

DRUG ERUPTIONS and other DERM IM CONDITIONS

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DRUG ERUPTIONS

Drug Reactions

3 things you need to know

1. Type of drug reaction

2. Statistics

- u What drugs are most likely to cause that type of reaction?

3. Timing

- u How long after the drug was started did the reaction begin?



Clinical Pearls

- u Drug eruptions are extremely common
- u Tend to be generalized/symmetric
 - u Maculopapular/morbilliform are most common
- u Best Intervention: Stop the Drug!
 - u Do not dose reduce
 - u Completely remove the exposure
- u How to spot the culprit?
 - u Drug started within days to a week prior to rash
 - u Can be difficult and take time
 - u Tip: can generally exclude all drugs started after onset of rash
- u Drug eruptions can continue for 1-2 weeks after stopping culprit drug

LITT's drug eruption database

com

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Litt's
DRUG ERUPTION & REACTION DATABASE

“ The most comprehensive database on adverse drug reactions for our profession.
David Adams MD ”

Log in

Home Search multiple drugs **Search class reactions** About Help Alerts Smartphones/tablets Subscribe Contact us

Search our database for **1619** drugs with **60128** documented drug reactions as evidenced by **116195** references on PubMed

Drug name Search GO

Drugs see all	Adverse Reaction see all	Drug Class see all
Brivaracetam NEW!	Ocular hypersensitivity [1]	Antibiotic, macrolide
Ixekizumab NEW!	Paranoia [26]	Anticonvulsant
Ledipasvir & Sofosbuvir	Palmar-plantar punctate keratoses [1]	Antipsychotic
Physostigmine	Amyotrophic lateral sclerosis [1]	Selective serotonin reuptake inhibitor (SSRIs)
Sulindac	Hypervolemia [3]	Statin
Deoxycholic Acid	Febrile neutropenia [113]	

Herbal see all	Reaction Category see all	Company see all
Omega-3 Fatty Acids	Skin	GSK
Red Clover	Cardiovascular	Novartis
Eucalyptus	Central Nervous System	Pfizer
Hawthorn (Fruit, Leaf, Flower Extract)	Mucosal	Sanofi-Aventis
Ginger	Nails	Wyeth
	Neuromuscular/skeletal	

Saved Searches

Watched Drugs

Litt's
DRUG ERUPTION & REACTION DATABASE

Mobile version is now available to subscribers!
Search drug eruptions and reactions data anytime, anywhere





Image Gallery



Drug Eruptions

- u Skin is one of the most common targets
- u **Antibiotics and anticonvulsants are most common**
 - u 1-5% of patients
- u 2% of all drug eruptions are “serious”
 - TEN, DRESS
- u More common in adult females and boys < 3 y/o
- u Not all drugs cause eruptions at same rate:
 - Aminopenicillins: 1.2-8% of exposures
 - TMP-SMX: 2.8-3.7%
 - NSAIDs: 1 in 200
 - Lamotrigine: 10%

Drug Eruptions

Three basic rules

1. **Stop** any unnecessary medications
2. **Ask about non-prescription medications**
 - u Eye drops, suppositories, implants, injections, patches, vitamins and health supplements, friend's medications
3. **ALWAYS consider medications as possible cause**
(no matter how atypical)

