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Dermatopathology

Clinicopathological Correlations

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For all my co-authors who did another amazing job with this book project. Also to Jerad and especially Talley for their late inning heroics in dragging this book across the finish line.

– Brian Hall

For my husband, Joseph

– Margaret Merola

I would like to thank my incredibly supportive and beautiful wife (Clarissa) whose sacrifices have allowed me to work on this endeavor, as well I would like to dedicate this book to her, my sons Carter and Clark (soon to be), and the memory of my late parents Don and Joyce.

– Chad Jessup

To Leann, Mackenzie, and Cayden: Thank you for enduring my academic pursuits and supporting me every step of the way. I love you all so much!

– Cary Chisholm

I dedicate this book to the memory of my great teachers including Thomas Fitzpatrick, Wallace Clark, Richard Reed, Ramzi Cotran, Hal Dvorak and the fellows/trainees I have taught over the years, as well as the future residents/ trainees that will learn from this book.

– Martin Mihm

To my former teacher and mentor, A. Bernard Ackerman.

– Clay Cockerell

Preface

It is often stated that a picture says a thousand words. In the original planning of this book, it was our goal to include hundreds of pictures in addition to clinical and histologic descriptions so as to signify a book that is greater than the sum of its individual parts. With over 1,000 images, we feel that we have accomplished our goal in this first edition. Each entity that is covered contains not only high-yield clinical information, but also high-yield, simplified histologic features, ancillary studies as well as differential diagnoses for each entity. As with each significant scholarly undertaking, an immense amount of effort was put forth not only by the authors on the cover of this book, but also by several other people, but especially Talley Whang and Jerad Gardner. This book would not have been possible without their significant contributions in the late stages of this book.

A famous pathologist once stated, "If it isn't in your differential diagnosis, you will miss it everytime." In holding true to that adage, we have included not only very common entities, but some much rarer ones as well, so that the in training dermatologist or pathologist who has a strong interest in dermatopathology will hopefully be confident enough after mastering this book to be comfortable not only with the extremely common entities, but also the less common entities that tend to sneak across our scopes from time to time and sometimes catch us off guard.

It is our hope that this work of science combined with the natural art of cutaneous pathology will provide an extremely valuable resource not only to the budding pathologist or dermatologist studying for their mock exams, in service exams, general boards, or subspecialty boards, but also serve as a valuable resource to all general pathologists, general dermatologists, and dermatopathologists that sign out dermatopathology on a regular basis. We hope that the reading and studying of this book brings the same enjoyment that the consummation of this large effort brought to all of us that took part in creating it and that it may serve as a valuable tool to help all of us become better diagnosticians and better physicians for our patients.

Dallas, TX, USA — Brian J. Hall, MD

Contents

Part I

Inflammatory Dermatopathology

Interface (Lichenoid) Dermatoses

1

Lichen Planus (LP)

Clinical: Commonly remembered as the "five P's" – **purple, polygonal, planar, and pruritic papules** (and/or plaques) with a fine overlying scale containing thin white lines (known as **Wickham's striae**). LP commonly manifests as grouped papules on the distal extremities and/or trunk, also with frequent involvement of oral mucosa.

Hypertrophic Lichen Planus – Typical inflammatory pattern with marked acanthosis of the epidermis which may mimic prurigo nodule or keratoacanthoma.

Lichen Planopilaris – Arises on the scalp and causes a scarring alopecia (discussed more in detail in the alopecia chapter).

Histopathology: Compact orthokeratotic hyperkeratosis except for oral lesions which may have parakeratosis (Fig. 1.1). Classically shows **wedge-shaped hypergranulosis** and irregular acanthosis of the epidermis ("**saw-toothed**" **rete ridges**). Underlying the epidermis is a **band-like** lymphocytic infiltrate which extends into the superficial epidermis causing **vacuolar interface change**. **Colloid or civatte bodies** can be found in the areas of liquefaction degeneration and sometimes in the superficial dermis. In more advanced lesions, the epidermis may become somewhat atrophic, and there may be a significant degree of pigment incontinence with dermal melnophages.

Ancillary Studies: Direct immunofluorescence studies may show positive IgM, complement, and fibrin in the **colloid bodies**.

