# Chapter 4
# Interface Dermatitis

**Keywords** Interface dermatitis • Lichen planus • Lichenoid drug eruption • Fixed drug eruption • Morbilliform drug eruption • Erythema multiforme • Stevens–Johnson syndrome • Toxic epidermal necrolysis • Lupus erythematosus • Dermatomyositis • Graft vs. host disease • Pityriasis lichenoides • PLEVA

Interface dermatitis is characterized by damage to the epidermis from the inflammatory infiltrate. Microscopically, this is characterized by basal vacuolization with or without necrotic keratinocytes. Interface dermatitis can be broadly grouped into two subgroups based on the pattern of the inflammatory infiltrate: (1) lichenoid, or band-like, in which, the infiltrate forms a dense layer parallel to the overlying epidermis (Fig. 4.1) and (2) perivascular, in which the infiltrate is concentrated around blood vessels in either a superficial or superficial and deep distribution (Fig. 4.2).

## Interface Dermatitis with Lichenoid Infiltrate

### *Lichen Planus*

#### Clinical Features

Lichen Planus is the prototypical lichenoid interface dermatitis. Lichen planus usually presents in adults as pruritic, polygonal violaceous papules. There is a predilection for extensor surfaces of the wrists and ankles, but the eruption may be widespread. Lichen planus involves the oral mucosa, especially the buccal mucosa, in about 60% of patients. In the oral cavity, lichen planus has as a reticulated, lace-like appearance; erosions and ulceration can also occur.

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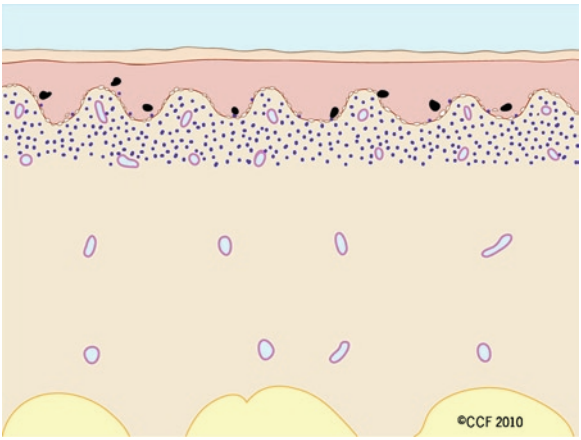


**Fig. 4.1** *Schematic representation of interface dermatitis with lichenoid pattern*. This pattern of interface dermatitis is characterized by basal vacuolization with scattered dyskeratotic keratinocytes and a band-like, or lichenoid, inflammatory infiltrate

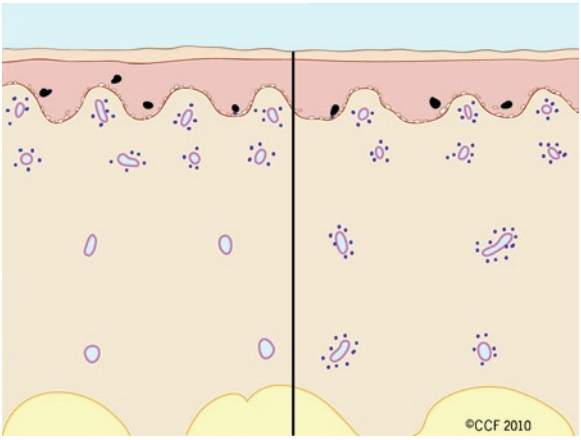


**Fig. 4.2** *Schematic representation of interface dermatitis with a perivascular pattern of inflammation*. This pattern can be roughly divided into those that have a predominantly superficial or superficial and deep infiltrate in addition to the interface change

## *Microscopic Features*

In cutaneous lichen planus, the stratum corneum shows compact hyperkeratosis, but not parakeratosis. The granular layer is thickened, often with a wedge-shaped pattern. The epidermis may show mild acanthosis. Within the dermis, there is a dense band-like pattern of mononuclear cells predominantly composed of lymphocytes (Fig. 4.3). Some admixed histiocytes may be present, but eosinophils are typically not seen. There is interface change manifested by basal vacuolization, exocytosis of lymphocytes and necrotic keratinocytes. These dyskeratotic cells may have lymphocytes "tagging" the keratinocytes, so-called satellite cell necrosis (Fig. 4.4). There may also be eosinophilic globules in the superficial dermis representing keratinocytes that have "dropped out" of the epidermis that are also referred to as Civatte bodies. As the epidermal damage evolves, the rete pegs lose their normal undulating pattern and take on a saw-tooth configuration (Table 4.1).

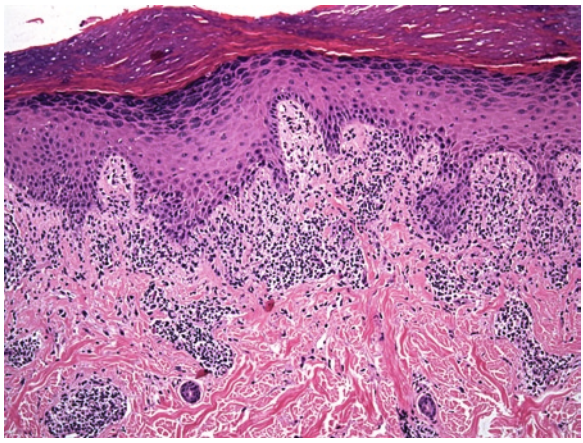

**Fig. 4.3** *Lichen planus* is characterized by compact hyperkeratosis without parakeratosis, a thickened granular layer and variable acanthosis. The rete pegs have an irregular sawtooth configuration and there is a dense lichenoid lymphocytic infiltrate with basal vacuolization and dyskeratotic cells

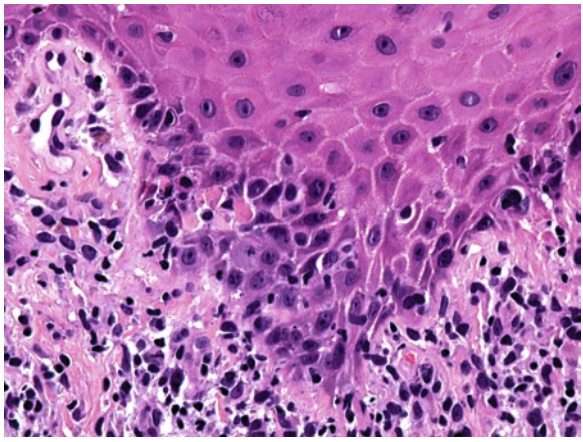

**Fig. 4.4** *Lichen planus*. This high power image of the interface change demonstrates the lymphocytic infiltrate and the scattered dyskeratotic keratinocytes in the epidermis

**Table 4.1** Key microscopic features: lichen planus

- Compact hyperkeratosis without parakeratosis
- Thickened granular layer
- Lichenoid infiltrate
- Interface change with basal vacuolization, dyskeratotic keratinocytes and saw-tooth pattern of dermoepidermal junction
- Typically no eosinophils

There are two histologic variants of cutaneous lichen planus to be aware of: hypertrophic and atrophic. In hypertrophic lichen planus, there is significant epidermal acanthosis in conjunction with other histologic findings of lichen planus (Fig. 4.5). In part, the epidermal hyperplasia may be the result of persistent excoriation as seen in lichen simplex chronicus and prurigo nodularis. Unlike typical lichen planus, eosinophils can be seen occasionally in hypertrophic lichen planus, but they should

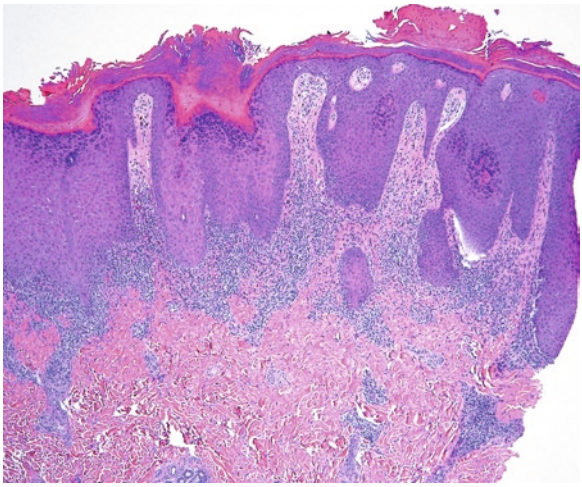

**Fig. 4.5** *Hypertrophic lichen planus*. This variant of lichen planus resembles conventional lichen planus but with marked acanthosis

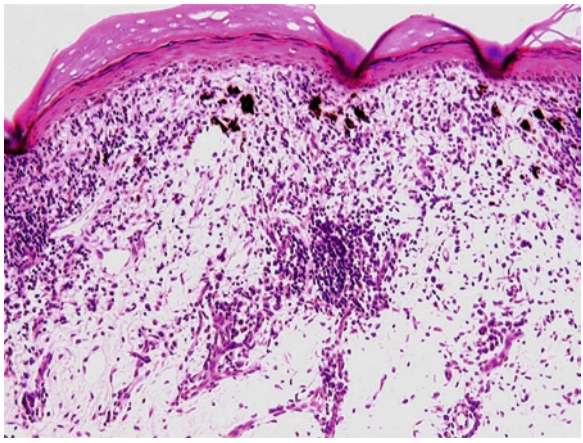

**Fig. 4.6** *Atrophic lichen planus*. In atrophic lichen planus the epidermis in thinner than normal, and the interface change is subtler. Melanophages are frequently present in the dermis reflecting chronic damage to the epidermis

be sparse in number. In atrophic lichen planus, the epidermis is thinned and there is less pronounced hyperkeratosis and hypergranulosis (Fig. 4.6). Within the dermis, the infiltrate is frequently less intense, and there may be scattered melanophages. Atrophic lichen planus, in many cases, represent a burned out or resolving lesion of lichen planus.