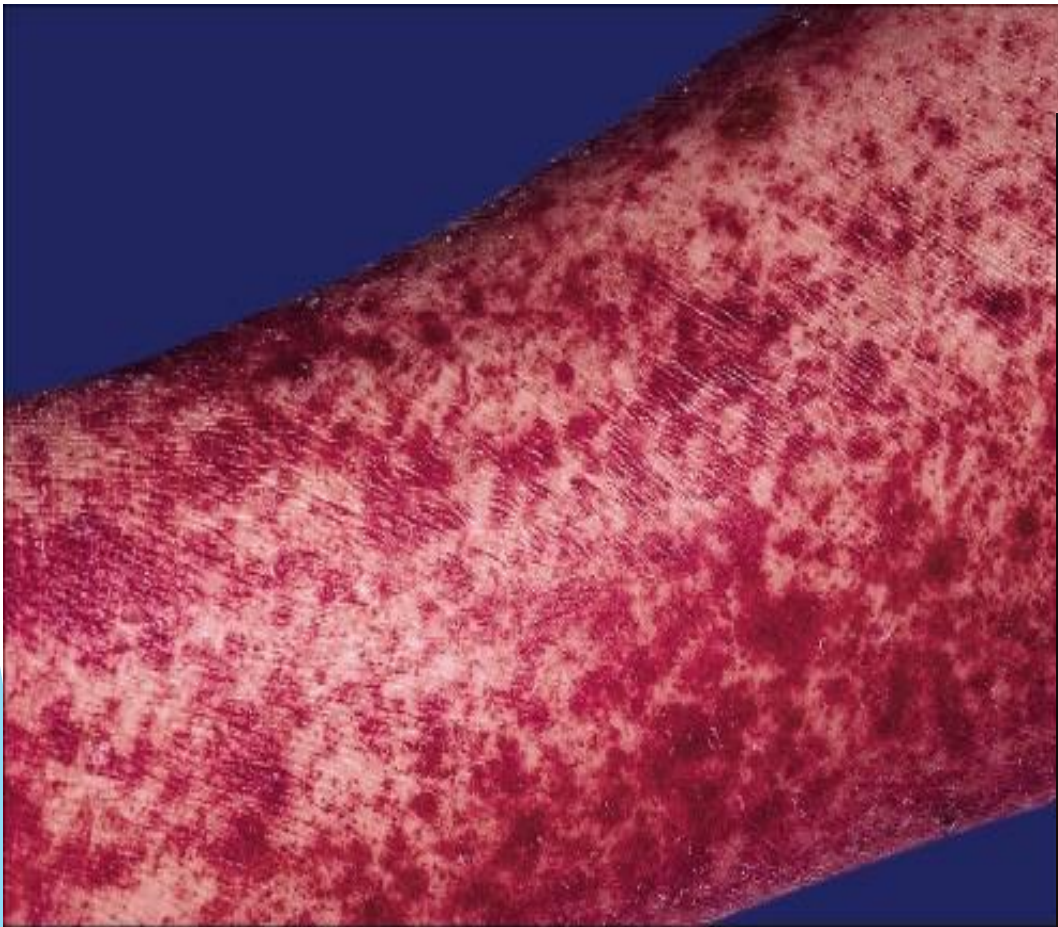


Note confluence of lesions on trunk

Morbilliform reaction to ampicillin-amoxicillin



Clinical presentation	Percentage that are drug-induced (%)	Time interval	Mortality (%)	Selected responsible drugs
Exanthematous eruption	Child: 10–20 Adult: 50–70	4–14 days	0	Aminopenicillins Sulfonamides Cephalosporins Anticonvulsants Allopurinol
Urticaria Anaphylaxis	<10 30	Min-hours Min-hours	0 5	Penicillins Cephalosporins NSAIDs Monoclonal Abs Contrast media
Fixed drug eruption	100	First exposure: 1–2 weeks Re-exposure: <48 hours, usually within 24 hours	0	TMP-SMX NSAIDs Tetracyclines Pseudoephedrine
Acute generalized exanthematous pustulosis (AGEP)	70-90	<4 days	1-2	β -Lactam antibiotics Macrolides Calcium channel block.
Drug reaction with eosinophilia and systemic symptoms (DRESS)	70-90	15-40 days	5-10	Anticonvulsants Sulfonamides Allopurinol Minocycline Lamotrigine
Stevens–Johnson syndrome (SJS) Toxic epidermal necrolysis (TEN)	70-90	7-21 days	5 30	Sulfa Anticonvulsants NSAIDS Allopurinol





Vasculitis

Drug Induced Vasculitis

- Palpable Purpura
 - Dependent area
 - Symmetrical distribution
 - Urticaria-like lesions, ulcers, nodules, hemorrhagic blisters, pustules and digital necrosis
- Typically small vessels
- 7 to 10 days after drug administration and < 3 days following rechallenge
- Medications associated:
 - PCNs, NSAIDs (oral and topical), sulfonamides and cephalosporins
 - Propylthiouracil, thiazide diuretics, furosemide, allopurinol, phenytoin
 - Fluoroquinolones and biologic agents [G-CSF, GM-CSF], interferons
- Treatment
 - Stop offending agent
 - Supportive care
 - NSAIDS, antihistamines



