SKIN

VESICULAR LESIONS

Kenneth Alonso, MD, FACP

Differentiation of vesicular lesions

- Granular layer:
- Friction blister
- Pemphigus foliaceus
- Subcorneal pustular dermatosis
- Staphylococcal scalded-skin syndrome/bullous impetigo
- Spinous layer:
- Eczematous dermatitis
- Herpesvirus infection
- Familial benign pemphigus

Differentiation of vesicular lesions

- Suprabasal:
- Pemphigus vulgaris
- Darier's disease
- Basal layer:
- Erythema multiforme
- Lupus erythematosus
- Lichen planus
- Epidermolysis bullosa

Differentiation of vesicular lesions

- Junctional (at the lamina lucida):
- Junctional epidermolysis bullosa
- Bullous pemphigoid
- Dermis (below basal lamina):
- Epidermolysis bullosa
- Porphyria cutanea tarda
- Dermatitis herpetiformis

Mechanisms

- Type I reaction
- IgE binds to specific antigen, activating mast cells (or basophils)
- Leads to release of histamine, leukotriene 4, and other cytokines.
- Edema is the result.
- Uriticaria is the clinical presentation in the skin.
- Type II reaction
- T-cell cytotoxicity leads to cell destruction and separation of dermal and epidermal layers.
- Bullous pemphigoid, eczematous dermatitis are clinical presentations in the skin.

Mechanisms

- Type III reaction
- Circulating immune complexes lead to tissue damage.
- Lupus erythematosis is a clinical presentation in the skin.
- Type IV reaction
- T-cell mediation is caused by inflammation from cytokines produced by CD4+ and CD8+ cells.
- Chemical sensitivities may not be immune mediated.

Aphthous ulcer

- Painful mucosal ulcerations
- Often during second decade, diminishing thereafter
- More common in women
- Usually bucal and labial mucosae
- May occur in both upper and lower GI tract and anogenital region
- Etiology unknown
- Recurrent

Aphthous ulcer

- If constant in oropharynx or anogenital region, complex aphthosis
- Consider Behçet's disease (especially if uveitis)
- Consider Crohn's disease

Uriticaria and angioedema

- Uriticaria
- Pruritic, edematous, erythematous plaques.
- Perivascular edema.
- Dermal lesion.
- Angioedema
- Pruritic, edematous, erythematous plaques.
- Perivascular edema.
- Subcutaneous lesion

Uriticaria

- IgE mediated in majority of cases
- Complement mediated
- Serum sickness (anaphylatoxin release)
- Circulating immune complexes (penicillin)
- Physical onset
- 4% dermatographism
- Cold uriticaria
- Usually in children
- Solar uriticaria
- Histamine mediation

Uriticaria

- Cholinergic uriticaria
- Exercise to point of sweating
- Flushing, burning, wheezing
- Vibratory uriticaria
- Pressure induced direct degranulation of mast cells
- Intense deep dermal inflammatory infiltrate
- Drug induced (non-IgE dependent)
- Opiates
- Radiocontrast material
- Antibiotics
- ACE inhibitors
- NSAIDs

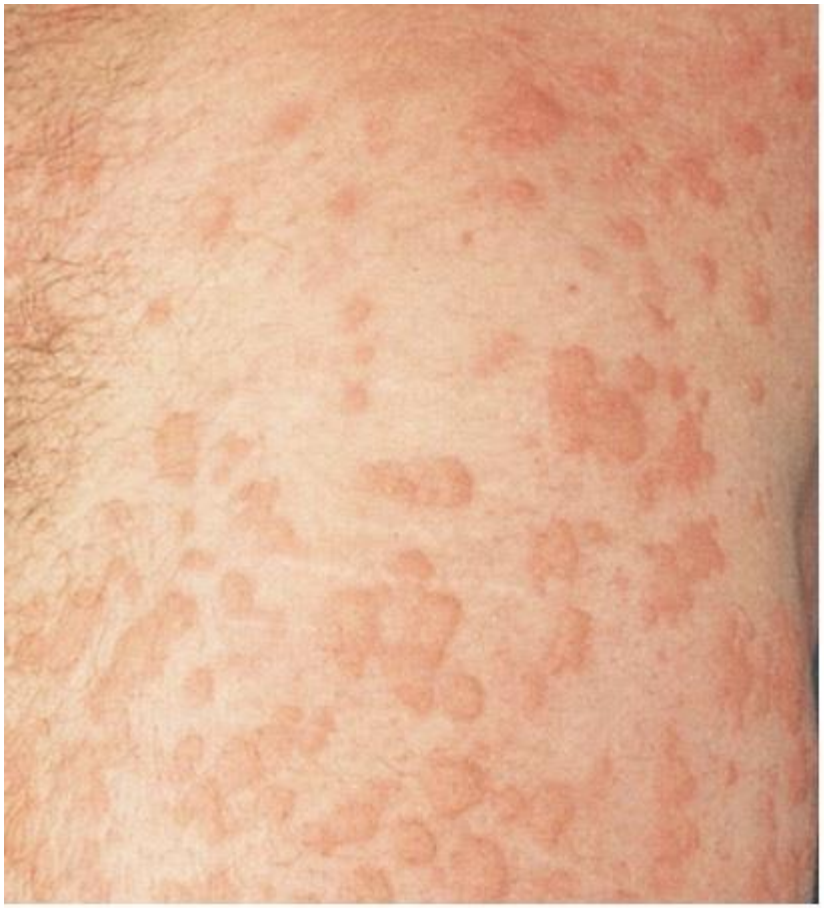
Uriticaria

- Hereditary Angioedema
- Facial swelling
- Laryngeal edema
- Stridor
- Abdominal pain
- Autosomal dominant
- C1 esterase low
- Angioedema-uriticaria-eosinophilia syndrome

Fever, water retention

Cyclic presentation

Fig. e-14 Accessed 07/16/2010



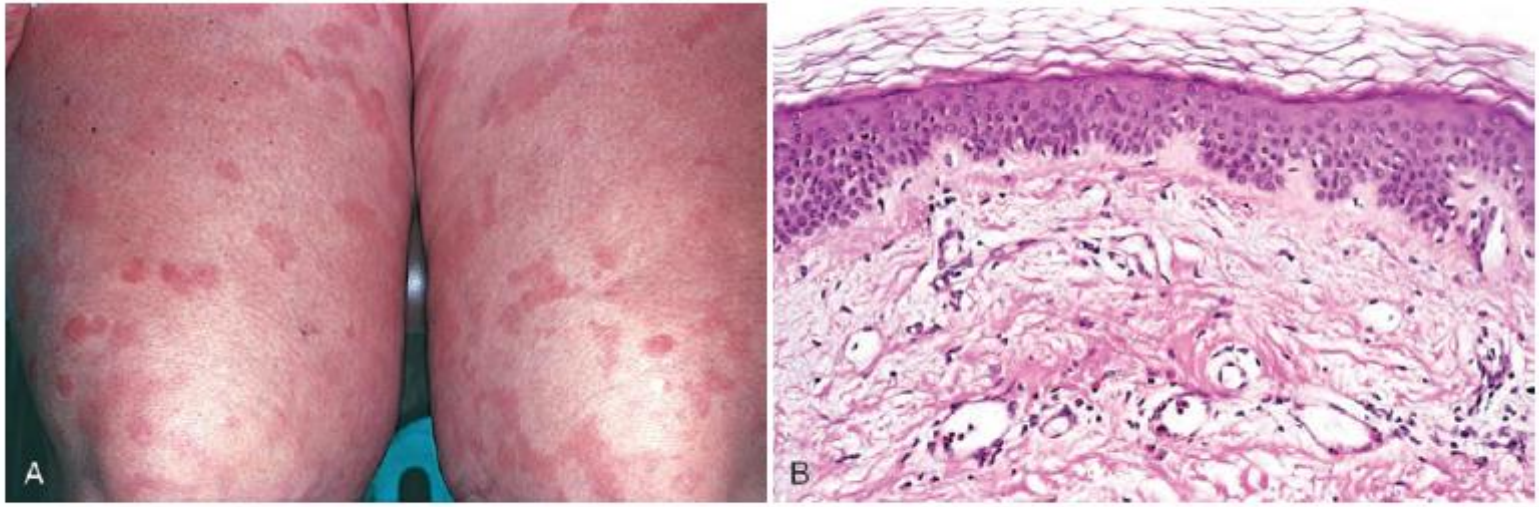
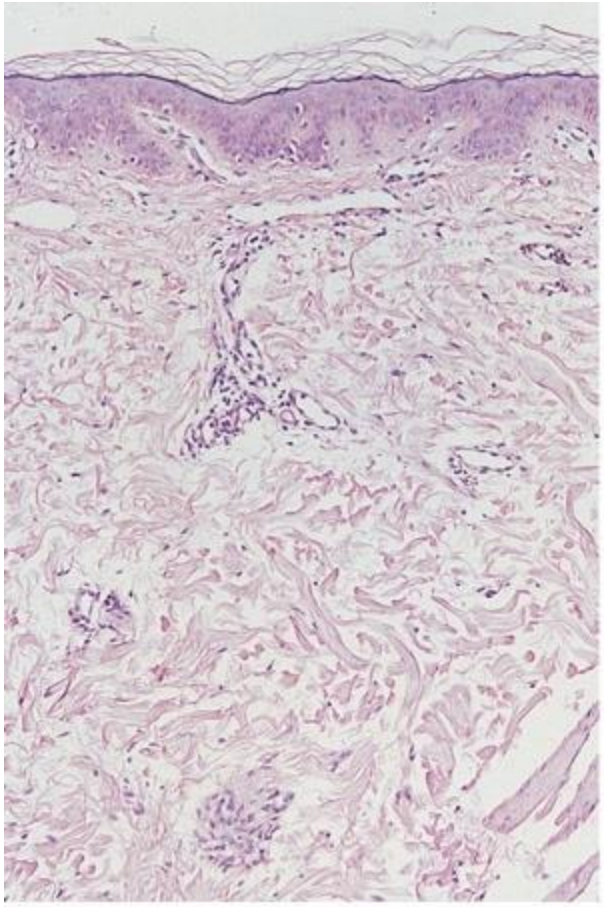


Figure 25-21 Urticaria. **A,** Erythematous, edematous, often circular plaques are characteristic. **B,** Histologically, there is superficial dermal edema, manifested by spaces between collagen bundles, and dilated lymphatic and blood-filled vascular spaces; the epithelium is normal.

Uriticaria



A

Characteristic of the reaction is a sparse, perivascular lymphocytic infiltrate with few eosinophils. Note the slight edema in the dermis and around the postcapillary venules.

Fig. 6-15A
Accessed 07/16/2010

Uriticaria pigmentosa

- Cutaneous form of mastocytosis.
- 0-2 years of age
- Multiple, widely distributed oval, red-brown non-scaling papules and small plaques.
- Soltary mastocytoma may be nodular, pruritic, and show blister formation.
- 10% systemic (C-Kit mutation)
- Dermatographism.
- Darier sign is wheal that occurs when skin is rubbed.

