Dermatology in the Hospitalized Patient

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Objectives

- To identify dermatologic conditions that may manifest in hospitalized patients.
- To layout a diagnostic and management approach to these dermatologic conditions.
- To provide appropriate treatment options to be used while the patient is hospitalized.

Disclaimer

- This presentation is not comprehensive of all dermatologic conditions that may be seen in the hospitalized patient, but does encompass many of the most commonly seen conditions.
- This presentation focuses on immediate and short term management of conditions while the patient is hospitalized and does not discuss comprehensive and outpatient management.

Dermatologic Conditions in Hospitalized Patients

Drug Reaction	**Infectious**
Morbilliform eruption Urticarial eruption Drug reaction with eosinophilia and systemic symptoms (DRESS) Acute generalized exanthematous pustulosis (AGEP) Stevens Johnson syndrome (SJS) / Toxic epidermal necrolysis (TEN)	Cellulitis Necrotizing fasciitis Herpes zoster Bacterial endocarditis
Inflammatory	**Vascular**
Atopic dermatitis Psoriasis Contact dermatitis Stasis dermatitis	Vasculitis Disseminated intravascular coagulation (DIC) Calciphylaxis
Ulcers	**Neoplastic**
Venous insufficiency Arterial insufficiency Diabetic ulcers Pressure ulcers	Basal cell carcinoma Squamous cell carcinoma Melanoma

Drug Reactions

Morbilliform eruption → Repeat exposure to causative drug ok – treat through recurrence of rash

Urticarial eruption → Repeat exposure to causative drug ok if not severe – treat through recurrence of rash

SCARs (severe cutaneous adverse reactions)

- Drug reaction with eosinophilia and systemic symptoms (DRESS)
- Acute generalized exanthematous pustulosis (AGEP)
- Stevens Johnson syndrome (SJS) / Toxic epidermal necrolysis (TEN)

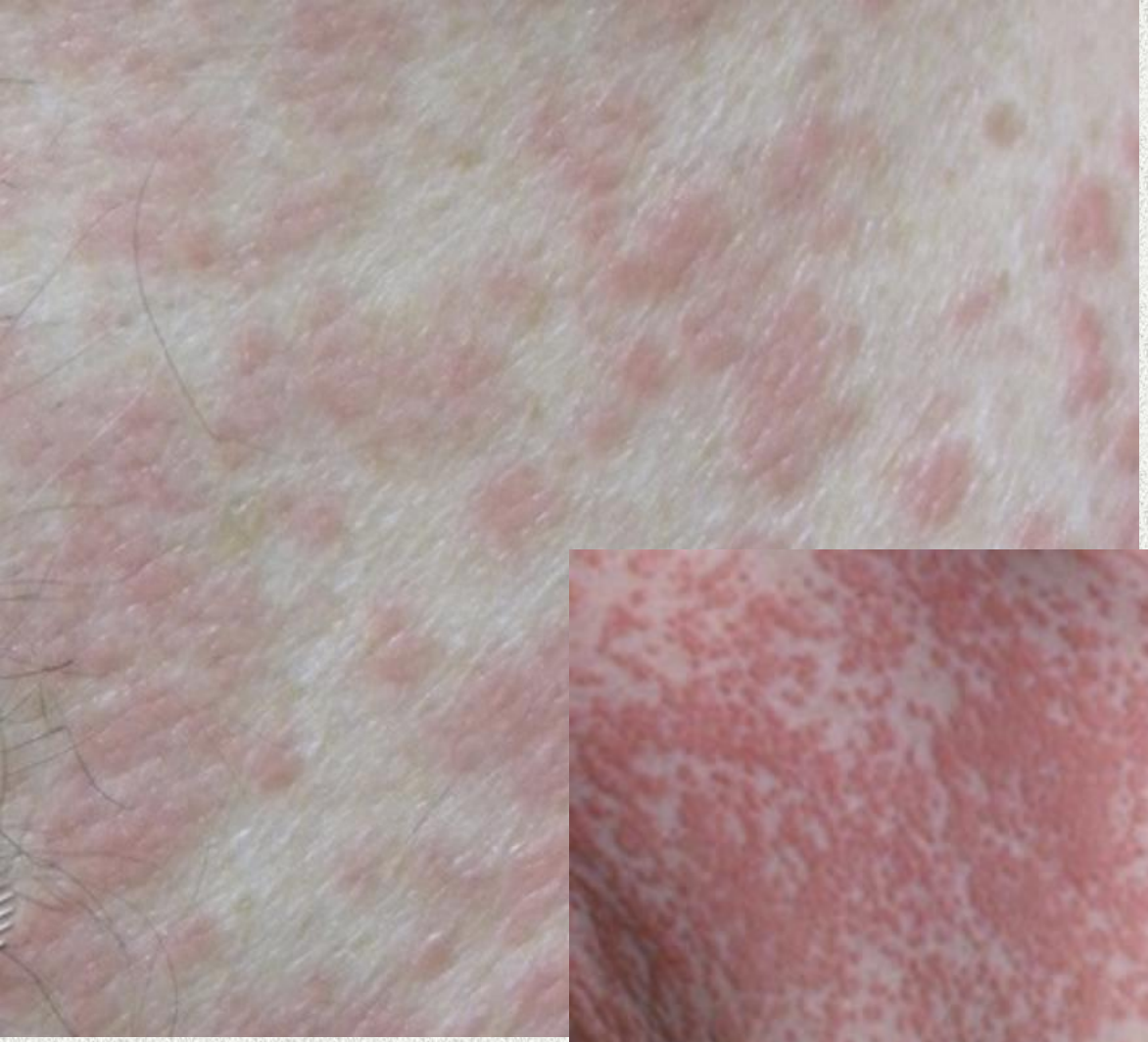
→ Never re-expose to causative agent – repeat exposures likely to be worse and more dangerous

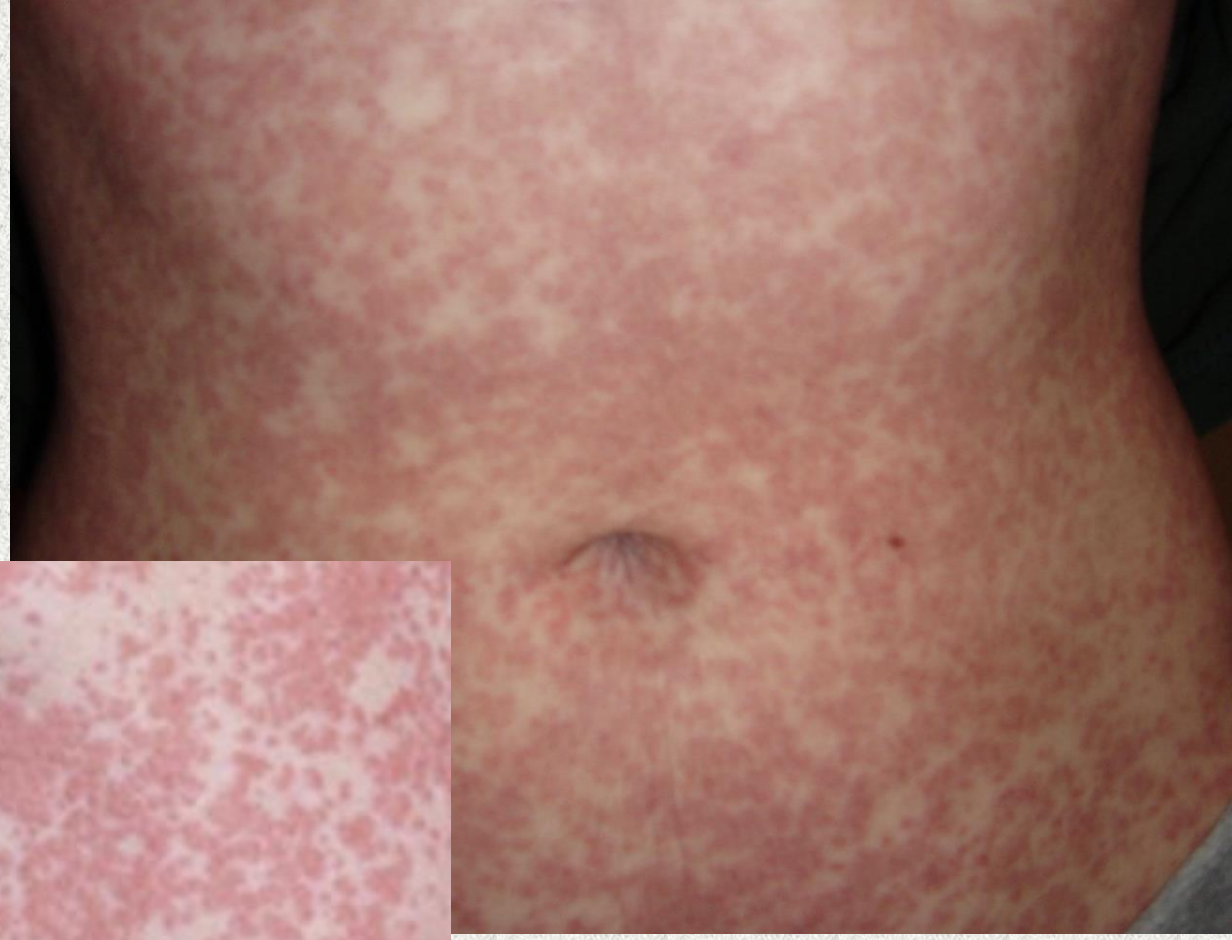
Morbilliform Drug Eruption

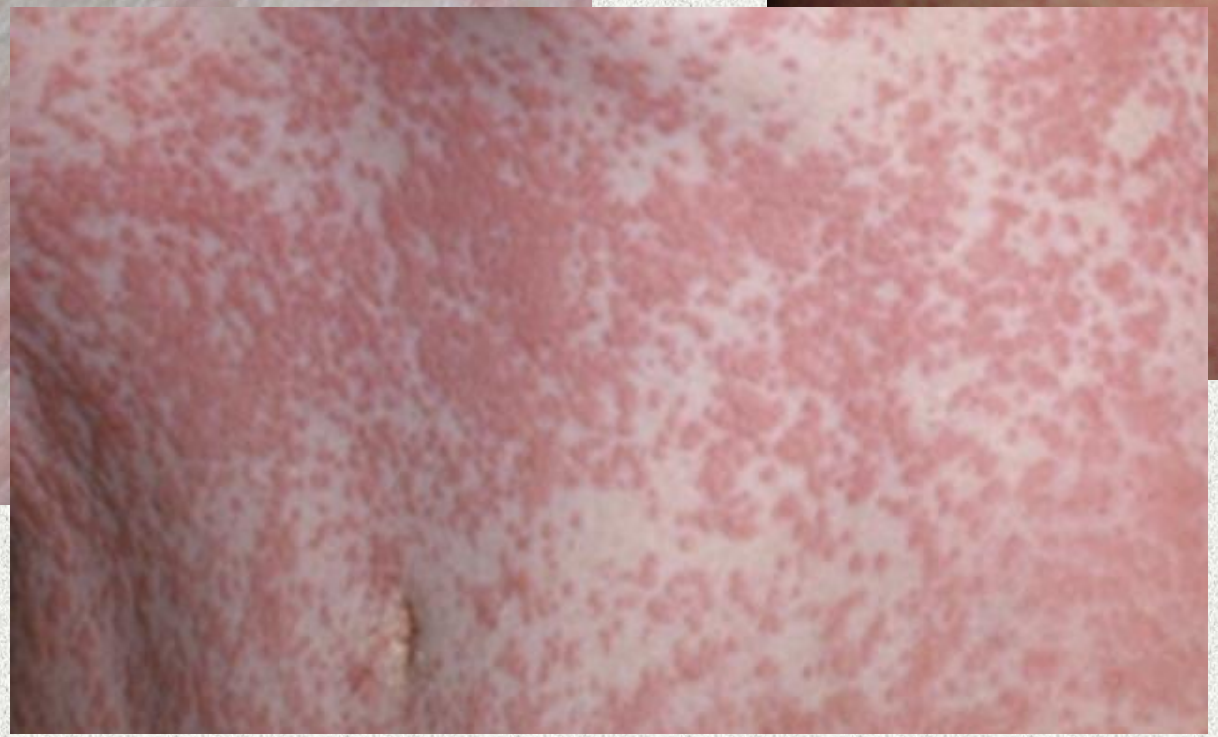
- Most common drug eruption
  - Not considered a SCAR (severe cutaneous adverse reaction) meaning acceptable to re-expose to offending drug and treat cutaneous reaction through course of exposure to drug
- Type IV (delayed) hypersensitivity reaction
- Causes – antibiotics (beta-lactams, sulfonamides), allopurinol, anti-epileptics, NSAIDs
  - Can also be seen in viral and bacterial infections (eg. viral exanthem) – more common in children
- Risk factors – prior morbilliform drug eruption, family history of drug allergy, concomitant EBV, HIV, cystic fibrosis

Morbilliform Drug Eruption – Presentation

- Onset (initial) – 1-2 weeks after starting offending drug
  - May even start after offending drug is stopped
  - Onset (re-exposure) – 1-3 days after starting drug
- Truncal onset with symmetric peripheral spread
- Erythematous macules and papules initially scattered but may coalesce into erythematous patches and plaques that then resolve over days to weeks with possible mild desquamation
  - Typically not petechial, but some patients may develop petechial lesions on lower extremities
  - No mucosal involvement
  - Can be more prominent on dependent locations – may be more heavily distributed on the back and posterior extremities of a pt confined to supine position in bed







https://dermnetnz.org/topics/morbilliform-drug-reaction
https://www.mdedge.com/familymedicine/article/65946/dermatology/body-rash
https://www.visualdx.com/blog/drug-eruptions-ten-tips-to-help-you-address-them/

Morbilliform Drug Eruption – Management in Hospitalized Patient

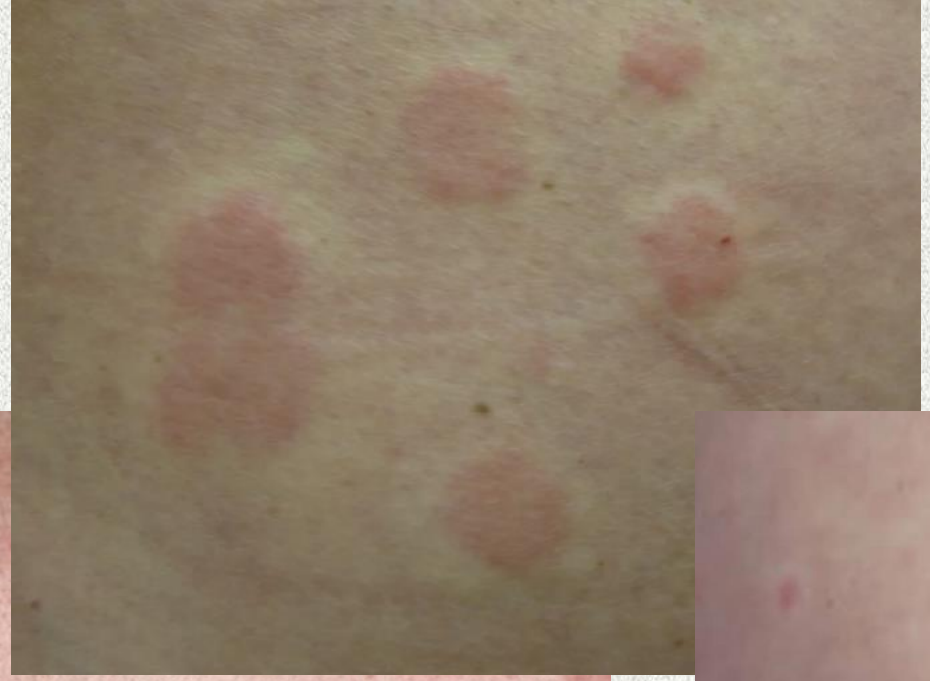
- Stop offending drug
- Labs to rule-out internal organ involvement
  - CBC, CMP, CRP, viral serology (EBV, measles, rubella, etc)
    - Eosinophilia can correlate with allergy
- Skin biopsy of diagnosis uncertain
- Monitor for signs indicating more severe and dangerous drug reaction
  - High fever, mucosal involvement, erythroderma, skin tenderness/duskiness, blistering, evidence of other organ involvement
- Topical steroids for pruritis (more benefit with occlusion) +/- antihistamines
- Continued use of offending agent and re-exposure – ok in morbilliform eruption → treat skin symptoms through course of exposure

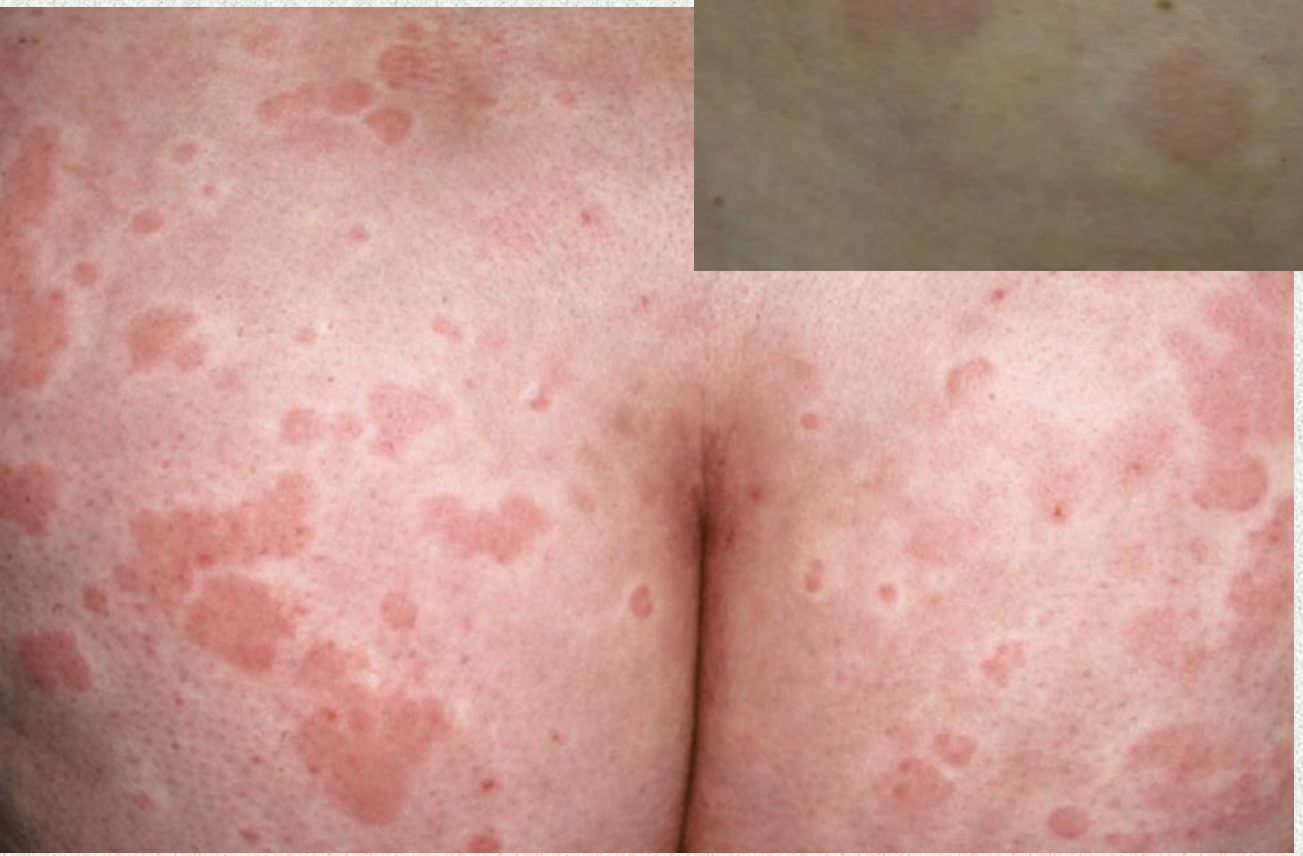
Urticarial Drug Eruption

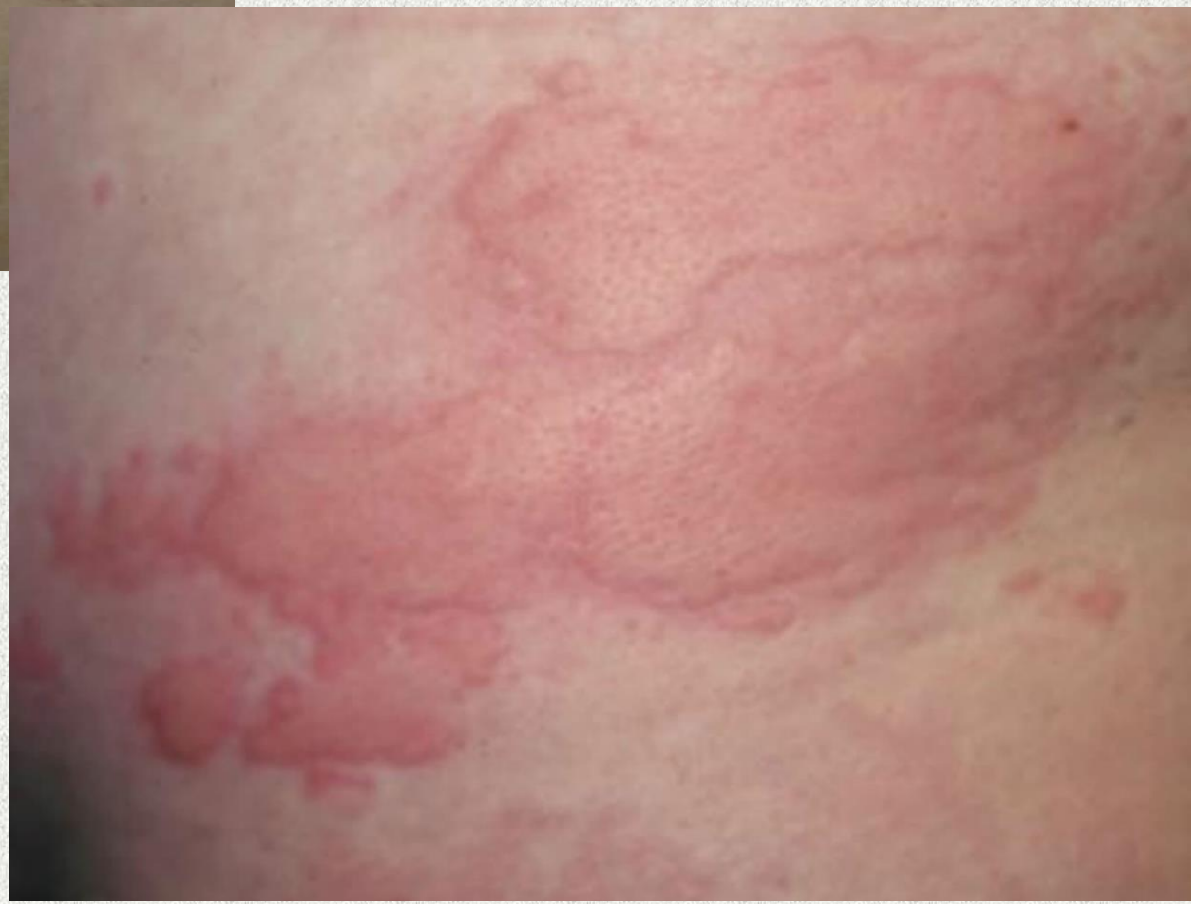
- Not considered a SCAR (severe cutaneous adverse reaction) meaning acceptable to re-expose to offending drug and treat cutaneous reaction through course of exposure to drug *ONLY IF it is not associated with angioedema, anaphylaxis, serum sickness, or severe systemic symptoms*
- Type I (immediate) hypersensitivity reaction, IgE mediated
  - Onset (initial) – within 3 weeks of exposure
  - Onset (re-exposure) – within minutes
- Causes – antibiotics (PCN, cephalosporins, sulfa), cytostatic agents, ACEIs, CCBs
- Other causes and variants
  - Mast-cell degranulators – ASA, NSAIDs, muscle relaxants, opiates
  - Angioedema – most commonly ACEIs
    - Delayed onset, localized and broad/confluent rather than scattered, primarily of eyelids, perioral, hands/feet, genitals, oral involvement (tongue, palate, larynx
  - Serum sickness – most commonly Cefaclor or blood transfusions
    - Urticaria with bruising, fever, arthralgias +/- LAD, nephritis, endocarditis

Urticarial Drug Eruption – Presentation

- Erythematous edematous pruritic papules/plaques with wheal & flare
  - Wheal – erythematous edematous plaque
  - Flare – white halo around wheal
- Individual lesions are transient – appear within minutes and resolve in <24 hours
- Can last weeks after offending agent removed
  - Acute – can last up to 6 weeks
  - Chronic – lasts longer than 6 weeks







https://www.dermatologytimes.com/view/tips-diagnosing-treating-urticaria
https://www.aafp.org/pubs/afp/issues/2017/0601/p717.html

Urticarial Drug Eruption – Management in Hospitalized Patient

- Stop offending drug
  - If not a/w angioedema or anaphylaxis, ok to continue offending medication and treat symptoms if medication absolutely needed
- Workup
  - Skin biopsy may confirm but may be non-specific and is often not necessary for diagnosis
  - No labs necessary in acute urticaria
  - Chronic urticaria – CBC and CRP
  - Angioedema – C4, C1-INH, C1q
  - Systemic symptoms (fever, arthralgias, malaise) – w/u for autoimmune conditions
  - Prick testing can be considered as outpatient

Urticarial Drug Eruption – Management in Hospitalized Patient

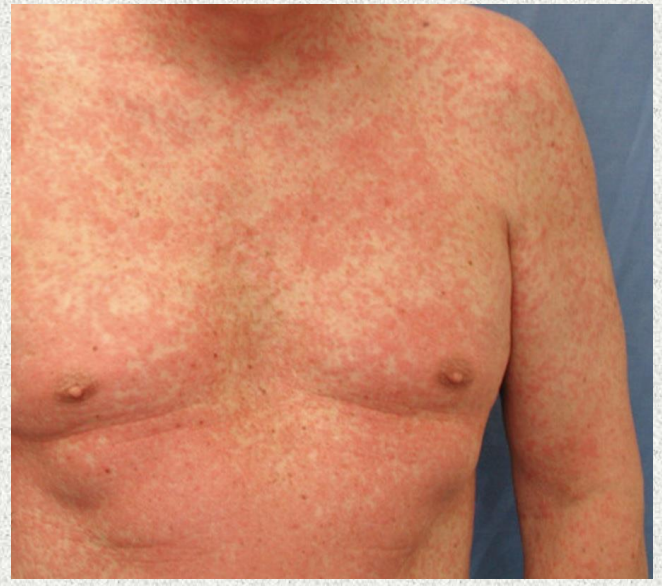
- Second-generation H1 antihistamines – some benefit with using both Cetirizine and Fexofenadine
  - Cetirizine – 10-40mg daily
  - Fexofenadine – 180-360mg daily
  - Others – Loratadine, Levocetirizine
- Add H2 antihistamines – Ranitidine 150-300mg daily
- Can add first-generation antihistamine – Diphenhydramine 100-300mg daily
  - More sedating and shorter duration of benefit than second-generation, not as beneficial or necessary
- Mid-potency to high-potency topical steroids for symptom (pruritis) control
- Prednisone taper for severe cases – up to 1mg/kg/d (typically max at 60mg/d) tapered over minimum of 3 weeks
  - Too short taper will lead to rebound flaring after course stopped

Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS)

- Acute, severe *systemic* adverse drug reaction
- SCAR – avoid re-exposure → absolute recurrence, likely worse, higher morbidity and mortality
- Causes – anticonvulsants (carbamazepine, phenobarbital, phenytoin), allopurinol, olanzapine, sulphonamide antibiotics
  - Onset – within 2 weeks with antibiotics, greater than 2 weeks with anticonvulsants and allopurinol
  - Anticonvulsants – estimated 1:10,000 patients
  - Allopurinol – risk increases in CKD and concomitant thiazide diuretics

DRESS – Presentation

- Symptoms develop over several days
- Fever of 38-40C followed by widespread rash which can last weeks
  - Morbilliform eruption
  - Facial edema
  - Mucosal involvement less common
  - Severity of cutaneous eruption does not
  - correlate with severity of systemic involvement



- Systemic symptoms may continue to worsen and persist for several weeks even after drug discontinuation
  - Generalized lymphadenopathy*
  - Atypical lymphocytes*, eosinophilia*, leukocytosis, thrombocytopenia, anemia
  - Transaminitis*, hepatitis, liver failure
  - Myocarditis*, pericarditis
  - Thyroiditis* (autoimmune)
  - Interstitial nephritis (mild and rare)
  - Interstitial pneumonitis, pleuritis, pneumonia, ARDS
  - Meningitis, encephalitis, polyneuritis
  - Gastroenteritis, pancreatitis, GI bleeding, actue colitis
  - Myositis
  - Uveitis

https://dermnetnz.org/topics/drug-hypersensitivity-syndrome

DRESS – Management in Hospitalized Patient

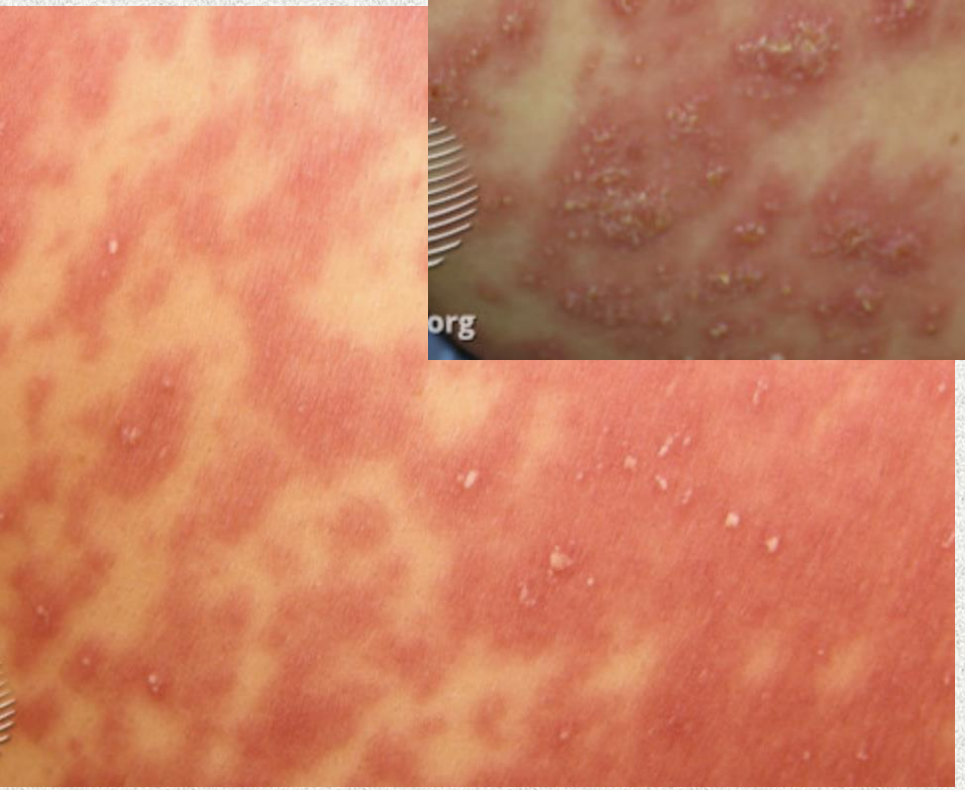
- Withdrawal of causative drug
- Systemic workup – evaluation depends on symptoms and early lab/imaging abnormalities
  - Delayed autoimmune thyroiditis – TSH/T4 during admission, at 6-12 weeks, as well as at 1 and 2 years post-DRESS
  - Fulminant myocarditis – echo during admission and at 3 weeks post-DRESS
  - Hemophagocytic syndrome, hepatic failure, multiorgan failure
- Topical steroids, emollients
- Oral antihistamines for pruritis
- Fluid, electrolyte and calorie management in severe systemic cases
- Prednisone in cases with severe systemic involvement
  - Benefit is uncertain, should be tapered slowly to prevent rebound
- Other systemic therapies – ciclosporin, IVIG, plasmapheresis, cyclophosphamide, mycophenolate, rituximab

