My approach to superficial inflammatory dermatoses

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Superficial inflammatory dermatoses are very common and comprise a wide, complex variety of clinical conditions. Accurate histological diagnosis, although it can sometimes be difficult to establish, is essential for clinical management. Knowledge of the microanatomy of the skin is important to recognize the variable histological patterns of inflammatory skin diseases. This article reviews the non-vesiculobullous/pustular inflammatory superficial dermatoses based on the compartmental microanatomy of the skin.

The histological diagnosis of cutaneous inflammatory diseases can be confusing, even for the most experienced pathologist. This is because the immune system within the skin has limited ways in which it reacts and responds to an antigenic stimulus, and many inflammatory diseases do not show specific histological features. In view of this complexity and commonality, histological patterns of recognition are beneficial for the rapid development of a differential diagnosis. This can be achievable by being familiar with the microanatomy of the skin and its regional variations, in addition to the basic structural alterations that can occur in different pathological conditions. This review is oriented towards the recognition of the histological patterns seen in non-vesiculobullous/pustular inflammatory superficial dermatoses that involve the epidermis and papillary dermis, using a schematic approach.

THE ANATOMICAL COMPARTMENTS/UNITS OF THE SKIN

From the practical point of view, the skin is divided into four anatomical compartments or units:

1. The first compartment/unit includes the epidermis, papillary dermis, and superficial vascular plexus. These structures are interrelated and mutual.
2. The second compartment/unit consists of the reticular dermis and the deep vascular plexus.
3. The third compartment/unit consists of the pilosebaceous units, the eccrine glands, and in certain anatomical locations, the apocrine glands.
4. The fourth compartment/unit is the subcutaneous tissue (panniculium).

Although using a compartmental approach to establish the reaction pattern facilitates the diagnosis of inflammatory skin diseases, there are limitations to this approach. The size of the skin biopsy should be adequate and representative of all four compartments and should also include hair follicles. A 2 mm punch biopsy is too small to represent all compartments, and often insufficient to demonstrate a recognizable pattern. A 4 mm punch biopsy is preferred, and usually adequate for the histological evaluation of most inflammatory dermatoses. However, a larger biopsy (6 mm punch biopsy), or even an incisional biopsy, might be necessary in panniculitis or cutaneous lymphoproliferative disorders. A superficial or shave biopsy should be avoided, because it might be misleading, producing an erroneous pattern and diagnosis.

PRACTICAL POINTS

In the histological evaluation of skin for inflammatory dermatoses, a clinicopathological correlation is essential, and plays an important role in achieving the diagnosis. The patient’s age and relevant clinical history, in addition to the site from which the skin biopsy was obtained, should be provided to the pathologist. After proper fixation and processing, three deeper haematoxylin and eosin-stained sections and one section stained with periodic acid Schiff (PAS) for evaluation of the epidermal basement membrane, blood vessels, and the presence of fungal organisms are considered sufficient for microscopic evaluation. The need for deeper sections and special stains varies from one case to another, and additional tasks can be ordered when necessary. Special stains that might be beneficial in establishing a diagnosis of inflammatory dermatoses include Martius-Scarlet blue stain for fibrin, colloidal iron Hale’s for acid mucin, Prussian blue for iron, toluidine blue for mast cells, Fontana Masson for melanin pigment, elastic stain for perforating disorders and demonstration of elastic tissue, Von Kossa for calcium deposits, and Congo red for amyloid. Gram stain and Gomori methenamine silver stain can be helpful in demonstrating bacterial and fungal microorganisms, respectively.

“Immunofluorescence studies have an essential role in diagnosing immunologically related inflammatory skin diseases, specifically vesiculo-bullous diseases and vasculitis.”

Immunohistochemistry has a limited role, although it can be useful in certain clinical situations.