Uriticaria pigmentosa

- Multiple and widely distributed
- Round to oval, red-brown, nons-caling papules and small plaques.
- Solitary mastocytoma presents as a pink to tan brown nodule that may be pruritic or show blister formation.
- Histopathology
- Stellate mast cells about superficial dermal blood vessels
- OR large numbers of closely packed mast cells in upper to mid-dermis.
- Granules best identified with Giemsa or toluidine blue stain

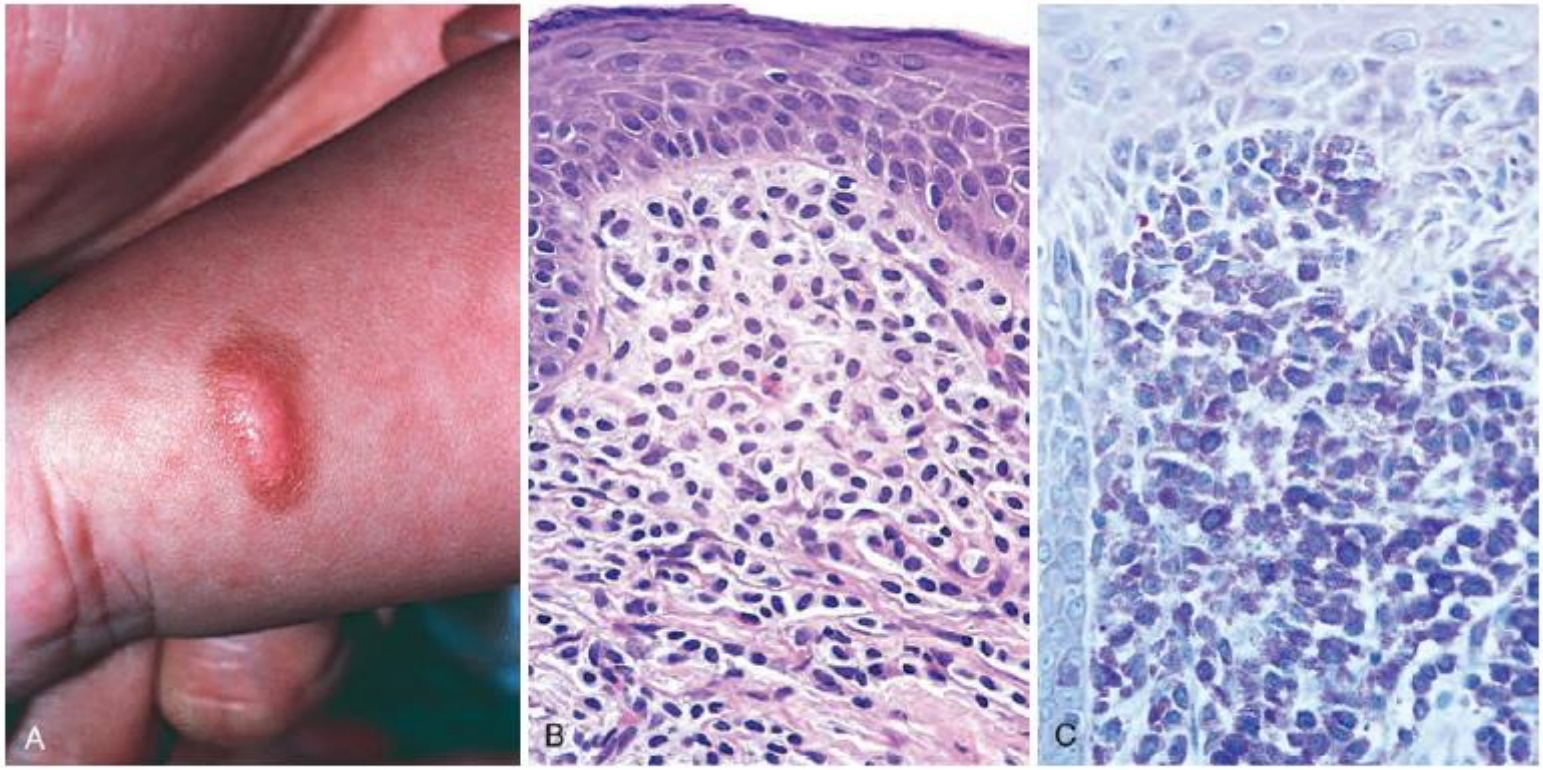
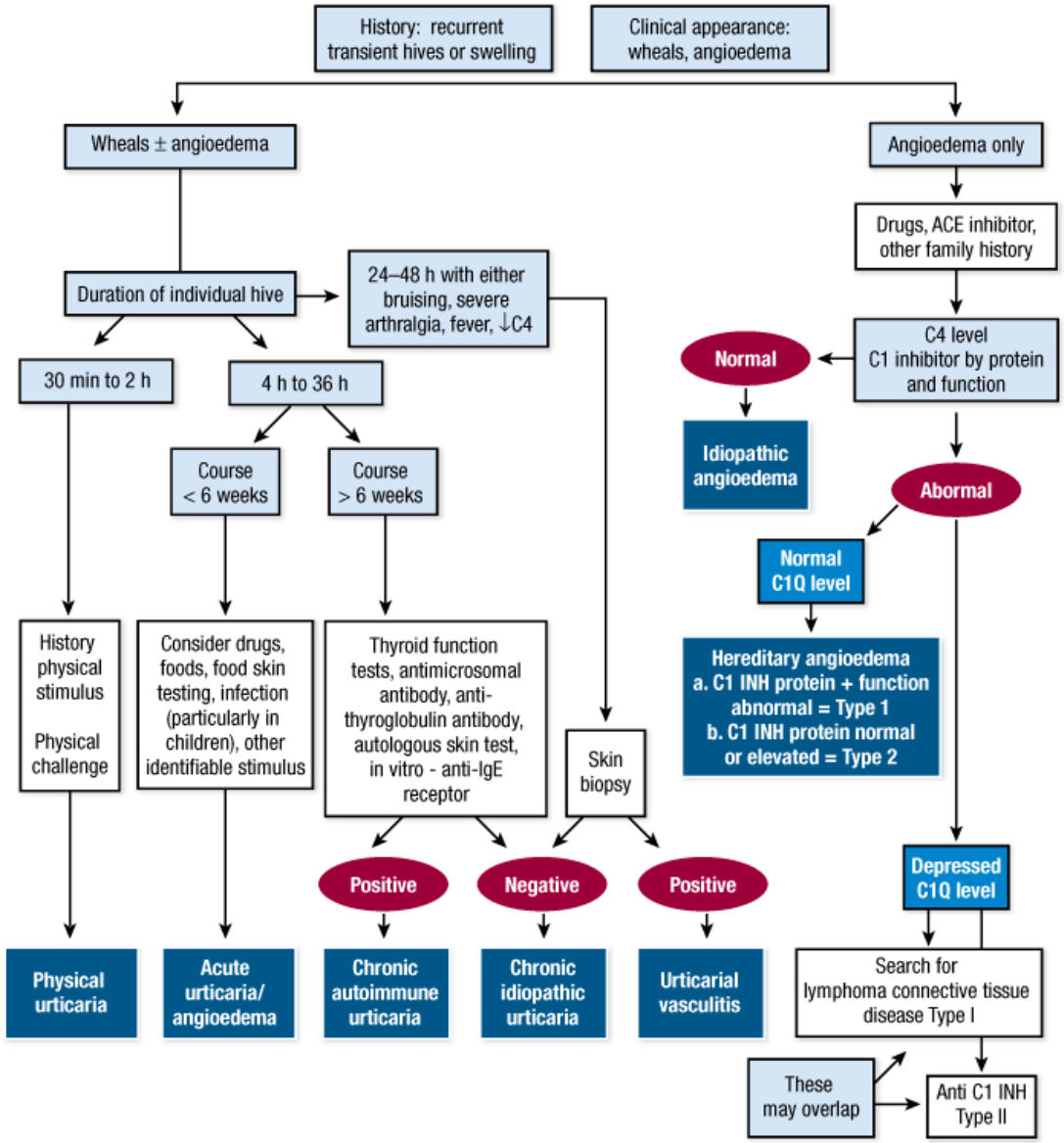


Figure 25-19 Mastocytosis. **A,** Solitary mastocytoma in a 1-year-old child. **B,** By histology, numerous ovoid cells with uniform, centrally located nuclei are observed in the dermis. **C,** Giemsa staining reveals purple "metachromatic" granules within the cytoplasm of the mast cells.



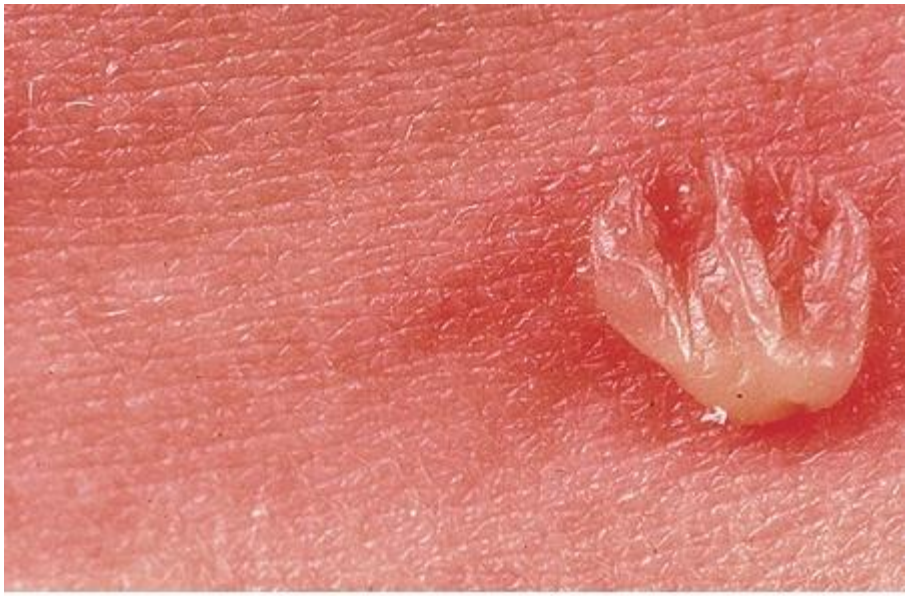
Source: Wolff K, Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell DJ: *Fitzpatrick's Dermatology in General Medicine*, 7th Edition: http://www.accessmedicine.com

Fig. 37-8
Accessed 07/16/2010

Pemphigus vulgaris

- 80% of all cases
- 40-60 years of age
- No sex predilection
- Presents with multiple flaccid vesicles and bullae that rupture easily
- Begins in oral mucosa
- Scalp, face usual sites
- Chest, axillae, groin as other sites
- No pruritis
- Life threatening disease
- Responds to corticosteroids and immunosuppresion

Pemphigus vulgaris



This is the classic initial lesion:

Flaccid, easily ruptured vesicle or bulla on normal-appearing skin.

Ruptured vesicles lead to erosions that subsequently crust.

Fig. 6-9 Accessed 07/16/2010

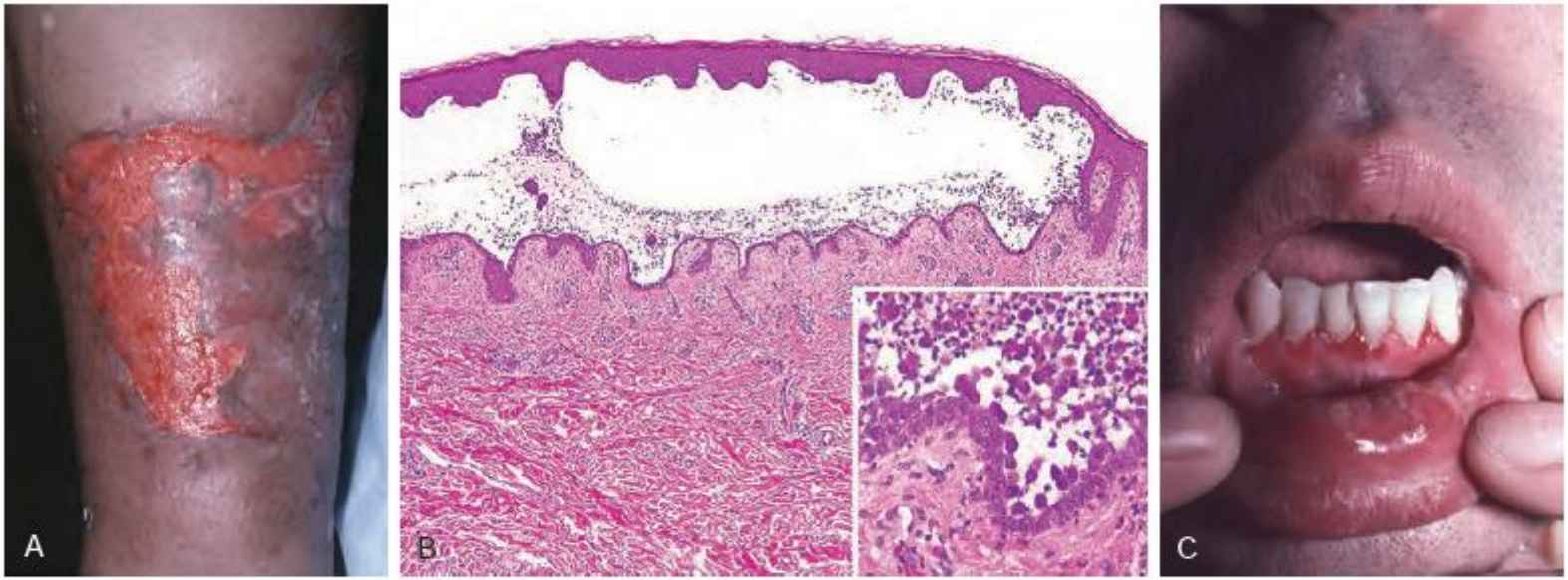


Figure 25-29 Pemphigus vulgaris. **A,** Eroded plaques are formed following the rupture of confluent, thin-roofed bullae, here affecting axillary skin. **B,** Suprabasal acantholysis results in an intraepidermal blister in which dyscohesive (acantholytic) epidermal cells are present *(inset)*. **C,** Ulcerated blisters in the oral mucosa are also common, as seen here on the lip.