Differential Diagnosis: benign lichenoid keratosis, lichenoid actinic keratosis, halo nevus, lichenoid drug eruption, lupus erythematosus, erythema multiforme.

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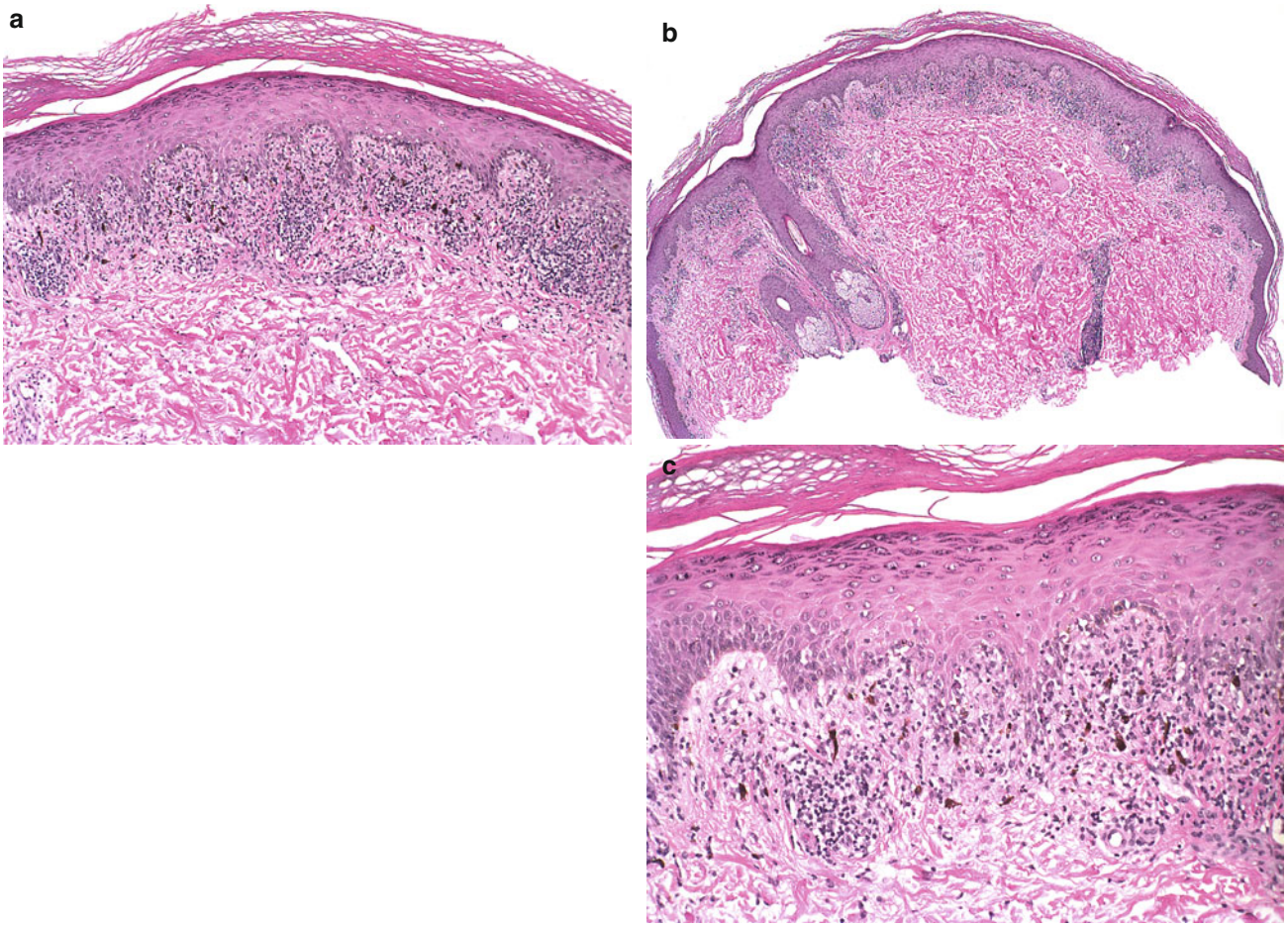

