Inflammatory Dermatopathology
This book is dedicated to my wife, Beth, and daughter, Maeve. I am ever grateful for all the joy you bring me and for your love and support.

Thank you:

I especially want to thank Drs. Yu-Hung Wu and Jasmin Jamora. This book would not have been written without your help. I would also like to thank my residents and fellows who always teach me as much as I teach them.

Steven D. Billings

To the three wonderful men in my life: my husband, Andrew and my sons Nathan and Jackson.

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Jenny Cotton

We would also both like to thank our mentors: Drs. Lawrence Roth, Thomas Ulbright, Evan Farmer, Sharon Weiss and William Moores. We especially thank Dr. Antoinette Hood for all her patient teaching, support, and enthusiasm.
When I was a first year resident, I emerged from my first dermatopathology conference with only one thought in mind: I would never study dermatopathology. It was all too confusing. Everything looked alike, and the terminology was impenetrable. Never say never. Somehow I was able to overcome that first shock with the help of my colleague Dr. Cotton and my mentor Dr. Antoinette Hood. Several years later, I am now charged with teaching dermatopathology to residents and fellows as well as CME courses for practicing pathologists and other dermatopathologists.

This brings us to this book. Clearly, there are other much more encyclopedic dermatopathology textbooks out there than this relatively thin tome. So, why this book? Probably because I keep running into residents and practicing pathologists who have the same reaction I had when I first encountered the subspecialty of dermatopathology. There is hope. Dermatopathology is hard, but it is not as hard as it is made out to be. A quick weekend of reading this book should help demystify inflammatory dermatopathology. It is meant to be a practical resource, a survival guide if you will, for surgical pathologists and residents for the approach of inflammatory diseases of the skin. Most of the entities commonly encountered in daily practice are covered in this book, with an emphasis on practical points that are useful in everyday practice. To increase the practical usefulness, we have also included a novel aspect. While most pathology texts do an admirable job describing histologic features and discussing differential diagnoses, writing the report is an art never discussed. Therefore, to increase the usefulness of the text, we have also included sample reports at the end of each chapter to provide examples on how we approach signing out our cases. We hope you enjoy this book. More importantly, we hope you find it useful in your practice.
## Contents

1. Introduction ......................................................... 1
2. Spongiotic Dermatitis ........................................... 5
3. Psoriasiform Dermatitis ......................................... 21
4. Interface Dermatitis ............................................. 37
5. Perivascular Dermatitis ......................................... 69
6. Vasculitis and Thrombotic Disorders ....................... 97
7. Nodular and Diffuse Dermatitis ............................. 119
8. Palisading Granulomatous Dermatitis .................... 133
9. Sclerosing Dermatitis ........................................... 145
10. Bullous Dermatitis ............................................. 157
11. Panniculitis ....................................................... 185
12. Infections ........................................................ 203
13. Miscellaneous Dermatoses: Invisible Dermatoses and Inflammatory Processes that Clinically Mimic Tumors ....... 233

Index ............................................................................. 247
Chapter 1
Introduction

Keywords Reaction pattern • Pathology report • Descriptive diagnosis

Dermatopathology is a hard subject and inflammatory dermatopathology is especially vexing. There is significant histologic overlap between the entities. The terminology can border on the impenetrable, and so, a specific diagnosis is often elusive. As a result we rely on diagnoses such as non-specific chronic dermatitis. Therein lies the problem. There is nothing that dermatologists or other clinicians hate more than the diagnosis of “non-specific chronic dermatitis.” It does not have to be this way. One can still make a descriptive diagnosis that is actually helpful to the clinician.

The key to interpreting biopsies of inflammatory dermatoses lies in understanding the concept of the basic reaction patterns. This book is generally organized according to these reaction patterns with some exceptions. Broadly speaking, most inflammatory dermatoses can be divided into two categories: epidermal and dermal patterns. In the epidermal patterns, there are three primary patterns: spongiotic, psoriasiform, and interface patterns. The spongiotic pattern is characterized by intraepidermal accumulation of edema fluid. The psoriasiform pattern is characterized by epidermal hyperplasia. The interface pattern is characterized by damage to the basal layer of the epidermis by an inflammatory infiltrate. The spongiotic and psoriasiform patterns frequently co-exist. Overlap with the interface pattern may also be seen.