Pemphigus vulgaris

- Histology
- Bulla forms above basal layer.
- Acantholysis as cells dissociated.
- Basal cells present as tombstones.
- Direct immunofluorescence to IgG (and C3) in the intercellular substance of the epidermis (desmoglein III)

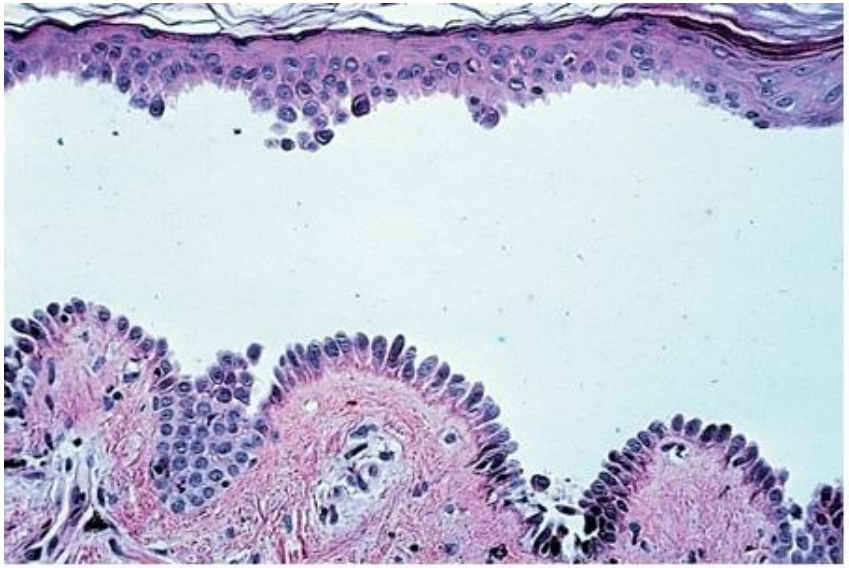


Fig. 52-7 Accessed 07/20/2010

Pemphigus vegetans

- Wart-like plaques.
- Groin, axilla, flexor surfaces.
- May evolve into Pemphigus vulgaris
- Antibody to desmoglein III.

Pemphigus foliaceus

- Erythematous patches and crusted erosions.
- Subcorneal separation of epithelial cells.
- No bullae.
- Usually scalp, face, chest, back
- BUT may present as exfoliative erythroderma
- Antibody to desmoglein 1.
- A common variant in Brazil.
- Arthropod borne

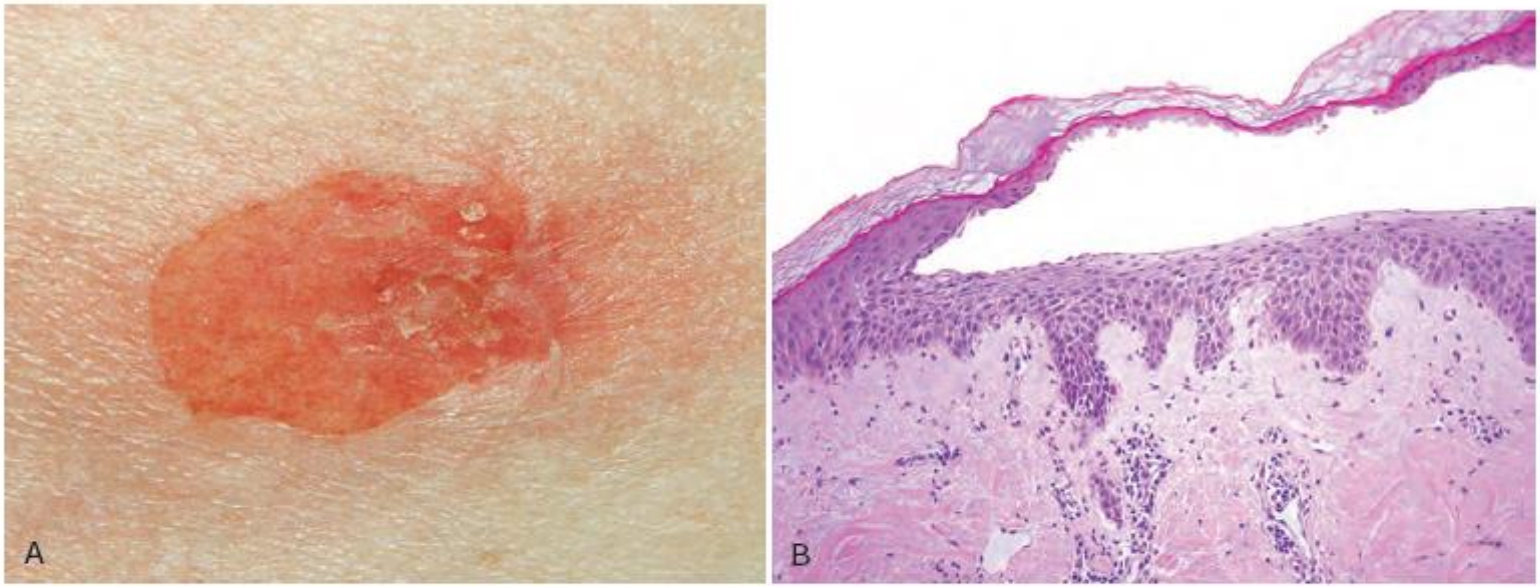


Figure 25-30 Pemphigus foliaceus. **A,** The delicate superficial (subcorneal) blisters are much less erosive than those seen in pemphigus vulgaris. **B,** Subcorneal separation of the epithelium is seen.

Pemphigus erythematosus

- May resemble pemphigus foliaceus
- Malar area of face.
- Confined to seborrheic sites.
- Antibodies against both Dsg-1 and Dsg-3
- Associated with thymoma
- Associated with myasthenia gravis

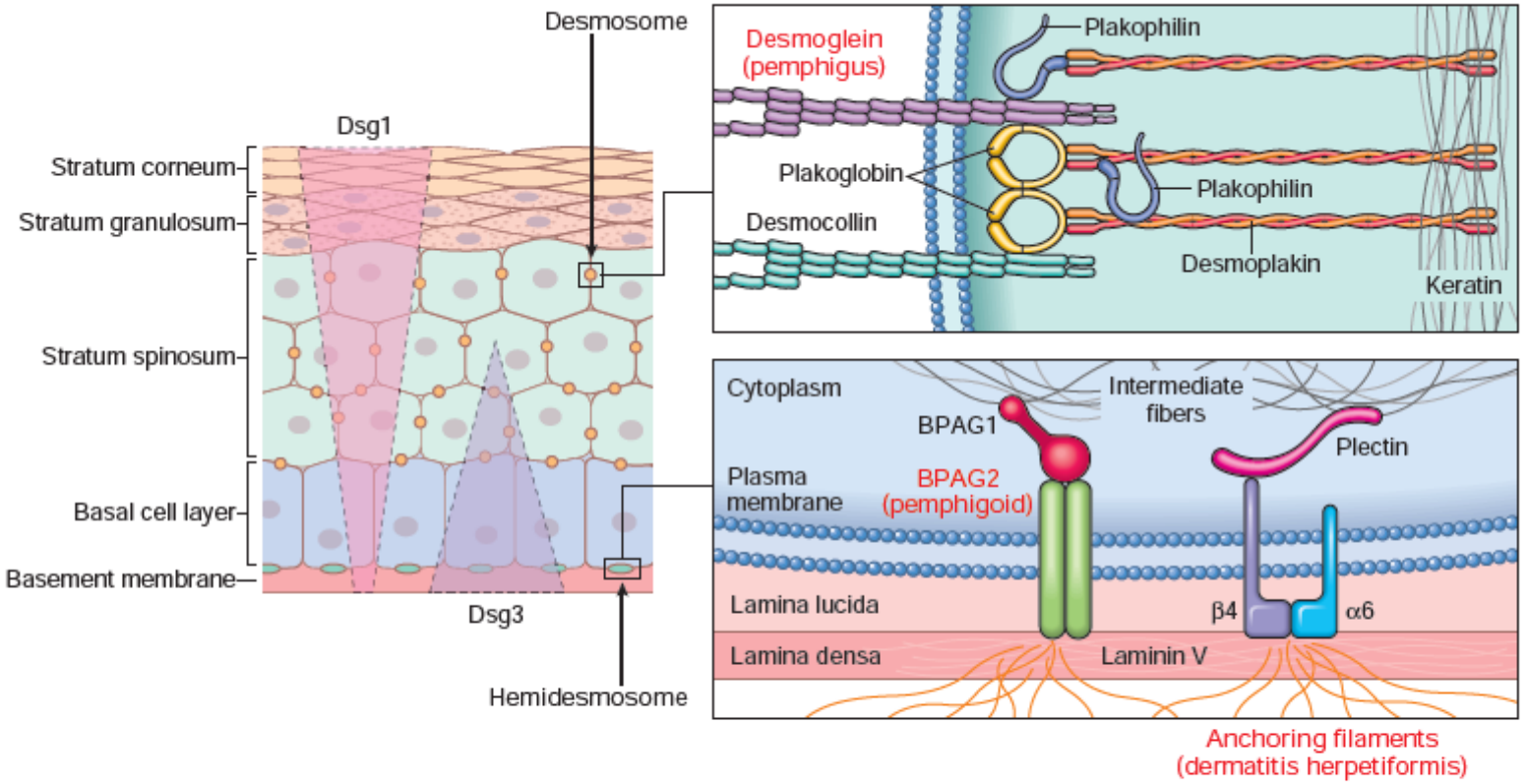


Figure 25-28 Keratinocyte adhesion molecules and blistering inflammatory disorders. Knowledge of the proteins composing desmosomes and hemidesmosomes is key to understanding blistering disorders. Desmogleins 1 and 3 (Dsg1, Dsg3) are functionally interchangeable components of desmosomes, but have different distributions within the epidermis *(left panel)*. The major structural proteins of desmosomes and hemidesmosomes are shown at *right*. In pemphigus vulgaris autoantibodies against Dsg1 and Dsg3 cause blisters in the deep suprabasal epidermis, whereas in pemphigus foliaceus the autoantibodies are against Dsg1 alone, leading to superficial, subcorneal blisters. In bullous pemphigoid autoantibodies bind BPAG2, a component of the hemidesmosomes, leading to blister formation at the level of the lamina lucida of the basement membrane. Dermatitis herpetiformis is caused by IgA autoantibodies to the fibrils that anchor hemidesmosomes to the dermis.

Pemphigus antibody patterns

- Anti-Dsg (desmoglein)-1 antibodies in pemphigus foliaceus cause acantholysis only in the superficial epidermis of skin.
- In the deep epidermis and in mucous membranes, Dsg-3 compensates for antibody-induced loss of function of Dsg-1.
- In early pemphigus vulgaris, antibodies are present only against Dsg-3, which cause blisters only in the deep mucous membrane where Dsg 3 is present without compensatory Dsg-1.

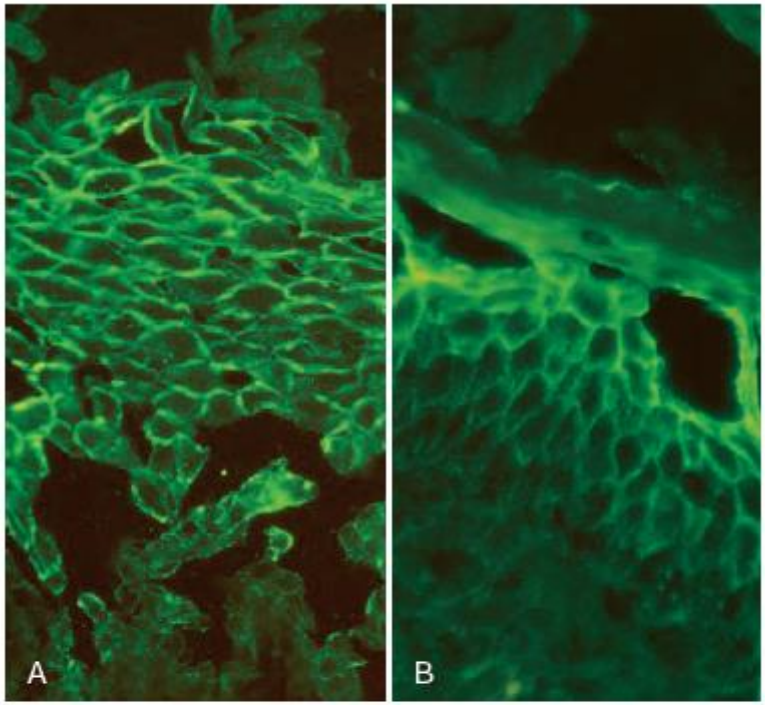


Figure 25-31 Direct immunofluorescence staining for immunoglobulin of epidermis involved by pemphigus. **A,** In pemphigus vulgaris there is deposition of immunoglobulin along the plasma membranes of keratinocytes in a reticular or fishnet-like pattern accompanied by suprabasalar loss of cell-to-cell adhesion (acantholysis). **B,** In pemphigus foliaceus the immunoglobulin deposits and acantholysis are more superficial.