In oral lichen planus, the findings are often more subtle. There is less hyperkeratosis, and, in contrast to cutaneous lichen planus, there may be some parakeratosis (Fig. 4.7). Frequently there is a subtle hypergranulosis characterized by a few keratohyaline granules in the superficial epidermis. Recognition of the granular layer may require examination at high power. Within the subepithelial stroma, there is a band-like infiltrate of lymphocytes admixed with plasma cells. Rare eosinophils may also be seen. Prominent saw-toothing is usually not present, and the degree of interface change may be milder in nature.

In lichen planus, there are characteristic, if not entirely specific, direct immunofluorescence findings. The most characteristic finding is shaggy deposition of

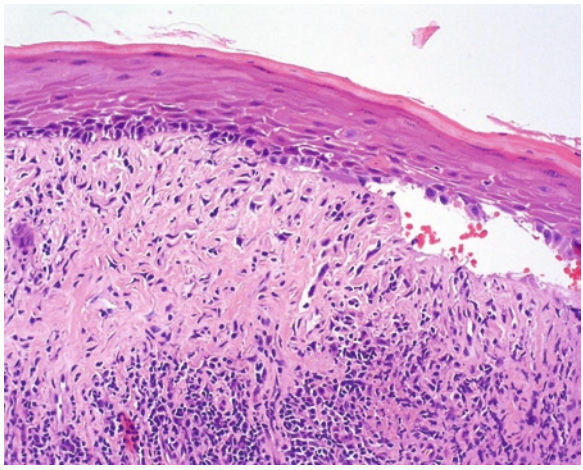

**Fig. 4.7** *Oral lichen planus*. The epithelial changes are more subtle than in conventional lichen planus. There is hyperkeratosis and subtle evidence of formation of a granular layer characterized by focal coarse keratohyaline granules in the upper part of the epithelium. Some cases may show parakeratosis unlike typical lichen planus. The epithelium has interface damage but usually dose not show the sawtooth pattern

fibrinogen along the dermoepidermal junction. There is usually some complement deposition, and there is variable IgM deposition. If necrotic keratinocytes are present in the dermis, they can non-specifically take up immunoglobulins, especially IgM, but IgG or IgA staining may also be present. It is important to point out that DIF findings are only supportive and not diagnostic without appropriate histologic findings.

## *Differential Diagnosis*

A common entity in the differential diagnosis is a benign lichenoid keratosis, also called lichen planus-like keratosis or lichenoid benign keratosis, depending on your preference. In some cases the histologic features may be indistinguishable. In other cases, they resemble a seborrheic keratosis that also has prominent interface change. Sometimes a recognizable component of solar lentigo is seen at the edges of the biopsy specimen. The clinical presentation is quite different. Benign lichenoid keratosis is a solitary lesion that usually presents on the trunk. Clinically, it mimics basal cell carcinoma, and the possibility of basal cell carcinoma is frequently suggested by the clinician. That can be a clue to the diagnosis.

Lichenoid drug eruption and a fixed drug can be confused with lichen planus. Lichenoid drug eruption may closely mimic lichen planus, but in addition to features resembling lichen planus there are eosinophils in the inflammatory infiltrate and usually some parakeratosis in the stratum corneum. Fixed drug eruptions lack the prominent epidermal changes and also have eosinophils in the infiltrate. The clinical history of fixed drug eruption is also distinctive as discussed below. Practical tips are summarized in Table 4.2.

**Table 4.2** Practical tips: lichen planus

- If the clinical history is a solitary lesion, think benign lichenoid keratosis
- Eosinophils are not a typical feature of lichen planus with perhaps the exception of hypertrophic lichen planus. If present, consider lichenoid drug eruption
- Parakeratosis is not typical a feature of lichen planus. If present, the possibility of a lichenoid drug eruption should be considered
- Oral/mucosal lichen planus is more subtle
  - Mucosal epithelium does not normally have a granular layer, so there is not prominent hypergranulosis
  - The presence of a subtle granular layer is a diagnostic clue
  - Parakeratosis is often present in mucosal lichen planus
- In cases where the histologic features or clinical history are not clear cut, use a descriptive diagnosis of "lichenoid interface dermatitis, see comment". Refer to sample reports

## Lichenoid Drug Eruption

### *Clinical Features*

The lesions of lichenoid drug eruptions can clinically resemble lichen planus. However, they tend to be larger and are more frequently distributed on the trunk; lesions on the extremities are not limited to the flexural surfaces. Oral mucosa involvement is usually absent. Some of the more common agents that can result in a lichenoid drug eruption include beta blockers, captopril, thiazides, and Lasix.

### *Microscopic Features*

Lichenoid drug eruptions, like lichen planus, are characterized by a band-like infiltrate with interface change, with evidence of epidermal damage including dyskeratotic keratinocytes and vacuolar change along the dermal-epidermal junction. There may also be acanthosis and hyperkeratosis. The histologic findings are essentially the same as lichen planus with some key exceptions (Fig. 4.8). There is often patchy parakeratosis, a feature not seen in lichen planus. Eosinophils are usually conspicuous, a feature that essentially excludes most cases of lichen planus. Thickening of the granular cell layer is usually, but not always present and is usually less prominent than in lichen planus (Table 4.3).

### *Differential Diagnosis*

As outlined above, the differential diagnosis is primarily lichen planus and a fixed drug eruption. Differentiating lichen planus from lichenoid drug requires identification of features not seen in lichen planus such as parakeratosis and conspicuous eosinophils.

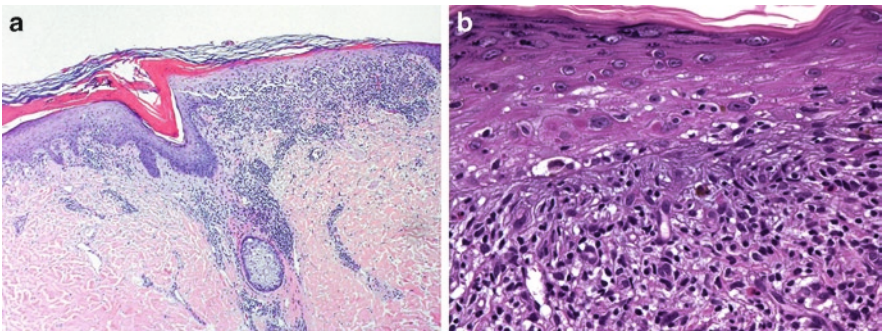


**Fig. 4.8** *Lichenoid drug reaction*. (**a**). in this lower power image, the lesion resembles lichen planus but there is conspicuous parakeratosis. (**b**). The higher power image demonstrates the interface change with a lichenoid infiltrate that contains conspicuous eosinophils

**Table 4.3** Key microscopic features: lichenoid drug eruption

- Compact hyperkeratosis and parakeratosis
- Lichenoid infiltrate of lymphocytes and eosinophils
- Interface change

Fixed drug eruptions tend to be localized (see below) and show less epidermal change. While still having the interface change and lichenoid infiltrate, they lack hyperkeratosis or granular layer thickening. See Table 4.4.

**Table 4.4** Practical tips: lichenoid drug eruption

- Parakeratosis is a frequent feature of lichenoid drug eruptions. Its presence argues for lichenoid drug eruption rather than lichen planus.
- Eosinophils are conspicuous in the great majority of lichenoid drug eruptions. If you can pick up the presence of eosinophils on medium power (10× objective), it favors lichenoid drug eruption over lichen planus.
- Lichenoid drug eruptions are typically more widespread than lichen planus.
- Oral mucosa involvement is uncommon in lichenoid drug eruptions.

## Fixed Drug Eruption

### *Clinical Features*

Fixed drug eruptions present as one or more violaceous plaques usually on the extremities or genitalia. On re-exposure to the drug the eruption recurs in the same locations. Common sensitizing agents include barbiturates, ibuprofen, and sulfa drugs.

## *Microscopic Features*

The stratum corneum of the epidermis varies from a normal basket weave pattern to having patchy parakeratosis. The epidermis may show ballooning degeneration of keratinocytes. Within the dermis, there is a lichenoid infiltrate composed of lymphocytes and eosinophils with interface damage to the overlying epidermis (Fig. 4.9). Melanophages are present as the lesion evolves (Table 4.5).