The dermal patterns lack significant epidermal change. The dermal patterns can generally be divided into perivascular, nodular and diffuse, palisading granulomatous, and sclerosing patterns. As expected, the perivascular pattern demonstrates an inflammatory infiltrate predominantly around dermal blood vessels in a superficial, or superficial and deep distribution. In the nodular and diffuse pattern, the infiltrate is less vasculocentric. There may be significant overlap between perivascular and nodular and diffuse patterns. The palisading granulomatous pattern has an infiltrate that surrounds zones of altered collagen. Sclerosing dermatoses are characterized by fibrosis of the dermis, usually with relatively little inflammation.
As a general rule the epidermal patterns trump the dermal patterns. In other words, if there is significant epidermal change, the lesion belongs to one of the epidermal patterns, and not one of the dermal patterns. Within the epidermal patterns, the interface pattern trumps the other two epidermal patterns. One must be careful not to overinterpret basilar spongiosis as true interface change. In general, interface change shows at least focal evidence of keratinocyte destruction.

There are also special patterns that are unique unto themselves. Panniculitis does not belong to the aforementioned patterns, but is subdivided into septal and lobular patterns. Similarly bullous disease has is its own patterns, divided into subepidermal and intraepidermal patterns.

Knowledge of these patterns and the common entities in the patterns is crucial in creating a good pathology report. What makes up an ideal surgical pathology report of an inflammatory dermatosis? In our opinion, all reports from biopsies of inflammatory dermatoses require three elements: (1) diagnosis, (2) microscopic description, and (3) comment.

Obviously, a diagnosis is required for any report. When possible, it is important to provide a specific diagnosis. Unfortunately, a specific diagnosis is often not possible. In such cases, it is perfectly acceptable to provide a descriptive diagnosis. However, the descriptive diagnosis needs to be couched in the appropriate terms. In other words, the diagnosis needs to be framed using the reaction pattern that is present (e.g., spongiotic dermatitis) rather than overly general terms such as “chronic dermatitis.” What gives meaning to the descriptive diagnosis are the accompanying microscopic description and the comment section of the report.

As a specialty, pathologists are increasingly turning away from microscopic descriptions and it is becoming a lost art. However, it is still important to provide this in pathology reports for inflammatory skin diseases for a number of reasons. First and foremost, dermatologists as a general rule are relatively high-end consumers of pathology reports. Unlike some surgeons, they often read the entire report. They expect a microscopic description and are looking for key descriptive terms in the body of the report. Sometimes a microscopic description will provide additional insights into a case for the clinician and might even prompt consideration of alternate clinical possibilities. Another reason to provide a report is the nature of inflammatory processes in general. Inflammatory skin disease is dynamic. A particular entity may have a completely different appearance early in the course of the disease from what it looks like late in the disease process. Occasionally, multiple biopsies may be required, and the descriptive historical record can be helpful in deciphering the diagnosis. As a general rule, we incorporate the microscopic description as the first part of the comment section. Part of the reason for doing it this way is the layout of the report format we use. The choice in the construction of your report is up to you.

In the comment section of the report, especially in cases where a descriptive diagnosis is rendered, one should provide a differential diagnosis if possible and what is favored if possible. The comment section is frequently the most important section of the report. It is the pathologist’s chance to truly enter a dialog with the clinician. As mentioned above, we combine the microscopic
description with the comment section. The first half of the comment section is the microscopic description while the second half is the discussion of the case.

