

Histopathologic spectrum of psoriasis

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S U M M A R Y

The many clinical faces of psoriasis are reflected by the spectrum of histopathologic changes ranging from guttate psoriasis to generalized pustular psoriasis. Psoriasis is a dynamic process and consequently the morphologic changes vary during the evolution and subsequent resolution of the individual lesions. For example, a fully developed psoriatic lesion reveals psoriasiform acanthosis of the epidermis characterized by elongated thin or club shaped rete ridges of equal length alternating with thin dermal papillae that are covered by thin suprapapillary plates. Typically, there is confluent parakeratosis, which contains accumulation of neutrophils. The granular layer is decreased or absent. In the upper part of the dermis there is a moderately dense perivascular and interstitial infiltrate of lymphocytes in association with dilated spiraled capillaries in the dermal papillae and marked edema of the papillary dermis.

Besides early, fully developed and late lesions of "classical" psoriasis, palmoplantar psoriasis, pustular psoriasis and erythrodermic psoriasis are all characterized by distinctive morphologic changes that, in many instances, allow a histopathologic diagnosis with certainty.

The most important differential diagnoses from a histopathologic point of view are as follows: psoriasiform trichophytia, pityriasis rubra pilaris, pityriasis rosea, nummular dermatitis, chronic contact dermatitis, lichen simplex chronicus and rupial syphilis.

In conclusion, the histopathologic findings of the many faces of psoriasis are distinctive and allow a definitive diagnosis by the experienced dermatopathologist in nearly all instances. Until today no laboratory methods including modern molecular technologies are replacing conventional histopathology in the diagnosis of psoriasis.

Introduction

Psoriasis vulgaris is a relatively common skin condition that affects about 1% to 2% of the general population (1,2). A genetic predisposition has been

shown, but no clear pattern of inheritance has yet been demonstrated. Psoriasis vulgaris manifests itself in the second and third decades of life mainly as generalized erythematous plaques (plaque type). The disease may also show several other clinical variants mainly including

K E Y W O R D S

psoriasis,
clinical
features,
histo-
pathology



An early lesion of psoriasis: Fig. 1a) There is psoriasiform hyperplasia of the epidermis with focal parakeratosis containing neutrophils (haematoxylin-eosin 50x).

eruptive guttate, pustular, erythrodermic and geographic types (1-4).

Because of this wide spectrum of clinical features, the correct diagnosis may sometimes not be suspected clinically. Histopathology is useful in confirming the diagnosis and excluding other conditions that may mimic psoriasis. Histopathologic findings vary depend-

A well- developed lesion of plaque type psoriasis: Fig. 2a) There is prominent psoriasiform epidermal hyperplasia, hypogranulosis and confluent parakeratosis with collections of neutrophils in stratum corneum (haematoxylin-eosin 50x).

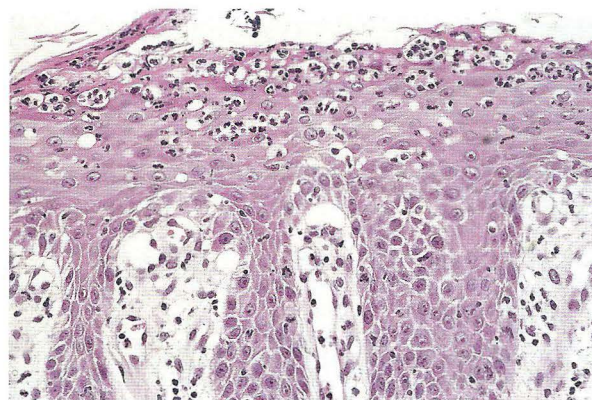
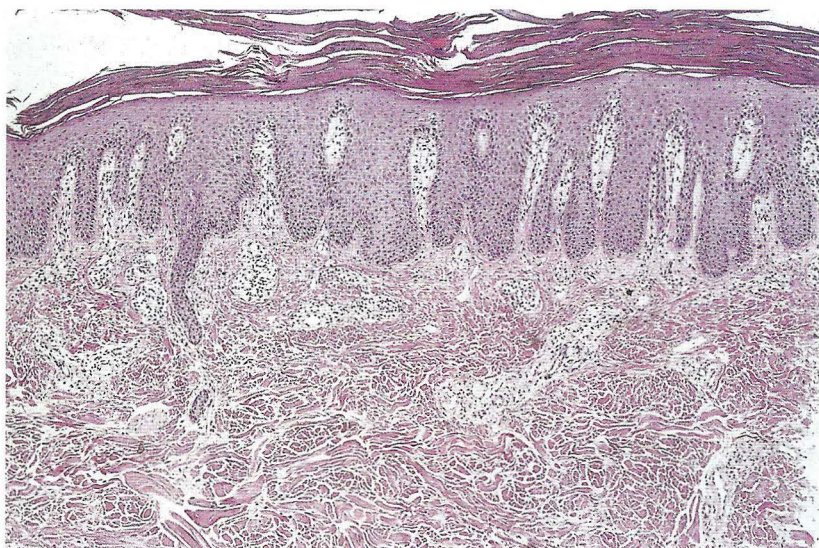


