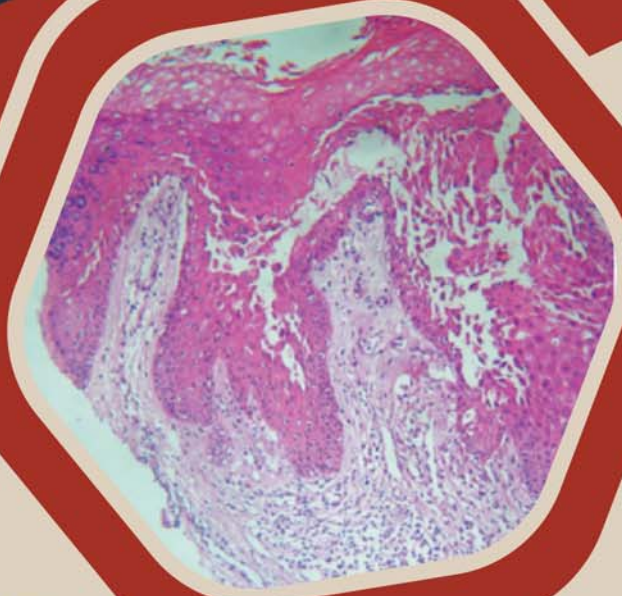
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6th Edition

Fundamentals of PATHOLOGY OF SKIN



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JAYPEE

Contents

SECTION 1: INFLAMMATORY DISEASES

1. **Biopsy** 3
   Rutvi Pandya
2. **Structure of Normal Skin** 7
   Yashika J Doshi
3. **Stains Used in Dermatology** 22
   Chandana Shajil
4. **Definitions in Dermatopathology** 29
   Kondeti Kavya
5. **Approach to Diagnosis** 64
   Shradha Singh
6. **Spongiotic Dermatosis** 75
   Pooja Kanumuru
7. **Reactive Erythemas** 84
   Pooja Kanumuru
8. **Lichenoid Tissue Reactions** 90
   Chandana Shajil
9. **Psoriasiform Reactions** 102
   Kondeti Kavya
10. **Bullous Diseases** 112
    Chandana Shajil
11. **Connective Tissue Diseases** 127
    Shradha Singh
12. **Vasculitis** 137
    Shradha Singh
13. **Granulomas** 155
    Kondeti Kavya
14. **Disorders of Pigmentation** 184
    Kondeti Kavya

15. **Appendageal Diseases** 189
Chandana Shajil

16. **Panniculitis** 204
Yashika J Doshi

17. **Genodermatoses** 215
Adrija Datta

18. **Deposition Disorders and Disorder of Elastic Fibers** 223
Padmini CV

19. **Perforating Disorders** 231
Anirudh Padmanabhachar

20. **Histopathology of Infections** 235
Padmini CV

21. **Immunofluorescence and Other Immunomethods of Diagnosis** 252
Raghavendra Rao, Saurodeep Basu

SECTION 2: TUMORS

22. **Tumors** 271
Anirudh Padmanabhachar

23. **Premalignant and Malignant Tumors of Epidermis** 280
Yashika J Doshi

24. **Appendageal Tumors** 294
Padmini CV

25. **Connective Tissue Tumors** 307
Anirudh Padmanabhachar

26. **Melanocytic Tumors** 320
Padmini CV

27. **Cutaneous Infiltration Disorders** 334
Padmini CV

28. **Lymphoid Infiltrates of Skin** 342
Chandana Shajil

29. **Clues in Dermatopathology** 357
Adrija Datta

Recommended Readings 363

Index 365

10 Bullous Diseases

Chandana Shajil

Bullous diseases are classified according to the anatomical level (intraepidermal vs. subepidermal) and pathomechanism of the blister [acantholysis, basement membrane zone (BMZ) destruction, cytolysis, reticular degeneration and spongiosis].

Table 10.1 presents different immunobullous diseases and their key features.

TABLE 10.1: Different immunobullous diseases and their features.

Disease	*Level of bulla formation*	*Antigen*	*Diagnosis by immunofluorescence*
Intraepidermal bullous diseases			
Pemphigus vulgaris	*Suprabasal:* Above basal layer	Adhesion molecule in ICS, desmoglein 3 when only mucous membrane is involved and desmoglein 1 and 3 in mucocutaneous involvement	*Fish-net pattern:* IgG and C3 deposits in ICS
Pemphigus foliaceus and pemphigus erythematosus	*Subcorneal:* Below stratum corneum	Adhesion molecule in ICS, desmoglein-1	*Fish-net pattern:* IgG deposits in ICS (more pronounced in the upper epidermis); additional deposits at DEJ in pemphigus erythematosus
IgA pemphigus (subcorneal pustular dermatosis variant)	Subcorneal	Desmocollin 1	*Fish-net pattern:* IgA deposits preferentially in ICS of upper epidermis
IgA pemphigus (intraepidermal neutrophilic dermatosis)	Spinous layer	Desmoglein 1 or 3	*Fish-net pattern:* IgA deposits preferentially in ICS of lower epidermis
Paraneoplastic pemphigus	Suprabasal	Desmoglein 1, 3, Desmoplakin 1, 2, Envoplakin, BP230, Periplakin	IgG and C3 deposit in ICS accompanied by linear or granular C3 deposit along DEJ
Subepidermal bullous diseases			
Bullous pemphigoid	Subepidermal	Antigen in hemidesmosomes; BPAg1, BPAg2	Linear IgG and C3 at DEJ

Continued

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Continued

Disease	*Level of bulla formation*	*Antigen*	*Diagnosis by immunofluorescence*
Herpes gestationis	Subepidermal	Antigen in hemidesmosomes; BPAg2	Linear IgG and C3 at DEJ
LABD	Subepidermal	Antigen in hemidesmosome; LABD97, LAD-1	Linear IgA deposit at DEJ
Dermatitis herpetiformis	Subepidermal	Tissue transglutaminase	Granular IgA at the dermal papillae
EBA	Subepidermal	Type VII collagen	Linear IgG and C3 at DEJ
Cicatricial pemphigoid	Subepidermal	Laminin 5, β4 subunit of the α6β4 integrin, BPAg2, BPAg1	Linear IgG and C3 at DEJ
Bullous systemic lupus erythematosus;	Subepidermal	Type VII collagen	Linear IgG and C3 at DEJ

(DEJ: dermoepidermal junction; EBA: epidermolysis bullosa acquisita; ICS: intercellular space; LABD: linear immunoglobulin A bullous dermatosis)

INTRAEPIDERMAL BULLAE

Table 10.2 shows the pathomechanism of different intraepidermal bullous diseases.