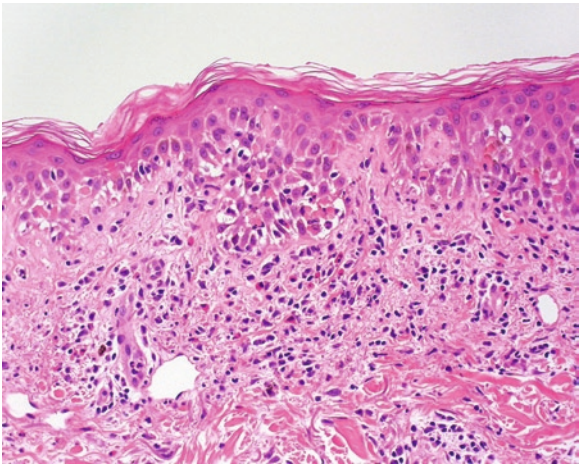

**Fig. 4.9** *Fixed drug eruption*. The epidermis has a normal basket weave stratum corneum. There is a lichenoid infiltrate with prominent interface change and frequent eosinophils. Scattered melanophages are present

**Table 4.5** Key microscopic features: fixed drug eruption

- Normal basket weave stratum corneum or parakeratosis; no hyperkeratosis
- Lichenoid infiltrate of lymphocytes and eosinophils
- Scattered melanophages
- Interface change

## *Differential Diagnosis*

The differential diagnosis includes lichen planus and a lichenoid drug eruption (see above). Unlike both lichen planus and lichenoid drug eruptions, fixed drug eruptions have a more limited distribution. The plaques of fixed drug eruption are larger than the papular lesions of lichen planus and lichenoid drug eruptions. Histologically, fixed drug eruption has less epidermal change. While having a similar degree of interface change, fixed drug eruptions do not have hyperkeratosis or hypergranulosis. Frequently, fixed drug eruptions have a normal basket weave pattern to the stratum corneum. A morbilliform drug eruption could also be considered. Morbilliform drug

eruptions are more widespread, and have a perivascular rather than lichenoid pattern. The degree of epidermal damage is less in morbilliform drug eruptions. Erythema multiforme (EM) and graft vs. host disease (GVHD) could also be considered. Again, these conditions have a less prominent infiltrate than seen in fixed drug eruption. These conditions are discussed in more detail below. See Table 4.6.

**Table 4.6** Practical tips: fixed drug eruption

- Clinically localized, not widespread
- Epidermal change limited
  - Patchy keratosis to normal stratum corneum
  - Granular layer not thickened
- Only make a diagnosis of fixed drug eruption with a solid clinical history. A phone call to the clinician is helpful in cases with inadequate history
- Melanophages can be a clue to an evolving or recurrent fixed drug eruption
- Fixed drug eruptions have more prominent interface change than morbilliform drug eruptions

## *Interface Dermatitis with Perivascular Infiltrate*

In this section, the entities have a predominantly perivascular pattern of inflammation rather than lichenoid pattern.

## Morbilliform Drug Eruption

### *Clinical Features*

Morbilliform drug eruptions present as widespread erythematous, blanchable macules or papules. They can present shortly after initiation of the offending medication or it can take several months for the hypersensitivity reaction to develop.

### *Microscopic Features*

The epidermis typically shows little change except for some mild basal vacuolization. Parakeratosis, acanthosis and spongiosis are not a typical feature except in rare eczematous drug eruptions (see Chap. 2). Occasional necrotic keratinocytes may be present, but this is not an invariable feature. Within the dermis, there is a mild superficial perivascular mixed inflammatory infiltrate of lymphocytes and eosinophils (Fig. 4.10). In many cases, there is no interface damage to the epidermis and the predominant finding is a superficial perivascular infiltrate (see Chap. 5) (Table 4.7).

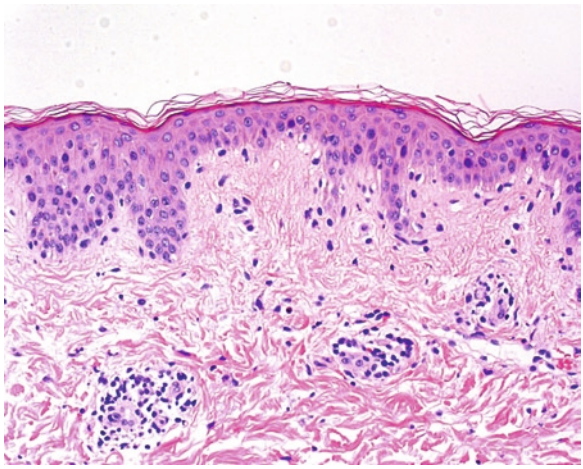

**Fig. 4.10** *Morbilliform drug eruption*. The epidermis appears relatively normal except for mild vacuolar change. With the dermis there is a mild superficial perivascular infiltrate of lymphocytes and eosinophils

**Table 4.7** Key microscopic features: morbilliform drug eruption

- Mild basal vacuolization or no epidermal change
- Superficial perivascular infiltrate of lymphocytes admixed with eosinophils

## *Differential Diagnosis*

In cases with interface damage, the differential diagnosis of morbilliform drug eruption includes acute GVHD, fixed drug eruption, lupus erythematosus, dermatomyositis, and viral exanthem. Acute GVHD typically occurs in the setting of bone marrow transplantations and rarely in solid organ transplants. It usually occurs relatively soon after the transplant, and in most cases, but by no means all, lack eosinophils. Connective tissue disease such as lupus erythematosus and dermatomyositis are characterized by interface change. However, they lack eosinophils and typically have increased dermal mucin. Viral exanthems usually lack eosinophils and are rarely biopsied.

In drug eruptions without interface change, the differential diagnosis includes dermal hypersensitivity reactions such as urticaria. Histologically, these entities are essentially indistinguishable and require clinical information (see Chap. 5). So-called papular dermatitis, also known as itchy red bump disease or papular eczema, has a similar pattern of perivascular infiltrate, but usually has reactive epidermal changes related to excoriation. See Table 4.8.

**Table 4.8** Practical tips: morbilliform drug eruption

- The interface change in most morbilliform drug eruptions is mild in nature. If numerous dyskeratotic keratinocytes are present, other entities should be considered.
- Interface change is not always present
- The dermal infiltrate is typically mild in nature and is composed of lymphocytes and eosinophils
- Eosinophils are not necessarily prominent
- Without a good history, it is best to give a descriptive diagnosis and suggest the possibility of a drug eruption in the report comment. See example reports.
- A phone call to the clinician can be helpful if a good history is not available

### *Erythema Multiforme, Stevens–Johnson Syndrome and Toxic Epidermal Necrolysis*

These entities are viewed by some as a spectrum of the same disease process and by others as distinct entities. Histologically, they are similar to identical and will be dealt with as a group.

### *Clinical Features*

In classic erythema multiforme (EM), the patient presents with episodic eruptions of macules, papules, or targetoid lesions on the extensor surfaces, palms, soles, and/or oral mucosa. If there is extensive mucosal involvement, the eruption can qualify for the designation of Stevens–Johnson syndrome (SJS). The eruption can be associated with herpes simplex virus infections (especially EM), mycoplasma infections, and drugs. SJS is typically associated with medications, with sulfa drugs being one of the most common triggers.

Toxic epidermal necrolysis (TEN) presents with widespread tender macular eruption with vesicles and bullae. Application of pressure to the skin can cause detachment of the epidermis (Nikolsky's sign). TEN is a medical emergency necessitating admission to a burn unit. The mortality ranges from 25 to 50%.

### *Microscopic Features*

All of the entities in this group have essentially the same histologic features. The epidermis is relatively normal with a basket weave stratum corneum lacking parakeratosis or hyperkeratosis. There is vacuolar interface damage with necrosis of keratinocytes, often at all levels of the epidermis, in association with a mild superficial perivascular lymphocytic infiltrate (Fig. 4.11). Eosinophils are sometimes present, especially in cases related to medications. In TEN, there is often full-thickness necrosis, but this is not a specific finding for this entity (Fig. 4.12) (Table 4.9).