Bullous pemphigoid

- 60-80 years-old
- No sex predilection
- Prodromal erythematous uriticarial lesion
- Evolves slowly then presents as a generalized eruption of serpiginous bullae
- Usual site are inner thighs and flexor surfaces
- Legs often first site manifest
- Occasionally oral or ocular involvement.
- May scar
- Early marker of HBV infection

Bullous pemphigoid

- Histology
- Subepidermal, non-acantholytic bulla
- Lysis at dermal-epidermal junction.
- Basal cell vacuolization.
- “Indian file” alignment of neutrophils at dermal-epidermal junction
- Perivascular infiltrate of neutrophils, lymphocytes, and eosinophils in papillary dermis

Bullous pemphigoid

- Antibodies to hemidesomosomes
- BPAg1 and BPAg2 or type XVII collagen
- Anti-laminin, if scarring
- Anti-BPAg 2 if ocular lesions or blister formation.
- Responds to topical steroids, tetracycline.

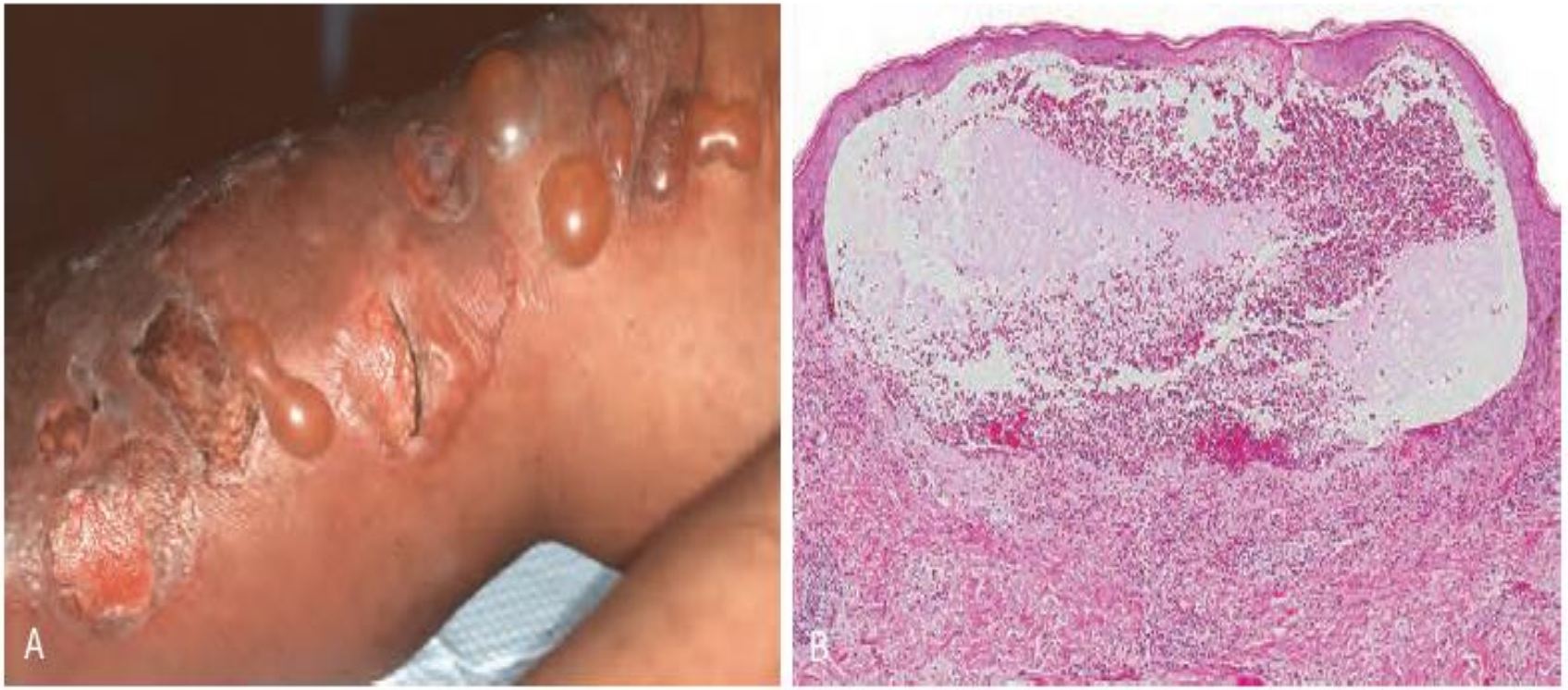
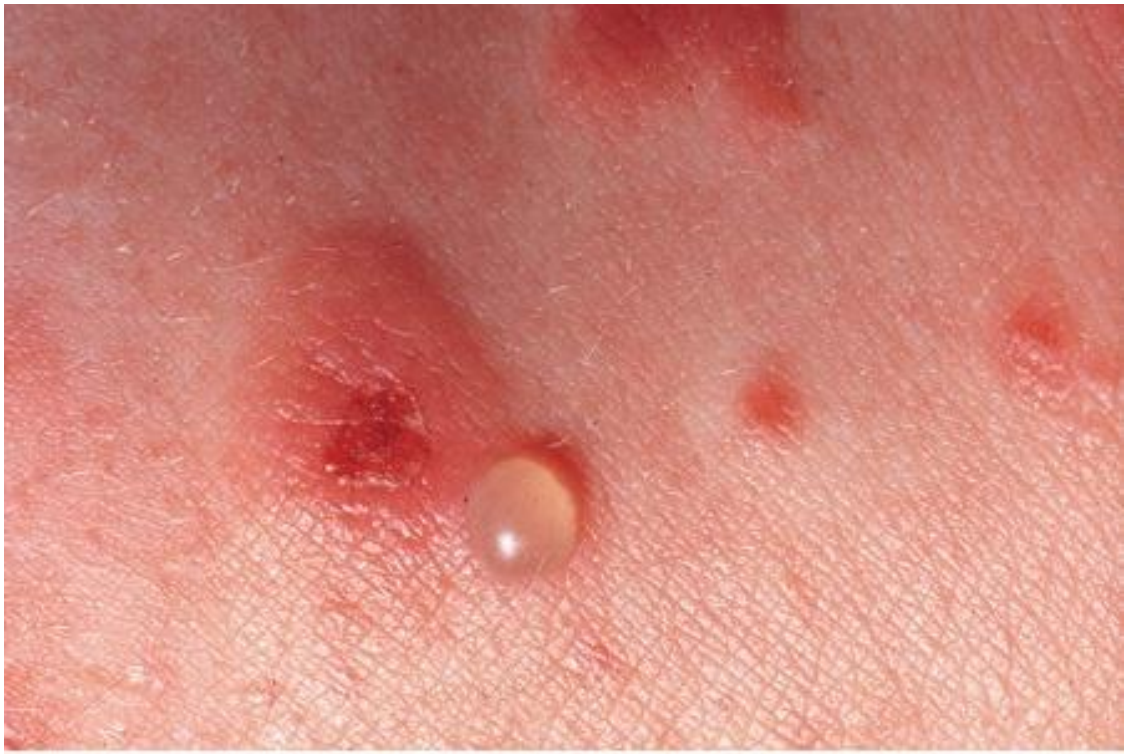


Figure 25-32 Bullous pemphigoid. **A,** Bullae consist of tense subepidermal blisters that usually fail to rupture, as their roof consists of the full epidermal thickness. Ulcers form when the blisters are rupture. **B,** An intact sub-basilar blister associated with eosinophils, lymphocytes and occasional neutrophils.

Bullous pemphigoid

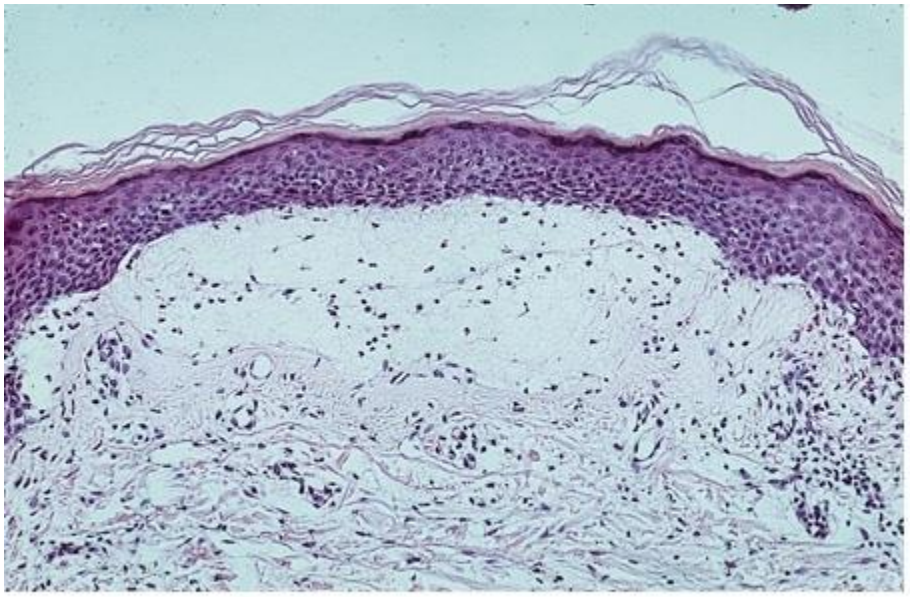


Source: Wolff K, Johnson RA: *Fitzpatrick's Color Atlas and Synopsis of Clinical Dermatology, 6th Edition:* http://www.accessmedicine.com

Urticarial plaques and a small, tense blister with a clear serous content.

Fig. 6-12 Accessed 07/16/2010

Bullous pemphigoid



- Subepidermal (junctional) cleft formation and a perivascular and interstitial lymphoeosinophilic infiltrate are characteristic.

- Linear deposits of IgG and C3 at the dermal-epidermal junction found in 80% of cases

Fig. 6-8 Accessed 07/16/2010

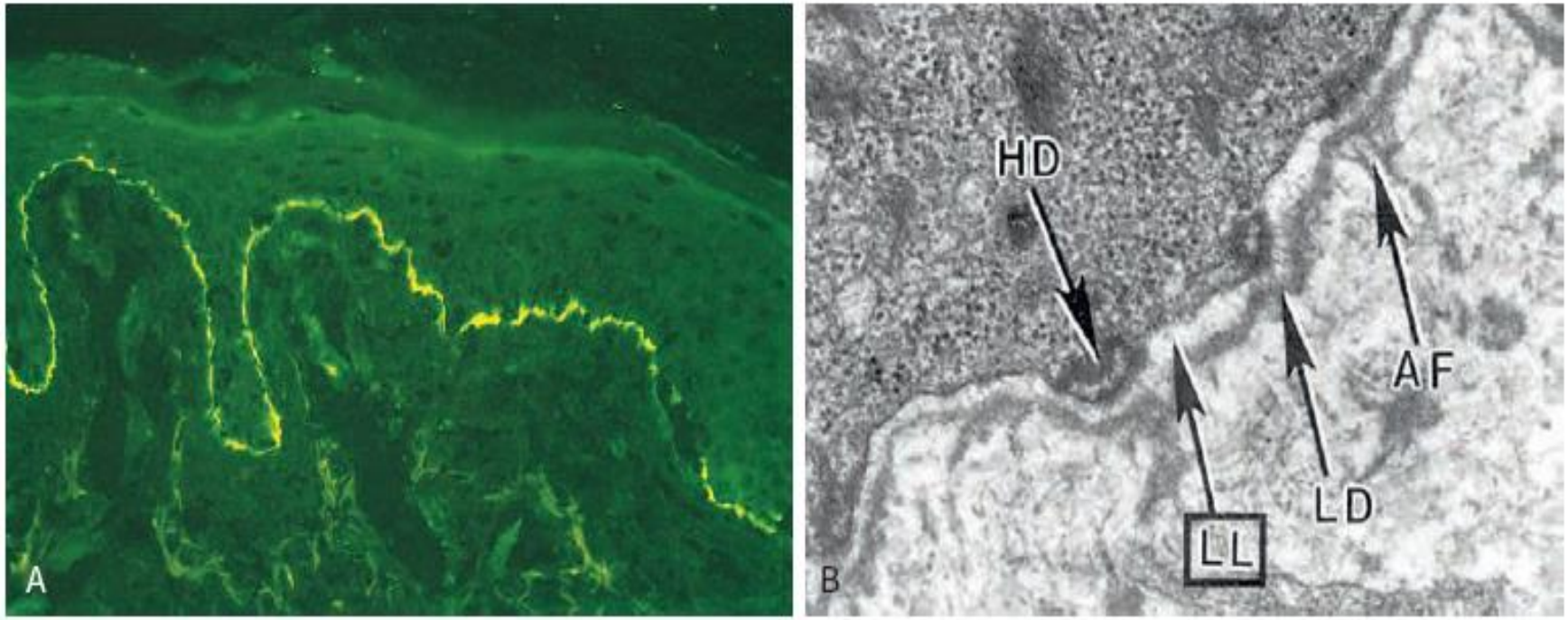


Figure 25-33 **A,** Linear deposition of complement along the dermoepidermal junction in bullous pemphigoid. **B,** Electron micrograph showing the ultrastructural features of the dermoepidermal junction. The bullous pemphigoid antigen (BPAG) is located in the basal portion of basal keratinocytes in association with hemidesmosomes (HD), which attach the epidermis to the lamina lucida (LL) of the basement membrane. AF, Anchoring fibrils; LD, lamina densa. (See also Fig. 25-31.)