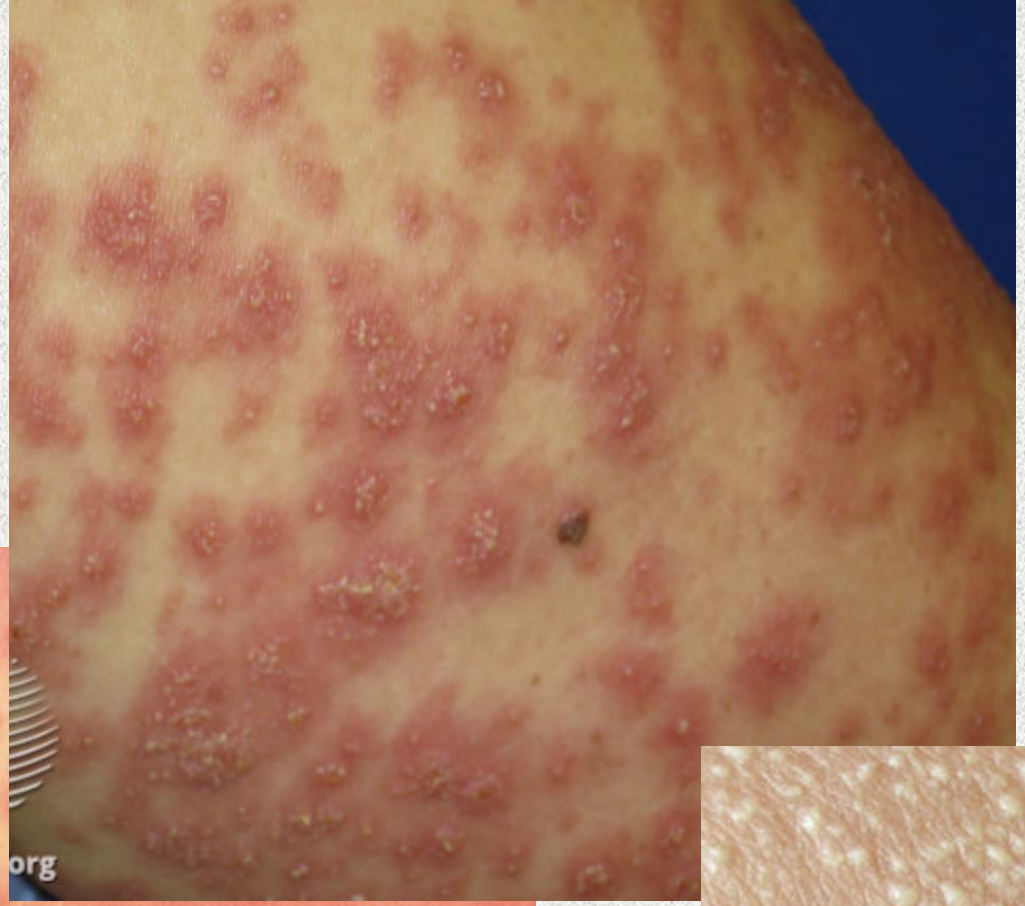
Acute Generalized Exanthematous Pustulosis (AGEP)

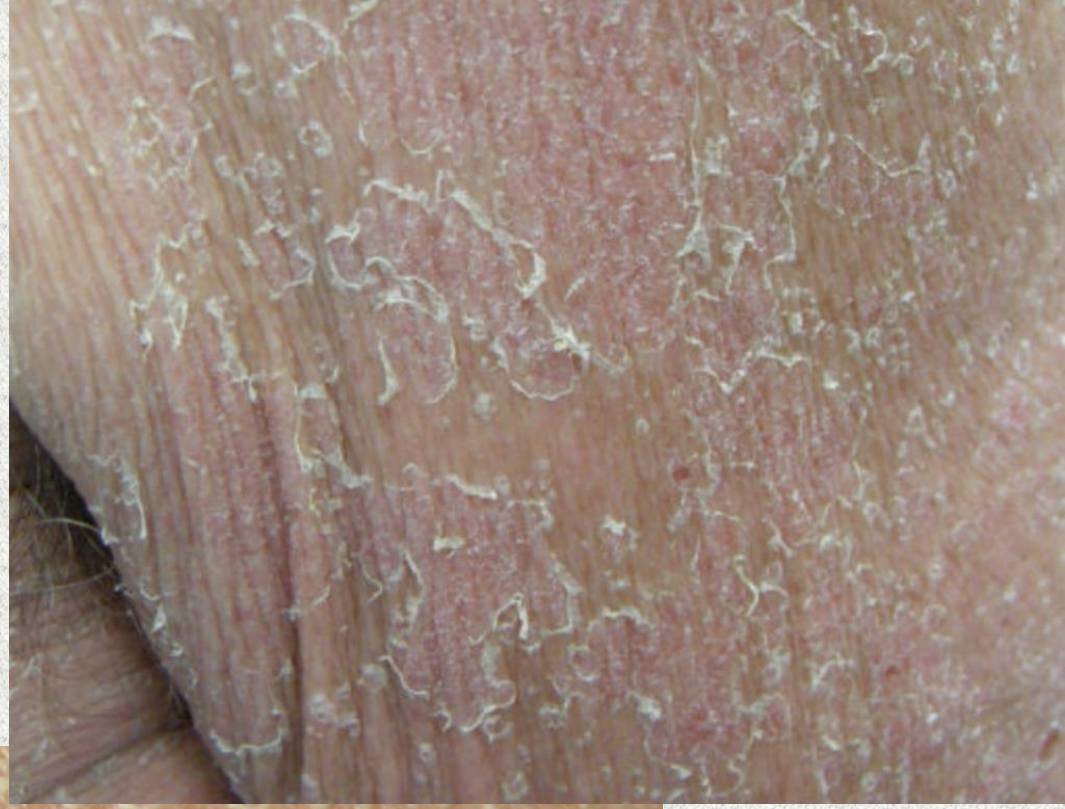
- Acute *cutaneous* eruption due to medication
- SCAR – avoid re-exposure → absolute recurrence, likely worse, higher morbidity and mortality
- Causes – beta-lactam antibiotics (penicillins, cephalosporins, quinolones), tetracyclines, sulfonamides, terbinafine, calcium channel blockers, hydroxychloroquine, carbamazepine, acetaminophen, viral infection (less common – EBV, HBV, CMV, enterovirus, adenovirus)

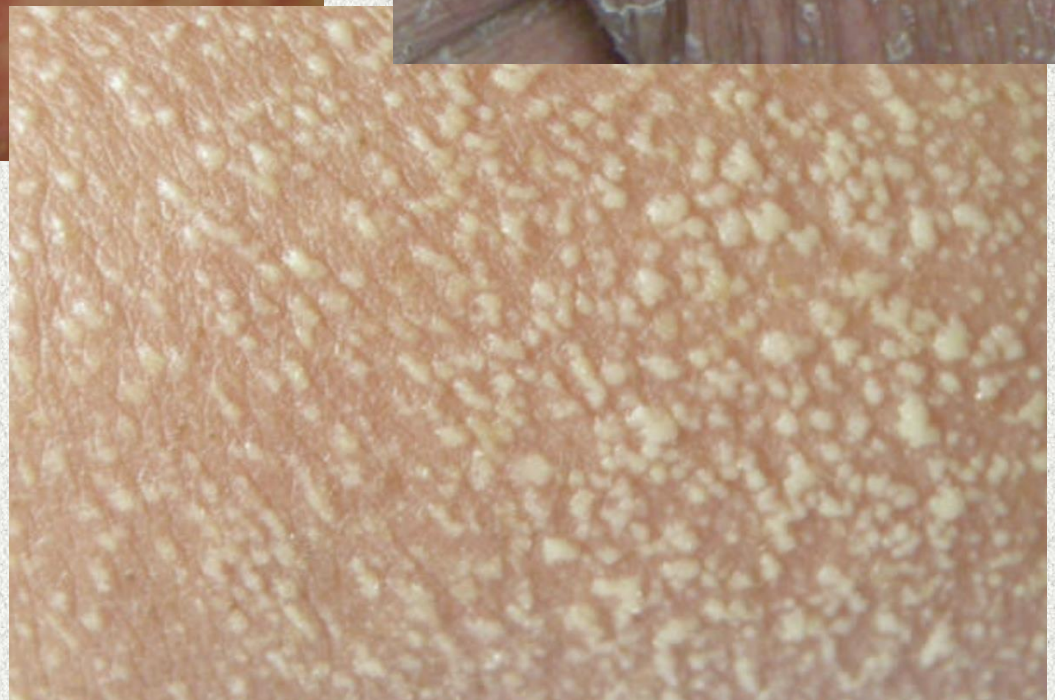
AGEP – Presentation

- Rapid onset
- Patchy erythema with tiny sterile pustules
  - Starts in armpits/groin then becomes more widespread, but remains more prominent in skin folds
- Facial swelling possible
- Oral lesions less common (~20%)
- Fever, malaise possible, but patient is generally not unwell
- Persists for 1-2 weeks followed by focal superficial skin desquamation
- Systemic involvement uncommon (~10%) – hepatic, renal, pulmonary, hematologic









https://dermnetnz.org/topics/acute-generalised-exanthematous-pustulosis

Bolognia JL, Schaffer JV and Ceroni L. (2018). Dermatology, 4th Ed: Ch 21.

AGEP – Management in Hospitalized Patients

- Discontinue causative drug
- Topical steroids, emollients, topical analgesics
- Oral antihistamines
- Systemic therapy rarely indicated

Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN)

- Acute severe, *systemic* eruption due to medication
- SCAR – avoid re-exposure → absolute recurrence, likely worse, higher morbidity and mortality
- Spectrum of diseases determined by % of skin involvement
  - SJS <10% TBSA -- SJS/TEN overlap 10-30% TBSA -- TEN >30% TBSA
- Causes
  - Medications (most common) –
    - Antibiotics (40%) – sulfonamides, penicillins, cephalosporins
    - Other common drugs – anticonvulsants and allopurinol
    - Less common drugs – acetaminophen, NSAIDs, nevirapine
  - Infections (very rare) – CMV
- Presentation
  - Onset variable – within 1 week with antibiotics, few days to 3 weeks with most other drugs, up to 2 months with anticonvulsants
  - Prodrome – flu-like symptoms (fever, sore throat, runny nose, cough, conjunctivitis, red sore eyes, malaise and myalgias) + painful skin

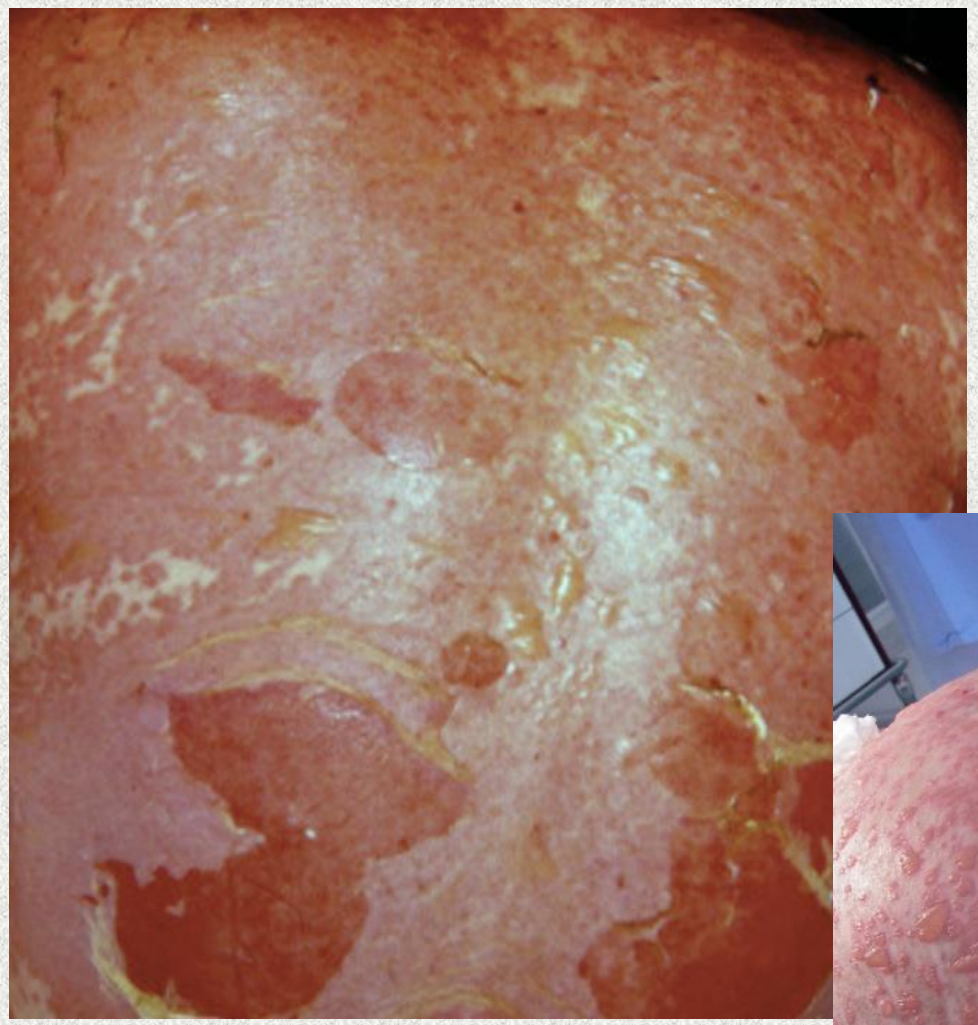
SJS/TEN – Presentation

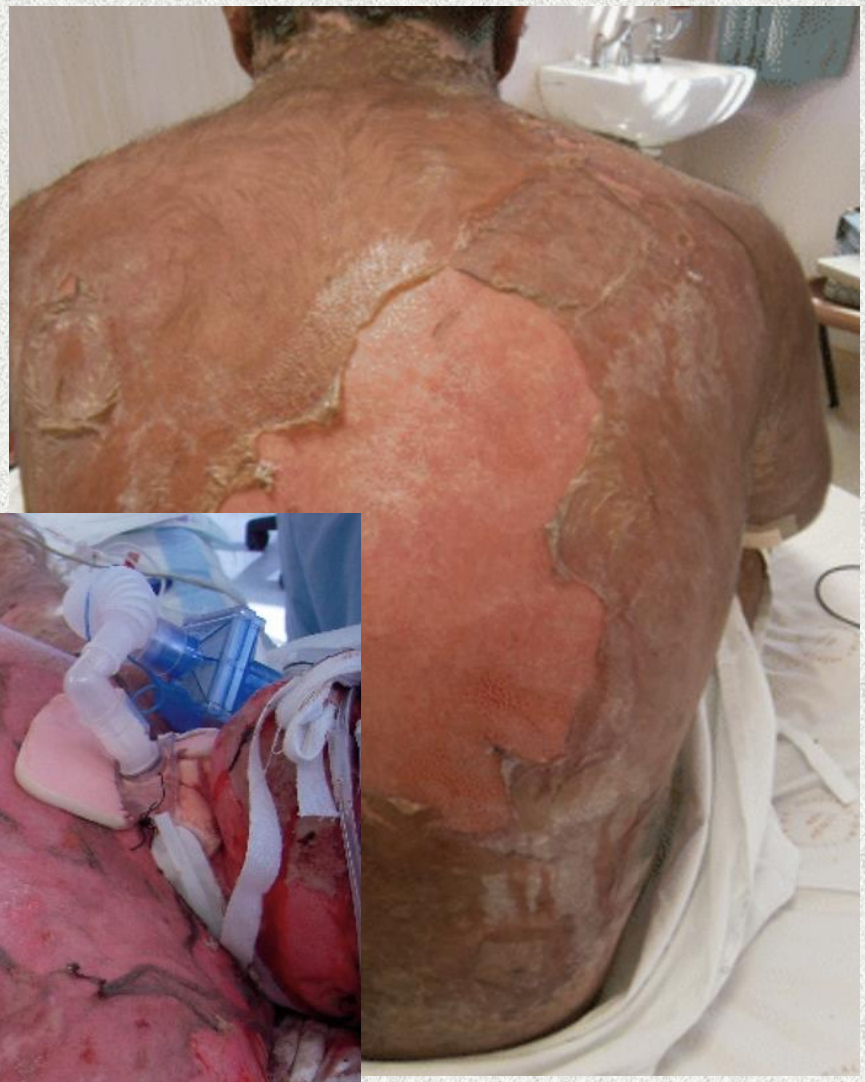
Cutaneous Presentation

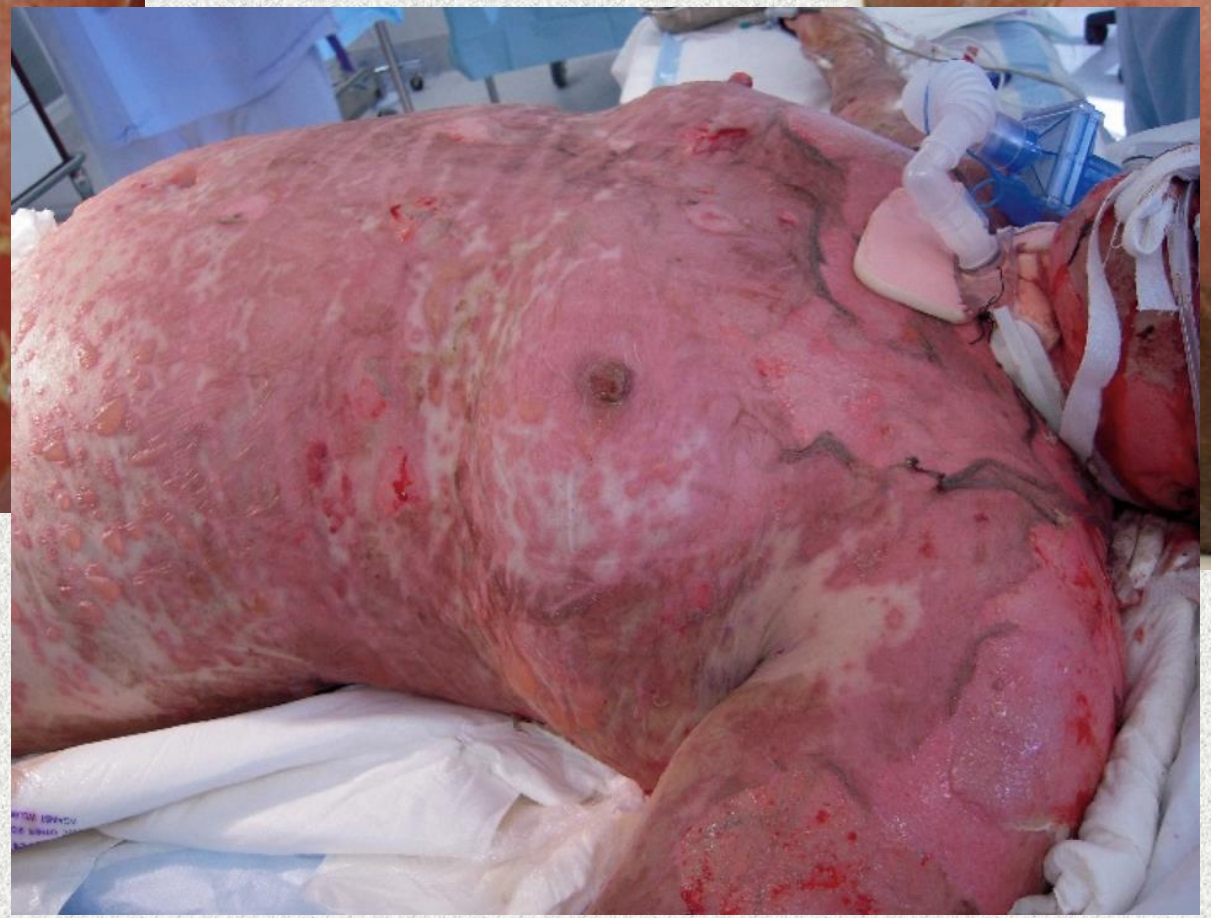
- Abrupt onset of tender skin lesions starting on trunk and rapidly spreading to face and extremities over few days
  - Scalp, palms/soles – not involved
- Morphology
  - Erythematous, dusky or purpuric macules
  - Diffuse erythema
  - Targetoid or bullous (flacid)
- Skin lesions coalesce → skin sloughs in sheets → oozing red raw dermis
- Nikolsky sign – rubbing of finger on skin causes shearing of epidermis

Mucosal Presentation

- Severe mucosal involvement classic with SJS/TEN
- Oral – red crusted painful lips and mouth ulcers → chelitis, stomatitis
- Ocular – red sore eyes → conjunctivitis, ulceration, uveitis, photosensitivity
- Genital – erosions/ulcers → urinary retention, scarring
- Respiratory – sloughing of trachea/bronchus → coughing, respiratory distress requiring intubation







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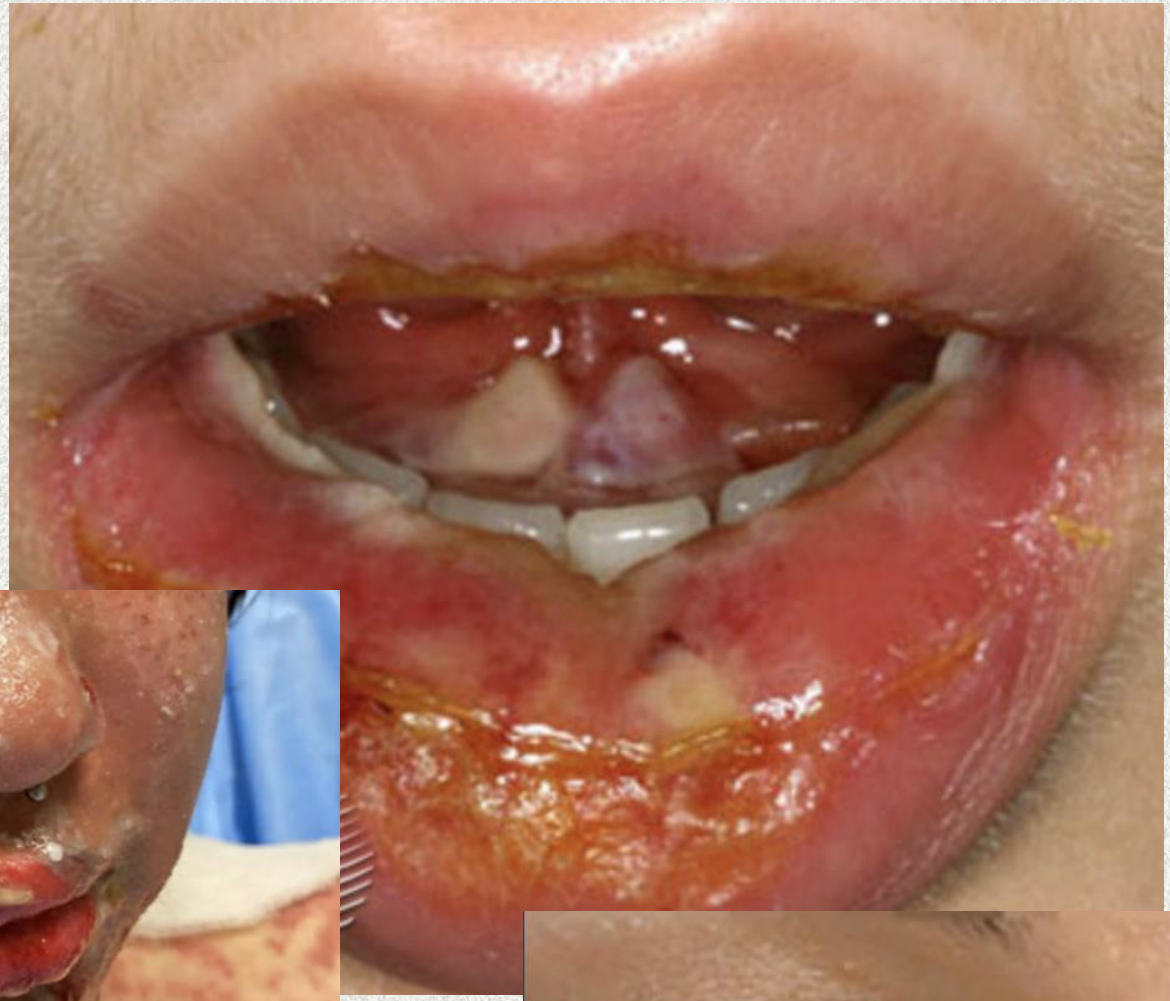

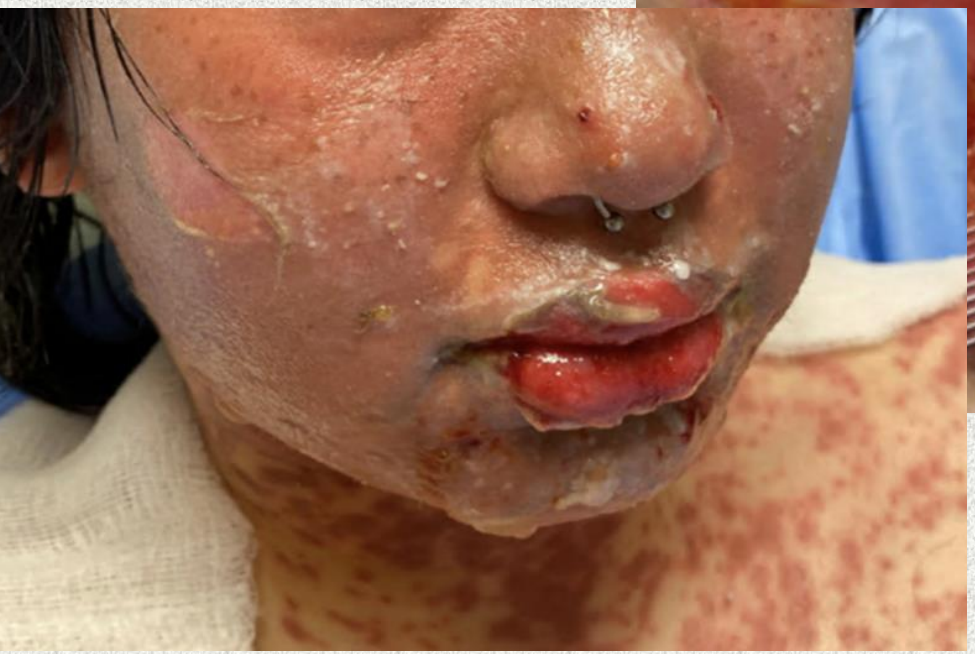

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SJS/TEN – Complications

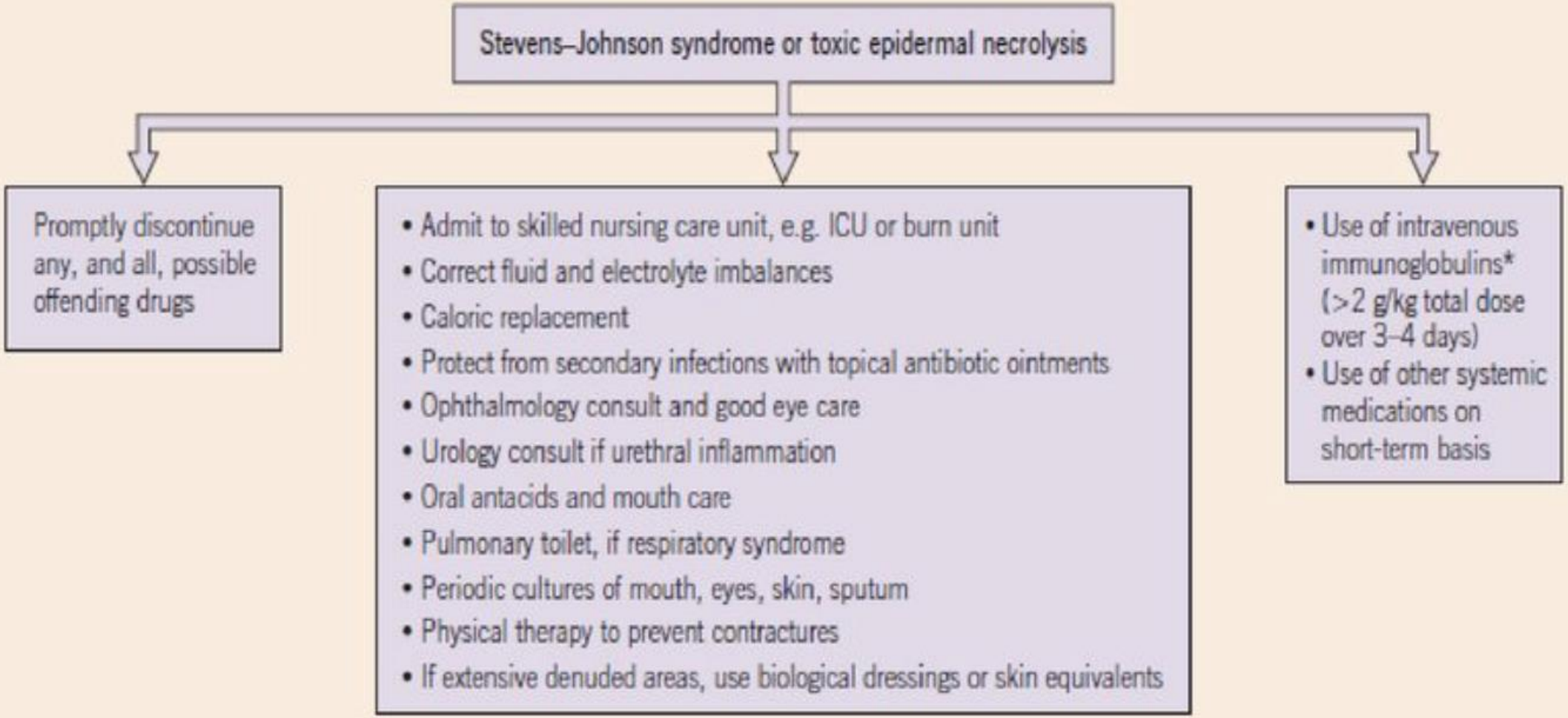
Early

- Mortality – 1-5% in SJS, 25-35% in TEN
- Dehydration
- Malnutrition
- Infection – cutaneous and hematologic
- Respiratory distress
- Gastrointestinal ulceration and perforation
- Shock, multiorgan failure

