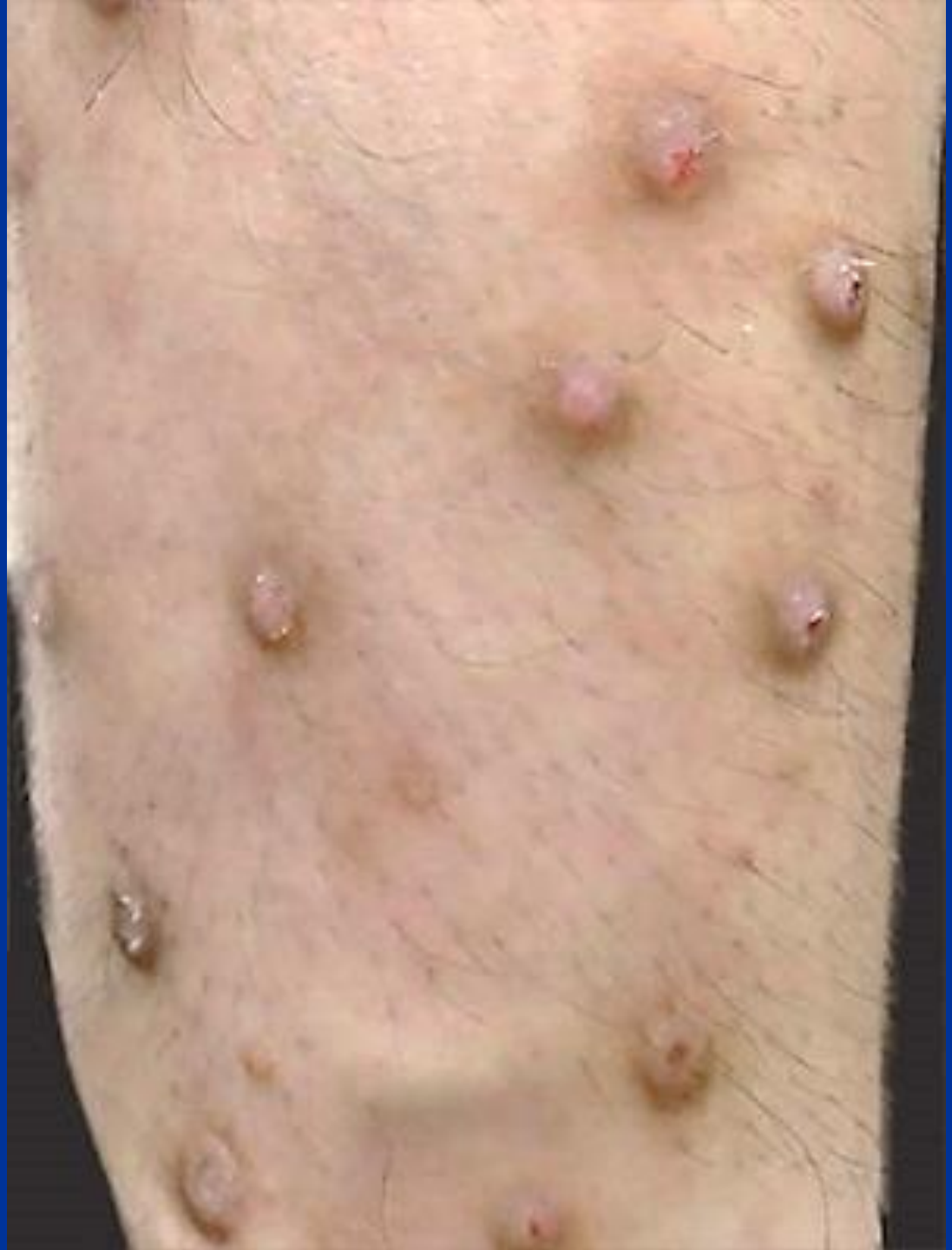


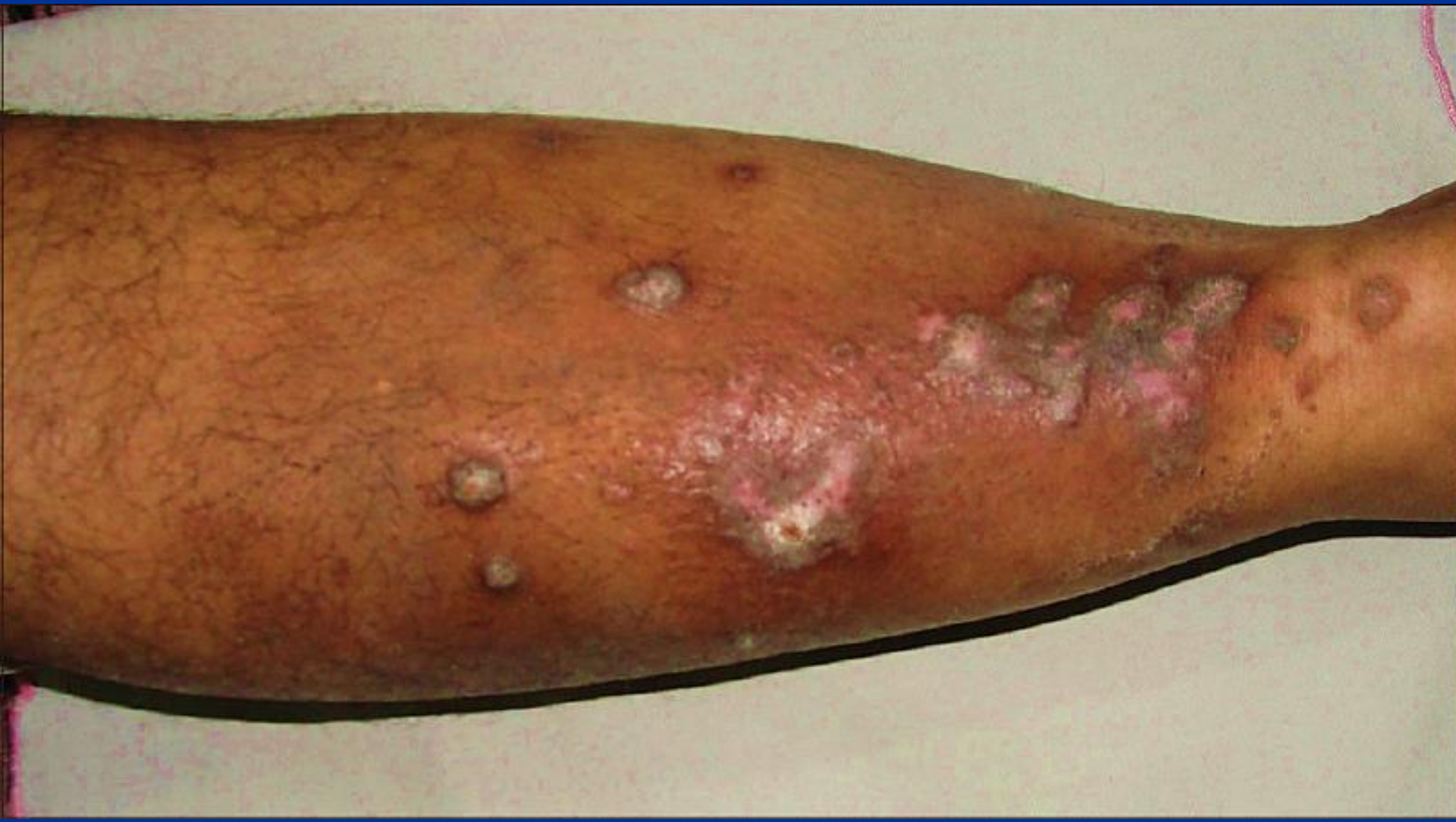


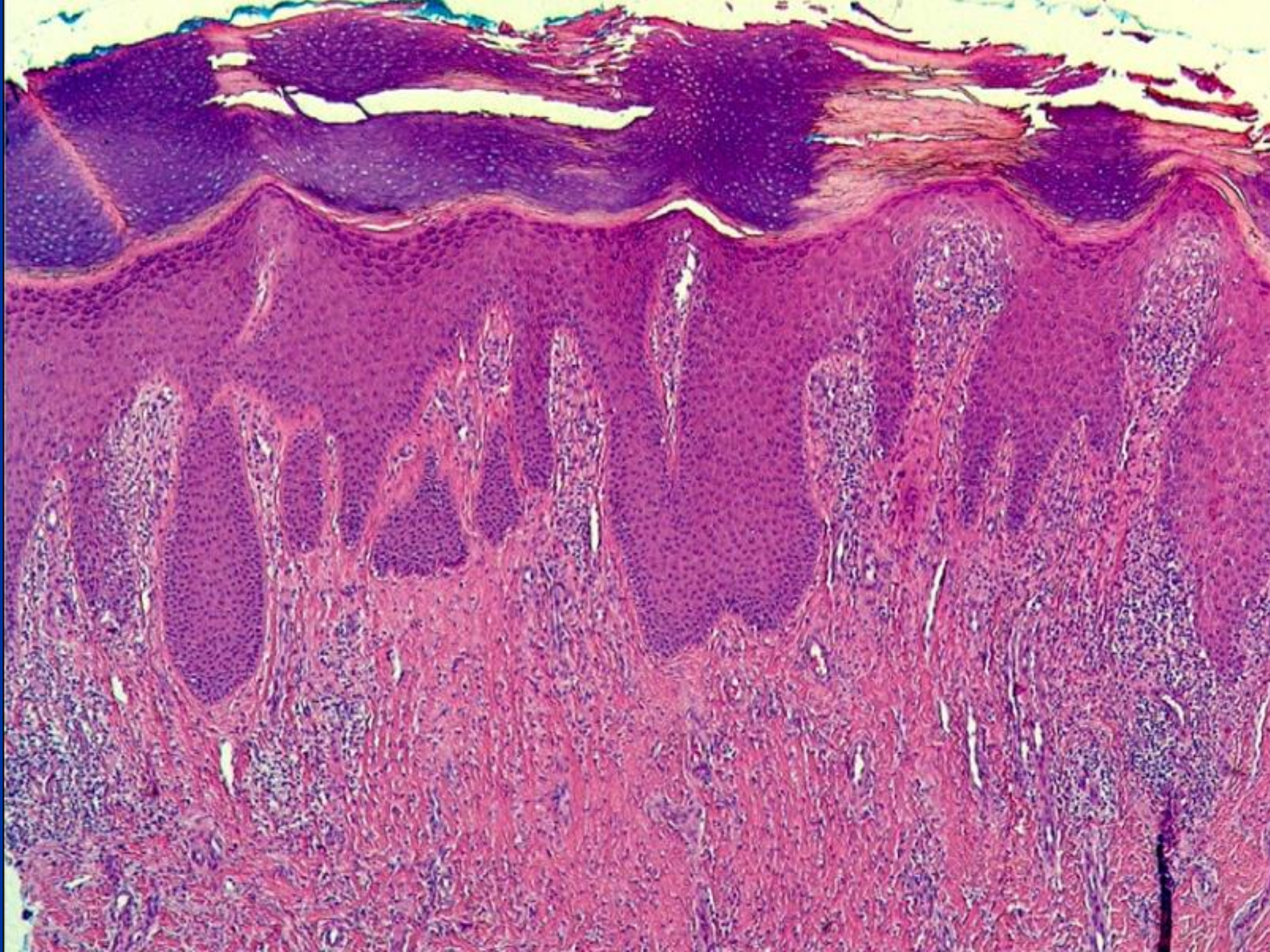


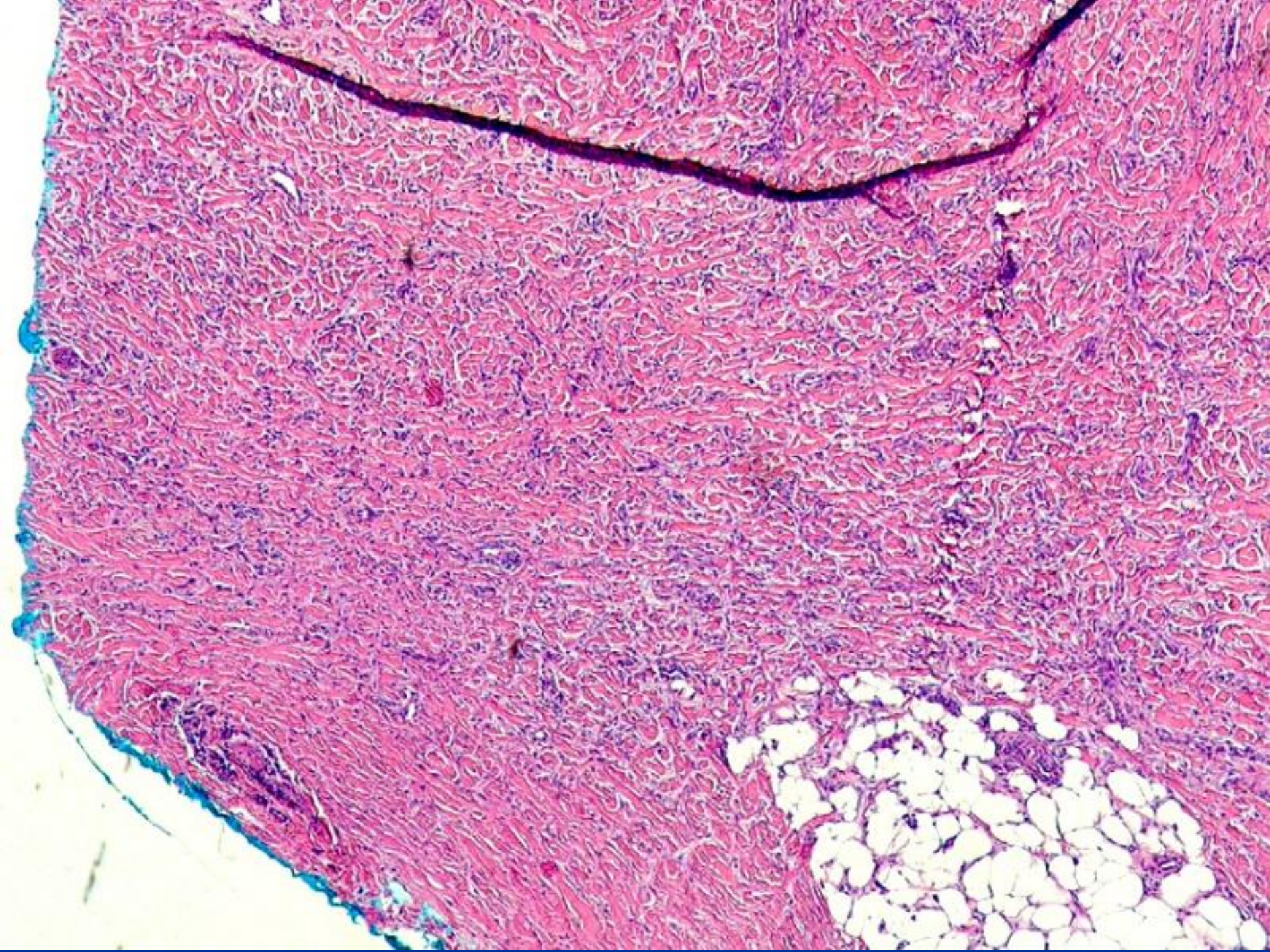


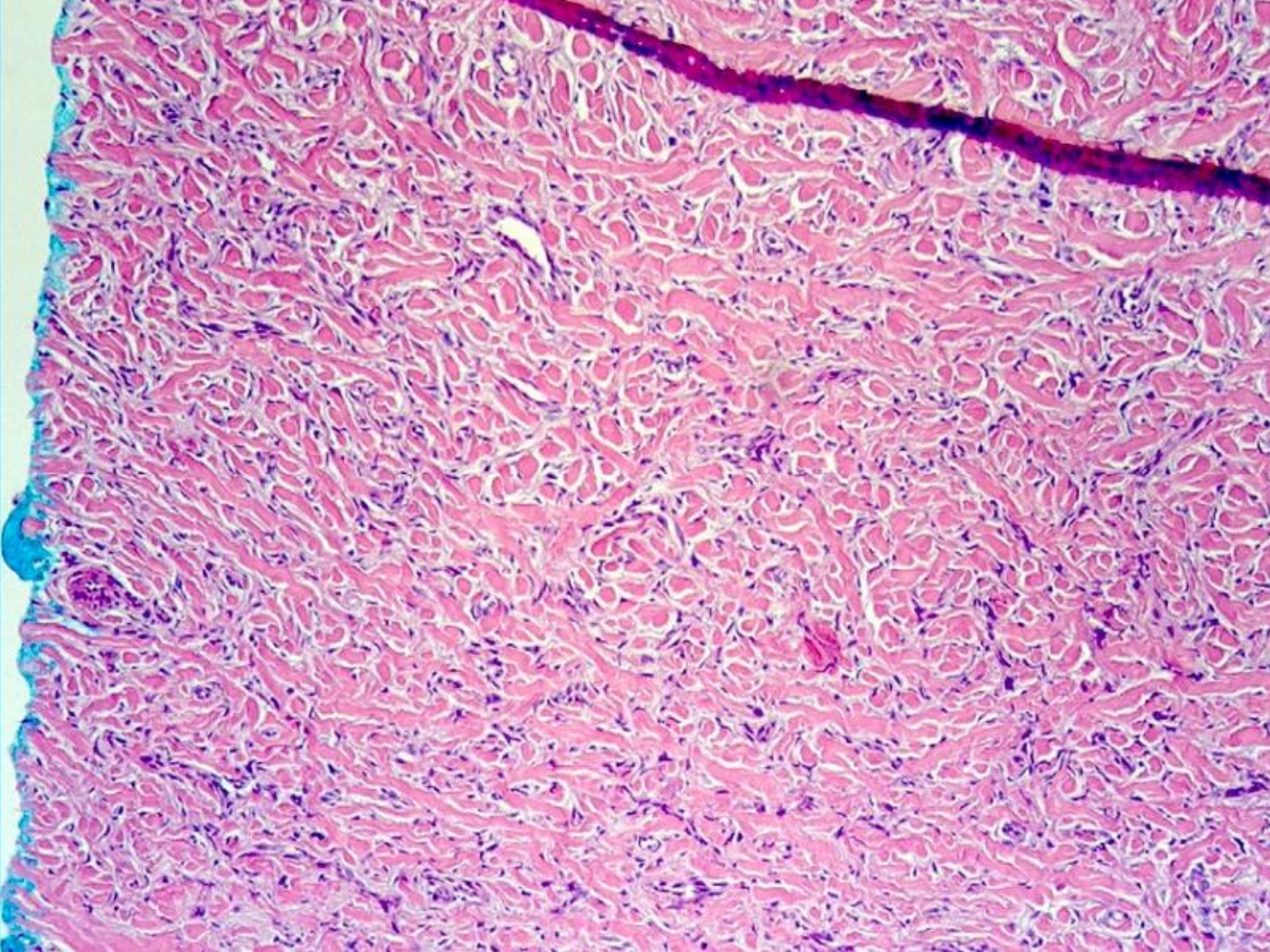












Ann Dermatol Vol. 22, No. 2, 2010 DOI: 10.5021/ad.2010.22.2.125

INVITED REVIEW ARTICLE

Atopic Dermatitis

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Histology

Histology of both forms of dermatitis is highly similar to that of allergic contact dermatitis and has no fundamental impact on the diagnosis of AD. Clinically "normal" appearing skin of AD patients contains a sparse perivascular T-cell infiltrate suggesting minimal inflammation[5]. "Acute" papular skin lesions are characterized by marked intercellular edema (spongiosis) of the epidermis. Langerhans cells (LC), in lesional and, to a lesser extent, in nonlesional skin of AD exhibit surface-bound IgE molecules in the IgE-associated form but not in the non-IgE-associated form. In the dermis of the acute lesion, there is a marked perivascular T-cell infiltrate with monocyte-macrophages. The lymphocytic infiltrate consists predominantly of activated memory T cells bearing CD3, CD4, HLA-DR, CD25 and CD45RO. Eosinophils are seen in the acute lesions but basophils and neutrophils are rarely present. Mast cells are present in various stages of degranulation.

"Chronic" lichenified lesions are characterized by a hyperplastic epidermis with elongation of the rete ridges, prominent hyperkeratosis, and minimal spongiosis. There is an increased number of IgE-bearing DC in the epidermis, and macrophages dominate the dermal mononuclear cell infiltrate. The number of mast cells is increased but the cells are generally fully granulated. Although they are hardly seen histologically, increased numbers of eosinophils are suspected in the dermis of chronic AD skin lesions since their products such as eosinophil major basic protein, eosinophil cationic protein and eosinophil-derived neurotoxin can be detected by immunostaining. Thus eosinophils may likely contribute to allergic skin inflammation by the secretion of cytokines and mediators that augment allergic inflammation and induce tissue injury in AD through the production of reactive oxygen intermediates and release of toxic granule proteins.

The American Journal of Dermatopathology 12(6): 544–551, 1990

The Cutaneous Pathology of Atopic Dermatitis

Robert M. Hurwitz, M.D., and Celestine DeTrana, M.D.

Sixteen skin biopsy specimens from early eruptive, evolving papules and well-developed plaques were obtained from eight patients with established atopic dermatitis. We found that the chronological and histopathological sequence begins with a perivascular interstitial spongiotic dermatitis, evolves into a psoriasiform microvesicular spongiotic dermatitis, which is sometimes focally lichenoid, and eventually concludes as a psoriasiform dermatitis. Thus, atopic dermatitis has characteristic and diagnostic histopathologic findings. These are portrayed and contrasted to what has been previously reported.

Key Words: Atopic dermatitis—Histopathology.

Atopic dermatitis is considered an eczematous disease of characteristic distribution and usually occurs in persons with a personal or family history of allergies (1). The cutaneous pathology of atopic dermatitis has been studied by many well-known dermatologists for over 100 years, yet there is no general agreement. Some authorities consider the changes nonspecific, and there is disagreement regarding the site of the primary process (i.e., dermal or epidermal). Therefore, biopsy specimens were obtained from what appeared to be early eruptive, evolving, and well-developed lesions in patients with established atopic dermatitis to help clarify this issue.

TABLE 2. *Histopathologic findings in atopic dermatitis*

Early eruptive lesion (Fig. 4)
Epidermal:
Basketweave orthokeratosis, slight spongiosis
Dermal:
Papillary edema, perivascular and interstitial mixed cell infiltrate with lymphocytes, histiocytes, neutrophils, eosinophils, a few mast cells, and dilated blood vessels
Evolving lesion (*Fig. 5a–c*)
Epidermal:
Compact orthokeratosis, focal parakeratosis, epidermal hyperplasia, focal hypergranulosis, spongiosis and microvesiculation, exocytosis of lymphocytes, neutrophils, and eosinophils, focal collection of neutrophils within the stratum corneum, focal vacuolar alteration at the dermoepidermal junction, especially at tips of dermal papillae, and sometimes across a broad front
Dermal:
Papillary dermis thickened with coarse collagen, edema, and a mixed-cell or lymphohistiocytic infiltrate which is sometimes focally lichenoid, perivascular and interstitial mixed-cell infiltrate with lymphocytes, neutrophils, and eosinophils, extravasated eosinophilic granules and nuclear fragments on occasion, and dilated blood vessels
Well-established lesion (*Fig. 6*)
Epidermal:
Compact orthokeratosis, focal parakeratosis, psoriasiform epidermal and follicular hyperplasia
Dermal:
Papillary dermis thickened with vertically oriented coarse collagen bundles, perivascular lymphohistiocytic infiltrate, and dilated blood vessels

Spongiotic dermatitis vs MF

Spongiotic dermatitis

- Spongiosis prevailing
- Flask-shaped, Langerhans' cell-rich intraepidermal collections
- Psoriasiform hyperplasia

Mycosis fungoides

- Intraepidermal lymphocytosis prevailing
- True Pautrier's without Langerhans' cells
- Psoriasiform+lichenoid (patchy)
- Psoriasiform+atrophic

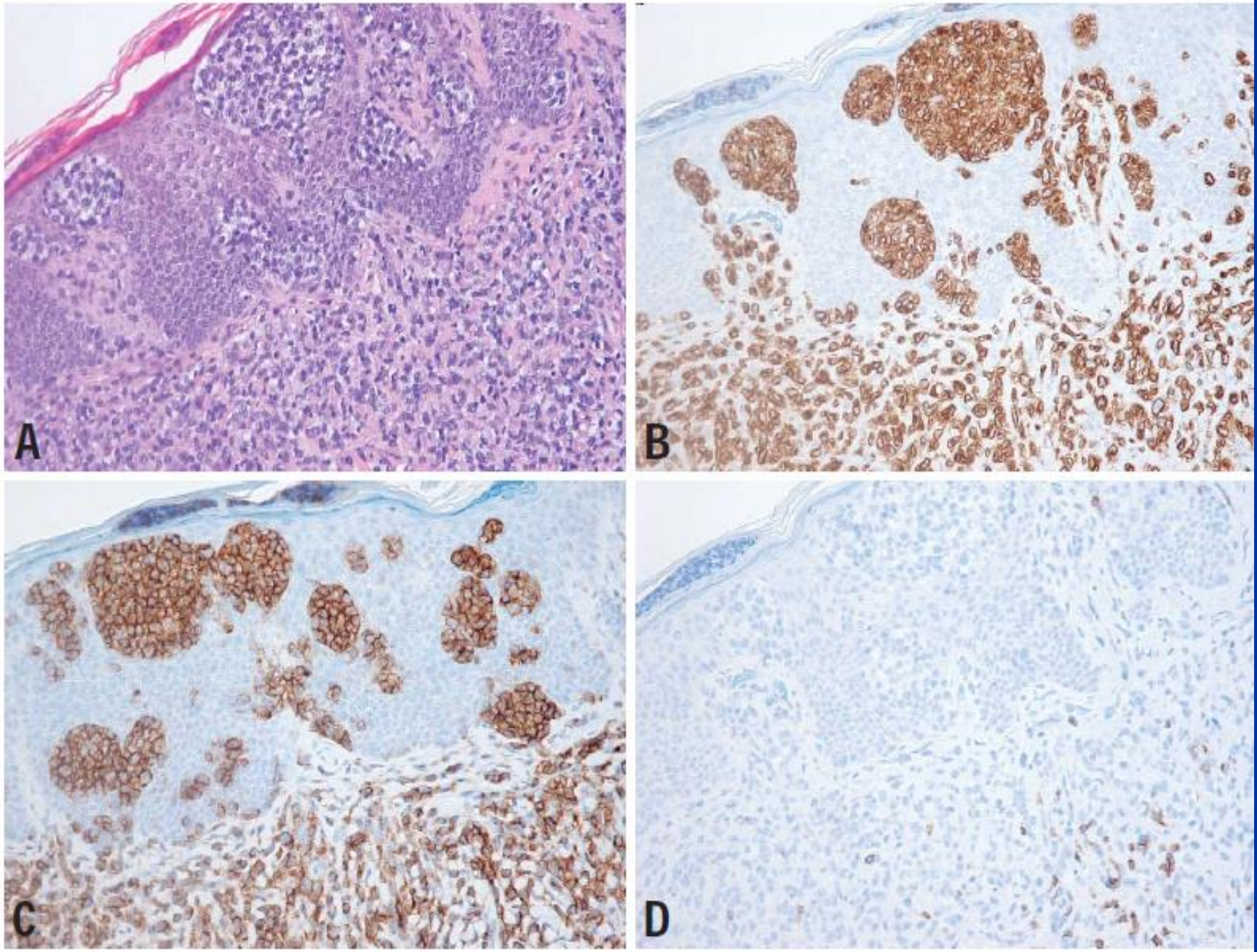
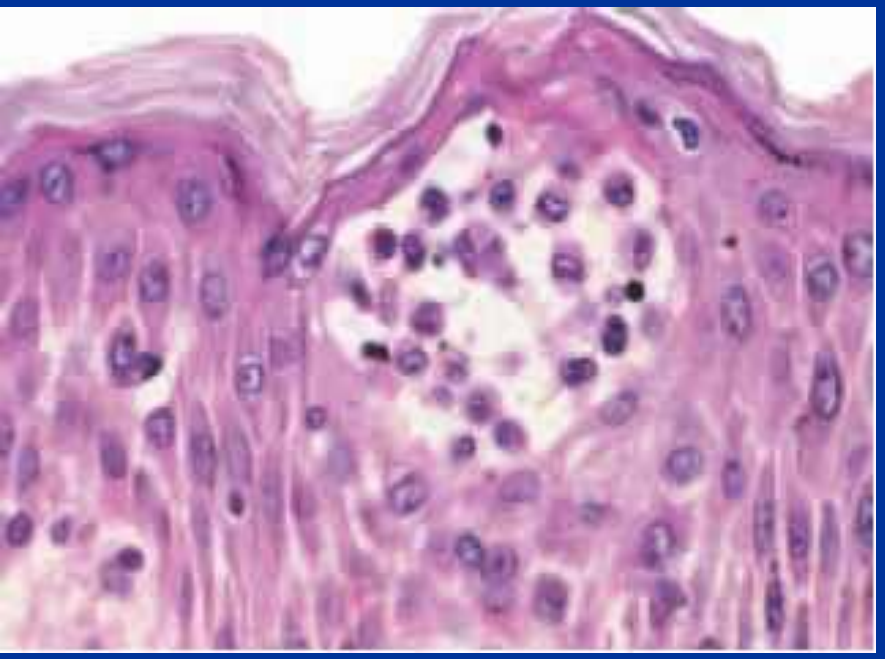
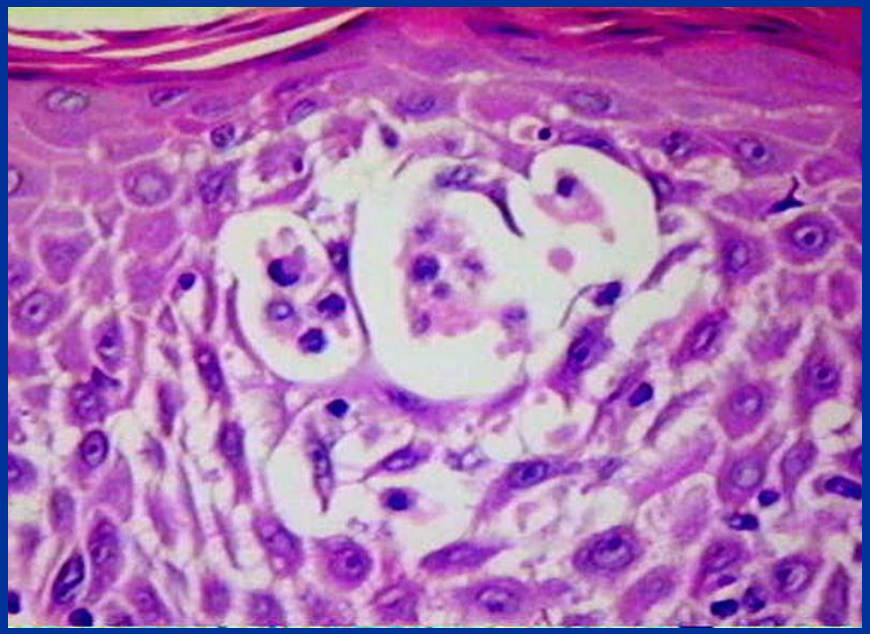