Cytology

Cytological examination is the first crucial step in the diagnosis of bullous diseases. *Tzanck test* is performed as follows:

- Rupture a bulla and remove the roof.
- Scrape the floor with a glass slide or blunt edge of a scalpel.
- Make a smear on a slide and stain with Leishman stain.
- Look for *Tzanck cell or acantholytic cell*: A large cell, 20 µm in size, rounded, with a dark central nucleus, surrounded by a zone of pink cytoplasm and a peripheral rim of dark pink condensed cytoplasm. These cells may be found either singly or in groups.

Note: Tzanck test is also useful in the following situations:
- Herpes zoster and simplex—multinucleated keratinocyte giant cells
- Molluscum contagiosum-Henderson-Patterson bodies
- Squamous cell carcinoma, basal cell carcinoma—atypical cells
- Mastocytoma—mast cells

Acantholytic Disorders

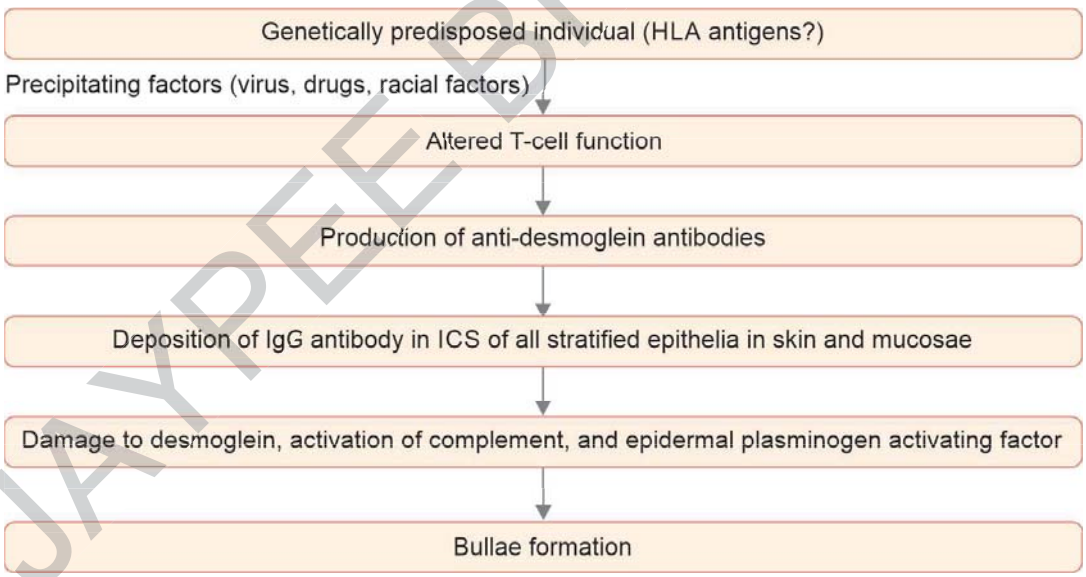
The pemphigus group of diseases is caused by loss of intercellular adhesion at different levels within epidermis either immunologically (as in pemphigus vulgaris, pemphigus vegetans, and pemphigus erythematosus) or nonimmunologically (Hailey–Hailey disease). Events in etiopathogenesis of pemphigus are presented in **Flowchart 10.1**.

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TABLE 10.2: Different intraepidermal bullous diseases and their pathomechanism.

Level	*Mechanism*		*Disease (predominant cell type)*
Subcorneal	Spongiotic		Miliaria crystallina (scant inflammatory cells)
	Acantholytic		Pemphigus foliaceous (acantholytic cells), SSSS (scant inflammatory cells)
Bullae in spinous layer	Spongiotic	Lymphocytic	CD, Miliaria rubra, eczematous drug rash
		Eosinophilic	Incontinentia pigmenti (first stage), spongiotic drug rash, preacantholytic stage of pemphigus
	Acantholytic		Hailey–Hailey disease, pemphigus vulgaris and foliaceus (extension of bullae)
	Cytolysis	With inflammation	Herpes group infection (mixed inflammatory cells)
		Without inflammation	Friction blister (scant inflammatory cells)
		Granular degeneration	Epidermolytic hyperkeratosis (scant inflammatory cells)
Suprabasal	Acantholytic		Pemphigus vulgaris and variants, Darier disease
	Cytolysis		Friction blister, epidermolytic hyperkeratosis

(CD: contact dermatitis; SSSS: staphylococcal scalded skin syndrome)



FLOWCHART 10.1: Etiopathogenesis of bullae in the pemphigus group of diseases.

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Pemphigus Vulgaris

The hallmark of pemphigus vulgaris is intraepidermal acantholytic bulla **(Figs. 10.1 and 10.2)** with minimal inflammation. Binding of antibodies to antigens in intercellular substance (ICS) activates plasminogen leading to lysis of ICS resulting in bulla formation. Complement involvement is seen in around 50% cases and hence the paucity of inflammation.