Long-Term

- Widespread pigment alteration
- Widespread scarring (may appear similar to burn scarring)
- Xerophthalmia, epiphora, conjunctivitis, corneal scarring, symblepharon, ectropion/entropion, trichiasis
- Scarring permanent loss of nails
- Phimosis (foreskin scarring), vaginal introitus scarring
- Joint contractures
- Chronic bronchitis, obstructive lung disease

APPROACH TO THE PATIENT WITH STEVENS–JOHNSON SYNDROME OR TOXIC EPIDERMAL NECROLYSIS



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Infectious

Cellulitis

Necrotizing fasciitis

Herpes zoster

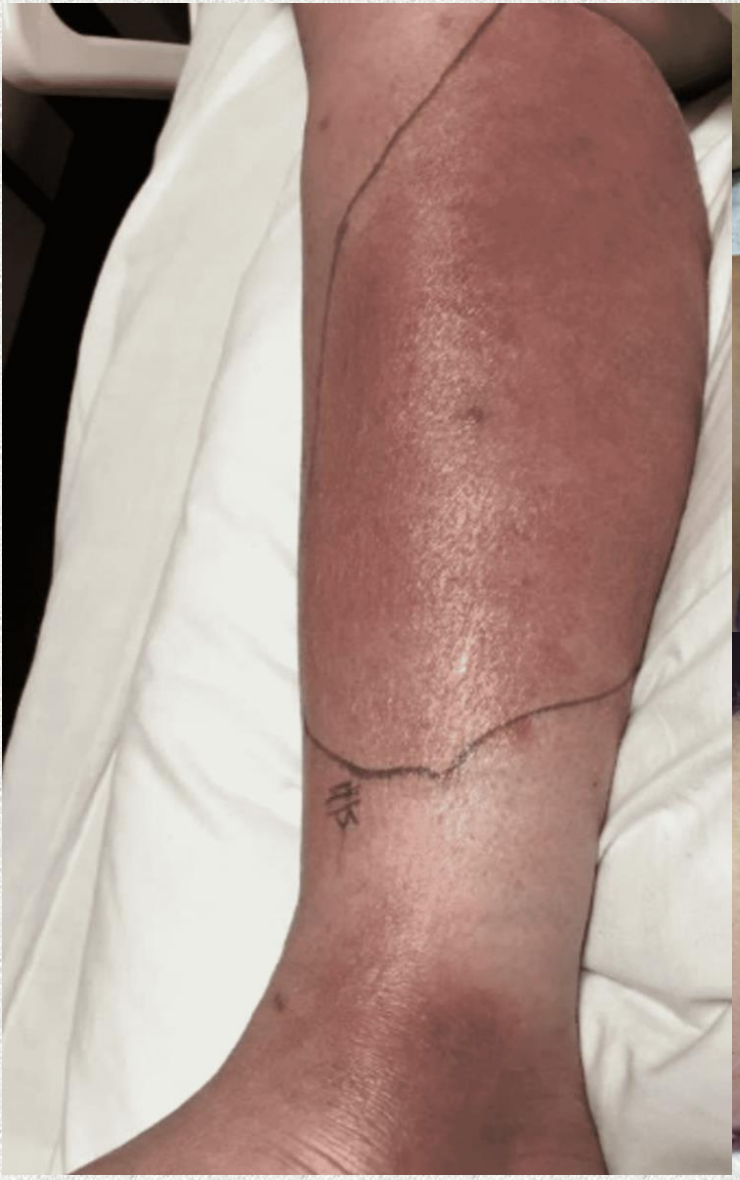
Bacterial endocarditis

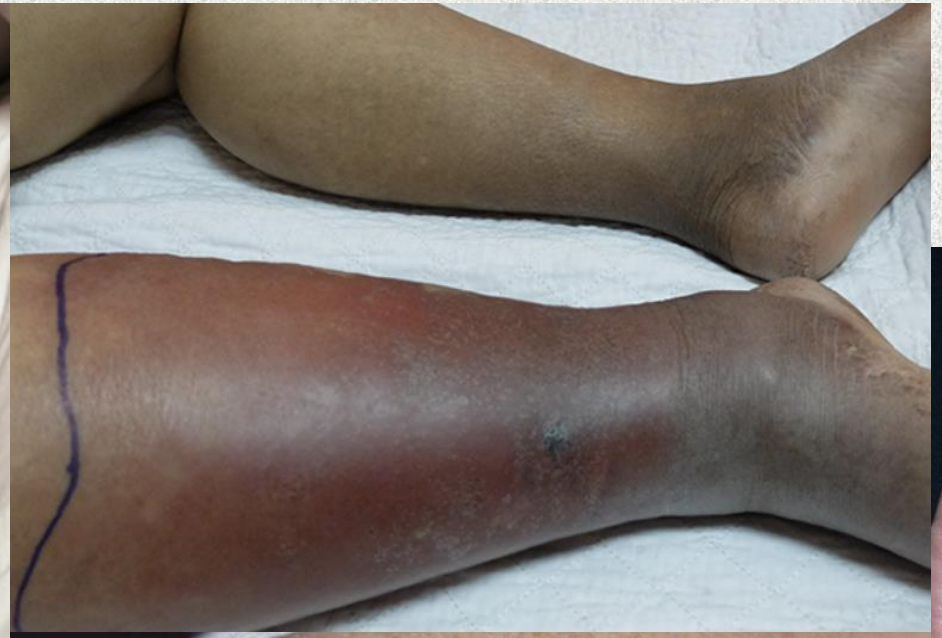
Cellulitis

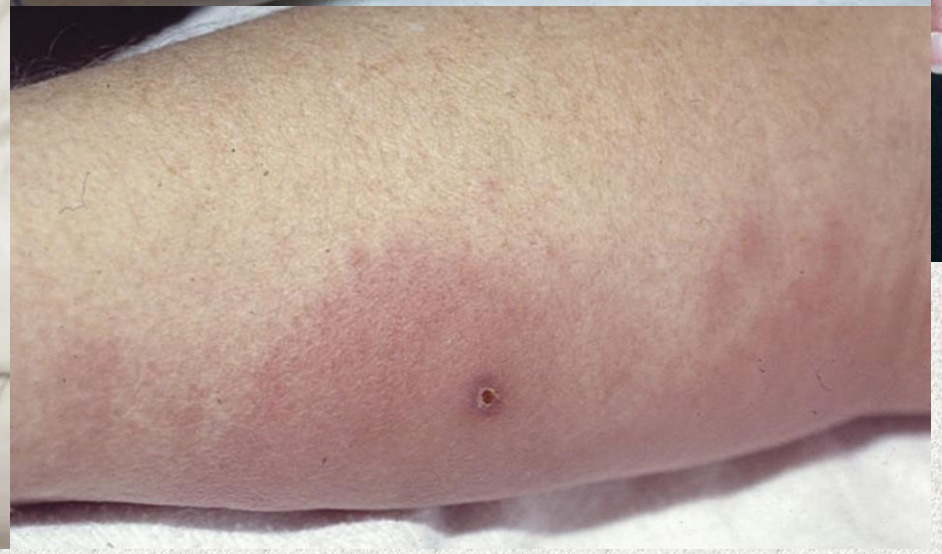
- Bacterial infection of deep dermis and subQ
  - Erysiplas – superficial bacterial infection of upper dermis
- Risk factors – hx cellulitis, broken-down skin (eg tinea, stasis ulcer), injury, surgical site, immunodeficiency/immunosuppression, DM, CKD/liver disease
- Causes – strep pyogenes and staph aureus (MC), pseudomonas, haemophilus, pasteurella, vibrio, erysipelothrix
- Presentation – localized, expanding, erythematous, edematous/indurated, dimpled (peau d'orange), warm, painful skin
  - Systemic symptoms may start first as a sign of bacteremia – fever, chills, rigors, malaise
  - Lymphangitis/lymphadenitis – erythematous streaking from cellulitis to nearby LAD
  - Bilateral disease unlikely → think stasis dermatitis instead

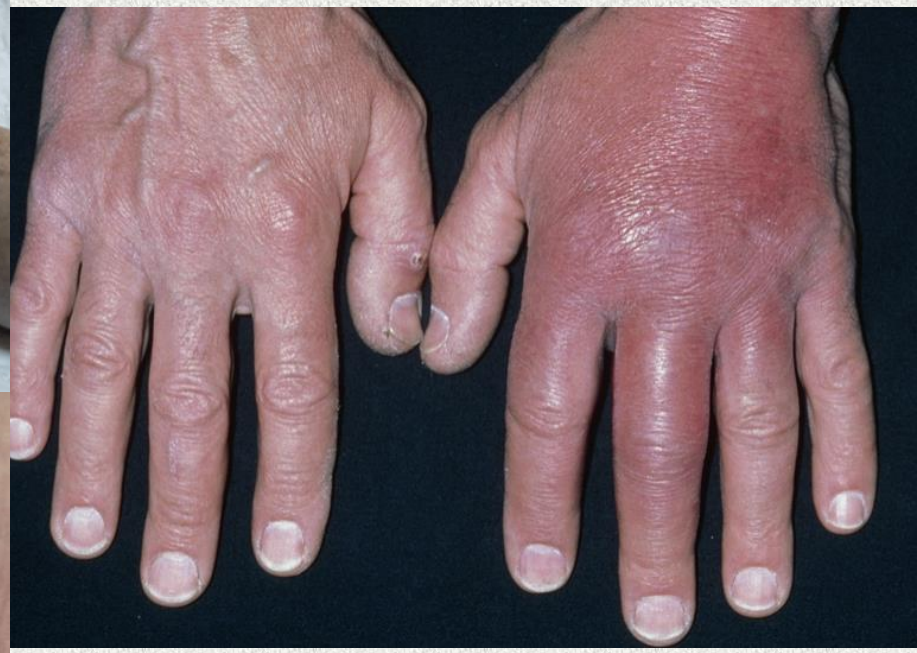
Cellulitis – Management

- Workup
  - Labs – leukocytosis, elevated CRP, culture (tissue, blood)
    - Skin swab will not diagnose cellulitis, often shows skin flora
- Management
  - Mark edges to monitor for progression or improvement
  - Uncomplicated (no systemic symptoms) – PO antibiotics
  - Complicated (systemic symptoms) – IV antibiotics, IV fluids, O2
  - Recommended antibiotic – PCN G, flucloxacillin, augmentin, ceftriaxone/cefotaxime/cefazolin, clindamycin, SMX/TMP, doxycycline, vancomycin, linezolid, daptomycin
  - Transition from IV to PO when fever subsides, cellulitis regresses, CRP declines









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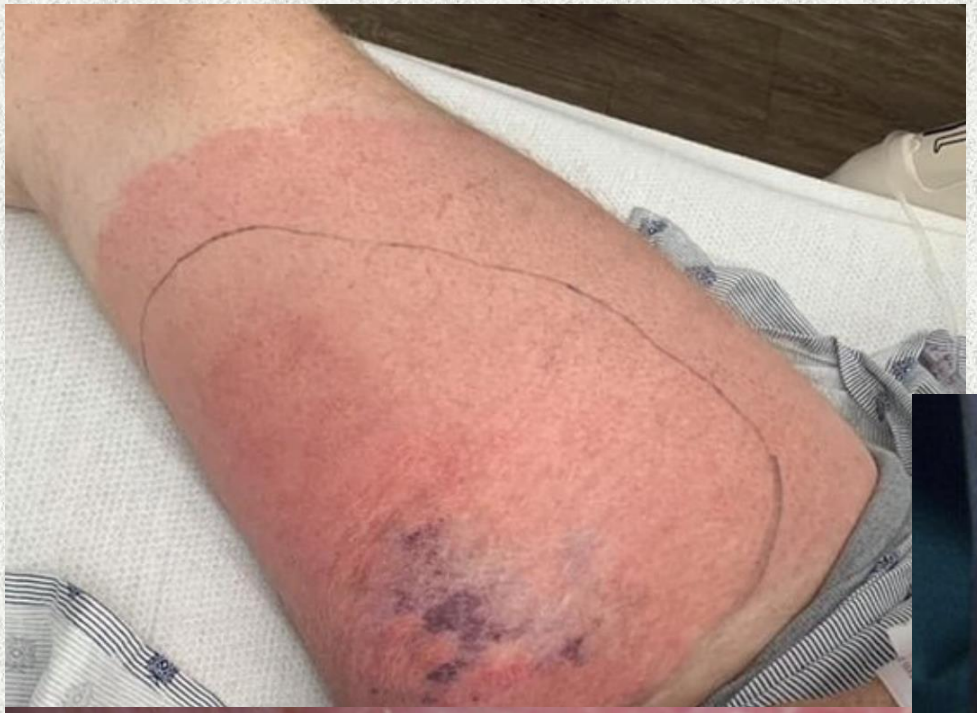
https://www.dermatologyadvisor.com/home/decision-support-in-medicine/dermatology/cellulitis-and-erysipelis/
https://www.nhs.uk/conditions/cellulitis/

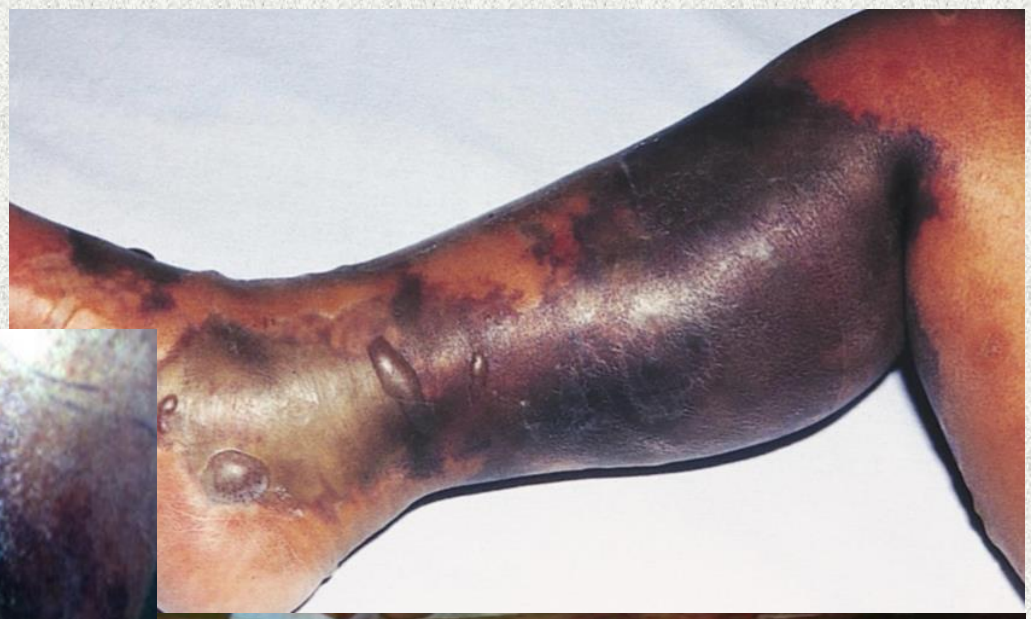
Necrotizing Fasciitis

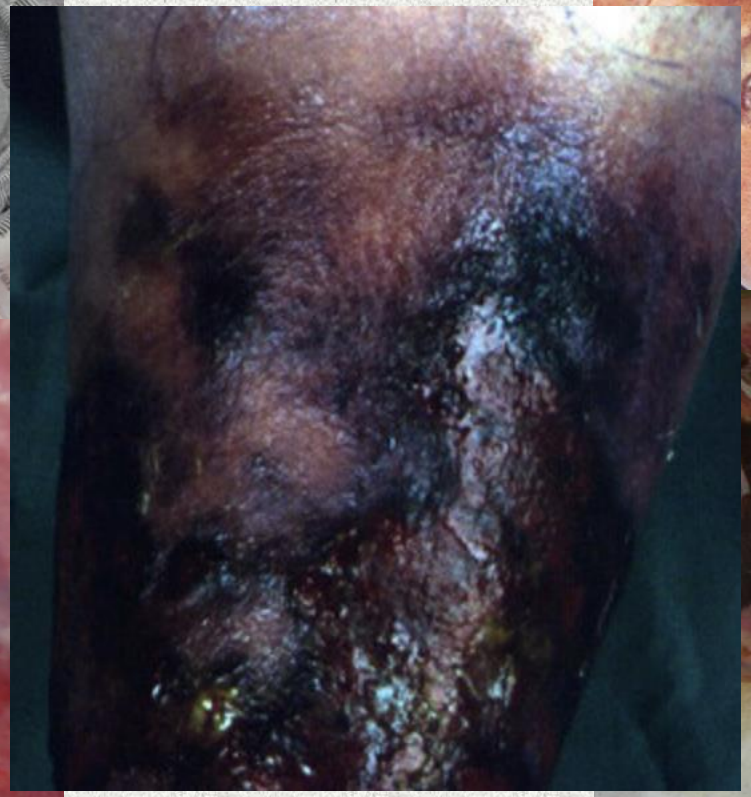
- Severe rapidly spreading bacterial infection of soft tissue and fascia resulting in vascular occlusion and necrosis of tissue with possible rapid progression to septic shock, multiorgan failure and death
  - Infection starts in superficial fascia then spreads horizontally through fascial plane before spreading vertically to overlying skin and underlying muscle → diagnosis may be delayed due to initial horizontal spread
  - Mortality up to 25%
- Can involve any location
  - Leg most common site
  - Fournier gangrene – perineal, genital, perianal → 15-50% mortality rate
- Risk factors – advanced age, obesity, DM, chronic illness, malignancy, immunosuppression, ASA/NSAIDs, drug abuse, traumatic injury
- Causes – 5 subtypes depending on cause
  - Hemolytic group A strep, staph, clostridium, haemophilus, vibrio, fungal, marine

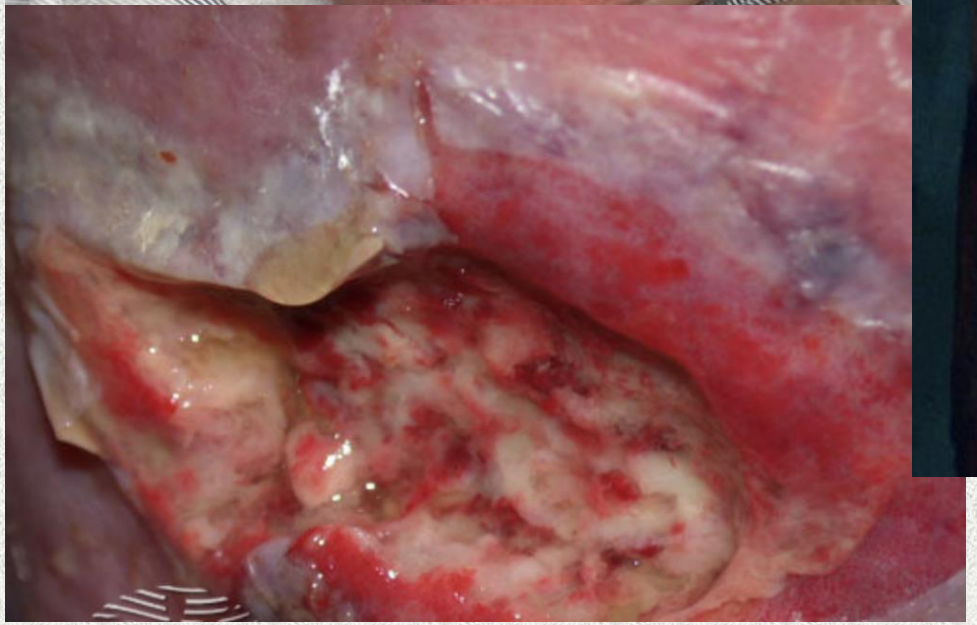
Necrotizing Fasciitis – Presentation

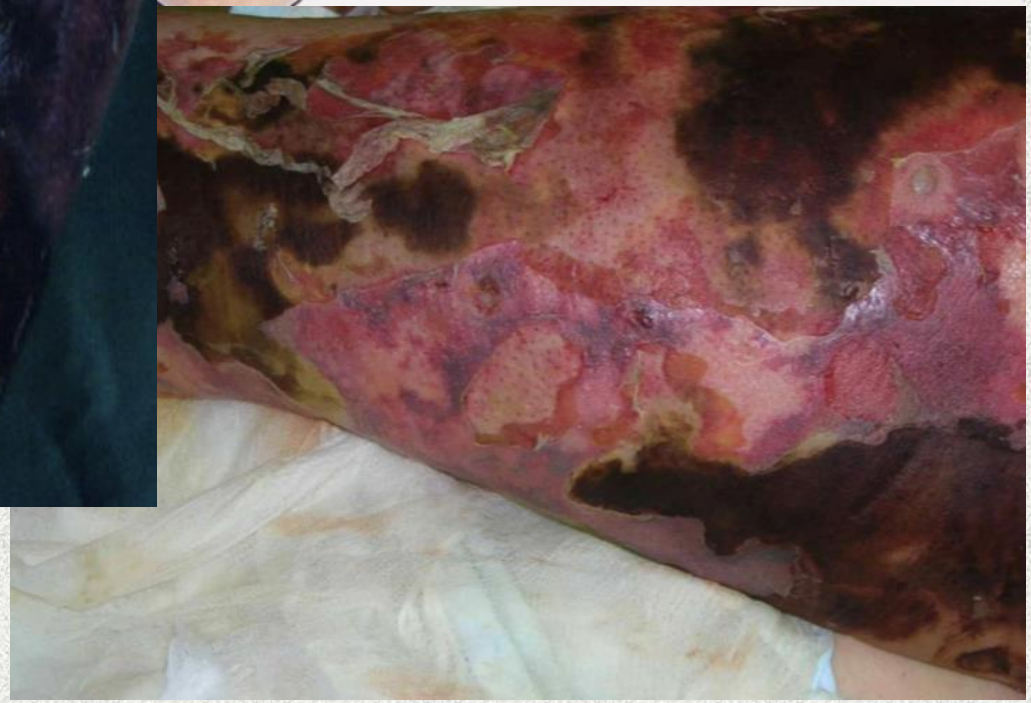
- Symptoms appear within 24 hours of injury with clinical features taking 3-4 days to appear
- Pain out of proportion to appearance that progressively worsens
- Flu-like symptoms
- Intense thirst common
- Site of infection – violaceous and edematous → duskiness with development of hemorrhagic and necrotic bulla → necrosis
  - Crepitus present due to gas within tissue
  - Severe pain followed by anesthesia due to necrosis involving sensory nerves
- Even with immediate antibiotic intervention, infection may not improve
  - Within 4-5 days – high fevers, hypotension → septic shock and multiorgan failure
  - Spread of abscesses to liver, lung, spleen, brain, pericardium, skin











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Necrotizing Fasciitis – Management

Diagnosis

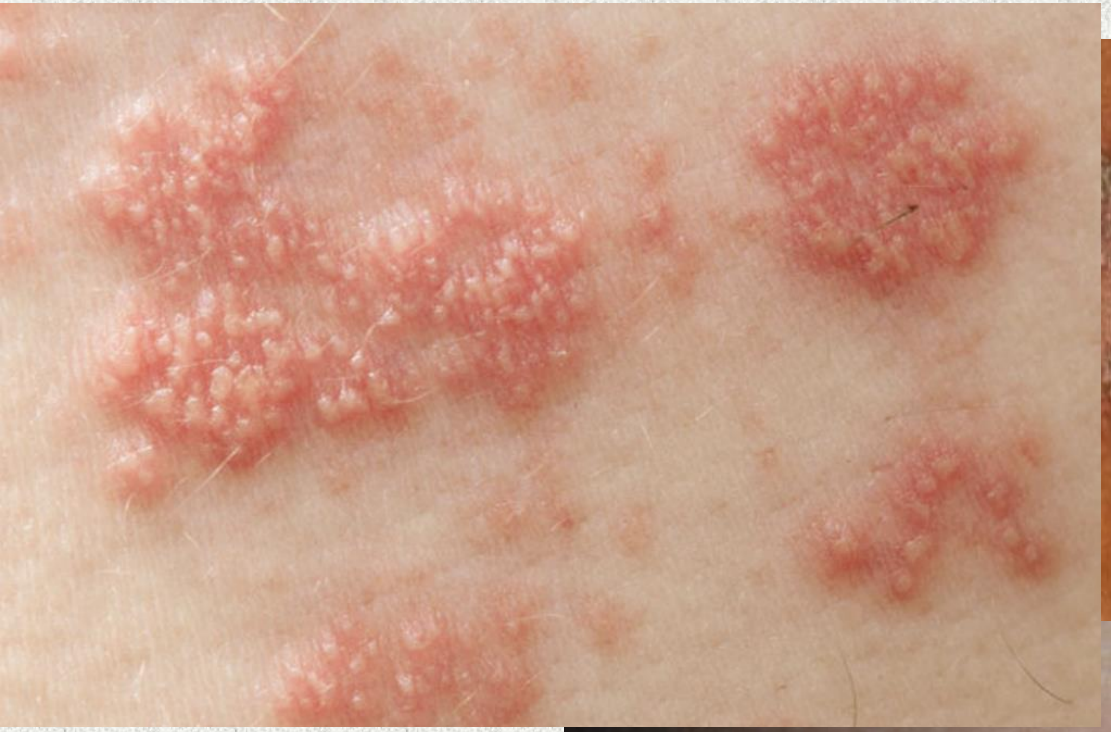
- Primarily clinical
- Labs – WBC >15.4x10^9/L, sodium <135mmol/L, CRP >16mg/dL, CK >600U/L, urea >18mg/dL
- Imaging – xray, CT, MRI → can identify gas in tissues
- Cultures – blood, tissue (with gram stain)

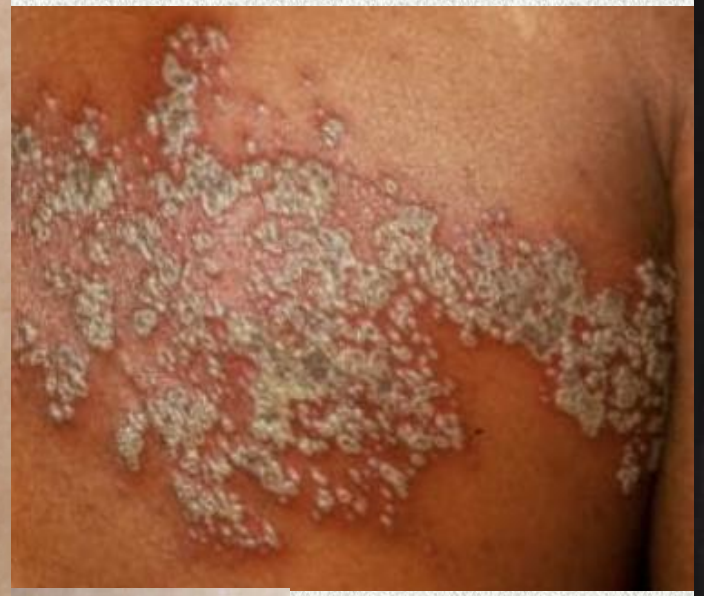
Management

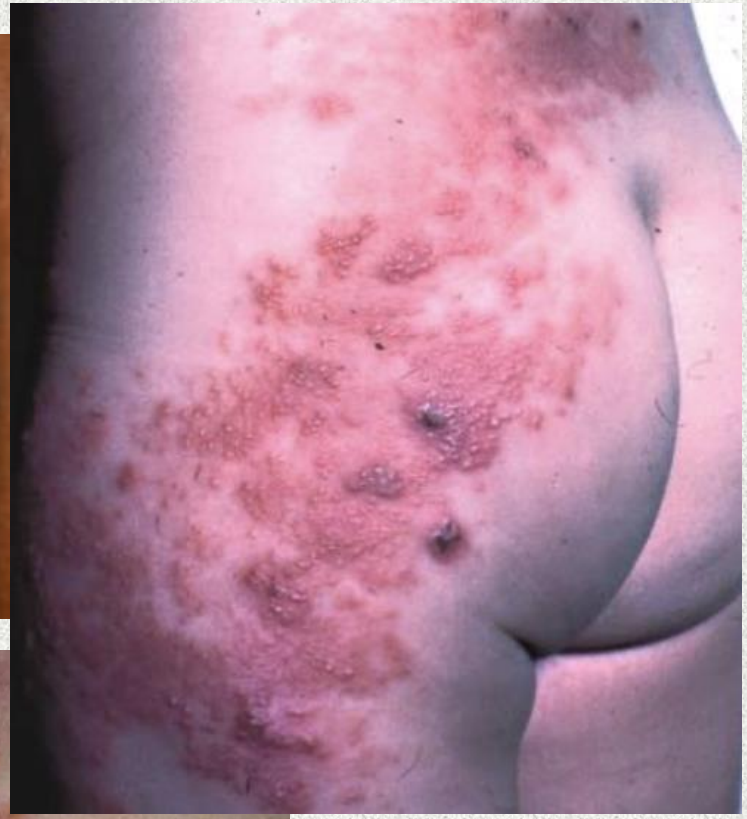
- ICU admission
- Supportive measures – oxygen, fluids, blood pressure management
- High dose IV antibiotics while awaiting tissue culture results
  - Penicillin, clindamycin, metronidazole, cephalosporins, carbapenems, vancomycin, linezolid
- Transition to high dose IV antibiotics specific to tissue culture results as soon as possible
- Urgent surgical debridement of all infected and necrotic tissue repeated frequently as disease progresses
  - Skin grafting usually required after infection completely cleared
- Other – hyperbaric O2, IVIg

