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Common spongiotic dermatoses

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ABSTRACT

This review article focuses on the spongiotic tissue reaction pattern and some of the common entities that practicing pathologists can frequently encounter in their practice. The spongiotic tissue reaction pattern is the single most common one encountered in the routine analysis of inflammatory dermatoses, and the most non-specific one as well. Spongiotic dermatoses include a very large list of disorders which are best grouped under neutrophilic, eosinophilic, miliarial, follicular and pityriasiform forms of spongiosis. The dermatoses that will be discussed under this category include contact dermatitis, eczema, pityriasis rosea, stasis dermatitis, seborrheic dermatitis and others.

Introduction

The spongiotic tissue reaction pattern is the single most common one encountered in the routine analysis of inflammatory dermatoses, and the most non-specific one as well. As pathologists, lack of specificity and vagueness on reporting are difficult issues to deal in the routine practice, as we have been trained to determine the 'final diagnosis'. It's hard to be faced with a pattern that often times seems to be 'non-specific…' At the end, can you effectively discriminate between eczema, contact dermatitis, allergic dermatitis, a spongiotic drug reaction or an id reaction? The answer is certainly 'no', but the reader should feel relieved by the fact that, while those differential diagnostic considerations are provided clinically, the dermatologist will be most satisfied with the demonstration of a spongiotic tissue reaction pattern.

The clinical presentation of these disorders is protean, and can range from crusted patches, plaques, papules, papulovesicles, or even blisters and bullae.[1,2] The term 'eczema' derives from the Greek word which means 'boiling over'.[3,4] The resolving spongiotic disorders show a characteristic collarette of scale.

Histologically, the spongiotic tissue reaction pattern involves the accumulation of fluid in the intercellular spaces of the epidermis, making the desmosomes junctions more easy to appreciate.[5] Parakeratosis forms above the areas of spongiosis. The foci of spongiosis can be microscopic to grossly identifiable clinically with vesicles or bullae. With spongiosis, some inflammatory cells can extend into the epidermis, a term that is called exocytosis. It is important to remember that other tissue reaction patterns can be accompanied by spongiosis. In lichenoid tissue reactions suprabasilar spongiosis is relatively common. In psoriasis, particularly in pustular forms, spongiform postulation is very characteristic. Certain viral exanthems and morbiliform drug reactions can be accompanied by mild spongiosis, usually more limited to the basal layer of the epidermis. Follicular mucinosis, the accumulation of mucopolysaccharides in the hair follicle epithelium, can simulate spongiosis, and is more easily recognized with the use of special stains. In cutaneous T-cell lymphomas, such as mycosis fungoides, extension of the lymphocytes into the epidermis, and particularly collections of them (so-called Pautrier microabscesses) can mimic inflammatory disorders.

There are 5 patterns of spongiosis where many inflammatory disorders are included and separated into.[5] Those patterns will be represented further but classic histologic examples which are encompassed within them:

a) Neutrophilic spongiosis (Table 1).
b) Eosinophilic spongiosis (Table 2).
c) Miliarial spongiosis (Table 3).
d) Follicular spongiosis (Table 4).
e) Pityriasiform spongiosis (Table 5).

Prurigo pigmentosa

Prurigo pigmentosa (PP) is a rare inflammatory skin disorder of unknown etiology. It's characterized by recurrent short lived pruritic erythematous macules and papules in the upper part of the back, sacrum, abdomen and chest, that leaves behind netlike

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Table 1
Common examples of spongiotic neutrophilic dermatoses.

1. Pustular psoriasis/palmoplantar pustulosis
2. Prurigo pigmentosa
3. IgA pemphigus
4. Acute generalized exanthematous pustulosis (AGEP)
5. Dermatophytosis
6. Pustular contact dermatitis
7. Nutritional deficiencies

Table 2
Common examples of spongiotic eosinophilic dermatoses.

1. Pemphigus (vulgaris, foliaceous, erythematosus, vegetans)
2. Bullous pemphioid/pemphigus gesationis/cicatricial and mucosal pemphigoid
3. Drug reactions
4. Allergic contact dermatitis and atopic dermatitis
5. Arthropod bite reactions
6. Eosinophilic folliculitis
7. Incontinentia pigmenti (first stage)
8. Id reactions
9. Wells syndrome

Table 3
Common examples of miliarial spongiotic dermatosis.

1. Miliaria

Table 4
Common examples of follicular spongiotic dermatoses.

1. Acute folliculitis of various etiologies (most common infectious, acne)
2. Atopic dermatitis with a follicular pattern
3. Eosinophilic folliculitis
4. Follicular mucinosis
5. Perioral dermatitis
6. Seborrheic dermatitis

Table 5
Common examples of pityriasiform spongiotic dermatosis.