Fig. 1b) Note the focal loss of the granular layer, and dilated, tortuous capillaries in the dermal papillae; spongiform pustule of Kogoj in the spinous layer (haematoxylin-eosin 250x).

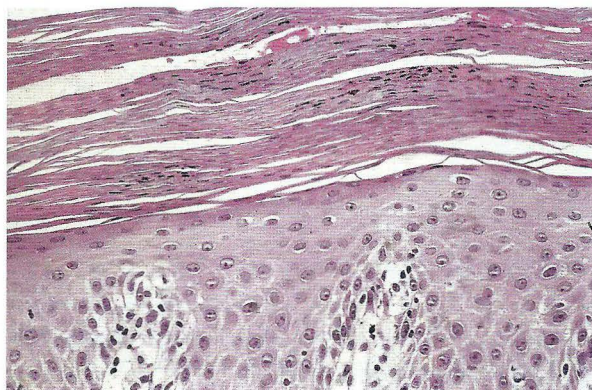
ding on the clinical type, age of the lesion, and with presence and degree of irritation or previous treatment (1,2,5-8). A correct diagnosis often requires a correlation between the clinical and histopathologic features.

In this article, we highlight the main histopathologic findings of psoriasis in the context of the different clinical types. In addition, a few clues that may be helpful in making a prompt diagnosis, will be mentioned.

Plaque type psoriasis

Plaque type of psoriasis vulgaris presents mainly as sharply demarcated erythematous plaques with silvery scales showing predilection for extensor surfaces of the elbows and knees, presacral areas and the scalp (3,4,9). It is further characterized by a chronic course with remissions and exacerbations. The nails are frequently affected (pits and onycholysis). About 15% of cases show joint involvement in the form of arthritis. The

Fig. 2b) Note keratinocytes with pale cytoplasm, and a few lymphocytes in the lower portion of the epidermis (haematoxylin-eosin 250x).



lesions may initially clinically simulate a variety of inflammatory skin conditions e.g., pityriasis rosea, secondary syphilis, pityriasis rubra pilaris, chronic nummular and dyshidrotic eczema, and lichen simplex chronicus.

Histopathologic findings vary mainly with the age of the lesions.

Early lesions

Early lesions are characterized mainly by slight epidermal hyperplasia with mounds of parakeratosis containing neutrophils, focal loss of granular layer, and slight exocytosis of inflammatory cells with spongiosis (Figs. 1a and b) (1,2,5-8,10). The epidermis reveals an increased number of mitotic figures. The papillary dermis sometimes shows slight edema and there are dilated, tortuous capillaries in the dermal papillae. A sparse, superficial perivascular inflammatory infiltrate consisting of mononuclear cells and neutrophils, but usually without eosinophils, is present in the upper dermis.

The main histopathologic differential diagnoses at this stage include conditions that reveal spongiotic dermatitis with focal parakeratosis, e.g., nummular and atopic dermatitis, pityriasis rosea, superficial gyrate erythemas, seborrheic dermatitis, and superficial fungal infections. In contrast to early psoriasis vulgaris, both nummular and atopic dermatitis exhibit more prominent acanthosis and may show scattered eosinophils within the dermal infiltrate. Pityriasis rosea and superficial gyrate erythemas reveal mounds of parakeratosis that do not contain neutrophils. Seborrheic dermatitis displays mounds of parakeratosis containing plasma located mainly at the edges of infundibular ostia.