When constructing a report, we recommend brevity. In general, the microscopic description/comment section can be provided in a handful of sentences. Verbose language is rarely required. Remember the axiom that the more you write, the less the consumer of the report reads. Another tip for generating effective reports is good communication with the clinician. Too often, pathologists forget to use one of their most important tools: the telephone. Rarely does a day go by where we do not pick up a phone and call a contributor to seek additional information to clarify the clinical situation of a case. It must be remembered that clinicians rarely fill out the specimen requisitions. Often it is a nurse or assistant who fills out the form and certain key information can be missing. Furthermore, the physical space on the specimen requisitions may be too small to provide sufficiently detailed important information. A brief 5-minute phone call can often clear up these matters. It also helps build a working relationship with the clinician, a vital aspect of successful practice for any pathologist or dermatopathologist.

To provide additional guideline in the formation of effective reports, there are sample reports at the end of each chapter. These sample reports merely represent guidelines and not specific ‘report language’ that can be used in the readers’ reports. As always, one must assess each case individually and apply observations unique to the individual case.
Chapter 2
Spongiotic Dermatitis

**Keywords**  Spongiosis • Spongiotic dermatitis • Eczema • Atopic dermatitis • Contact dermatitis • Nummular dermatitis • Dyshidrotic eczema • Drug eruption • Mycosis fungoides • Pityriasis rosea • Stasis dermatitis • Psoriasiform dermatitis

**Spongiotic Dermatitis**

A variety of entities are in this group of inflammatory diseases. This chapter will focus on the group of entities encompassing the eczematous family of dermatitis, but also discuss other important and distinct diseases with spongiosis as its predominant finding.

The spongiotic reaction pattern is characterized by epidermal changes related to the accumulation of intraepidermal edema. The resulting hydrostatic forces cause separation of the keratinocytes revealing the intercellular desmosomal attachments. This appearance has been likened to the cut surface of a sponge, hence the term spongiotic (Fig. 2.1). The epidermal change in spongiotic dermatitis is a dynamic process that evolves over time. It can be divided into three phases: acute, subacute, and chronic. It should be recognized that these divisions are somewhat arbitrary and merely represent a means to conceptualize the histological changes.

**Acute Spongiotic Dermatitis**

This represents the earliest phase and consequently the least frequently biopsied phase. In the earliest timeframe the epidermis retains its normal basket-weave stratum corneum. The epidermis proper has variable amounts of spongiosis ranging from minimal to spongiotic microvesicles (Fig. 2.2). Spongiotic microvesicles are collections of edema fluid in the epidermis. They form when the hydrostatic pressure from the intraepidermal edema fluid is such that the intercellular junctions between keratinocytes...
are ruptured. Clinically, this can result in the appearance of blisters. In addition to the intraepidermal spongiosis, there is usually a superficial perivascular inflammatory infiltrate composed of a mixture of lymphocytes, some histiocytes, and often some eosinophils. In some cases, a few neutrophils may be present. The infiltrate is usually concentrated around the superficial vascular plexus, but the pattern of the infiltrate can be somewhat variable. In lesions with an intense infiltrate, it can have the appearance of a more lichenoid pattern. There may also be some extension of the inflammation into the mid-dermis. There is usually some exocytosis of inflammatory cells into the overlying epidermis, usually lymphocytes, but can be other inflammatory cells as well. The superficial dermis usually shows some edema in the earlier phases of the process (Table 2.1).
Subacute Spongiotic Dermatitis

One of the ways the epidermis reacts to inflammatory insults is by proliferation. This results in additional changes including acanthosis (hyperplasia) and parakeratosis (Fig. 2.3). In subacute spongiotic dermatitis the epidermis has had time to react to the inflammatory process. The epidermis shows variable parakeratosis and acanthosis. There is spongiosis, but it varies. There can be spongiotic microvesicles, but more often, the degree of spongiosis is less than what is seen in acute spongiotic dermatitis. Within the dermis, there is less edema, but otherwise a similar pattern of inflammation (Table 2.2).