Fig. 1.1 (**a**) There is a lichenoid lymphocytic infiltrate with saw-toothed rete ridges. There is also confluent orthokeratosis (hematoxylin and eosin, 10×). (**b**) Lichen planus is the prototype for lichenoid infiltrates. There is a superficial band-like lymphocytic infiltrate which obscures the dermoepidermal junction (hematoxylin and eosin, 4×). (**c**) Pigment incontinence and melanophages are frequently seen. (hematoxylin and eosin, 20×)

Benign Lichenoid Keratosis (Lichen Planus-Like Keratosis)

Clinical: Usually a solitary erythematous or violaceous papule which may arise on the arms or trunk. Patients may complain of pain, burning, or pruritis.

Histopathology: Nearly identical to lichen planus, except it is a **solitary lesion** (Fig. 1.2). BLK represents a lichenoid immune reaction to a previously existing lesion (like seborrheic keratosis or solar lentigo). If there is keratinocytic atypia, then a dysplastic lesion (such as lichenoid AK) must be excluded. A careful search for a melanocytic proliferation should be negative.

Differential Diagnosis: Lichen planus, lichenoid actinic keratosis, halo nevus, lichenoid drug eruption, lupus erythematosus, erythema multiforme.

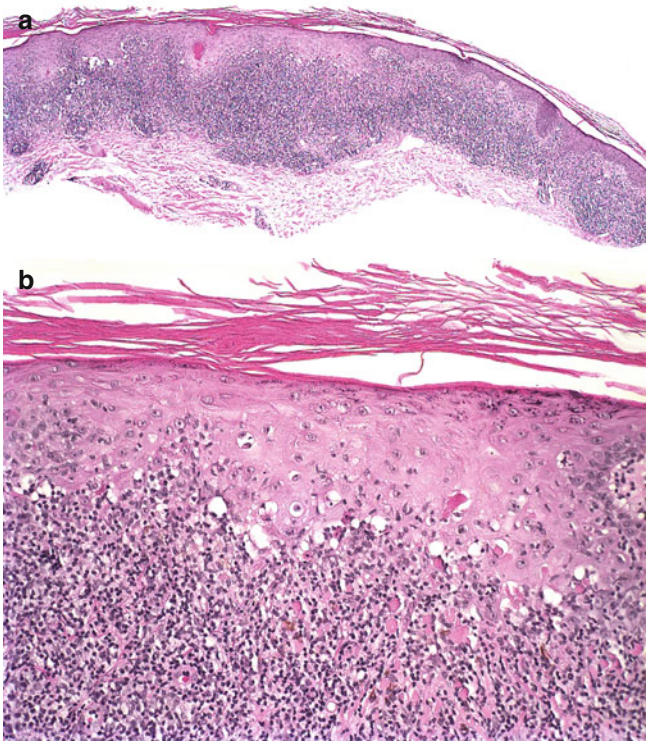

Fig. 1.2 (**a**) A lichenoid keratosis has a lichenoid lymphocytic infiltrate with saw-toothed epidermis at low power (hematoxylin and eosin, 4×). (**b**) Civatte bodies, confluent orthokeratosis, a lichenoid lymphocytic infiltrate, and an irregular dermoepidermal junction can be seen. (hematoxylin and eosin, 20×)

Lichen Striatus

Clinical: Usually a childhood disease which manifests as a **linear plaque** with overlying scale. There may be hypopigmentation or hyperpigmentation. Typically resolves after several months.

Histopathology: The epidermis is acanthotic or psoriasiform with overlying hyperkeratosis with or without focal parakeratosis (Fig. 1.3). Spongiosis is a frequent finding. As in other lichenoid eruptions, there is **vacuolar interface degeneration** with **dyskeratotic keratinocytes**. The lymphocytic infiltrate is more perivascular, perifollicular, and, characteristically **syringocentric** (around the sweat ducts).

Differential Diagnosis: Lichen planus, benign lichenoid keratosis, lichenoid actinic keratosis, halo nevus.