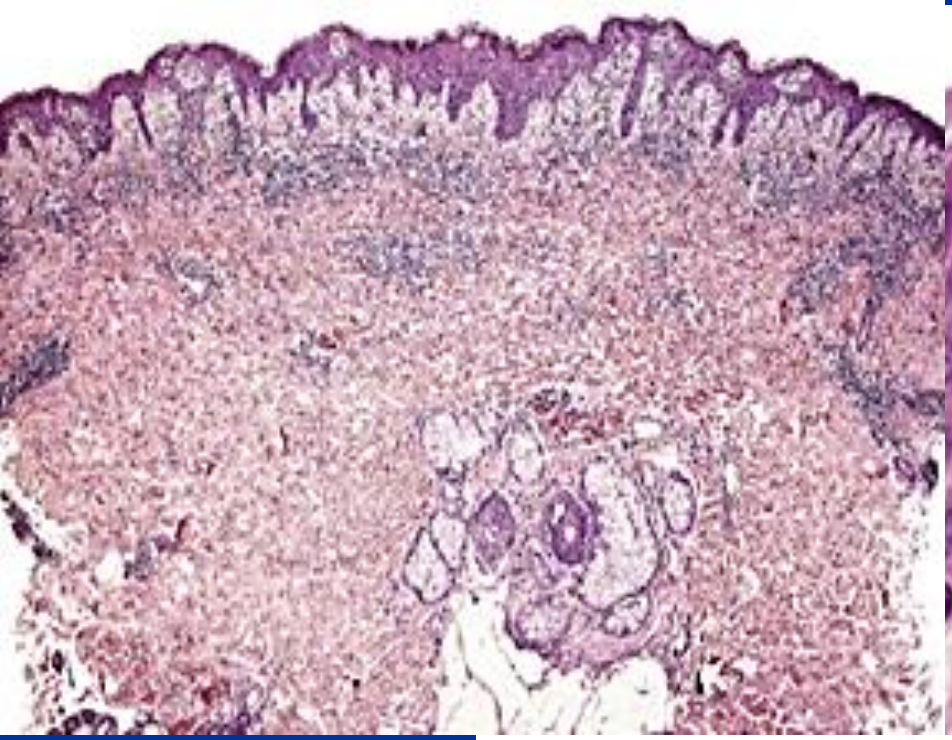
Fig. 4.2 Mycosis fungoides (MF). **A** Typical Pautrier abscesses. The neoplastic cells are strongly positive for **B** CD3 and **C** CD4, while **D** CD8 is negative.

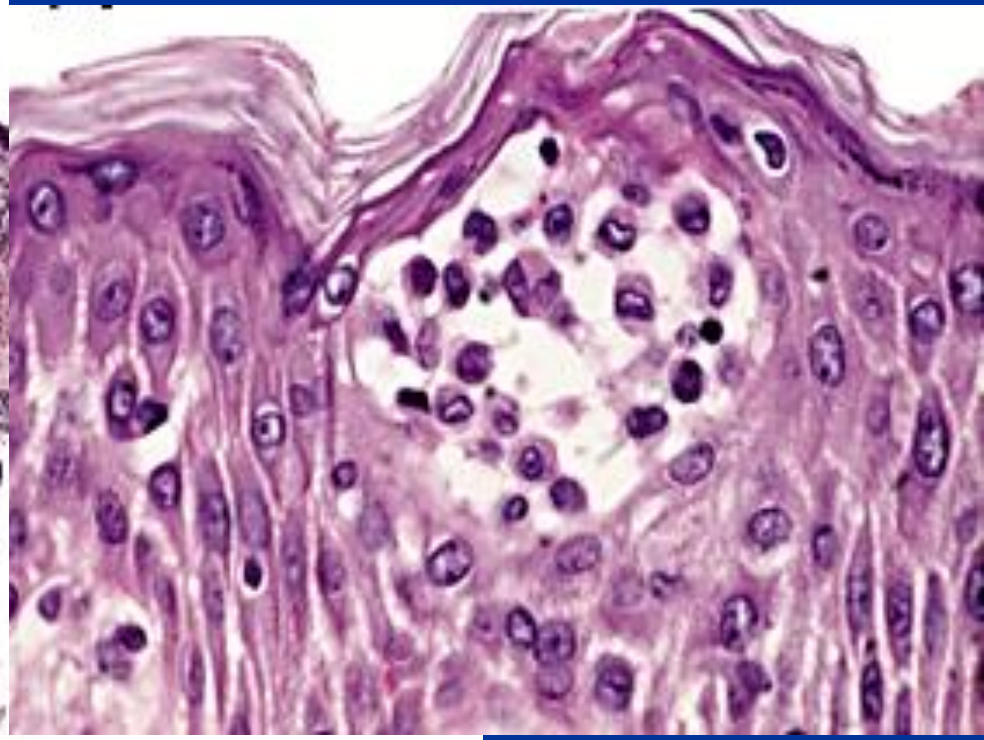


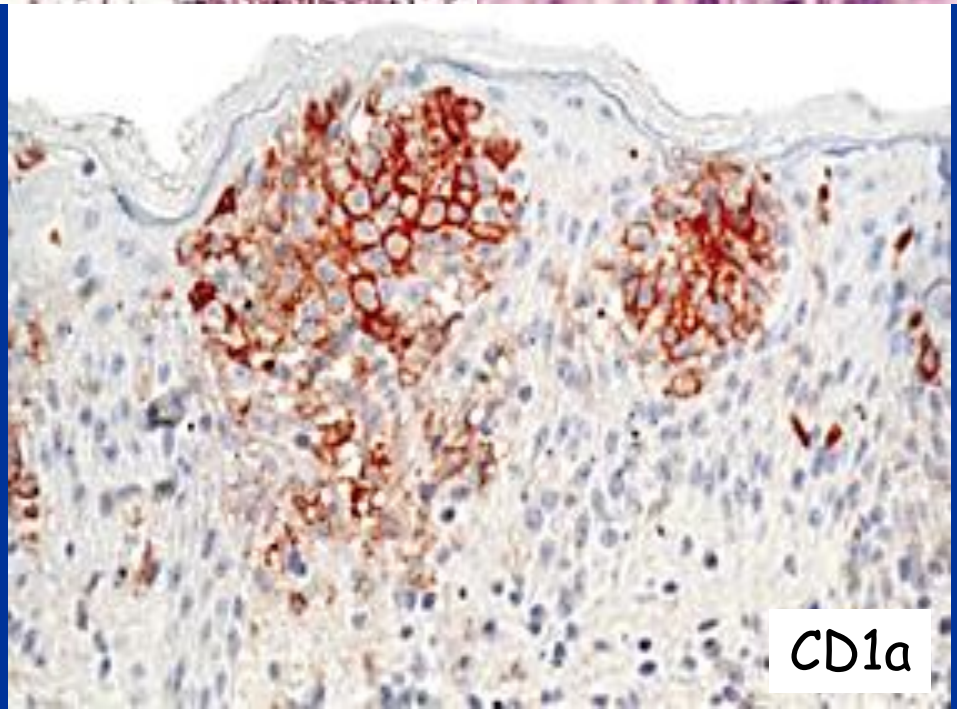


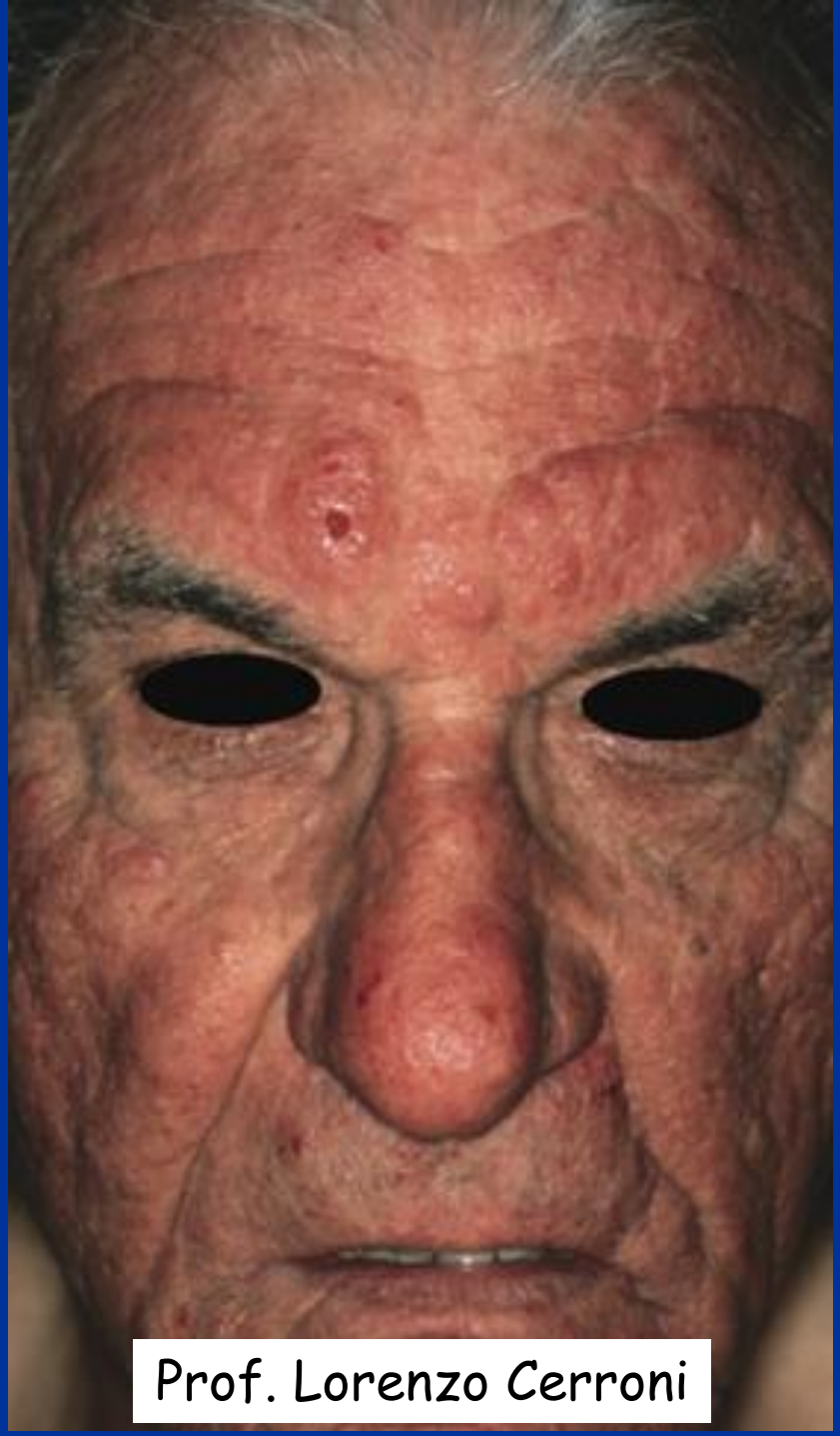
An example of pseudo-Patrier's microabscess provided by another famous dermatopathologist

An example of Patrier's microabscess provided by a famous dermatopathologist






CD1a


Prof. Lorenzo Cerroni

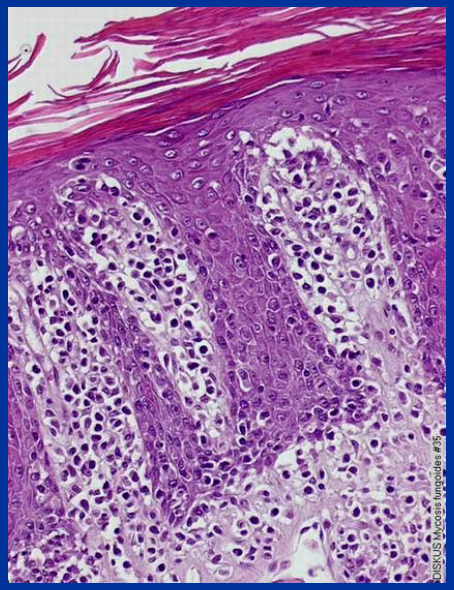
Pattern psoriasiforme puro

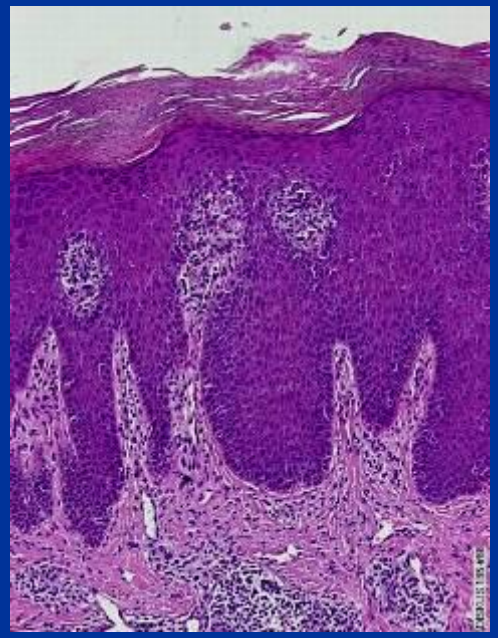
Psoriasi
Dermatite seborroica
Pitiriasi rubra pilare
Dermatofitosi

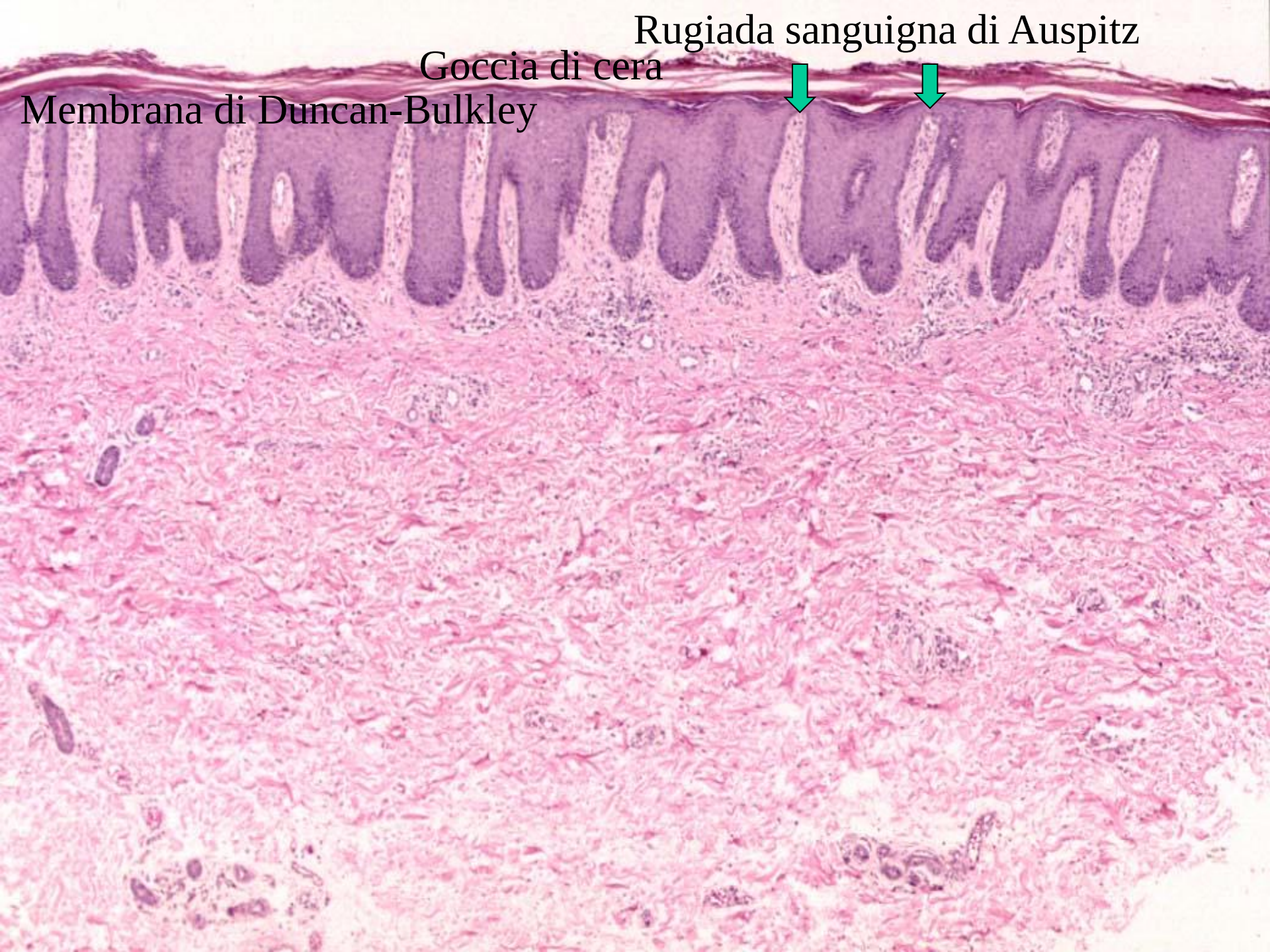
Psoriasiform pattern

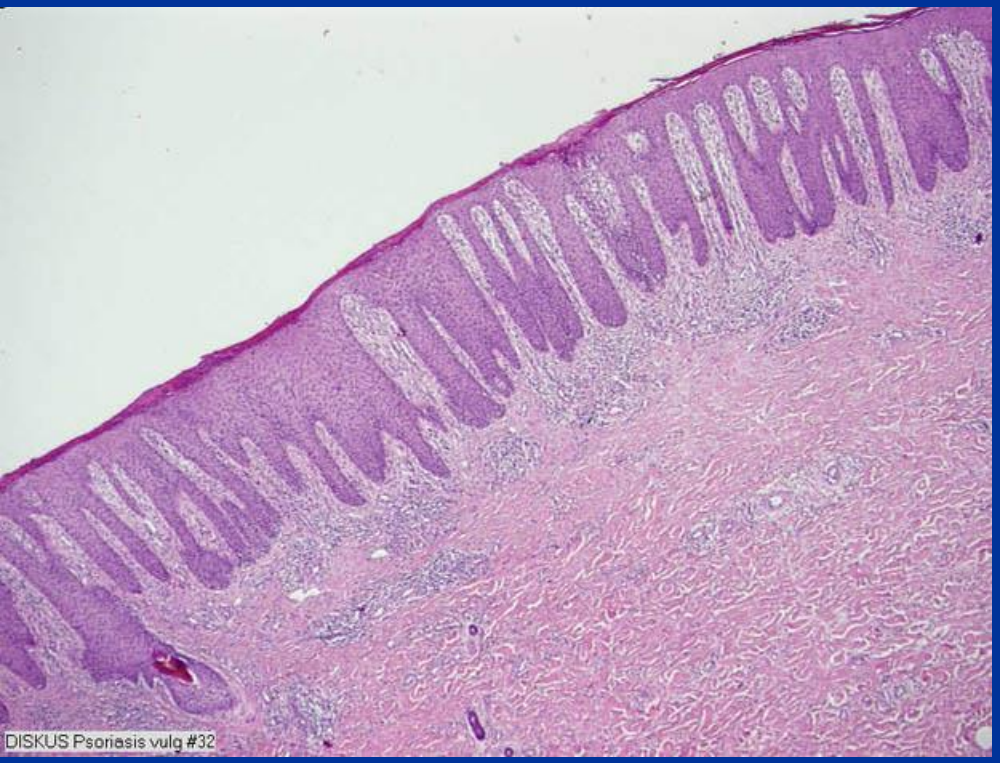
- Regular acanthosis of the epidermis, but with suprapapillary thinning
- Parakeratosis and/or orthokeratosis
- Variable acute inflammation, especially involving the epidermis & stratum corneum ("Munro" & "Kogoj" microabscesses) in psoriasis vulgaris *(the 3 primary diseases featuring neutrophils in the stratum corneum are psoriasis, the superficial dermatophytoses, & PLEVA)*
- Perivascular chronic dermal inflammation
- Papillary dermal hypervascularity

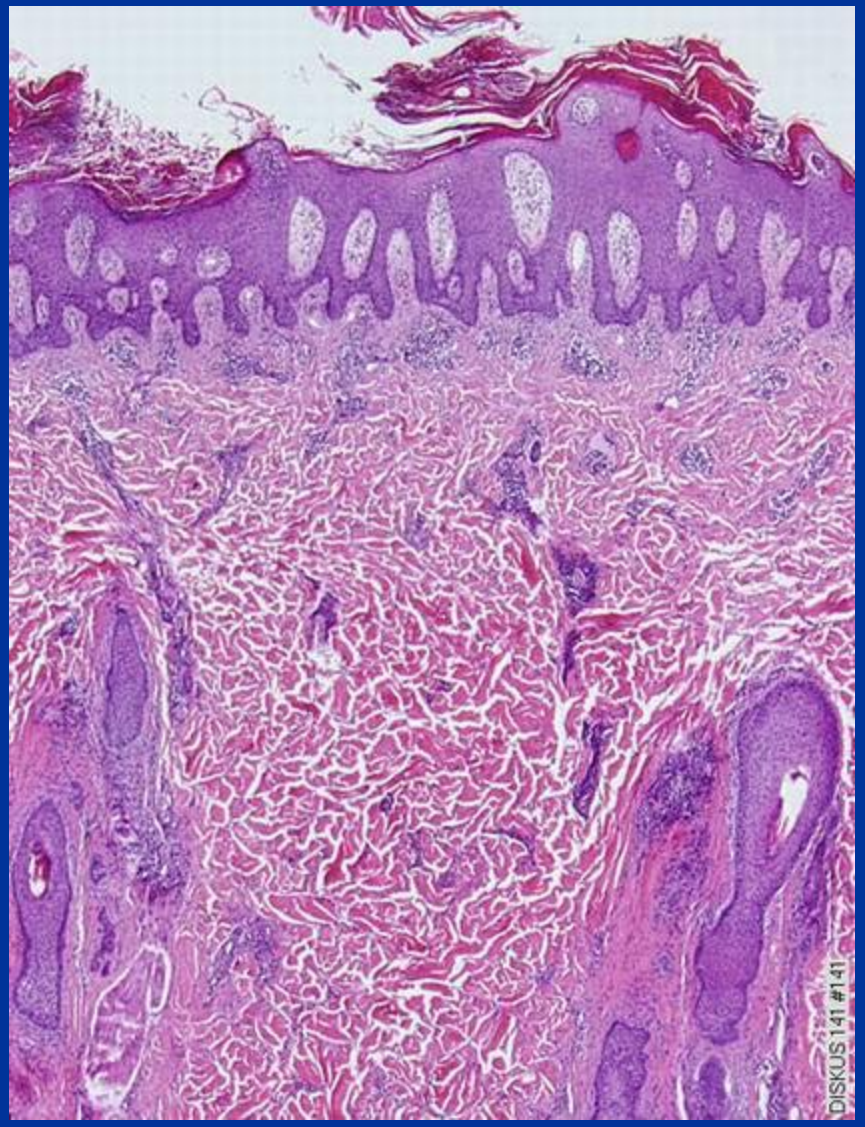

Che cosa significa pattern psoriasiforme???