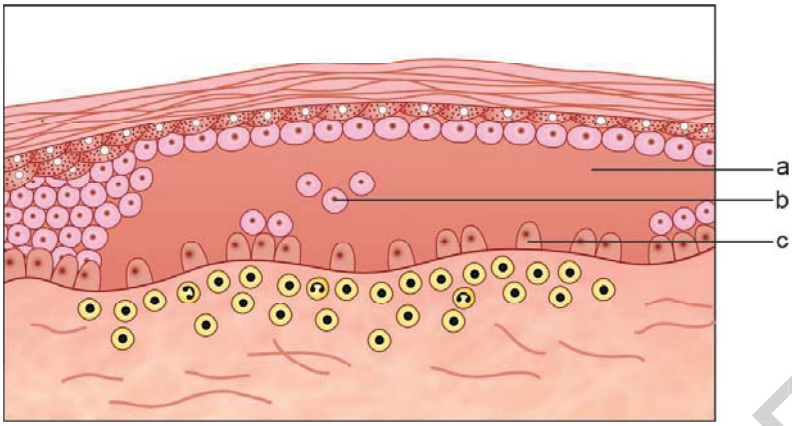
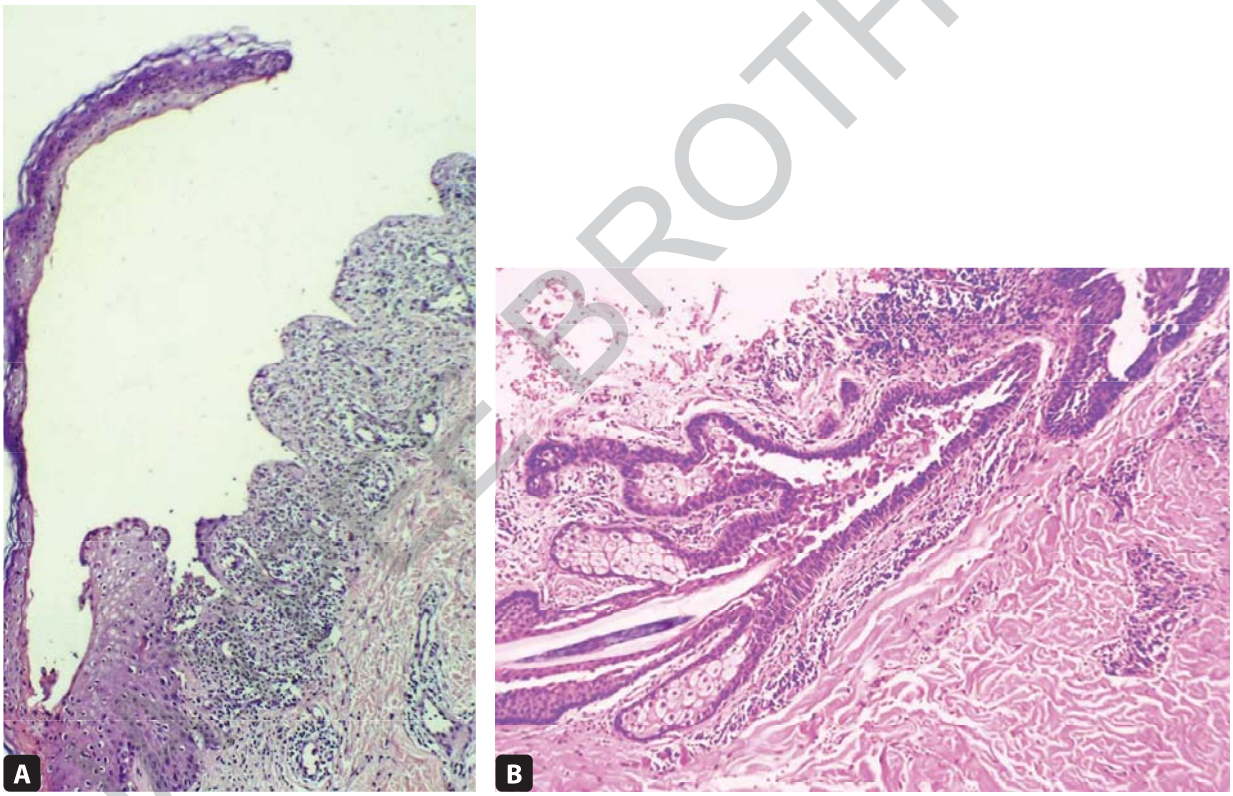


FIG. 10.1: Schematic diagram of pemphigus vulgaris showing: (a) Suprabasal cleft with (b) Acantholytic cells and (c) Tombstone appearance of basal cells with minimal dermal infiltrate.



FIGS. 10.2A AND B: (A) Pemphigus: Low-power view showing suprabasal cleft with tombstone appearance (10×). Note the villous projections. (B) Follicular involvement in pemphigus vulgaris (10×).

- Intraepidermal bulla/cleft in the basal layer and lower layers of stratum malpighi containing acantholytic cells and a few inflammatory cells
- Basal cells lining the floor of the bulla appear like a "row of tombstones" due to loss of intercellular adhesion, without complete detachment from the dermoepidermal junction (DEJ)
- Acantholytic cells (Tzanck cells) in bulla cavity are keratinocytes that have detached from each other. Characteristic features of acantholytic cells:

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- Rounded shape (polygonal keratinocyte becomes rounded after losing their desmosomal attachments).
- Dense eosinophilic cytoplasm, particularly in the periphery
- Central pyknotic nucleus with perinuclear halo

These changes, when progressive, lead to dyskeratosis as seen in Darier's disease.

- Acantholytic changes involving hair follicle
- Dermis shows mild perivascular chronic inflammatory infiltrate that includes eosinophils.

Other less common features in pemphigus include the following:

- Dermal papilla lined by a single layer of basal cells forming finger-like projection into blister cavity, called villi **(Fig. 10.3A)** (more common in Hailey–Hailey disease and Darier's disease) with the basal cells appearing like a 'row of tombstones' **(Fig. 10.3B)**.
- Eosinophilic spongiosis (earliest recognizable change), i.e., the presence of eosinophils amidst keratinocyte showing spongiosis—seen in early lesions of pemphigus
  As lesions age, erosion and ulceration may be seen with a mixed inflammatory reaction.

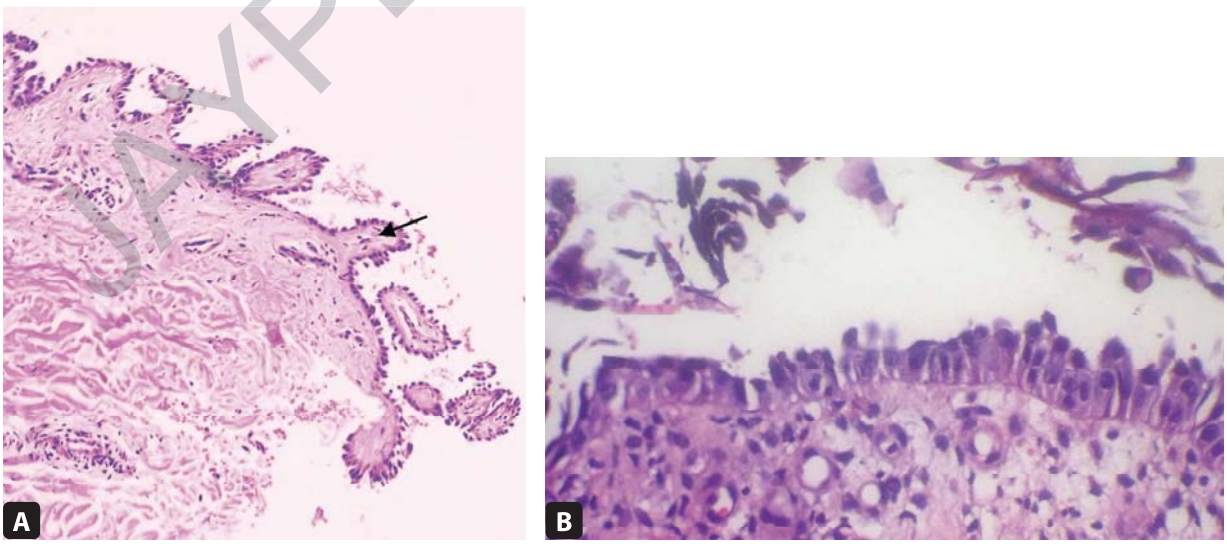
Direct immunofluorescence (DIF): DIF demonstrates a fishnet-like deposition of IgG and, to a lesser extent, C3, IgM, and IgA. DIF positivity is more pronounced in the deeper layers of epidermis.