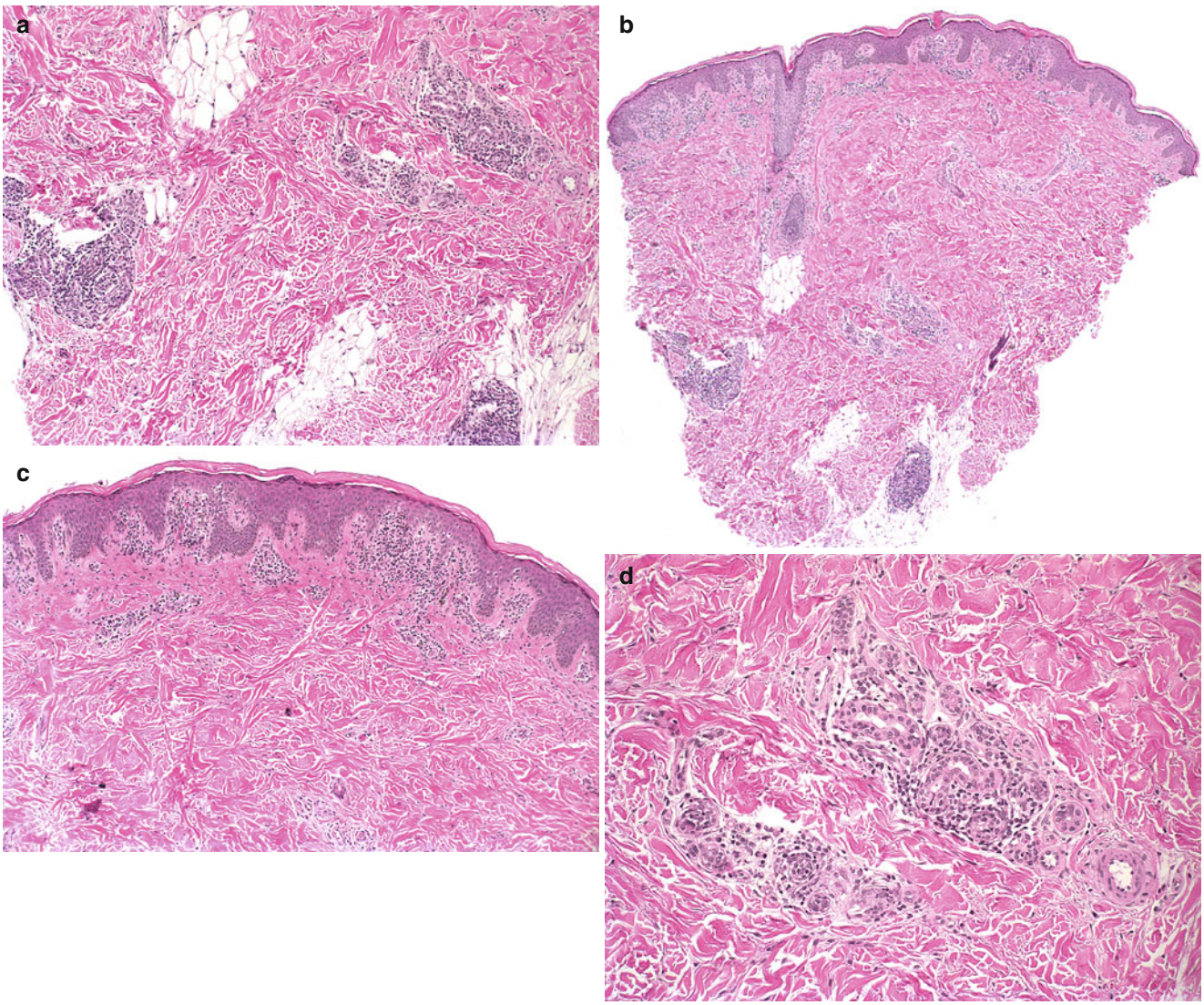

Fig. 1.3 (**a**) The inflammation in lichen striatus also involves the underlying sweat glands in addition to the overlying lichenoid infiltrate (hematoxylin and eosin, 10×). (**b**) There is a lichenoid lymphocytic infiltrate which also extends down to the sweat glands (hematoxylin and eosin, 4×). (**c**) There is a lichenoid lymphocytic infiltrate superficially (hematoxylin and eosin, 10×). (**d**) This is a close up view of the lymphocytic infiltrate within the sweat glands. (hematoxylin and eosin, 20×)

Lichen Nitidus

Clinical: Typically a childhood eruption of innumerable 1–2 mm skin-colored papules on the trunk and/or penis. Resolves after several months.