### *Differential Diagnosis*

The histologic differential diagnosis can include morbilliform drug eruption, graft versus host disease (GVHD) and connective tissue disease such as lupus erythematosus or dermatomyositis. The pronounced epidermal damage helps exclude a typical drug eruption. GVHD has the appropriate clinical history. In connective tissue disease, there are epidermal changes (e.g., parakeratosis, thickened basement

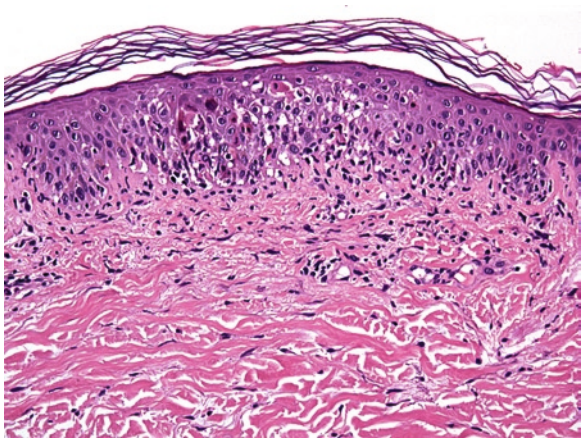

**Fig. 4.11** *Erythema multiforme*. The epidermis has a normal stratum corneum. There is prominent epidermal damage characterized by dyskeratotic keratinocytes at all levels of the epidermis. Note the disproportionately sparse superficial lymphocytic infiltrate in comparison to the degree of epidermal damage

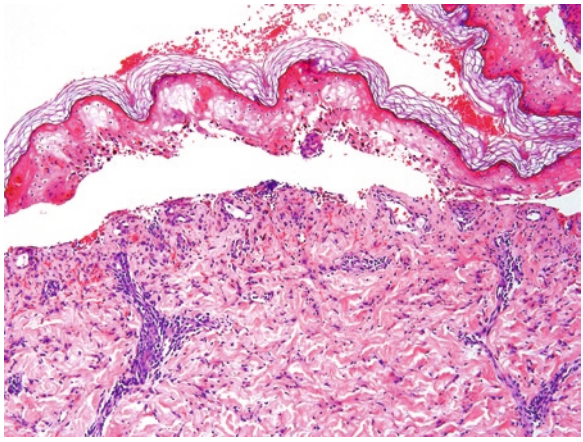

**Fig. 4.12** *Toxic epidermal necrolysis*. In this case there is full thickness acute necrosis of the epidermis and a sparse superficial perivascular lymphocytic infiltrate

**Table 4.9** Key microscopic features: erythema multiforme, Stevens–Johnson syndrome, toxic epidermal necrolysis

- Normal basket-weave stratum corneum
- Mild perivascular lymphocytic infiltrate (with or without scattered eosinophils)
- Basal vacuolization with dyskeratotic keratinocytes at all levels of the epidermis
- May have full thickness necrosis of epidermis

membrane) that are not seen in the EM/SJS/TEN spectrum. Clinically, TEN and staphylococcal scalded skin syndrome (SSSS) can look alike. This clinical difference can be the source of a middle of the night frozen section, and so familiarity with this differential diagnosis is important. In SSSS a bacterial toxin causes a split between the stratum corneum and underlying epidermis. There is no dyskeratosis or interface change (Fig. 4.13). Practical tips are summarized in Table 4.10.

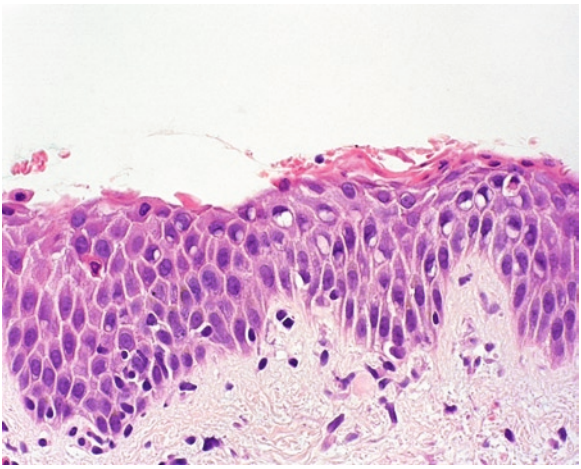

**Fig. 4.13** *Staphylococcal scalded skin syndrome (SSSS).* SSSS is characterized by a split between the stratum spinosum and stratum corneum. It does not have interface change or prominent keratinocyte necrosis in contrast to toxic epidermal necrolysis

**Table 4.10** Practical tips: erythema multiforme, Stevens–Johnson syndrome, toxic epidermal necrolysis

- Degree of epidermal damage is disproportionate to the density of the infiltrate
- Because this group has an acute onset, the epidermis retains its normal basket-weave pattern in the stratum corneum
- If there are large areas of full thickness necrosis, SJS or TEN is more likely

## Lupus Erythematosus

### *Clinical Features*

Cutaneous lupus erythematosus can be subdivided into chronic (discoid), subacute, and systemic (acute) forms. There is a clinical overlap, and patients with discoid or subacute lupus erythematosus can progress to systemic disease, though it is less common in discoid lupus erythematosus.

Chronic, or discoid, lupus erythematosus is characterized by sharply demarcated erythematous scaly plaques usually involving the head and neck, often involving the face in a butterfly pattern. Lesions on the scalp can result in scarring alopecia. A variant of discoid lupus erythematosus called tumid lupus presents as juicy papules and plaques on the upper trunk, and head, and neck. The tumid variant has less scale. Chronic forms of lupus erythematosus are usually not associated with underlying systemic disease. Progression to systemic disease is seen in roughly 5–10% of cases. Antinuclear antibody (ANA) titers are positive in approximately 70% of the cases.

The cutaneous lesions of subacute lupus erythematosus manifest as annular lesions or plaques in photodistributed areas on the head and neck, upper trunk, and upper extremities. The patients often have mild musculoskeletal symptoms. Central nervous system involvement is usually absent and renal involvement is variable. Traditionally renal involvement was not considered common, but some reports have

refuted this finding. Positive ANA titers are seen in about 50% of cases. Patients may develop lesions of discoid lupus erythematosus or progress to fully developed systemic lupus erythematosus.

Cutaneous lesions are present in about 80% of patients with systemic lupus erythematosus. The cutaneous lesions are less well defined as in the other forms of cutaneous lupus erythematosus. They present as erythematous patches with little scale. As in other forms of cutaneous lupus erythematosus, the cutaneous lesions are in photodistributed areas, especially the malar face. Positive ANA titers are seen in approximately 90% of cases and >50% have anti-double stranded DNA antibodies.

## *Microscopic Features*

Similar to the clinical manifestations, there is significant histologic overlap in the different clinical subtypes of cutaneous lupus erythematosus. From a practical standpoint, the overlap may preclude subclassification based on histologic features alone. All are a characterized by interface change of basal vacuolization and a perivascular lymphocytic infiltrate with increased dermal mucin (Fig. 4.14). Dermal mucin appears as stringy blue–gray material between the dermal collagen of the reticular dermis. It is not to be confused with solar elastosis; solar elastosis does not have the delicate appearance of dermal mucin, but resembles the structure of collagen fibers. Dermal mucin may be variably identifiable on routine H&E stained sections; it depends on the slide preparation technique of individual laboratories. Colloidal iron stains can be helpful in highlighting dermal mucin when it is not evident on routine H&E stains, but in most cases, it is not necessary (Table 4.11).

In discoid lupus erythematosus, the epidermis shows hyperkeratosis, variable epidermal atrophy alternating with acanthosis and follicular plugging (Fig. 4.15). The basement membrane is often thickened. The inflammatory infiltrate has a superficial and deep pattern, and frequently involves adnexal structures. In older "burned out" lesions, there may be less active interface change. In such cases, the evidence of epidermal change includes the thickened basement membrane, epidermal atrophy and melanophages

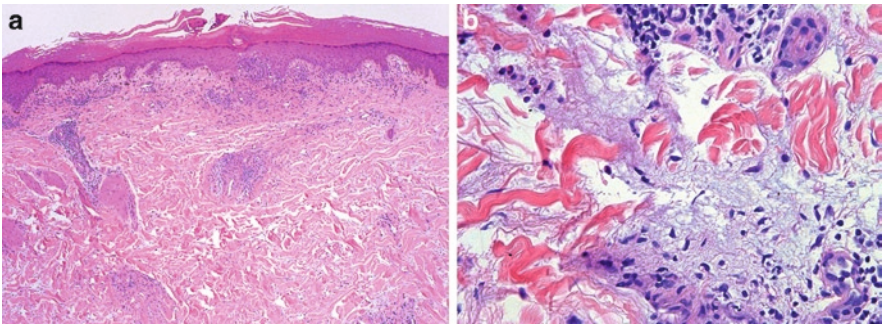


**Fig. 4.14** *Lupus erythematosus*. (**a**). There is interface change and a superficial and deep perivascular lymphocytic infiltrate. (**b**). Between the collagen bundles there is deposition of dermal mucin characterized by blue-gray, somewhat delicate, stringy material

**Table 4.11** Key microscopic features: lupus erythematosus

- Variable hyperkeratosis and parakeratosis
- Interface change with basal vacuolization
- Epidermal basement membrane often thickened
- Superficial or superficial and deep perivascular or perivascular and periadnexal lymphocytic infiltrate
- Increased dermal mucin

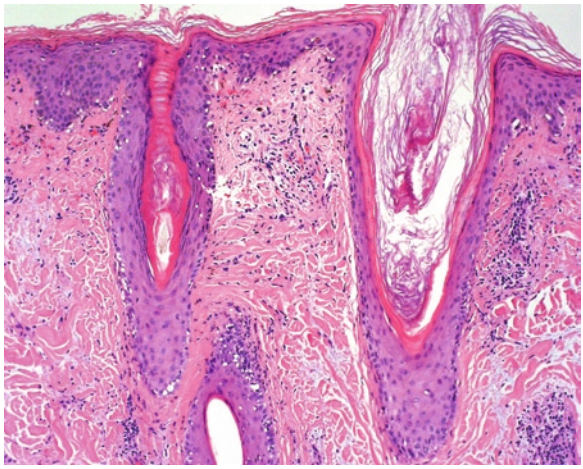

**Fig. 4.15** *Lupus erythematosus*. In lupus erythematosus, especially discoid forms, the epidermis demonstrates follicular plugging and alternating acanthosis and atrophy in addition to the interface change. Note the blue-gray dermal mucin between the collagen bundles

in the upper dermis. In the tumid form, significant interface change is typically absent; the combination of a superficial and deep infiltrate with increased dermal mucin is an important clue (Fig. 4.16). Subacute lupus erythematosus differs from the discoid form only slightly. There is usually a less intense inflammatory infiltrate and more prominent atrophy. In systemic lupus erythematosus, there is prominent basal vacuolization but necrotic keratinocytes are rare. The infiltrate is typically less intense and usually in a superficial perivascular distribution.