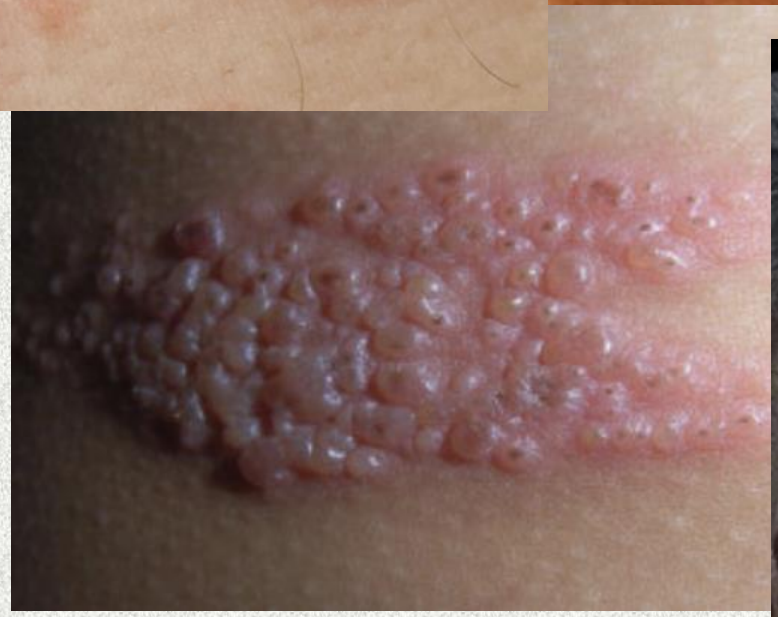
Herpes Zoster

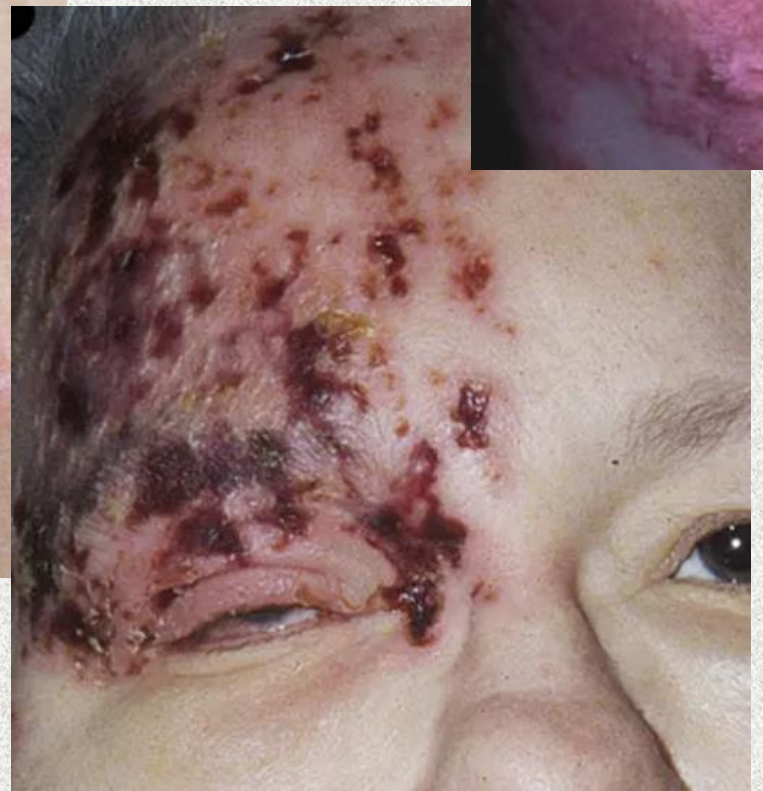
- VZV dormant in dorsal root ganglia → reactivation and spread down sensory nerves to skin
- Risk factors – elderly, malignancy, immunosuppression/ immunodeficiency
- Contagious to those who have not had chickenpox or vaccination until all vesicles are crusted over
  - Avoid exposure to pregnant women, children, elderly
- Presentation – chest, neck, forehead, lumbosacral locations most common
  - Prodrome – pain localized along 1-2 sensory nerves, may be focal or spread along track of nerve, non-tender, no cutaneous lesions; fever, headache, possible LAD
  - Clustered and scattered erythematous papules → progress to vesicles on erythematous base spread along 1-2 unilateral dermatomes → crust over before resolving
    - Stops at anterior and posterior midlines (however, may cross midline by few cm but is not bilateral)
    - Blisters appear within 3 days of pain, new lesions appear as old lesions resolve over 3-4 weeks











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Herpes Zoster – Management

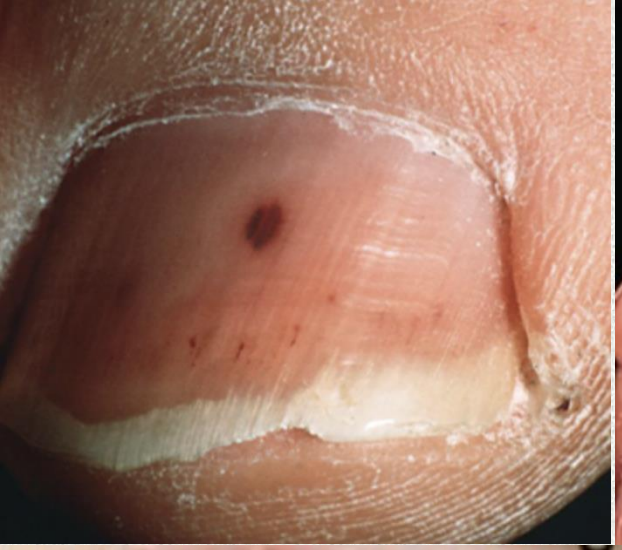
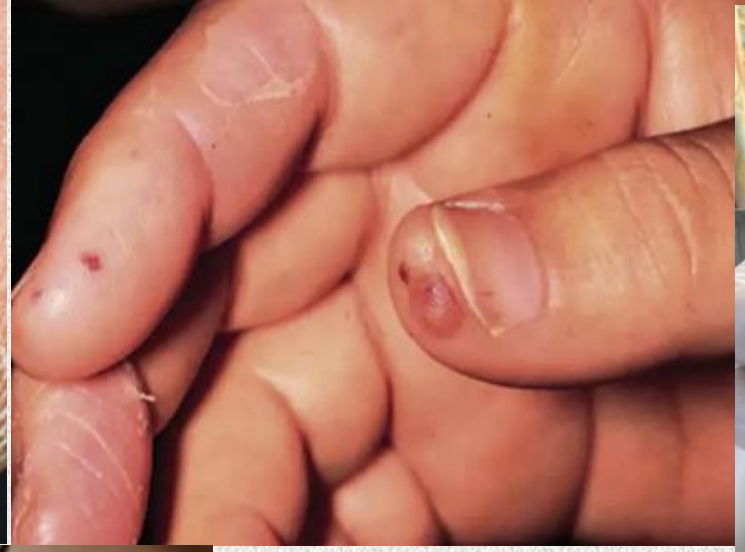
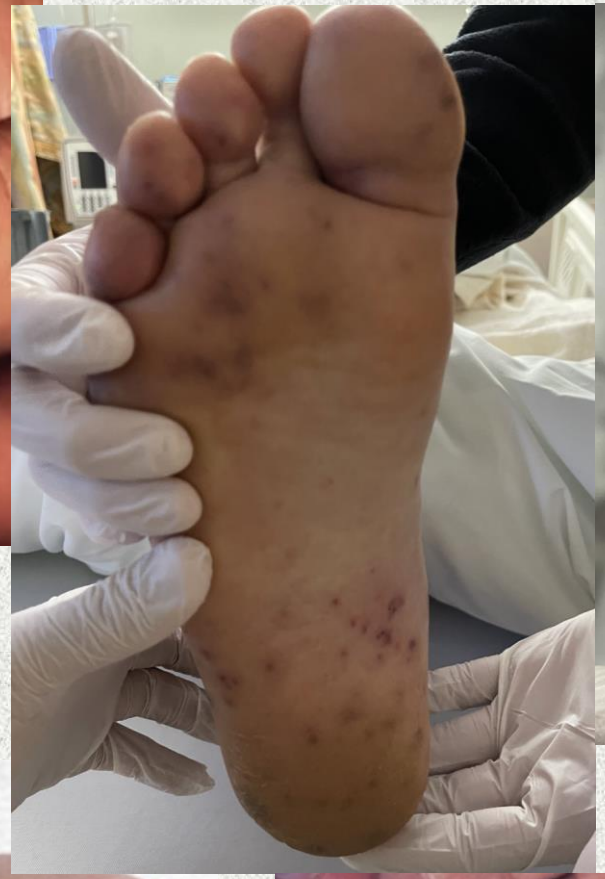
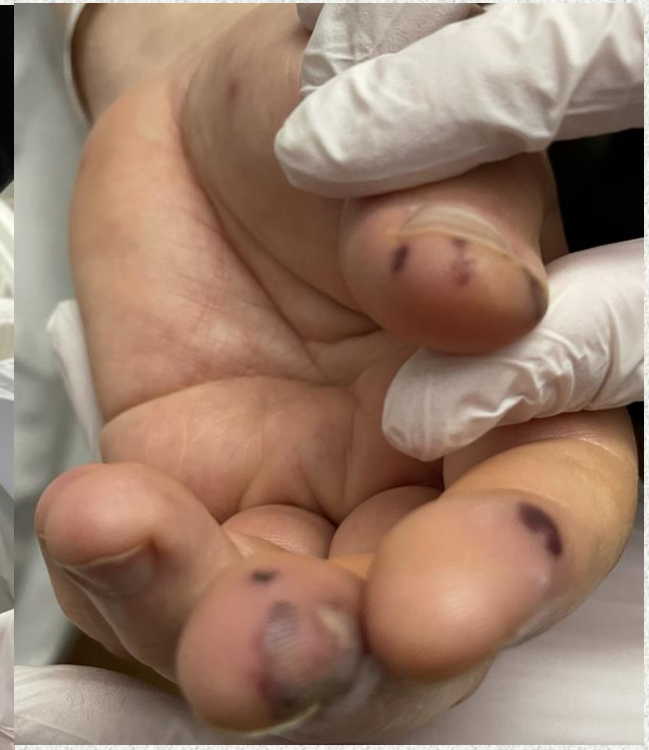
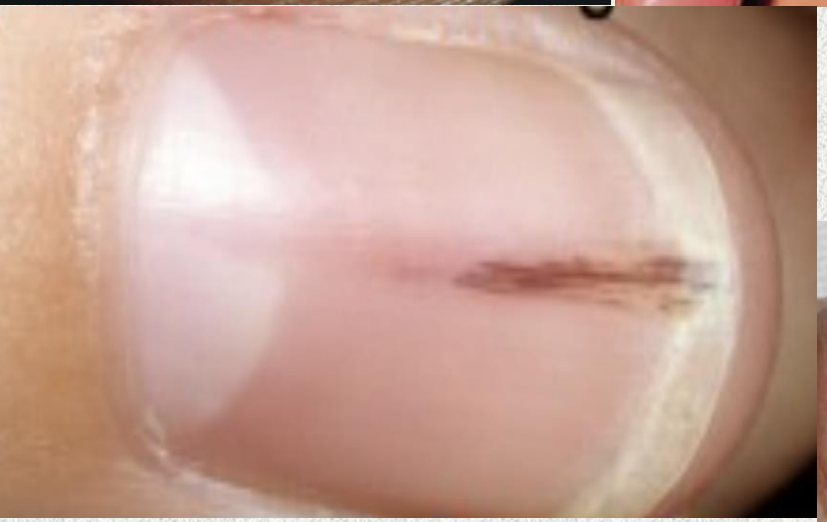
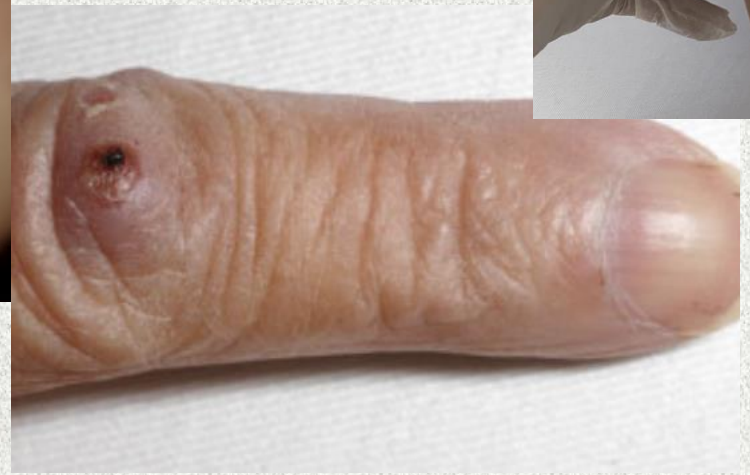
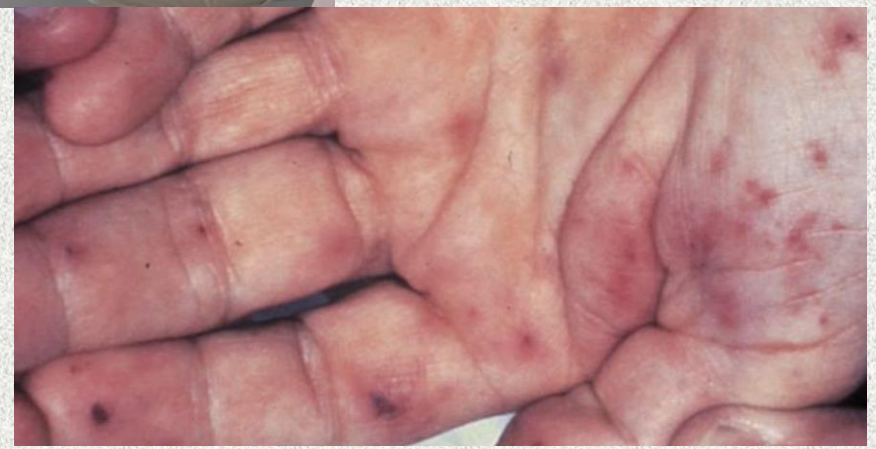
- Emergencies
  - Immunocompromised → at risk for disseminated and visceral zoster and is considered an emergency → IV acyclovir or foscarnet (acyclovir-resistant)
    - VZV vaccine contraindicated d/t risk of disseminated zoster
  - Herpes zoster ophthalmicus – Hutchinson's sign (lesions on tip/side of nose) → zoster of the anterior ethmoidal branch of the nasociliary branch of the ophthalmic division of the trigeminal nerve → high risk of ocular involvement (keratitis, uveitis, blindness, ocular cranial nerve palsy) → emergent Ophtho consultation
  - Ramsay Hunt syndrome (herpes zoster oticus) – lesions in ear canal → involvement of geniculate ganglion of cranial nerve VII → unilateral facial weakness/paralysis, dizziness → failure to initiate antivirals within 72 hours increases risk of permanent weakness/paralysis
- Complications
  - Post-herpetic neuralgia (PHN) – pain or pruritis persisting >1mo after onset, greater risk in older pts and in facial distribution, pain is either continuous burning or intermittent shooting, a/w skin numbness or hyperesthesia
    - Initiating antivirals greater than 72 hours after onset much less likely to minimize risk of PHN
  - Pregnancy – most harmful in 1st trimester, chickenpox in fetus/newborn in 3rd trimester (risks of scarring)

Herpes Zoster – Management

- Aciclovir 800mg q5 hours x 7 days OR Valaciclovir 1g q8 hours x 7 days, must be started within 72 hours
- PO steroids – no proven efficacy
- PHN – topical lidocaine, capsaicin, gabapentin/pregabalin, amitriptyline
  - NSAIDs and opioids not helpful

Bacterial Endocarditis

- Bacteria in bloodstream infect heart valves
- Risk factors – abnormal valves d/t rheumatic fever, valve replacement/repair, congenital abnormalities
- Causes – staph, strep viridans, enterococcus, pseudomonas, bartonella, others possible
- Presentation – fever, malaise, fatigue, SOB, CP, palpitations
- Cutaneous presentation
  - Splinter hemorrhages of proximal nail beds
  - Osler nodes – erythematous-violaceous painful nodules with central palor on fingers and toes, last for hours to days, suspected to be septic micro-emboli
  - Janeway lesions – non-tender petechia/purpura on palms (thenar and hypothenar eminences) and soles of feet, last days to weeks, suspected to be septic micro-emboli
- Treatment – IV abx based on species and susceptibility of bacteria and type of valve involved (native or prosthetic)
  - PCN or Ceftriaxone (MC), PCN w/ Gentamicin (enterococci), Vancomycin (high level resistance)Rifampin+Gentamicin+Nafcillin/Oxacillin/Cefazolin/Vanc (prosthetic valve w/ staph)

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Inflammatory

Atopic dermatitis

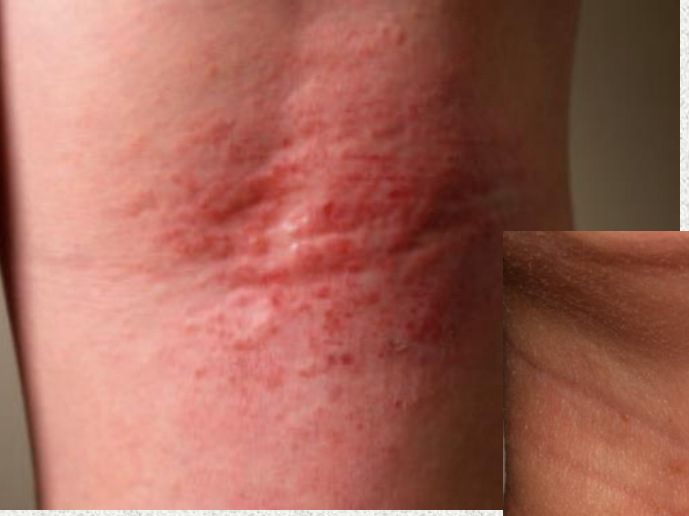
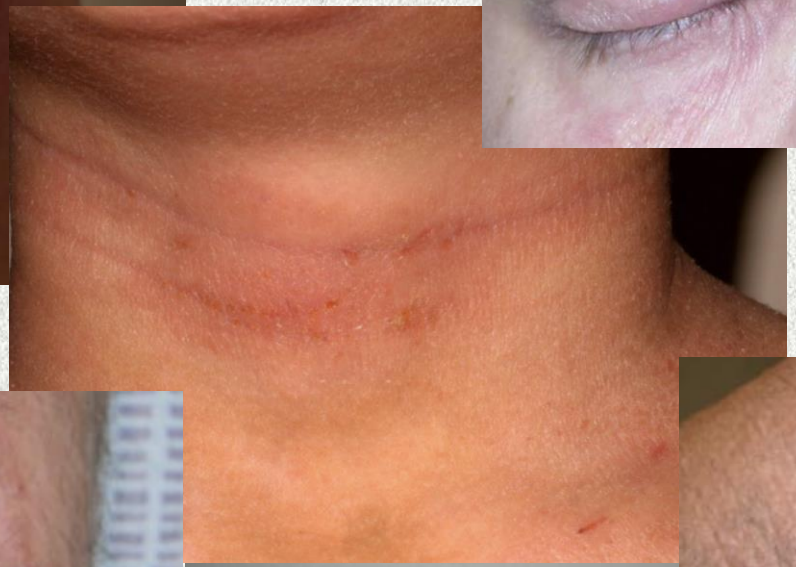
Psoriasis

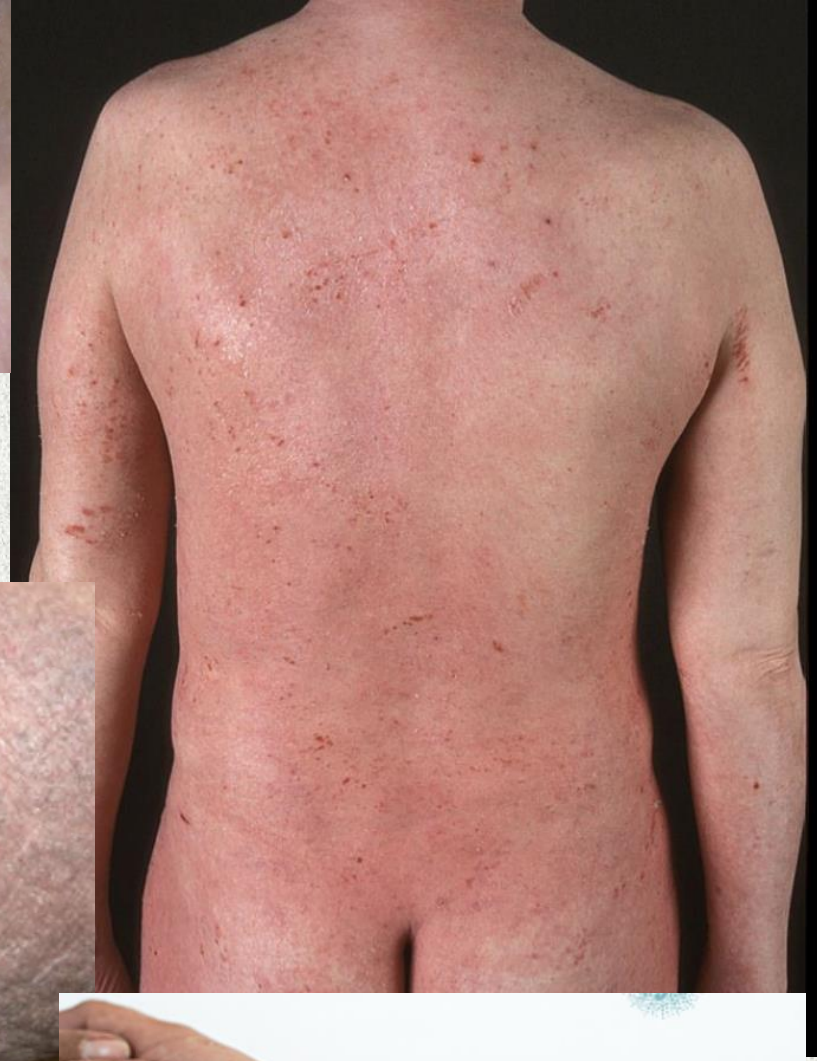
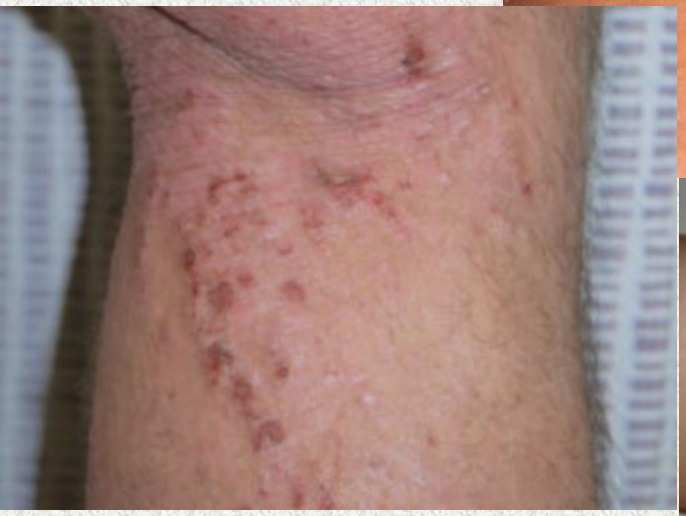
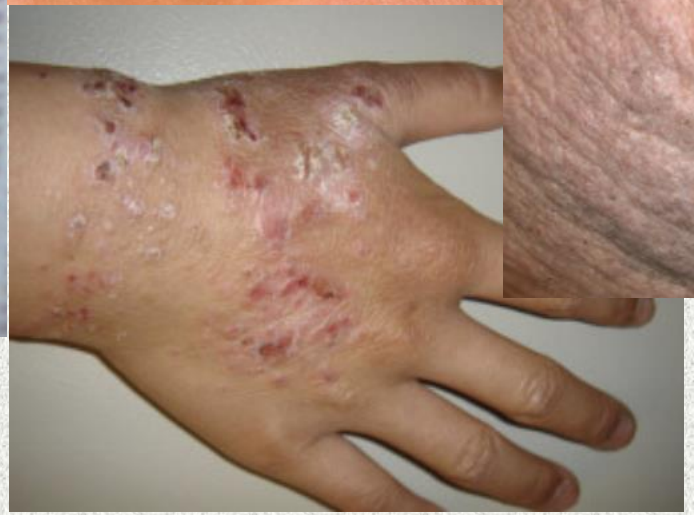
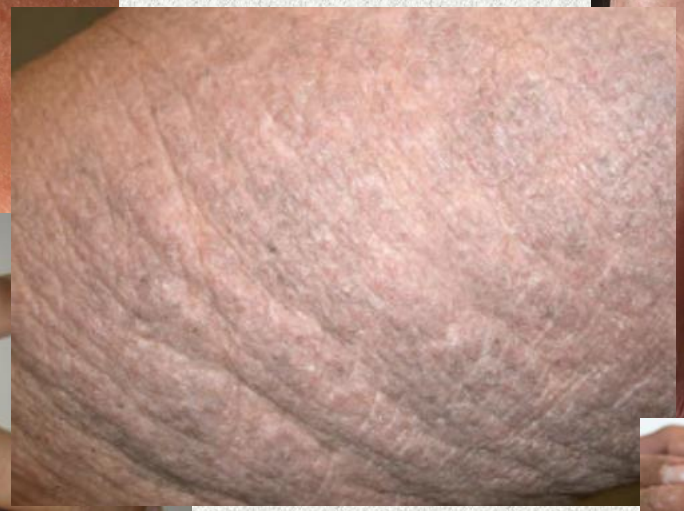
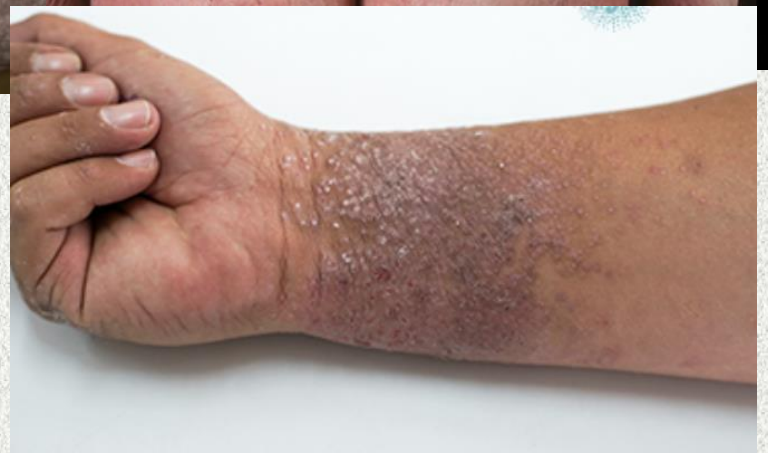
Contact dermatitis

Stasis dermatitis

Atopic Dermatitis

- Erythematous, scaly, sometimes excoriated or fissured, sometimes weeping or crusted, moderate-extremely pruritic plaques, borders can be poorly defined
- Flexural (antecubital fossa, popliteal fossa, flexor wrist, neck), periorbital, trunk, dorsal hands
  - Can be broader and more confluent
- Importance in hospitalized patients – in patients with history of AD, physiologic stressors such as illness, hospitalization, surgery, etc can cause flares while inpatient
- Management in hospitalized patient
  - Mild-moderate – mid-potent to potent topical steroids (eg. triamcinolone, clobetasol) BID, can occlude for faster results
  - Severe – can start with potent topical steroids (eg. clobetasol), but if fails, consider prednisone as long as it won't be detrimental to underlying reason for hospitalization or other comorbidities
  - Monitor for impetiginization (secondary staph or strep infection) – add Mupirocin or if needed, PO abx (eg. keflex, doxycycline usually effective)
  - Emollients frequently, if possible avoid potential irritants to skin (harsh soaps, adhesives over eczematous skin, etc)