Pemphigus Vegetans

Pemphigus vegetans is a variant of pemphigus vulgaris showing additional features:

- Papillomatosis
- Epidermal hyperplasia [The reason for this epidermal hyperplasia is not precisely known. It may either be due to regional predisposition (as pemphigus vegetans is mostly seen in intertriginous areas) or due to influence of cytokines.]
- Pronounced villi formation

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FIGS. 10.3A AND B: (A) Pemphigus showing a villous projection (black arrow) within the bullous cavity (10×). Tombstone arrangement of basal cells is characteristic. (B) Higher magnification view of row of tombstone appearance.

- Focal heavy collections of inflammatory cells—eosinophil and sometimes neutrophil, often forming microabscesses in the epidermis. Eosinophilic spongiosis is more prominent in the early lesions of Hallopeau type than the Neumann type.
- Acantholysis is localized and may be missed easily.
- Upper epidermis contains a heavy infiltrate of lymphocytes and eosinophils, with some neutrophils.

Differential Diagnosis

"Pustular vegetating lesion" is an important clinical problem and is frequently biopsied with the following differential diagnosis:

- Pyoderma vegetans and blastomycosis-like pyoderma are distinguished by the absence of acantholysis, predominance of neutrophils over eosinophils in epidermis, and DIF negativity. The polymorphs may be accumulated as small microabscesses in epidermis as well as in dermis.
- Halogenoderma (bromoderma and iododerma) are characterized by pseudoepitheliomatous hyperplasia, intraepidermal microabscesses (neutrophils, eosinophils, desquamated keratinocytes), and granulomatous inflammation. A clinical history of past exposure to iodine or bromide containing topical agent, systemic drug (expectorant/tonic), or radiocontrast agent (intravenous pyelogram or myelogram) is helpful.
- Infective conditions such as sporotrichosis and atypical mycobacterial infection show a suppurative granuloma, and special stains for microbes are sometimes helpful in identifying these conditions.

Pemphigus Foliaceus (Fig. 10.4)

- Subcorneal clefts are present. As the cleavage is more superficial within the granular layer, the roof is thinner and therefore gets removed easily—intact bulla may not always be seen.
- Acantholytic cells are smaller, fewer, and diamond shaped as they are derived from upper stratum malpighi.

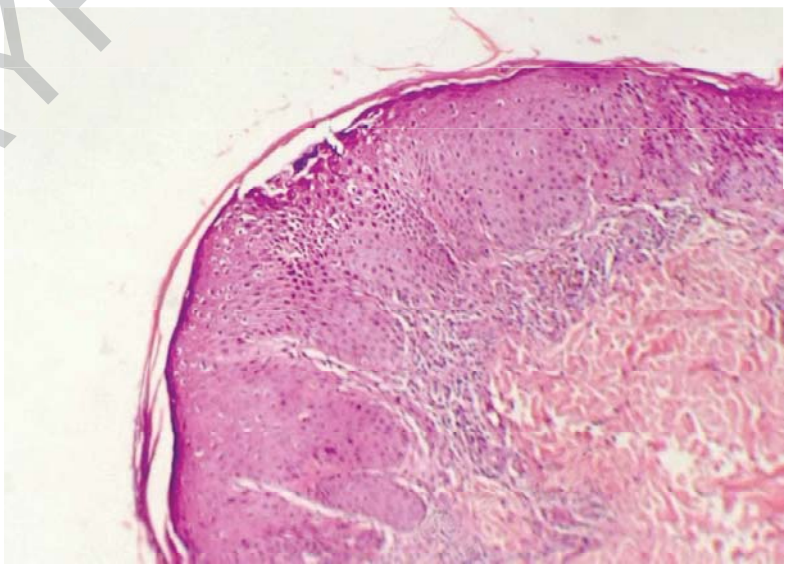


FIG. 10.4: Pemphigus foliaceus showing a subcorneal cleft with few acantholytic cells (10×).

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- Dyskeratotic granular keratinocytes are more common, as acantholytic cells are derived from a keratinized stratum granulosum, unlike in pemphigus vulgaris (keratinization has just started in basal cells).
- As in pemphigus vulgaris, eosinophilic spongiosis may be seen in early lesions.
- *DIF:* Intercellular staining for IgG and C3 is more intense in the superficial layers of epidermis.

Pemphigus Erythematosus

Pemphigus erythematosus is primarily a clinical and immunological diagnosis. Histological signs are indistinguishable from those of pemphigus foliaceus, of which this is a variant. DIF demonstrates a linear IgG band at the DEJ, in addition to the intercellular deposition of IgG in a fish-net pattern, prominent in superficial epidermis.

Pemphigus Herpetiformis

Pemphigus herpetiformis is a variant of pemphigus foliaceus showing dermatitis herpetiformis-like clinical features and pemphigus-like DIF findings. Histopathology shows eosinophilic spongiosis with intraepidermal vesicle formation and minimal acantholysis.

Paraneoplastic Pemphigus

Paraneoplastic pemphigus shows polymorphic features clinically and histologically and may be difficult to diagnose. It exhibits varying histological features depending on the clinical presentation:

- Suprabasal acantholysis and clefting like in pemphigus vulgaris
- Apoptotic/necrotic keratinocytes with vacuolar interface dermatitis like erythema multiforme or lichenoid interface dermatitis like lichen planus
- Gross epithelial destruction with interface dermatitis mimicking severe graft-versus-host disease is sometimes seen.
- A subepidermal clefting in pemphigoid-like lesions may be seen rarely.

Note: Acantholysis may be focal and missed in a single biopsy. So, multiple biopsies with an increased number of sections are recommended in a suspected case of paraneoplastic pemphigus.

Hailey–Hailey Disease (Chronic Benign Familial Pemphigus) (Fig. 10.5)

- Intraepidermal cleft formation
- Extensive acantholysis occurring throughout stratum malpighi (unlike suprabasal acantholysis of pemphigus vulgaris) resulting in a dilapidated brick wall appearance. It occurs at all levels within stratum malpighi (unlike suprabasal acantholysis of pemphigus vulgaris). Unlike pemphigus vulgaris, it rarely results in total separation of cells, and hence keratinocytes are held loosely together by few intact intercellular bridges, giving rise to this appearance.
- Dyskeratotic cells appearing as *corp ronds* are present.
- Differentiating features of pemphigus vulgaris and Hailey–Hailey disease are given in **Table 10.3**.