Rugiada sanguigna di Auspitz
Goccia di cera
Membrana di Duncan-Bulkley


DISKUS Psoriasis vulg #32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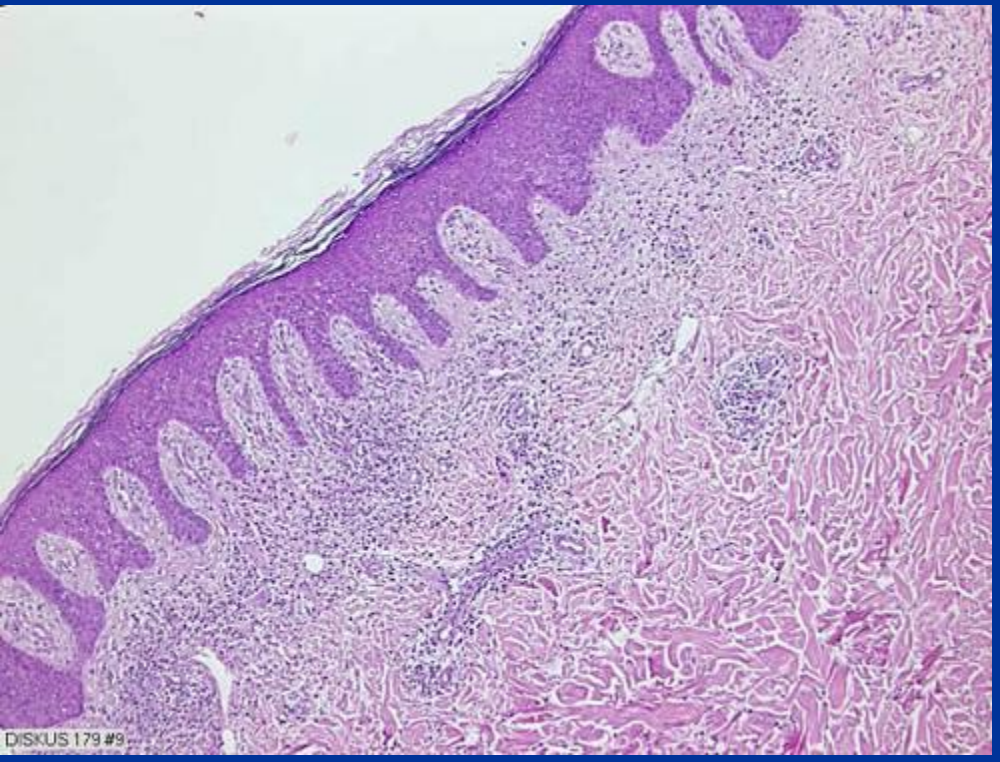
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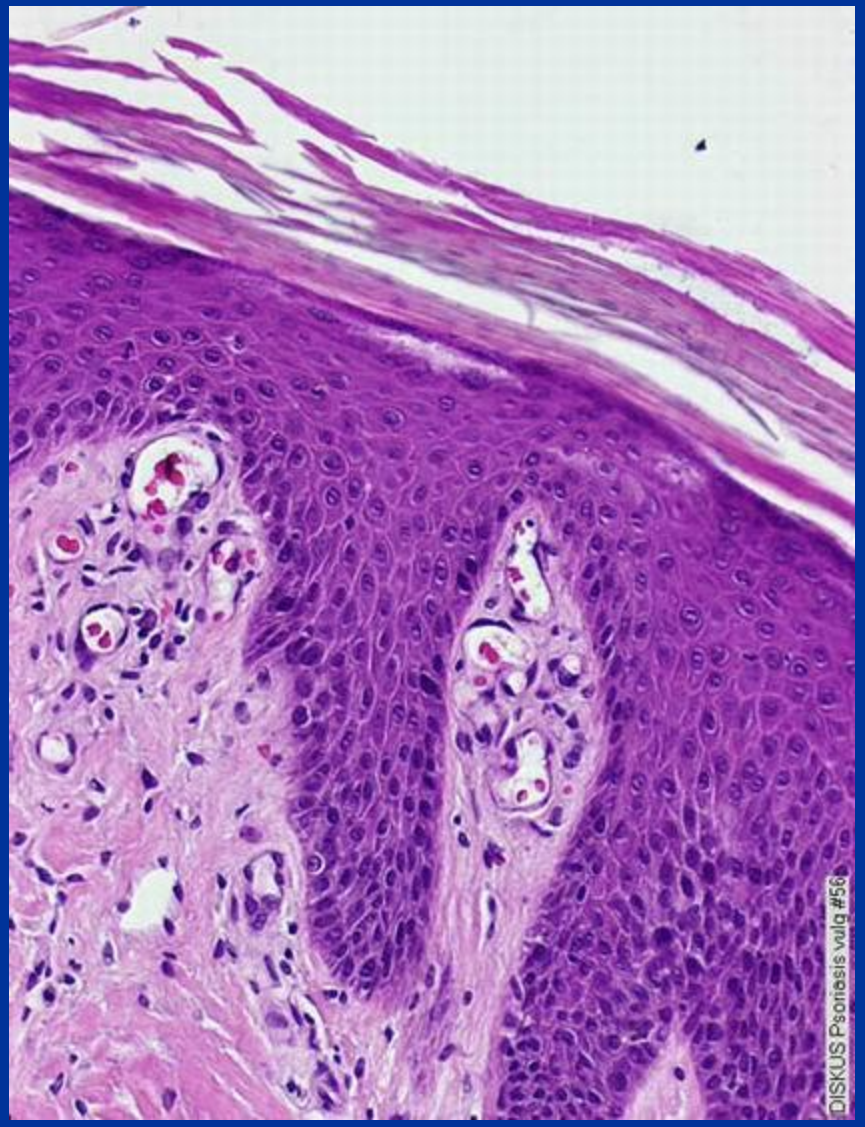
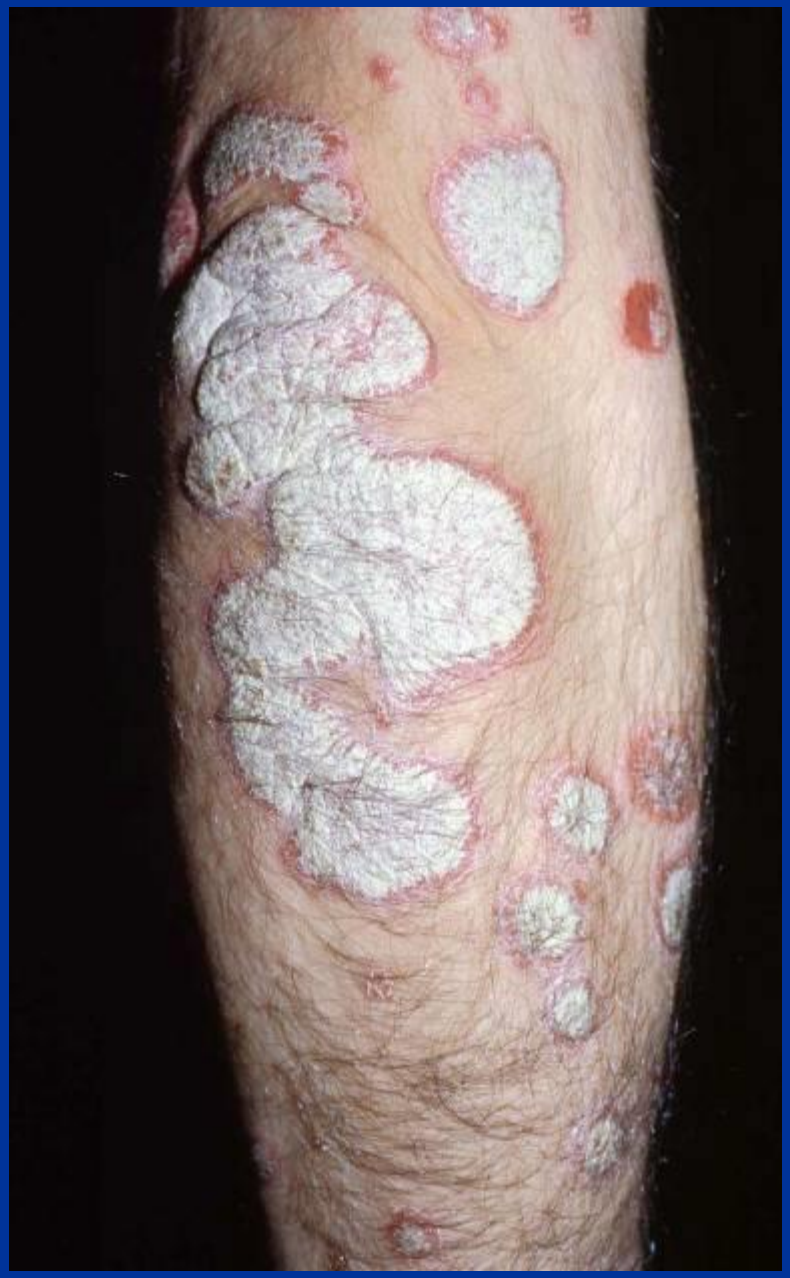
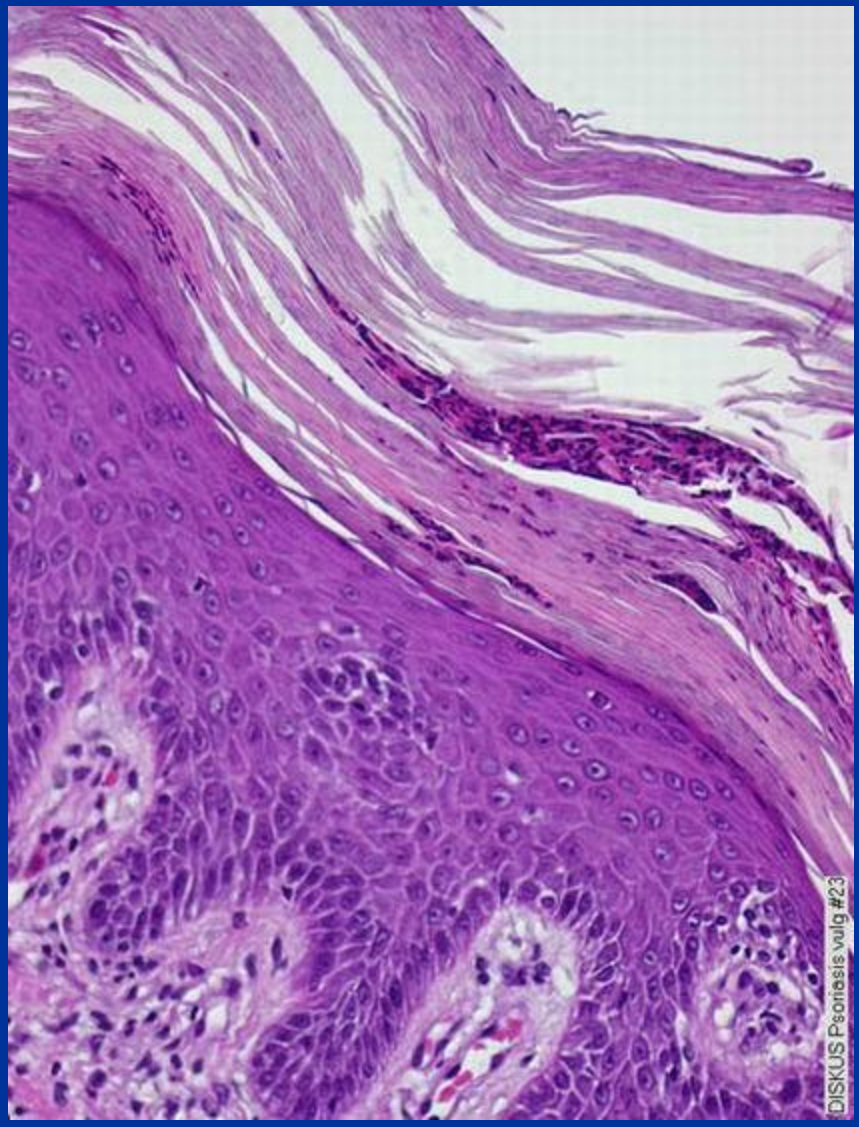
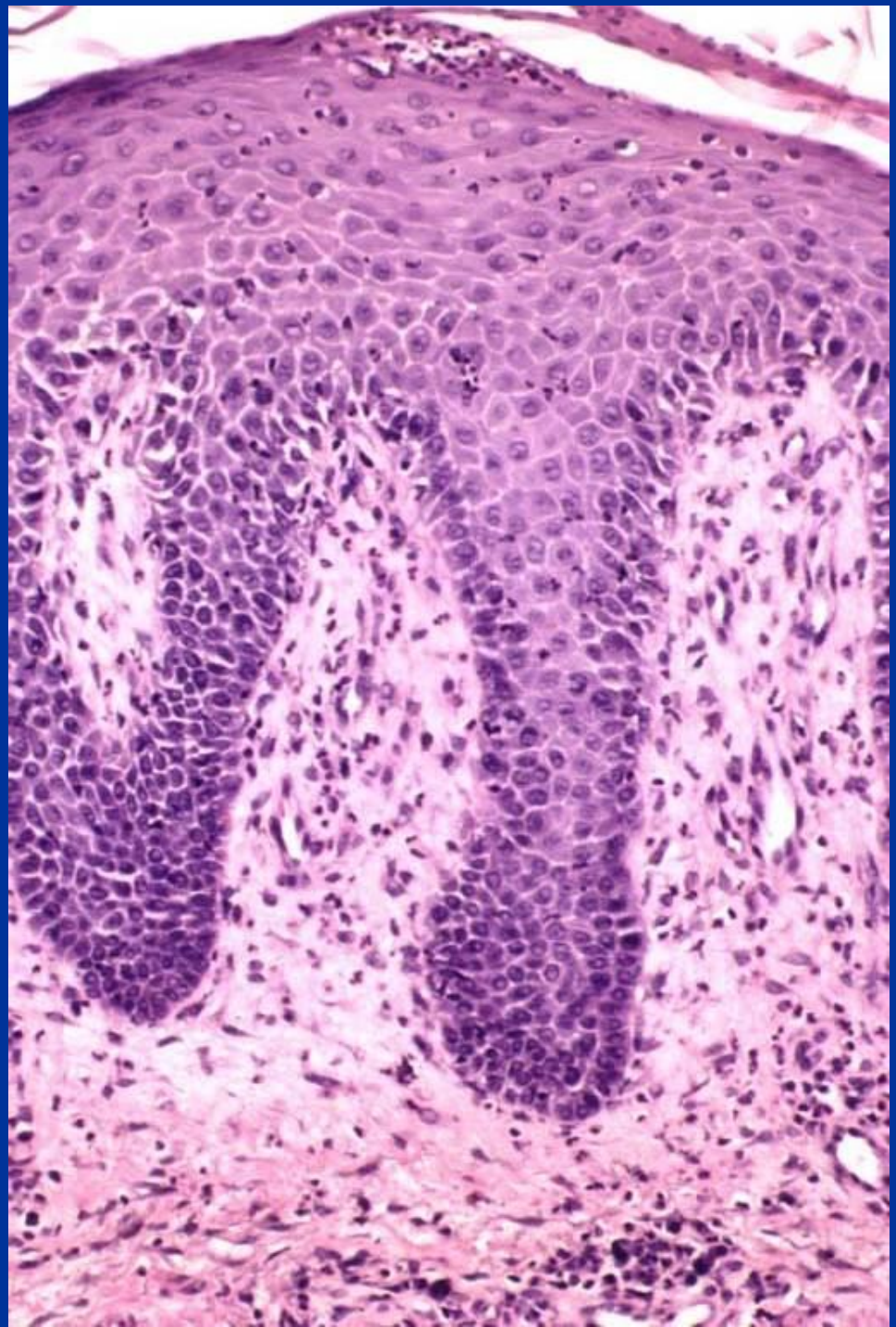

DISKUS Psoriasis vulg #56




DISKUS Psoriasis vulg #23



Ann Dermatol Vol. 27, No. 1, 2015 http://dx.doi.org/10.5021/ad.2015.27.1.26

ORIGINAL ARTICLE

Histopathological Findings Are Associated with the Clinical Types of Psoriasis but Not with the Corresponding Lesional Psoriasis Severity Index

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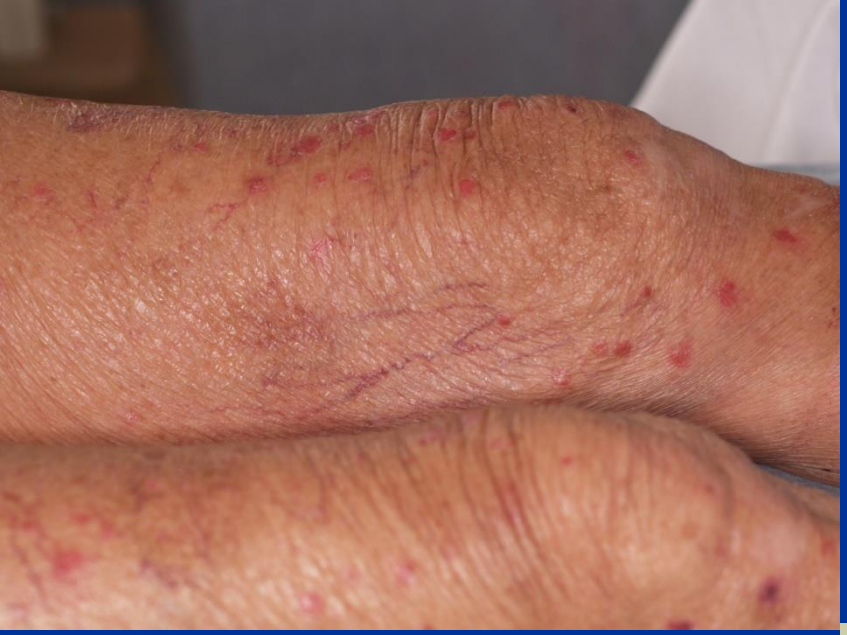
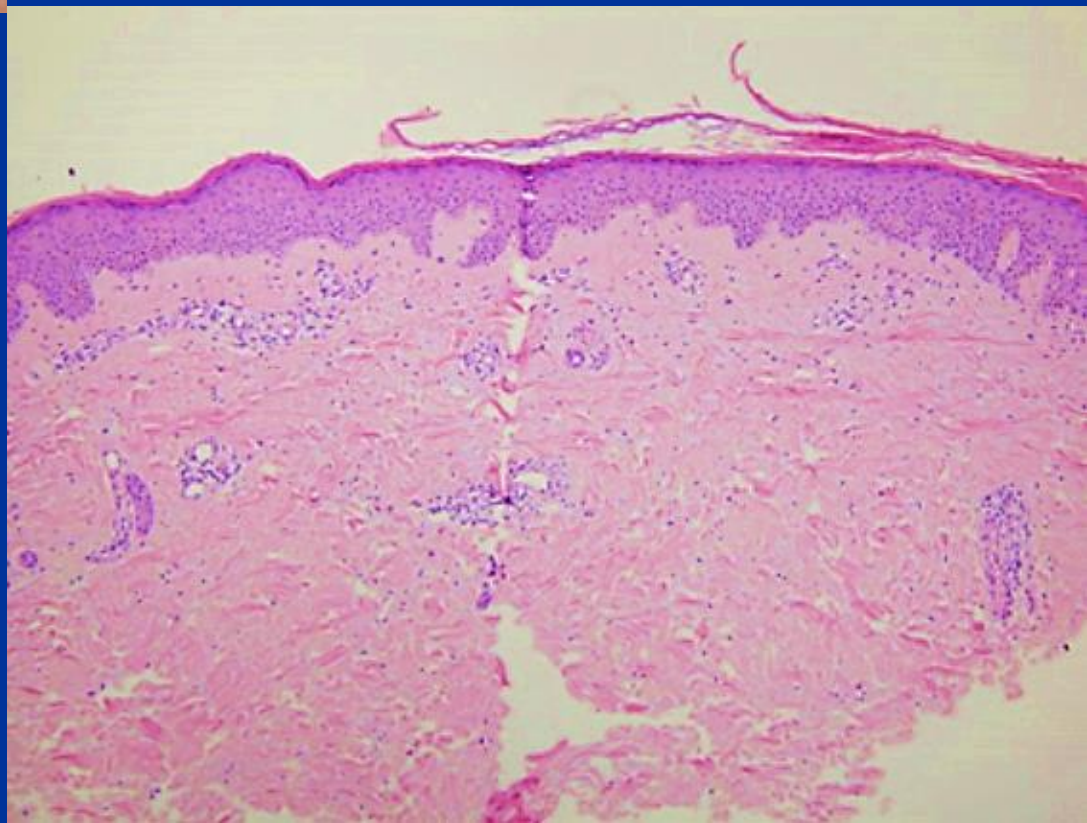
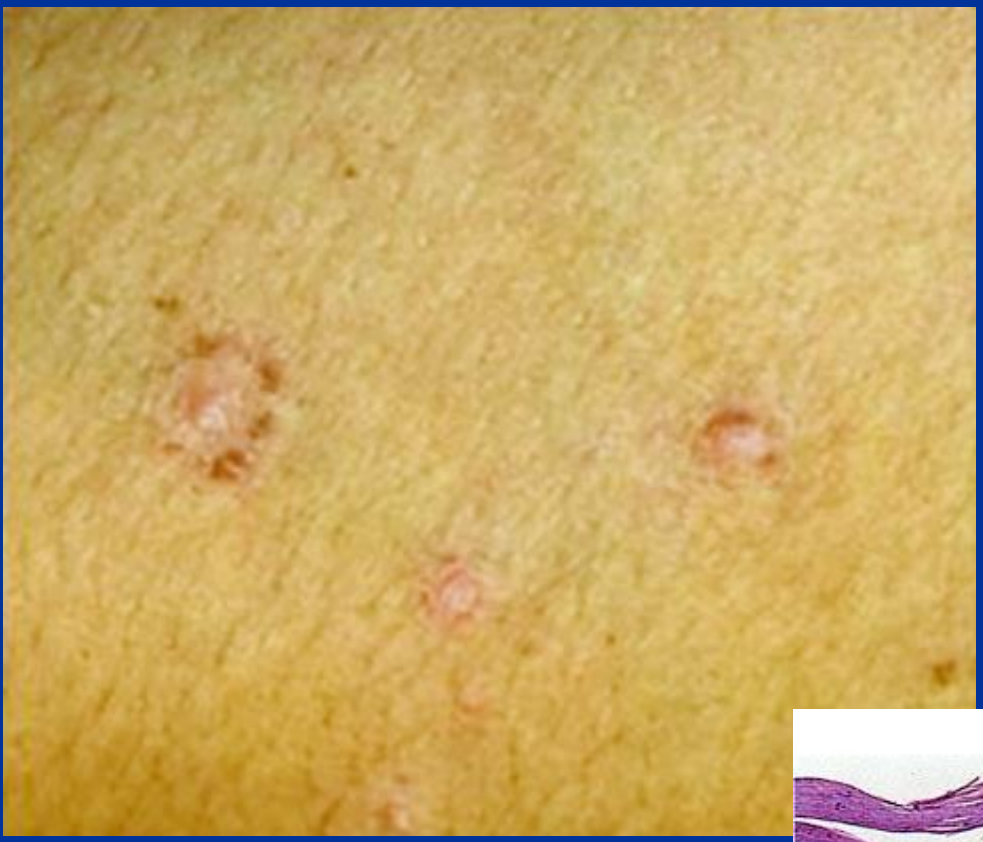
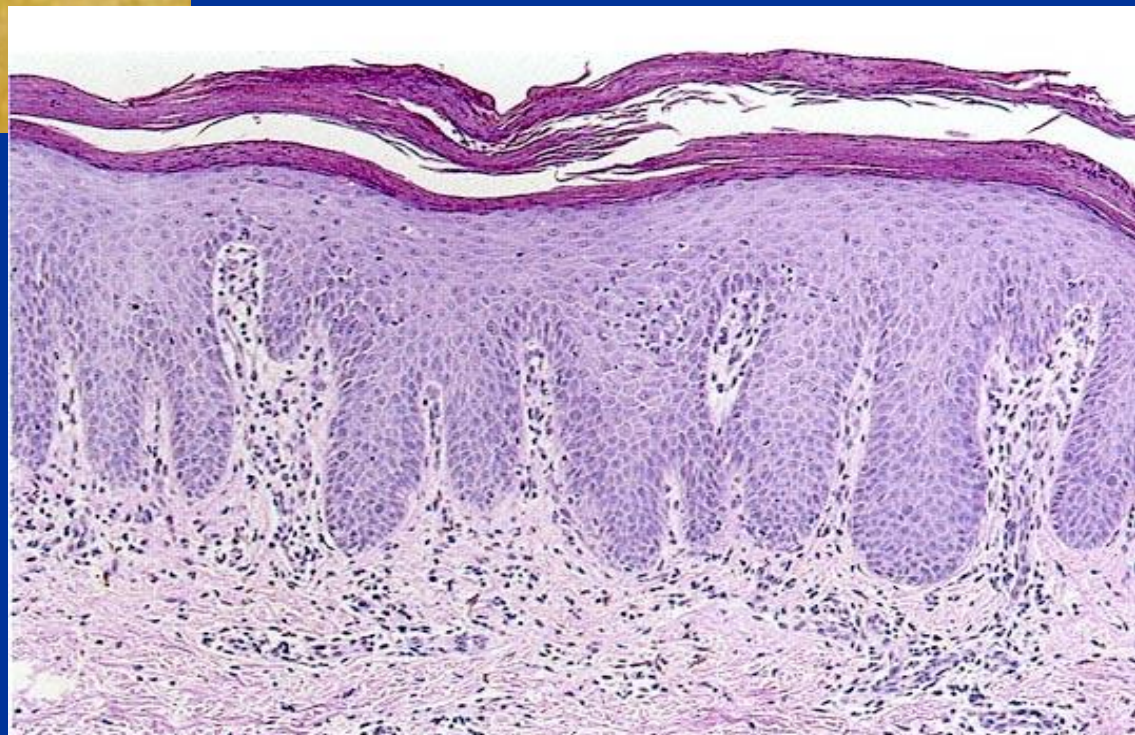
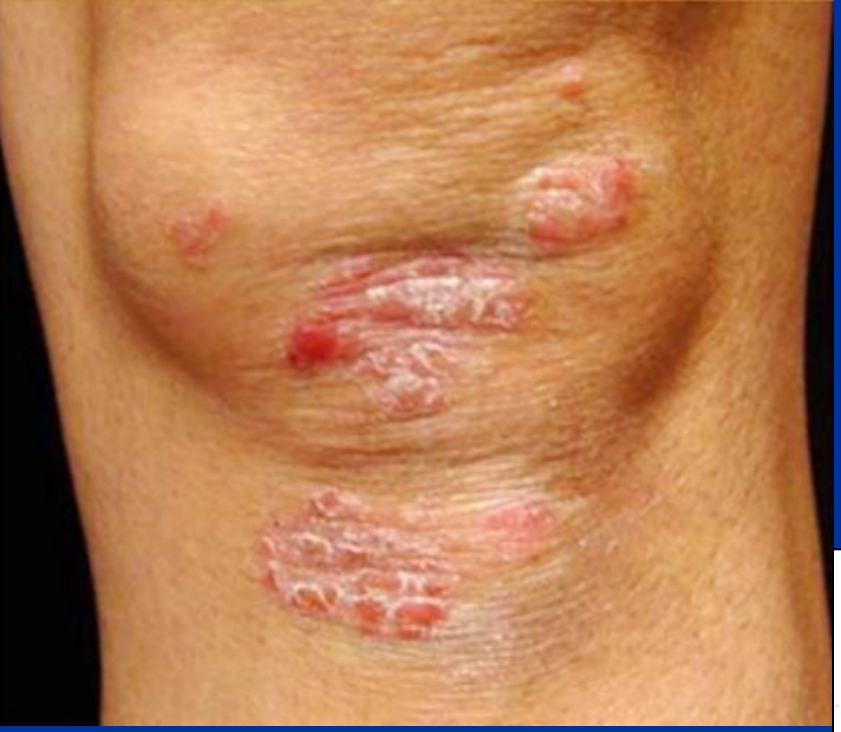
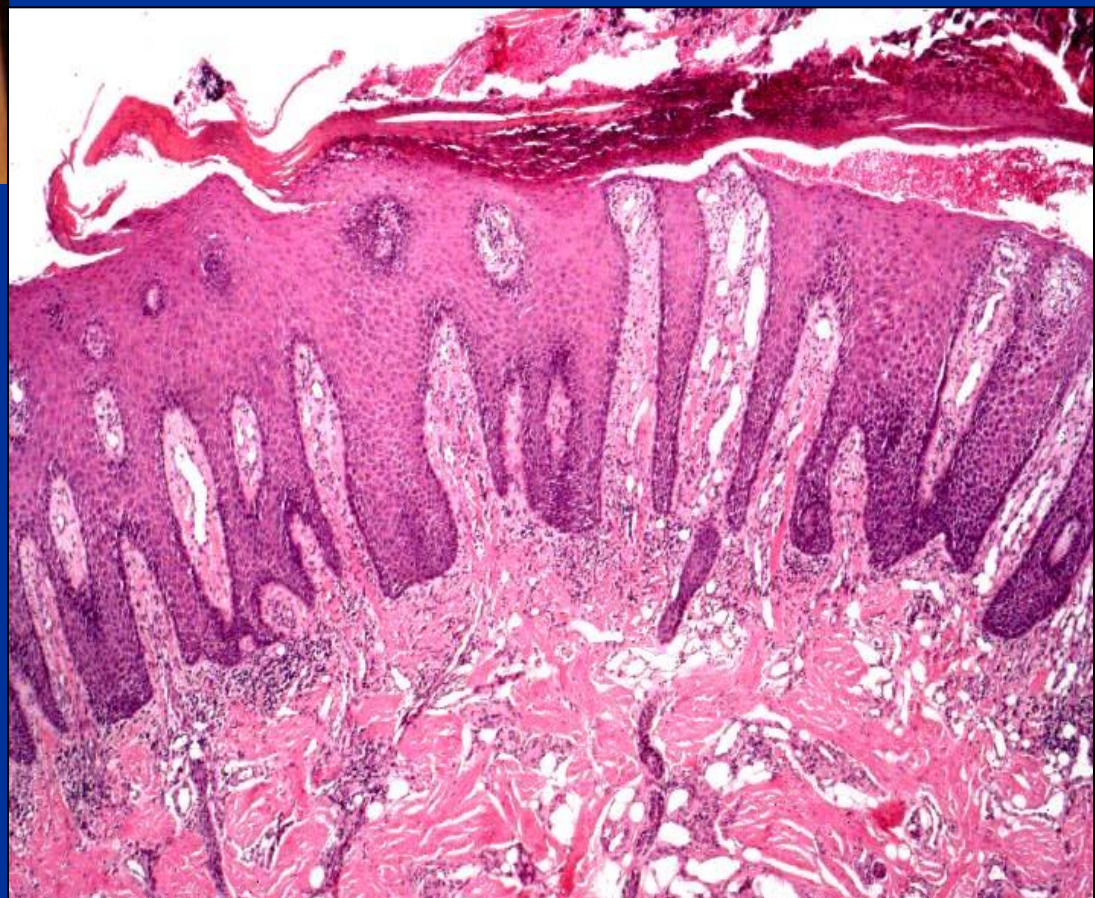