1. Pityriasis rosea
2. Pityriasiform drug reactions
3. Erythema annulare centrifugum (superficial variant)
4. Allergic contact dermatitis
5. Nummular dermatitis
6. Gianotti-Crosti syndrome

hyperpigmentation upon resolution of the lesions.[6–8] It appears to be more common in young Japanese women. A number of exogenous (contact allergens, bismuth salicylate ingestion, sweating, trauma) and systemic conditions (ketosis from diabetes, fasting, pregnancy, anorexia, and H. pylori gastritis) are associated with PP. Some have considered this entity as an 'inflammatory' variant of confluent and reticulated papillomatosis.[9] Histologically the lesions begin with a perivascular and superficial neutrophilic inflammatory infiltrate. The neutrophils cluster shortly in the dermal papillae and extend to the epidermis with the presence of neutrophilic spongiosis. In addition to spongiosis, ballooning and necrotic keratinocytes are present. Subsequently, eosinophils and lymphocytes become the predominant infiltrate. Sometimes a lichenoid band of lymphocytes, in addition to vacuolar alteration of the basal keratinocytes is present. Later on the lesions leave melanophages in the dermis, and the epidermis show changes of acanthosis, parakeratosis and hyperpigmentation.[10,11] Direct immunofluorescence studies are characteristically negative (Fig. 1).

Dermatophyte infections

Dermatophyte infections represent approximately 4% of dermatological consultations in the routine clinical practice. The clinical appearance is variable, and depends on a number of different factors, which include the species of fungi, the site, immunological status, and prior use of topical corticosteroids. The most classic clinical lesion is an erythematous (and sometimes vesicular) annular macule or plaque with peripheral scaling, desquamation and central clearing. Broken hairs and dystrophic nails are also frequent. The term Majocchi's granuloma is used to describe nodular and plaque-like lesions on the lower leg, histologically showing the image of a granulomatous perifolliculitis. The agents related to dermatophyte infections include three genera of fungi: *Epidermophyton, Microsporum* and *Trichophyton.* T. rubrum accounts for approximately 40% of all dermatophyte infections worldwide.[12–14]

Histologically, dermatophyte infections can be characterized by the presence of spongiosis +/- psoriasiform acanthosis with the presence of neutrophils.[5] Intraepidermal vesicles can sometimes be noted. Compact basket-weave orthokeratosis is more frequent than parakeratosis. The presence of neutrophils in the stratum corneum leads to the formation of a classic histologic sign, referred to as 'sandwich sign'. It's capital to remember that any new biopsy with the clinical or histologic picture of 'psoriasis' should have additional special stains done (GMS, PAS) to exclude the possibility of a fungal infection. The presence of folliculitis can sometimes be seen. Within the dermis, there is a variable inflammatory infiltrate, with a perivascular or diffuse mixed infiltrate that includes lymphocytes, histiocytes, neutrophils and/or eosinophils (Fig. 2). GMS and PAS stains can help identify the organisms in the stratum corneum.

Incontinentia pigmenti

Incontinentia pigmenti (IP) is an X-linked dominant genodermatoses. A mutation of the *NEMO* gene is noted, which encodes inhibitors of the transcription factor NFkB, causing a transactivation of the pathway.[15–17] Clinically the disease follows a rash that changes according to the age of the kid. From birth to the first 2 weeks of life, the rash has a vesicular pattern (Fig. 3). The next 4 weeks of life are characterized by the presence of verrucous lesions. Between the first 3–6 months of life, most of the lesions show hyperpigmentation, which later on translate to areas of hypopigmentation in the next 2nd and 3rd decades of life. Other clinical findings include scarring alopecia, nail dystrophy, teeth abnormalities, hypopigmented changes in the retina, CNS abnormalities and later on subungueal tumors.[18–23]

Histologically, IP is characterized by eosinophilic spongiosis on its first stage.[17,24–27] The presence of intraepidermal vesicles containing eosinophils is particularly common (Fig. 3). Occasional dyskeratotic cells with eosinophilic hyaline cytoplasm in the epidermis adjacent to the vesicles is also seen. The superficial dermis has an infiltrate particularly rich in eosinophils. The verrucous stage is best demonstrated by the presence of acanthosis, hyperkeratosis, mild irregular papillomatosis and abundant dyskeratotic cells. The third stage shows pigmentary incontinence.

Apocrine miliaria (Fox-Fordyce disease)

This is a chronic papular eruption usually limited to the axilla that almost exclusively affects young adult women. The miliarias