Fixed Drug Eruption

Fixed Drug Eruptions

- Lesions reoccur at the same site with each exposure to medication
 - 1 to 2 weeks after first exposure
 - Within 24 hours, after subsequent exposures
- Clinically:
 - One or a few, round, sharply demarcated erythematous & edematous plaques
 - Dusky, violaceous hue, central blister or detached epidermis
- Anywhere on the body,
- Favor the lips, face, hands, feet and genitalia
 - 50% on oral or genital mucosa
 - 2% of all genital ulcers (especially young boys)
- Treatment
 - Lesions fade, leaving a residual postinflammatory brown pigmentation

Fixed Drug Eruptions

u Usually intermittent drugs:

- ❖ NSAIDs
- ❖ Sulfonamides (TMP) → majority of genital fixed drug eruptions
- ❖ Barbiturates, TCNs, phenolphthalein, acetaminophen, cetirizine, celecoxib, dextromethorphan, hydroxyzine, lamotrigine, phenylpropanolamine, erythromycin, herbs



Acneiform Eruption

- Epidermal growth factor receptor (EGFR)
 - Treatment of advanced lung, pancreatic, colorectal, and head and neck cancers
 - Monoclonal antibodies
 - Cetuximab, panitumumab
 - Small-molecule tyrosine kinase inhibitors
 - Gefitinib, erlotinib, lapatinib
 - Cutaneous adverse events to EGFR inhibitors are frequent
 - Abundant expression of EGFR in the skin and adnexal structures.

Acneiform



This lung cancer patient who has been receiving radiation therapy is on a **tea and toast diet** and complains of **myalgias**. History is positive for **soft tissue bleeding**. Hb is 8 gm. He is deficient in vitamin:

- A. B6
- B. A
- C. D
- D. E
- E. C**



Gingival hyperplasia



“Corkscrew hairs”

NUTRITIONAL DISORDERS

- u Vitamin K Deficiency
- u Vitamin B3 Deficiency
- u Vitamin C Deficiency
- u Zinc Deficiency
- u Iron Deficiency

Vitamin K Deficiency

- *Infants*
 - Premature, uncolonized GI tract
- *Adults*
 - Malabsorption; liver dz
- Clinical:
 - Purpura
 - Massive hemorrhage
- Dx: elevated PT and PTT
- Tx:
 - Adults = Vitamin K 5-10 mg/day IM x several days
 - Kids = 2 mg/day (0.5 to 1mg in newborns)
 - Acute crisis = Fresh Frozen Plasma (FFP)



Vitamin B3 Deficiency- Pellagra

- *Skin manifestations may be the 1st sign*
- **Glossitis**: inflammation of the tongue
- The 4 D's: **diarrhea, dementia, dermatitis** → DEATH
 - **Diarrhea**
 - Acute inflammation of the small intestine and colon
 - **Dementia**
 - Patchy demyelination and degeneration of the affected nervous system
 - **Dermatitis**
 - Four types of dermatitis
 - **Photosensitive eruptions**
 - *Perineal lesions*
 - Thickening and pigmentation over bony prominences
 - Seborrheic-like dermatitis of the face

Pellagra



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Casal's necklace: Photosensitive eruption on face, neck, and upper chest

Pellagra

- u Additional manifestations:
 - u Depression, apathy, psychosis, coma
 - u Death in 4-5 yrs if untreated
- u Dx: clinical
 - u Low serum niacin, tryptophan
- u Tx: Niacin 50-300mg/day PO (rapid reversal of pellagra)
 - u 100mg/day IV for malnourished

VITAMIN C DEFICIENCY

Scurvy



- Water-soluble vitamin found in fresh fruits, vegetables
- Roles:
 - Collagen & ground substance formation
 - Synthesis of epinephrine & carnitine
 - Leukocyte function, iron absorption, folic acid metabolism
- Reducing agent:
 - Cofactor for hydroxylation of procollagen → collagen
- **Elderly male alcoholics (MC)**, psych patients on restrictive diets, children 6-24mo