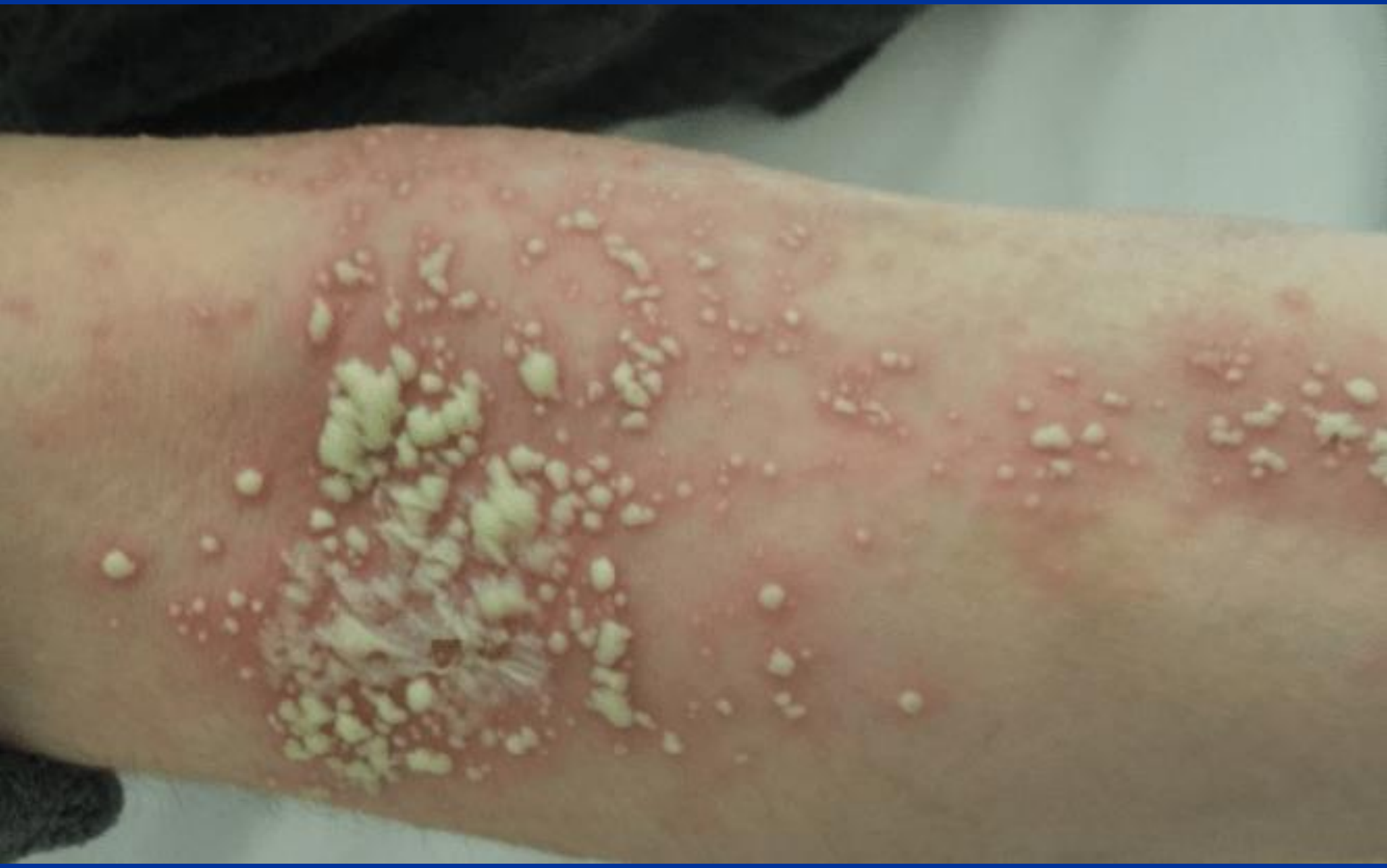


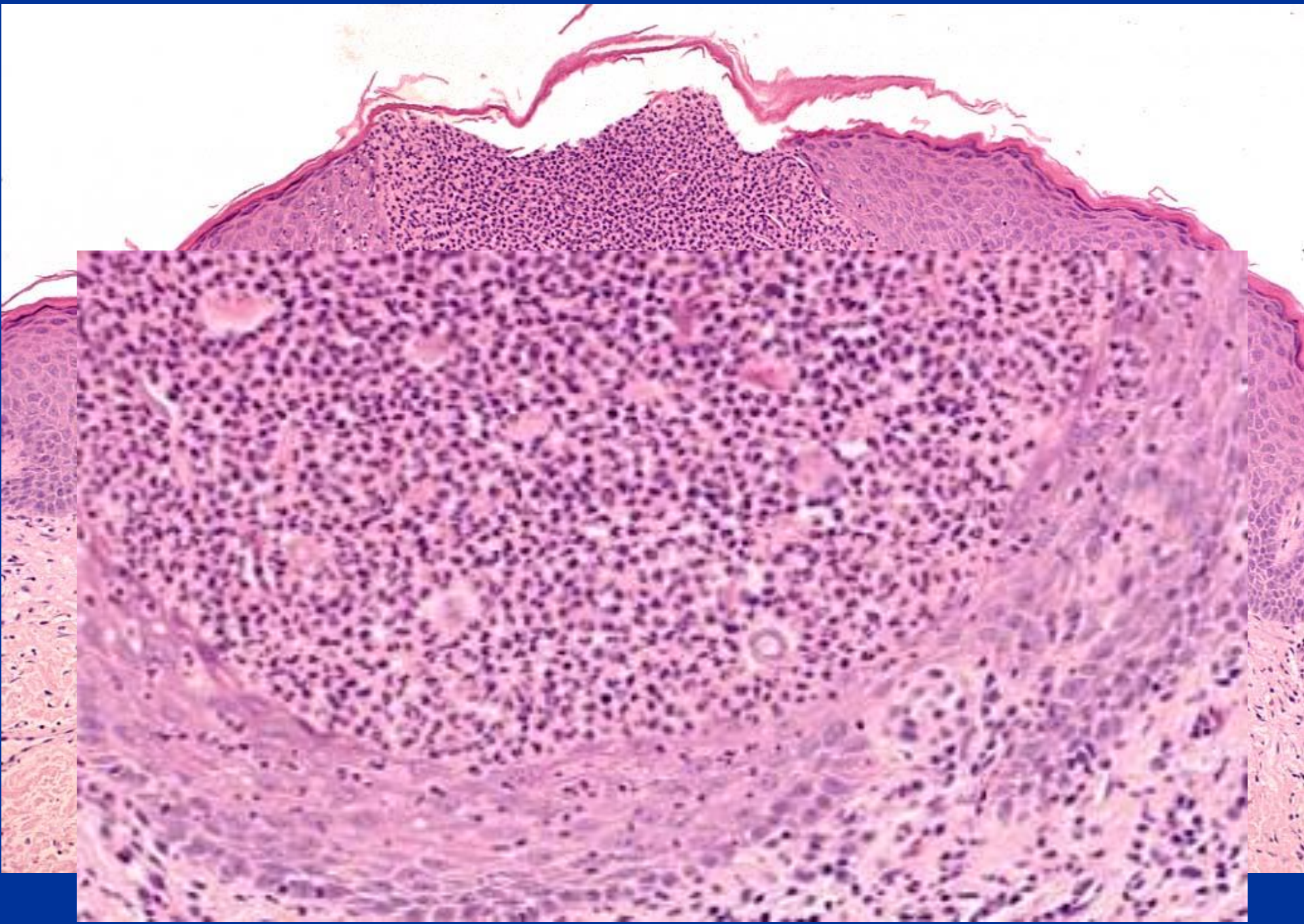




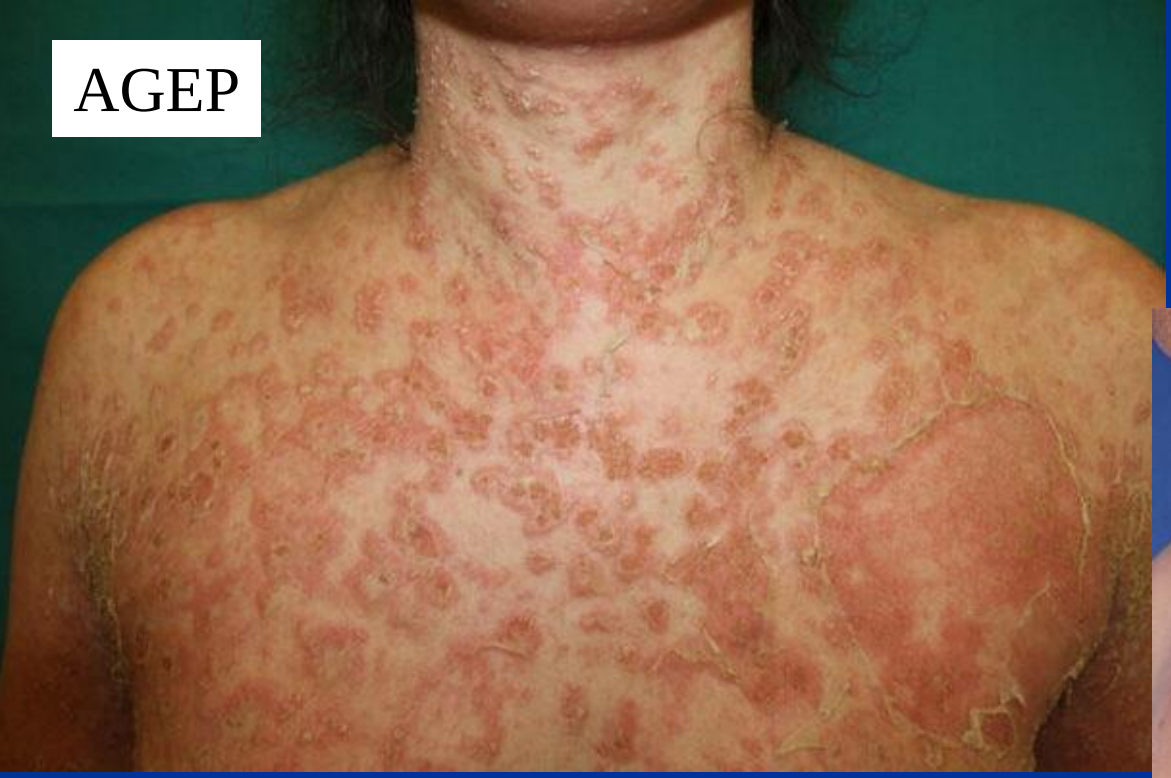




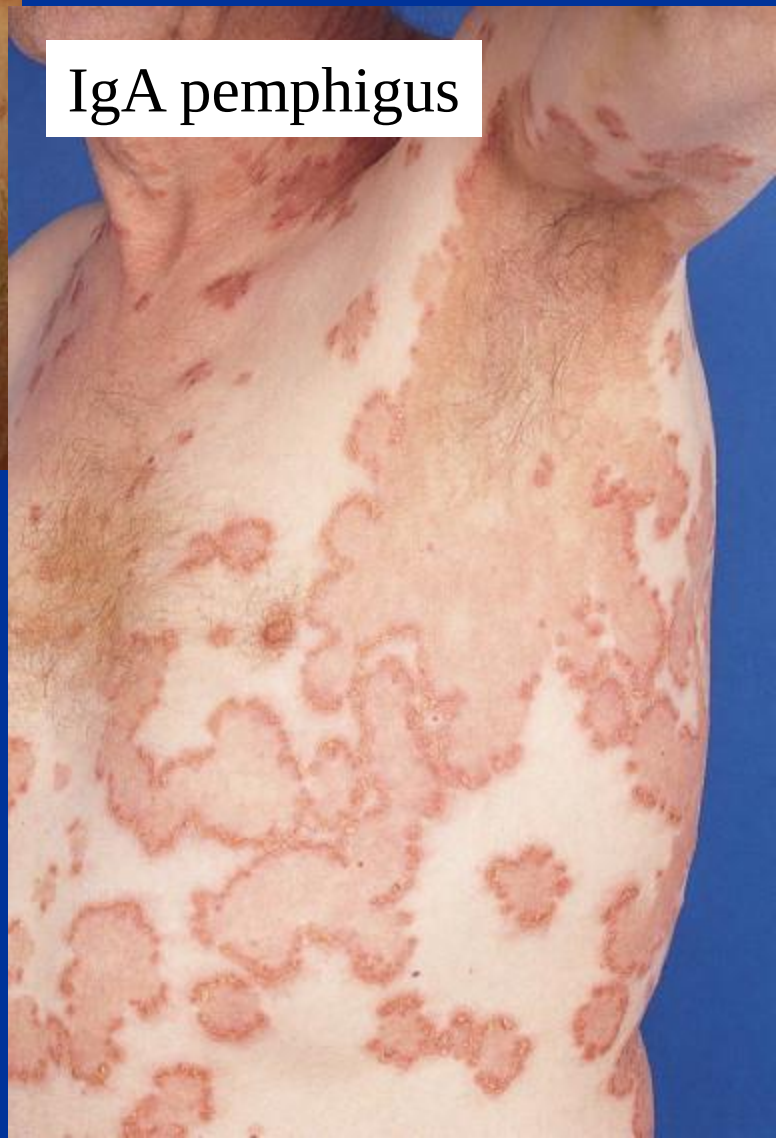




AGEP



IgA pemphigus



Clinical and Laboratory Investigations

Dermatology

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Psoriatic Erythroderma: A Histopathologic Study of Forty-Five Patients

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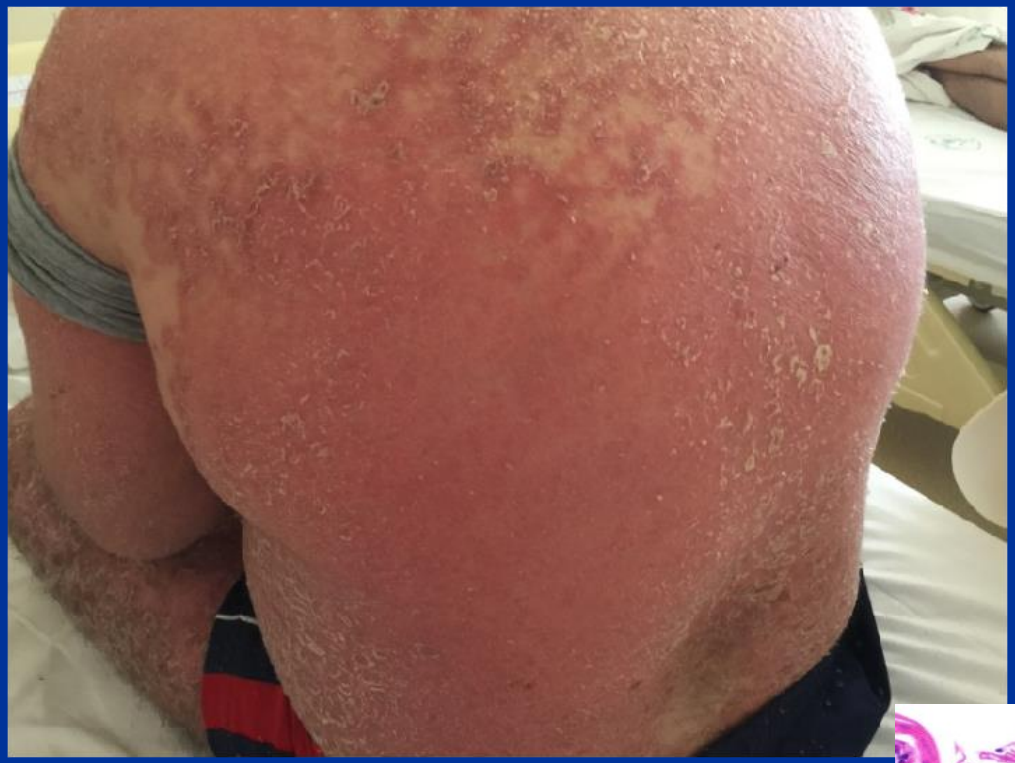
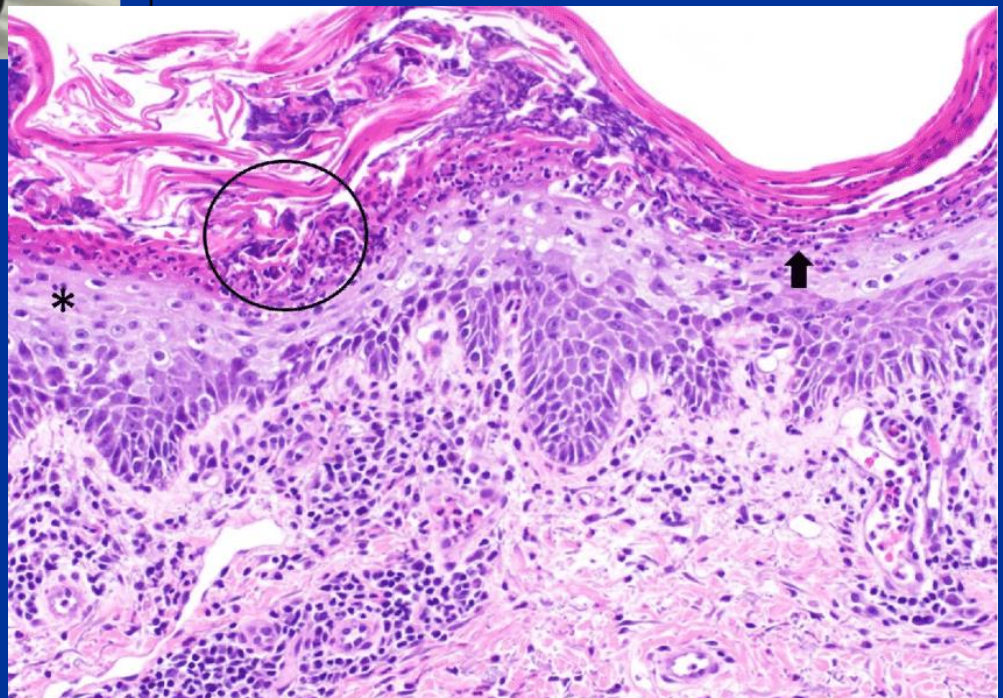
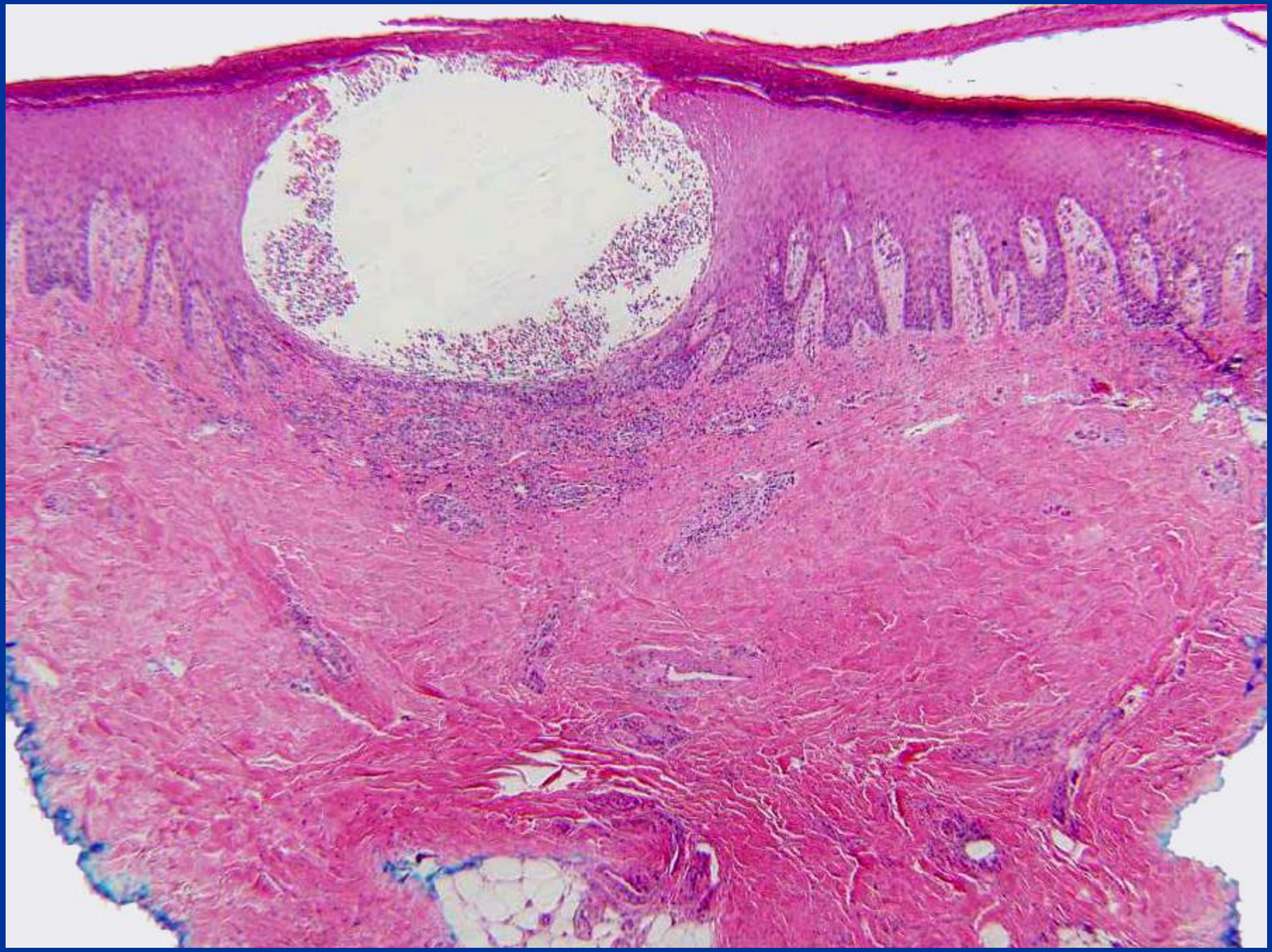
Key Words
Erythroderma
Psoriasis
Histopathology