Abbreviations: GVHD, graft versus host disease; HIV, human immunodeficiency virus; PAS, periodic acid Schiff; PVI, perivasculare inflammatory infiltrate.
conditions, such as characterising the neoplastic CD4+ T cell population in mycosis fungoides and the CD4+ and CD8+ T cell populations in patients with human immunodeficiency disorder (HIV). It is worthwhile mentioning that the CD8+ T cell population is the predominant subtype in HIV dermatoses. Immunofluorescence studies have an essential role in diagnosing immunologically related inflammatory skin diseases, specifically vesiculo-bullous diseases and vasculitis. Electron microscopy is of limited value, but may be helpful in certain vesiculo-bullous diseases, mycosis fungoides, and Langerhans cell histiocytosis.

A PRAGMATIC APPROACH

Most common superficial inflammatory dermatoses involve the first compartment/unit of the skin. The most common pattern of reaction encountered is the superficial perivascular inflammatory infiltrate. A transient inflammatory stimulus results in slight hyperaemia and a mild perivascular lymphocytic infiltrate. If the stimulus persists, interstitial oedema and endothelial swelling develop. With further stimulation, involvement of the overlying epidermis occurs. Inflammatory dermatoses involving the first compartment of the skin are divided into three main categories (fig 1): (1) non-vesiculobul lous/pustular lesions, (2) vesiculobul lous lesions. The non-vesiculobul lous/pustular lesions, the focus of this review, are divided into two categories based on the presence or absence of epidermal changes. When epidermal changes are present, they are further subdivided into spongiotic dermatitis, interface dermatitis, and psoriasiform dermatitis (fig 2).

Inflammatory dermatoses without epidermal changes

The inflammatory skin dermatoses without epidermal changes are manifested histologically by a superficial perivascular inflammatory infiltrate (PVI). This reactive pattern is induced by many conditions. The type of inflammatory cell infiltrate is different from one condition to another, allowing further subclassification of the PVI into six groups (table 1).

PVI with a predominant lymphocytic infiltrate

This is the most common type of PVI. Many conditions can result in a lymphocytic PVI. Common causes are immunological and non-immunological cutaneous drug eruption—particularly secondary to antibacterials—urticarial reactions, viral exanthema, infestation, and insect bites. Other less common conditions are fungal infections, pigmented purpuric dermatoses, erythema annulare centrifugum, and other gyrate erythemas.

PVI with lymphoeosinophilic infiltrate

Many conditions that cause PVI with a predominant lymphocytic infiltrate can present with a lympho eosinophilic infiltrate. These conditions include drug reactions (fig 4), urticarial reactions, a prevesicular early stage of bullous pemphigoid, insect bites (fig 5), infestations, and HIV related dermatoses. In pregnant women, pruritic urticarial papules and plaques of pregnancy are characterised by a perivascular lympho eosinophilic inflammatory infiltrate in skin biopsies.

PVI with lymphoplasmacytic infiltrate

A prominent plasma cell component of the inflammatory infiltrate may be seen adjacent to an area of trauma, ulceration, or scar. It is also seen in cases of rosacea, secondary syphilis, and erythema chronicum migrans, which is pathognomonic of Lyme’s disease. The patch stage of Kaposi’s sarcoma may be associated with increased
plasmacytic cell infiltration. Occasionally, early cutaneous connective tissue diseases can be associated with an increased plasma cell population. It is worth mentioning that relatively larger numbers of plasma cells are usually present in the mucosal biopsies and may be non-specific.

**PVI with increased mast cells**

Mastocytosis is a rare disease characterised by primary pathological accumulation of excessive numbers of mast cells in different tissues. Urticaria pigmentosa is the most common form of cutaneous mastocytosis. The mast cells are present in the interstitium and around the superficial vascular channels, and some of the mast cells show degranulation. A toluidine blue stain is usually used to demonstrate mast cells.

**PVI with lymphohistiocytic infiltrate**

This is the most confusing type of PVI. Conditions that are associated with a lymphohistiocytic inflammatory cell infiltrate include drug reactions, viral infections and post-viral reactions, HIV dermatoses, and leprosy. The mast cells are present in the interstitium and around the superficial vascular channels, and some of the mast cells show degranulation. A toluidine blue stain is usually used to demonstrate mast cells.