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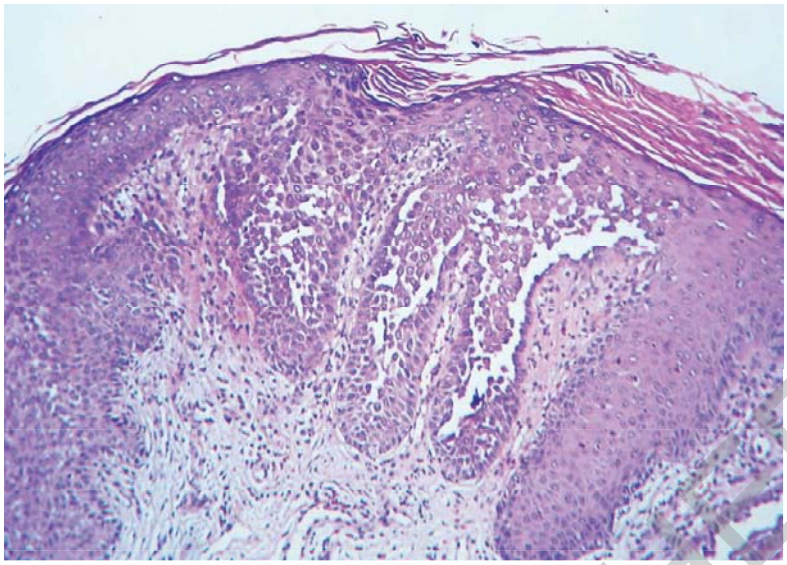


FIG. 10.5: Hailey–Hailey disease (10×) showing a dilapidated brick wall appearance caused by acantholysis at all levels.

TABLE 10.3: Differentiating features of pemphigus vulgaris and Hailey–Hailey disease.

Feature	*Pemphigus vulgaris*	*Hailey–Hailey disease*
Acantholysis	Suprabasal	At all levels in stratum malpighi and more extensive
Appearance	Row of tombstones	Dilapidated brick wall
Type of bulla	One large bulla	Multiple clefts
Clefts and villi	Less common	More common
Dyskeratotic cells	Less common	More common
Acantholytic cell	Common	Less common
Acantholysis in hair follicle	Present	Absent
Direct immunofluorescence	Positive	Negative

Note: Pemphigus vulgaris, Hailey–Hailey disease, and Darier's disease display a spectrum of acantholytic–dyskeratotic cells. Acantholysis is prominent in pemphigus vulgaris, while dyskeratosis is pronounced in Darier's disease.

Darier's Disease

Darier's disease is characterized by suprabasal clefting with acantholysis and prominent keratinocyte dyskeratosis appearing as corps ronds and grains.

Transient Acantholytic Dyskeratosis

Transient acantholytic dyskeratosis is characterized by small foci of acantholysis (usually suprabasal) and dyskeratosis along with superficial dermal infiltrate of lymphocytes and eosinophils. It shows following histological patterns:

- Hailey–Hailey disease-like pattern
- Darier's disease-like pattern
- Spongiotic dermatitis pattern

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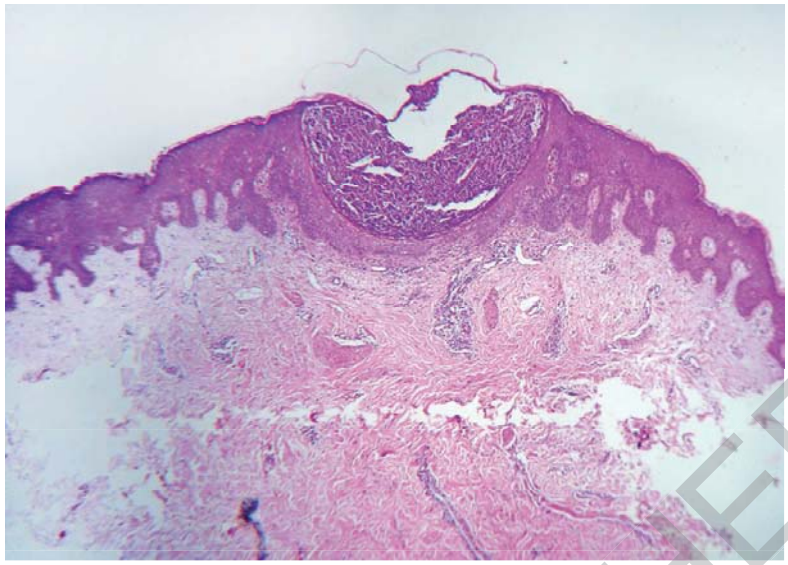


FIG. 10.6: Subcorneal pustular dermatosis showing a unilocular subcorneal pustule.

- Pemphigus-like pattern
- Nonspecific pattern

IgA Pemphigus

IgA pemphigus has two subtypes:

1. Intraepidermal neutrophilic dermatosis type with pustules in suprabasal–stratum spinosum. This variant shows intercellular IgA deposits on DIF.
2. Subcorneal pustular dermatosis (SCPD) which shows subcorneal pustules. Classical SCPD **(Fig. 10.6)** reveals a unilocular subcorneal pustule and is distinguished from SCPD variant IgA pemphigus by the absence of intercellular IgA deposition on DIF and lack of circulating antibodies to epidermal antigens.

SUBEPIDERMAL BULLOUS DISEASES

Classification of subepidermal bullous diseases is shown in **Box 10.1**.

Approach to Diagnosis

Subepidermal bullae can cause confusion in diagnosis, and often DIF is required for confirmation. An early bulla should always be preferred for biopsy, as a late bulla may falsely reveal an intraepidermal blister due to epithelial regeneration. The classification presented in **Box 10.1**, based on cell type forming the inflammatory infiltrate, provides a stepwise approach to diagnosis. However, histology alone may not always be adequate, and DIF and electron microscopy may be necessary to arrive at a final diagnosis.

Bullous Pemphigoid

Histology varies between inflammatory and noninflammatory lesions.

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BOX 10.1: Classification of subepidermal bullous diseases.

- *Subepidermal bullous diseases with acellular dermis:*
  - Porphyria cutanea tarda
  - Pseudoporphyria
  - Epidermolysis bullosa
  - Noninflammatory bullous pemphigoid
  - Noninflammatory epidermolysis bullosa acquisita (EBA)
  - Diabetic bullae
  - Burns and ischemic bulla
- *Subepidermal bullous diseases with cellular dermis:*
  - *Subepidermal bullous diseases with eosinophils:* Bullous pemphigoid, herpes gestationis, some cases of IgA bullous dermatoses of childhood, bullous arthropod bite, bullous drug reaction, Wells' syndrome
  - *Subepidermal bullous diseases with lymphocytes:* Erythema multiforme, bullous fixed-drug reaction (FDR), lichen sclerosus et atrophicans (LSEA), lichen planus pemphigoides, EBA (inflammatory variant), bullous mycosis fungoides
  - *Subepidermal bullous diseases with polymorphs:* Dermatitis herpetiformis, linear IgA bullous disease anti-p200 pemphigoid, anti-p105 pemphigoid, bullous lupus erythematosus (LE), Sweet's syndrome
  - *Subepidermal bullous diseases with mixed infiltrate:* Cicatricial pemphigoid, EBA/bullous LE, bullous pemphigoid, pemphigoid gestationis
- *Subepidermal bullous diseases with mast cells:* Bullous mastocytosis

Inflammatory Lesions (Figs. 10.7 and 10.8)

- Subepidermal unilocular bulla
- Roof usually consists of intact viable epidermis
- Predominantly eosinophilic infiltrate, with few mast cells in dermis. Occasionally, polymorphs/lymphocytes can occur.
- Eosinophilic spongiosis may be seen in early urticarial lesions of bullous pemphigoid.
- Eosinophilic "flame figures" are rarely seen.