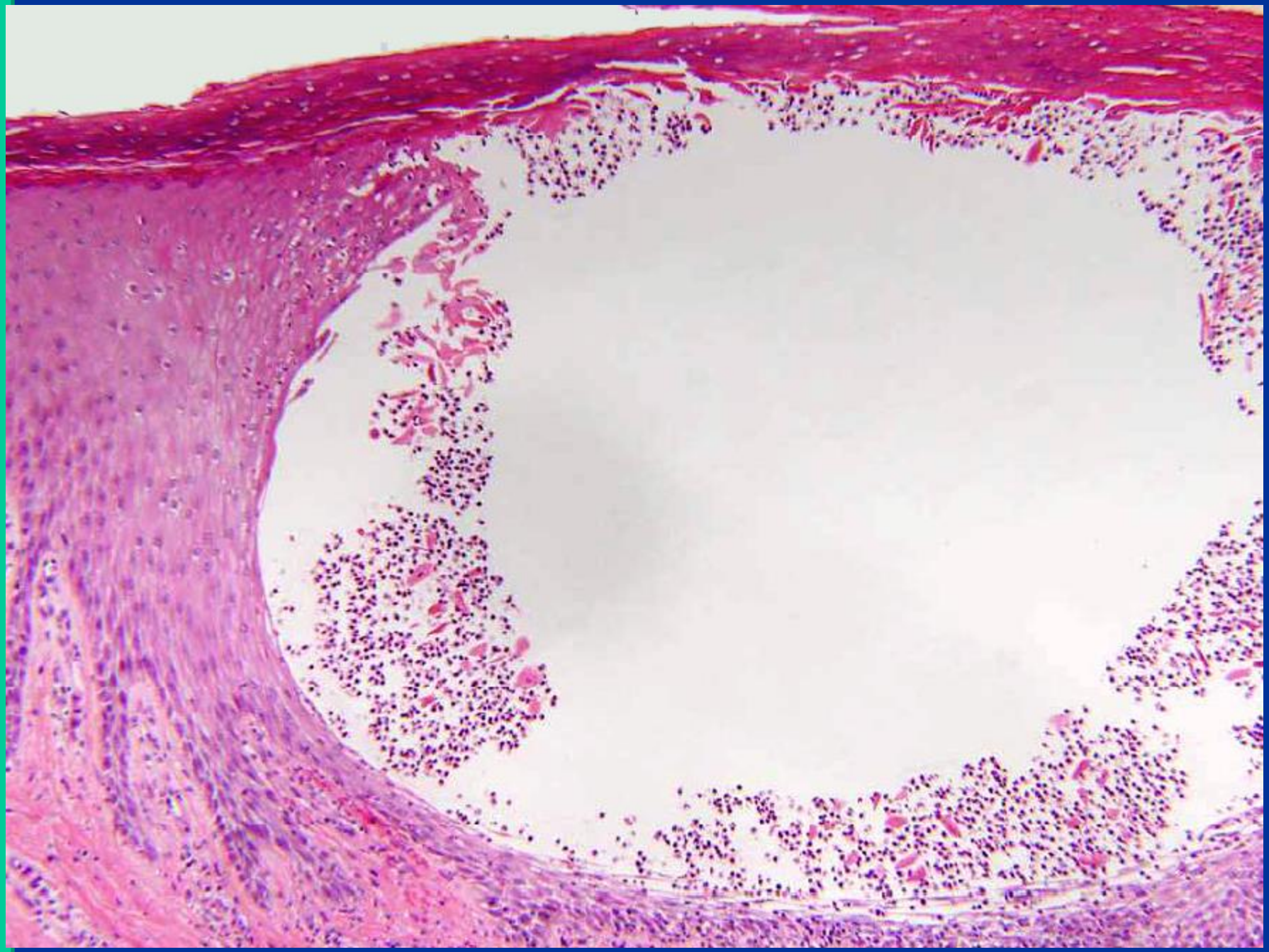
Abstract

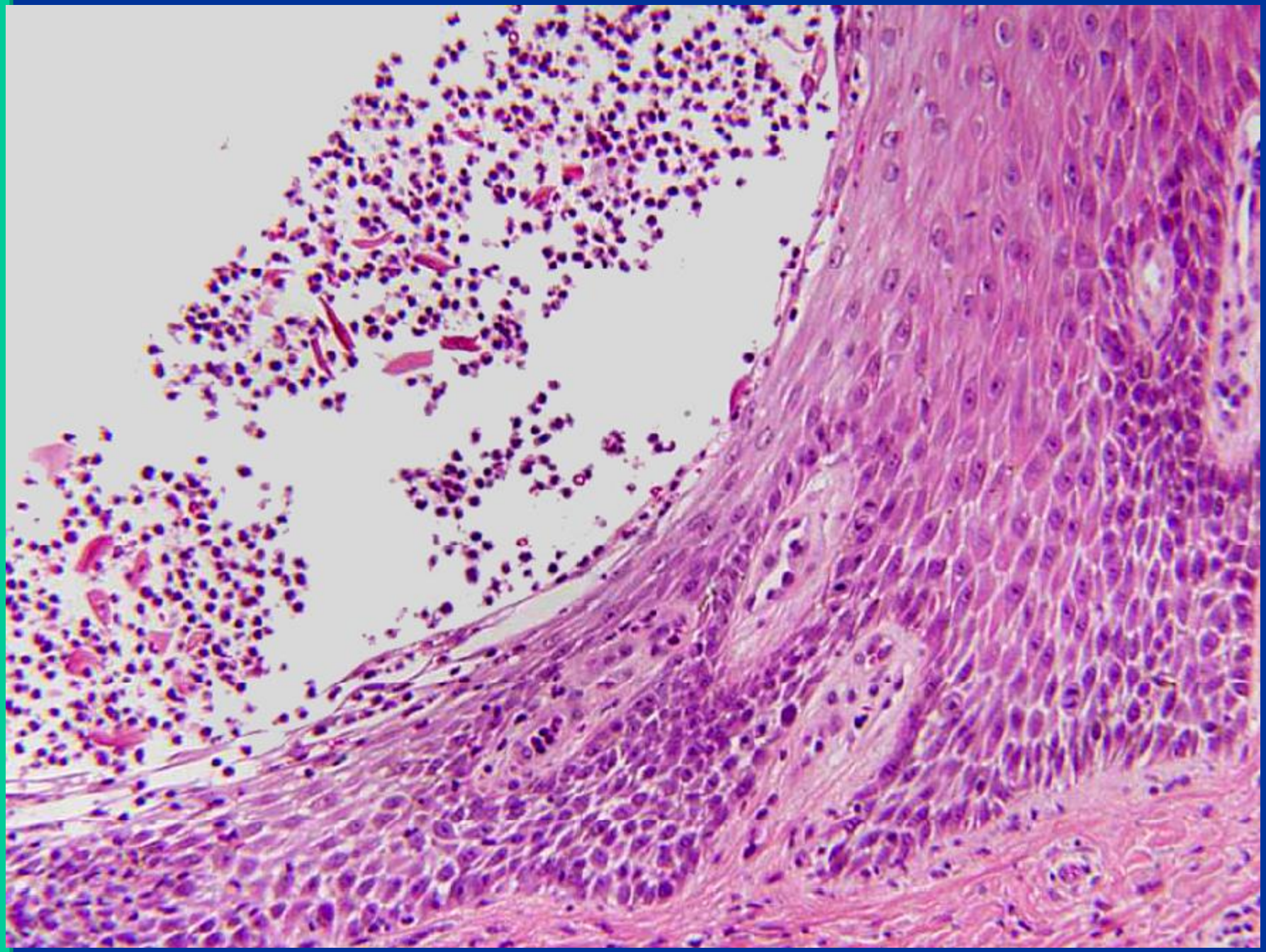
Background: There are conflicting opinions about the diagnostic value of skin biopsy in erythrodermic psoriasis. **Objective:** The purpose of the present study was to establish the specificity of the histopathologic changes of psoriatic erythroderma. **Methods:** We reviewed 52 skin biopsies from 45 erythrodermic patients having a final diagnosis of psoriasis on the basis of combined clinical and laboratory data, in addition to response to therapy and follow-up. In 5 patients, erythroderma was the presenting sign of psoriasis. A control group of nonpsoriatic erythrodermic patients was also included in the study. **Results:** Among the group of patients with a discharge diagnosis of psoriatic erythroderma, the histopathologic changes were specific for psoriasis in 40 cases (88%). The changes of early macular and squamous lesions of psoriasis were more often found in the biopsy specimens of our series than those of fully developed or late lesions of psoriasis. They included mainly slight epidermal hyperplasia, focal disappearance of the granular layer, mounds of parakeratosis and extravasated erythrocytes within edematous dermal papillae associated with perivascular and interstitial infiltration of lymphocytes and histiocytes. **Conclusion:** When features of early lesions of psoriasis are found during the evaluation of a biopsy specimen from a patient with a clinically nonspecific erythroderma, the dermatopathologist should be aware that this patient could have psoriasis and a renewed anamnesis and a close follow-up should be made.

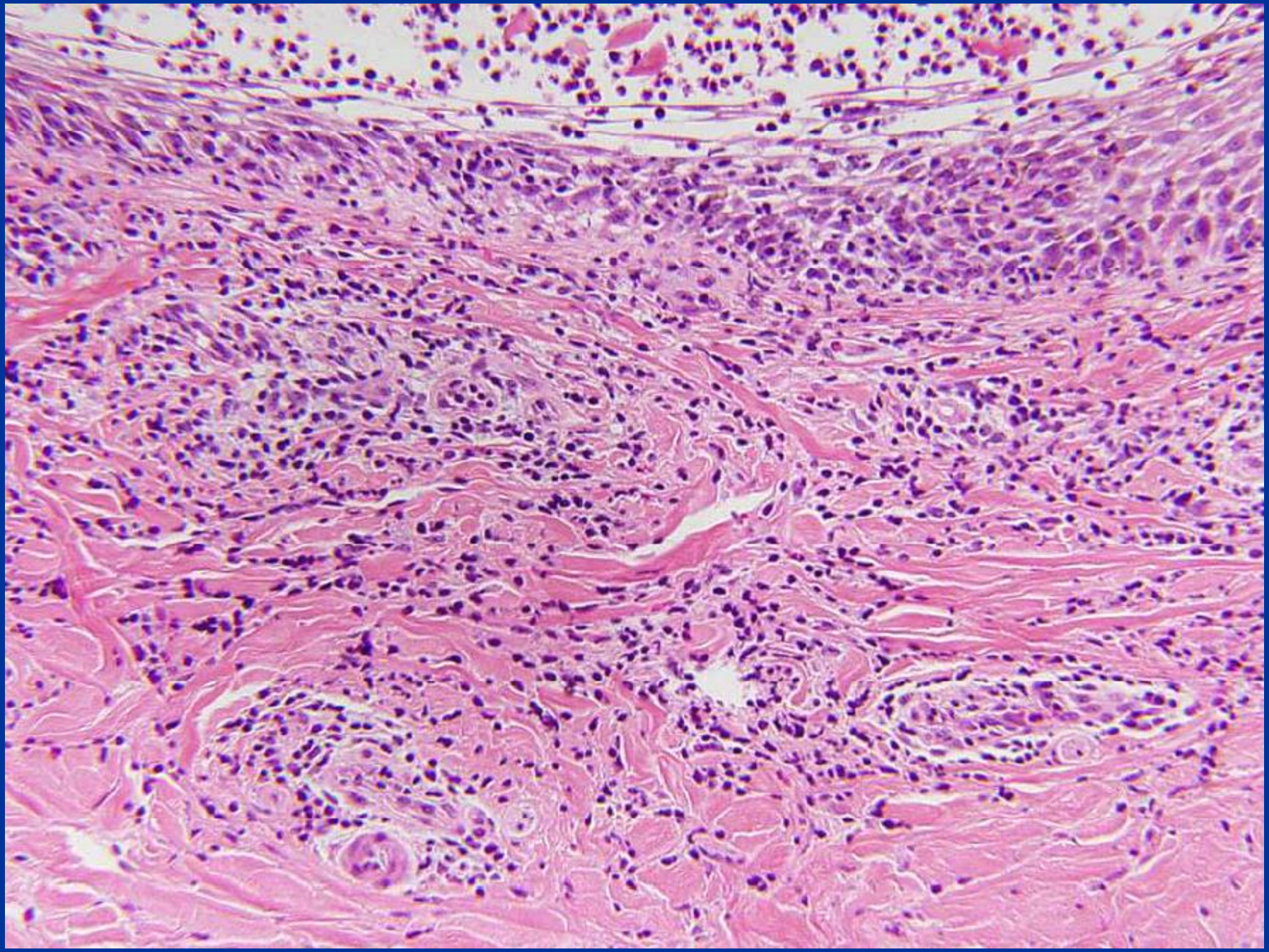


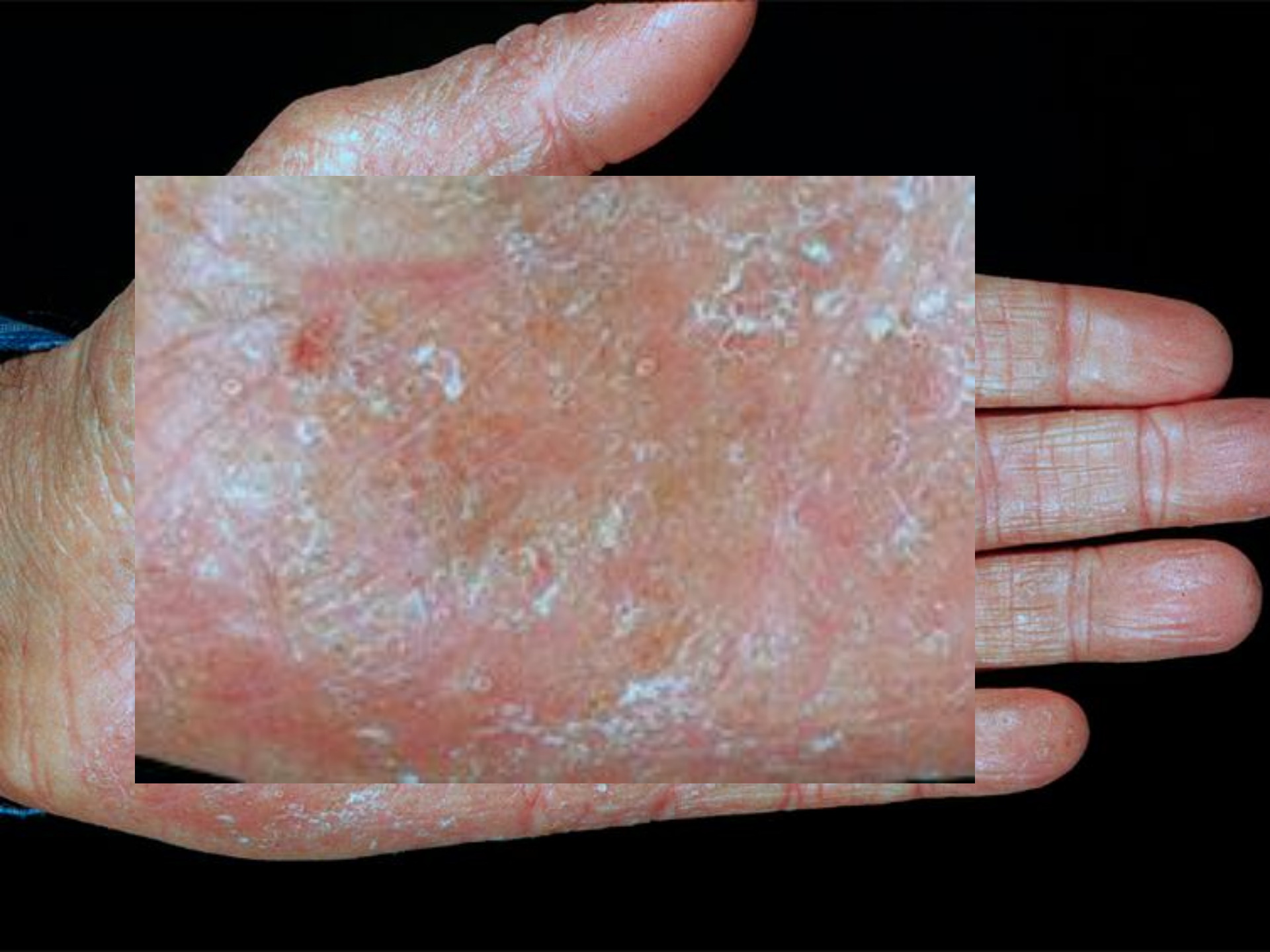






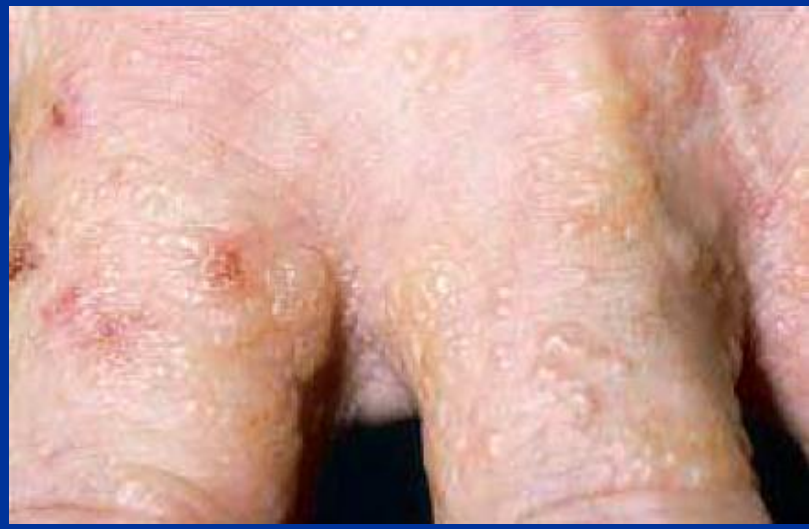






Dyshidrotic eczema

- Age of nset: <40 years
- M=F
- Preciptating factors (emotional stress, hot humid weather)
- Pruritus, pain in fissures, secondary infections
- Hands (80%) and feet (involvement of the lateral aspects is very characteristic
- Confluent tapioca-like vesicles and crusted erosions
- Are pustules part of the spectrum?

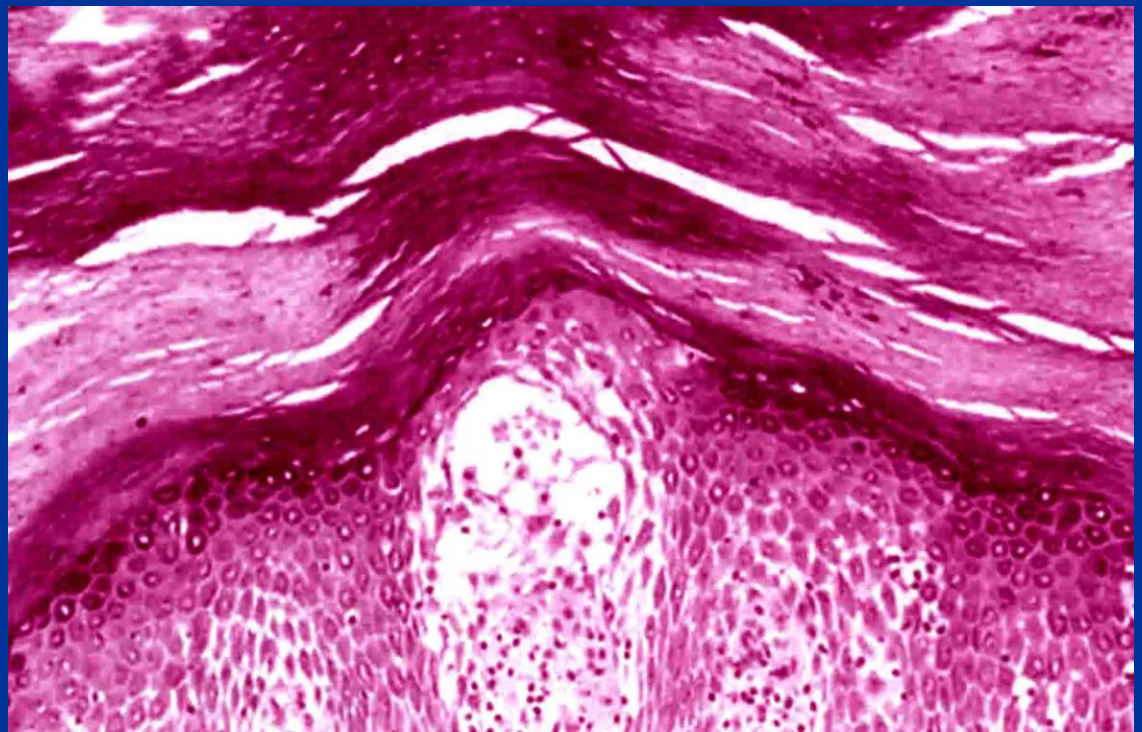


Histopathology

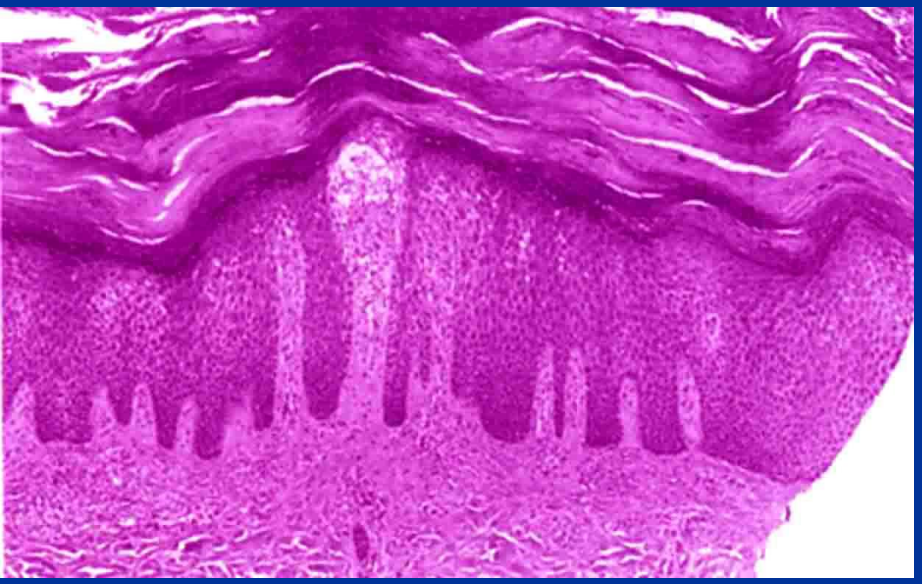
- **Spongiosis**
- **Vesicles**
- **Lymphocytes**
- **DDx allergic contact dermatitis: VERY DIFFICULT (absence of eosinophils)**
- **DDx acral psoriasis: CAN BE VIRTUALLY IMPOSSIBLE**

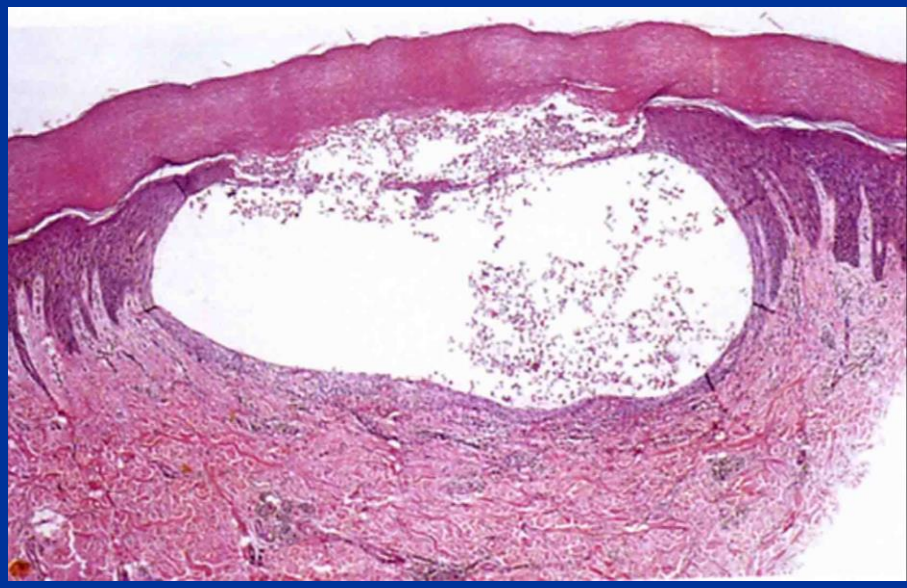
Acral psoriasis

- **Epidermal hyperplasia more diffiult to evaluate**
- **(Striking) spongiosis**



Dyshidrotic eczema vs acral psoriasis





Two ‘entities’ which are hardly differentiated histopathologically might be the same

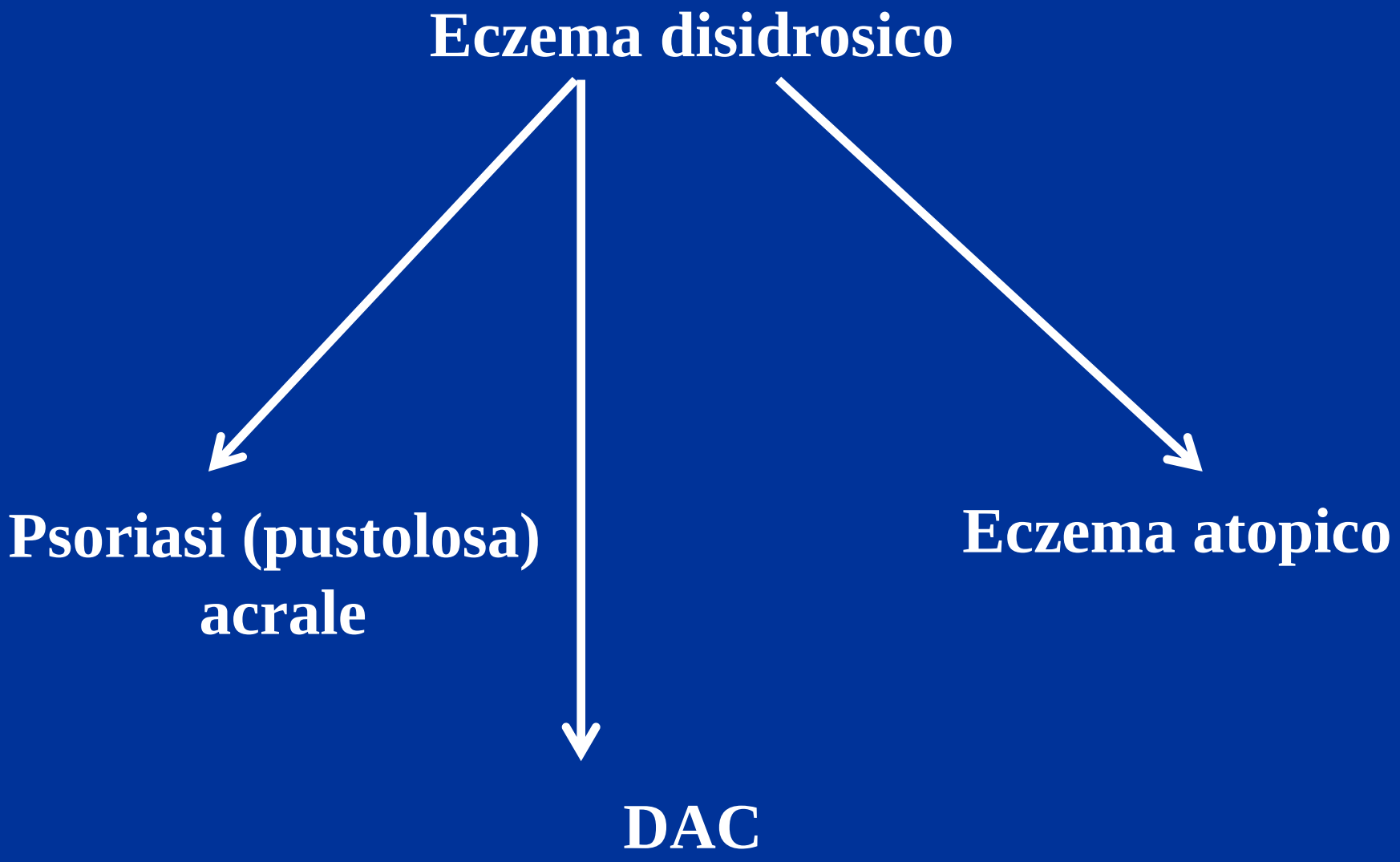

Eczema disidrosico
Psoriasi (pustolosa) acrale
DAC
Eczema atopico

Table 5.5 Variants of psoriasis with spongiosis
Early psoriasis/guttate psoriasis
Psoriasis of palms and soles
Erythrodermic psoriasis
Flexural psoriasis
Established psoriasis (rare, may have subtle histopathological features of glucagonoma syndrome as well)
Spongiotic psoriasis

Psoriasis May Have Classic and Non-Classic Histopathologic Features

Journal of Cutaneous Pathology

1 Expert Comment

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TAKE-HOME MESSAGE

- Classic and non-classic histologic features of psoriasis were evaluated in this study of 51 biopsies from 46 patients with clinically confirmed psoriasis. Classic histologic features were present in the majority of cases and included club-shaped rete ridges (96%), hypogranulosis (96%), Munro microabscess (78%), and spongiform pustule of Kogoj (53%). However, regular acanthosis was only present in 14% of cases, and irregular acanthosis was found in 84% of cases. Hypergranulosis was present in 65% of cases, usually in association with hypogranulosis. Other atypical findings included junctional vacuolar alteration (76%), spongiosis (76%), dermal neutrophils (69%), necrotic keratinocytes (67%), and dermal eosinophils (49%).
- In this study there was a broad histologic spectrum of psoriasis, including both classic and non-classic features.

Paradoxical autoimmunity

- Paradoxical onset of autoimmune disease in the course of anti TNFalpha therapy for autommune disease (Crohn, RA)
- 2-5% of pts
- Usually resolve after discontinuation
- In 0.5-1% of pts: lupus-like syndrome

Tumor necrosis factor-α inhibitor-induced psoriasis: Systematic review of clinical features, histopathological findings, and management experience

Gabrielle Brown, MD, MS,[a] Eva Wang, MD,[a] Argentina Leon, MD,[a] Monica Huynh, DO,[a]
Mackenzie Wehner, MD,[a] Rebecca Matro, MD,[b] Eleni Linos, MD, PhD,[a]
Wilson Liao, MD,[a] and Anna Haemel, MD[a]
San Francisco, California

Background: Tumor necrosis factor-α (TNF-α) inhibitors have been reported to induce new-onset psoriasis.

Objective: To better define the demographic, clinical features, and treatment approach of TNF-α inhibitor-induced psoriasis.

Methods: Systematic review of published cases of TNF-α inhibitor-induced psoriasis.

Results: We identified 88 articles with 216 cases of new-onset TNF-α inhibitor-induced psoriasis. The mean age at psoriasis onset was 38.5 years. The most common underlying diseases were Crohn disease (40.7%) and rheumatoid arthritis (37.0%). Patients underwent TNF-α therapy for an average of 14.0 months before psoriasis onset with 69.9% of patients experiencing onset within the first year. The majority of patients received skin-directed therapy, though patients who discontinued TNF therapy had the greatest resolution of symptoms (47.7%) compared with those who switched to a different TNF agent (36.7%) or continued therapy (32.9%).

Limitations: Retrospective review that relies on case reports and series.

Conclusion: While TNF-α inhibitor cessation may result in resolution of induced psoriasis, lesions may persist. Decisions regarding treatment should be weighed against the treatability of TNF-α inhibitor-induced psoriasis, the severity of the background rheumatologic or gastrointestinal disease, and possible loss of efficacy with cessation followed by retreatment. Skin-directed therapy is a reasonable initial strategy except in severe cases. (J Am Acad Dermatol http://dx.doi.org/10.1016/j.jaad.2016.08.012.)