Table 2.1  Acute spongiotic dermatitis: key microscopic features

- Normal basket-weave stratum corneum
- Epidermal spongiosis with or without spongiotic microvesicles
- Variable papillary derma edema
- Superficial perivascular inflammatory infiltrate of lymphocytes and often with admixed eosinophils

Subacute Spongiotic Dermatitis

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Table 2.2  Subacute spongiotic dermatitis: key microscopic features

- Parakeratosis
- Spongiosis
- Acanthosis
- Little to no papillary dermal edema
- Superficial perivascular inflammatory infiltrate of lymphocytes and often with admixed eosinophils
Chronic Spongiotic Dermatitis

In chronic spongiotic dermatitis, there is much less spongiosis. The spongiosis is minimal to mild in nature. In this phase, the reactive epidermal changes are more prominent (Fig. 2.4). There is compact hyperkeratosis, variable parakeratosis, thickening of the granular layer, and more pronounced acanthosis. The superficial dermis does not demonstrate evidence of edema and may be slightly fibrotic. The inflammatory infiltrate is less intense but otherwise composed of the same constituent cells (Table 2.3).

Fig. 2.4 Chronic spongiotic dermatitis. In chronic spongiotic dermatitis, there is compact hyperkeratosis with no or minimal parakeratosis. The epidermis is acanthotic and there is little to no apparent spongiosis. The papillary dermis may be fibrotic and there is a variable, usually mild, superficial perivascular infiltrate.

Table 2.3 Chronic spongiotic dermatitis: key microscopic features

- Compact hyperkeratosis and variable parakeratosis
- Acanthosis
- Minimal spongiosis
- Superficial perivascular inflammatory infiltrate of lymphocytes and often with admixed eosinophils. Usually mild in nature
- Variable superficial dermal fibrosis

Overlap With Psoriasiform Pattern

In subacute and chronic spongiotic dermatitis, the acanthosis of the epidermis can cause significant overlap with the psoriasiform pattern (Chap. 3). This issue is primarily an issue in construction of the pathology report and will be dealt with at the end of the chapter.
Eczematous Dermatitis

The group of inflammatory disorders in the eczematous family of skin diseases includes a wide range of entities including, atopic dermatitis, nummular dermatitis, contact dermatitis (both allergic and irritant contact dermatitis), dyshidrotic dermatitis (pompholyx), id reaction, and eczematous drug eruptions. Here is one of the secrets of dermatopathology: all of these entities are essentially histologically identical. They all can demonstrate the three patterns of spongiotic dermatitis depending on when the lesion is biopsied. With some of the entities, there can be clues to the diagnosis histologically, but clinical information is often crucial to the diagnosis. With that in mind, it is important to review some of the clinical aspects of these diseases.

Atopic Dermatitis

Atopic dermatitis is a chronic, relapsing, pruritic dermatitis in patients with a familial history of atopy. Atopy is characterized by variable combinations of dermatitis, asthma, sinusitis, and allergic rhinitis. In children, the eruption favors flexural areas such as the antecubital fossa. In adults, the presentation is more variable including very mild periorbital dermatitis to full body erythroderma.

Contact Dermatitis

Contact dermatitis is a result of an exogenous stimulus and can be subdivided into allergic or irritant types. Allergic contact dermatitis is the result of a type IV hypersensitivity reaction that requires exposure to a specific antigen. The prototypical allergic contact dermatitis includes reactions to substances such as poison ivy or latex. Nickel allergies are also common and tend to present where people come into contact with the metal (e.g., earlobes, waistline near blue jeans snaps).

Irritant contact dermatitis results from direct damage to the epidermis from the offending substance rather than an immune mediated response. Detergents are one of the most common causes of irritant dermatitis (so-called dishpan hands). Diaper rash is another prototypical example.

Histologically, both show features of spongiotic dermatitis. Clinically, there are frequent clues to the diagnosis. For example, in poison ivy, the eruption often has a linear arrangement corresponding to the edge of the offending leaf that brushed along the skin. Depending on the offending agent, there may be peculiar distributions such as with allergic reactions to latex gloves or nickel containing jewelry. A histological clue to the diagnosis of allergic contact dermatitis is the presence of Langerhans cell microabscesses within the epidermis (Fig. 2.5) (not to be confused with the Pautrier’s microabscesses of mycosis fungoides which are composed of neoplastic lymphocytes). Langerhans cell microabscesses are not always present in allergic contact dermatitis, and they are not entirely specific. In irritant contact dermatitis,
**Fig. 2.5** *Langerhans cell microabscess.* Allergic contact dermatitis often has Langerhans cell microabscesses, characterized by collections of Langerhans cells within the spongiotic epidermis. Langerhans cells have reniform nuclei and relatively abundant pale eosinophilic cytoplasm.

