Trauma, infection, inflammation → **BLISTERS** → erosion or ulceration

LOCALIZED

Herpes Zoster (shingles)

Epidemiology: evolving due to immunization
- 10-20% incidence

Pathophysiology: eruption of latent varicella zoster virus
- Sensory ganglia affected becomes lifelong host → reactivated by trauma, tumor, radiation, immunosuppress

Clinical: **prodrome** - pain, tenderness, paresthesia
Active - papules → vesicles → pustules → crust (>50% thoracic)
PHN - constant, severe, stabbing, dysesthetic pain
± Hutchinson's sign (tip of nose)

Diagnosis: dermatomal, grouped, pain/burning

Treatment: (val)acyclovir
PHN - gabapentin

Herpes Simplex

Epidemiology: **HSV1** favors mouth/nose. **HSV2** favors genitalia, buttock, thigh

Pathophysiology: herpes simplex viral infection

Clinical: Painful, grouped vesicles on erythematous base
can be **pustular**
- **perianal** in immunosuppressed
- **recurrent**

Diagnosis: painful, recurrent

Treatment: **acyclovir** used episodically or prophylactically
Also **famciclovir** and **valacyclovir** ($$$)

Dyshidrotic eczema

Etiology: hx of atopic dermatitis and/or tinea pedis ITCHY

Clinical: pruritic vesiculo papules on palms, soles, and sides of fingers
↳ after healing → post-inflammatory hyperpigmentation

Diagnosis: history and clinical

Treatment: potent topical steroids

HERPES VIRUS diagnosis

- **Tzanck prep** → multinucleated giant cell
  bedside scraping and microscopic exam
- **Viral culture** - only performed if fluid present
- **Direct fluorescent antibody test** - differentiate HSV1/2 and VZ
- **Fluid swab for PCR** - can differentiate HSV1/2 and VZ

LOCATION CLUES

Feet
- Dorsum - contact derm, insect bites
- Sides/toes - dyshidrotic eczema
- Soles - tinea pedis
- Balls/heels - friction blister

Mouth/nose/eyes - HSV, bullous impetigo
Chest/back - VZV
Arms/legs - contact derm

GENERALIZED/EXTENSIVE

Pemphigus Vulgaris

Epidemiology: middle aged adults

Pathophysiology: **IgG antibodies** → loss of cell to cell adhesion → superficial bullae and erosions
- autoantibodies to desmogleins 1, 3 between keratinocytes

Clinical: lesions usually start in oral mucosa → skin (6-12 mon later)
- Superficial bullae and erosions

Diagnosis: +Nikolosky sign. Diagnose with direct immunofluorescence

Treatment: **prednisone**
± Immunosuppressive treatment
- azathioprine, methotrexate

Bullous Pemphigoid

Epidemiology: older adults (>50 yo)

Pathophysiology: **autoimmune**
Auto-antibodies, complement fixation, neutrophils, and eosinophils cause bullae
- autoantibodies against hemidesmosome

Clinical: deep, tense bullae on pruritic, urticarial base
Prodrome - urticarial/papular lesions.

Diagnosis: bacterial culture
Immunofluorescence → BPAG2 antibodies

Treatment: Prednisone
topical steroids if mild or localized

IMMEDIATE REACTIONS

URTICARIA hives

hives. mast cell. IgE

Pathophysiology: vascular reaction causing swelling of upper dermis
usually mastcell involvement
Immunologic - antigen binds IgE on mast → degranulation and histamine release
Non-Immunologic - direct mast cell degranulation by pharm/physical mechanism

Etiology: Acute - <6 weeks - idiopathic, post infection (viral, strep), food/drug/detergent reactions
Chronic - >6 weeks - >50% idiopathic. Physical: pressure, cold, heat, dermatographism
Autoimmune: IgE receptor antibodies

Clinical: wheals surrounded by a blanchable red halo or flare. Pruritic.

Diagnosis: Clinical - history is important.

Treatment: oral antihistamines. 1st generation can cause sedation

IF SYSTEMIC SYMPTOMS

ANGIOEDEMA

Pathophysiology: Same as urticaria but in the deep dermis

Clinical: Subcutaneous swelling usually affecting face or extremities
- Painful/burning, but not pruritic
- may last several days

CAN BE CUTANEOUS PRESENTATION OF

ANAPHYLAXIS

Pathophysiology: Serious allergic reaction

Clinical: rapid onset. chest tightness, difficulty breathing, hoarse voice, throat tightness, nausea, vomiting, abdominal pain, lightheadedness

Treatment: epinephrine, IV fluids, and oxygen. Monitored in hospital

DRUG REACTIONS

cutaneous drug reactions are inflammatory, generalized, symmetric

DRUG ERUPTIONS

Exanthematous eruptions

Epidemiology: most common

Pathophysiology: >2 days after drug initiation (usually 8-11)

Clinical: erythematous macules and infiltrated papules. ± fever, pruritis
- limited to skin