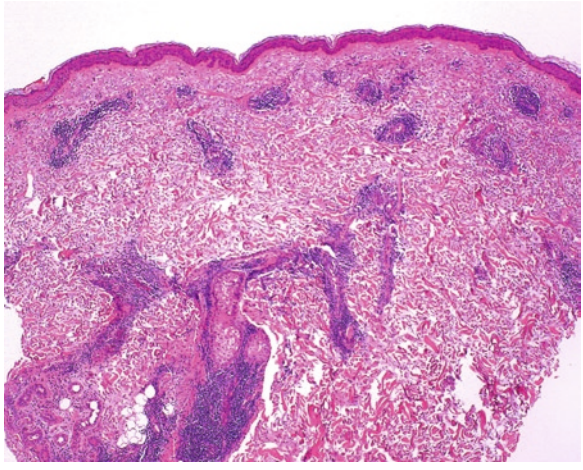

**Fig. 4.16** *Tumid lupus erythematosus*. In the tumid form of lupus erythematosus, interface change is focal or absent. The key features are the superficial and deep lymphocytic infiltrate and dermal mucin deposition

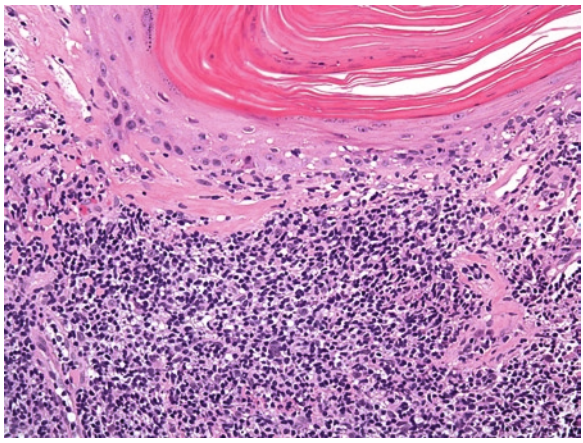

**Fig. 4.17** *Reactive epidermal atypia in lupus erythematosus*. In some cases of lupus erythematosus, the interface change can result in reactive atypia of the epidermis

### *Differential Diagnosis*

In cases with relatively numerous necrotic keratinocytes, the differential diagnosis includes EM. However, EM is an acute process and does not show the other epidermal changes of lupus erythematosus such as hyperkeratosis, atrophy, or basement membrane thickening. In cases with a dense inflammatory infiltrate, lichen planus could be considered, but the presence of dermal mucin and a deep inflammatory component are against lichen planus. In both instances, clinical history is also helpful. Dermatomyositis can be remarkably similar to lupus erythematosus (see below). It also shows interface dermatitis with increased dermal mucin. The inflammatory infiltrate in dermatomyositis is generally mild and restricted to the superficial dermis. In some cases, it may not be possible to distinguish between these entities except by clinical history. Some cases of lupus erythematosus, especially discoid lupus erythematosus, show reactive atypia in the keratinocytes of the epidermis (Fig. 4.17). The reactive epithelial atypia can mimic the dysplasia of actinic keratosis or even squamous cell carcinoma. Confusion with an actinic keratosis is usually more of a risk in superficial shave biopsies. The clinical history and the presence of other findings of lupus erythematosus will allow for distinction (Table 4.12).

## Dermatomyositis

### *Clinical Features*

Dermatomyositis is characterized by the combination of muscle weakness and characteristic cutaneous findings of erythematous to violaceous slightly scaly lesions. The face, shoulders and extensor surfaces of the extremities are most commonly

**Table 4.12** Practical tips: lupus erythematosus

- Distribution is important: Lupus erythematosus is a photo-distributed disease
- Eosinophils are not a feature of lupus erythematosus except in the rare cases of drug-induced lupus erythematosus. The presence of eosinophils raises the possibility of dermal hypersensitivity reactions such as an arthropod bite reaction or drug eruption.
- The "actinic keratosis clue." Remember that some cases of lupus erythematous can superficially resemble actinic keratosis. If there is interface change and squamous atypia, consider the possibility of lupus erythematosus.
- Remember that biopsies from old lesions may not show active vacuolar interface change. Look for evidence of past interface damage such as atrophy, basement membrane thickening, and melanophages.
- Colloidal iron studies may help highlight the dermal mucin
- Some cases of dermatomyositis and lupus erythematosus are histologically indistinguishable
- Tumid lupus erythematosus lacks interface change

involved. Involvement of the face frequently takes the form of a periorbital heliotrope rash. Involvement of the shoulders is often diffuse causing the shawl sign. Periungual erythema and Gottron's papules are common findings on the hands. Muscle weakness, when present, involves proximal muscles. Cutaneous involvement can precede muscle involvement by months to years, and some patients never develop muscle weakness (so-called dermatomyositis sine myositis).

## *Microscopic Features*

The histologic features are characterized by basal vacuolization, a minimal to mild superficial perivascular lymphocytic infiltrate and increased dermal mucin (Fig. 4.18). The basement membrane may be thickened, and melanophages may be seen in the upper dermis. Occasional neutrophils may be present. In some cases, interface change is not apparent on the biopsy specimen. In cases such as this, the prominent dermal mucin and scant to mild perivascular lymphocytic infiltrate should serve as a clue to the diagnosis (Table 4.13).

## *Differential Diagnosis*

The primary differential diagnosis is lupus erythematosus. Unfortunately, it is not possible to unequivocally differentiate dermatomyositis from lupus erythematosus. (See also above section on lupus erythematosus). The same comments above on differentiating lupus erythematosus from other forms of interface dermatitis apply to dermatomyositis (see Table 4.14).

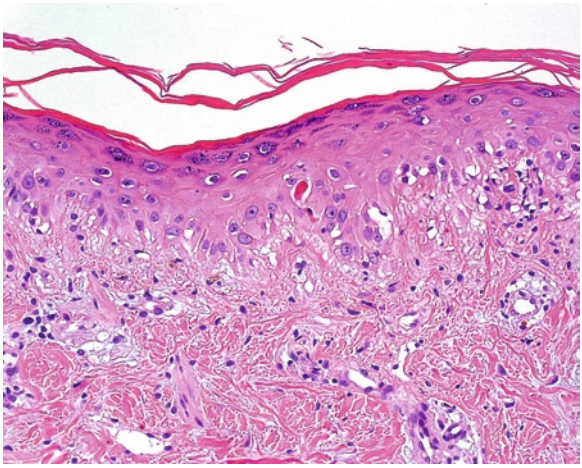

**Fig. 4.18** *Dermatomyositis*. In dermatomyositis there is typically interface change with basal vacuolization, but the infiltrate is sparse or mild in nature. Dermal mucin deposition is present

**Table 4.13** Dermatomyositis: key microscopic features

- Basal vacuolization
- Mild superficial perivascular lymphocytic infiltrate
- Increased dermal mucin

**Table 4.14** Dermatomyositis: practical tips

- The infiltrate in dermatomyositis is usually mild and restricted to the superficial dermis. If there is a deep component, consider the diagnosis of lupus erythematosus.
- Eosinophils are not a feature of dermatomyositis. If present consider the diagnosis of a drug eruption.
- Colloidal iron stains may help highlight the dermal mucin.
- Dermatomyositis is pruritic; this clinical information can be a clue.

## Graft Versus Host Disease

### *Clinical Features*

Cutaneous graft versus host disease (GVHD) usually occurs in the setting of bone marrow transplant, but can sometimes occur in the setting of solid organ transplants. GVHD can be subdivided into acute GVHD and chronic GVHD. Acute GVHD typically occurs 2–4 weeks after transplantation, but it can be quite variable and may be several weeks to months after transplantation. Another variable that is increasingly seen is the practice of donor lymphocyte reinfusion. In this setting, acute GVHD can present many months after the original transplant. The reinfusion of donor lymphocytes essentially resets the GVHD clock. The eruption of acute GVHD is characterized by an erythematous macular to papular eruption involving the face, posterior neck, ears, hands and feet. The eruption often starts with facial erythema that subsequently involves other parts of the body with a maculopapular eruption. Co-existing diarrhea is often present, and may precede the cutaneous eruption. Laboratory tests frequently show elevated liver enzymes.