Well-developed lesions

The histopathologic features of most well-developed lesions of plaque type psoriasis are characteristic and include prominent psoriasiform epidermal hyperplasia, mainly involving the rete ridges and sparing the epidermis over the dermal papillae, hypogranulosis, and confluent parakeratosis with collections of neutrophils in stratum corneum (Fig. 2a) (1,2,5-8). Neutrophils may also be observed in the malpighian layer of the epidermis (spongiform pustules of Kogoj) as well as dermal papillae. The upper portions of the epidermis may reveal keratinocytes with a pale cytoplasm (Fig. 2b). Exocytosis of lymphocytes may be observed in the lower portions of the epidermis (6). The basal layer often reveals a focal increase in mitotic figures. The dermal papillae are typically thinned and elongated, and contain fibrillary

collagen and dilated capillaries. A perivascular inflammatory infiltrate of lymphocytes, histiocytes and neutrophils is usually present in the upper dermis. Histopathologic differential diagnoses mainly include subacute contact eczema, nummular dermatitis and psoriasiform fungal infections.

Late lesions

Late lesions of plaque type psoriasis are characterized mainly by acanthosis with elongation of the rete ridges. Instead of parakeratosis, compact orthokeratosis is usually present (Figs. 3a and b) (11). There is conspicuous bulbous enlargement of the tips of the evenly elongated rete ridges with narrowing at the bases, and sometimes "bridging" of the rete ridges. The suprapapillary plate remains thinned slightly and there are dilated tortuous capillaries in the dermal papillae.

Late lesions of plaque type psoriasis may histopathologically simulate lichen simplex chronicus. However, lichen simplex chronicus displays more prominent fibrosis of the papillary dermal collagen and marked hypergranulosis, whereas late chronic plaque type of psoriasis exhibits only subtle fibroplasia in the papillary dermis.

Treatment and irritation (rubbing and scratching) may alter slightly the histopathologic features of psoriasis vulgaris. Pretreated psoriatic plaques often show a minimal inflammatory infiltrate, a reduction in amount of epidermal hyperplasia and a less reduced granular layer (1,5). In some of these cases, there is also an increase of fibroblasts in the papillary dermis.

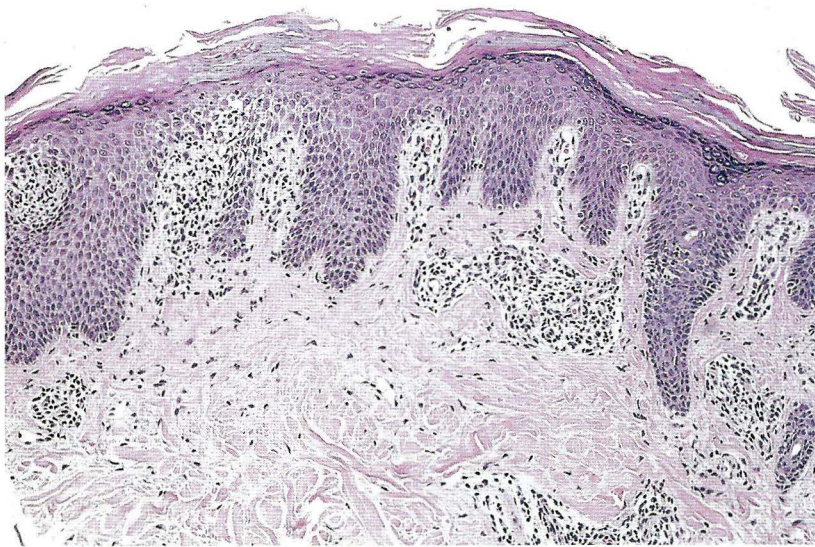
Guttate psoriasis

Eruptive guttate psoriasis commonly affects relatively younger individuals aged between 10 to 20 years. The skin condition is characterized by an acute outbreak of generalized, small, red, nonconfluent papules and plaques, which often accompany a streptococcal throat infection. The main clinical differential diagnoses include pityriasis rosea and lichen planus.

Histopathologic findings in guttate psoriasis are similar to those in early lesions of plaque type psoriasis (see above) and will not be discussed in further details.

Pustular psoriasis

Pustular psoriasis may develop in the setting of well-established plaque-type psoriasis. The skin condition is characterized by pustules commonly localized



A late lesion of plaque type psoriasis: Fig. 3a) There is acanthosis with compact orthokeratosis (haematoxylin-eosin 50).

exclusively on the palms and soles. In few patients, however, a generalized pustular form develops, often accompanied by fever and an elevated white blood cell count. Recently, Ackerman et al. have also proposed that a number of pustular skin conditions previously considered to be distinctive entities including acrodermatitis continua of Hallopeau, dermatitis repens of Crocker, impetigo herpetiformis, keratoderma blenorrhagicum, pustular bacterid of Andrews, subcorneal pustular dermatosis of Sneddon and Wilkinson, and geographic tongue actually represent different expressions of pustular psoriasis (5). Pustular psoriasis should clinically mainly be differentiated from superficial fungal infections.