**PVI with a neutrophilic infiltrate**

A prominent perivascular neutrophilic inflammatory cell infiltrate can be associated with neutrophilic urticarial reactions (fig 7A), dermatitis herpetiformis, early IgA dermatosis, early Sweet’s syndrome, early connective tissue disorders such as lupus erythematosus, early herpetic infection, and acute generalised exanthematous pustulosis (fig 7B). The last three conditions are usually associated with epidermal changes. Leucocytoclastic vasculitis should be ruled out in all cases of neutrophilic dermatosis (fig 7C). The examination of multiple haematoxylin and eosin stained deeper sections is helpful.

**Inflammatory dermatoses with epidermal changes**

The inflammatory dermatoses with epidermal changes are classified histologically into spongiotic dermatitis, interface dermatitis, and psoriasiform dermatitis.

**Spongiotic dermatitis**

Spongiotic dermatitis is defined by the presence of epithelial intercellular oedema. It is caused by a variety of clinical conditions. These include allergic/contact dermatitis, atopic dermatitis, nummular dermatitis, dyshidrotic dermatitis, seborrheic dermatitis, drug reactions, lichen reaction, dermatophytosis, miliaria, Gianotti-Crosti syndrome, and pityriasis rosea.

Spongiotic dermatitis is further subclassified into acute, subacute, and chronic, depending on the histological features and the time the biopsy was performed.

**Acute spongiotic dermatitis**

This shows a variable degree of epidermal spongiosis and vesicle formation; the vesicles are filled with proteinaceous fluid containing lymphocytes and histiocytes (fig 8A). Superficial dermal oedema with a perivascular lymphocytic inflammatory cell infiltrate is usually present. Exocytosis of the inflammatory cells is present, and there is no acanthosis or parakeratosis. In allergic/contact dermatitis and atopic dermatitis, eosinophils may be present in the dermis and epidermis (eosinophilic spongiosis).

**Subacute spongiotic dermatitis**

This is the most frequently encountered type of spongiotic dermatitis. The degree of spongiosis and exocytosis of the
inflammatory cells is mild to moderate, and compared with acute spongiotic dermatitis there is irregular acanthosis and parakeratosis. A superficial dermal perivascular lymphohistiocytic inflammatory infiltrate, swelling of the endothelial cells, and papillary dermal oedema are present (fig 8B, C).

**Chronic spongiotic dermatitis**
The spongiosis is mild to absent but there is pronounced irregular acanthosis, hyperkeratosis, and parakeratosis (fig 8D). Minimal dermal inflammation and exocytosis of the inflammatory cells are present. Fibrosis of the papillary...
dermis may be present. The PAS stain is essential to exclude fungal infection.

All three histological subtypes of spongiotic dermatitis can be caused by various clinical conditions. The microscopic features of the chronicity of the lesion depend largely on the time of biopsy and the clinical course of the lesion. The role of the pathologist is to recognise the histological features of spongiotic dermatitis. However, a clinical correlation is crucial for a definitive diagnosis.

**Interface dermatitis**

This is histologically classified into two categories: (a) interface dermatitis with vacuolar change, and (b) interface dermatitis with lichenoid inflammation (fig 2). Each category has distinctive morphological features, and is caused by various clinical conditions (table 2).

**Interface dermatitis with vacuolar alteration**

This pattern of inflammatory dermatosis is characterised by the presence of a mild inflammatory cell infiltrate along the dermoepidermal junction, with vacuolar change within the basal keratinocytes. Individual necrotic, predominantly basal, keratinocytes are frequently seen because the inflammatory process causes injury to the basal keratinocytes (colloid or Civatte bodies). Numerous clinical conditions can result in vacuolar alteration of the basal keratinocytes, including: viral exanthems, phototoxic dermatitis, acute radiation dermatitis, erythema multiforme,37 erythema multiforme-like drug eruption (fig 9), and fixed drug eruption.41 Vacuolar alteration is often present in cutaneous connective tissue disorders such as lupus erythematosus42 43 (fig 10A–D), and dermatomyositis.44 Acute cutaneous graft versus host disease (GVHD) (table 3) characteristically shows vacuolar alteration (fig 11), which ranges in severity from focal or diffuse vacuolation of the
basal keratinocytes (grade I), to separation at the dermo-epidermal junction (grade III).\(^4^5\) Drug reactions can be difficult to differentiate from acute GVHD based on morphological appearances. The presence of an eosinophilic inflammatory component favours a drug reaction, whereas involvement of the hair follicles is in keeping with acute GVHD. The clinical history is essential to distinguish between these two entities.