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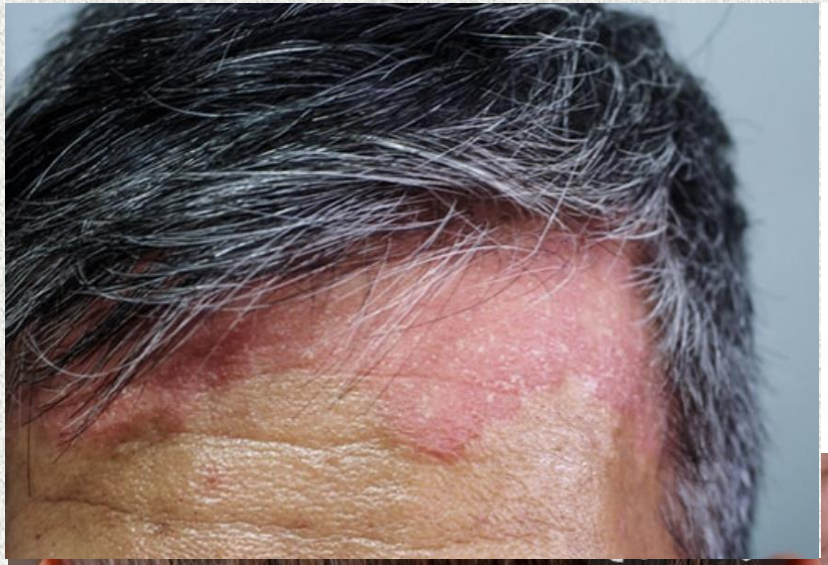
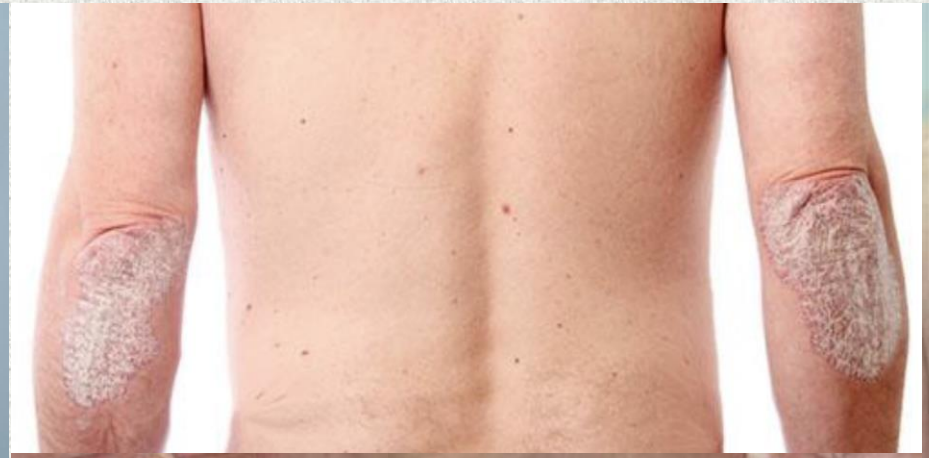
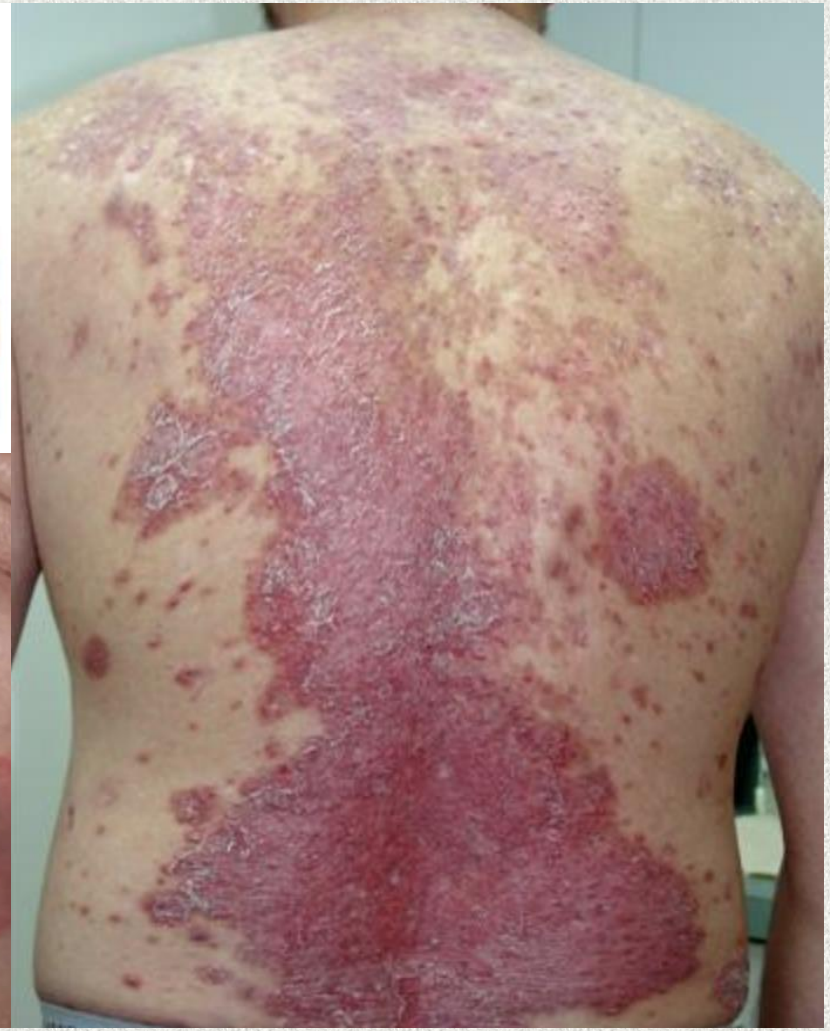
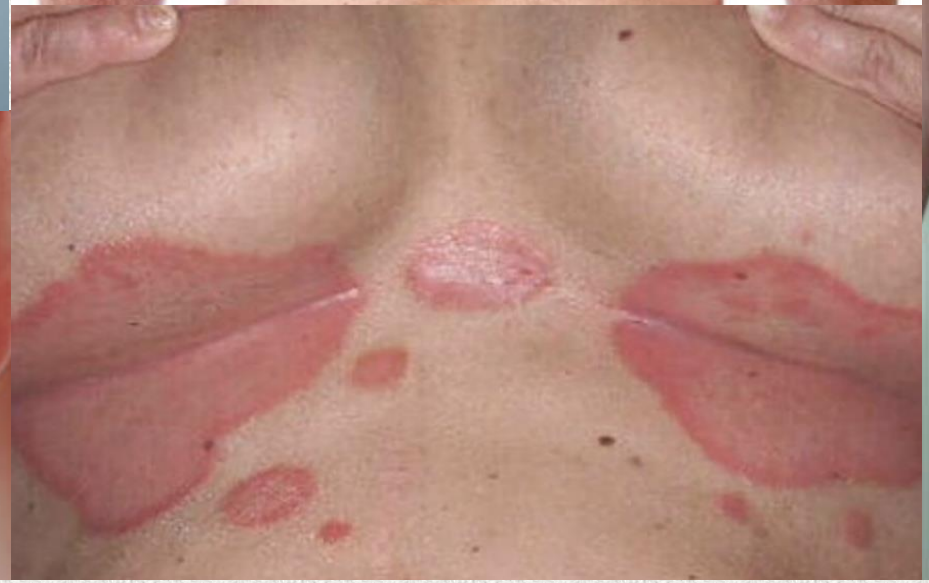
Psoriasis

- Well defined, pink plaques with thicker whitish scale, may fissure but less common than AD, typically less pruritic than AD
- Extensor (knees, elbows), intertriginous/inverse (inframammary, inguinal), trunk, scalp, ears
- Importance in hospitalized patients – in patients with history of psoriasis, physiologic stressors such as illness, hospitalization, surgery, etc can cause flares while inpatient
  - If systemic steroids are needed for any other reason, it is important to withdraw slowly as rapid cessation could result in flare of psoriasis
- Management in hospitalized patient
  - Low-mid potency topical steroids (hydrocortisone 2.5%, triamcinolone 0.025%) for face and genitals
  - Mid potency (triamcinolone 0.1%) for mild-moderate lesions
  - High potency (clobetasol 0.05%) for more severe lesions
  - Emollients frequently, if possible avoid potential irritants to skin (harsh soaps, adhesives over eczematous skin, etc)









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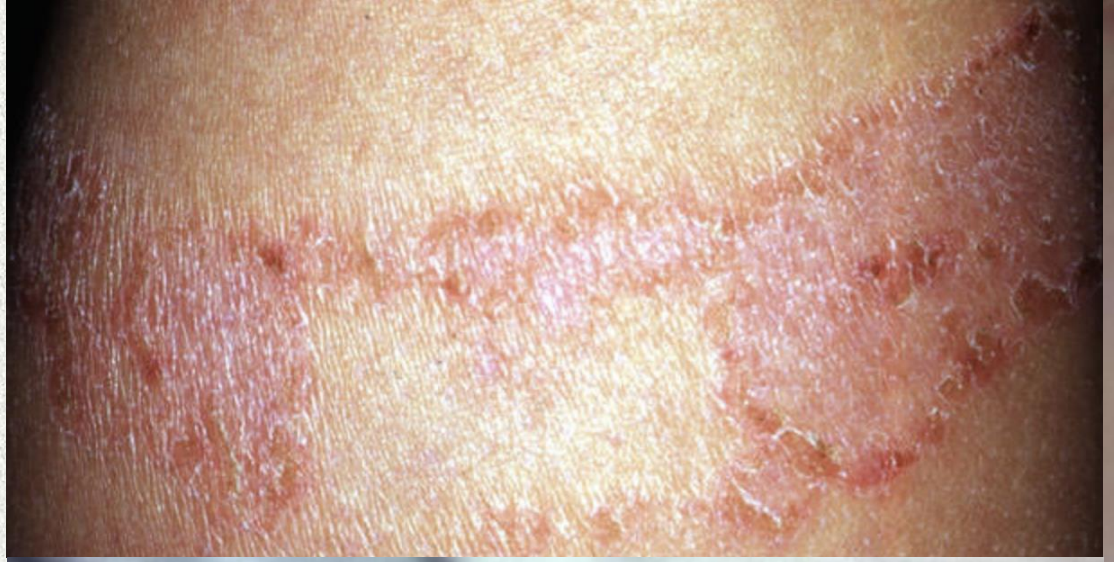
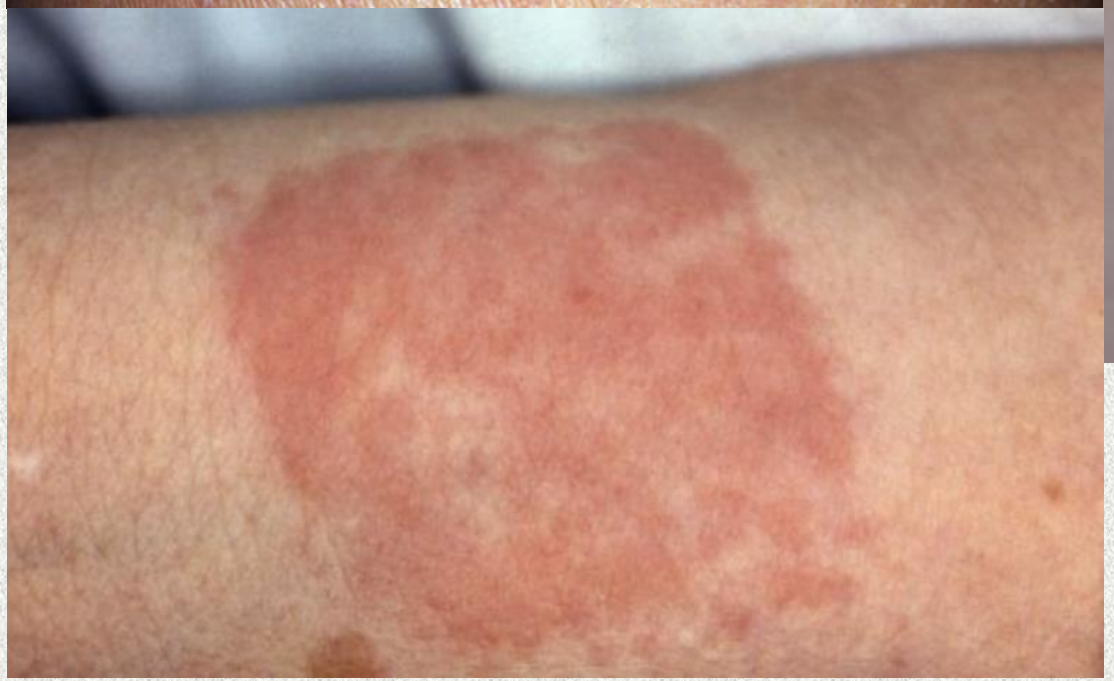
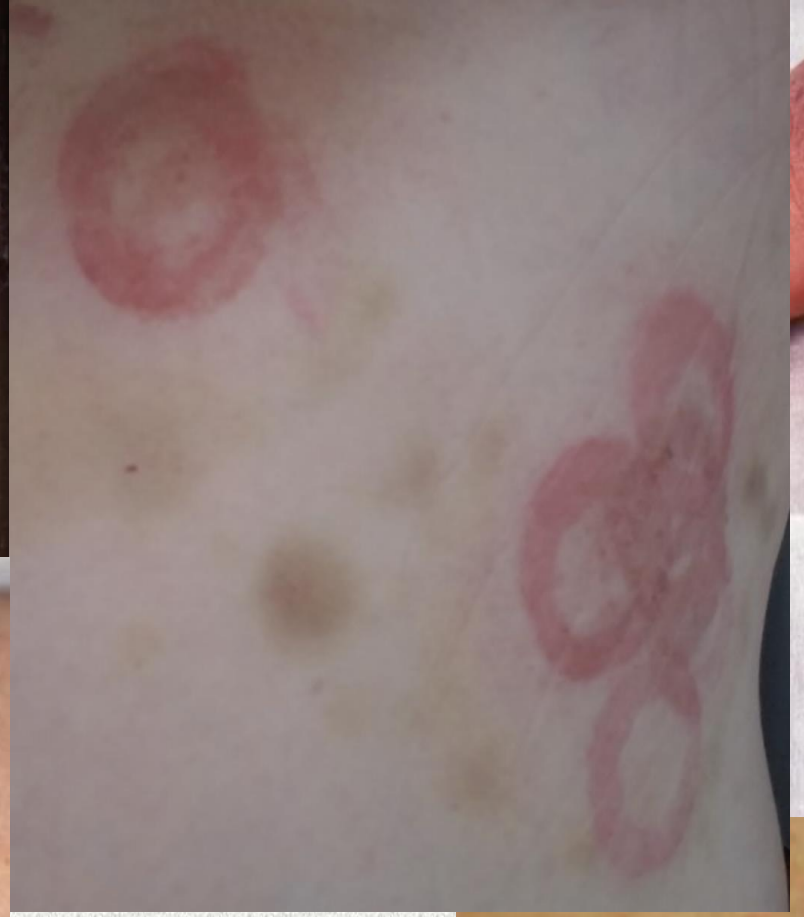
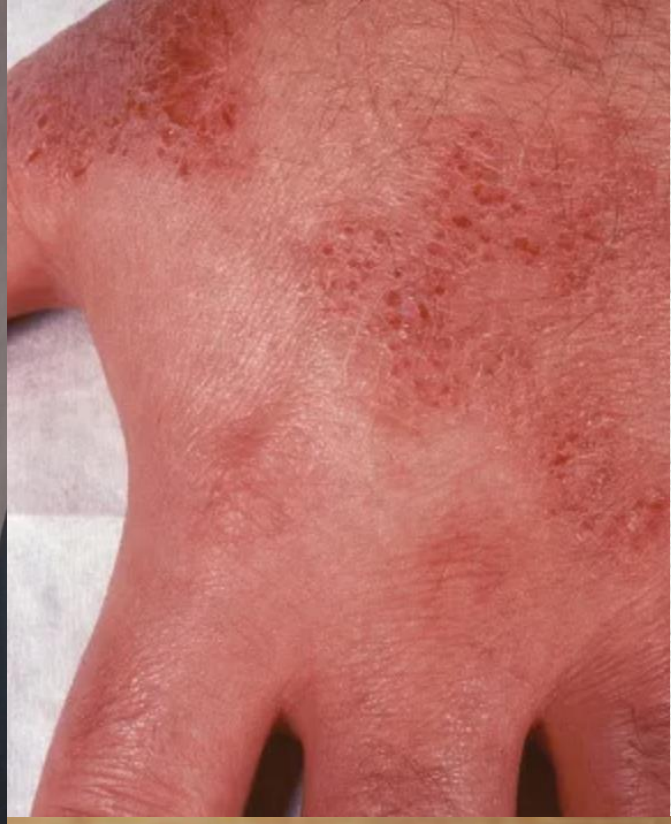
Contact Dermatitis

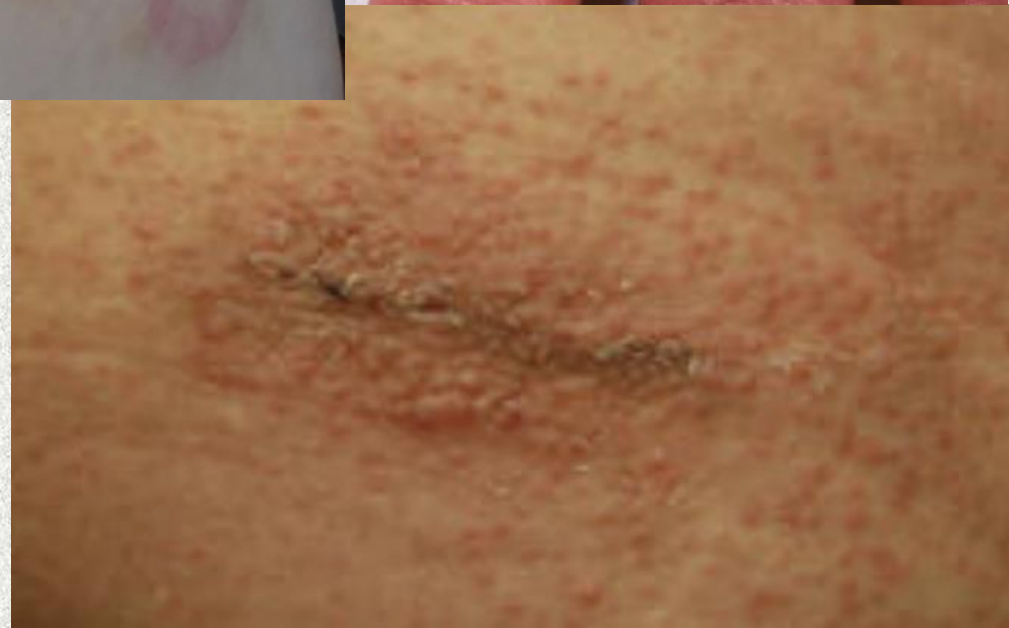
- Well defined geometric pattern, erythematous, scaly plaques, may have vesicles, marked pruritis
  - Geometric pattern – looks like an “outside job” (follows pattern of something that was put on the skin)
- Irritant or allergic
  - Irritant – non-allergic, exposure to anyone’s skin will start to cause breakdown and inflammation
  - Allergic – type IV (delayed) hypersensitivity reaction
- Causes – topical antibiotics, antiseptics, anesthetics, rubber, cleansers, fragrances in lotions, adhesives in bandages, skin glue
  - Latex and iodine cause a type I (immediate) hypersensitivity reaction resulting in urticaria
- Management in hospitalized patient
  - Remove and avoid suspected triggering agent
  - Mild-moderate – mid-potent to potent topical steroids (eg. triamcinolone, clobetasol) BID, can occlude for faster results
  - Severe – can start with potent topical steroids (eg. clobetasol), but if fails, consider prednisone as long as it won’t be detrimental to underlying reason for hospitalization or other comorbidities











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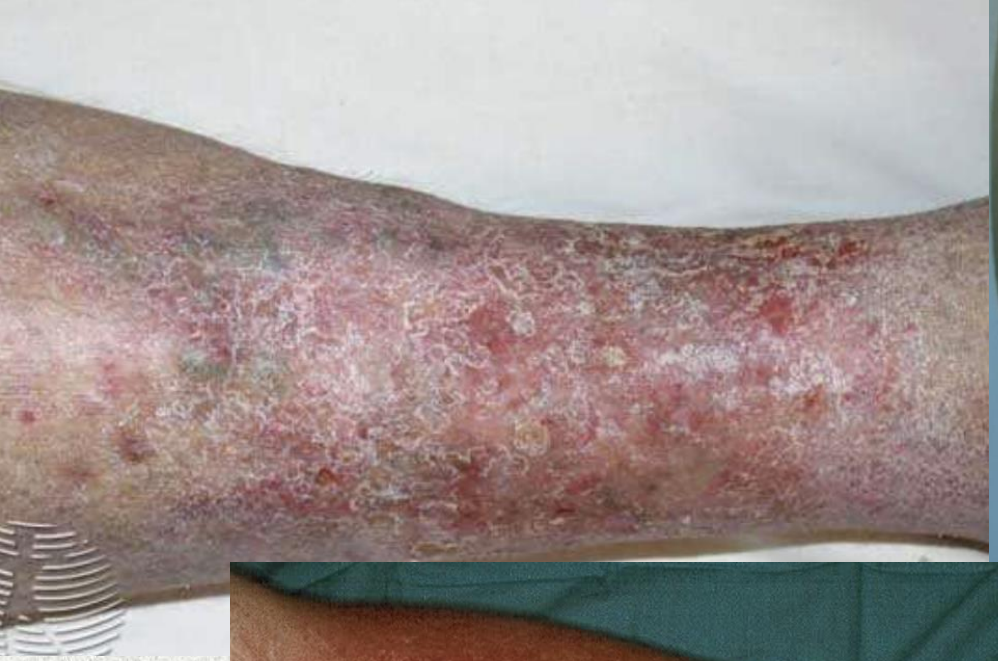
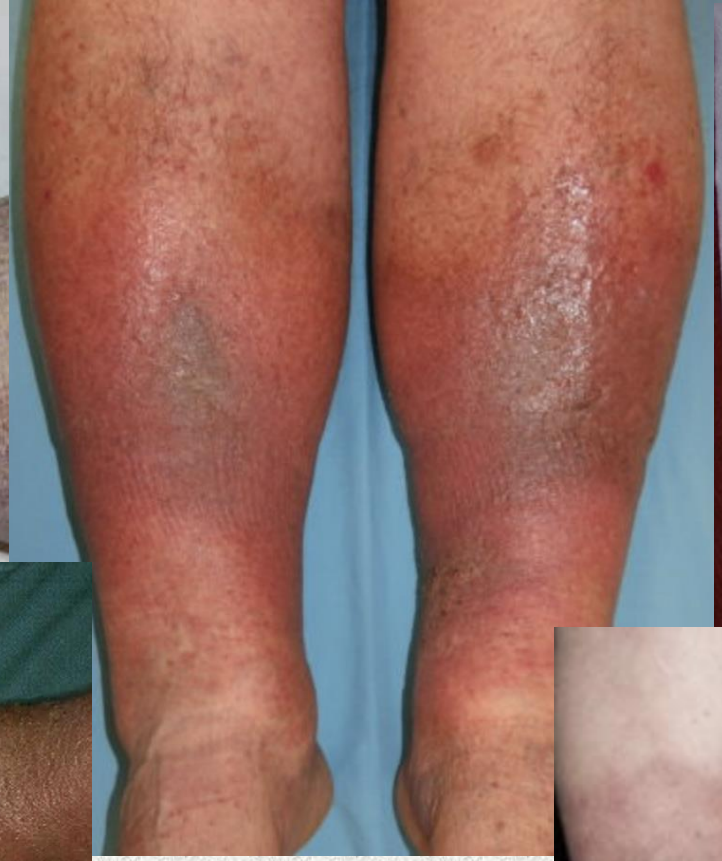
Stasis Dermatitis

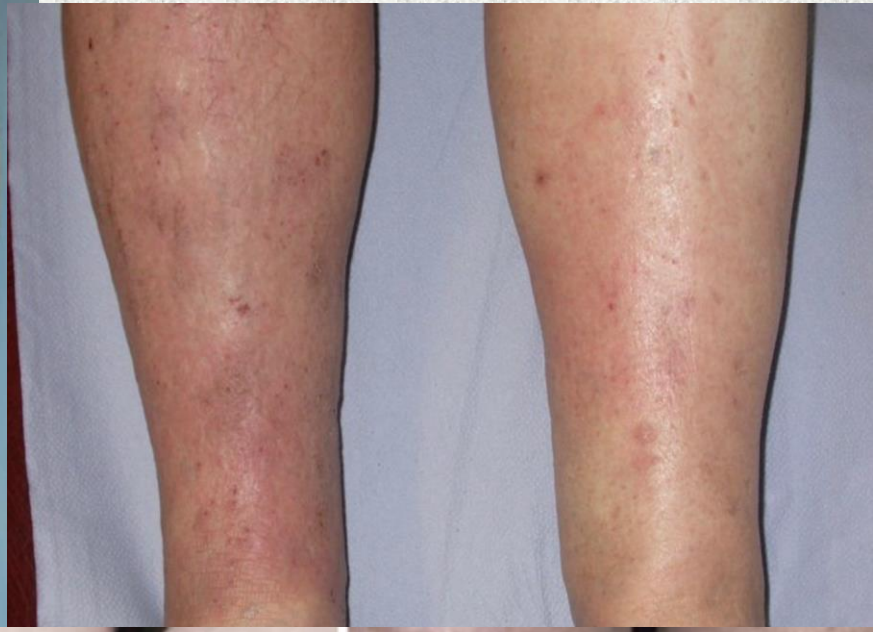
- Etiology – venous insufficiency and reflux → venous hypertension → leakage of fluid into tissue → edema → tensile stress on skin → breakdown and inflammation of skin
- Typically bilateral lower legs, but can be worse on one side
  - *If it is bilateral, it is much less likely to be bilateral cellulitis and much more likely to be stasis dermatitis. If you think cellulitis, ok to start antibiotics but also consider adding topical steroids.
- Risk factors – advanced age, hx DVT, hx cellulitis in lower leg, chronic LEE, varicose veins
- Presentation – pruritic erythematous scaly confluent plaques circumferentially around distal-mid lower legs, may have vesiculation and bulla, may have fissuring and ulceration, a/w LEE, a/w hemosiderin deposition
  - May have atrophie blanche – white irregular scars
  - May have lipodermatosclerosis – tense sclerotic skin of distal lower leg creating an inverted wine bottle appearance

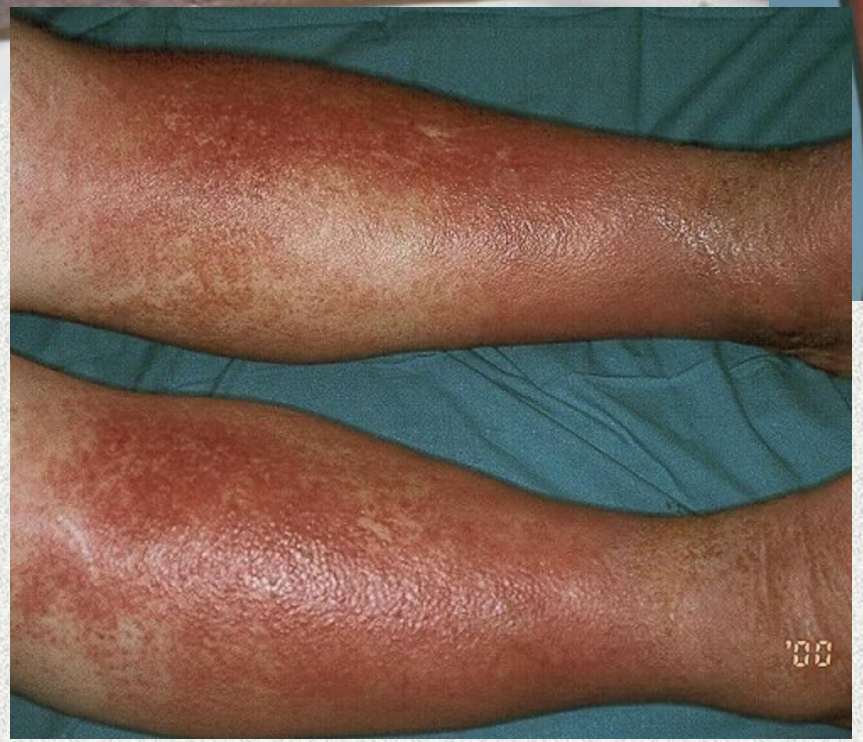
Stasis Dermatitis – Management in Hospitalized Patient

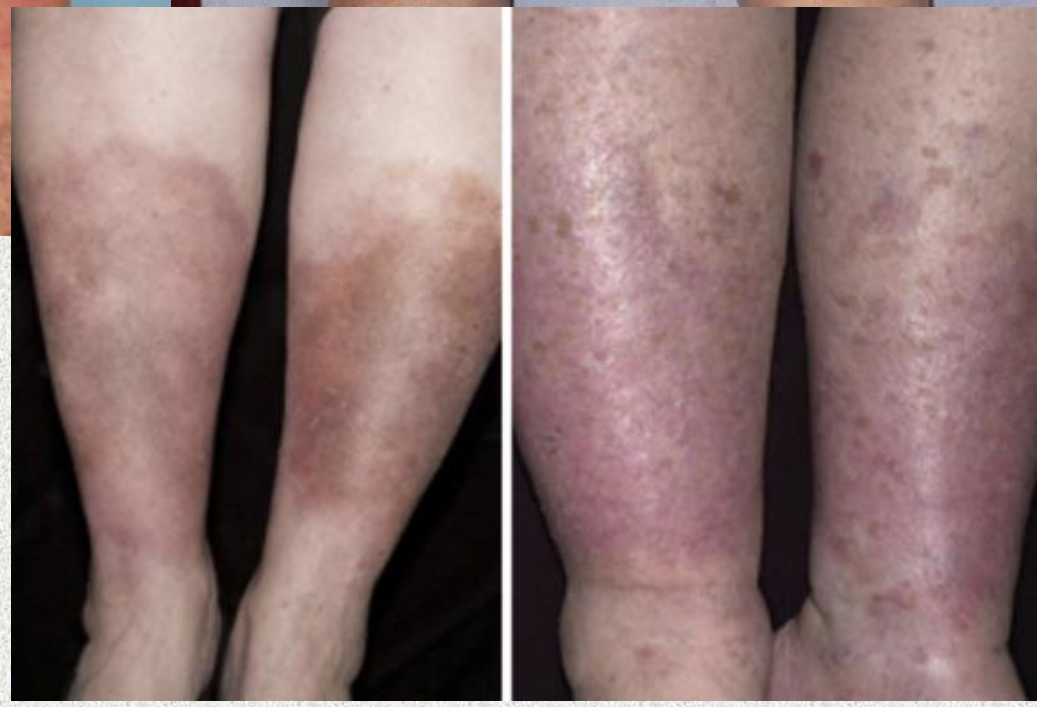
- Mid-potent to potent topical steroids (eg. triamcinolone, clobetasol) BID, can occlude for faster results
- Compression stockings/wraps significantly important to remove fluid and decrease compressive and stretching forces on skin
- Monitor for impetiginization and cellulitis – add PO abx (eg. keflex, doxycycline)
- Emollients, if possible avoid potential irritants to skin (harsh soaps, adhesives over eczematous skin, etc)











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Vascular

Vasculitis

Disseminated Intravascular Coagulation (DIC)

Calciphylaxis

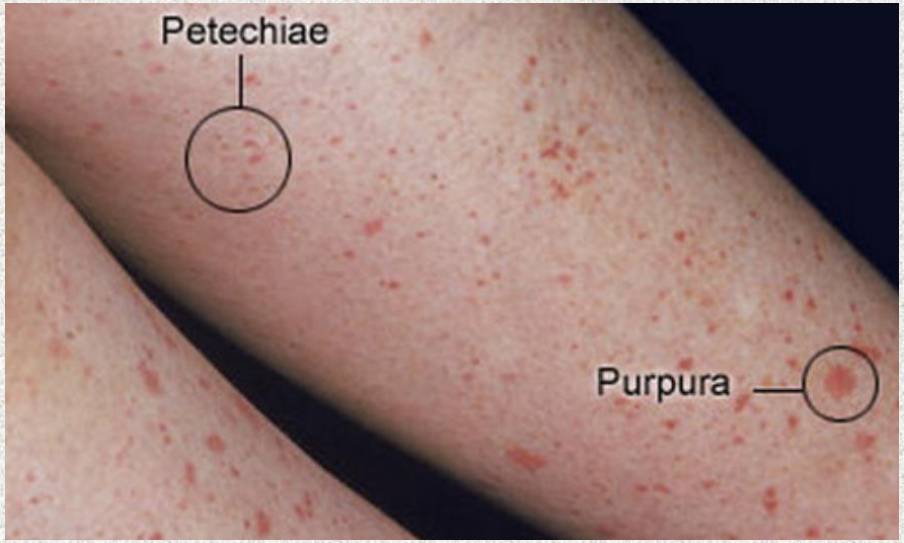
Vasculitis

- Clinicopathologic process characterized by inflammation and necrosis of blood vessels
- Palpable purpura may be the presenting manifestation of a systemic vasculitic process and should trigger systemic workup to both determine etiology and monitor for end-organ sequale
- Classified by size of blood vessel affected
  - Small-vessel vasculitis – affects postcapillary venules → urticarial lesions and palpable purpura
  - Small artery vasculitis – affects small arteries → subcutaneous nodules
  - Medium-sized artery vasculitis – affects medium-sized arteries → levido, purpura, necrosis of major organs, and mononeuritis multiplex
  - Large-vessel vasculitis – affects large vessels → necrosis and claudication