Diagnosis: Clinical

Treatment: topical steroids, antinistamines

Usually resolve without sequelae

Fixed drug eruptions

quicker onset

Causes: NSAIDs, tetracyclines, metronidazole, sulfonamides

Pathophysiology: lesions appear 30min-8hr after exposure

Clinical: solitary erythematous patch or plaque that recurs at same site with reexposure ± bullae and erosion

Treatment: should resolve when discontinue drug. Healed lesions are dark brown w/ violet hue

Non-eroded - potent topical glucocorticoid

Eroded - antimicrobial ointment

SYSTEMIC REACTIONS

Stevens-Johnson Sydrome / Toxic Epidermal Necrolysis (SJS/TEN)

5-12% mortality | >20% mortality

Sulfa abx **A**llopurinol **T**etracyclines **A**nticonvulsants **N**SAIDs

Pathophysiology: extensive necrosis and detachment of epidermis AND mucousal surfaces

Clinical: erythematous, irregularly shaped, dusky macules with rapid expansion

1-3 day prodrome - fever, headache, myalgias

Skin detaches ↓ + Nikolsky

Diagnosis: <10% body surface → SJS. >30% body surface → TEN.

Treatment: early withdrawal of offending agent. Burn unit if >25-30% BSA

Drug-induced Hypersensitivity Syndrome

Causes: allopurinol, sulfa abx, penicillin, anticonvulsants, NSAIDs, abacavir

eosinophils

Pathophysiology: skin eruption

Clinical: systemic symptoms (fever) and internal organ involvement (liver, kidney, heart)
- macular exanthem, centrofacial swelling, fever, malaise, lymphadenopathy

Diagnosis: elevated eosinophils.

More delayed than drug eruption

Treatment: fatality rate up to 10%.

Mild - topical steroids and anti-histamines ± systemic steroids with slow taper

Erythema multiforme

Epidemiology: 50% <20yo. M > F.

Pathophysiology: Cutaneous reaction to antigenic stimuli (HSV)
- less commonly sulfonamides, phenytoin, barbiturates, phenylbutazone, PCN, allopurinol

Clinical: Painful targetoid papules and oral ulceration
- acral/extremities > trunk
- ± bullae
- ± fever

HSV

Treatment: topical steroid if mild. Systemic steroid and analgesia if severe
- Acyclovir for prevention

PHOTOSENSITIVITY: agents absorb UVA energy → erythema or inflammation

- **Phototoxicity**: phototoxic agent + UV radiation.
  - retinoids, fluorouracil, antibiotics, thiazides, amiodarone, etc
- **Photoallergic**: immune reaction to UVA-modified chemical. Type IV hypersensitivity
  - sunscreens, disinfectants, fragrances, NSAIDs, antifungals

PURPURA

Epidemiology: **fam history** of bleeding/thrombotic disorders. **Drug/meds** that affect coagulation. Conditions that result in **altered coagulation**

Pathophysiology: **extravasation** of blood into skin or mucous membranes
- Can be due to **hyper/hypocoag states**, **vascular dysfunction**, or **extravascular**

Clinical: **red-purple** (violaceous) color

Diagnosis: **diascopy** - **does not blanch** → **erythrocyte extravasation**
- **CBC w/ diff**, **PT/PTT**, evaluate coag states

Treatment:

Macular: non-inflammatory
- **Petechie** <3mm
- **Ecchymosis** >5mm

Causes: nutritional deficiency, meningitis, tickborn illness

Palpable Purpura OR

VASCULITIS

hallmark lesion of **leukocytoclastic vasculitis**

Pathophysiology: **inflammation** of small cutaneous vessels

Clinical: **Small** → **palpable** purpura. **Medium** → subcutaneous nodules, purpura, and **fixed livedo reticularis**. **Large** → **claudication**, **ulceration**, **necrosis**

Diagnosis: **CBC** with platelets
ESR
Autoimmune testing
Skin biopsy

Treatment: **underlying cause**, remove agent, immune suppression

Henoch-Schönlein Purpura

Epidemiology: **children** (3-15 yo)

Pathophysiology: usually due to association with preceding **viral/bacterial infection**

Clinical: **vasculitis**, **arthritis**, **abdominal pain**, and **kidney disease**

Diagnosis: **Clinical ± biopsy**-leukocytoclastic vasculitis of small dermal blood vessels
Immune complexes contain **IgA deposition**.

Treatment: **Supportive** ± prednisone

Polyarthritis Nodosa

Epidemiology: **HBV**, **HCV**, **HIV**, **parvovirus**

Pathophysiology: systemic **necrotizing** vasculitis of **medium**-sized arteries

Clinical: **painful**, **subcutaneous nodules**.
Fever, paresthesias, decreased reflexes

Diagnosis: **Biopsy** - inflammation of medium sized artery of skin

Treatment: **Chronic**. Local wound care.
± **prednisone** depending on severity