Histopathology: Dense lymphohistiocytic infiltrate in the superficial dermis which is localized or patchy (Fig. 1.4). The overlying epidermis is usually atrophic with **vacuolar interface change** and lymphocytic infiltration. The epidermal retia are elongated around the lymphohystiocytic infiltrate forming a **collarette** – the so-called "**ball in claw**" lesion. Multinucleated giant cells may also be present.

Differential Diagnosis: Lichen planus, benign lichenoid keratosis, lichenoid actinic keratosis, halo nevus.

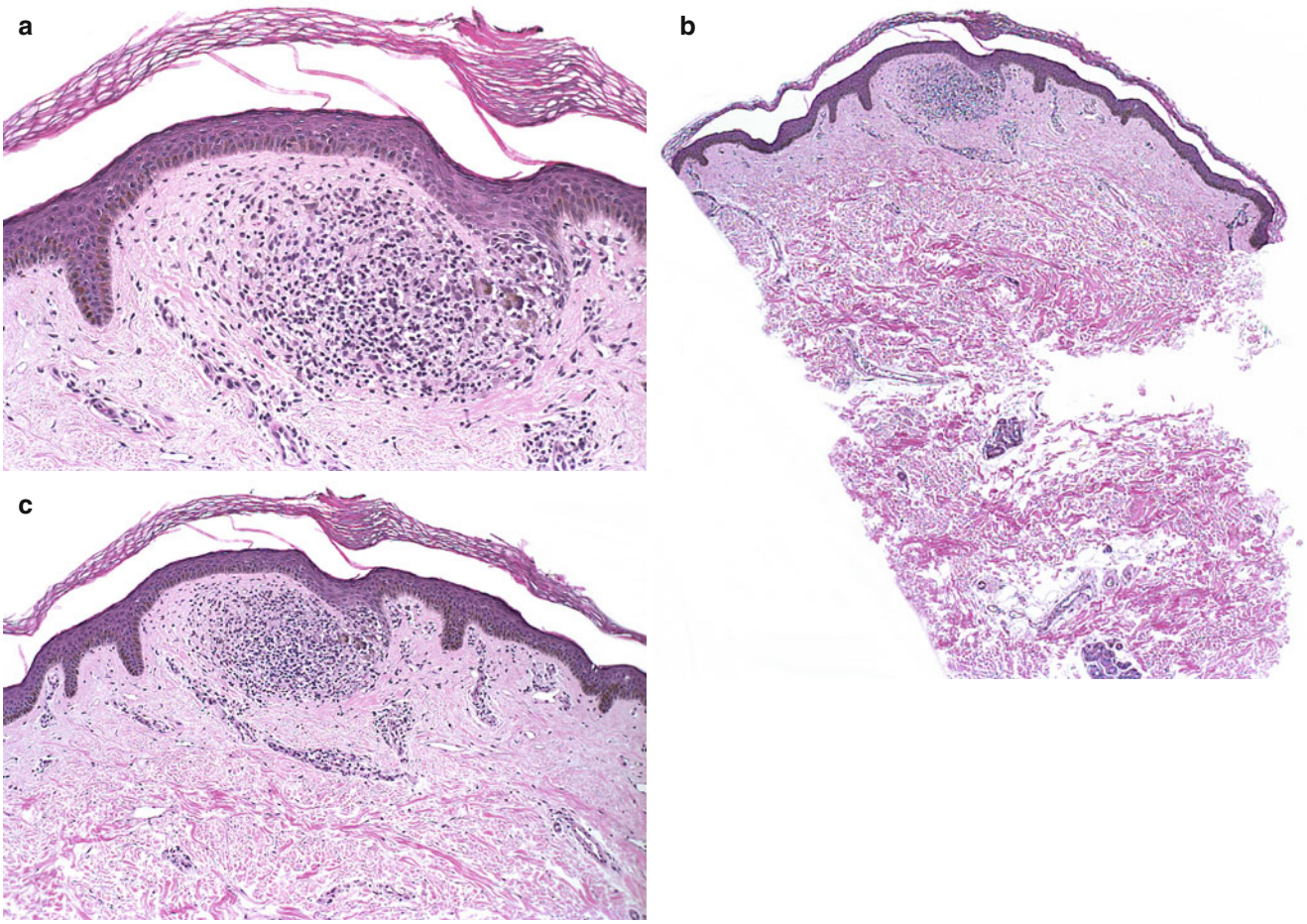

Fig. 1.4 (**a**) The epidermal retia are beginning to extend around the granuloma. This is the beginning of the "ball and clutch" that is typical of lichen nitidus (hematoxylin and eosin, 10×). (**b**) There is a small granuloma located immediately beneath the epidermis (hematoxylin and eosin, 4×). (**c**) The epidermal retia are beginning to extend around the granuloma. This is the beginning of the "ball and clutch" that is typical of lichen nitidus. (hematoxylin and eosin, 10×)

Lichenoid Drug Eruption