Dermatitis herpetiformis

- Presents with intensely pruritic vesicles, papules, and wheals
- Often in a "butterfly" fashion on scalp, face, and hairline.
- Typical are lesions on extensor areas (elbows, knees)
- Buttocks, scapular, and sacral areas
- Bilateral, symmetrical.
- 20-60 years of age
- Males 2:1

Dermatitis herpetiformis

- Histology
- Clusters of neutrophils and fibrin at tips of dermal papillae (microabsceses).
- Severe dermal infiltration of neutrophils and eosinophils as lesion progresses
- Subepidermal blisters.
- Serum antibodies to gliadin
- IgA anti-endomysial antibodies present as granular pattern in tips of papillae
- Correlates with disease severity in celiac disease
- IgA linear pattern at the dermal-epidermal junction is not associated with celiac disease

Dermatitis herpetiformis

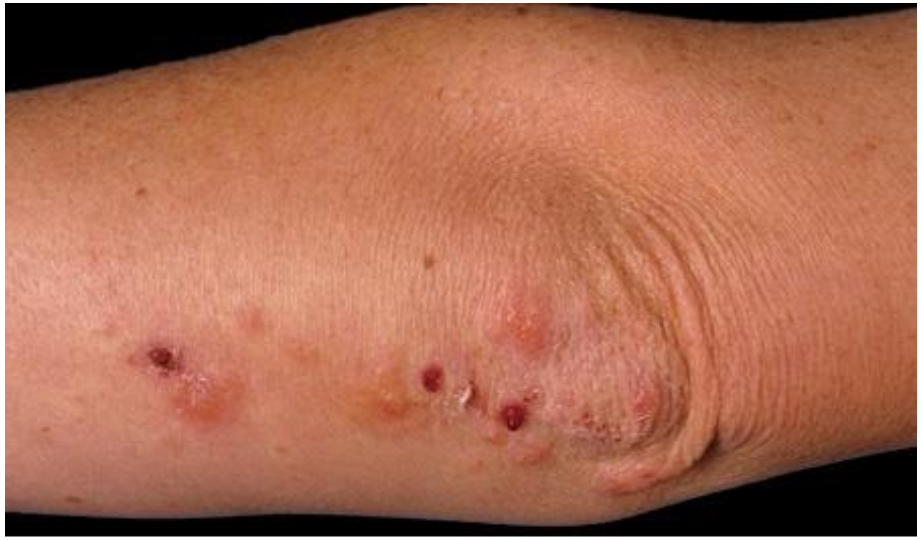
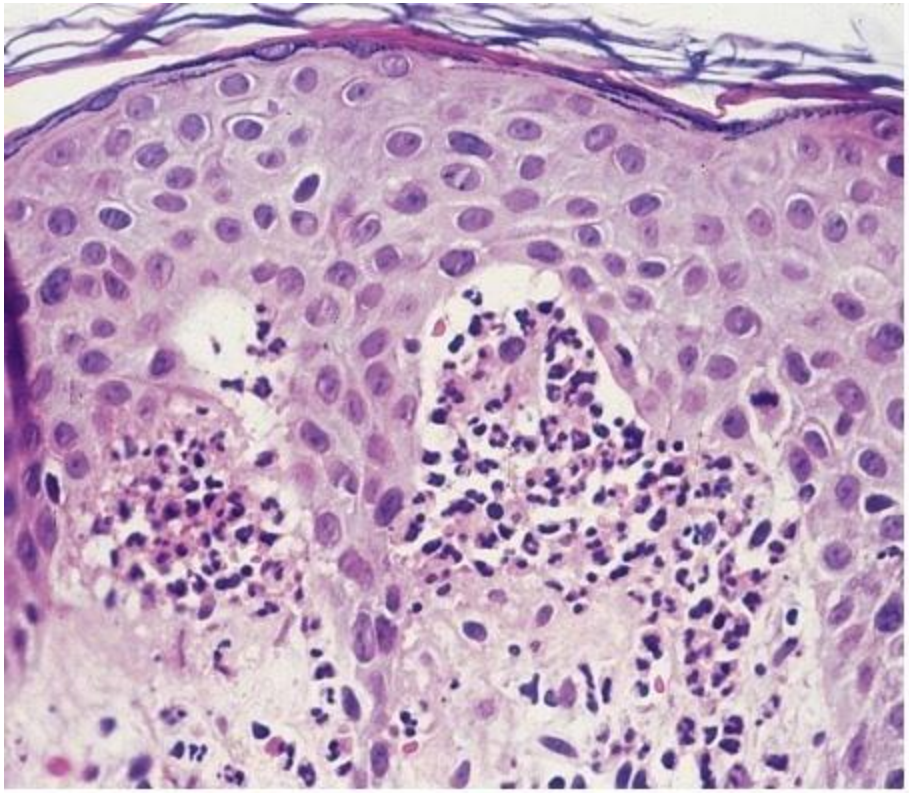


Fig. 6-16 Accessed 07/16/2010

Classic early lesions:

Papules, urticarial plaques, small grouped vesicles, and crusts on the elbow

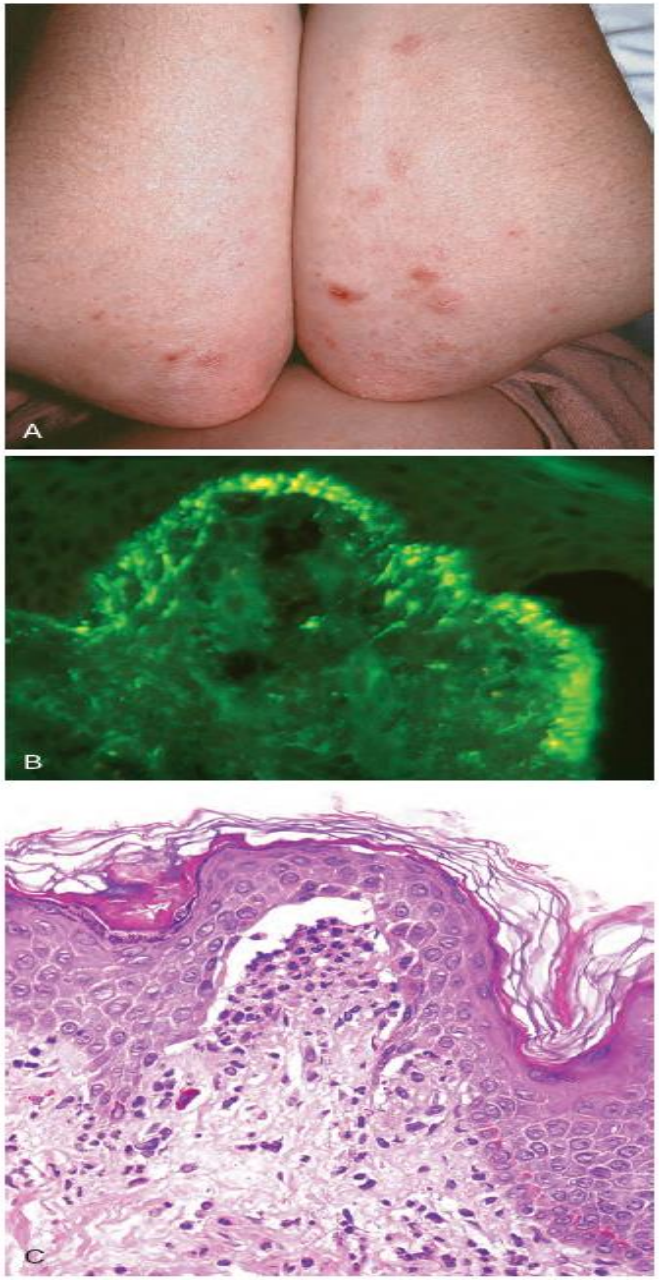
Dermatitis herpetiformis



Source: Wolff K, Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell DJ: *Fitzpatrick's Dermatology in General Medicine*, 7th Edition: http://www.accessmedicine.com

Two papillae show microabscesses composed of neutrophils. Vacuolization and early cleft formation are evident in both papillae.

Fig. 6-13 Accessed 07/16/2010



Dermatitis herpetiformis.
A, Lesions consist of intact and eroded (usually scratched) erythematous blisters, often grouped (seen here on elbows and arms).
B, Selective deposition of IgA autoantibody at the tips of dermal papillae is characteristic.
C, The blisters are associated with the accumulation of neutrophils (microabscesses) at the tips of dermal papillae.

(B, Courtesy Dr. Victor G. Prieto, Houston, Texas.)

Epidermolysis bullosa

- Proclivity to form blisters at sites of pressure, rubbing, or trauma, at or soon after birth.
- Not inflammatory
- Basal cell layer degenerates
- Simplex type
- Weber-Cockayne is mild form
- Begins in childhood
- Dowling-Meara is more extensive form
- Kindler syndrome is less extensive form
- Begin in infancy
- 75-85% of cases

Epidermolysis bullosa

- Autosomal dominant
- Mutations in KRT5 gene at 12q13.13 and KRT14 gene at 17q21.2
- Defects of the basal cell layer of the epidermis of type I (acidic) interfilament proteins (keratins)
- KRT5 also affects melanosome transport

Epidermolysis bullosa

- Junctional type (Herlitz)
- Three subtypes range in severity
- Extensive blistering of skin and oral mucosa
- Associated with death in infancy
- Defect at level of lamina lucida
- Autosomal recessive
- Mutation of LAMB3 gene at 1q32.2 most common
- Anchoring protein (laminin 332)

Epidermolysis bullosa

- Dominant dystrophic
- Blistering may be localized to the hands, feet, elbows and knees or it may be generalized. Common findings include scarring, milia (tiny white bumps), mucous membrane involvement, and abnormal or absent nails
- Mutation in COL7A1 gene at 3p21.31
- Type VII collagen affected

Epidermolysis bullosa

- Recessive dystrophic
- Scarring, milia, mucous membrane involvement and nail dystrophy
- Common manifestations include malnutrition, anemia, esophageal strictures, growth retardation, webbing or fusion of the fingers and toes causing mitten deformity (pseudosyndactyly) with loss of function, development of contractures, malformation of teeth, microstomia and corneal abrasions.
- Bi-allelic loss of COL7A1 gene

Epidermolysis bullosa

- Ogna involves all layers of skin
- Mutation of PLEC gene at 8q24.3 (plectin)
- impairs attachment of epidermis at hemidesmosome