Scurvy

- Four Hs:
 - Hemorrhage
 - Hemorrhagic gingivitis
 - Epistaxis
 - Perifollicular petechiae
 - Subungual, IM, and intraarticular hemorrhage
 - Subperiosteal hemorrhage leading to pseudoparalysis
 - Hyperkeratosis of the hair follicles
 - Hypochondriasis
 - Hematologic abnormalities



- Clinical

- Woody edema
- Corkscrew hairs → plugging of hair follicles by curled hairs
 - Forearms, abdomen, thighs
- Delayed wound healing
 - due to secondary defect in collagen formation
- ***Depression***
- **ANEMIA** (secondary to bleeding)

- Tx:

- Ascorbic acid 1000 mg/day
- Maintenance dose of 100 mg/day should be considered



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Zinc Deficiency

- u Functions of Zinc:

- u **Wound healing**

- u Immune/reproductive/neuropsychiatric function

- u **Inc. zinc requirements:**

- u infections, post-surgery, pregnancy, cancer

- u *Largely dependent on food intake*

- u Nuts, whole grains, green leafy vegetables, shellfish, human milk

- u **Presents most commonly in infancy**

- u Premature

- u suboptimal absorption, high zinc requirements, inadequate body stores

- u Breast milk generally provides adequate zinc

- u Occurs at the time of weaning from breast milk to cow's milk



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Zinc Deficiency

Genetic

**“acrodermatitis
enteropathica”**

zinc transporter mutation

Acquired

alcoholics,
malnourished,
CRF,
malignancies,
pregnancy, drugs,
HIV



Zinc Deficiency

- u Triad: *Dermatitis, diarrhea* and *alopecia*

- u Dermatitis:

- u **Acral and periorificial distribution**

- u Patchy, red, dry scaling with exudation and crusting

- u Angular cheilitis and stomatitis; drooling

- u Diarrhea: *suspect in infant with chronic diaper rash & diarrhea*

- u Alopecia: generalized

- u Additional Sx:

- u Growth retardation

- u Impaired wound healing

- u CNS findings

- u Emotional lability & irritability



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Zinc Deficiency

Dx:

- u Low serum zinc levels but not diagnostic
- u **LOW alkaline phos** (zinc-dependent enzyme)

Tx:

- u Zinc sulfate PO 1 to 2 mg/kg/day
- u **Acrodermatitis enteropathica: 3 mg/kg/day lifelong**
- u Warm compresses and petroleum applied TID to areas of weeping or crusted dermatitis to support reepithelialization

Iron Deficiency

- u Decreased total-body iron content
- u Common
 - u Menstruating women
 - u Diet low in red meat or diminished absorbable dietary iron
- u Iron balance: achieved by regulation of iron absorption in the proximal small intestine
- u Dx: check serum iron, TIBC and ferritin
- u Tx: Iron sulfate 325 mg PO TID
- u Plummer-Vinson: middle-aged women
 - u Microcytic anemia
 - u Dysphagia/**esophageal webs**
 - u Thin lips with small inelastic opening of the mouth

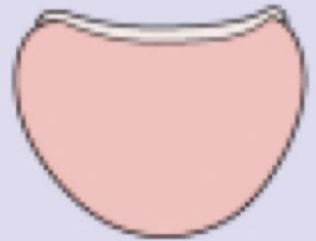
Iron Deficiency

u Clinical Manifestations

- u koilonychia
 - u “spoon nails”
 - u 40-50%
 - u *physiologic in kids*
- u Glossitis
- u Angular cheilitis
- u Pruritus
- u Telogen effluvium



Koilonychia



Case

- u 84 y.o. obese, caucasian female
- u Multiple enlarging, necrotic, ulcerated plaques on the lower extremities
- u Rapidly expanding up to 15 cm over several weeks
- u Painful upon palpation, indurated

Initial Presentation



Initial Presentation



Patient History

- u Past medical history
 - u Atrial fibrillation, hypercholesterolemia, lower extremity edema
- u Past dermatological history
 - u none
- u Medications
 - u Warfarin, simvastatin, spironolactone, torsemide