Noninflammatory Lesions

In the cell-poor pattern, only few inflammatory cells are found within the blister lumen and dermis **(Fig. 10.9)**.

Bullous Pemphigoid Variants

- Acanthotic epidermis with pseudoepitheliomatous hyperplasia, in pemphigoid vegetans
- Hyperkeratosis, acanthosis, and mild papillomatosis in pemphigoid nodularis
- Lichen planus pemphigoides is a clinical coexistence of lichen planus and bullous pemphigoid lesions in the same patient
- *Bullous pemphigoid–lupus erythematosus overlap:* This is a coexistence of lesions of bullous pemphigoid and lupus erythematosus.
- Vesicular pemphigoid is a clinical variant with small vesicles instead of large bullae.
- Dyshidrosiform pemphigoid is a clinical variant occurring on palms and soles, with histology as above.
- Pretibial pemphigoid is a clinical variant with lesions limited to the pretibial region.

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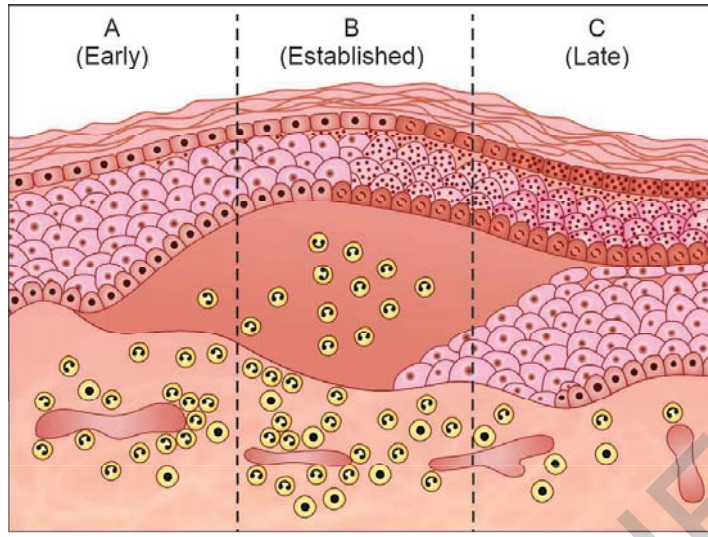


FIG. 10.7: Bullous pemphigoid: (A) Subepidermal cleft; (B) Subepidermal bulla with eosinophilic infiltrate; and (C) Re-epithelization resulting in subepidermal bulla becoming intraepidermal bulla.

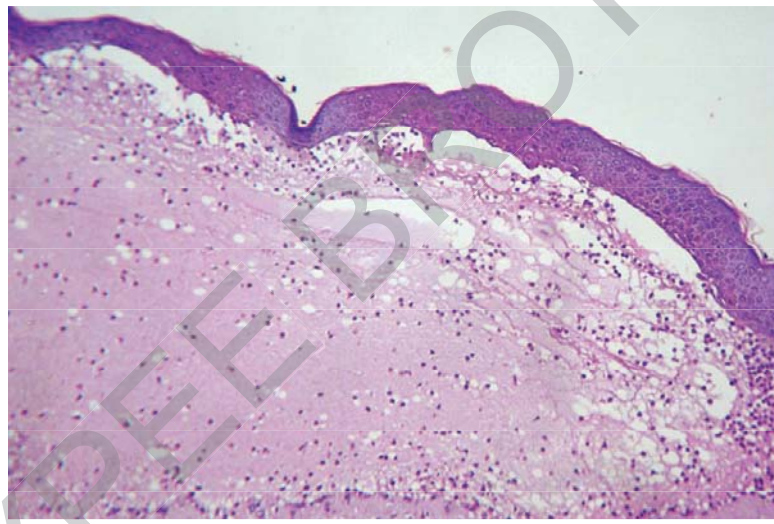


FIG. 10.8: Bullous pemphigoid showing subepidermal bulla with many eosinophils and lymphocytes (10×).

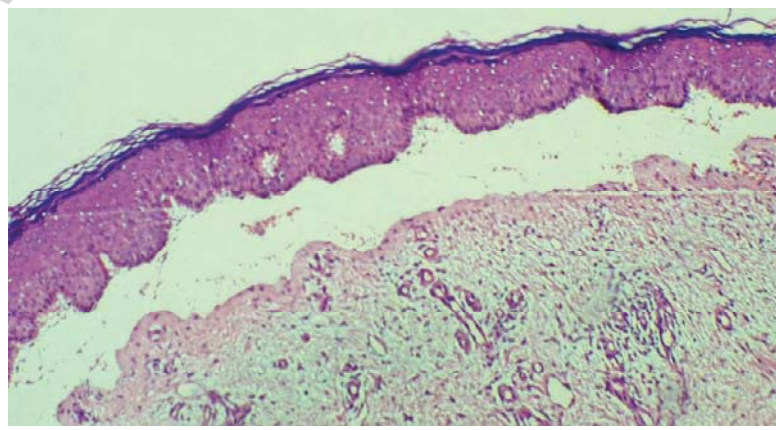


FIG. 10.9: Bullous pemphigoid showing a subepidermal blister with minimal infiltrate (20×).

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Herpes Gestationis

Herpes gestationis is a pruritic vesiculobullous dermatosis of pregnancy due to formation of antibodies against bullous pemphigoid-antigen 2 (180 kD).

- *Early urticarial lesion shows:*
  - Intense edema in papillary dermis
  - Mixed infiltrate (huge number of eosinophils with a mixture of lymphocytes and histiocytes) in upper and mid-dermis with perivascular distribution
  - Necrotic basal keratinocytes and eosinophilic spongiosis are seen occasionally.
- *Vesiculobullous lesions show in addition to features of early urticarial phase:*
  - Small subepidermal clefts/bullae
  - Eosinophilic microabscess in dermal papillae

Note

- Herpes gestationis, like dermatitis herpetiformis, is an intensely pruritic disorder and hence only small clefts are seen; large bullae are rare.
- Pruritic urticarial papules and plaques of pregnancy (PUPPP) is an important differential diagnosis for early lesions of herpes gestationis. PUPPP shows mild spongiosis with eosinophils in epidermis and dermis. It is differentiated from herpes gestationis by the absence of subepidermal blister and DIF negativity.