Chronic GVHD classically occurs greater than 6 months after transplantation. Chronic GVHD is subdivided into lichenoid and sclerodermoid forms. Classically, chronic GVHD first manifests with the lichenoid form, with the sclerodermoid form following. Some patients present with both forms simultaneously. Lichenoid chronic GVHD presents as polygonal violaceous papules reminiscent of lichen planus. Oral mucosal involvement is seen in approximately 90% of patients. Sclerodermoid GVHD presents as areas of dermal sclerosis similar to morphea/scleroderma (see Chap. 9).

## *Microscopic Features*

### Acute GVHD

In acute GVHD, the epidermis is relatively normal, but some keratinocyte atypia may be seen as a result of prior chemotherapy. Within the dermis, there is a sparse infiltrate of lymphocytes. Occasionally eosinophils may be part of the infiltrate. The interface change is manifested by basal vacuolization. As the eruption progresses, necrotic keratinocytes are seen often with lymphocyte satellitosis (Fig. 4.19). Severe cases can show cleft formation between the epidermis and dermis or even full thickness necrosis of the epidermis. Fortunately, this is rarely seen. Acute GVHD is graded by the following scheme:

Grade 0: Normal skin.
Grade 1: Basal vacuolization with a mild superficial perivascular lymphocytic infiltrate.
Grade 2: Same features as Grade 1 with scattered necrotic keratinocytes and satellite cell necrosis.
Grade 3: Same features as Grade 2 but with cleft formation between epidermis and dermis.
Grade 4: Same as features as Grade 2 or 3 with complete separation of the epidermis from the dermis.

Unlike acute GVHD, there is no grading scheme for chronic GVHD. Lichenoid chronic GVHD shows epidermal changes of hyperkeratosis and hypergranulosis in addition to interface change of basal vacuolization with necrotic keratinocytes and satellite cell necrosis (Fig. 4.20). The infiltrate in the dermis is usually mild, but is often more dense than seen with acute GVHD, and in some cases, there is a dense band-like infiltrate similar to lichen planus. Rarely some biopsies may show transitional forms with histologic features of acute GVHD and lichenoid chronic GVHD in the same biopsy or different concurrent biopsies from the same patient.

Sclerodermoid chronic GVHD resembles morphea or scleroderma (see below). There is epidermal atrophy with dermal sclerosis characterized by fibrosis with compaction of collagen fibers in the reticular dermis (Fig. 4.21). There is a loss of adnexal structures. Microscopic features are summarized in Table 4.15.

**Table 4.15** Key microscopic features: graft vs. host disease (GVHD)

- Basal vacuolization
- Variable amount of dyskeratotic keratinocytes
- Satellite cell necrosis
- Mild superficial perivascular lymphocytic infiltrate
- Lichenoid chronic GVHD has a thickened granular layer and hyperkeratosis
- Sclerodermoid chronic GVHD has thickened and compacted dermal collagen bundles

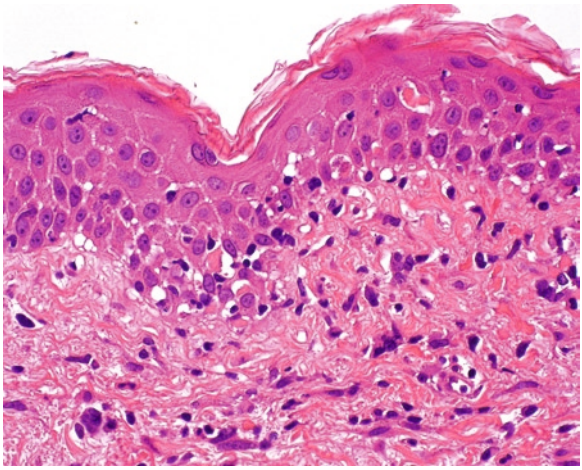

**Fig. 4.19** *Acute graft vs. host disease*. In acute graft vs. host disease there is variable interface change with basal vacuolization and dyskeratotic keratinocytes. Satellite cell necrosis, characterized by lymphocytes tagging dyskeratotic cells, is commonly seen. The dermal infiltrate is usually mild in nature. This lesion would be considered grade 2

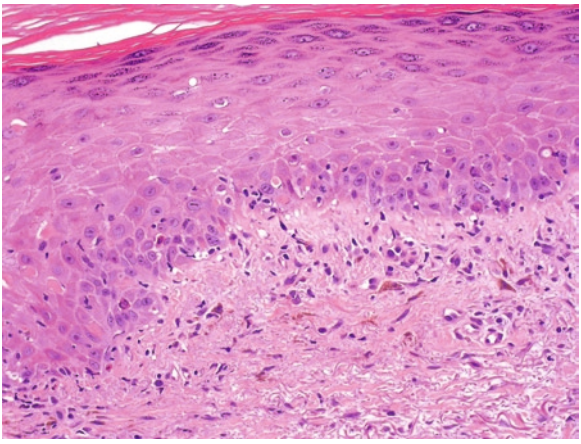

**Fig. 4.20** *Lichenoid chronic graft vs. host disease*. This form of chronic graft vs. host disease has features that overlap with lichen planus, namely compact hyperkeratosis, a thickened granular layer and interface change. The infiltrate in lichenoid graft vs. host disease is milder in nature

## *Differential Diagnosis*

In acute GVHD, the histologic and clinical differential diagnosis is usually a drug eruption. In most cases of acute GVHD, there are no eosinophils unlike typical drug eruptions. However, acute GVHD can sometimes have eosinophils as part of the infiltrate, and the presence of eosinophils is by no means diagnostic of a drug eruption in this setting as it was once believed. Satellite cell necrosis is more common in

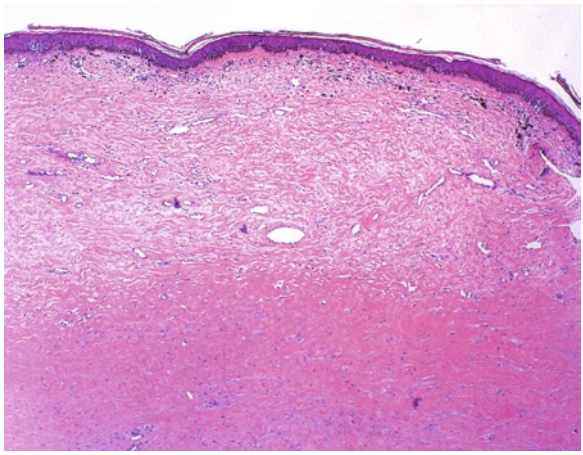

**Fig. 4.21** *Sclerodermoid chronic graft vs. host disease*. This form of chronic graft vs. host disease resembles morphea/scleroderma. There is sclerosis of the dermis characterized by compactin of the collagen fibers with loss of the normal space between collagen fibers of the reticular dermis and loss of adnexal structures. Inflammation is absent to mild. There is often no active interface change

GVHD, but unequivocal distinction may not always be possible. From a practical perspective, most patients for whom acute GVHD is a diagnostic consideration do not have a sufficiently reconstituted immune system to mount a drug eruption and the bias should be toward the diagnosis of acute GVHD. EM could be considered from a histologic standpoint, but the clinical situation typically negates EM from consideration.

In lichenoid chronic GVHD, the primary differential diagnosis is lichen planus. Lichen planus typically has a denser infiltrate. Clinical history is also helpful. It is not possible to distinguish sclerodermoid chronic GVHD from morphea or scleroderma histologically; clinical information is essential. See Table 4.16.

**Table 4.16** Practical tips: graft vs. host disease (GVHD)

- It is rare to see acute GVHD before 14 days after transplantation.
- The histologic features may lag the clinical presentation. In very early biopsies of GVHD, the skin may show no histologic abnormalities.
- Deeper levels or subsequent biopsies may show classic GVHD.
- Late onset acute GVHD (> 6 months after transplantation) may be seen in the setting of donor lymphocyte reinfusion, an increasingly common practice.
- Eosinophils may sometimes be seen in GVHD and does not exclude the diagnosis in the appropriate clinical setting. From a practical viewpoint, many of these patients may not have a sufficient immune system to mount a drug eruption. Our bias is that the eruption in this clinical setting is GVHD until proven otherwise.
- Additional clinical information (e.g., diarrhea or elevated liver enzymes) can help corroborate the diagnosis.

## Pityriasis Lichenoides

### *Clinical Features*

Pityriasis lichenoides is most common in young adult men and typically involves the extremities, trunk, and buttocks. Pityriasis lichenoides exists in two forms: pityriasis

lichenoides et varioliformis acuta (PLEVA) and pityriasis lichenoides chronica (PLC). PLEVA presents as recurrent crops of skin-colored papules that become hemorrhagic or crusted, ulcerate, and leave behind varioliform (smallpox-like) scars. PLC is less hemorrhagic consisting of red-brown, scaly macules to papules. In PLC, the lesions heal without scarring, but there may be post-inflammatory changes.