Clinical: Numerous medications, chemicals, and some occupational exposures have been reported to cause lichenoid drug eruptions. It may be similar clinically to lichen planus, or it may present in a sun-exposed distribution. Withdrawal of the offending medication usually leads to resolution.

Histopathology: Very similar to lichen planus or benign lichenoid keratosis, except **eosinophils** and sometimes plasma cells are scattered throughout the inflammatory infiltrate (Fig. 1.5). The overall infiltrate is typically **less dense** and not quite as band-like as in lichen planus. There is usually also overlying parakeratosis, and the vacuolar interface change may be less prominent. Apoptotic cells may be found higher in the epidermis than in lichen planus. Melanin incontinence is usually marked.

Differential Diagnosis: Arthropod assault, lichen planus, benign lichenoid keratosis, fixed drug eruption.

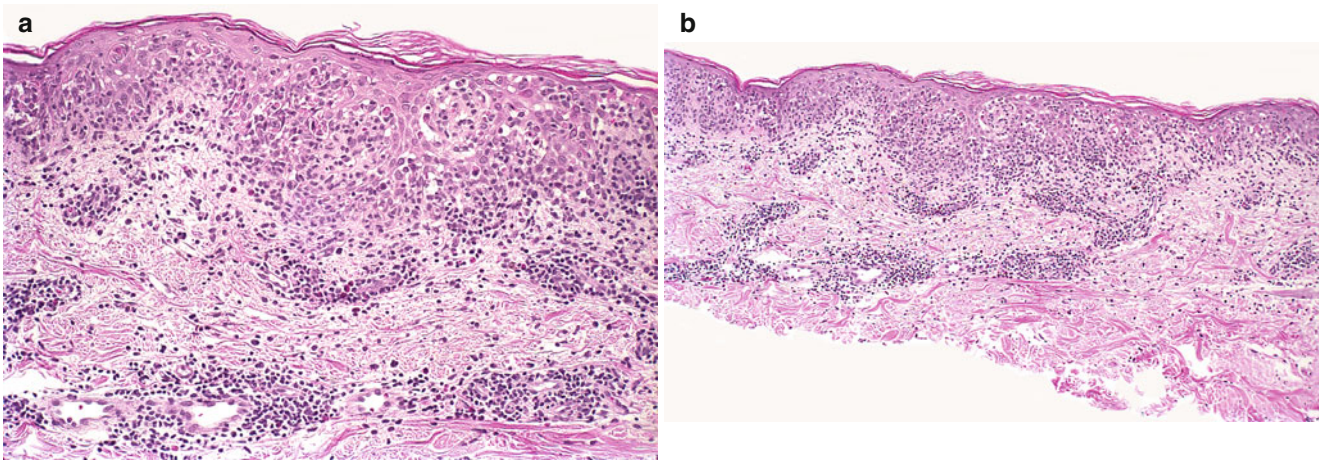

Fig. 1.5 (**a**) At higher power, numerous eosinophils can be identified within the infiltrate (hematoxylin and eosin, 20×). (**b**) This lichenoid process mirrors lichenoid dermatitis, lichen planus, and lichenoid keratosis histologically at low power. (hematoxylin and eosin, 10×)