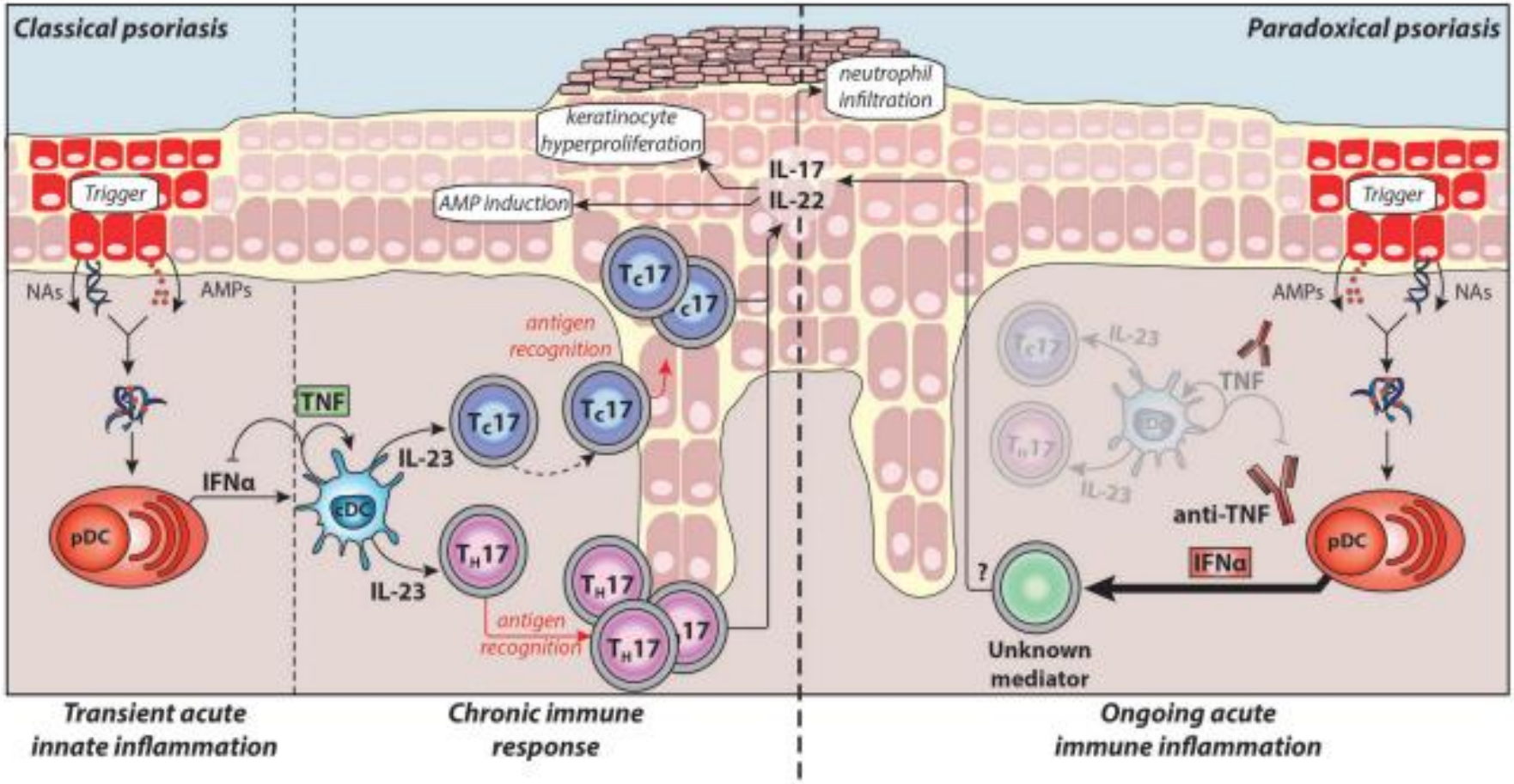


FIGURE 1 | Pathogenesis of classical plaque psoriasis and paradoxical psoriasis. Antimicrobial peptides (AMPs), which are produced by keratinocytes upon skin injury or released by neutrophils, form complexes with nucleic acids (NAs) released by dying cells. These complexes activate plasmacytoid dendritic cells (pDC) to produce large amounts IFNα during the acute/early phase of psoriasis pathogenesis. IFNα activates conventional dendritic cells (cDCs), which in turn produce TNF and IL-23. TNF induces the maturation of cDCs and pDCs, which lose their ability to produce IFNα. Thus, in classical psoriasis, early IFNα production gets relayed by TNF that controls and limits the IFNα production by pDCs via a negative feedback loop (through induction of pDC maturation). Subsequently, IL-23 and other pro-inflammatory cytokines produced by cDCs drive the activation of potentially autoreactive T-cells, which proliferate and, particularly $CD8^+$ T_C cells, migrate into the epidermis. Upon antigen recognition they produce the T_H17 cytokines IL-17 and IL-22 that induce keratinocyte hyperproliferation, attract neutrophils to the skin, and upregulate AMP production providing a positive feedback loop eventually resulting in the psoriatic phenotype (chronic/late phase). Normally, during anti-TNF therapy, the absence of TNF and consequently of downstream cytokines suppresses pathogenic T-cells thereby alleviating classical psoriasis. However, in patients developing paradoxical psoriasis, TNF blockade inhibits pDC maturation and leads to sustained IFNα production. In addition, as cDC cannot mature in absence of TNF, paradoxical psoriasis fails to elicit a T cell mediated autoimmune response. Thus, paradoxical psoriasis remains in an ongoing IFNα-driven acute immune inflammation independent of T-cells. The exact pathogenic downstream mechanism of IFNα-driven paradoxical psoriasis skin lesions remains to be fully elucidated.

Abbreviations used:

AS:	ankylosing spondylitis
AZA:	azathioprine
CD:	Crohn disease
IFN-α:	interferon α
IL:	interleukin
IL-23R:	interleukin-23 receptor
MeSH:	medical subject headings
MTX:	methotrexate
RA:	rheumatoid arthritis
T_H:	T helper
TNF-α:	tumor necrosis factor-α
UC:	ulcerative colitis

The psoriasis presentations were variable, and 26.9% of patients had more than one reported morphological type. The two most commonly observed clinical presentations included plaque (44.8%) and palmoplantar pustular (36.3%) psoriasis. Other presentations included ill-defined psoriasiform dermatitis (19.9%), severe scalp involvement associated with alopecia (7.5%), and generalized pustular psoriasis (10.9%). The anatomical sites commonly involved included soles (45.8%), extremities (45.4%), palms (44.9%), scalp (36.1%), and trunk (32.4%).

Of the 102 cases confirmed with biopsy, 54.9% revealed plaque psoriasis and 33.3% were consistent with pustular psoriasis with the remainder interpreted as psoriasiform dermatitis. An eosinophil-rich infiltrate was noted in 3 of the plaque psoriasis biopsies and 3 of the pustular psoriasis biopsies.

TNF-α inhibitor treatment characteristics

The majority of cases were associated with common concomitant immunomodulators at the time of psoriasis onset included methotrexate (MTX; 33.7%), azathioprine (AZA; 18.9%), and leflunomide (13.7%). Seventeen cases reported administration of concomitant systemic steroids.

Management and outcomes

Therapeutic management and outcomes of TNF-α inhibitor-induced psoriasis are summarized in Table II. Resolution of psoriasis (or no evidence of recurrence at time of follow-up) was reported in patients who discontinued TNF therapy (47.7%), switched to a different TNF agent (36.7%), and continued the same TNF therapy (32.9%). Improvement but incomplete resolution of psoriasis was reported in patients who continued the same TNF therapy (57.3%), discontinued TNF therapy (46.2%), and switched to a different TNF agent (18.4%). Regardless of the TNF-α inhibitor treatment decision, the majority of patients received skin-directed therapy with one or more agents including topical steroids (76.5%), vitamin D analogues (17.6%), MTX (17.2%), phototherapy (8.3%), cyclosporine (5.4%), acitretin (2.0%), and coal tar (1.0%).

Management strategies and resulting outcomes were analyzed for patients with more severe presentations, including alopecia and/or generalized pustular psoriasis (Table III). Half of the patients with alopecia had resolution of symptoms regardless of continuing or discontinuing TNF therapy (resolution in 2/4 who continued vs 4/8 who discontinued therapy). Patients with generalized pustular psoriasis who continued therapy experienced resolution in 33.3% of cases (2/6). Of patients with generalized pustular psoriasis who discontinued therapy, 63.6%

J Cutan Pathol. 2021 Jun;48(6):813-816. doi: 10.1111/cup.13919. Epub 2020 Dec 2.

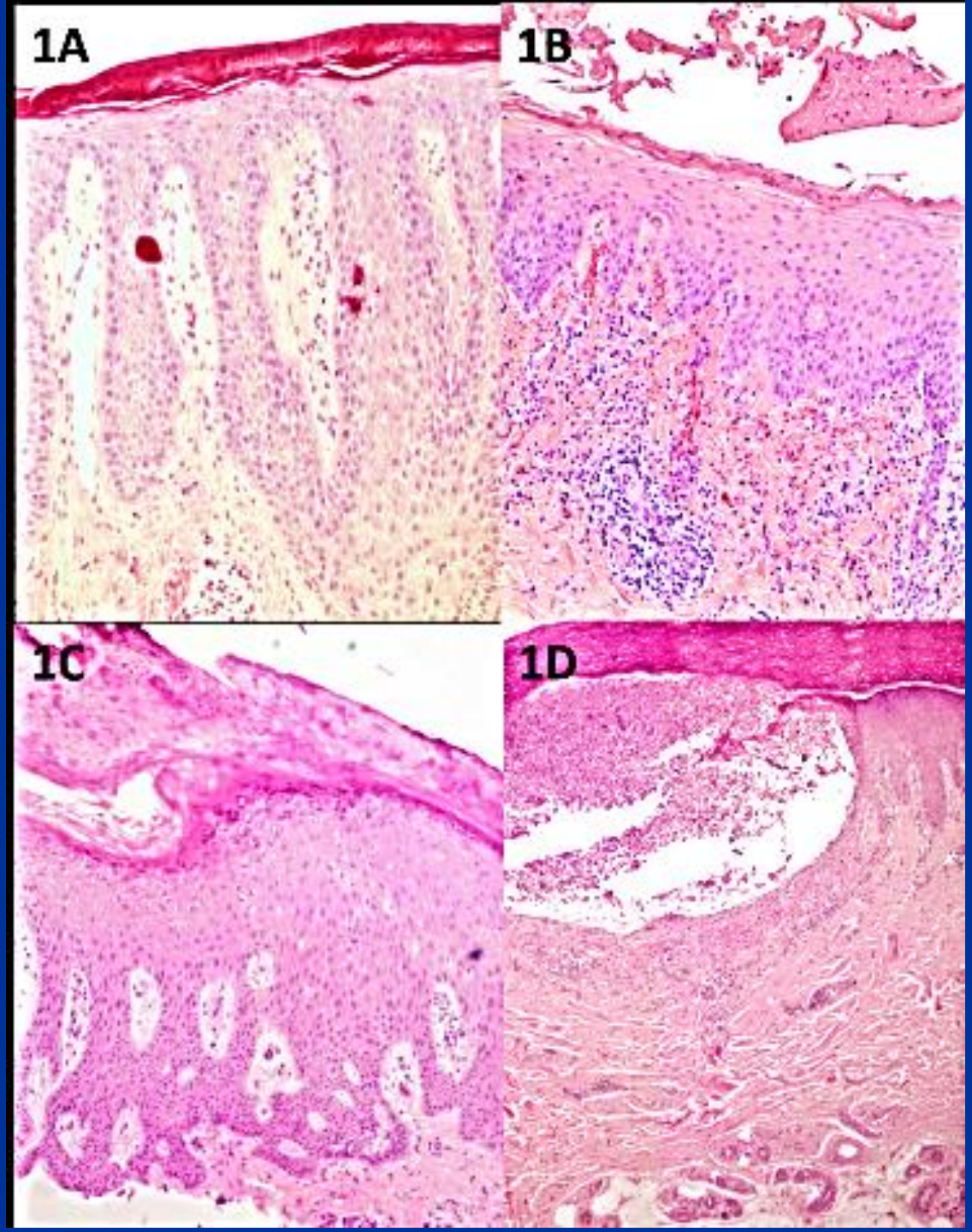
Histopathologic study of paradoxical psoriasis induced by antitumor necrosis factor alpha therapy: Is it true psoriasis?

Raquel Navarro [1], Karen Villar-Zarra [2], África Juarez [1], Esteban Daudén [1]

		Induced plaque psoriasis (%)	Plaque psoriasis (controls) (%)	P value[1]	Induced palmoplantar pustulosis (%)	Palmoplantar pustulosis (controls) (%)	P value[1]
Number of patients		6	8		7	13	
Number of biopsies		8	8		10	13	
	Complete	7 (87.5)	6 (75)	0.522	8 (80)	13 (100)	0.241
	Incomplete	1 (12.5)	2 (25)		1 (10)	0 (0)	
Epidermal hyperplasia	Absent	0 (0)	0 (0)		1 (10)	0 (0)	
	Present	5 (62.5)	6 (75)	1	4 (40)	8 (61.5)	0.414
Spongiosis	Absent	3 (37.5)	2 (25)		6 (60)	5 (38.5)	
	Complete	5 (62.5)	6 (75)	0.352	5 (50)	5 (38.5)	0.265
	Incomplete	3 (37.5)	1 (12.5)		0 (0)	3 (23)	
Hypogranulosis	Absent	0 (0)	1 (12.5)		5 (50)	5 (38.5)	
	Focal	6 (75)	4 (50)	0.301	6 (60)	5 (38.5)	0.071
	Generalized	2 (25)	2 (25)		3 (30)	1 (7.7)	
Parakeratosis	Absent	0 (0)	2 (25)		1 (10)	7 (53.8)	
	Present	3 (37.5)	1 (12.5)	0.569	10 (100)	13 (100)	--[2]
Neutrophil aggregates	Absent	5 (62.5)	7 (87.5)		0 (0)	0 (0)	
	Perivascular	8 (100)	6 (75)	0.319	8 (80)	8 (61.5)	0.619
	Lichenoid	0 (0)	0 (0)		1 (10)	2 (15.4)	
	Perivascular + Lichenoid	0 (0)	1 (12.5)		1 (10)	3 (23.1)	
Inflammatory infiltrate	Absent	0 (0)	1 (12.5)		0 (0)	0 (0)	
	>2 cells /field	0 (0)	1 (12.5)	1	0 (0)	1 (7.7)	1
Eosinophils	Absent	8 (100)	7 (87.5)		10 (100)	12 (92.3)	
	Present	8 (100)	4 (50)	0.77	8 (80)	10 (76.9)	1
Vascular dilatation	Absent	0 (0)	4 (50)		2 (20)	3 (23.1)	
Erythrocyte	Present	0 (0)	0 (0)	--[2]	4 (40)	2 (15.4)	0.341
extravasation	Absent	8 (100)	8 (100)		6 (60)	11 (84.6)	

[1] p-value from chi-square test or Fisher´s exact test as appropriate.

[2] No statistical hypothesis test is calculated because the value of that variable is a constant.


1A
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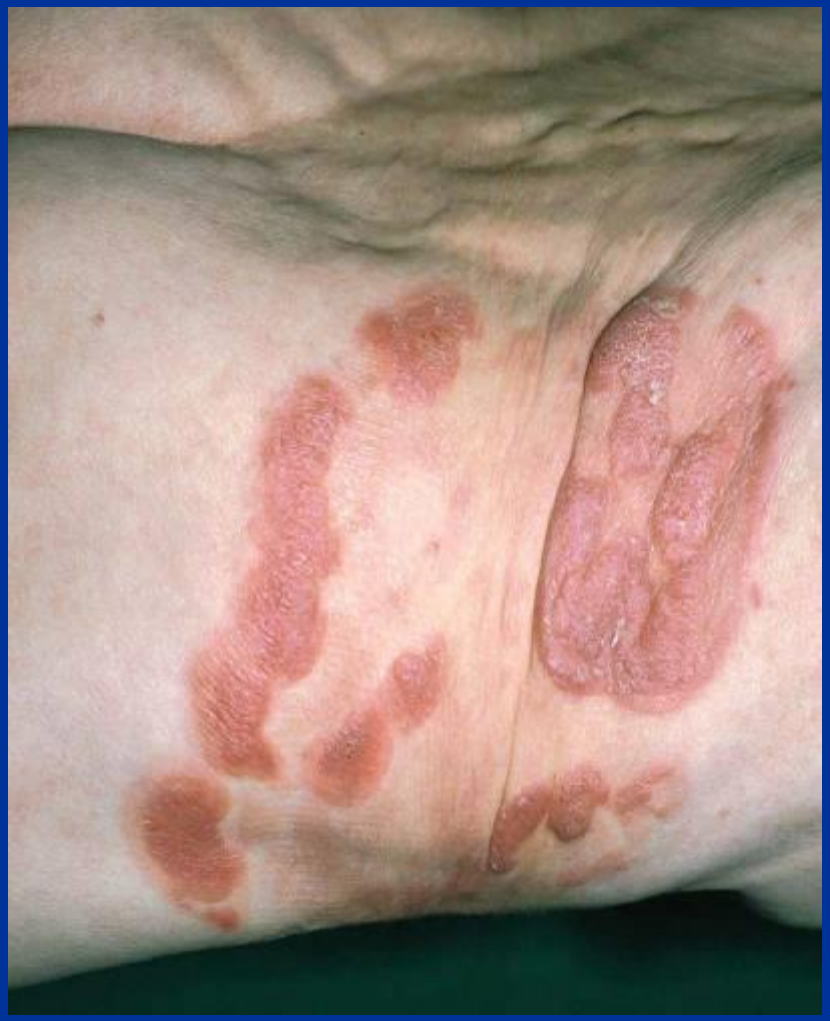
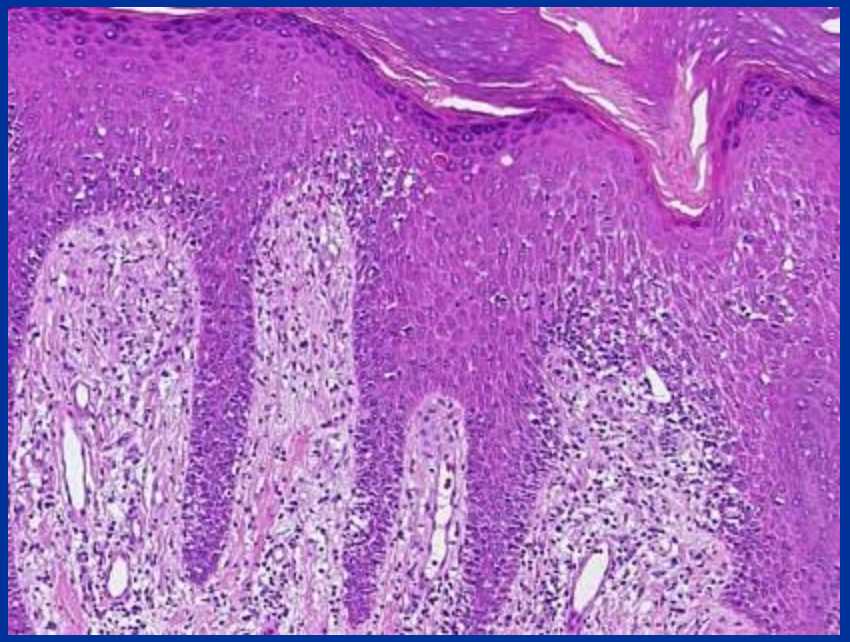
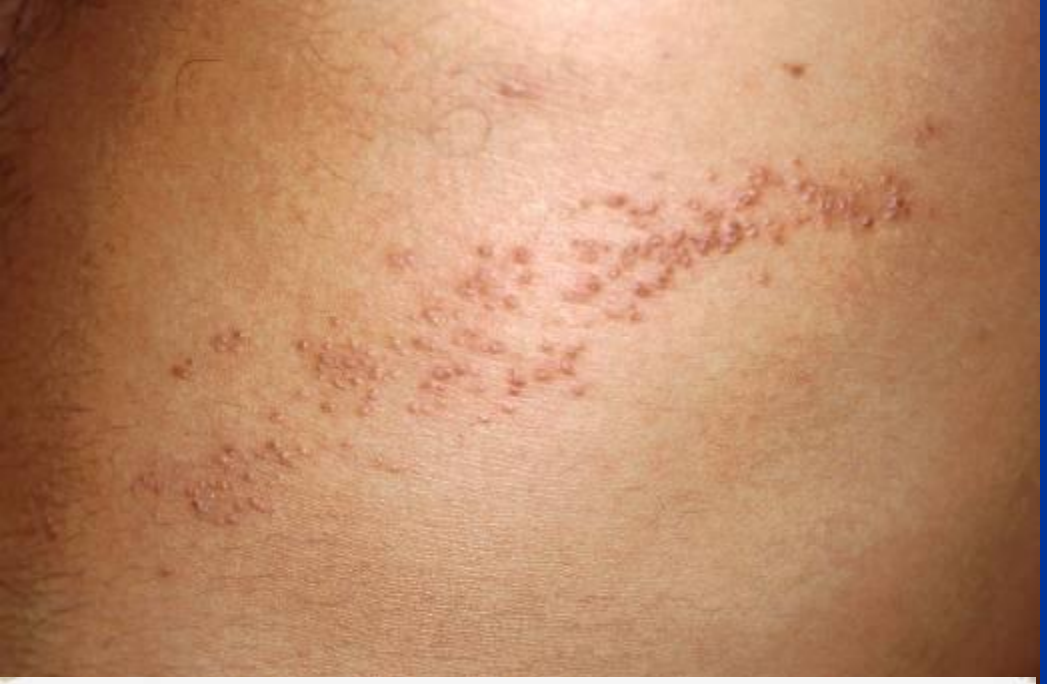
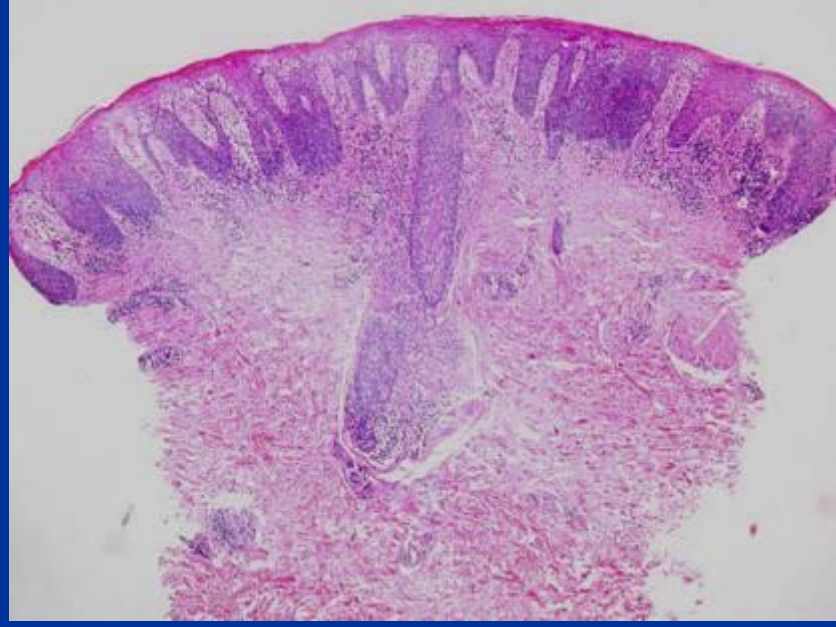
Some differentials