Cicatricial Pemphigoid

Cicatricial pemphigoid is a rare blistering disorder involving mucosa and skin, leading to scarring. Diagnosis of cicatricial pemphigoid is often difficult as it shows features similar to bullous pemphigoid/epidermolysis bullosa acquisita (EBA). DIF findings are the same as those of bullous pemphigoid.

Main Features

- Subepidermal bulla
- Mild-to-moderate mixed infiltrate (predominantly neutrophils and lymphocytes ± eosinophils) at the base and edge of bullae. Plasma cells are present in mucosal biopsy.
- Evidence of scarring—thick collagen bundles
- Sometimes, epidermal change mimics lichenoid lymphocytic infiltrate of lichen planus.

Dermatitis Herpetiformis (Figs. 10.10 and 10.11)

Because of intense pruritus, vesicles and bullae are rarely present. Papules/papulovesicles are the lesions to be biopsied for the best results. Histopathology finding depends on the age and type of lesion.

- Early erythematous lesions show:
  - Papillary tip microabscess (tip of dermal papilla contains cluster of neutrophils)
  - Vacuolar degeneration of basal cells
  - Papillary dermal edema
  - Fibrin in dermal papillae imparting a necrotic appearance
- *Late papulovesicle shows:*
  - Subepidermal vesicle with intact interpapillary ridge in early stage resulting in multilocular blister; later, the ridges are destroyed resulting in unilocular blister.
  - The blister cavity contains fibrin, neutrophil, and leukocytoclastic debris.

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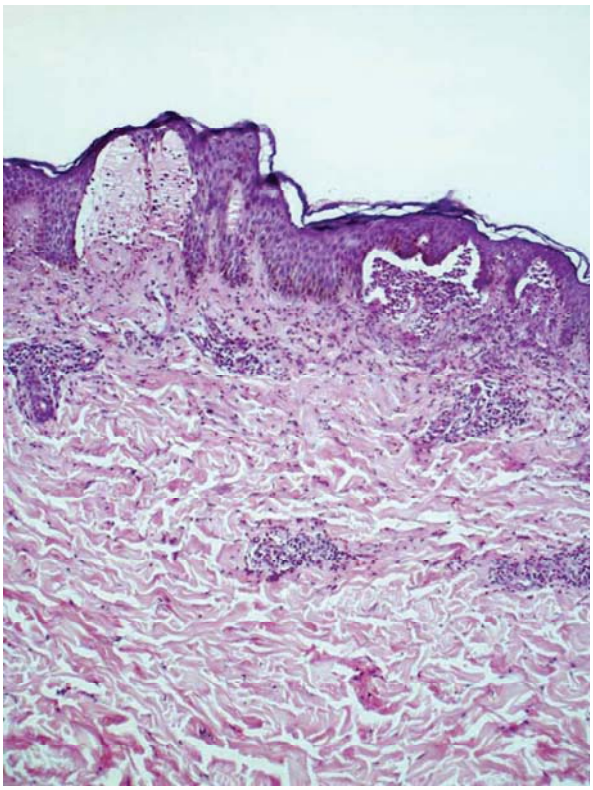


FIG. 10.10: Dermatitis herpetiformis showing papillary microabscess (40×).
Courtesy: Professor Jag Bhawan, USA.

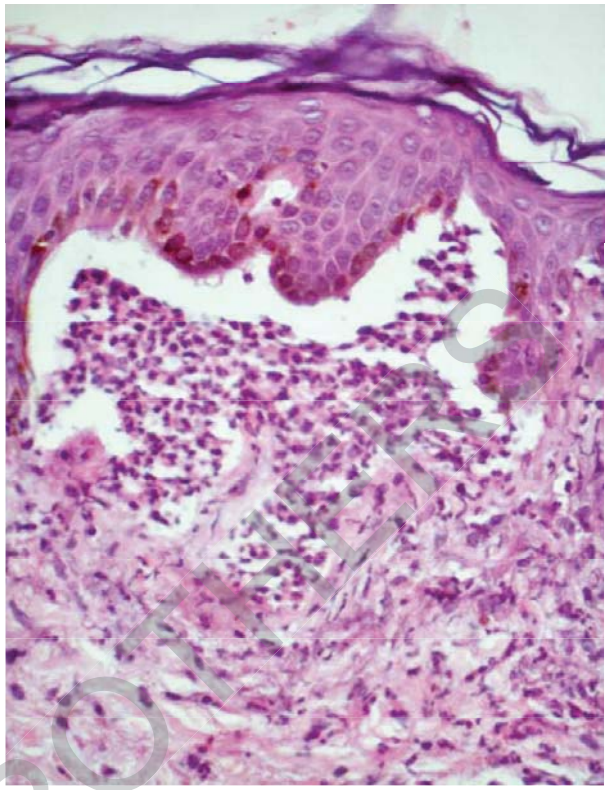


FIG. 10.11: Dermatitis herpetiformis: Close-up view of neutrophilic papillary microabscess (10×).
Courtesy: Professor Jag Bhawan, USA.

- Perivascular lymphocytic/lymphohistiocytic infiltrate with presence of eosinophils and neutrophils occasionally
- DIF shows granular deposits of IgA in the tips of dermal papilla.

Linear IgA Bullous Disease of Adults and Chronic Bullous Dermatosis of Childhood

Histology resembles dermatitis herpetiformis; however, the neutrophils are more commonly seen as uniform widespread infiltration along DEJ, instead of forming microabscesses. DIF to demonstrate homogenous linear IgA deposition along DEJ is required to diagnose these cases.

Epidermolysis Bullosa Acquisita

- Subepidermal bullae containing fibrin, few inflammatory cells and having an intact roof.
- Inflammatory lesions show predominantly neutrophils with lymphocytes containing infiltrate, while noninflammatory lesions reveal sparse lymphocytic infiltrate.

Epidermolysis bullosa acquisita cannot be distinguished from bullous pemphigoid with certainty on the basis of histology alone and needs DIF examination on salt-split skin for accurate diagnosis.

Note: Linear IgG deposition follows a n-serrated pattern in bullous pemphigoid and u-serrated pattern in EBA. In a salt-split skin, the immunoreactants line the roof of the induced split in bullous pemphigoid and floor of the split in EBA.