## *Microscopic Features*

The microscopic features show significant overlap between the two entities. Both show epidermal changes of parakeratosis, basal vacuolization and necrotic keratinocytes. The epidermal change is more pronounced in PLEVA with more numerous necrotic keratinocytes and prominent exocytosis of lymphocytes and often erythrocytes (Fig. 4.22). In PLEVA, the infiltrate is superficial and deep and often has a wedge-shaped configuration. Extravasation of erythrocytes is commonly present, especially in PLEVA, but fibrinoid necrosis of blood vessels is absent. In late lesions of PLEVA the biopsy may show ulceration of the epidermis. The changes in PLC are subtler than in PLEVA (Fig. 4.23). There is often confluent parakeratosis, and scattered dyskeratotic keratinocytes in the epidermis. There may be variable acanthosis and the interface change consisting of basal vacuolization is usually subtle. The dermal inflammatory infiltrate is predominantly composed of lymphocytes and usually restricted to the superficial dermis (Table 4.17).

## *Differential Diagnosis*

For PLEVA, the differential diagnosis includes lymphomatoid papulosis (LYP) (see Chap. 5). Both have similar clinical histories of recurrent crops of papules that can ulcerate and both can show a superficial and deep infiltrate with interface

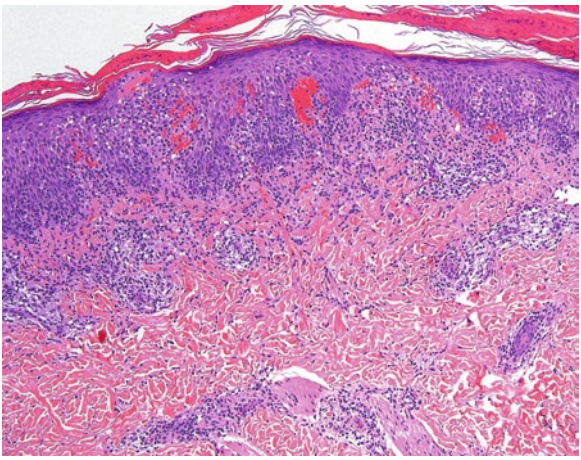

**Fig. 4.22** *Pityriasis lichenoides et varioliformis acuta (PLEVA)*. The histologic features of PLEVA are variable depending on which stage the biopsy is taken. The most characteristic findings include parakeratosis and serum crust scale overlying the epidermis in association with interface change, a superficial and deep perivascular lymphocytic infiltrate, and prominent hemorrhage in the superficial dermis

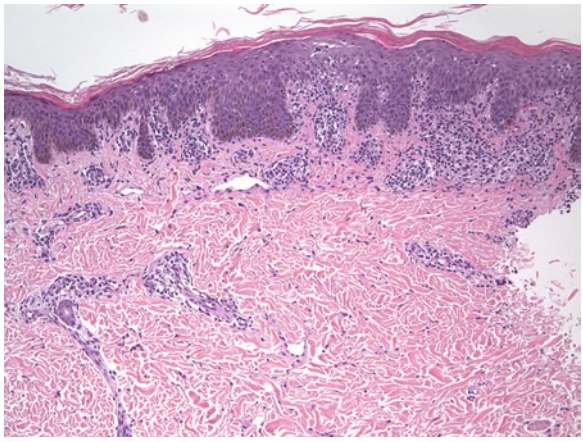

**Fig. 4.23** *Pityriasis lichenoides chronica (PLC).* In PLC, there is parakeratosis overlying the epidermis. There is interface change and usually a mild to moderate lymphocytic infiltrate in the upper dermis

**Table 4.17** Key microscopic features: pityriasis lichenoides et varioliformis acuta (PLEVA) and pityriasis lichenoides chronica (PLC)

| |
|---|
| PLEVA |
| • Parakeratosis, spongiosis, and basal vacuolization |
| • Dyskeratotic keratinocytes |
| • Superficial and deep perivascular lymphocytic infiltrate |
| • Extravasation of erythrocytes in papillary dermis |
| PLC |
| • Parakeratosis |
| • Variable acanthosis |
| • Occasional dyskeratotic cells |
| • Mild basal vacuolization |
| • Superficial perivascular lymphocytic infiltrate |

change. LYP usually has a population of large atypical CD30+ cells. Lupus erythematosus could be considered, but the clinical presentation is different, the degree of epidermal damage is more pronounced in PLEVA, and PLEVA does not have increased dermal mucin. EM is in the differential diagnosis, but PLEVA has more epidermal changes (e.g., parakeratosis) and a denser inflammatory infiltrate. For PLC, the differential diagnosis includes pityriasis rosea, spongiotic/eczematous dermatitis, and guttate psoriasis. Pityriasis rosea has more discrete mounds of parakeratosis and lacks interface change. Similarly, spongiotic dermatitis lacks interface change and has more pronounced spongiosis. Guttate psoriasis has mounds of parakeratosis that can have collections of neutrophils. Dyskeratotic cells or interface change are not features of guttate psoriasis.

Obviously with the overlapping histologic features, PLEVA and PLC can be confused, as these entities represent ends of a morphologic spectrum. Knowledge of the clinical presentation may be required to distinguish them. Classification of a given lesion as PLEVA or PLC may not be possible. In such a setting, the more generic term pityriasis lichenoides can be used. See Table 4.18.

**Table 4.18** Practical tips: PLEVA and PLC

- Maintain a high index of suspicion
- The presence of interface change with hemorrhage is an important clue especially for PLEVA
- Knowledge of the clinical history is especially helpful for the diagnosis of PLEVA or PLC
  - PLEVA presents as hemorrhagic papules
  - PLC presents as papules or small plaques
- An ulcerated lesion of PLEVA has non-specific histologic features. Suggest re-biopsy of a more recent lesion

## Sample Reports: Lichen Planus

Example 1:

*Clinical history*: Pruritic papules on wrist; rule-out lichen planus.
*Diagnosis*: Lichen planus, see comment.
*Comment*: Sections demonstrate an epidermis with compact hyperkeratosis and a thickened granular layer. Within the dermis, there is a lichenoid infiltrate of lymphocytes with prominent interface change with saw-toothing of the rete pegs and scattered dyskeratotic keratinocytes. The histologic features are consistent with lichen planus. Clinicopathologic correlation is recommended.

Example 2:

*Clinical history*: Lesion on chest.
*Diagnosis*: Lichenoid interface dermatitis, see comment.
*Comment*: The biopsy demonstrates many features of lichen planus including compact hyperkeratosis, a thickened granular layer and a lichenoid infiltrate of lymphocytes with prominent interface change. If there are multiple lesions, this could be compatible with lichen planus. If this is a solitary lesion, a benign lichenoid keratosis is favored. Clinicopathologic correlation is recommended.

Example 3:

*Clinical history*: Leukoplakia, rule out malignancy.
*Diagnosis*: Lichenoid mucositis, see comment.
*Comment*: There is some parakeratosis and a subtle granular layer. Within the subepithelial stroma, there is a lichenoid infiltrate of lymphocytes with interface change characterized by basal vacuolization and scattered dyskeratotic cells. No atypia or dysplasia is seen. The histologic features are consistent with oral lichen planus. Clinicopathologic correlation is recommended.

## Sample Reports: Lichenoid Drug Eruption

Example 1:

*Clinical history*: Rule out drug eruption.

*Diagnosis*: Lichenoid interface dermatitis consistent with lichenoid drug eruption, see comment.

*Comment*: There is compact hyperkeratosis and parakeratosis overlying the epidermis. Within the dermis, there is a lichenoid infiltrate of lymphocytes and eosinophils with prominent interface change. The histologic features are consistent with a lichenoid drug eruption. Clinicopathologic correlation is recommended.

Example 2:

*Clinical history*: Rule out lichen planus.

*Diagnosis*: Lichenoid interface dermatitis, see comment.

*Comment*: There is focal parakeratosis and compact hyperkeratosis overlying an epidermis with a thickened granular layer. Within the dermis, there is a lichenoid infiltrate of lymphocytes admixed with eosinophils in association with interface change. The presence of parakeratosis and eosinophils favors a lichenoid drug eruption over lichen planus. Clinicopathologic correlation is recommended.

## Sample Report: Fixed Drug Eruption

Example 1:

*Clinical history*: Recurrent lesion, rule out fixed drug eruption.

*Diagnosis*: Interface dermatitis consistent with fixed drug eruption, see comment.

*Comment*: There is normal basket-weave stratum corneum overlying the epidermis. Within the dermis, there is a lichenoid mixed infiltrate of lymphocytes and eosinophils with prominent interface change. Scattered melanophages are present in the dermis. The histologic features are consistent with the clinical impression of a fixed drug eruption.

Example 2:

*Clinical history*: Rule out drug eruption vs. other.

*Diagnosis*: Interface dermatitis, see comment.

*Comment*: The epidermis has focal parakeratosis. Within the dermis, there is a lichenoid infiltrate of lymphocytes and eosinophils in association with prominent interface change with basal vacuolization and dyskeratotic cells. Also within the dermis, there are

scattered melanophages. The histologic features are compatible with a dermal hypersensitivity reaction such as a drug eruption. The prominent interface change and melanophages could suggest the possibility of a fixed drug eruption in the appropriate clinical context. Clinicopathologic correlation is recommended.