Pustular psoriasis: Fig. 4a) There is a prominent collection of neutrophils in the upper half of the epidermis (spongiform pustules of Kogoj) and spinous zone (Munro's microabscesses) (haematoxylin-eosin 50x).

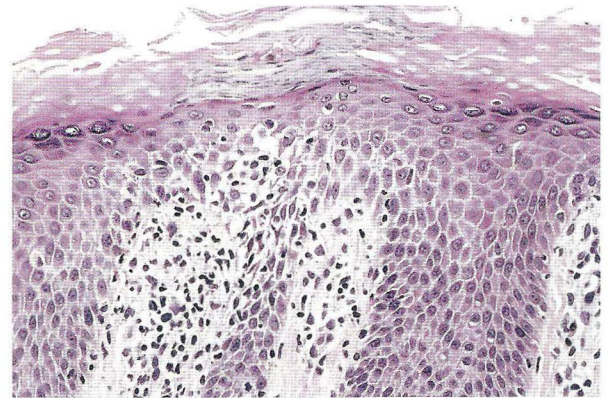
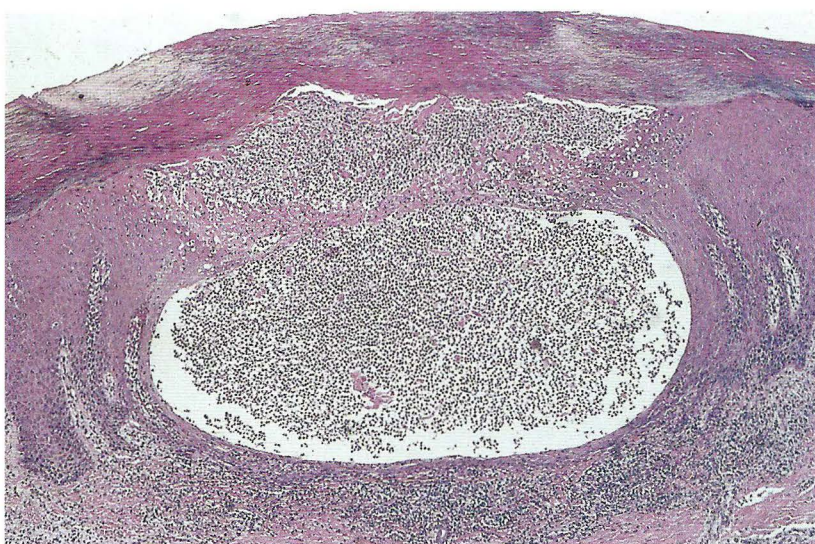
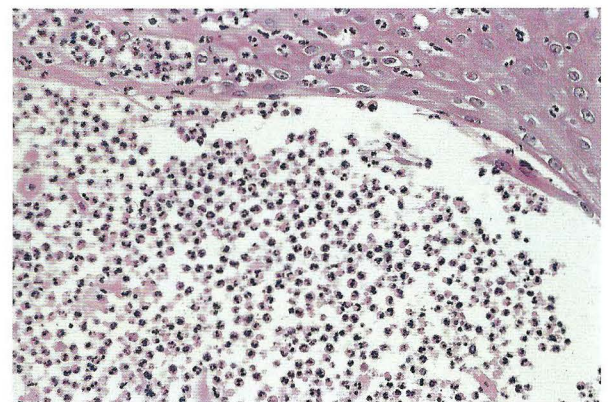


Fig. 3b) Higher magnification shows focal hypergranulosis and an inflammatory infiltrate in the upper dermis composed mainly of lymphocytes and histiocytes (haematoxylin-eosin 250x).

Histopathologically, pustular psoriasis generally shows similar features to those of plaque-type psoriasis except for the discrete collections of neutrophils in the spinous zone (Munro's microabscesses), sponge-like array of neutrophils in the upper half of the epidermis (spongiform pustule of Kogoj), collections of neutrophils beneath the cornified layer (subcorneal pustules), and/or presence of pockets of neutrophils within the cornified layer (intracorneal pustules) (Figs. 4a and b). Pustular lesions display less marked epidermal hyperplasia when compared to plaque type psoriasis (5,12).