Vasculitis – Definitions

Petechia vs Purpura vs Ecchymoses

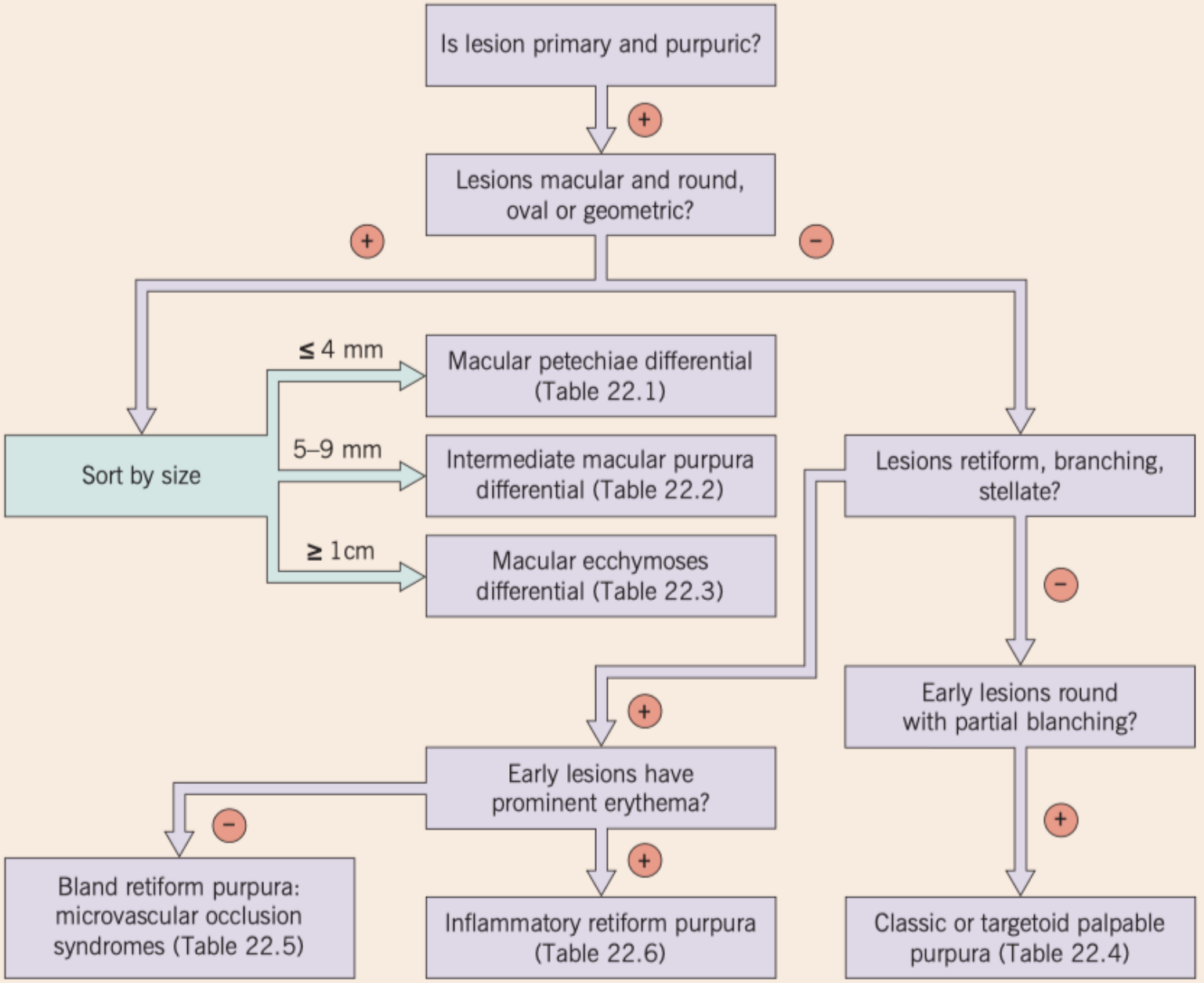
- All are caused by leakage of RBCs from vessels into skin
- Petechia – 4mm or smaller
- Purpura – 5-9mm
- Ecchymoses – 1cm or larger

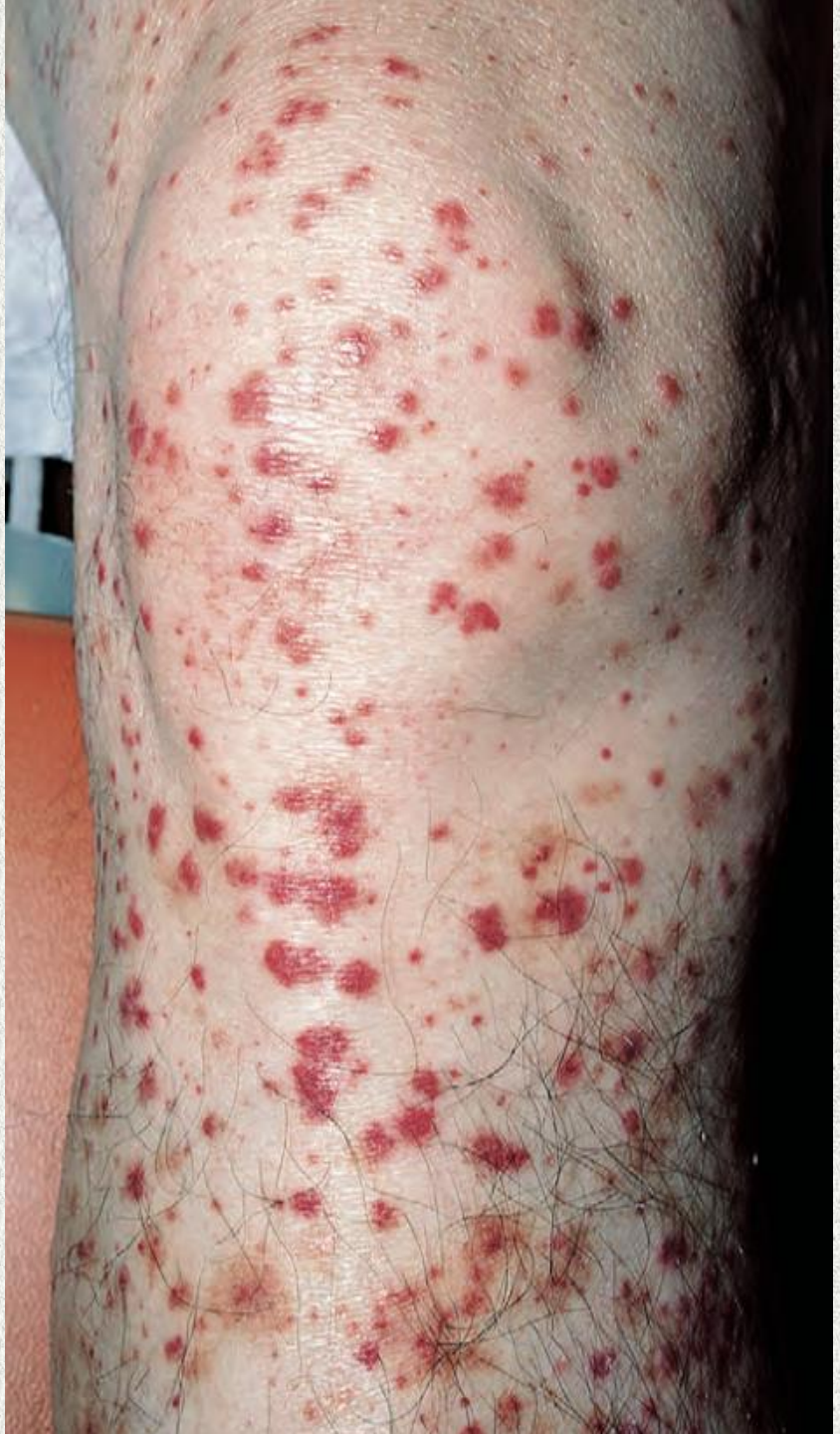


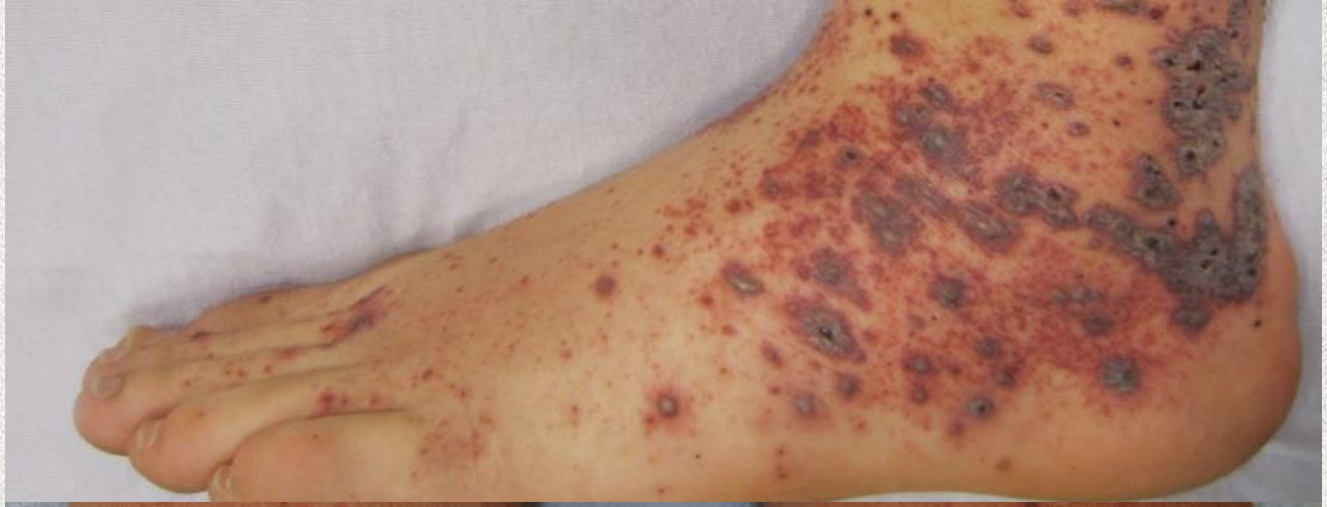
Palpable vs Non-Palpable Purpura

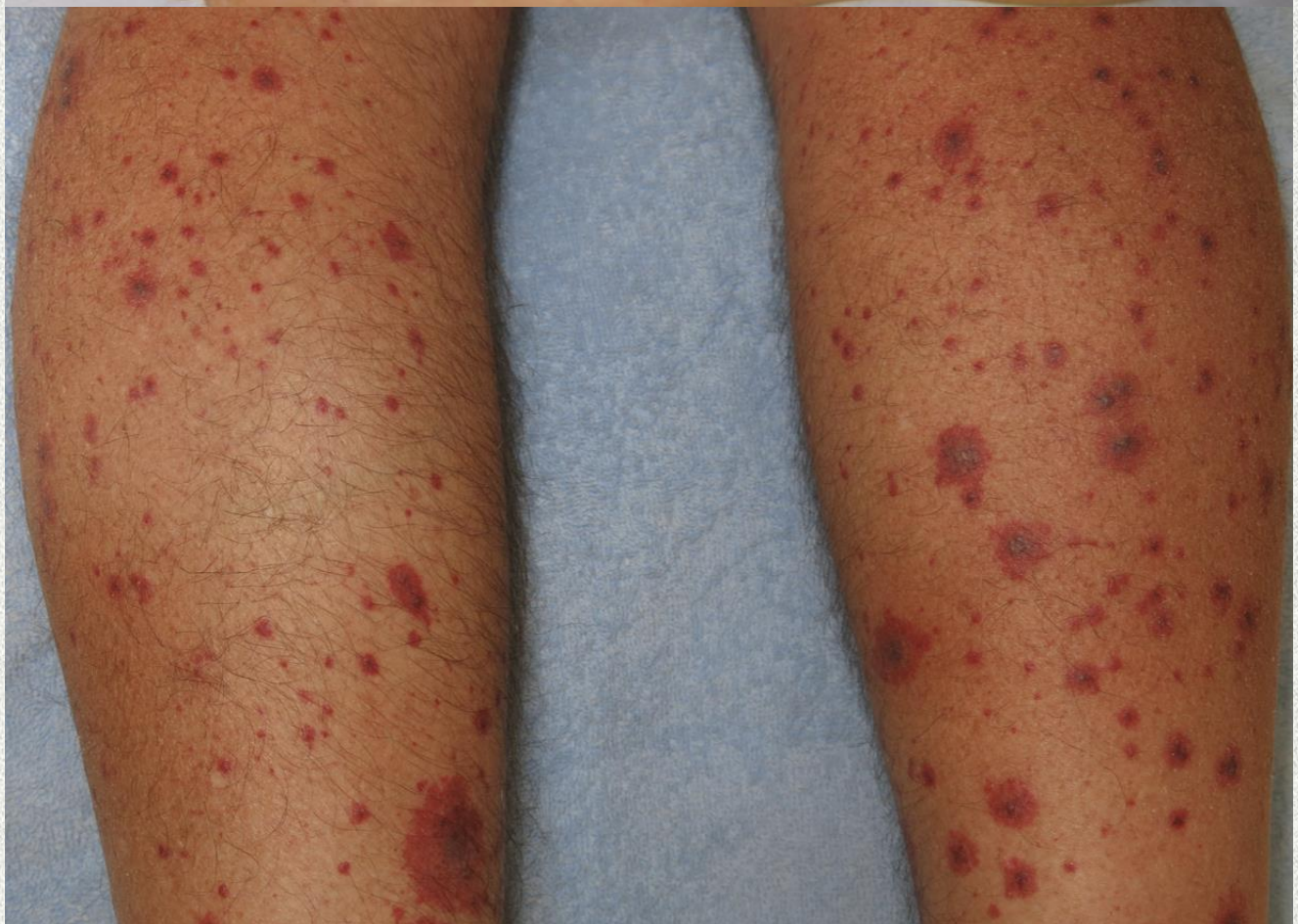
- Because purpura are caused by leakage of blood cells into the skin, the blood cells cannot be "compressed" back into the vessels → non-blanchable
- Purpura that are papular are palpable
  - Elevation of lesion is a result of inflammation
  - Inflammation = vasculitis = palpable purpura
- Purpura that are macular are non-palpable
  - Lack of elevation indicates no underlying inflammation
  - No inflammation = vasculopathy = non-palpable purpura

DIFFERENTIAL DIAGNOSIS OF PURPURA









Classifications of Vasculitis

Cutaneous Small-Vessel (postcapillary venule)

- Idiopathic cutaneous small-vessel vasculitis
- Henoch-Schonlein purpura
- Urticarial vasculitis
- Cryoglobulinemic vasculitis
- Other – drug-induced vasculitis, malignancy (lymphoreticular>solid tumor), AICTD, hyperglobulinemic purpura, IBD, bowel-associated dermatitis-arthritis syndrome, HIV, neutrophilic dermatosis (Behcet, Sweet, septic vasculitis, serum sickness, autoinflammatory, familial Mediterranean fever, erythema nodosum leprosum)

Classifications of Vasculitis

Medium-Vessel

- Polyarteritis nodosa
  - Benign cutaneous variant
  - Systemic variant

Large-Vessel

- Giant-cell arteritis
- Takayasu arteritis

Mixed-Size (Medium and Small) Vessel Disease

- Connective tissue disease associated (usually rheumatoid vasculitis)
- Septic vasculitis
- ANCA-associated
  - Microscopic polyangiitis
  - Wegener granulomatosis
  - Allergic granulomatosis (Churg-Strauss)
  - Occasional drug-induced

Systemic Vasculitis

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The systemic vasculitides are characterized by inflammation of blood vessel walls. Vessels of any type, in any organ can be affected, resulting in a broad spectrum of signs and symptoms. The heterogenous nature of vasculitides presents a diagnostic challenge. The American College of Rheumatology classification criteria and the Chapel Hill Consensus Conference nomenclature are the most widely used to distinguish different forms of vasculitis. The Chapel Hill Consensus Conference nomenclature defines 10 primary vasculitides based on vessel size (large, medium, and small). The diagnosis relies on the recognition of a compatible clinical presentation supported by specific laboratory or imaging tests and confirmatory histology. Antineutrophilic cytoplasmic antibody testing has been of particular benefit in defining a subgroup of small vessel vasculitides. Treatment is based on clinical presentation and the pattern of organ involvement. Glucocorticoids are the primary treatment for many forms of vasculitis. Additional immunosuppressive agents, including methotrexate and cyclophosphamide, are sometimes required. Newer approaches, such as the use of anti-tumor necrosis factor or B cell therapies, are being tried in resistant cases. Patients can experience considerable treatment-related toxicity, especially infection from immunosuppressive therapy and adverse effects from steroids (e.g., osteoporosis, diabetes mellitus, cataract). Vitamin D and calcium prophylaxis are recommended in patients on long-term steroid therapy. (*Am Fam Physician.* 2011;83(5):556-565. Copyright © 2011 American Academy of Family Physicians.)

Extent of subtypes, etiologies, work-up and management for systemic vasculitides is beyond the scope of this presentation. The above article offers a comprehensive review of classification, pathogenesis, features, diagnostic approach, and treatment. *Am Fam Physician.* 2011;83(5):556-565

Table 1. Classification of Primary Systemic Vasculitis (Chapel Hill Consensus Conference Nomenclature)

Vasculitis	*Description*
Small vessel	
Churg-Strauss syndrome	Eosinophil-rich and granulomatous inflammation involving the respiratory tract; necrotizing vasculitis of small to medium vessels; associated with asthma
Cutaneous leukocytoclastic angiitis	Isolated cutaneous leukocytoclastic angiitis without systemic vasculitis or glomerulonephritis
Essential cryoglobulinemic vasculitis	Vasculitis, with cryoglobulin immune deposits, affecting capillaries, venules, or arterioles; associated with serum cryoglobulins; skin and glomeruli are often involved
Henoch-Schönlein purpura	Immunoglobulin A–dominant immune deposits, affecting capillaries, venules, or arterioles; typically involves skin, gut, and glomeruli; associated with arthralgias or arthritis
Microscopic polyangiitis	Necrotizing vasculitis, with few or no immune deposits, affecting capillaries, venules, or arterioles, but may involve small and medium arteries; necrotizing glomerulonephritis is very common; pulmonary capillaritis often occurs
Wegener granulomatosis	Granulomatous inflammation involving the respiratory tract, and necrotizing vasculitis affecting capillaries, venules, arterioles, and arteries; necrotizing glomerulonephritis is common
Medium vessel	
Kawasaki disease	Arteritis involving coronary arteries, but aorta and veins may be involved; associated with mucocutaneous lymph node syndrome
Polyarteritis nodosa	Necrotizing inflammation of medium or small arteries without glomerulonephritis or vasculitis in arterioles, capillaries, or venules
Large vessel	
Giant cell (temporal) arteritis	Granulomatous arteritis of the aorta and its major branches, with a predilection for the extracranial branches of the carotid artery; often involves the temporal artery; associated with polymyalgia rheumatica
Takayasu arteritis	Granulomatous inflammation of the aorta and its major branches

Adpated with permission from Jennette JC, Falk RJ, Andrassy K, et al. Nomenclature of systemic vasculitides. Proposal of an international consensus conference. Arthritis Rheum. *1994;37(2):189.*

https://www.aafp.org/pubs/afp/issues/2011/0301/p556.html

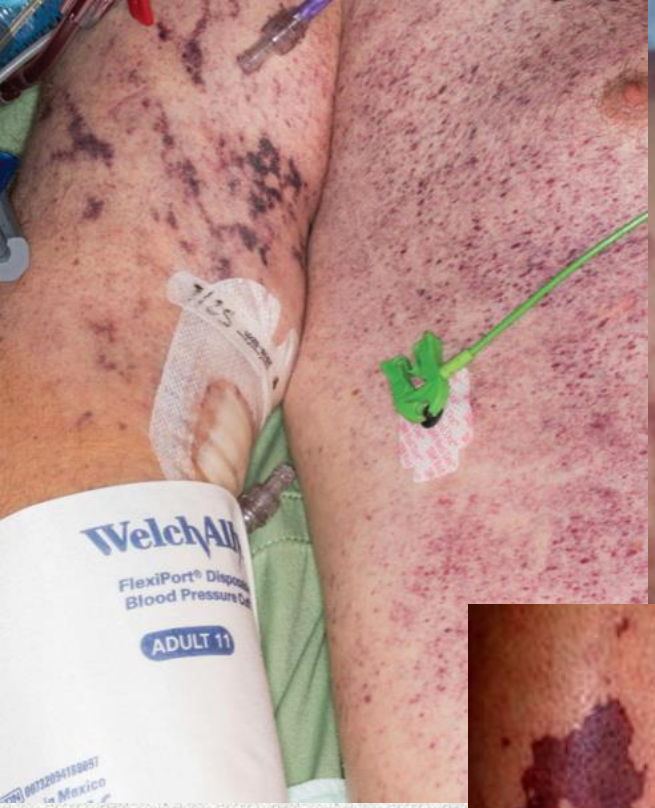
Table 2. Clinical Features of Major Systemic Vasculitides

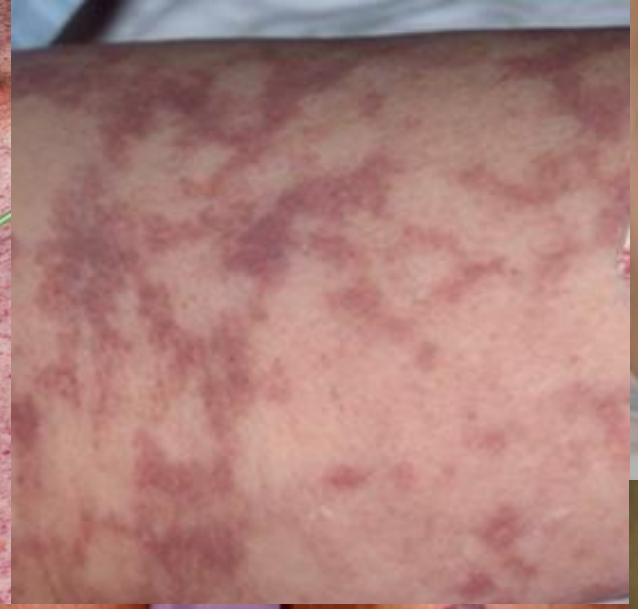
Vasculitis	*Organ involvement*	*Age (years)*	*Clinical features*
Small vessel			
Churg-Strauss syndrome	Respiratory tract, heart	50 to 60	Allergic rhinitis, asthma, peripheral eosinophilia
Cryoglobulinemic vasculitis	Skin, kidney	40 to 50	Recurrent palpable purpura, polyarthralgia, glomerulonephritis
Cutaneous leuko-cytoclastic angiitis	Skin	Any age	Palpable purpura, cutaneous infarcts, necrotic papules, urticaria
Henoch-Schönlein purpura	Skin, gastrointestinal tract, kidney, joint	3 to 8	Purpura, arthritis, abdominal pain, gastrointestinal bleeding, glomerulonephritis
Microscopic polyangiitis	Skin, lung, heart, kidney, liver, gastrointestinal tract	50 to 60	Palpable purpura, pulmonary hemorrhage, glomerulonephritis
Wegener granulomatosis	Upper and lower respiratory tracts, kidney	40 to 50	Pneumonitis with bilateral nodular and cavitary infiltrates, mucosal ulceration of nasopharynx, chronic sinusitis, glomerulonephritis
Medium vessel			
Kawasaki disease	Coronary arteries, aorta and its branches	2 to 4	Fever, conjunctivitis, desquamating skin rash, enlarged cervical lymph nodes
Polyarteritis nodosa	Renal and visceral organs, spares lung	30 to 40	Fever, weight loss, hypertension, abdominal pain, melena, peripheral neuritis, renal ischemia
Large vessel			
Giant cell arteritis	Extracranial branches of carotid artery, often involves temporal artery	50 to 60	Fever, visual disturbances, facial pain and headache (often along the course of superficial temporal artery)
Takayasu arteritis	Aorta and its major branches	30 to 40	More common in young Asian women Markedly lower blood pressure and weaker pulse in upper extremities, with coldness and numbness of fingers, visual disturbances, hypertension, neurologic deficit

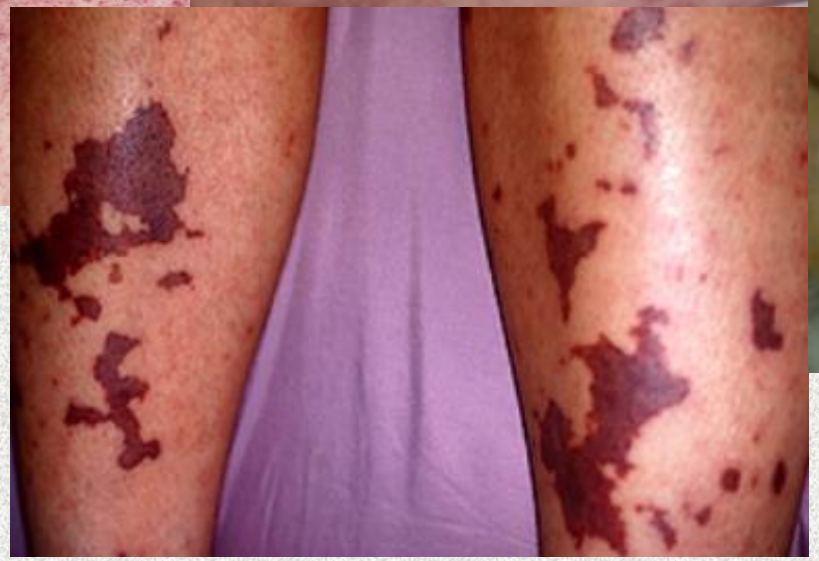
https://www.aafp.org/pubs/afp/issues/2011/0301/p556.html

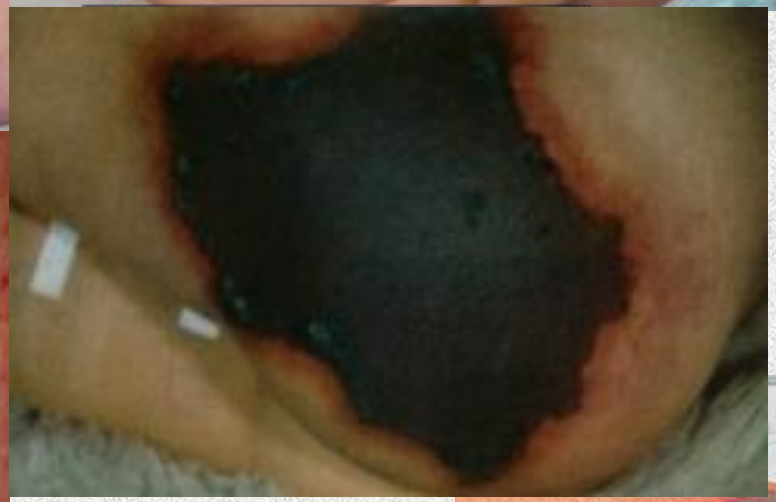
Disseminated Intravascular Coagulation (DIC)

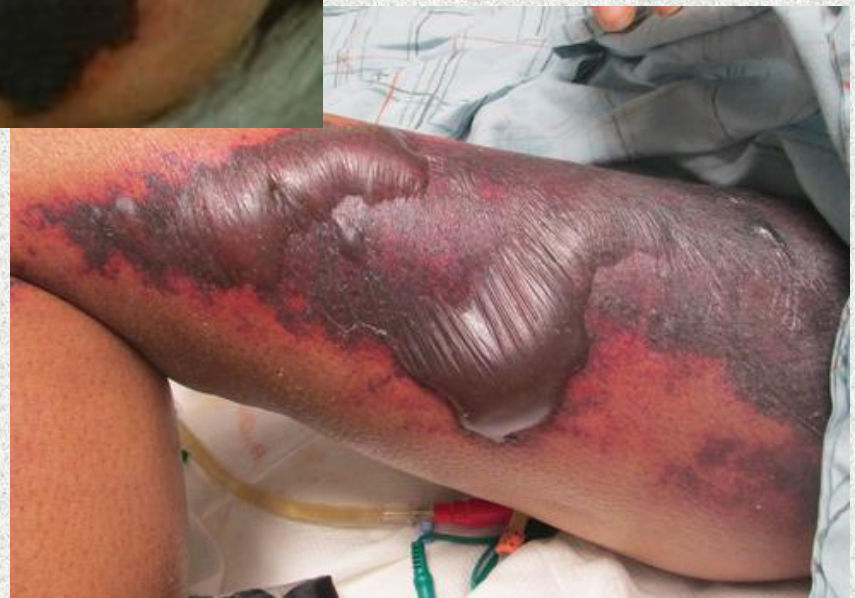
- Etiology – uncontrolled activation of clotting factors in vessels causing clotting throughout body → depletes platelets and coagulation factors resulting in hemorrhage
  - Imbalance between thrombin (clotting) and plasmin (clot lysing)
  - Acute and chronic variants – this presentation will focus on acute DIC in the hospitalized patient
  - Purpura fulminans – severe and rapidly fatal form
- Causes – infection (sepsis, meningococcemia, RMSF, viremia), complicated delivery (placental abruption, amniotic embolism, eclampsia), crush injury, TBI, renal transplant rejection, heat stroke, snakebites
- Presentation – high fever, severe malaise, purpura and ecchymoses, oral and genital mucosal bleeding, hemorrhagic bulla, progresses to foci of cutaneous ischemia and necrosis, symptoms of internal organ hemorrhage
- Treatment
  - No hemorrhage/ischemia – observation only
  - With hemorrhage – FFP, cryoprecipitate, PLT transfusion
  - With ischemia – heparin, LMWH (only after hemorrhage is corrected)