## Sample Reports: Morbilliform Drug Eruption

Example 1:

*Clinical history*: Connective tissue disease (code word for lupus erythematosus or dermatomyositis) vs. drug eruption.

*Diagnosis*: Mild interface dermatitis consistent with a drug eruption, see comment.

*Comment*: Within the dermis, there is a mild superficial perivascular mixed infiltrate of lymphocytes and scattered eosinophils. There is focal interface change characterized by basal vacuolization. The histologic features are consistent with a drug eruption. The presence of eosinophils argues against the diagnosis of connective tissue disease such as lupus erythematosus or dermatomyositis. Clinicopathologic correlation is recommended.

Example 2:

*Clinical history*: Rule-out eczema.

*Diagnosis*: Skin, trunk, punch biopsy: Superficial perivascular mixed infiltrate with focal interface change, see comment.

*Comment*: The epidermis is relatively normal without significant spongiosis. Within the dermis, there is a superficial perivascular infiltrate of lymphocytes and scattered eosinophils in association with focal basal vacuolization. The histologic features are most consistent with a dermal hypersensitivity reaction such as a drug eruption. Papular eczema could be considered, but the absence of reactive epidermal change consistent with excoriation argues against this diagnosis. The absence of epidermal spongiosis is against the possibility of an eczematous dermatitis. Clinicopathologic correlation is recommended.

## Sample Reports: Erythema Multiforme, Toxic Epidermal Necrolysis

Example 1:

*Clinical history*: Rule-out EM.

*Diagnosis*: Erythema multiforme, see comment.

*Comment*: There is a normal basket-weave stratum corneum overlying the epidermis. Within the dermis, there is a mild superficial perivascular infiltrate of lymphocytes. There is interface change with basal vacuolization and dyskeratotic keratinocytes scattered throughout all levels of the epidermis. The histologic features are those of Erythema multiforme. Clinicopathologic correlation is recommended.

Example 2:

*Clinical history*: EM vs. drug eruption.

*Diagnosis*: Interface dermatitis, see comment.

*Comment*: There is a normal basket-weave stratum corneum overlying the epidermis. Within the dermis, there is a superficial perivascular infiltrate of lymphocytes with occasional eosinophils. There is prominent basal vacuolization with numerous dyskeratotic keratinocytes. Given the degree of epidermal damage, the biopsy findings are most consistent with Erythema multiforme rather than a typical drug eruption. Eosinophils may be seen in Erythema multiforme and do not exclude the diagnosis. Clinicopathologic correlation is recommended.

Example 3:

*Clinical history*: SSSS vs. TEN.

*Diagnosis*: Consistent with toxic epidermal necrolysis, see comment.

*Comment*: The stratum corneum is intact. Within the dermis, there is a sparse superficial perivascular lymphocytic infiltrate in association with basal vacuolization and numerous dyskeratotic keratinocytes. The histologic features are diagnostic of toxic epidermal necrolysis in the appropriate clinical context.

## Sample Reports: Lupus Erythematosus

Example 1:

*Clinical history*: Lupus erythematosus vs. dermatomyositis.

*Diagnosis*: Interface dermatitis, see comment.

*Comment*: There is focal parakeratosis overlying the epidermis. Within the dermis, there is a superficial and deep perivascular lymphocytic infiltrate with increased dermal mucin. There is interface change characterized by basal vacuolization. The histologic features are consistent with connective tissue disease. The presence of a deep inflammatory component favors the diagnosis of lupus erythematosus over dermatomyositis. Clinicopathologic correlation is recommended.

Example 2:

*Clinical history*: Plaque on scalp.

*Diagnosis*: Interface change consistent with lupus erythematosus, see comment.

*Comment*: There is parakeratosis and compact hyperkeratosis overlying the epidermis. Follicular plugging is noted. There is interface change characterized by basal vacuolization and basement membrane thickening. Within the dermis, there is a superficial and deep perivascular lymphocytic infiltrate with increased dermal mucin. The histologic features are characteristic of discoid lupus erythematosus. Clinicopathologic correlation is recommended.

Example 3:

*Clinical history*: Annular lesion.

*Diagnosis*: Skin, arm, punch biopsy: lupus erythematosus, see comment.

*Comment*: The epidermis shows some parakeratosis and hyperkeratosis. There is interface change characterized by basal vacuolization. Within the dermis, there is a superficial perivascular lymphocytic infiltrate and increased dermal mucin. The histologic features and clinical history of an annular lesion are characteristic of lupus erythematosus. Clinicopathologic correlation is recommended.

## Sample Reports: Dermatomyositis (See Also Sample Reports for Lupus Erythematosus)

Example 1:

*Clinical history*: Rule out dermatomyositis.

*Diagnosis*: Skin, arm, punch biopsy: Interface dermatitis consistent with dermatomyositis, see comment.

*Comment*: There is interface change characterized by basal vacuolization in association with a mild superficial perivascular lymphocytic infiltrate and increased dermal mucin. The histologic features are consistent with the diagnosis of dermatomyositis in the appropriate clinical context. Clinicopathologic correlation is recommended.

Example 2:

*Clinical history*: Dermatomyositis vs. lupus erythematosus.

*Diagnosis*: Skin, arm, punch biopsy: Interface dermatitis, see comment.

*Comment*: There is interface change characterized by basal vacuolization in association with a mild superficial perivascular lymphocytic infiltrate and increased dermal mucin. The mild nature of the inflammatory infiltrate could slightly favor dermatomyositis, but lupus erythematosus cannot be excluded. Clinicopathologic correlation is recommended.

## Sample Reports: Graft Versus Host Disease

Example 1:

*Clinical history*: Bone marrow transplant 4 weeks ago now with new rash. Rule out GVHD vs. drug eruption.

*Diagnosis*: Skin, arm, punch biopsy: Acute graft versus host disease, grade 2 of 4, see comment.

*Comment*: The epidermis has a normal basket-weave stratum corneum. Within the dermis, there is a mild superficial perivascular infiltrate with interface change characterized by basal vacuolization and focal satellite cell necrosis. The histologic features are characteristic of acute graft versus host disease, grade 2 of 4. Clinicopathologic correlation is recommended.

Example 2:

*Clinical history*: Drug eruption vs. GVHD.

*Diagnosis*: Skin, arm, punch biopsy: Interface dermatitis, see comment.

*Comment*: Sections demonstrate an interface dermatitis characterized by basal vacuolization with focal satellite cell necrosis. Within the dermis, there is a mild superficial perivascular lymphocytic infiltrate admixed with eosinophils. Given the clinical context of recent stem cell transplantation, the diagnosis of acute graft versus host disease, grade 2 of 4, is favored despite the presence of eosinophils. Clinicopathologic correlation is recommended.

Example 3:

*Clinical history*: Bone marrow transplantation 7 months ago; rule out GVHD.

*Diagnosis*: Skin, arm, biopsy: Lichenoid chronic graft versus host disease, see comment.

*Comment*: The epidermis shows compact hyperkeratosis and a thickened granular layer. Within the dermis there is a mild perivascular to lichenoid lymphocytic infiltrate with scattered melanophages in association with interface change characterized by basal vacuolization and scattered dyskeratotic cells. The histologic features are compatible with lichenoid chronic graft versus host disease.

## PLEVA and PLC: Sample Reports

Example 1:

*Clinical history*: Rule-out LYP vs. PLEVA.

*Diagnosis*: Skin, buttock, punch biopsy: Pityriasis lichenoides et varioliformis acuta (PLEVA), see comment.

*Comment*: There is parakeratosis overlying the epidermis. Within the dermis, there is a superficial and deep perivascular lymphocytic infiltrate with prominent interface change and papillary dermal hemorrhage. Because of the clinical suspicion for possible lymphomatoid papulosis, an immunohistochemical stain for CD30 was performed and compared to appropriate controls. No significant immunoreactivity for CD30 was seen in the dermal infiltrate. In the appropriate clinical context, the histologic features are consistent with the diagnosis of PLEVA. Clinicopathologic correlation is recommended.

Example 2:

*Clinical history*: Rule erythema multiforme vs. PLEVA.

*Diagnosis*: Skin, arm, punch biopsy: Interface dermatitis consistent with Pityriasis lichenoides et varioliformis acuta (PLEVA), see comment.

*Comment*: There is parakeratosis overlying the epidermis. Within the dermis, there is a superficial and deep perivascular lymphocytic infiltrate associated with papillary dermal hemorrhage and interface change with numerous dyskeratotic cells. The histologic features are consistent with PLEVA. The presence of parakeratosis, papillary dermal hemorrhage and the density of the inflammatory infiltrate are against erythema multiforme (EM). Clinicopathologic correlation is recommended.

Example 3:

*Clinical history*: Pityriasis rosea vs. PLC.

*Diagnosis*: Skin, buttock, punch biopsy: Interface dermatitis consistent with pityriasis lichenoides chronica, see comment.

*Comment*: There is near confluent parakeratosis overlying a mildly acanthotic epidermis. Scattered dyskeratotic keratinocytes are present. Within the dermis, there is a superficial perivascular lymphocytic infiltrate associated with interface change characterized by focal basal vacuolization.

## Selected References

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