**Fig. 2.6** *Irritant contact dermatitis.* Within the upper epidermis there are scattered dyskeratotic cells. This is a common but nonspecific finding in irritant contact dermatitis.

the inflammatory infiltrate tends to be less intense and there may be ballooning degeneration of keratinocytes and/or occasional dyskeratotic keratinocytes in the epidermis, especially the upper half of the stratum spinosum (Fig. 2.6).

**Nummular Dermatitis**

This is one of the most common types of spongiotic dermatitis that is biopsied. Nummular dermatitis is characterized by round (coin-shaped) to oval patches variably composed of papules and vesicles usually on the extremities. As the eruption evolves, there may be central clearing, clinically resembling dermatophyte infection.
(tinea). In patients with atopic dermatitis, they may have flares of their disease presenting as nummular dermatitis. Nummular dermatitis almost always has a component of epidermal acanthosis. Microscopically, it typically has the features of subacute or chronic spongiotic dermatitis. Clinically and histologically the differential diagnosis of nummular dermatitis is psoriasis vulgaris.

**Dyshidrotic Eczema (Pompholyx or Palmoplantar Dermatitis)**

Dyshidrotic eczema is characterized by a recurrent pruritic, often vesicular, eruption of the palms, soles, or digits. Clinically, the vesicles have a papular appearance. Over time, scaling and cracking can develop. In many patients, this is a manifestation of atopy. A significant proportion of dyshidrotic eczema is the result of an allergic contact dermatitis. Spongiotic vesicles are a very common histologic feature. It is important to always exclude dermatophyte infection, especially in eruptions from the feet. A PAS or GMS stain is recommended to exclude the possibility of an underlying fungal infection.

**Id Reactions (Autoeczematization)**

Id reactions are the development of an eczematous dermatitis in regions away from the primary inflammatory focus. Dermatophyte infections of the feet (tinea pedis) and stasis dermatitis are two of the most common inciting conditions for id reactions. The patient can develop eczematous dermatitis on the upper extremities, or trunk, far away from the triggering process. In the case of dermatophyte-triggered eczematous dermatitis, no fungi are detectable in the dermatitis representing the id reaction. The id reaction component is difficult to treat without addressing the underlying trigger.

**Eczematous Drug Reactions**

Drug reactions will be dealt with in more detail in a later section of the book. A minority of drug reactions may be histologically indistinguishable from other forms of eczematous dermatitis. Depending on what sources you read, eczematous drug eruptions can account for <5–10% of all new drug eruptions. In our personal experience, it is relatively uncommon, and the rate is <5%. Association with new medications can help correlation with the diagnosis. In the absence of clinical information implicating a medication, it is not possible to differentiate an eczematous drug reaction from other eczematous dermatitides.
Differential Diagnosis

Eczematous dermatitis is frequently secondarily impetiginized resulting in neutrophils in the stratum corneum. This is also a key feature of a dermatophyte infection. When neutrophils are present in the stratum corneum or upper epidermis, a PAS or GMS should be performed to exclude a possible fungal infection.

Nummular dermatitis and psoriasis may have significant clinical overlap and differentiating these entities is often a diagnostic problem. Nummular dermatitis has more edema fluid in the stratum corneum, less uniform hyperplasia, a retained or thickened granular layer and usually has eosinophils in the dermal infiltrate. These are not features of psoriasis (see Chap. 3).

A minority of cutaneous drug eruptions is eczematous (spongiotic) in nature. They can be indistinguishable from other forms of eczematous dermatitis. Diagnosis requires good correlation with medication history. Unfortunately, some drug eruptions commence months after initiation of a new medication. In that case it is ultimately up to the clinician to sort out the diagnosis; it is beyond the scope of histology in that situation.