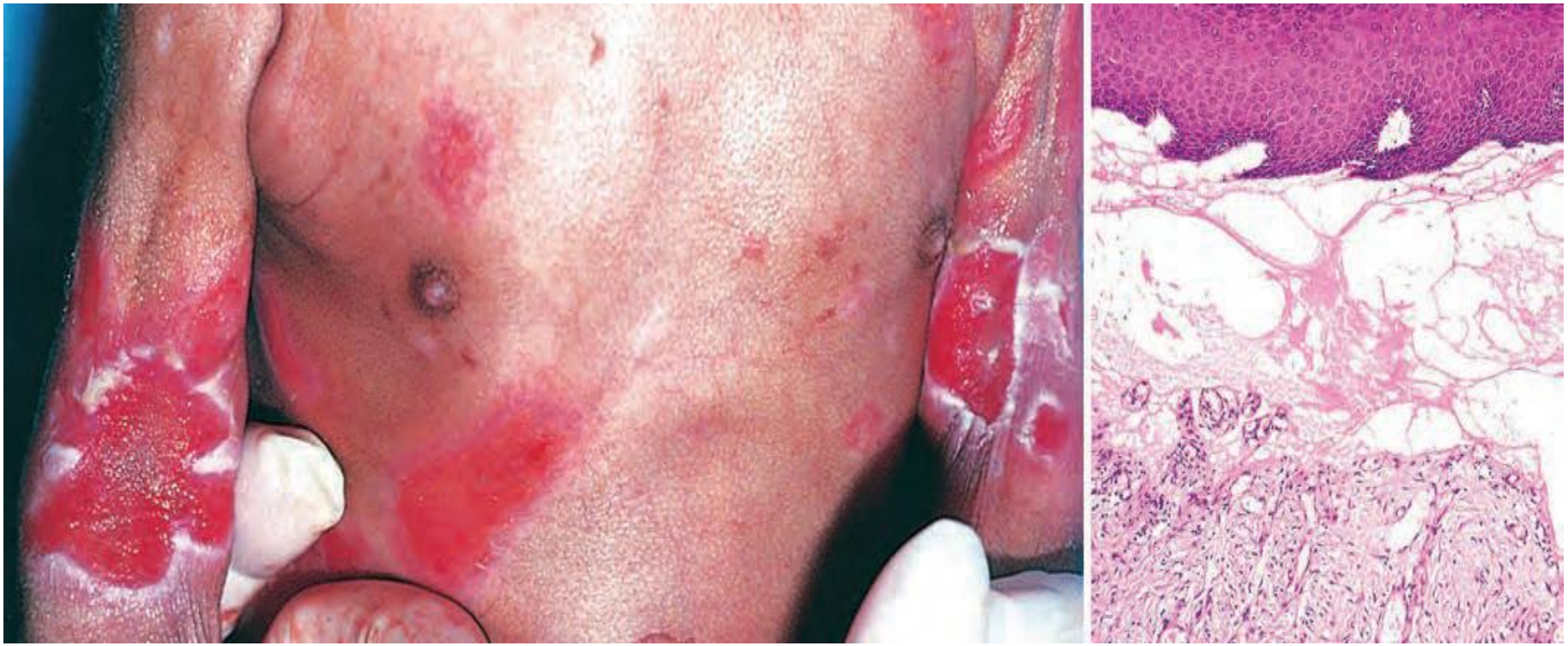


Figure 25-35 Epidermolysis bullosa. A, Junctional epidermolysis bullosa showing typical erosions in flexural creases. B, A subepidermal blister at the level of the lamina lucida. There is no associated inflammation.

Porphyria

- The five major types are:
- (1) congenital erythropoietic porphyria (CEP)
- Severe photosensitivity with erythema, swelling and blistering (painful).
- Hemolytic anemia
- (2) erythrohepatic protoporphyria (EPP)
- Photosensitivity (painful)
- Gallstones
- (3) acute intermittent porphyria (AIP)
- Abdominal pain
- Peripheral neuropathy
- Psychiatric disorders

Porphyria

- (4) porphyria cutanea tarda (PCT)
- Photosensitivity with vesicles and bullae
- (5) variegated porphyria (VP)
- Cutaneous manifestations consist of urticaria and vesicles associated with scarring that are exacerbated by exposure to sunlight.
- The vesicles are subepidermal. The adjacent dermis contains vessels with walls that are thickened by glassy deposits of serum proteins, including immunoglobulins

Porphyria cutanea tarda

- Most common
- 30-50 years of age
- Autosomal dominant (familial form)
- Estrogen use, Hepatitis C may precipitate
- “Fragile skin”
- Vesicles and bullae on dorsa of hands
- Following minor trauma or sun exposure
- Mutation in UROD gene at 1p34.1

	Biochemical Finding
Deficient enzyme	Hepatic uroporphyrinogen decarboxylase
Enzyme activity	< ~20% of normal
Plasma	↑ Uroporphyrin, heptacarboxylporphyrin (~620 nm) [1]
Urine	↑ Uroporphyrin, heptacarboxylporphyrin
Stool	↑ Heptacarboxylporphyrin, isocoproporphyrins + pentaporphyrins

1. Fluorescence emission peak of diluted plasma at neutral pH, following excitation at 400-410 nm

https://www.ncbi.nlm.nih.gov/books/NBK143129/

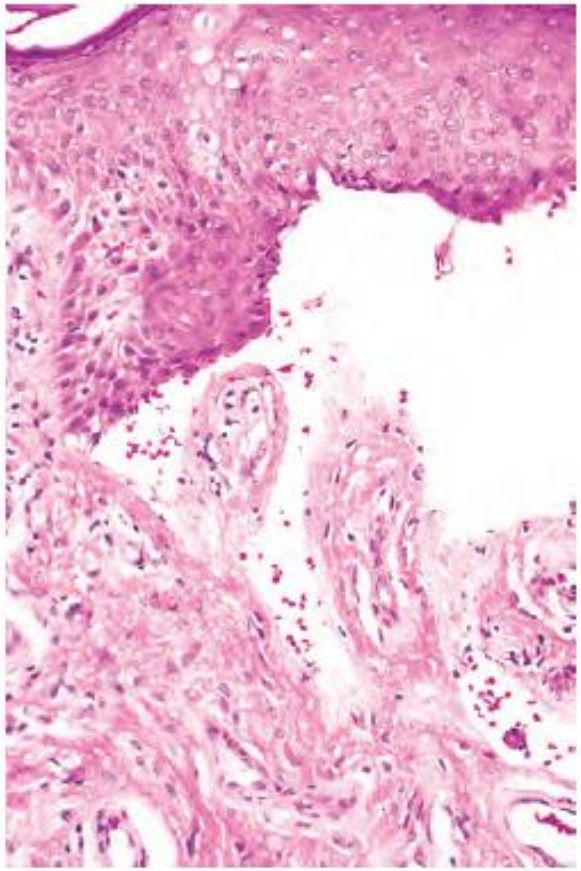


Figure 25-36 Porphyria. A noninflammatory blister at the dermoepidermal junction; note the seemingly rigid dermal papillae at the base that contain abnormal superficial vessels.

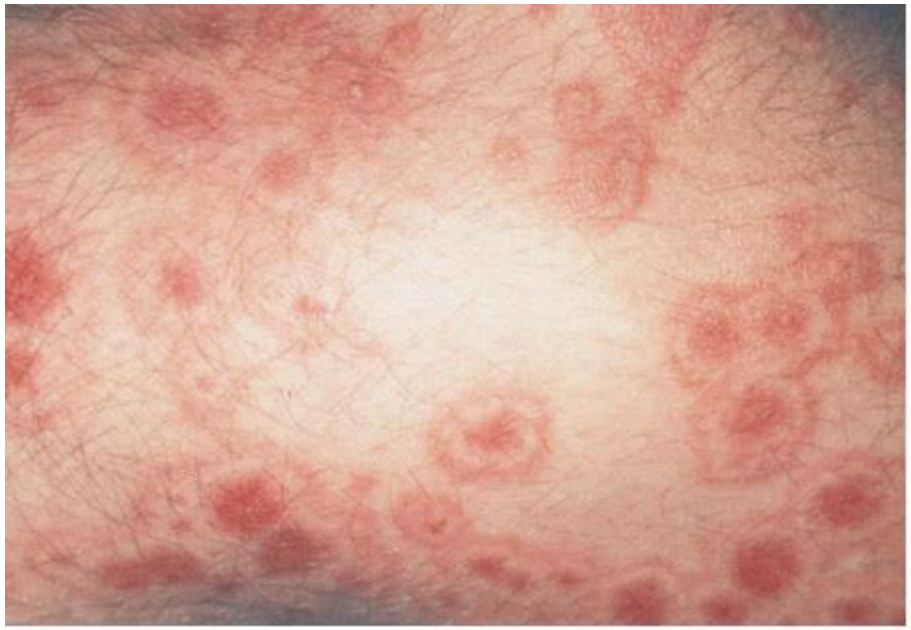
Erythema multiforme syndrome

- 20-30 years of age
- Male predilection
- Lesions may be pruritic or painful
- Macule evolves to papule within 48 hours
- Then multiform erythematous plaques characterized by a target lesion.
- Generally symmetric involvement of dorsa of hands, palms, and soles (mild form)
- May involve forearms, face, elbows, and knees
- 50% have genital involvement (severe form)

Erythema multiforme syndrome

- Consider Lyme disease.
- Causes
- Usually Herpes simplex (with mucosal lesions)
- Sulfur-containing drugs
- Penicillin
- Allopurinol
- May be difficult to distinguish from toxic epidermal necrolysis if oral mucosal lesions predominate
- Perivascular CD8+ lymphocytic infiltrate with degeneration of the dermal-epidermal junction (interface dermatitis)
- IgE may be demonstrated in uriticarial lesions.

Erythema multiforme



Multiple erythematous plaques with a target or iris morphology usually represents a hypersensitivity reaction to drugs or infections (especially herpes simplex virus).

(Courtesy of the Yale Resident's Slide Collection; with permission.)

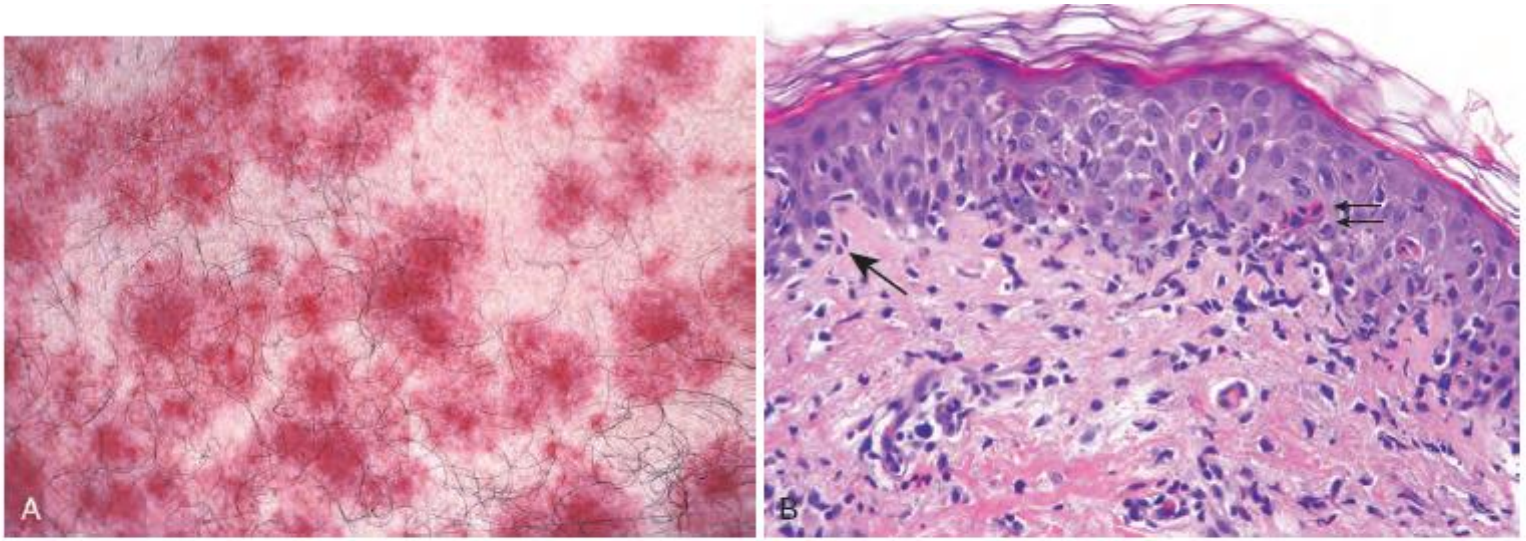
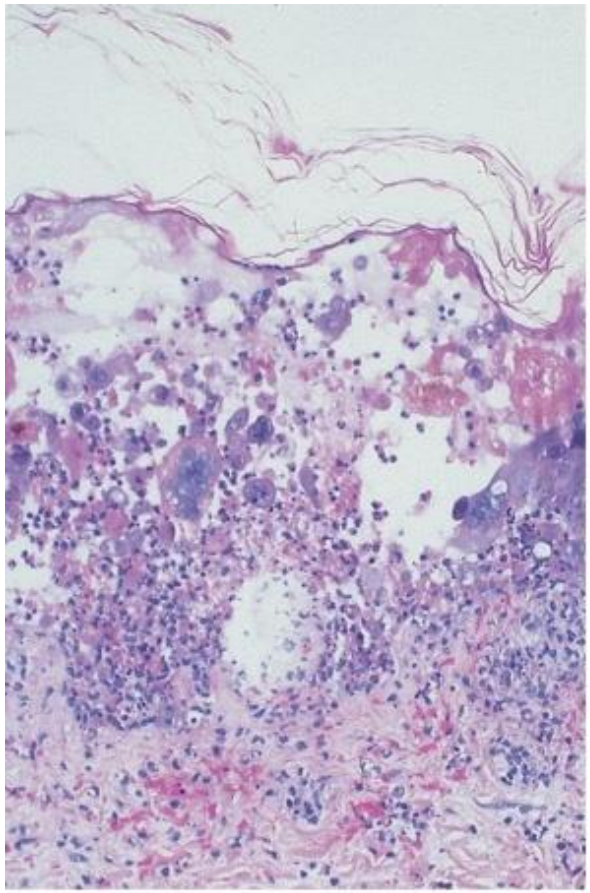


Figure 25-24 Erythema multiforme. **A,** The target-like lesions consist of a central blister or zone of epidermal necrosis surrounded by macular erythema. **B,** An early lesion shows lymphocytes accumulating along the dermoepidermal junction where basal keratinocytes have begun to become vacuolated (arrow). With time, necrotic/apoptotic keratinocytes appear in the overlying epithelium (double arrow).