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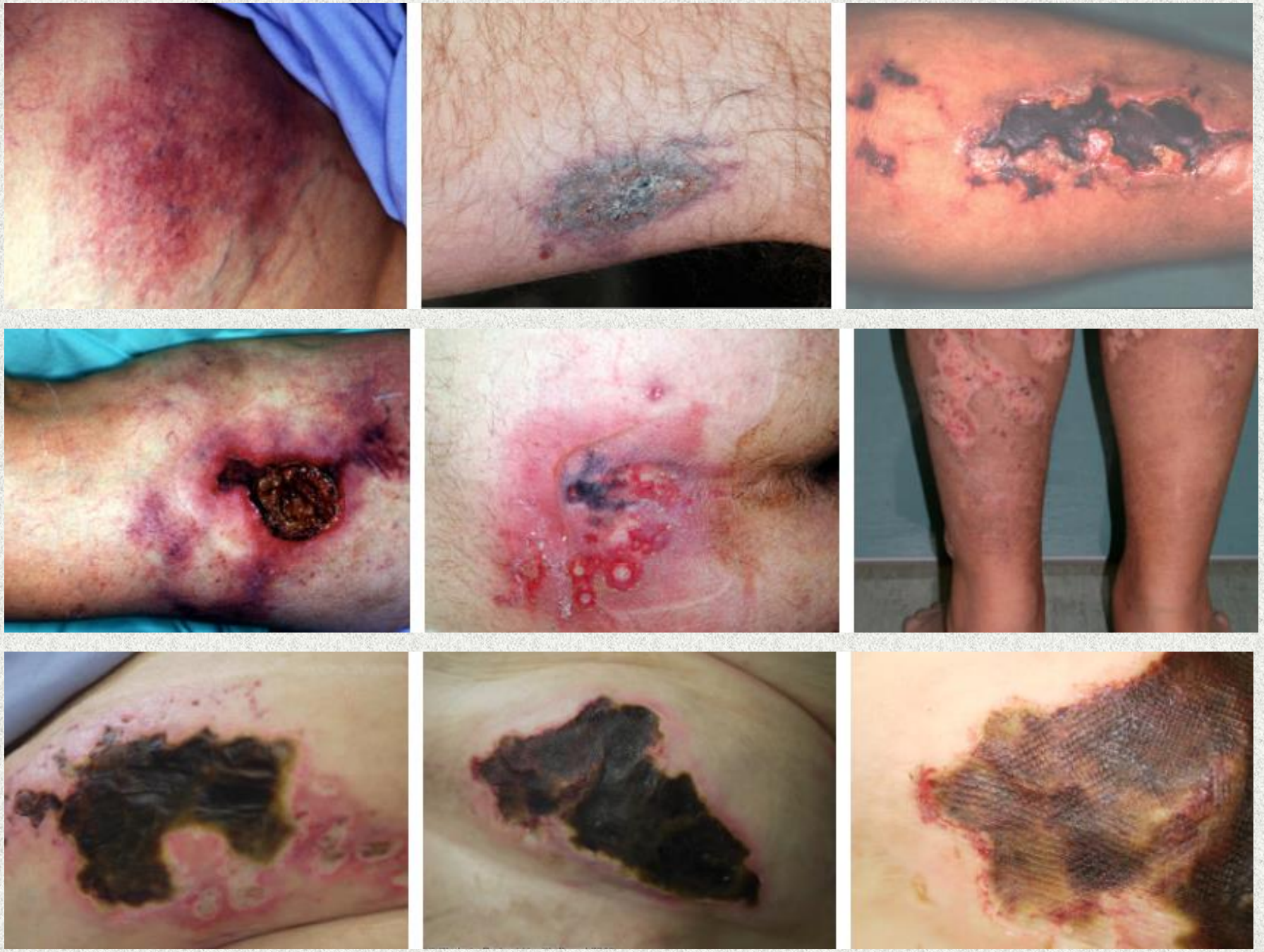
Calciphylaxis

- AKA calcific uremic arteriolopathy or calcific vasculopathy
- Etiology – vascular wall calcification leads to thrombosis leading to spreading ischemia and necrosis
  - CKD – decreased renal function → increased phosphate → binds with calcium → vit D reduced d/t renal failure and decreased GI absorption → bones resistant to parathyroid hormone → secondary hyperparathyroidism → increased circulating calcium
  - Possible to occur in pts with high or normal calcium or phos, with or w/o vit D replacement, with or without dialysis or after renal transplant
- Risk factors – CKD (MC population), primary hyperparathyroidism, malignancy, alcoholic hepatic failure, DM, coumadin, immunosuppressants, corticosteroids
- Presentation – retiform purpura → hemorrhage into skin, hemorrhagic bulla → skin necrosis → stellate eschar with dry gangrene and deep extensive ulceration
  - Associated with severe pain
  - Extremities most common location
  - Fattier locations (trunk, abdomen, buttocks, thighs) have higher morbidity and mortality

Calciphylaxis – Management in Hospitalized Patient

- Complications – infection with sepsis, death
  - Mortality – 60-80% in CKD, closer to 15% in coumadin-associated
- Diagnosis – deep wedge biopsy (calcium deposits in subQ vessels), xray (vascular calcification in skin), bone scintigraphy w/ technetium (radiotracer uptake in soft tissue)
- Treatment
  - IV sodium thiosulfate to chelate calcium
  - Dialysis with lower dialysate calcium concentration
  - Calcium and phosphate restricted diet
  - Surgical debridement of necrotic tissue (caution d/t risk of exacerbating calciphylaxis) or amputation if extensive
  - Parathyroidectomy or cinacalcet – only if hyperparathyroidism
  - Switch to heparin if on coumadin
  - Antibiotics if infection
  - Possible benefit with doxycycline to inhibit matrix metalloproteinases



https://dermnetnz.org/topics/calciphylaxis

Ulcers

Venous insufficiency

Arterial insufficiency

Diabetic ulcers

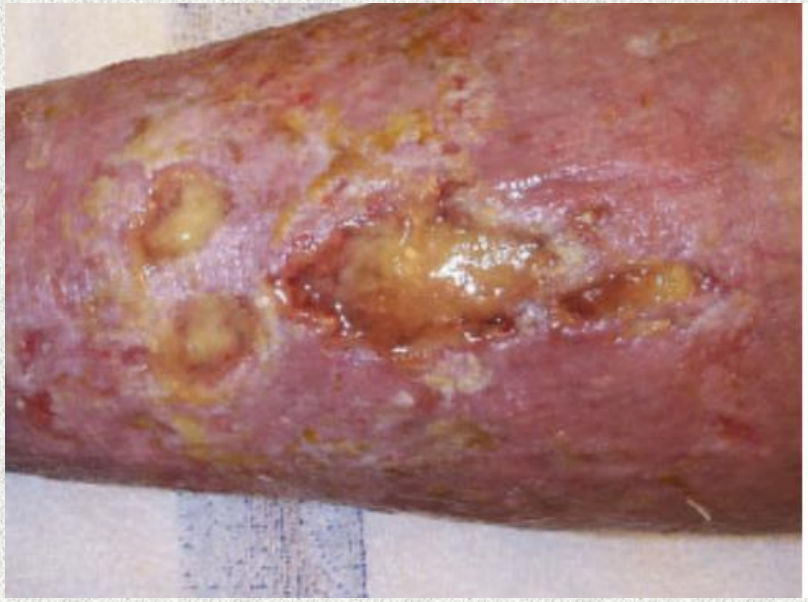
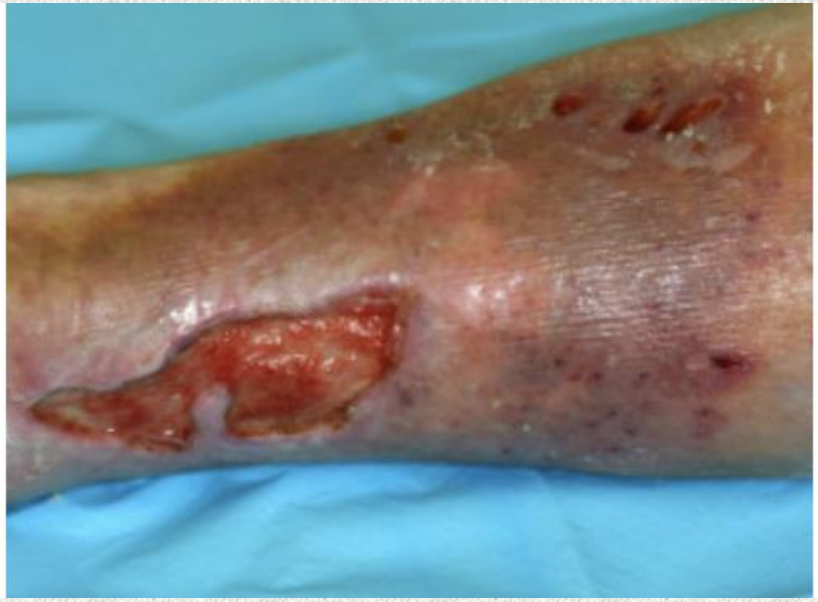
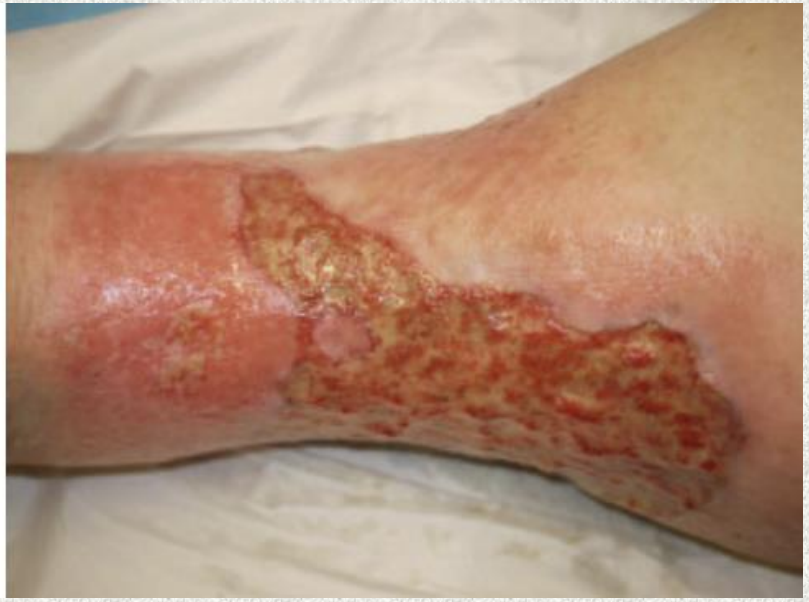
Pressure ulcers

Venous Insufficiency Ulcers

- Etiology – venous insufficiency and reflux → venous hypertension →
  - Leakage of fluid into tissue → edema → tensile stress on skin → inflammation of skin → breakdown of skin and ulceration
  - Vessel wall fibrosis → reduced O2 delivery to tissue and reduced waste removal from tissue → impaired healing
- Risk factors – obesity, HTN, varicose veins, multiparous, immobility, lower extremity injury/surgery/fracture, prior DVT
- Characteristics – *medial* lower leg, jagged and shallow, heavy exudate and weeping, minimal pain
  - Surrounding skin – LEE, varicose veins, hemosiderin deposition, stasis dermatitis, lipodermatosclerosis

Venous Insufficiency Ulcers – Management in Hospitalized Patient

- Workup – duplex US to confirm reflux and r/o thrombosis, ABI to r/o concomitant arterial disease
- Treatment
  - Rule out and treat for infection
  - Wound care with specialized dressings (alginates, foams), chemical debridement – consider Wound Care consultation
  - Physical (surgical) debridement – consider Gen Surg consultation
  - Topical steroids for surrounding stasis dermatitis
  - Compression wraps/socks
  - Other – ASA, hyperbaric O2, pentoxifylline

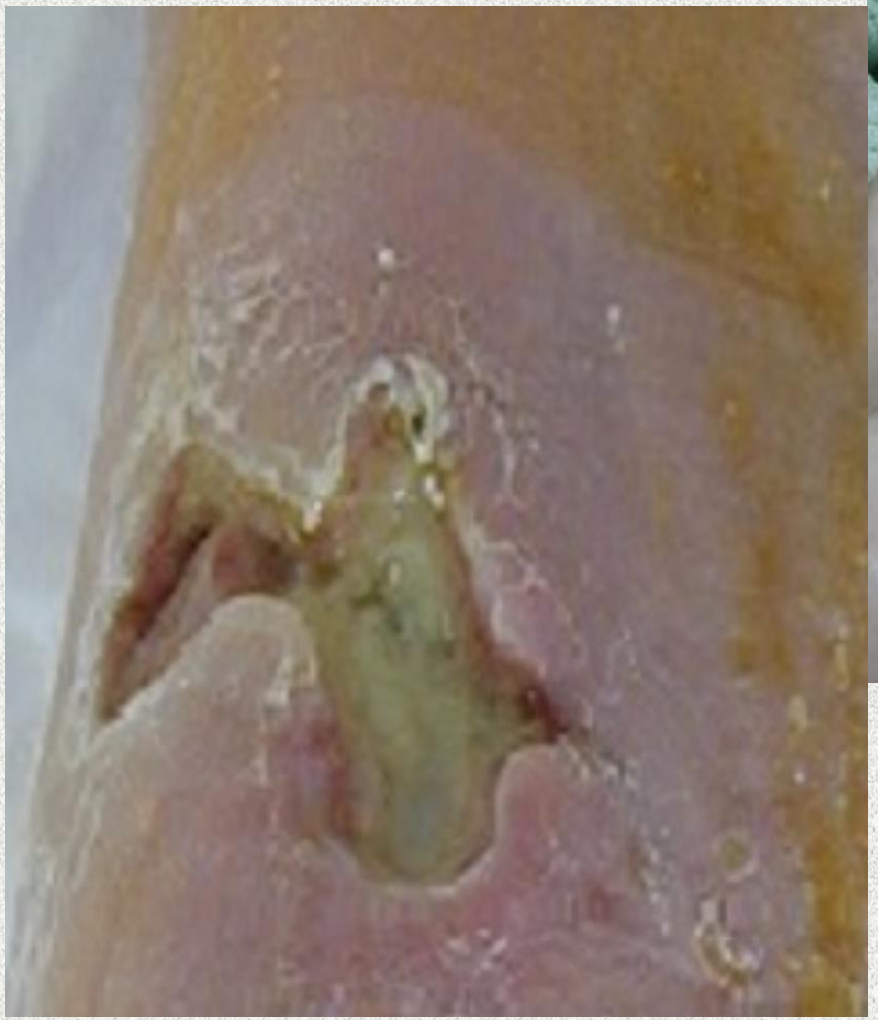
https://dermnetnz.org/topics/stasis-ulcer

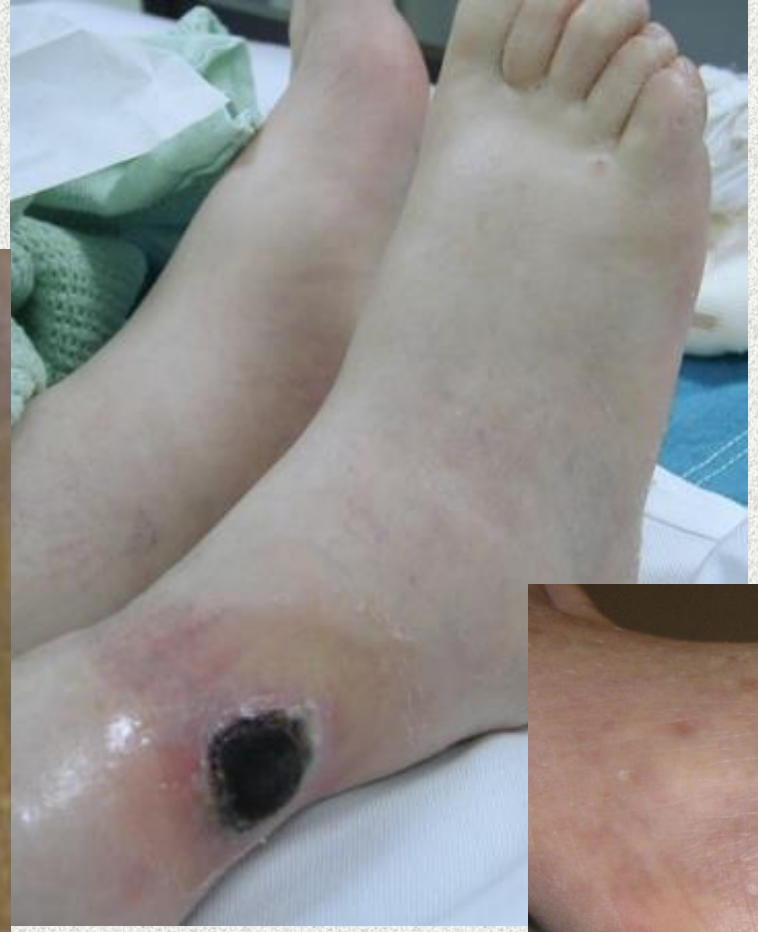
Arterial Insufficiency Ulcers

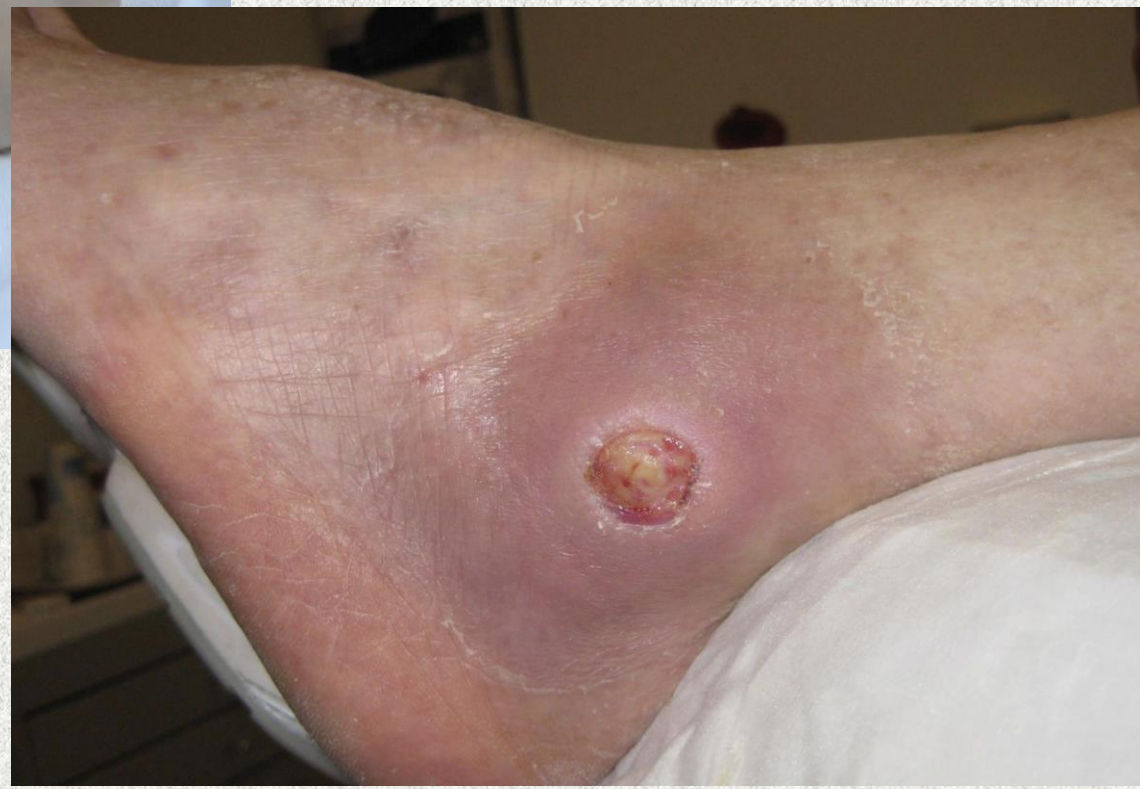
- Etiology – atherosclerosis → arterial insufficiency → poor supply of O2 and nutrients
  - Minor injury → slow healing due to poor blood supply
  - Severe arterial disease → spontaneous cutaneous death and ulceration
  - Atherosclerotic plaque embolization → complete occlusion of smaller cutaneous vessels → tissue ischemia, necrosis and ulceration
- Risk factors – obesity, HTN, high cholesterol, diabetes, smoking, clotting disorders, renal failure, RA, other vascular disorders
- Characteristics – *lateral* lower leg and dorsal feet, punched-out and deeper, dry with minimal bleeding, painful especially when legs elevated (relieved by lowering legs below heart level)
  - Surrounding skin – cool and pale, shiny with loss of hair, faint or absent pulses
  - Claudication common → relieved by rest

Arterial Insufficiency Ulcers

- Workup
  - Prolonged capillary refill time
  - Ankle brachial pressure index (ABPI) – <0.9 likely arterial disease, <0.5 severe disease
  - Transcutaneous oximetry around ulcer– <40mmHg vascular insufficiency, <20mmHg severe insufficiency
  - Buerger test – raising leg 45 degrees in supine position for 1min → palor of foot, followed by bright redness when lowered below table
- Treatment
  - Rule out and treat for infection
  - Wound care with specialized dressings (hydrocolloid, hydrogel, film), chemical debridement – consider Wound Care consultation
  - Physical (surgical) debridement – consider Gen Surg consultation
  - Other – control BP and cholesterol, ASA, weight loss, smoking cessation







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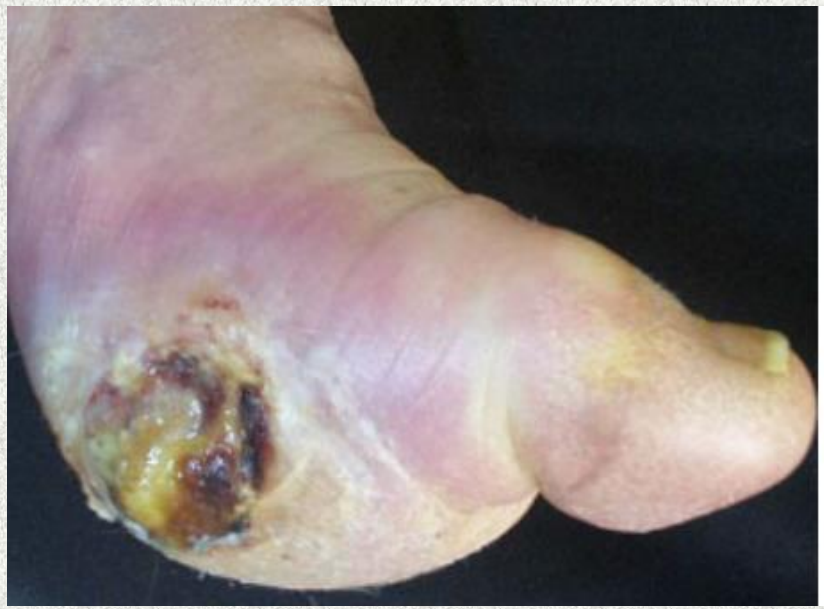
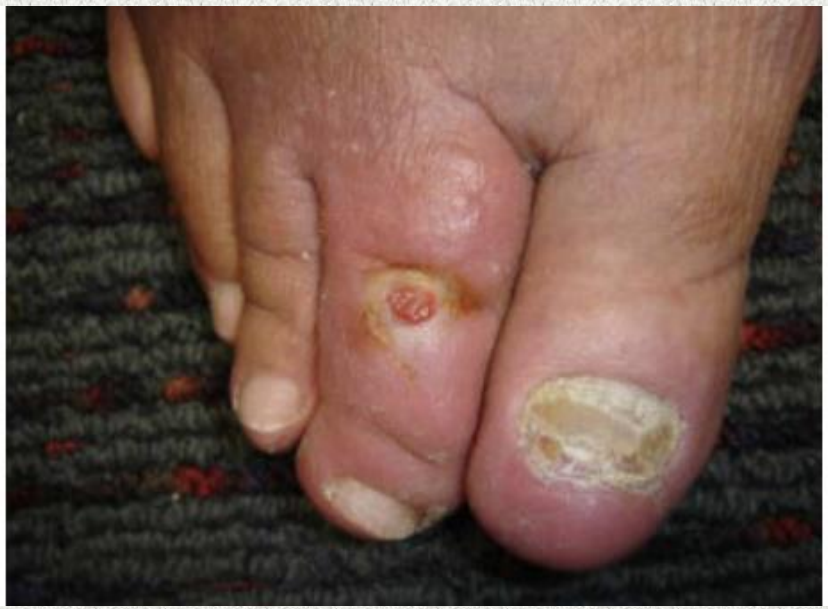
https://www.vascularsociety.org.uk/patients/conditions/12/arterial_ulcer
https://medicine.wright.edu/sites/medicine.wright.edu/files/page/attachments/LLoretzArterialandVenousLowerExtUlcers-final%5B2%5D_0.pdf

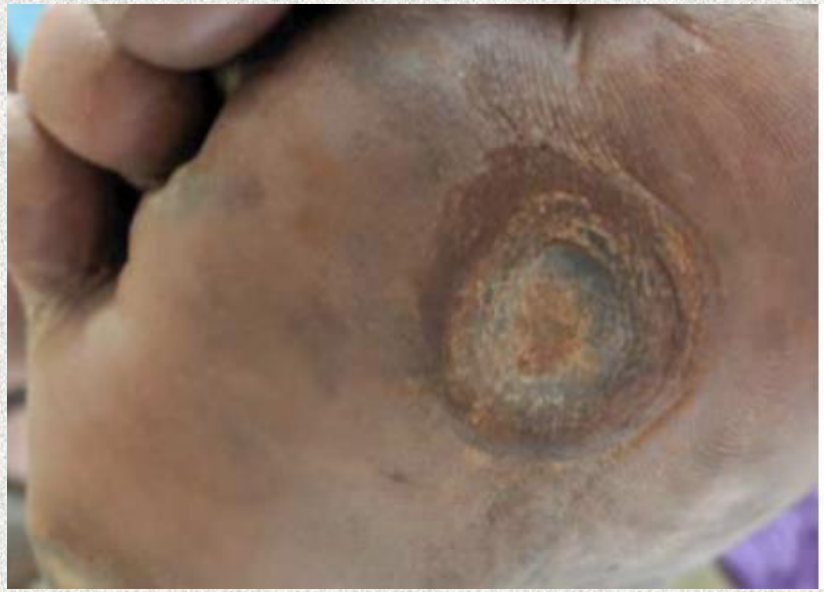
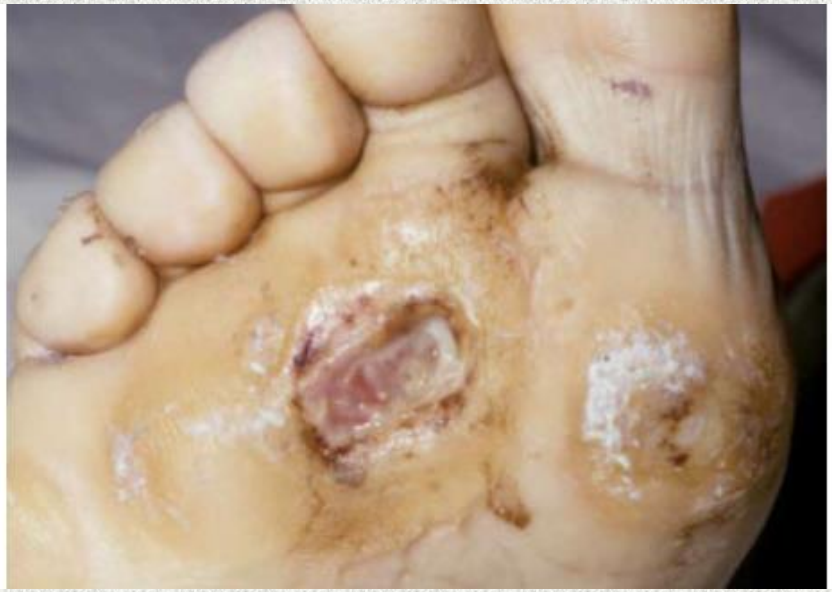
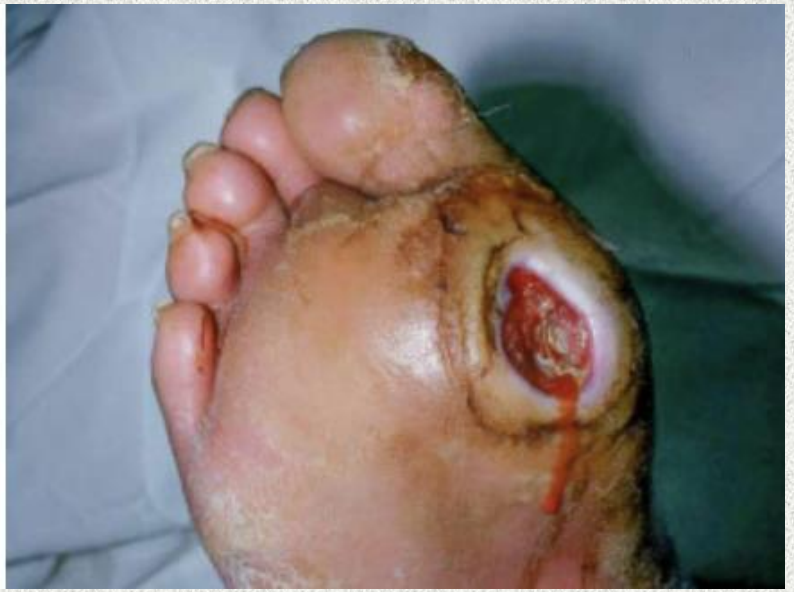
Diabetic Ulcers

- Etiology
  - Neuropathic – uncontrolled high blood sugar → damage to sensory nerves → loss of sensation → increased risk of injury or pressure
  - Vascular – uncontrolled high blood sugar → damage to blood vessels → decreased blood flow → ischemia, microvascular ischemia, necrosis and ulceration similar to arterial ulcers
- Risk factors – M > F, type 2 > type 1 diabetes, longer duration of diabetes, poor control, high A1c
- Characteristics – pressure points of feet, circular punched-out surrounded by keratotic callus, painless
  - Surrounding skin – faint pulses, reduced sensation

Diabetic Ulcers

- Complications – wound infection, cellulitis, osteomyelitis
- Workup – xray to r/o osteomyelitis, angiography
- Treatment
  - Rule out and treat for infection
  - Wound care with specialized dressings depending on level of exudate, chemical debridement – consider Wound Care consultation
  - Physical (surgical) debridement, grafting, amputation if necessary – consider Gen Surg consultation
  - Other – hyperbaric O2, Podiatry for pressure boot