Plan

u Biopsy

- u A punch biopsy and incisional biopsy were performed in the office

u Labs

- u CBC, CMP, phosphorus, PT/PTT/INR, protein C & S, antithrombin III, anticardiolipin antibody, lupus anticoagulant, factor V leiden, serum cryoglobulins, hepatitis C antibody, vitamin D, parathyroid hormone

u Hospital admission

- u Further evaluation and treatment

Incisional Biopsy



Pathology Report

- u Punch biopsy and incisional biopsy revealed fibrin thrombi with a broad ddx
 - u Protein C & S deficiency
 - u Warfarin induced necrosis
 - u DIC
 - u Purpura fulminans
 - u Cryoglobulinemia
 - u Antiphospholipid syndrome
 - u Factor V leiden deficiency

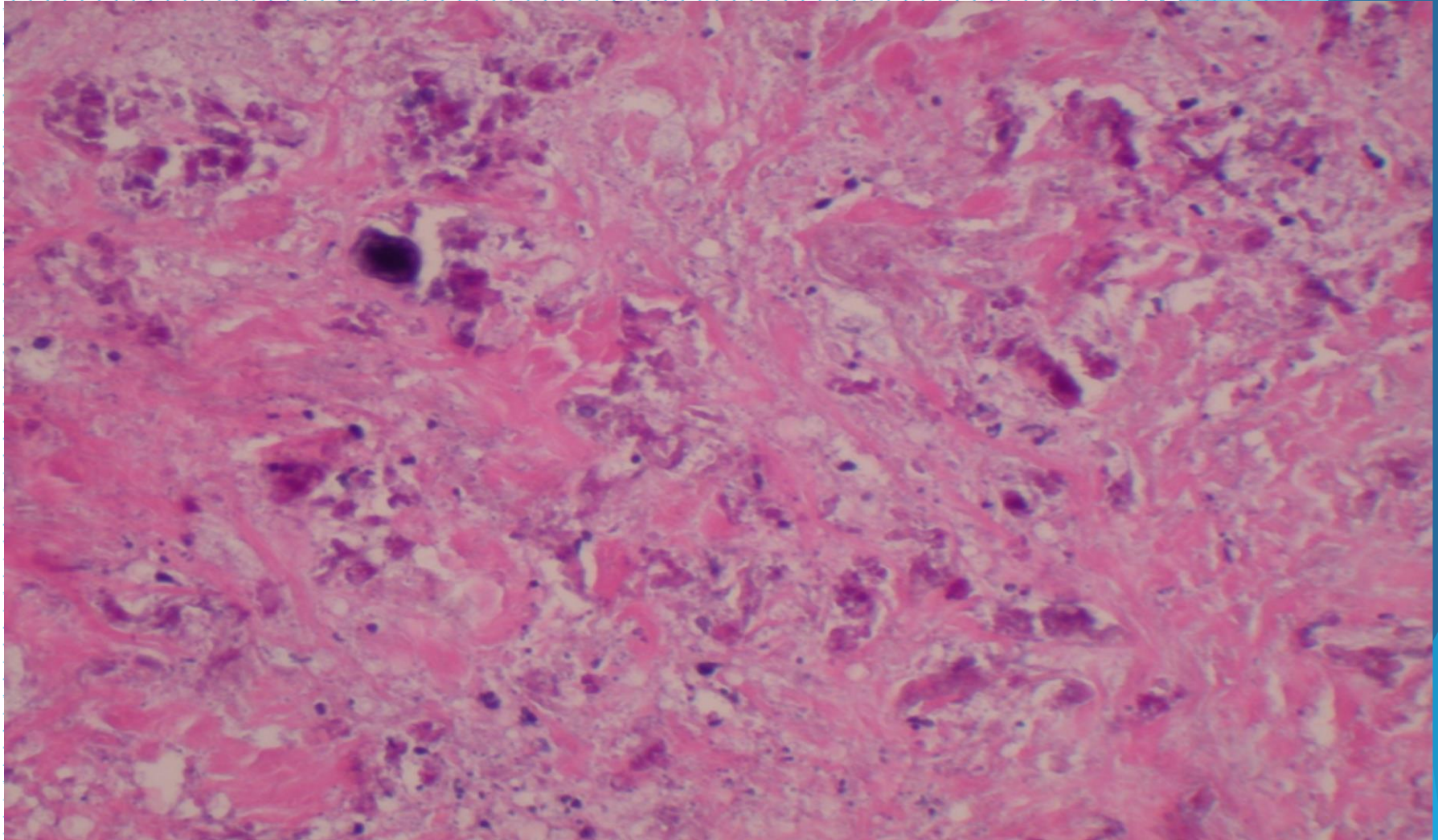
Labs

- u Significant for:
 - u Increased: serum creatinine, alk phos, PT, anticardiolipin IgM antibody, lupus anticoagulant
 - u Normal: calcium, phosphorus, INR, parathyroid hormone, factor V leiden, cryoglobulins
 - u Decreased: GFR (29), protein C & S

Hospital Course

- u Excisional biopsy performed by general surgery on one of the ulcerated, necrotic plaques
 - u Consistent with **calciophylaxis**