**Interface dermatitis with lichenoid alteration**

This is another pattern of cutaneous inflammatory reaction, which is characterised by a confluent, band-like, dense accumulation of inflammatory cells in the papillary dermis, mainly consisting of small lymphocytes and a few histiocytes along or hugging the dermo-epidermal junction. The lichenoid reaction is often accompanied by vacuolar degeneration of the basal keratinocytes and the presence of apoptotic bodies (colloid or Civatte bodies). Many clinical conditions can result in the lichenoid alteration. These include lichen planus\(^4^6\) (fig 12), lichen planus-like keratosis,\(^4^7\) lichenoid actinic keratosis, lichenoid drug eruption, lichenoid lupus erythematosus, lichenoid GVHD (chronic GVHD),\(^4^8\) lichen nitidus,\(^4^9\) pigmented purpuric dermatosis,\(^1^0\) lichen amyloidosus,\(^5^0\) pityriasis rosea,\(^5^1\) and pityriasis lichenoides chronica.\(^5^2\) Other unusual conditions that can be associated with a lichenoid inflammatory cell infiltrate are HIV dermatitis,\(^5^3\) syphilis, mycosis fungoides,\(^5^4\) urticaria pigmentosa,\(^5^5\) and post-inflammmatory hyperpigmentation.\(^5^6\) In cases of post-inflammatory hyperpigmentation, care should be taken not to miss the diagnosis of a mimic, such as regressed melanocytic lesion or lichenoid pigmented actinic keratosis. A detailed and appropriate clinical history is essential for the accurate diagnosis of interface dermatitis.

**Psoriasiform dermatitis (tables 4 and 5)**

This type of inflammatory dermatosis is characterised by the presence of regular epidermal hyperplasia, elongation of the rete ridges, hyperkeratosis, and parakeratosis. The portion of the epidermal cell layer that overlies the tips of dermal papillae (suprapapillary plates), and dilated, tortuous blood vessels within these papillae are often present. A superficial perivascular inflammatory cell infiltrate is usually encountered. Numerous conditions can result in psoriasiform dermatitis. These include psoriasis\(^5^8\) (fig 13), seborrheic dermatitis, psoriasiform drug eruption, chronic fungal infections, lichen simplex chronicus, chronic spongiotic dermatitis, secondary syphilis, pellagra\(^5^9\) and other nutritional deficiencies, and pityriasis rubra pilaris.\(^6^0\) Pityriasis lichenoides chronica,\(^5^1\) acrodermatitis enteropathica,\(^6^2\) inflammatory linear verrucous epidermal naevus,\(^6^3\) and rarely subacute psoriasiform cutaneous lupus erythematosus are also associated with psoriasiform dermatitis.

A systemic approach is important for pathological diagnosis when evaluating skin psoriasiform dermatites. Examining multiple deeper levels is recommended if the histological features in the initial cuts do not correlate well with the clinical history. Recognising various histological alterations in the epidermal layers can help in narrowing down the differential diagnosis of psoriasiform dermatitis. Agranulosis or hypogranulosis of the epidermis is a feature of
of psoriasis. However, in partially treated psoriasis, the granular cell layer may be present, so that clinical–pathological correlation is crucial for accurate diagnosis. An increase in the intensity of the granular cell layer is associated with various clinical conditions (table 6). The microscopic findings of seborrheic dermatitis often show features of both spongiform and psoriasiform changes. Evaluation of the dermoepidermal junction and dermis is important. Psoriasiform dermatitis with interface changes can be seen in drug reactions, cutaneous lupus erythematosus, and syphilis.