Mycosis fungoides can figure into the differential diagnosis of eczematous dermatitis. A detailed discussion of mycosis fungoides is beyond the scope of this text, but references are provided at the end of this chapter. That being said, the differential diagnosis of mycosis fungoides vs. an eczematous dermatitis is relatively common. Lesions of mycosis fungoides have disproportionate amount of intraepidermal lymphocytes within a relatively non-spongiotic epidermis (Fig. 2.7). The intraepidermal lymphocytes have a halo artifact and frequently tag the basal layer of the epidermis. The neoplastic lymphocytes tend to have more irregular, cerebriform nuclei, but this is less helpful in actual practice. Immunostains and clonality studies can also be helpful. A shift in the ratio of CD4 to CD8 positive cells of >4–6:1 in the appropriate context favors mycosis fungoides over eczematous

![Fig. 2.7 Mycosis fungoides](image)

Mycosis fungoides is characterized by epidermotropism of lymphocytes into the epidermis that is disproportionate to the amount of spongiosis
Other Forms of Spongiotic Dermatitis

Stasis dermatitis and pityriasis rosea are also in the differential diagnosis. See below for discussion of these entities.

Stasis Dermatitis

Clinical Features

Stasis dermatitis typically presents on the medial aspect of the lower extremities in association with evidence of venous insufficiency. It usually presents in older patients or obese patients. Usually, stasis dermatitis presents as pruritic, scaly plaques. Rarely it presents as a more circumscribed process clinically, and can be confused with a neoplasm. Acroangiodermatitis, a specific form of stasis dermatitis, presents as violaceous macules, nodules or plaques on the dorsal feet. It can be clinically and histologically confused with Kaposi’s sarcoma.
**Microscopic Features**

The epidermis shows features of subacute or chronic spongiotic dermatitis as described above. The key differentiating features are found in the dermis. Within the papillary dermis, there is a lobular proliferation of relatively thick-walled vessels (Fig. 2.8). There may be evidence of tissue edema or, in long-standing cases, fibrosis. There is extravasation of erythrocytes and associated perivascular siderophages to varying degrees. A perivascular lymphocytic infiltrate is present. The infiltrate is variable, but is usually less intense than the infiltrate of the eczematous dermatitides described above (Table 2.5).

**Fig. 2.8** *Stasis dermatitis.* (a) The epidermis shows variable spongiosis and acanthosis. Within the dermis there is a lobular proliferation of relatively thick-walled blood vessels and a perivascular lymphocytic infiltrate. (b) Higher power view of papillary dermal blood vessels with surrounding siderophages, a frequent finding in long-standing cases of stasis dermatitis.
Other Forms of Spongiotic Dermatitis

**Differential Diagnosis**

The differential diagnosis of most cases includes the forms of spongiotic dermatitis in the eczematous dermatitis group outlined above. In some cases, there may be a combination of eczematous dermatitis superimposed on underlying stasis change. In rare cases, the vascular proliferation is so prominent as to mimic a vascular neoplasm such as Kaposi’s sarcoma. This form of stasis dermatitis is referred to as acroangiodermatitis (Fig. 2.9). Careful attention to histological features allows distinction. Acroangiodermatitis does not have the dense proliferation of spindled endothelial cells with slit-like vascular spaces, lacks the promontory sign of early Kaposi’s sarcoma and does not express latent nuclear antigen of HHV-8. Recognition of more conventional areas of stasis change can be helpful (Table 2.6).

![Acroangiodermatitis](image)

*Fig. 2.9 Acroangiodermatitis.* The reactive vascular proliferation due to stasis can sometimes be quite prominent and cause confusion with a vascular neoplasm.