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Porphyria Cutanea Tarda

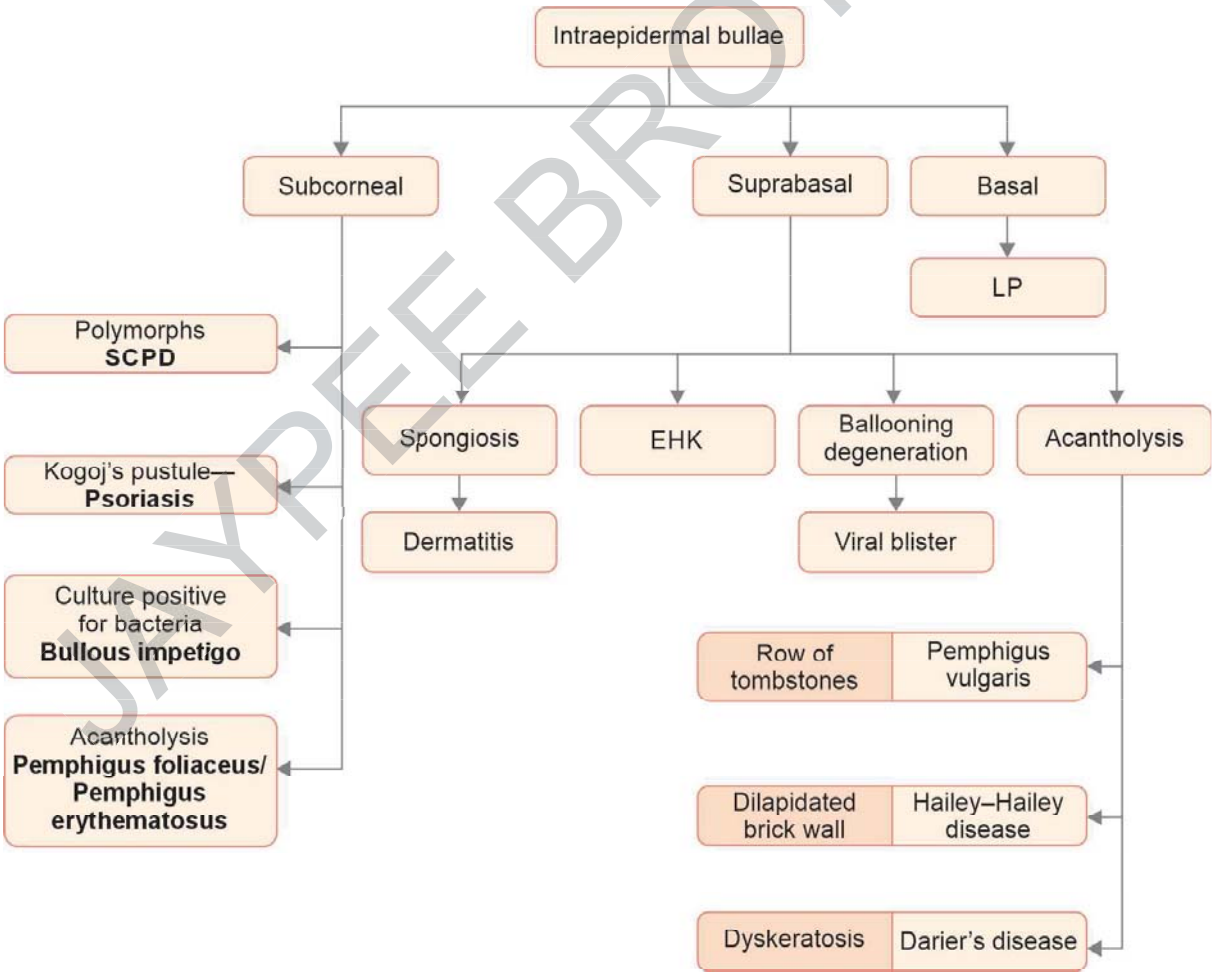
1. Subepidermal bullae
2. Due to deposition of periodic acid–Schiff (PAS)-positive material in the basement membranes, dermal papillae become taut and protrude into bulla cavity. This is called *festooning* or *naked villae.*
3. Mild perivascular mononuclear infiltrate
4. PAS-positive material in BMZ and around upper dermal blood vessels.

Bullous Mastocytosis

Bullous mastocytosis shows intense edema with strikingly monomorphic mast cell infiltrate.

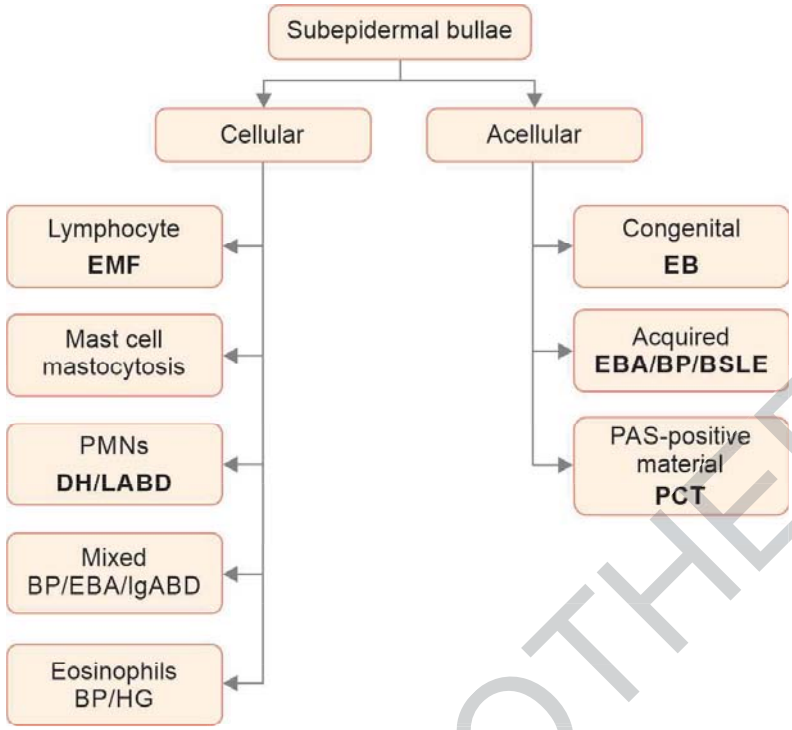
DIAGNOSTIC ALGORITHMS

Algorithms for diagnosis of intraepidermal bullous diseases and subepidermal bullous diseases are shown in **Flowcharts 10.2 and 10.3**, respectively.



FLOWCHART 10.2: Algorithm for intraepidermal bullous diseases.
(EHK: epidermolytic hyperkeratosis; LP: lichen planus; SCPD: subcorneal pustular dermatosis)

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FLOWCHART 10.3: Algorithm of subepidermal bullous diseases.

(BP: bullous pemphigoid; BSLE: bullous systemic lupus erythematosus; CBDC: chronic bullous dermatosis of childhood; DH: dermatitis herpetiformis; EBA: epidermolysis bullous aquisita; EMF: erythema multiforme; HG: herpes gestationis; PCT: porphyria cutanea tarda)

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- It includes a large number of colored photographs and schematic diagrams, wherever required.
- Key points are highlighted in italics and textboxes.
- Simple explanations are provided for concepts, wherever necessary.
- The book will continue to serve as a ready reckoner for students as well as for the practicing dermatologists and pathologists.

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