- u Patient was started on sodium thiosulfate 25g IV daily
- u Patient deferred further treatment and was discharged to a nursing home for palliative care
- u Patient died within 1 month of initial visit

Excisional Biopsy



Calciophylaxis

- u *AKA calcific uremic arteriopathy*
- u Rare and serious disorder that features systemic medial calcification of arterioles that causes ischemia and subcutaneous necrosis
- u Most commonly occurs in ESRD patients on hemodialysis
- u Pathogenesis
 - u Poorly understood, multifactorial
 - u Vascular calcification
- u Risk factors
 - u ESRD, female sex, obesity, hyperparathyroidism, hypercoaguable states, hyperphosphatemia, medications
 - u **warfarin**, vit D analogs, systemic glucocorticosteroids
 - u warfarin: 10 fold increased risk of calciophylaxis

Discussion Cont

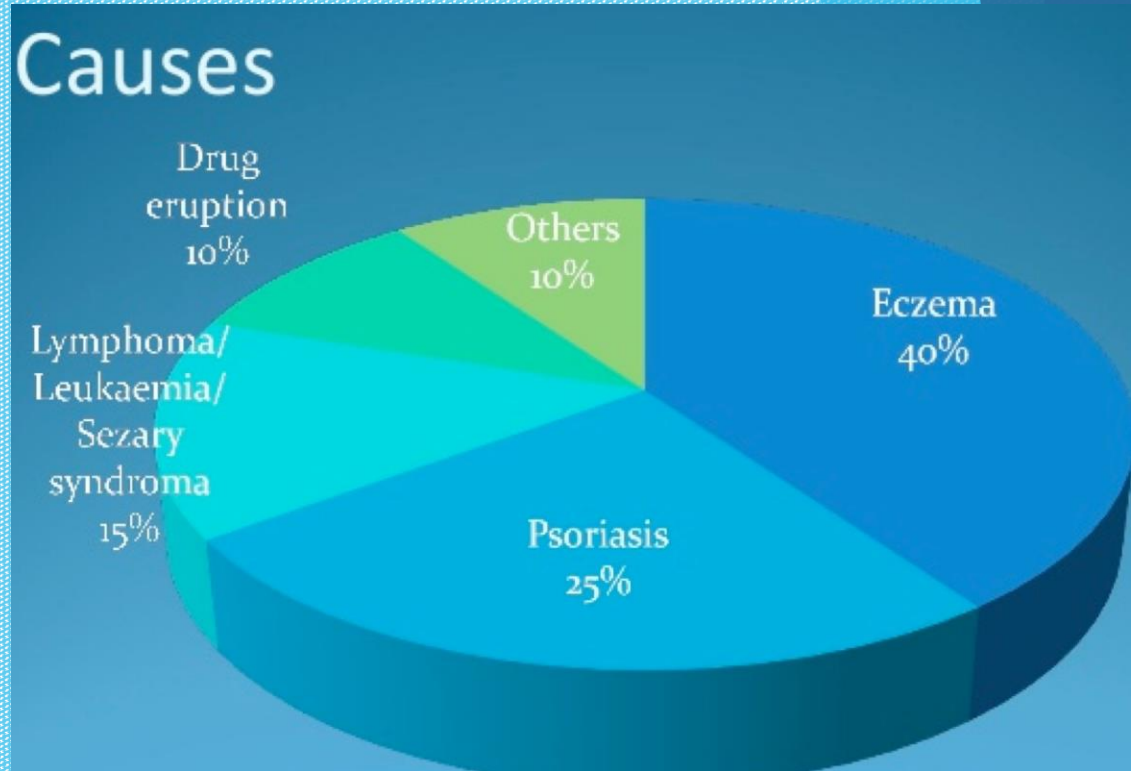
- u Treatment
 - u Evidence based guidelines have yet to be determined
 - u IV Sodium thiosulfate - calcium binder and antioxidant
 - u Wound care and pain management
 - u Oxygen therapy
 - u Correct any underlying lab abnormalities

