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Introduction to inflammatory dermatoses: Histological clues for the practicing pathologist

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Introduction

The discipline of dermatopathology is complex and very frequently practicing surgical pathologists feel overwhelmed by the complexity of the inflammatory disorders. While the easy approach to handling those cases relies on having an 'expert' dermatopathology opinion, in many circumstances such cases can be managed using a descriptive approach and having an open dialogue and communication with the referring clinicians. Most frequently, dermatologists, who have a broad clinical and pathologic experience to the diagnosis and therapy of skin diseases, can adequately orient the pathologist to the understanding and recognition of specific findings in the biopsy that can point towards a specific direction. As clinical medicine evolves, other specialties frequently face and encounter patients with dermatologic conditions (family practitioners, urologists, gynecologists, etc.). In some instances, the clinical description and differential diagnoses provided is more limited, and pathologists feel the challenge of providing a reliable answer to a protean character of histologic findings.

In this review and introduction, a focus is placed on the common terminology used in the description of inflammatory conditions. The use of tissue reaction patterns is the mainstay for the categorization of inflammatory dermatoses. It is important to emphasize over and over that for certain patterns of inflammation an innumerable number of different entities can be encompassed within that reaction pattern. The prototype of such are spongiotic dermatoses. General pathologists and practitioners, however, should feel relieved by the fact that clinicians understand the notorious lack of specificity of the pattern. Therefore, there are no particular expectations to 'get to the right diagnosis'. To this extent, a dermatologist is not expecting that a pathologist will state on the report whether the biopsy shows contact dermatitis, an id reaction, eczema or irritant contact dermatitis. They will just feel relieved by the adequate description of a spongiotic pattern. Similarly, some particular patterns are so classic under the microscope, where only few entities can be included, where a pathologist can make a more accurate interpretation and help to arrive to a specific diagnosis. An example of the latter is the finding of axillary granular parakeratosis, where distinctive abnormal keratin hyaline granules are retained on a stratum corneum that shows an abnormal retention of the nuclei (parakeratosis).

The majority of inflammatory dermatoses can be grouped according to six specific patterns, so-called major tissue reactive patterns[1,2]. Those include: 1) lichenoid / interface (band-like inflammation / basal cell vacuolar damage); 2) psoriasiform (regular epidermal hyperplasia); 3) spongiotic (intraepidermal cellular edema); 4) vesiculobullous (blistering); 5) granulomatous (chronic granulomatous inflammation); and 6) vasculopathic (injury to the blood vessels).

To this extent, a detailed histopathologic description of common terms used in skin biopsy diagnosis will be described further. ***Acanthosis***refers to an increased thickness of the epidermis (Fig. 1). Acanthosis always implies increased number of cells within specific layers, therefore, it's equivalent to hyperplasia of the epidermis[3]. The term ***psoriasiform acanthosis or hyperplasia*** (Fig. 2) is used when the rete ridges are elongated and hyperplastic with a ***regular*** pattern. By regular it means that each individual rete redges has the same length. Many use modifier adjectives such as 'vague', 'like', when the psoriasiform changes are somewhat irregular or incomplete. ***Papillomatosis*** implies regular acanthosis with papillary changes on the surface (Fig. 3). Papillomatosis is particularly common in certain benign neoplastic conditions such as verrucae, and epidermal nevi [2,4]. ***Orthokeratosis*** refers to the corneal layer sitting on the surface of the epidermis. It implies a woven pattern of keratin above the granular cell layer, and absence of nuclei in this layer of the epidermis (Fig. 4). Under normal circumstances, in certain areas of the skin (palms or soles), a thickened corneal layer with an orthokeratotic pattern is present. When there is an abnormal retention of the nuclei in the stratum corneum, the term ***parakeratosis*** is used (Fig. 5). ***Dyskeratosis*** is a pathological phenomenon that occurs when there is an abnormal, hypereosinophilic condensation of the cytoplasm of the keratinocytes, characteristically in the basal layer, in association with displacement of the nuclei to the side of the cell, or even sometimes complete disappearance of the nuclei (Fig. 6). Dyskeratosis is associated with interface tissue reactions. A lichenoid tissue reaction describes a dense band of typically lymphocytes and histiocytes that often times obscures the dermal-epidermal junction. ***Acantholytic dyskeratosis*** is characterized by suprabasilar clefting with

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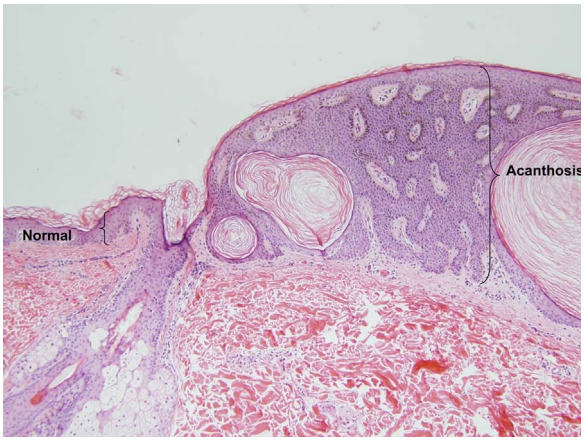

Fig. 1. An example of epidermal hyperplasia (right side) or acanthosis in the setting of a benign seborrheic keratosis (H&E, 100x).