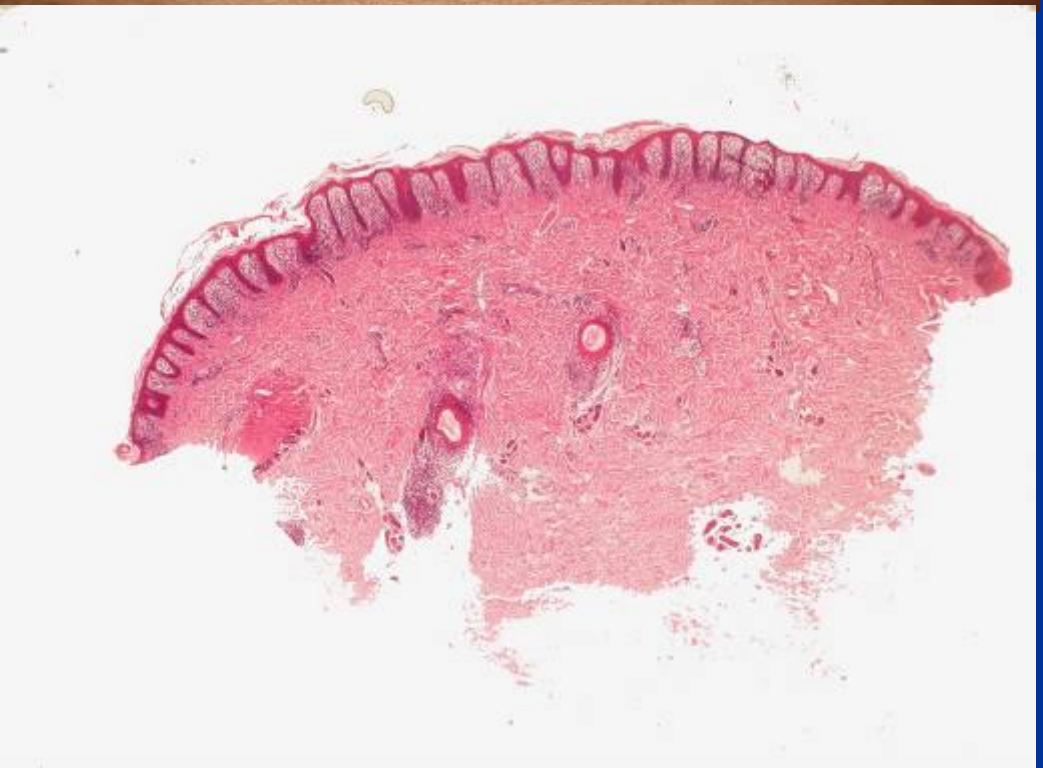


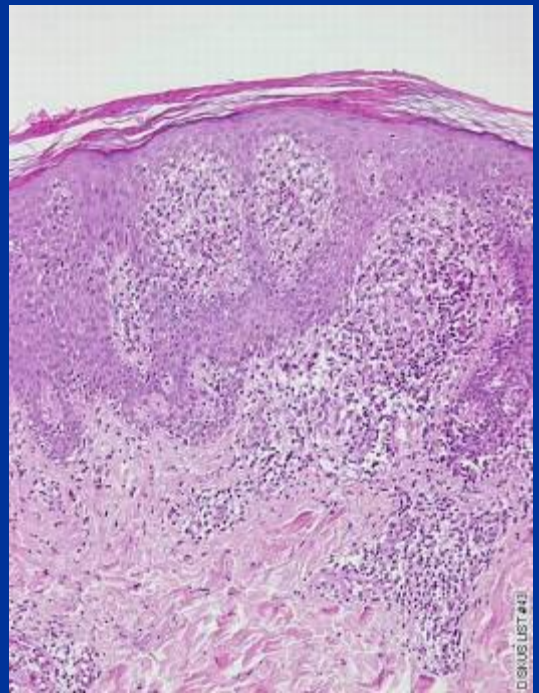

DISKUS spon-psor-bend #44

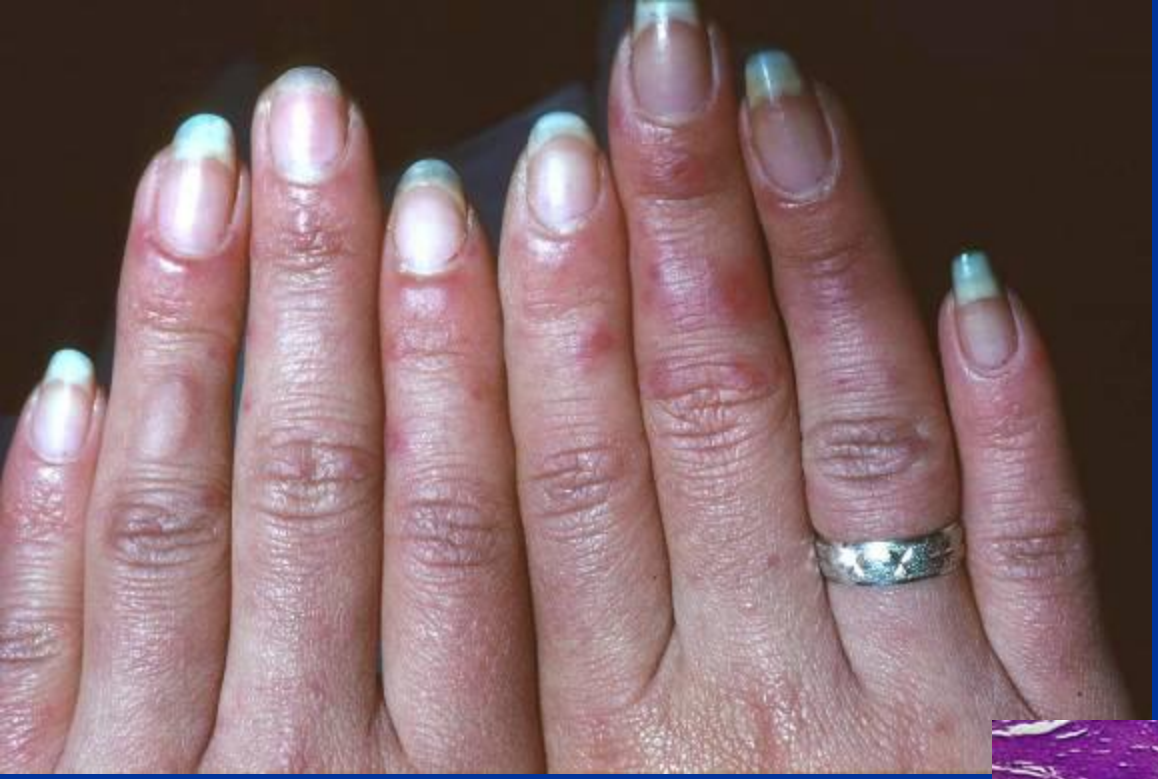


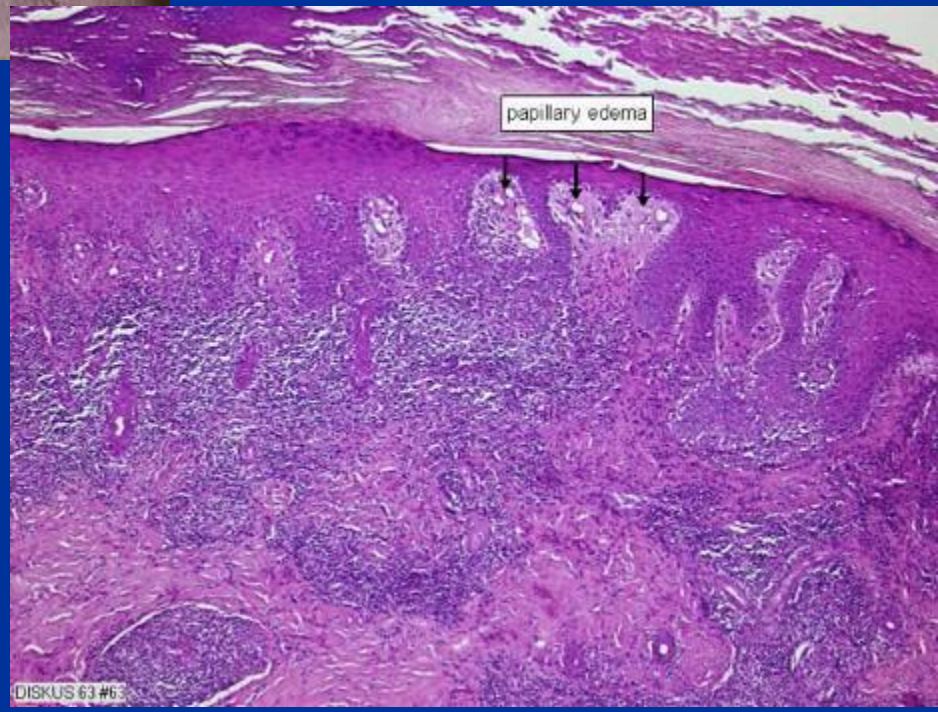











papillary edema
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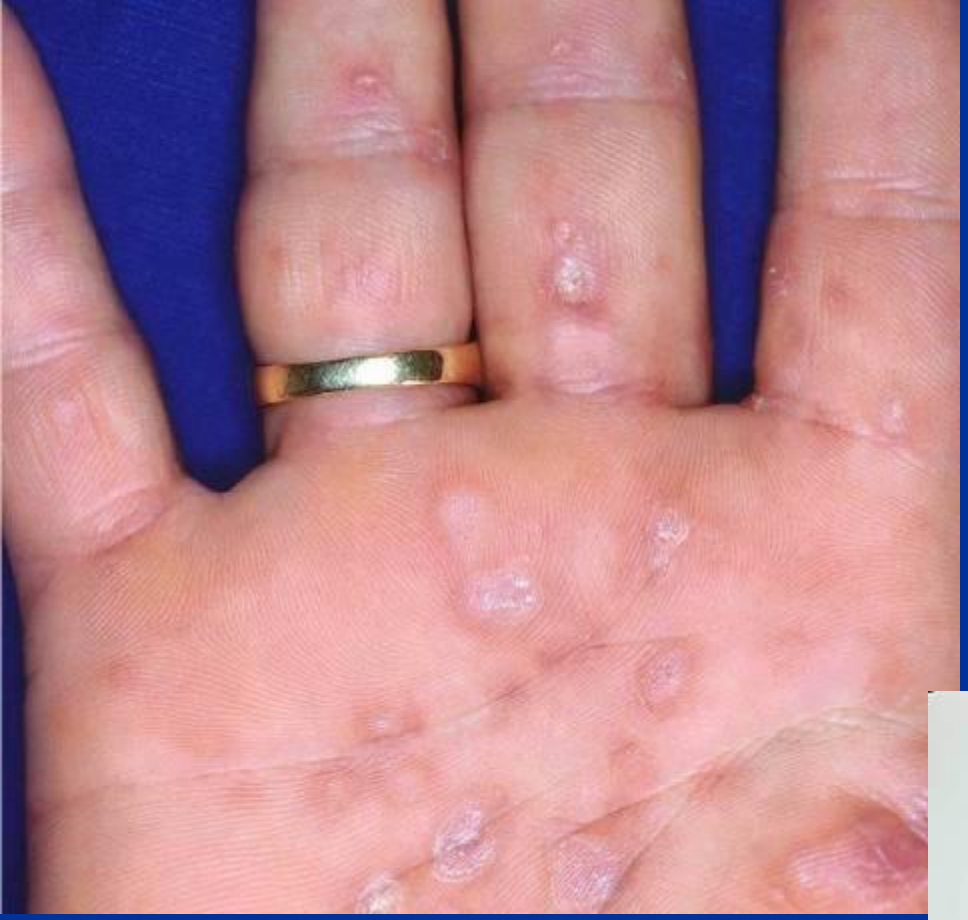

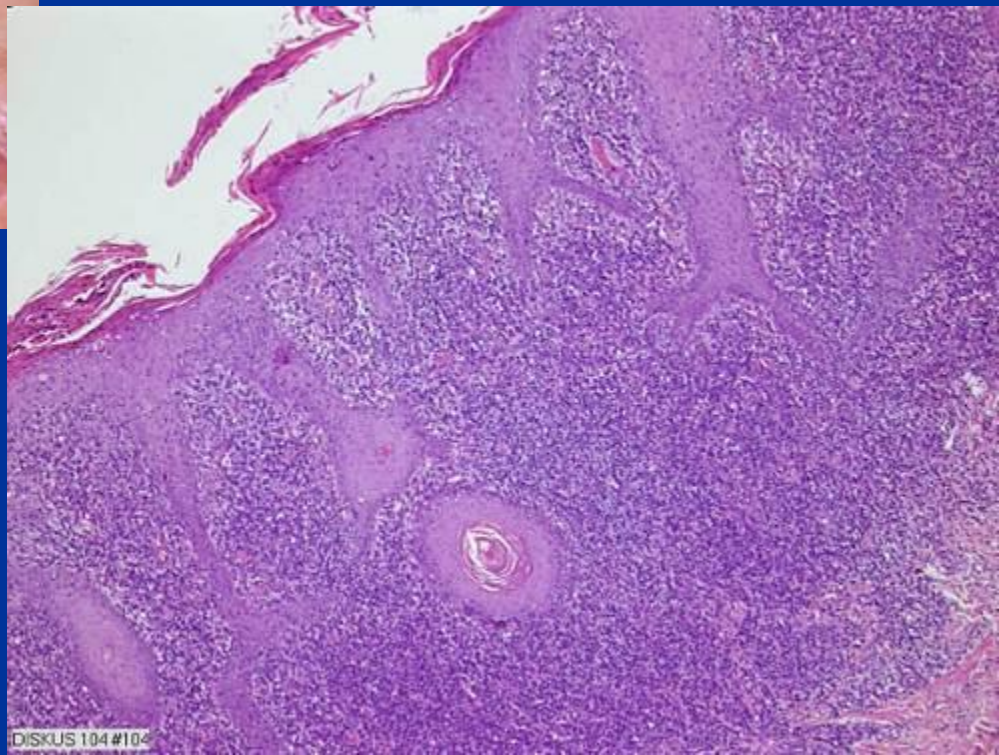
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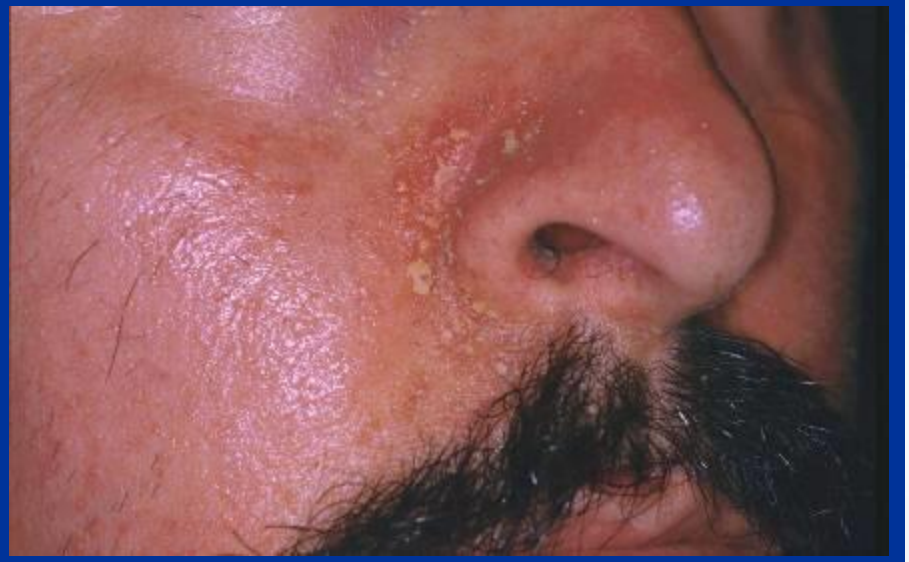
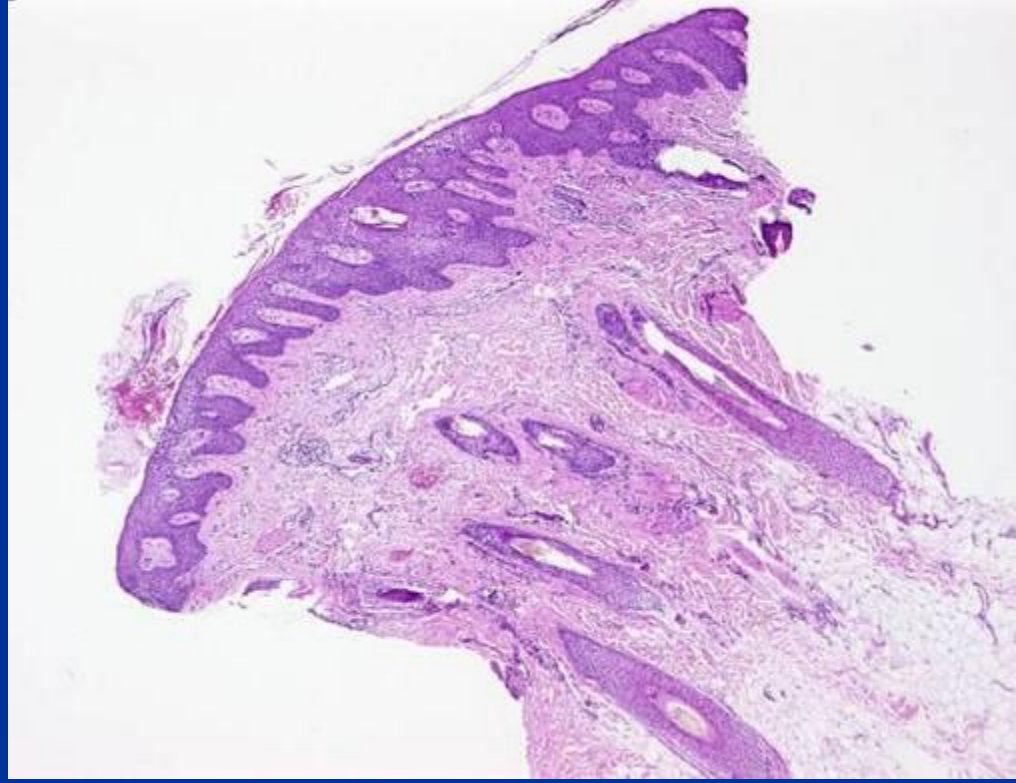
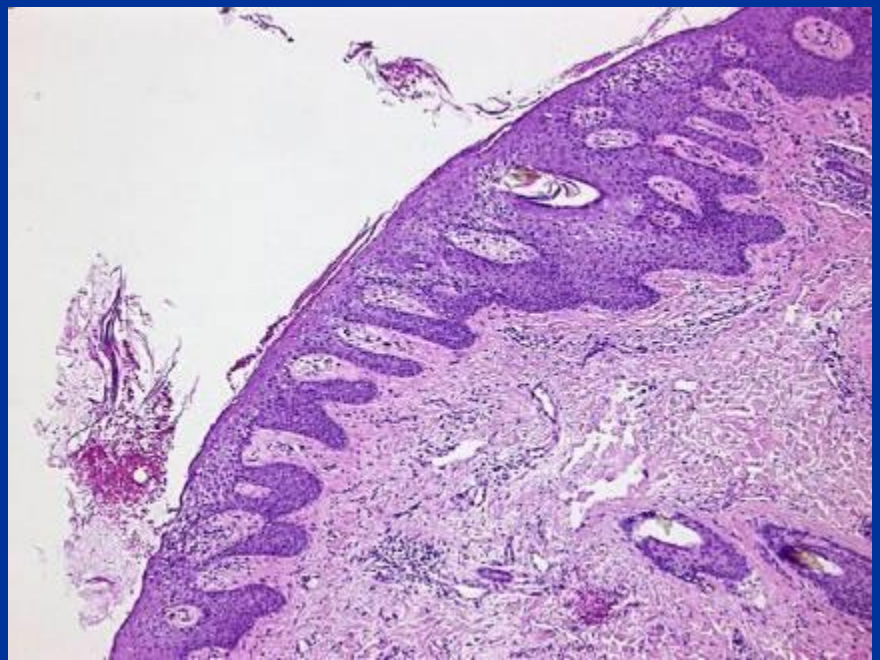
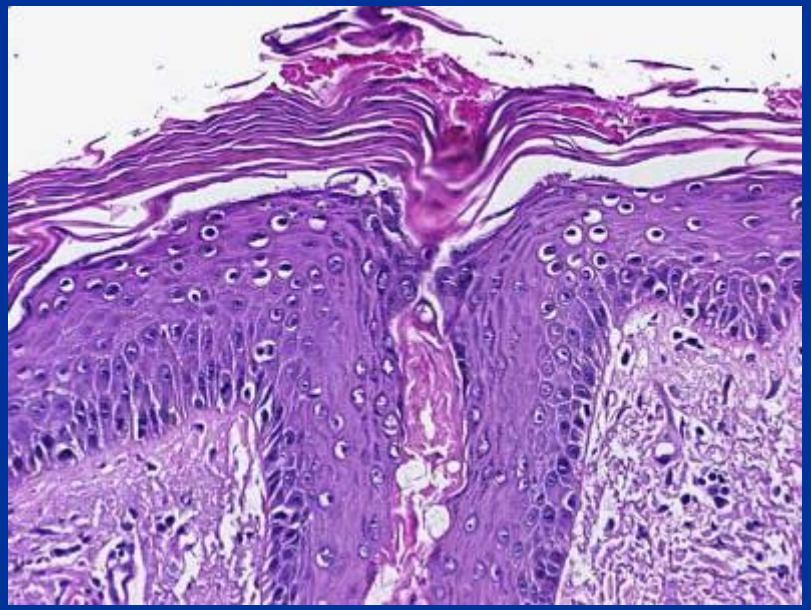
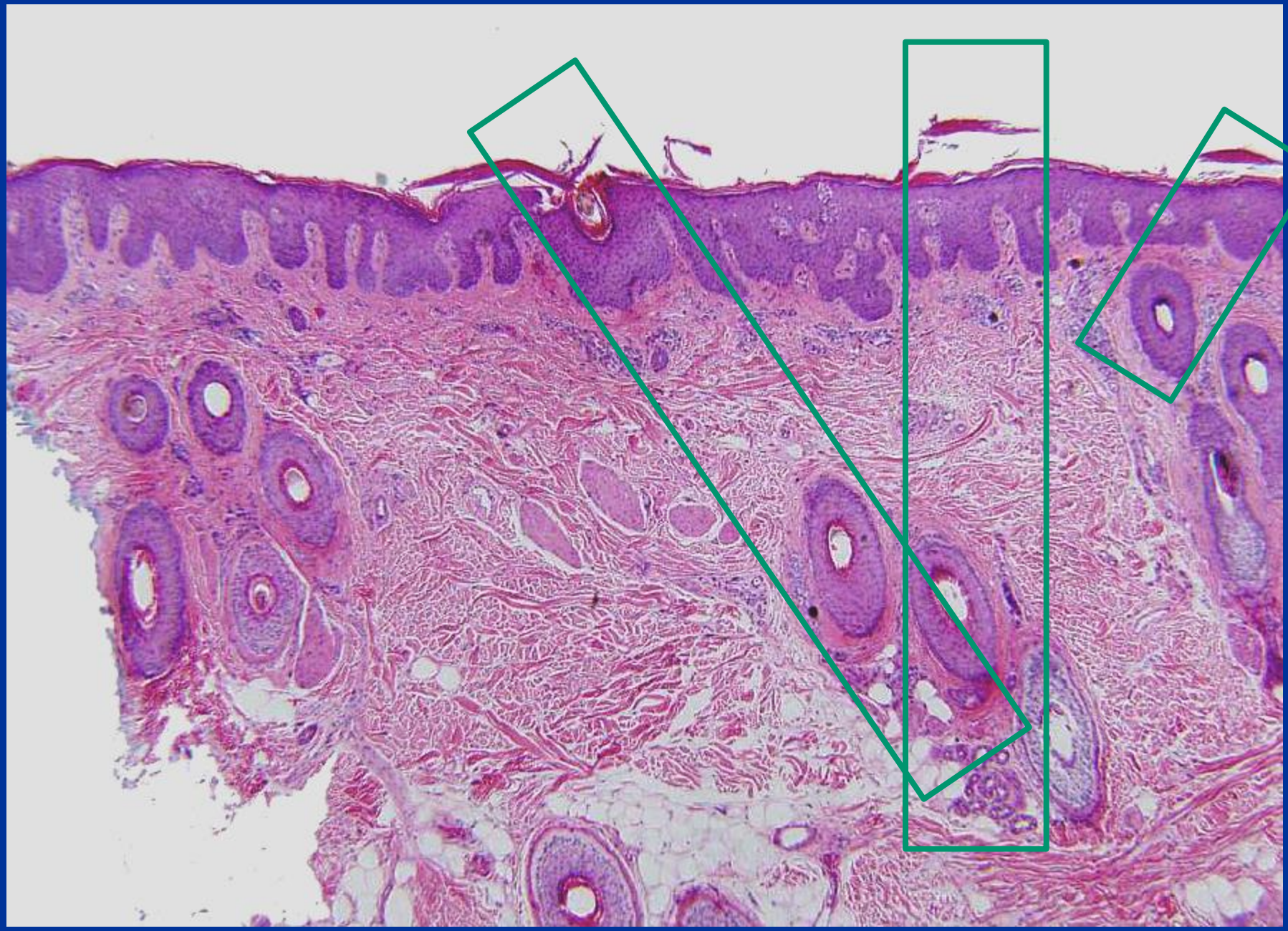
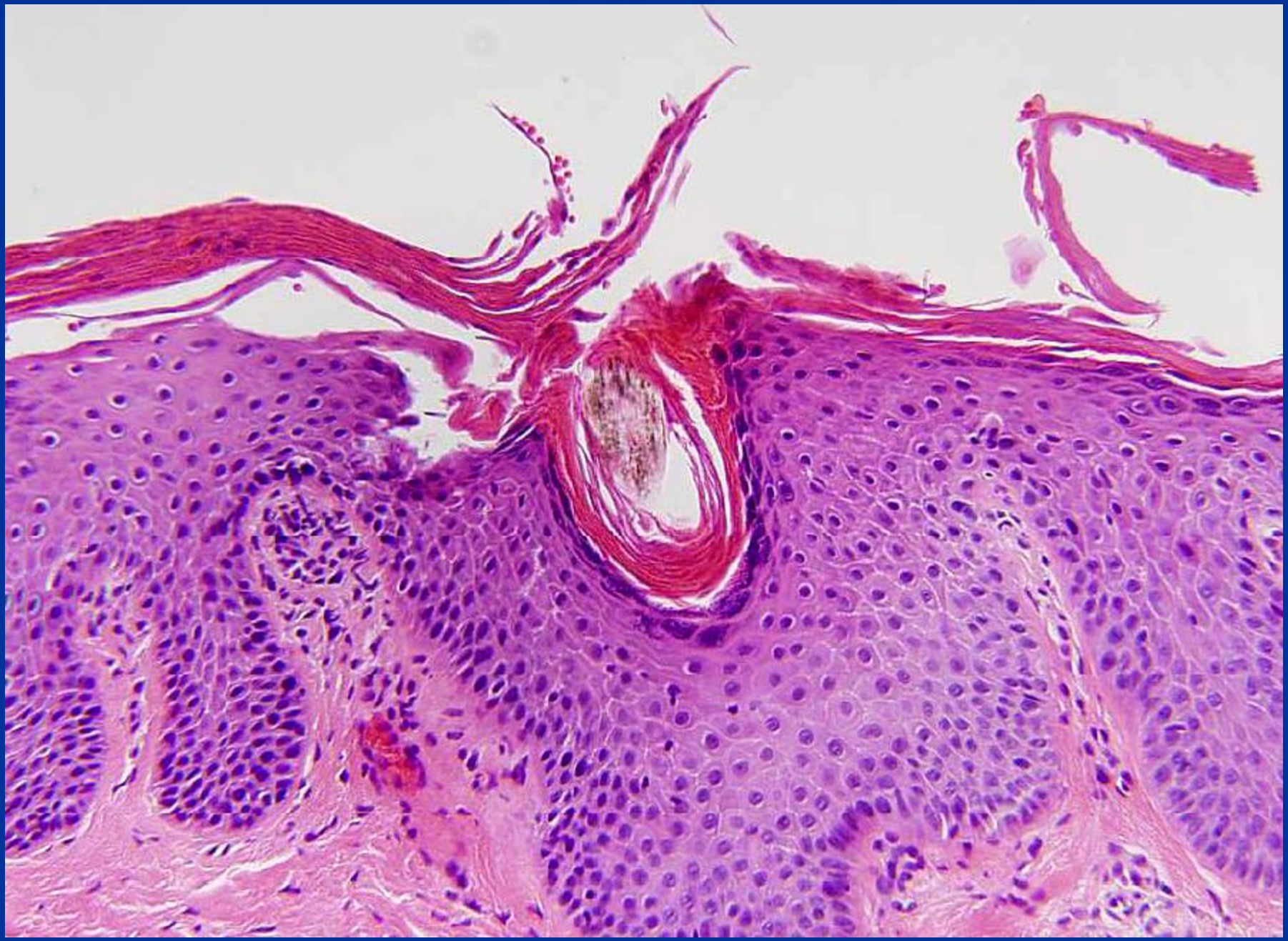