Herpes virus infection

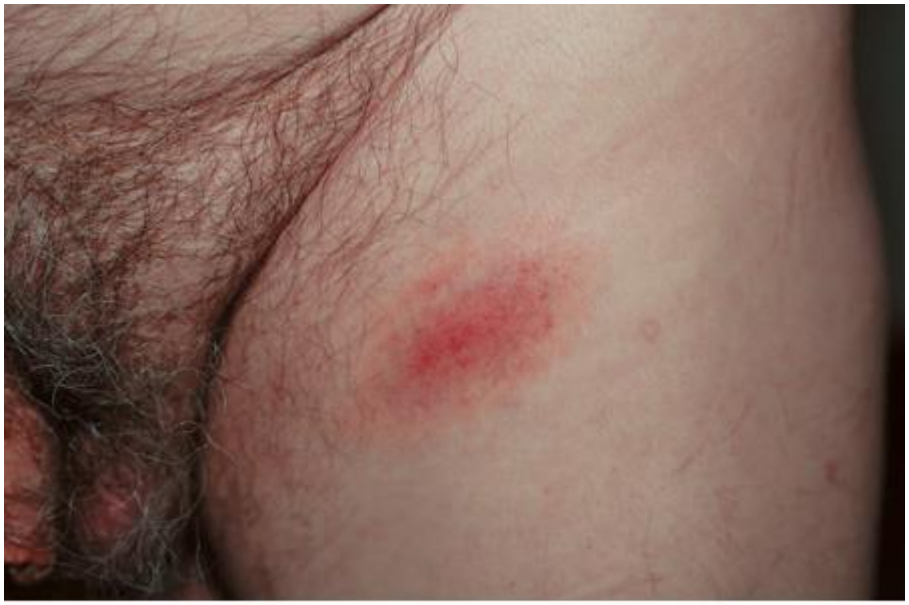


The epidermis shows marked ballooning degeneration, cytolysis, and intraepidermal vesiculation. Perivascular lymphocytic infiltrate.

<u>Acantholytic and multinucleated epidermal giant cells are a clue to herpetic infection.</u>

Fig. 6-7 Accessed 07/16/2010

Erythema chronicum migrans



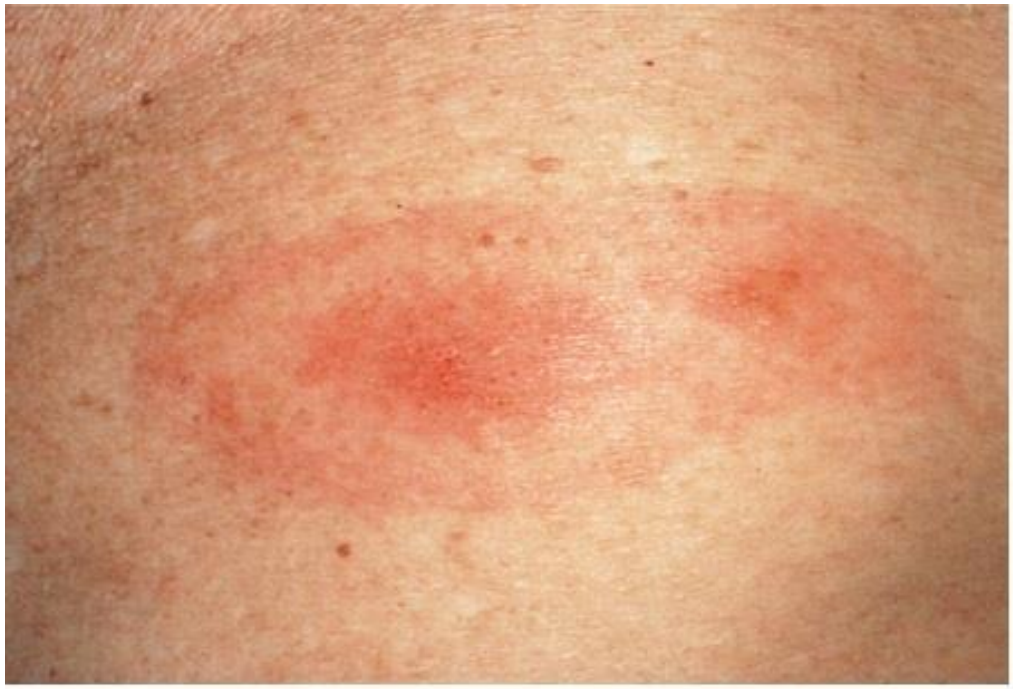
A

Target lesion:

Erythematous annular patch, often with a central erythematous papule

Fig. 24-76A Accessed 07/16/2010

Lyme disease

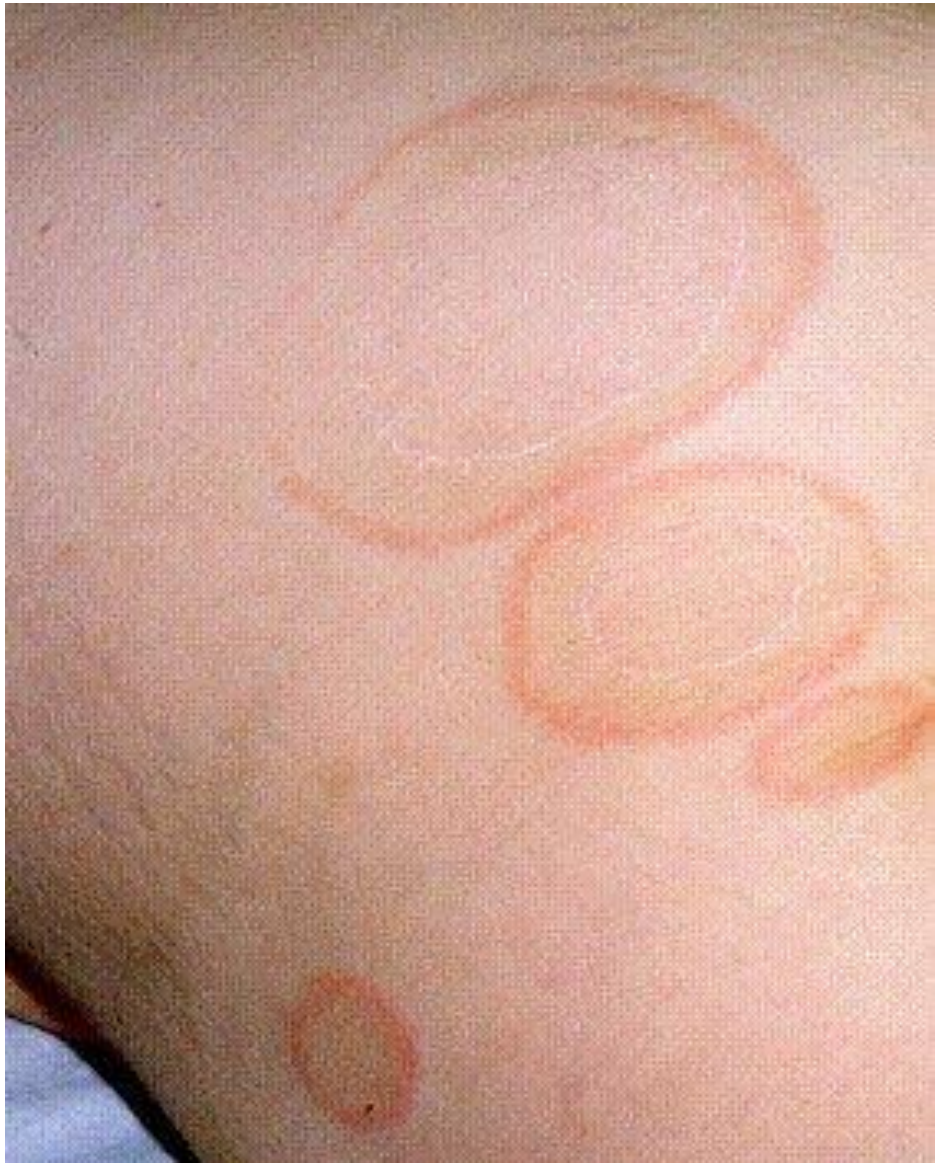


Erythema chronicum migrans is the early cutaneous manifestation of Lyme disease

Target lesion at the tick bite site (Borrellia burgdorferi)

(Courtesy of Yale Resident's Slide Collection; with permission.)

Erythema marginatum



Erythematous serpiginous macular lesions with pale centers

Not pruritic.

Accentuated by skin warming.

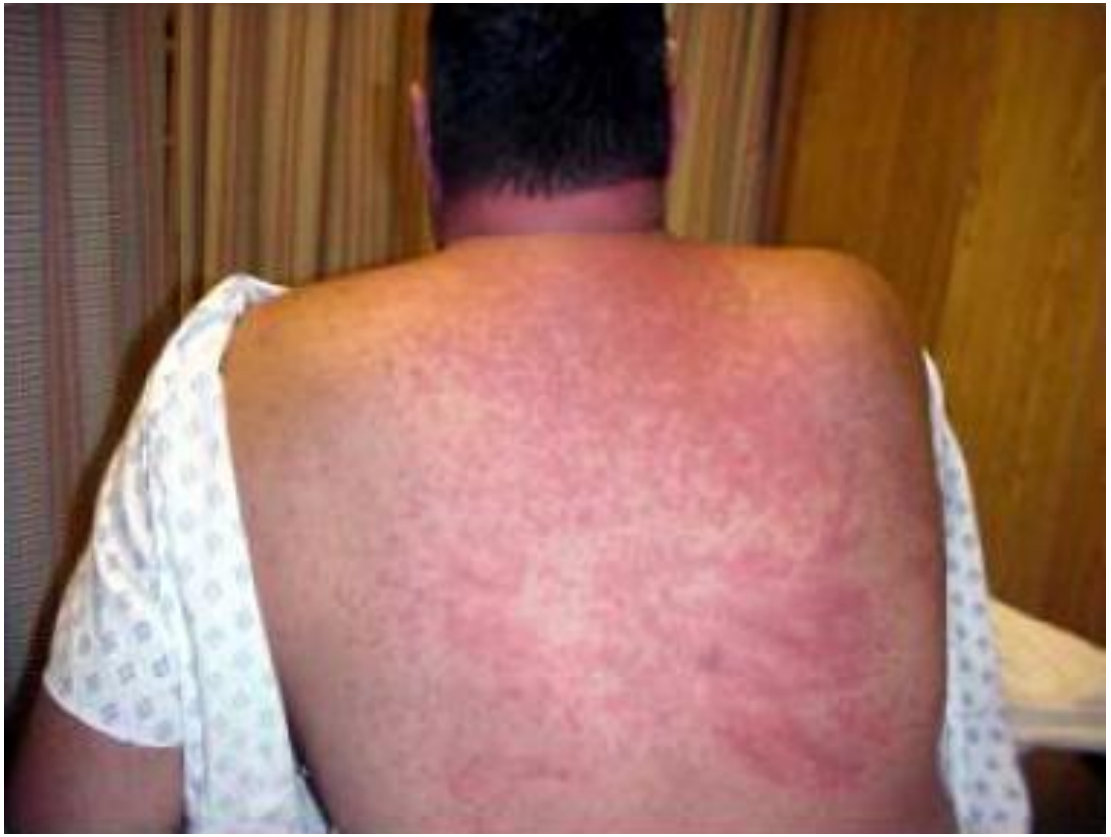
Acute rheumatic fever

https://www.hxbenefit.com/wp-content/uploads/2012/07/Erythema-marginatum-Picture.jpg
Accessed 12/07/2019