Table 4 Epidermal changes in psoriasiform dermatoses

<table>
<thead>
<tr>
<th>Condition</th>
<th>Hyperkeratosis</th>
<th>Parakeratosis</th>
<th>Acanthosis</th>
<th>Suprapapillary plate</th>
<th>Microabscess formation</th>
<th>Granular cell layer changes</th>
<th>Spinous cell layer changes (table 6)</th>
<th>Basal cell layer changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psoriasis</td>
<td>+</td>
<td>Diffuse</td>
<td>Regular</td>
<td>Thin</td>
<td>+</td>
<td>Decreased or absent</td>
<td>Increased mitotic activity; minimal spongiosis</td>
<td>Inflammatory cell exocytosis, single keratinocyte necrosis</td>
</tr>
<tr>
<td>Drug reaction</td>
<td>+</td>
<td>Focal</td>
<td>Regular and irregular</td>
<td>Normal or thick</td>
<td>–</td>
<td>Normal</td>
<td>Spongiosis; eosinophilic infiltrate</td>
<td>–</td>
</tr>
<tr>
<td>Chronic allergic/contact and atopic dermatitis</td>
<td>+</td>
<td>Focal; crust may be present</td>
<td>Irregular</td>
<td>Normal or thick</td>
<td>–</td>
<td>Normal</td>
<td>Spongiosis; eosinophilic infiltrate</td>
<td>–</td>
</tr>
<tr>
<td>Fungal infection</td>
<td>Compact</td>
<td>Focal; crust may be present</td>
<td>Irregular</td>
<td>Normal or thick</td>
<td>–</td>
<td>Normal</td>
<td>Occasional neutrophilic exocytosis; ± minimal exocytosis</td>
<td>–</td>
</tr>
<tr>
<td>LSC</td>
<td>+</td>
<td>Focal; thick crust</td>
<td>Regular or irregular</td>
<td>Thin or thick</td>
<td>–</td>
<td>Thickened, hypergranulosis</td>
<td>Spongiosis; eosinophilic infiltrate</td>
<td>–</td>
</tr>
<tr>
<td>Scabies</td>
<td>+</td>
<td>Focal or diffuse</td>
<td>Irregular</td>
<td>Normal or thick</td>
<td>–</td>
<td>Normal</td>
<td>Exocytosis; spongiosis; eosinophilic infiltrate</td>
<td>–</td>
</tr>
<tr>
<td>Seborrheic dermatitis and HIV dermatitis</td>
<td>+</td>
<td>Focal</td>
<td>Irregular</td>
<td>Normal or thick</td>
<td>–</td>
<td>Normal</td>
<td>Spongiosis; lymphocytic and neutrophilic infiltrate</td>
<td>–</td>
</tr>
</tbody>
</table>

Table 5 Dermal changes in psoriasiform dermatitis

<table>
<thead>
<tr>
<th>Psoriasiform dermatitis</th>
<th>Dermal changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psoriasis</td>
<td>PVU; mild oedema, dilated vessels</td>
</tr>
<tr>
<td>Drug reaction</td>
<td>PVU or band-like infiltrate; ± eosinophilic infiltrate</td>
</tr>
<tr>
<td>Chronic allergic/contact and atopic dermatitis</td>
<td>PVU; ± eosinophilic infiltrate</td>
</tr>
<tr>
<td>Fungal infection</td>
<td>PVU; ± neutrophilic infiltrate</td>
</tr>
<tr>
<td>LSC</td>
<td>PVU; reactive fibroblasts; papillary fibrosis</td>
</tr>
<tr>
<td>Scabies</td>
<td>Moderate PVU; numerous eosinophils in deep dermis</td>
</tr>
<tr>
<td>Seborrheic dermatitis and HIV dermatitis</td>
<td>Mild PVU</td>
</tr>
<tr>
<td>PR</td>
<td>Mild PVU</td>
</tr>
<tr>
<td>PR</td>
<td>PVU; focal extravasated RBCs</td>
</tr>
<tr>
<td>Syphilis</td>
<td>Band-like lymphocytic infiltrate; numerous plasma cells; vascular endothelial proliferation; thick BVs; histiocytes, rare eosinophils</td>
</tr>
<tr>
<td>PLC</td>
<td>PVU</td>
</tr>
<tr>
<td>MF</td>
<td>PVU; atypical lymphoid cells</td>
</tr>
</tbody>
</table>