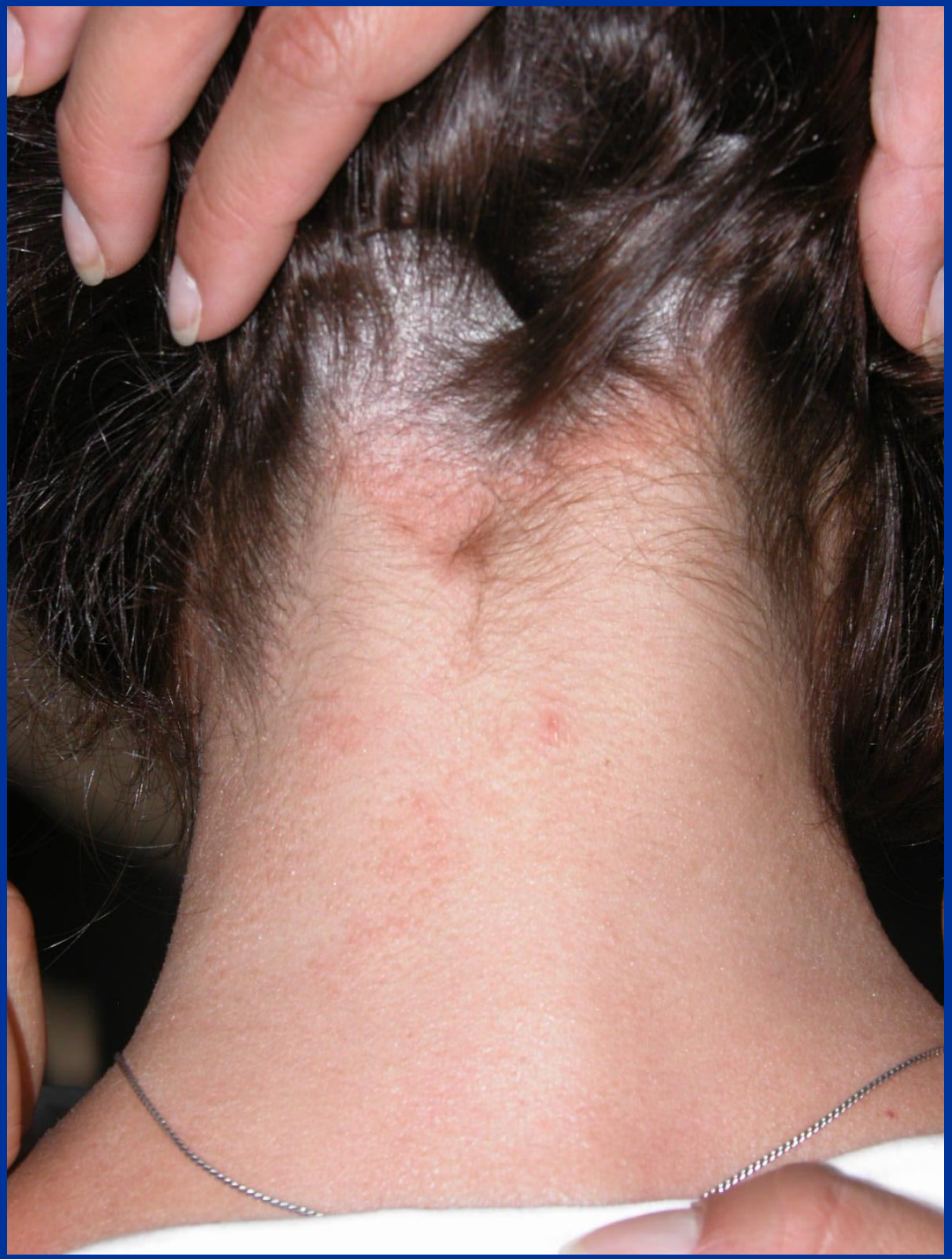


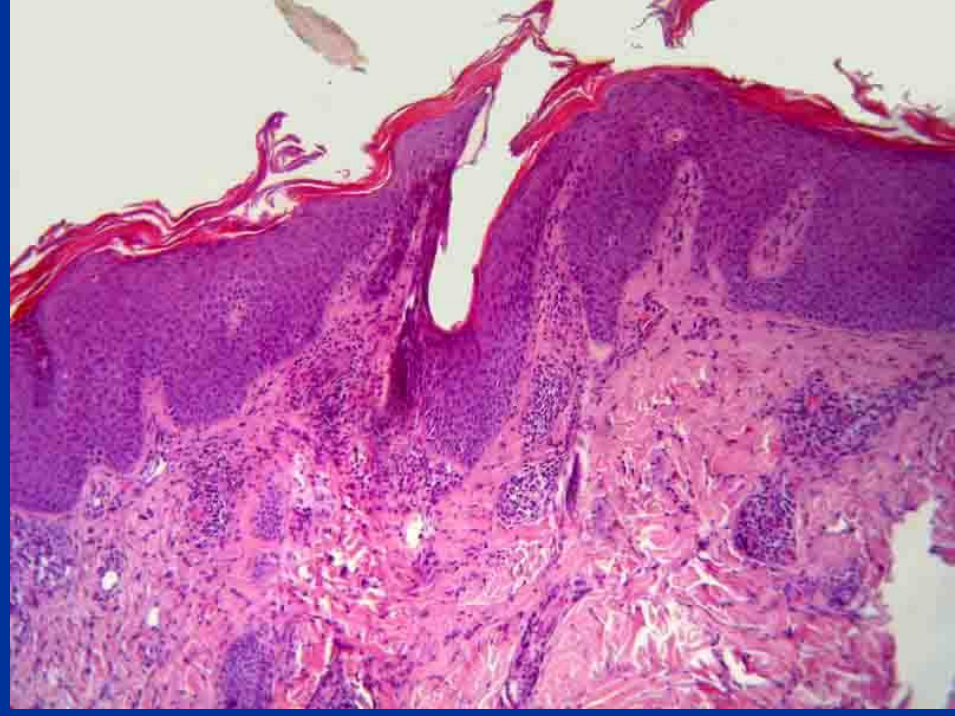
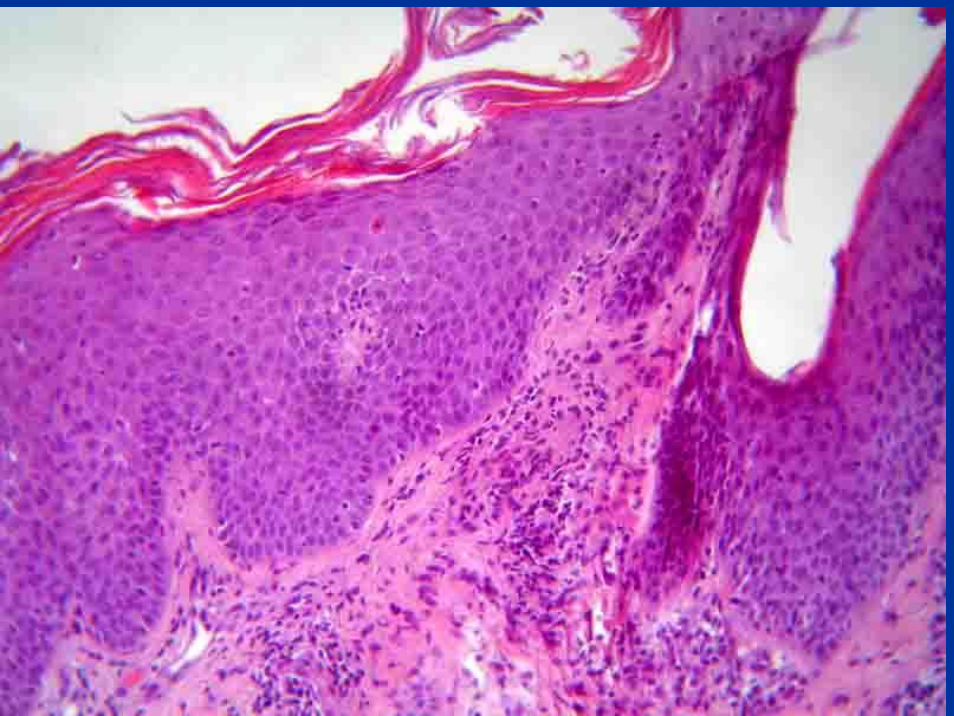
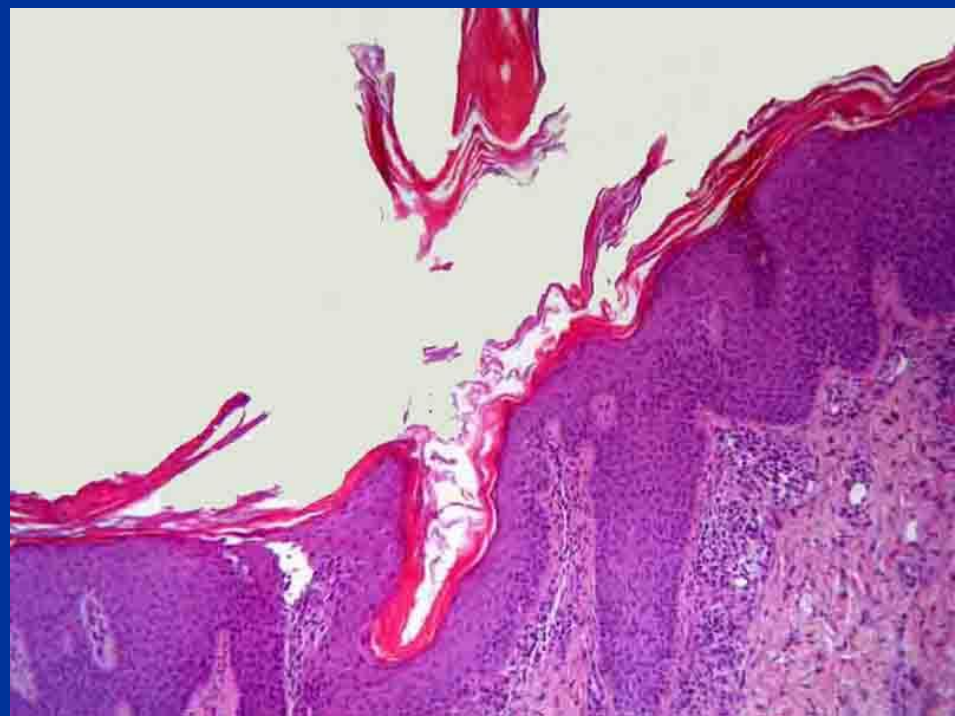


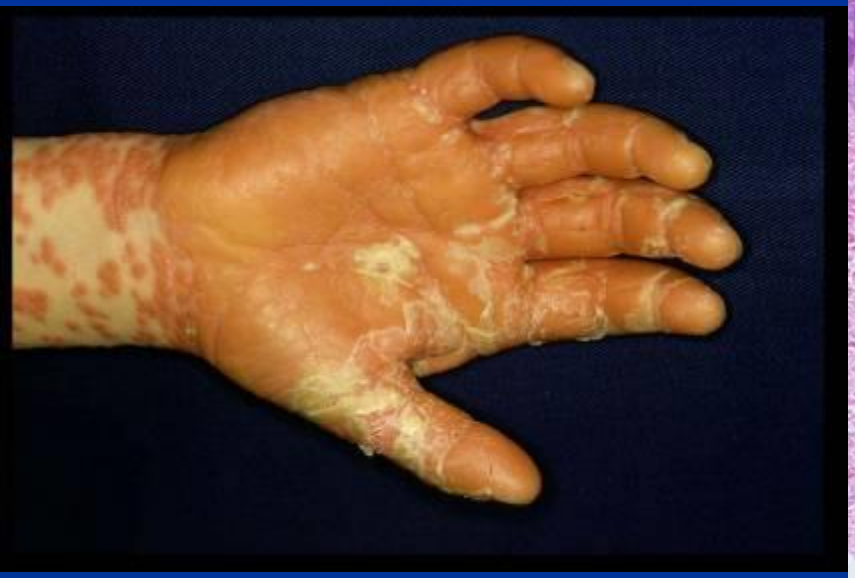


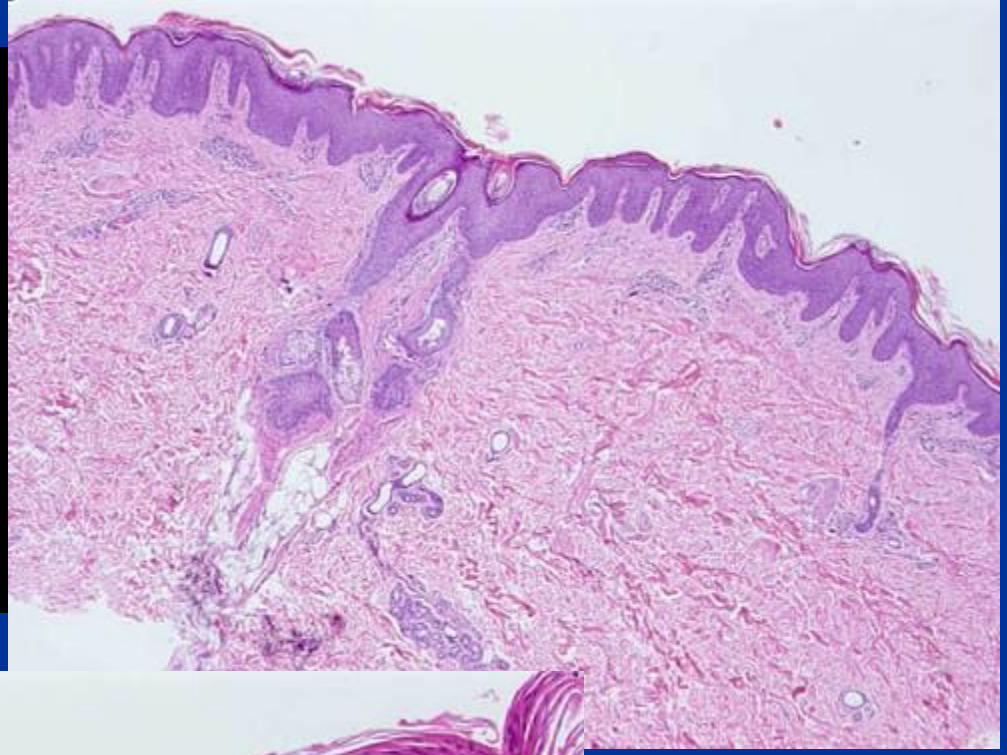


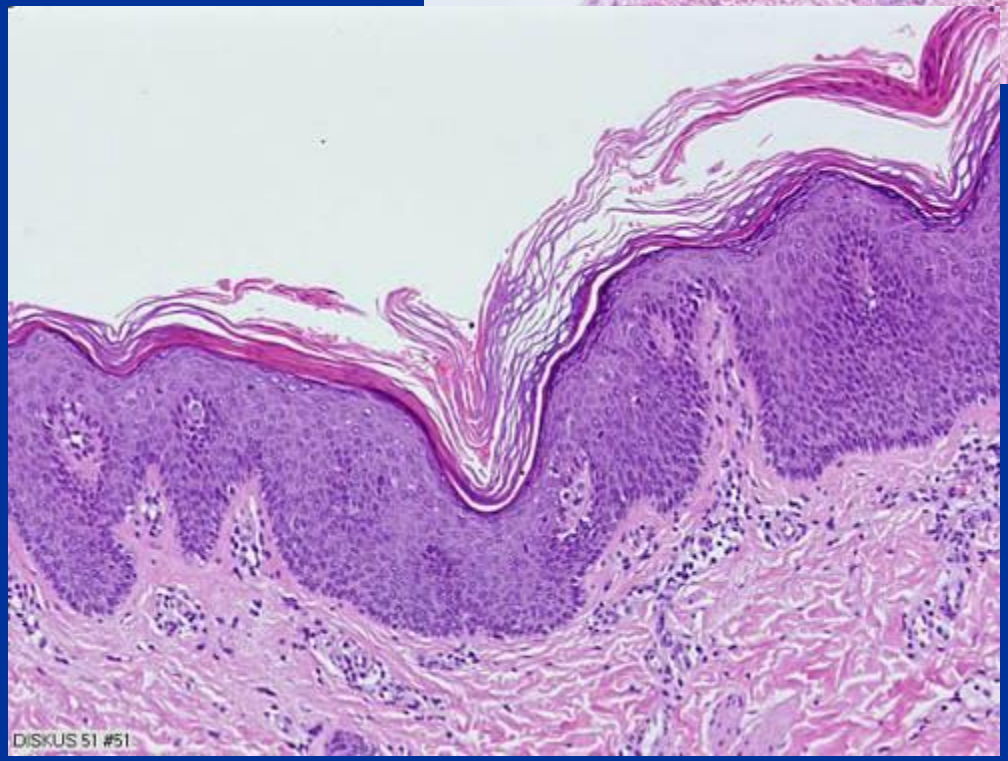




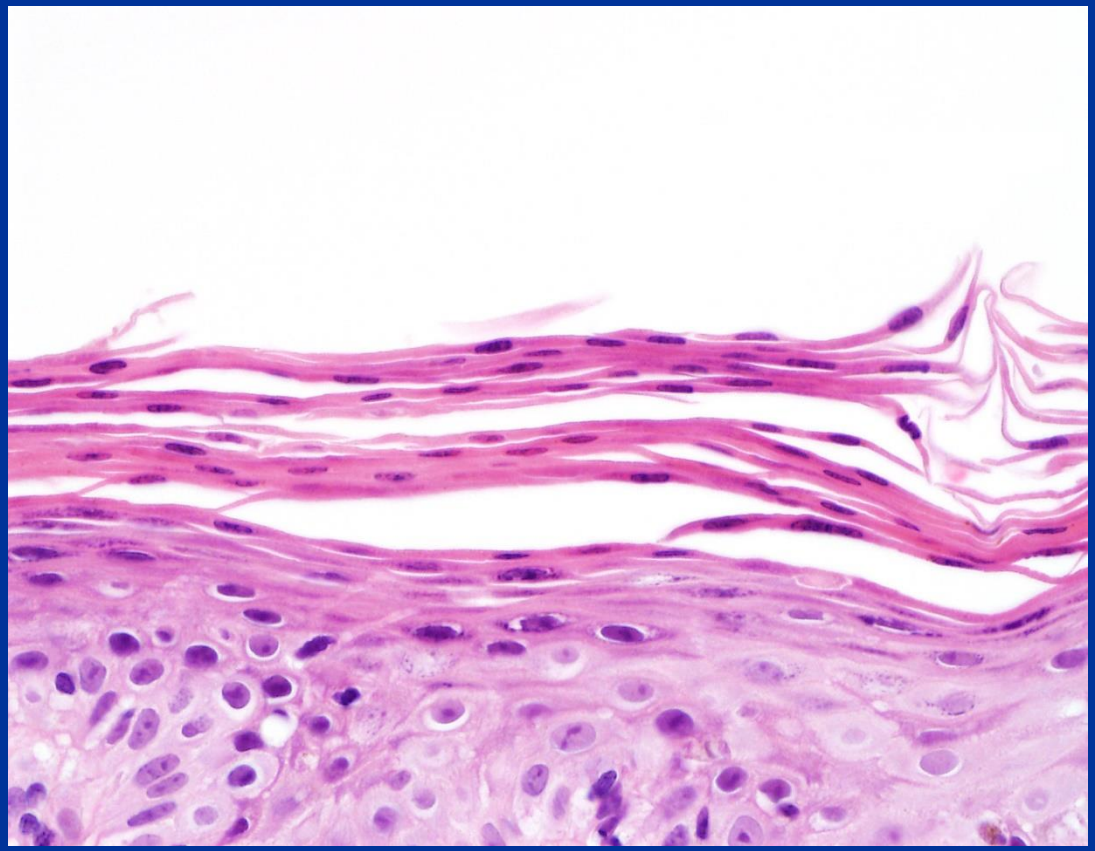





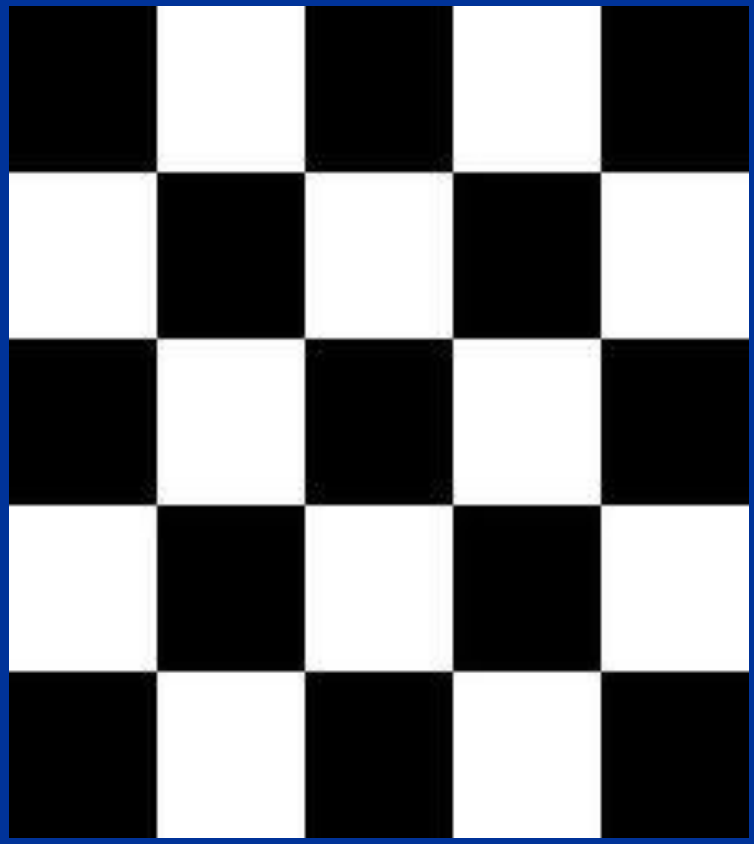


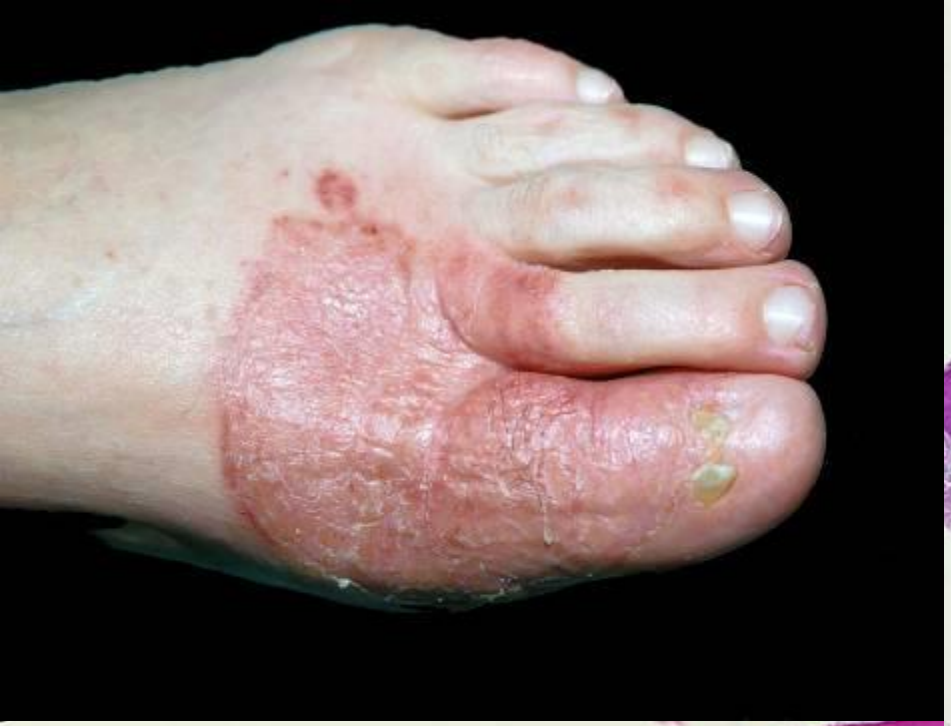


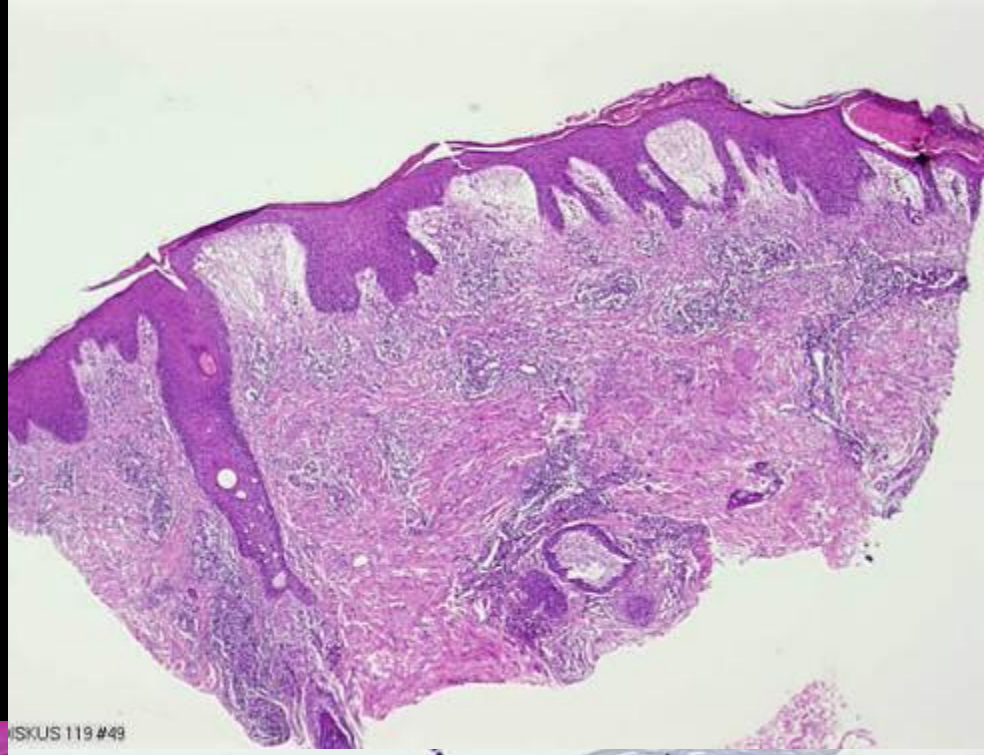

DISKUS 51 #51

‘CHECKERBOARD’ PATTERN





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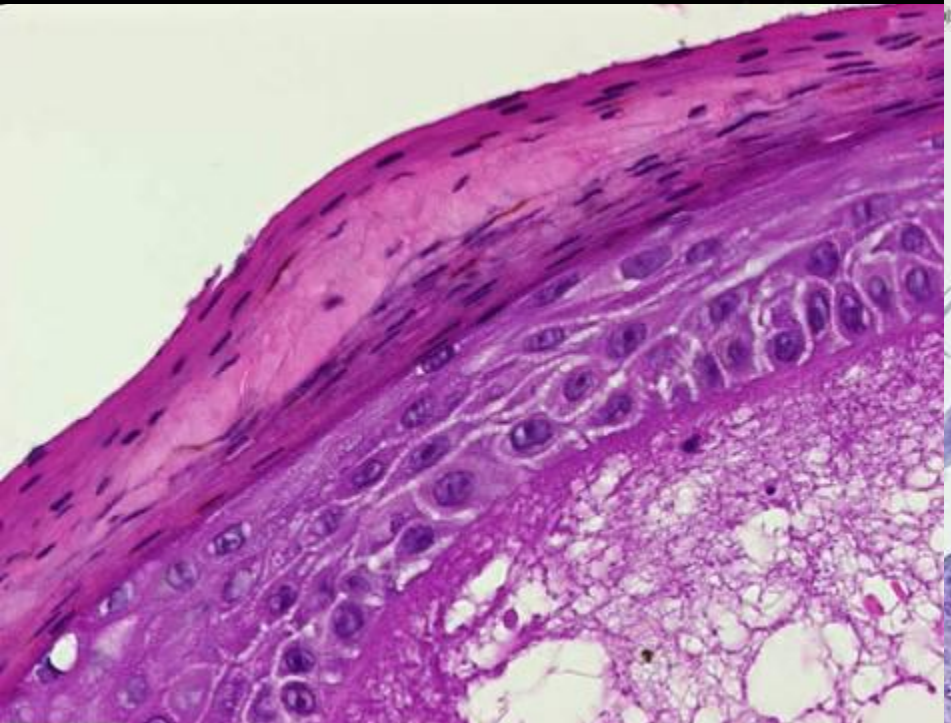

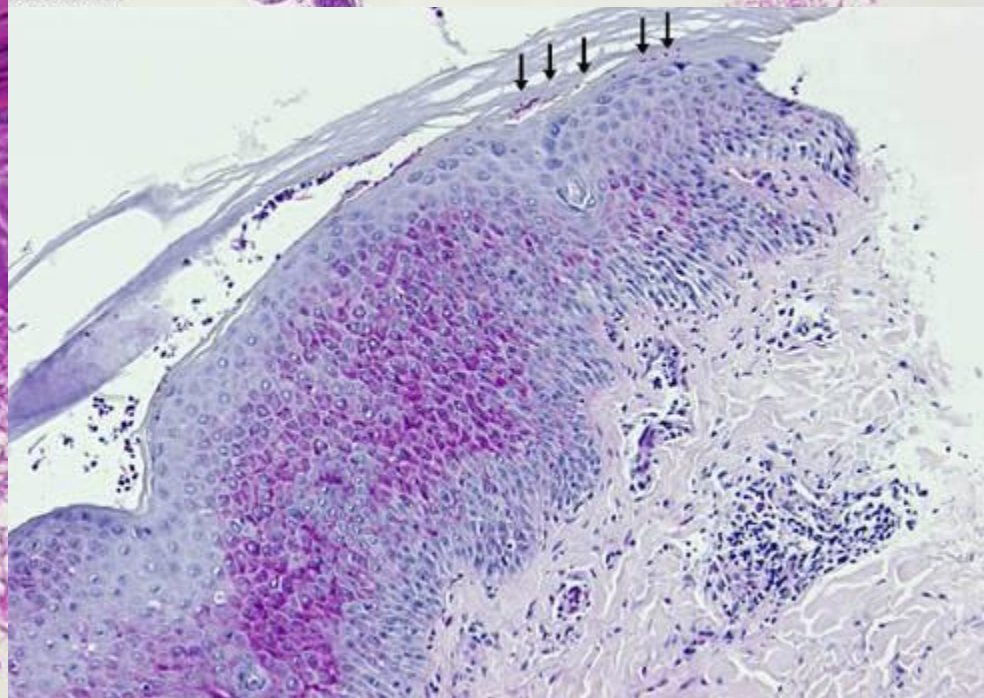

Conclusioni

- **La conoscenza dei patterns e dei sub-patterns delle dermatosi infiammatorie è crciale per un corretto inquadramento istopatologico**
- **Tuttavia, neanche la massima expertise istopatologica può sostituirsi al vero ‘gold standard’, che è la correlazione clinico-patologica**

2022
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