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**Table 2.5** Key microscopic findings: stasis dermatitis

- Variable acanthosis and spongiosis
- Lobular proliferation of relatively thick-walled vessels in superficial dermis
- Extravasation of erythrocytes and siderophages common

**Table 2.6** Practical tips: stasis dermatitis

- Keep a high index of suspicion on biopsies from the lower legs
- The vascular changes are the most important feature
- Patients can have an eczematous dermatitis superimposed on underlying stasis change
- Occasionally stasis dermatitis can clinically mimic a neoplasm and the clinician may submit with a clinical diagnosis of squamous cell carcinoma
**Pityriasis Rosea**

**Clinical Features**

Pityriasis rosea usually presents in young adults, though a wide age range may be affected. The eruption starts as a salmon-colored herald patch that in the next 7–14 days is followed by a widespread, symmetric eruption of numerous small pink to red scaly plaques. The eruption usually starts on the trunk and then spread to the abdomen and proximal extremities.

**Microscopic Features**

The most characteristic feature of pityriasis rosea is the presence of discrete mounds of parakeratosis in the stratum corneum (Fig. 2.10). The epidermis shows mild spongiosis and mild acanthosis. Within the dermis, there is a superficial perivascular lymphocytic infiltrate. Eosinophils are rarely present. There may be extravasation of erythrocytes in the papillary dermis and exocytosis of erythrocytes into the overlying epidermis and, when present, is a helpful feature (Table 2.7).

![Fig. 2.10 Pityriasis rosea](image)

*Fig. 2.10 Pityriasis rosea. In the stratum corneum, there are discrete mounds of parakeratosis. The epidermis is mildly spongiotic and acanthotic. The dermal infiltrate is predominantly composed of lymphocytes. Extravasation of erythrocytes is commonly seen in the papillary dermis, and frequently there is exocytosis of erythrocytes into the epidermis.*

**Table 2.7 Key microscopic findings: pityriasis rosea**

- Discrete mounds of parakeratosis
- Spongiosis
- Papillary dermal hemorrhage common
- Mild perivascular lymphocytic infiltrate

**Differential Diagnosis**

The differential diagnosis includes subacute spongiotic dermatitis and it is often not possible to distinguish without clinical history. Guttate psoriasis (see Chap. 3) also resembles pityriasis rosea. Classically, guttate psoriasis has mounds of parakeratosis.
surmounted by collections of neutrophils. Neutrophils are not a feature of pityriasis rosea (Table 2.8).

**Table 2.8** Practical tips: pityriasis rosea

- Discrete mounds of parakeratosis are the key histologic feature
- A specific diagnosis of pityriasis rosea is not possible without the appropriate clinical history
- In the absence of a sufficient history, the case should be signed out descriptively as “spongiotic dermatitis” (see sample reports at the end of the chapter)

**Vesicular Dermatophytosis**

Dermatophyte infections can sometimes present with prominent spongiosis. Usually, there are neutrophils in the stratum corneum and eosinophils are found as part of the dermal infiltrate. Dermatophyte infection will be discussed in more detail in Chaps. 3 and 12. As a general rule, a PAS or GMS stain should always be considered when examining a spongiotic dermatitis involving the feet.

**Sample Reports: Spongiotic Dermatitis NOS (Eczematous Dermatitis)**

Example 1:

**Clinical history:** Rule out psoriasis.
**Diagnosis:** Spongiotic dermatitis, see comment.
**Comment:** The epidermis shows parakeratosis with irregular acanthosis, a maintained granular layer and diffuse mild spongiosis. Within the dermis, there is a superficial perivascular mixed inflammatory infiltrate of lymphocytes and scattered eosinophils. The degree of spongiosis, intact granular layer, and presence of eosinophils in the infiltrate argue against the possibility of psoriasis. The histological features are most consistent with an eczematous dermatitis such as nummular dermatitis. Clinicopathologic correlation is recommended.

Example 2:

**Clinical history:** Rule out dermatitis, drug eruption, etc.
**Diagnosis:** Spongiotic dermatitis, see comment.
**Comment:** The epidermis shows parakeratosis, some hyperkeratosis, spongiosis, and occasional Langerhans cell microabscesses. Within the dermis, there is a superficial predominantly perivascular mixed infiltrate of lymphocytes and eosinophils. The histological features are compatible with an eczematous dermatitis. The presence of Langerhans cell microabscesses in the epidermis