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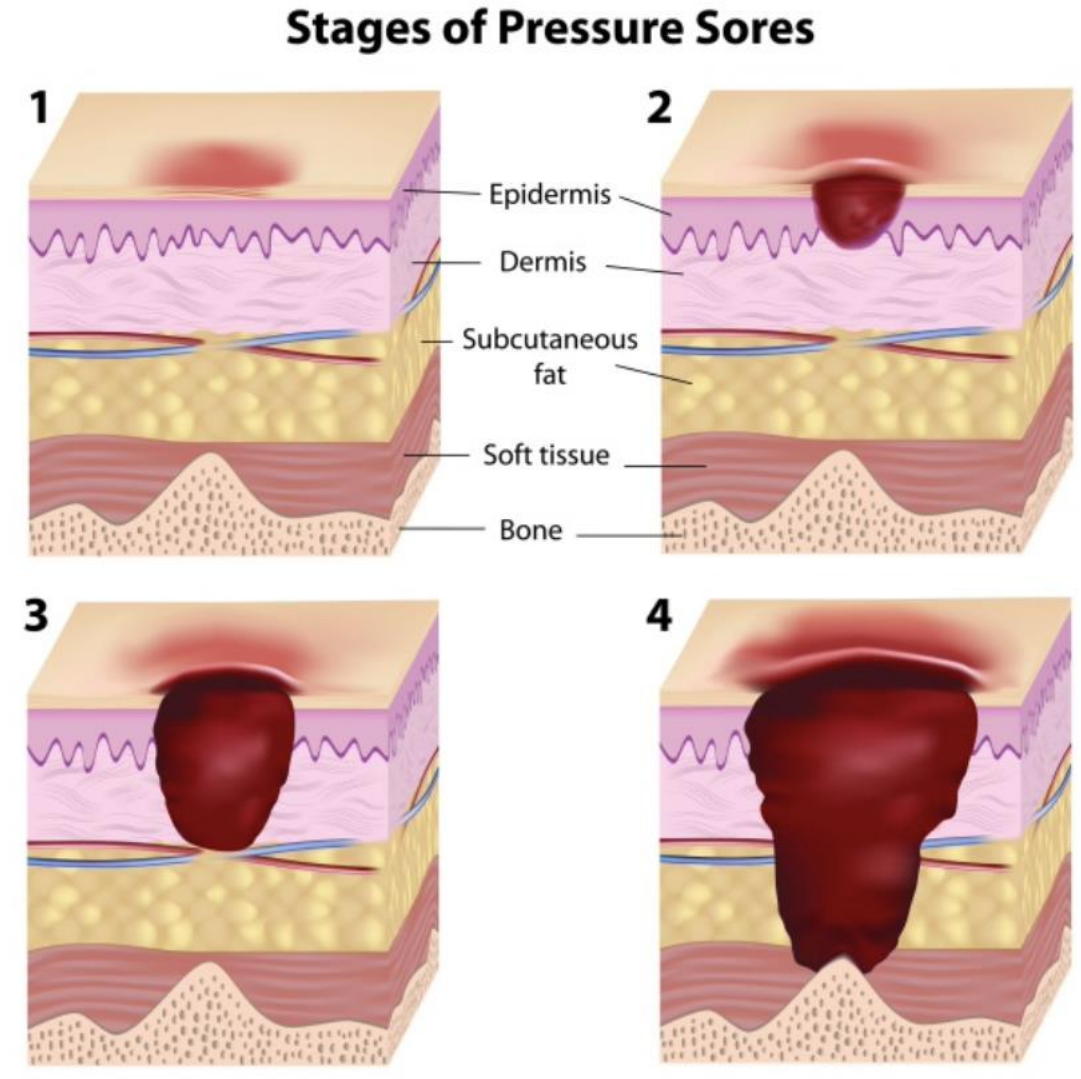
Pressure Ulcers

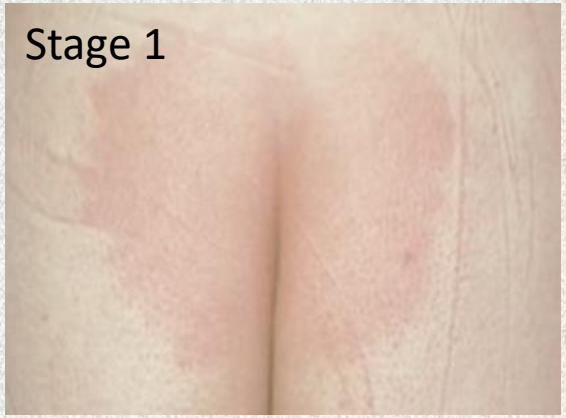
- Etiology – prolonged pressure (>2-3 hours) of skin/soft-tissue over bone → compression of blood flow → ischemia and tissue death
  - Factors that increase risk – friction, wrinkling/folding in bedding/clothing, moisture (sweat, blood, urine, feces), non-communicative pts, immobile, neuropathy, elderly

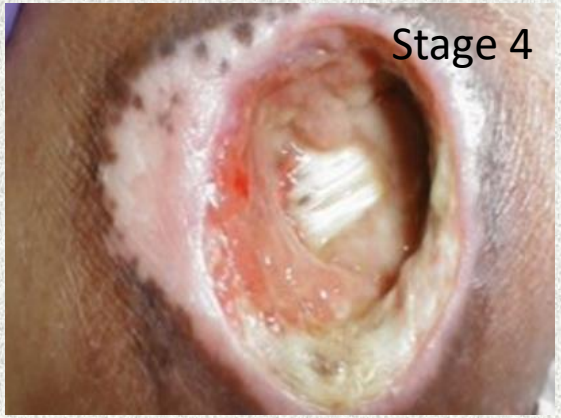
Stage 1	**Stage 2**	**Stage 3**
Pink to red skin, unbroken Tender, itchy or painful	Red, swollen skin Painful Bulla possible Erosion or shallow ulceration to superficial dermis	Ulceration into deeper dermis
Stage 4	**Deep Tissue Injury**	**Unstageable**
Ulceration into fat, muscle or bone Eschar formation	Persistent non-blanchable deep red/maroon/ purple discoloration Epidermal separation with dark wound bed Pain and temperature change precede color change	Extent of damage obscured by eschar

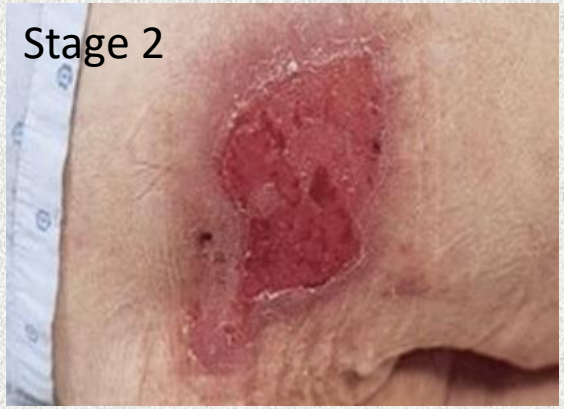
Pressure Ulcers

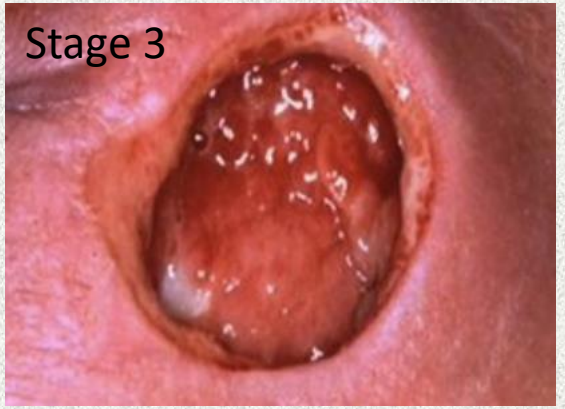
- Stage 1 and early stage 2 (before ulceration) – self-resolving once pressure removed, emollient and gentle skin care
- Late stage 2 – stage 4 (after ulceration)
  - Prevent, rule-out and treat infection
  - Wound care with specialized dressings depending on level of exudate, chemical debridement, woundVAC – consider Wound Care consultation
  - Physical (surgical) debridement, grafting – consider Gen Surg consultation
  - Other – alternating pressure bed, scheduled rotation of patient











https://sanaramedtech.com/blog/pressure-ulcer-injury-staging-classifications/

Neoplastic

Basal cell carcinoma

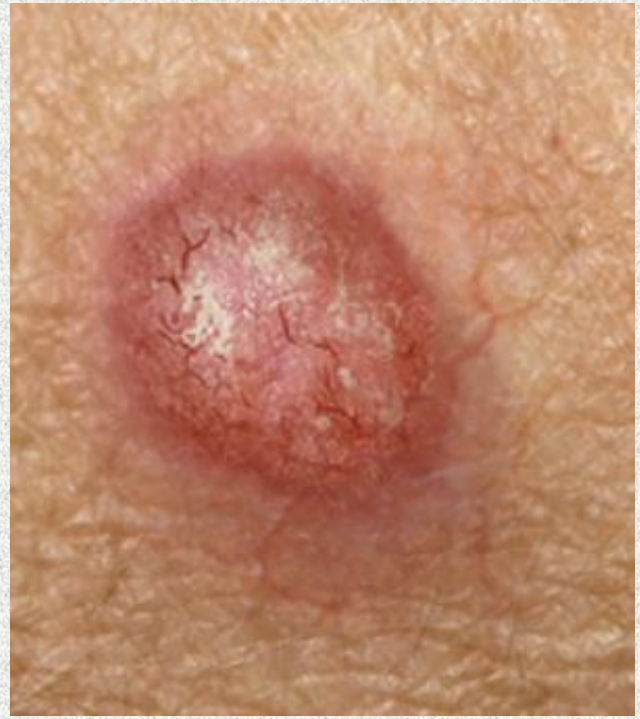
Squamous cell carcinoma

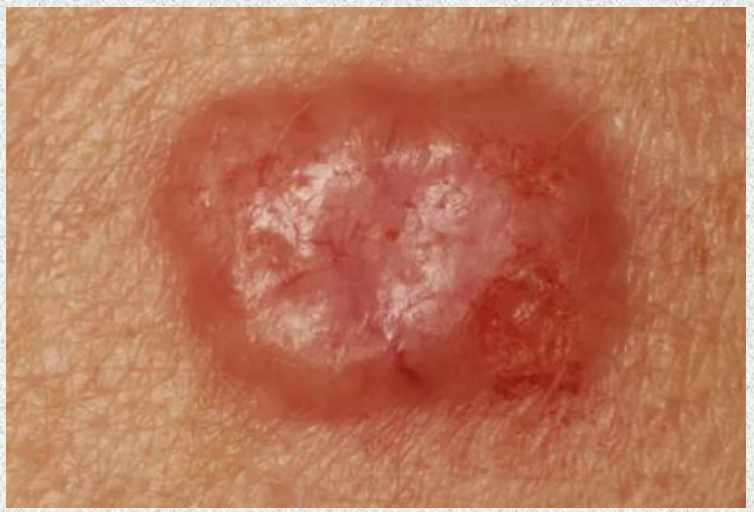
Melanoma

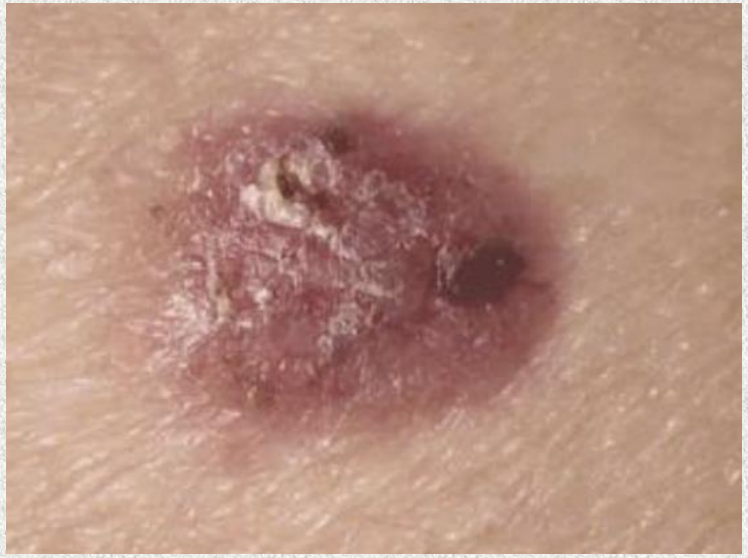
*Most do not require inpatient management but identification and referral is important, especially if high suspicion for large or rapidly growing SCC or melanoma given risk of metastatic disease

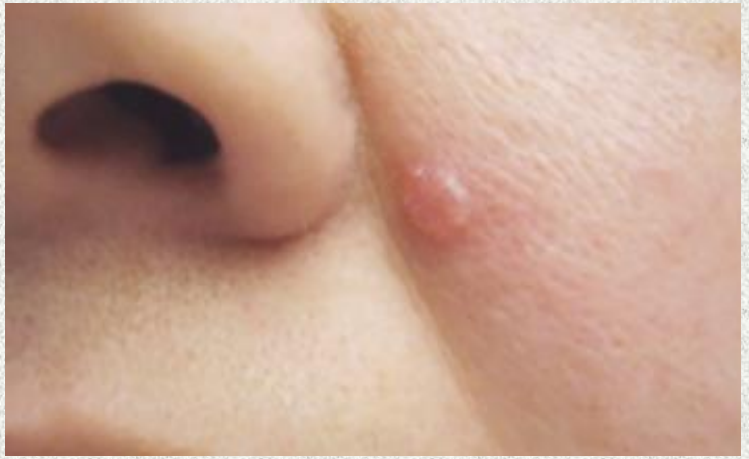
Basal Cell Carcinoma

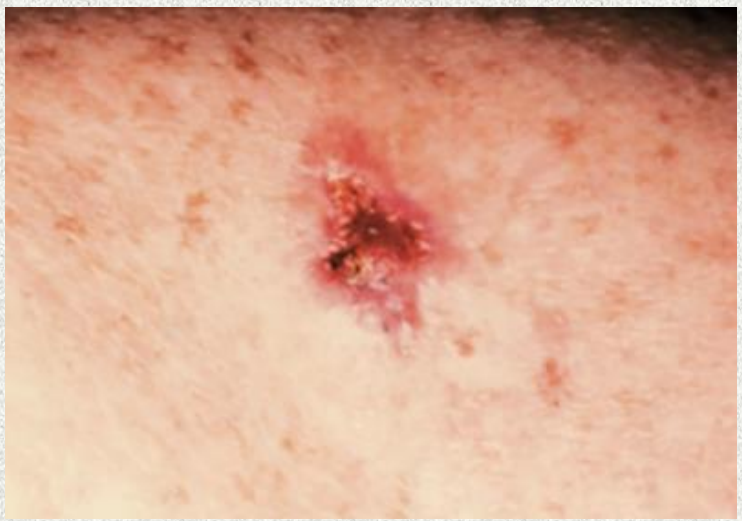
- Most common type of skin cancer
- Caused by UV exposure, often in combination with genetic defects
- Very low metastatic potential – rely on connections to basement membrane zone → minimal ability to spread through lymphatics
- Features – pearly or translucent pink papules or plaques with arborizing (branching) telangiectasias, may have rolled borders, central ulceration, crusting, friable (easy bleeding)
- Locations – more common in chronically sun exposed areas
  - Scalp, face, ears, nose, neck, upper chest, upper back, forearms, hands, lower legs











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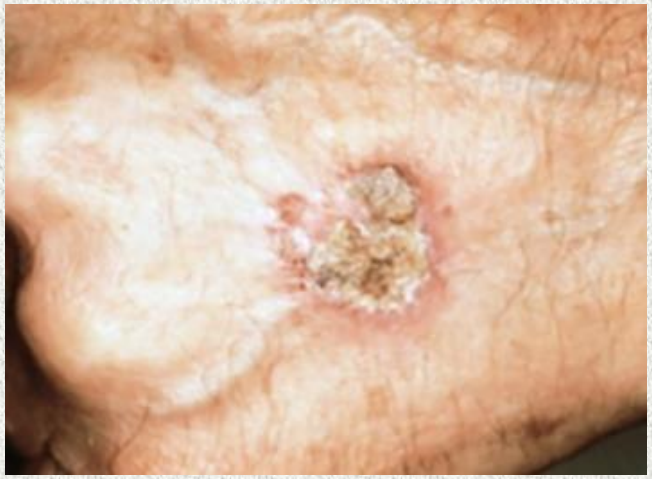
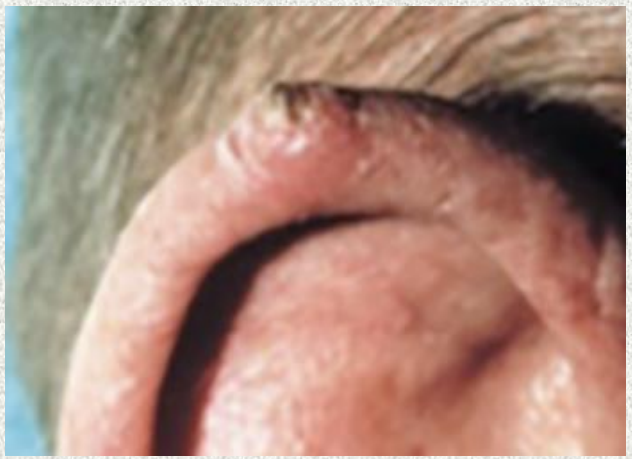
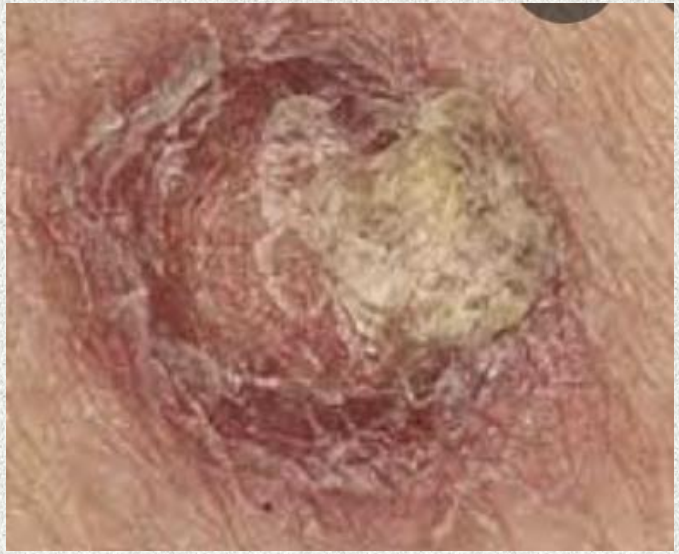
https://www.skincancer.org/skin-cancer-information/basal-cell-carcinoma/

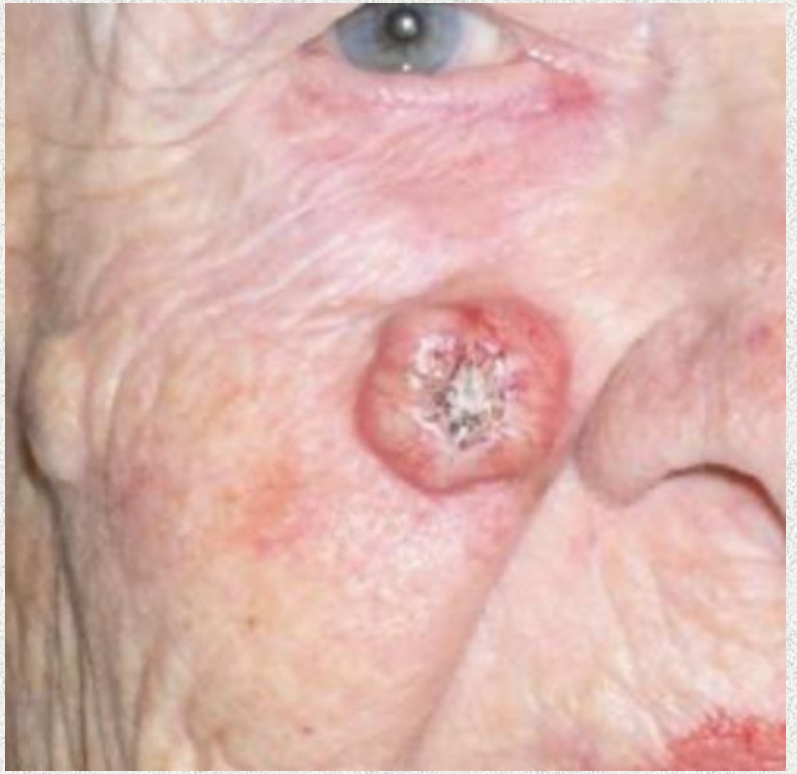
Squamous Cell Carcinoma

- Second most common type of skin cancer
- Caused by UV exposure
- Risk of perineural invasion and metastasis
- Features – pink scaly papule or plaque, may be hyperkeratotic, may have crusting
- Locations – more common in chronically sun exposed areas
  - Scalp, face, ears, nose, neck, upper chest, upper back, forearms, hands, lower legs
- May be associated with easy bleeding (eg. friable), splinter-like sensation, tenderness or may be a non-healing wound









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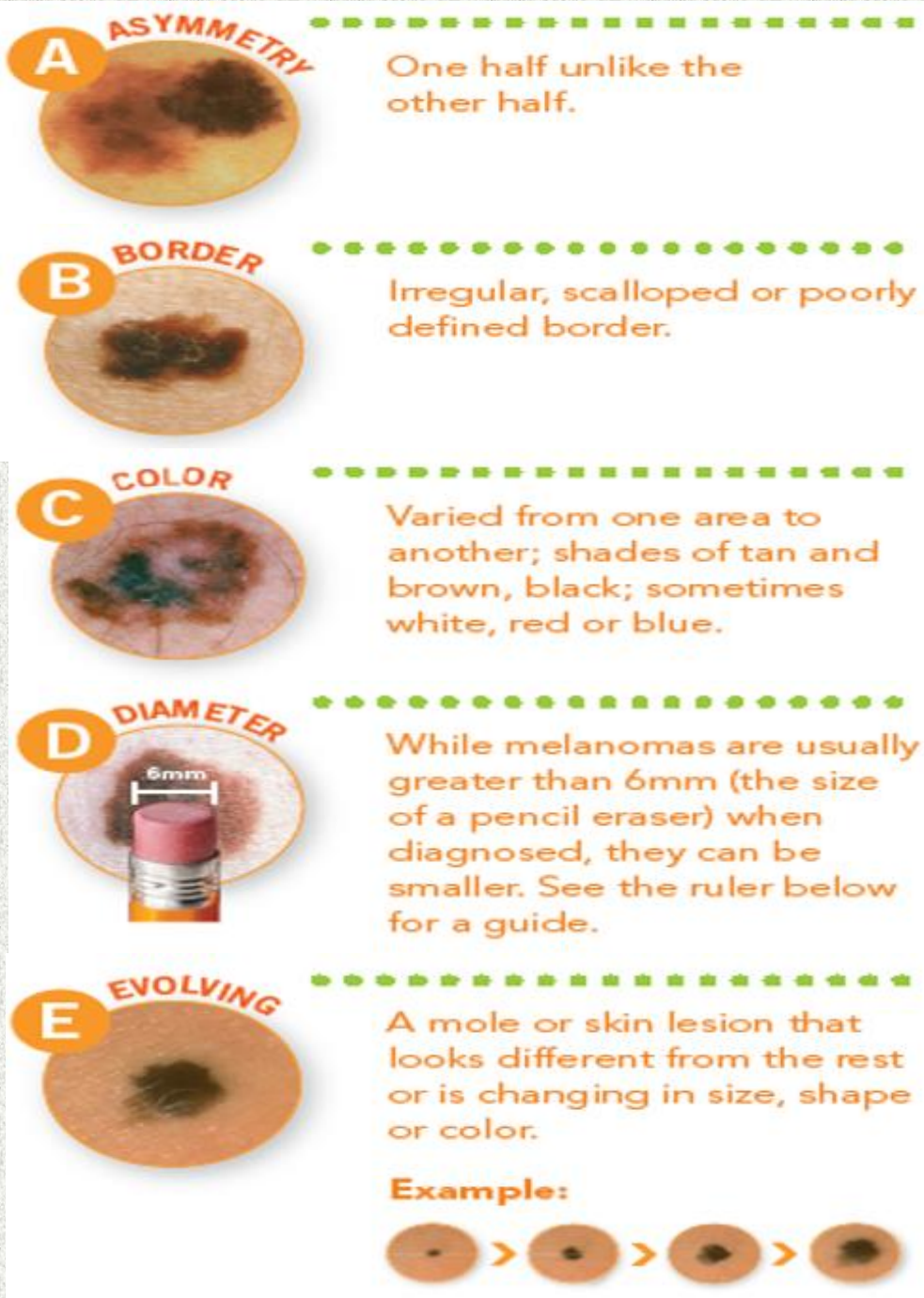
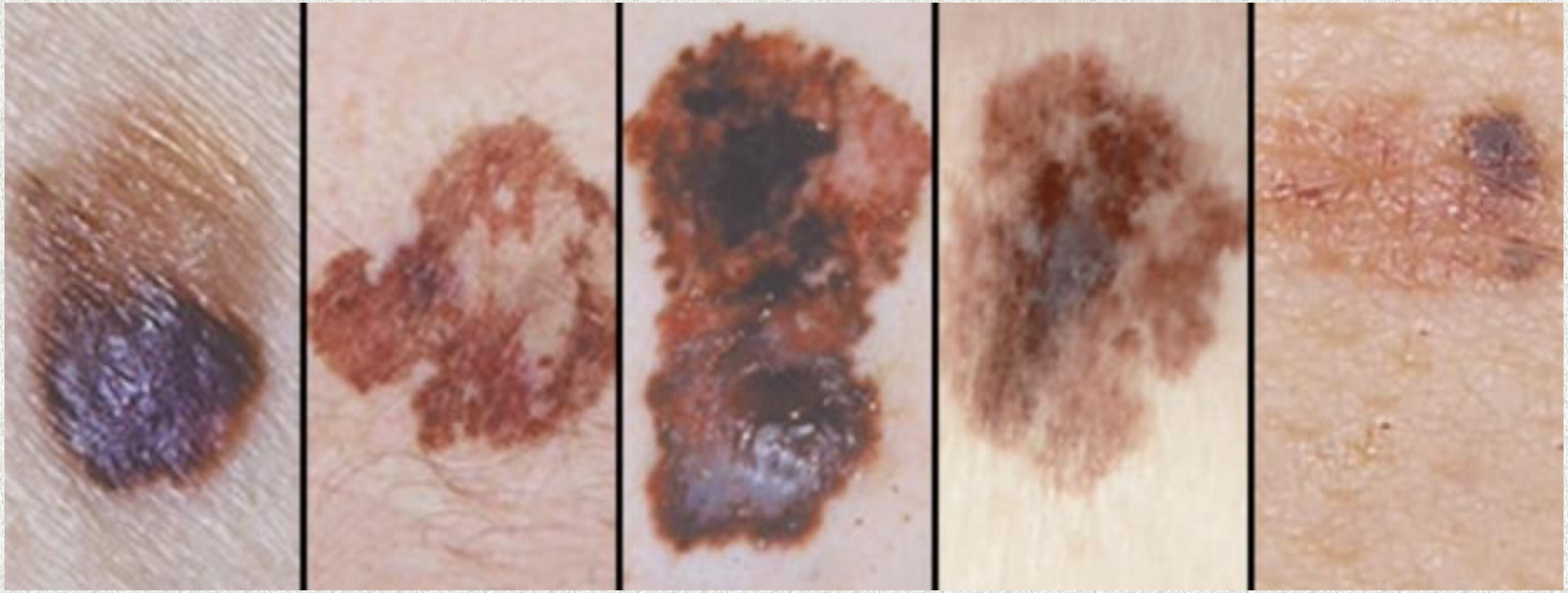
Melanoma

- Collection of atypical, mitotically active melanocytes in the epidermis alone (melanoma in situ) or epidermis and dermis (invasive)
- Often a/w UV exposure, however many have genetic mutations can occur in non-sun exposed sites (including eyes, oral mucosa, genitals)
- Aggressive, rapidly growing cancers with significant risk for metastasis
- Features – irregular, asymmetric macules, patches, papules, plaques or nodules with variegated color
- Locations – more common in sun exposed areas but can occur anywhere
  - MC location in males = back
  - MC location in females = lower legs

Signs of Melanoma

- New pigmented lesion
- Growing pigmented lesion
- Pigmented lesion with changing size, shape, color, thickness





https://www.mayoclinic.org/diseases-conditions/melanoma/multimedia/melanoma/sls-20076095

Question 1

With which drug reaction is it acceptable to re-expose the patient to the offending medication?

1. DRESS
2. SJS/TEN
3. Morbilliform
4. Urticaria with angioedema and/or systemic symptoms
5. AGEP

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Morbilliform drug eruptions are not considered SCARs and therefore rechallenge is acceptable with treatment of skin symptoms through course of offending agent. DRESS, AGEP and SJS/TEN are considered SCARs and rechallenge is likely to be worse and associated with higher morbidity and mortality. Non-complicated urticarial drug eruption can be rechallenged, but urticarial reaction with angioedema, systemic symptoms or anaphylaxis should not be rechallenged.

Question 2

A 73 year-old male admitted for PE is noted to have erythema and edema of both lower legs. Which of the following statements is true.

1. Diagnosis is cellulitis and IV antibiotics are the treatment of choice
2. Diagnosis is stasis dermatitis and PO antibiotics are the treatment of choice
3. Diagnosis is contact dermatitis and topical steroids are the treatment of choice
4. Diagnosis is stasis dermatitis and topical steroids are the treatment of choice

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Stasis dermatitis is more commonly bilateral whereas cellulitis is much more commonly unilateral. Treatment of choice for stasis dermatitis is topical steroids.

Question 3

Which of the following are signs of bacterial endocarditis?

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2. High fever and chest pain
3. Osler nodes
4. Janeway lesions
5. All of the above can be seen in bacterial endocarditis

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Bacterial endocarditis can present with fever, CP, proximal nail splinter hemorrhages, Osler nodes and Janeway lesions.

Question 4

Which of the following is not appropriate in the management of calciphylaxis?

1. Sodium thiosulfate
2. High calcium diet
3. Switching to heparin
4. Parathyroidectomy
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As hypercalcemia is part of the pathogenesis in calciphylaxis, diet must have limited added calcium.

Question 5

Which characteristics do not correctly describe the ulcer type?

1. Stage 3 pressure ulcer – red swollen painful skin with ulceration extending into superficial dermis
2. Venous insufficiency ulcer – medial lower leg, jagged and shallow, heavy exudate and weeping, minimal pain
3. Diabetic ulcer – circular punched-out surrounded by keratotic callus, painless
4. Arterial insufficiency ulcer – lateral lower leg and dorsal feet, punched-out and deeper, dry with minimal bleeding, painful especially when legs elevated

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Stage 2 pressure ulcers present with red swollen painful skin with possible ulceration into the superficial dermis. Stage 3 pressure ulcers have ulceration into the deep dermis.

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