Pustular lesions of psoriasis are histopathologically

Fig. 4b) Note the sponge-like array of neutrophils in the upper half of the epidermis (spongiform pustules of Kogoj) (haematoxylin-eosin 250x).



indistinguishable from skin lesions of Reiter's syndrome. Spongiform pustules are not specific for pustular psoriasis but may also be found in a variety of other skin conditions (e.g., dermatophytes, spirochetes).

Erythrodermic psoriasis

Erythrodermic psoriasis develops in about 2% of psoriatics usually following pustular or plaque types. In a minority of patients, erythrodermic psoriasis presents initially with no former history of the disease. Clinically, patients with erythrodermic psoriasis show extensive erythema with desquamation. These skin changes are often accompanied by fever, fatigue and muscle pains. The main clinical differential diagnoses include conditions that present as generalized erythroderma (e.g., Sezary syndrome, drug reactions).

Histopathologically, erythrodermic psoriasis ge-



Erythrodermic psoriasis: Fig. 5 a) There is epidermal hyperplasia and parakeratosis containing neutrophils, slight exocytosis of inflammatory cells, and prominent dilation of the superficial blood vessels in the upper dermis (haematoxylin-eosin 50x).

Table 1. Clues to histopathologic diagnosis of psoriasis*

| Histopathologic Feature |
|---|
| Mounds of parakeratosis with neutrophils at their summits |
| Thin, elongated rete ridges of nearly equal length |
| Striking pallor of cells in the upper part of the spinous layer |
| Dilated tortuous capillaries within thinned dermal papillae |

*The listing of clues was modified according to Ackerman et al. (5,6).

nerally shows less characteristic features (13). The subset of cases with more typical histopathologic changes display epidermal hyperplasia and parakeratosis containing neutrophils, slight exocytosis of inflammatory cells with spongiosis, and prominent dilation of the superficial blood vessels (Figs. 5a and b) (1,2,5). The amount of parakeratosis still adherent to the epidermal surface is usually scant. Moreover, the stratum corneum may be totally absent.

Table 1 summarizes important clues to histopathologic diagnosis of psoriasis, which have been compiled mainly from publications by AB Ackerman et al. (5,6).

Fig. 5 b) Note the scant amount of parakeratosis more or less adherent to the epidermal surface (haematoxylin-eosin 100x).



REFERENCES

1. Weedon D. Skin pathology. Churchill Livingstone, Edinburgh, London, 1997.
2. Maize JC, Burgdorf WHC, Hurt MA, LeBoit PE, Metcalf JS, Smith T, Solomon AR. Cutaneous pathology. Churchill Livingstone, Philadelphia 1998.
3. Kerkof van de PCM. Psoriasis: a spectrum of expressions. Acta Dermatovenorologica APA 1998;7:57-64.
4. Kerkof van de PCM. The differential diagnosis of psoriasis. Acta Dermatovenorologica APA 1999;8:50-8.
5. Ackerman AB, Chongchitnant N, Sanchez J, Guo, Bennin B, Reichel M, Randall MB. Histologic diagnosis of inflammatory skin diseases: an alogarithmic method based on pattern analysis. Lea & Febiger, Philadelphia, 1997.
6. Ackerman AB, Ragaz A. The lives of lesions, chronology in dermatopathology. Masson, New York 1984.
7. Cox AJ, Watson W. Histological variations in lesions of psoriasis. Arch Dermatol 1972; 106:503-6.
8. Ragaz A, Ackerman AB. Evolution, maturation, and regression of lesions of psoriasis. Am J Dermatopathol 1979; 1: 199-214.
9. Stern RS. Epidemiology of psoriasis. Dermatol Clin 1995; 13: 717-22.
10. Jablonska S, Chowaniec O, Maciejowska E. Histology of psoriasis: the role of polymorphoneutrophils. In: Beutner E, editor. Autoimmunity in psoriasis. Boca Raton, Fla: CRC Press, 1982: 21-36.
11. Griffin TD, Lattanand A, VanScott EF. Clinical and histologic heterogeneity of psoriatic plaques: therapeutic relevance. Arch Dermatol 1988; 124: 216-20.
12. Prystowsky JH, Cohen PR. Pustular and erythrodermic psoriasis. Dermatol Clin 1995; 13: 757-70.
13. Abrahams I, McCathy JT, Sanders SL. 101 cases of exfoliative dermatitis. Arch Dermatol 1963; 87: 96-101.

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