Toxic epidermal necrolysis

- Prodroma of conjunctivitis, pharyngitis, pruritis
- Mucous membrane erosions precede erythema multiforme skin lesions by several days.
- Ocular and genital involvement
- Desquamation
- Drug reaction
- Allopurinol
- Carbamazepine and phenytoin
- Sulfur-containing drugs
- Aminopenicillins
- Oxicam NSAIDs
- Niverapine (NNRTI)

Toxic epidermal necrolysis



https://emedicine.medscape.com/article/229698-overview
Accessed 12/07/2019

Toxic epidermal necrolysis

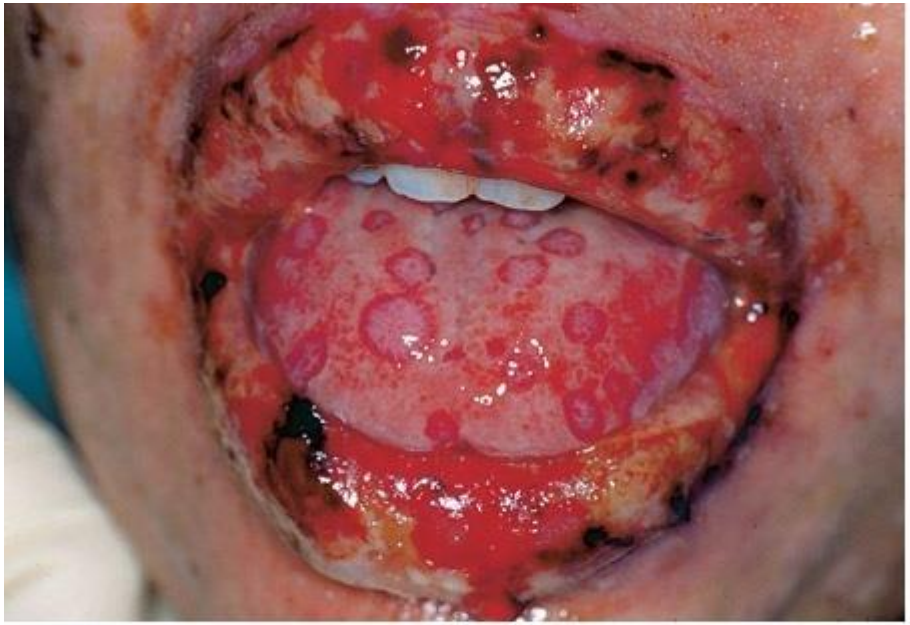


Fig. e7-4 Accessed 07/16/2010

Stevens-Johnson syndrome is part of the TEN spectrum.

Flaccid bullae and vesicles that develop centrally within a pre-existing target lesion.

Widespread apoptosis of keratinocytes provoked by the activation of a cell-mediated cytotoxic reaction.

Toxic epidermal necrolysis

- CD8+ cells in blister fluid
- Granulysin (cytokine) level elevated
- Pathogenesis:
- FAS ligand pathway activation
- Granule mediated exocytosis
- Histopathology:
- Apoptotic keratinocyte cell death in the epidermis with dermal-epidermal separation that results in bullae formation.
- Full-thickness lesion
- No antibody demonstrated on direct immunoflourescent stain

Stratification

- Widespread purpuric macules or flat atypical targets
- Stevens-Johnson syndrome (SJS)
- <10% Body surface area (BSA) involved
- TEN
- 10-30% BSA involved
- TEN with spots
- >30% BSA involved
- Large epithelial sheets but no purpuric macules
- TEN without spots
- >10% BSA involved

Mortality prediction score

Table 3. ABCD-10 SJS/TEN Mortality Prediction Model Score

ABCD-10[a]	Predicted Mortality Rate, % (95% CI)[b]
0	2.3 (1.1-4.6)
1	5.4 (3.2-8.7)
2	12.3 (8.9-16.6)
3	25.5 (19.6-32.5)
4	45.7 (34.2-57.8)
5	67.4 (50.8-80.6)
6	83.6 (66.7-92.8)

Abbreviations: ABCD-10, age, bicarbonate level, cancer, dialysis, and BSA greater than 10%; BSA, body surface area; SJS/TEN, Stevens-Johnson syndrome/toxic epidermal necrolysis.

[a] Calculated by taking the sum of 1 point each for age 50 years or older, epidermal detachment greater than10% of BSA, and serum bicarbonate level lower than 20 mmol/L; 2 points for the presence of active/ongoing cancer; and 3 points for dialysis prior to admission.

[b] $Pr(death) = e^{logit}/(1 + e^{logit})$ where logit = −3.764 + 0.898 (ABCD-10).

https://www.dermnetnz.org/topics/stevens-johnson-syndrome-toxic-epidermal-necrolysis/ Accessed 12/09/2019

Scalded skin syndrome

- Red blistering skin (resembles burn)
- <5 years-old
- Ritter or Lyell disease another name
- Release of epidermolytic toxins A and B from Staph. Aureus
- Bind to desmoglein.
- Acantholysis
- Desquamation
- Penicillinase resistant antibiotic therapy

Toxic shock syndrome

- Healthy patients
- 20-50 yearsold
- TSST1 exotoxin as well as enterotoxin B and C from Staph. Aureus lead to massive cytokine release
- TSS-1 menstrual form (women)
- TSST1
- TSS-2 non-menstrual form (men and women)
- Enterotoxin B and C as well as TSST1
- Patient has no anti-exotoxin antibodies
- Diaphragms, vaginal sponges predispose
- 30% recurrence rate

CDC diagnostic criteria

- Fever
- Hypotension
- Widespread red, flat rash
- Desquamation on palms and soles 1-2 weeks following onset
- Other system involvement
- Disease mimicked by M-protein release from Strep. Pyogenes.
- Functions as superantigen
- Isolation of Strep. Pyogenes as well as evidence of other organ involvement and rash
- Penicillin therapy (with clindamycin if Strep.)

Varicella-zoster (chickenpox)

- May have coryza.
- Vesicular lesions on an erythematous base present in succesive crops.
- Ulcerate and crust.
- Pruritic
- Begin on face and spread downward.
- Most profuse on pressure bearing areas

Varicella-zoster (chickenpox)

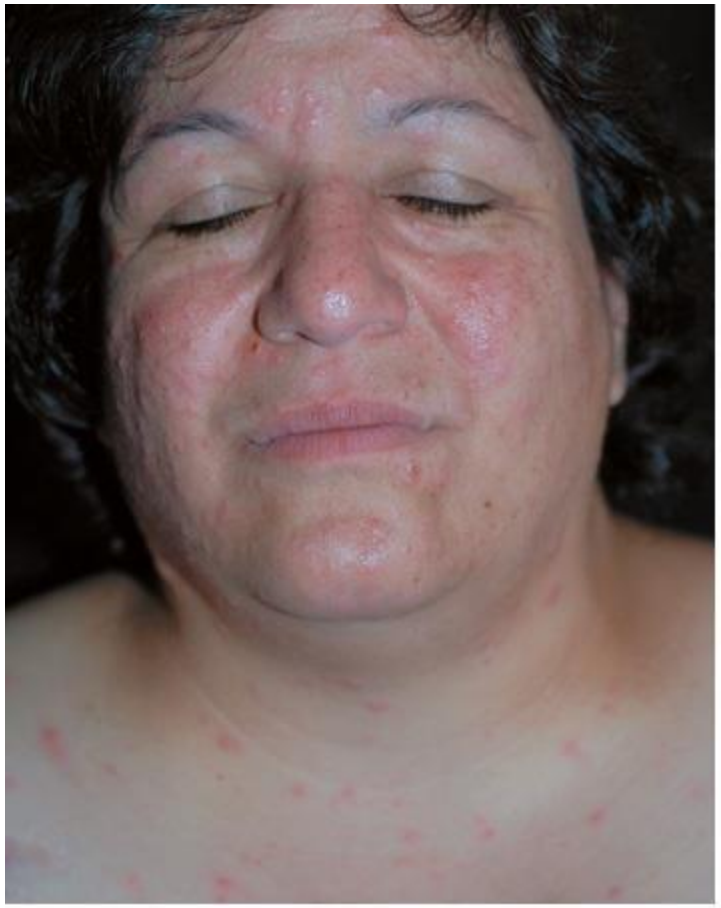
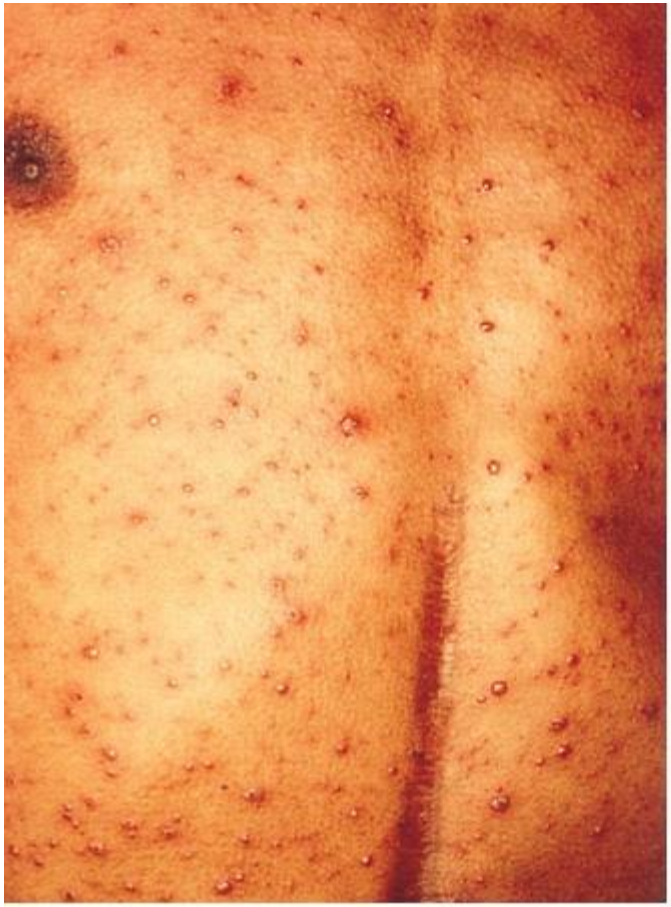


Fig. 27-39 Accessed 07/16/2010

Varicella-zoster (chickenpox)



(Courtesy of R. Hartman; with permission.)

- Fig. 173-1 Accessed 07/01/2010

Varicella-zoster infection

- Hemorrhagic vesicles and pustules on an erythematous base
- Dermatomal distribution.
- Pain often precedes eruption.
- Reactivation of vaccinia virus dormant in neuron soma.
- Systemic antivirals useful within first 72 hours of eruption.
- Prevent dissemination.
- Vaccine for primary protection.

Varicella-zoster infection (Shingles)

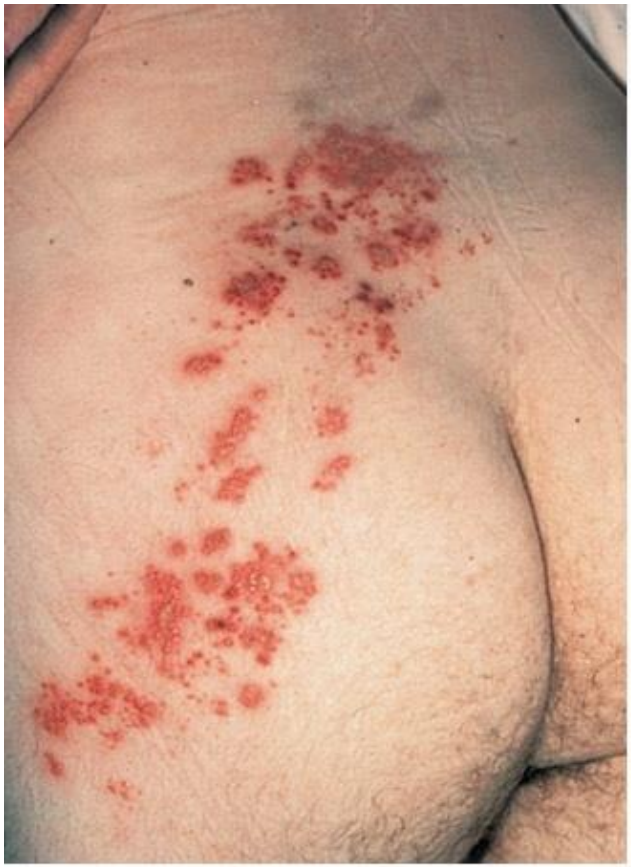


Fig. 173-3 Accessed 07/16/2010