*Parakeratotic mounds at the edge of follicular ostia.*

PVU, blood vessels; LSC, lichen simplex chronicus; MF, mycosis fungoides; PLC, pityriasis lichenoides chronicus; PR, pityriasis rosea; PRP, pityriasis rubra pilaris; +, present; –, absent.

Table 6 Epidermal histological alteration in psoriasiform dermatitis

<table>
<thead>
<tr>
<th>Alterations in stratum spinosum layer</th>
<th>Clinical conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased mitotic figures</td>
<td>Psoriasis</td>
</tr>
<tr>
<td>Spongiosis and exocytosis of lymphocytes</td>
<td>Chronic spongiotic dermatitis</td>
</tr>
<tr>
<td>Fungal infection</td>
<td>Drug reaction</td>
</tr>
<tr>
<td>Seborrheic dermatitis</td>
<td></td>
</tr>
<tr>
<td>Spongiosis and exocytosis of eosinophils</td>
<td>Atopic dermatitis</td>
</tr>
<tr>
<td>Allergic contact dermatitis</td>
<td>Nummular dermatitis</td>
</tr>
<tr>
<td>Drug reaction</td>
<td>Nevocytic keratinocytes</td>
</tr>
<tr>
<td>Anthralop bite</td>
<td>Scabies</td>
</tr>
<tr>
<td>Prebullous bullous pemphigoid</td>
<td></td>
</tr>
<tr>
<td>Spangiosis and exocytosis of neutrophils</td>
<td>Psoriasis</td>
</tr>
<tr>
<td>Fungal infection</td>
<td>Secondary syphilis (uncommon)</td>
</tr>
<tr>
<td>Atypical mononuclear cells</td>
<td>Mycosis fungoides</td>
</tr>
<tr>
<td>Langerhans cell histiocytosis</td>
<td>Cutaneous CD30 lymphoproliferative disorders</td>
</tr>
<tr>
<td>Single keratinocytes apoptosis</td>
<td>Drug reaction</td>
</tr>
<tr>
<td>Psoriasiform subacute lupus erythematosus</td>
<td></td>
</tr>
<tr>
<td>Pale keratinocytes</td>
<td>Nutritional dermopathies</td>
</tr>
</tbody>
</table>

BVs, blood vessels; LSC, lichen simplex chronicus; MF, mycosis fungoides; PLC, pityriasis lichenoides chronicus; PR, pityriasis rosea; PRP, pityriasis rubra pilaris; PVU, perivascular lymphocytic infiltrate; RBCs, red blood cells; +, present; –, absent.
Psoriasisform dermatitis with interface changes can be seen in drug reactions, cutaneous lupus erythematosus, and syphilis.

CONCLUSIONS
Inflammatory skin diseases are complex and create a diagnostic challenge for the pathologist. Knowledge of the clinical information, microanatomy of the skin, and the biological behaviour of various inflammatory dermatoses, in addition the use of a systematic approach during histological evaluation, are essential to narrow the differential diagnosis, thereby achieving the most accurate and appropriate diagnosis. An effort should be made to distinguish and exclude various entities that may simulate inflammatory skin conditions. These entities include stasis dermatitis, dermal haemorrhage and reactive endothelial cells, early Kaposi’s sarcoma with abundant plasma cells, cryoglobulinaemia, and mycosis fungoides. Special stains and immunohistochemical stains should be applied as needed.

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