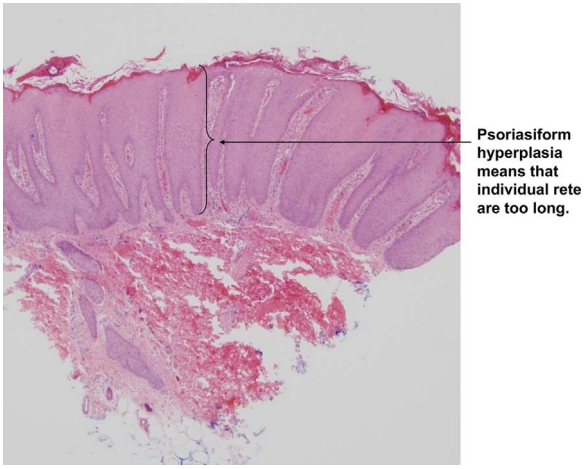

Fig. 2. In psoriasiform acanthosis/hyperplasia the individual rete are elongated with the same length across them (H&E, 100x).

Fig. 3. Papillomatosis indicates that the top of the epidermis is irregular and has a "peaks and valleys" appearance (H&E, 100x).

acantholytic and dyskeratotic cells at all levels of the epidermis (Fig. 7). It's commonly an incidental finding but can also be present in the vicinity of neoplasms (particularly architecturally disordered nevi), generalized dermatoses (Darier's disease), as a single neoplasm (acanthoma), transient disorders (Grover's disease), etc. However, acantholysis can occur without dyskeratosis in association with spongiosis, autoimmune disorders and viral infections (Fig. 8). The term ***epidermolytic hyperkeratosis*** is used to describe the presence of compact hyperkeratosis in association with granular and vacuolar degeneration of the spinous and granular cell layers (Fig. 9). **Cornoid lamellation** is a localized abnormal form of keratinization that creates a column of parakeratosis with an absent or diminished granular cell layer underneath the column. It's characteristic of the group of porokeratosis (Fig. 10).

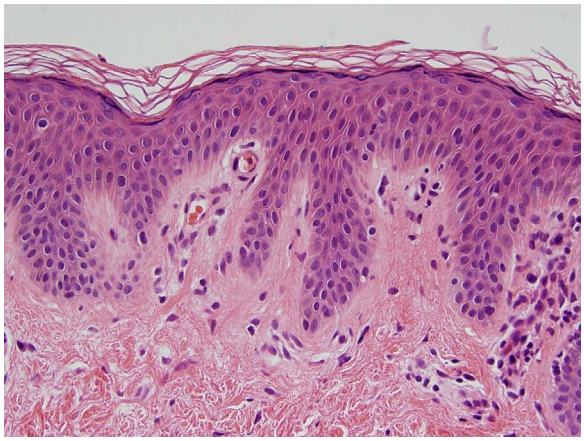

Fig. 4. An example of ortho (correct, right) keratosis. Note the woven pattern of the keratin above the granular cell layer. Note the absence of nuclei in those keratinocytes (H&E, 200x).

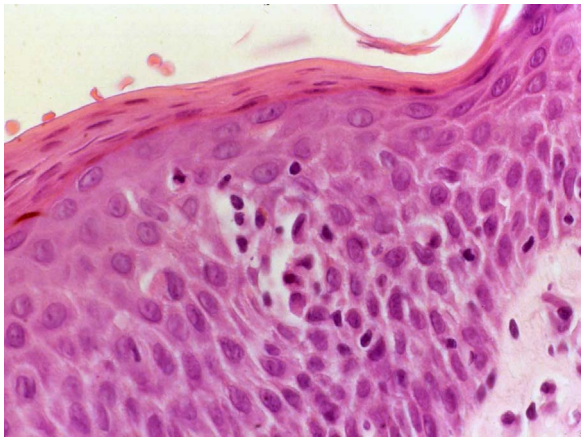

Fig. 5. The cornified layer/stratum corneum is the most superficial layer of the epidermis. In this example, it is abnormal because it has retained nuclei – that is to say it is parakeratotic (H&E, 400x).

The term ***spongiosis*** indicates the presence of fluid/edema between neighboring keratinocytes (Fig. 11)[5]. This causes a slight separation between adjacent keratinocytes so that the spinous processes/desmosomes become apparent and/or prominent. Sometimes, in addition to fluid, some inflammatory cells are present in the epidermis. When describing a biopsy of an inflammatory process, the word ***exocytosis***is commonly adopted (neutrophilic / eosinophilic exocytosis) to document that. However, when discussing a possible 'neoplastic' condition, particularly in the setting of neoplastic lymphocytic infiltrates, the word ***epidermotropism*** is preferred (Fig. 12). Regardless, both terms are used to describe the presence of inflammatory cells in the epidermis. A ***blister*** indicates the presence of a watter-filled space +/- inflammatory cells at all levels of the epidermis. Clinically, most of the blisters that occur superficially are very fragile, and

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