Calciophylaxis Conclusion

- u Elderly, obese female with an acute onset of calciophylaxis
- u Multiple cofactors that might have contributed to the pathogenesis
- u Treatments range from case report experience to the correction of underlying etiologies
- u Calciophylaxis is an aggressive and poorly prognostic diagnosis that still leaves a lot to be learned

Erythroderma

- What is it?
 - Exfoliative dermatitis that involves >90% surface area
- Causes
 - Psoriasis
 - Seborrheic Dermatitis
 - Drug Eruptions
 - Pityriasis rubra pilaris
 - Lymphoma
 - Eczema
 - Infection
 - Bacterial
 - Fungal
 - Viral
 - Autoimmune bullous dz



Psoriasis



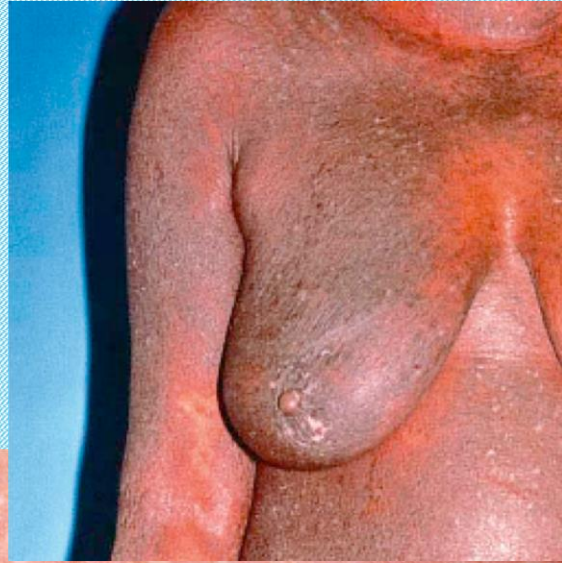
Seborrheic Dermatitis



Drug Eruption



Pityriasis Rubra Pilaris



Lymphoma



Case 1: 65 y/o Female presents w/ “tender skin all over” which began after few days of coughing and fever and chills



Case 1: Continued...

- u ROS: Photophobia & dysphagia/odynophagia
- u Recently Rx an antibiotic for a “large boil on her leg”
- u PMHx: seizure disorder
- u Meds: Trimethoprim/sulfamethoxazole, Lamotrigine



Stevens-Johnson (SJS) & Toxic Epidermal Necrolysis (TEN)



Characteristic dusky red color of the early macular eruption

Erythema Multiforme, Stevens-Johnson Syndrome, Toxic Epidermal Necrolysis

- u **Spectrum of Disease**
 - u Histologically indistinguishable
 - u **More severe reactions are likely to be drug induced (50% of SJS, 80% of TEN)**

- u **Definitions**
 - u **SJS = less than 10% BSA**
 - u **SJS/TEN overlap = 10-30% BSA**
 - u **TEN = greater than 30% BSA**

SPECTRUM OF DISEASE BASED UPON SURFACE AREA OF EPIDERMAL DETACHMENT

SJS



<10%

SJS-TEN
overlap



10-30%

TEN



>30%

 = Surface area of epidermal detachment  = detached epidermis

SJS = Stevens-Johnson syndrome TEN = Toxic epidermal necrolysis

Associated Medications SJS/TEN

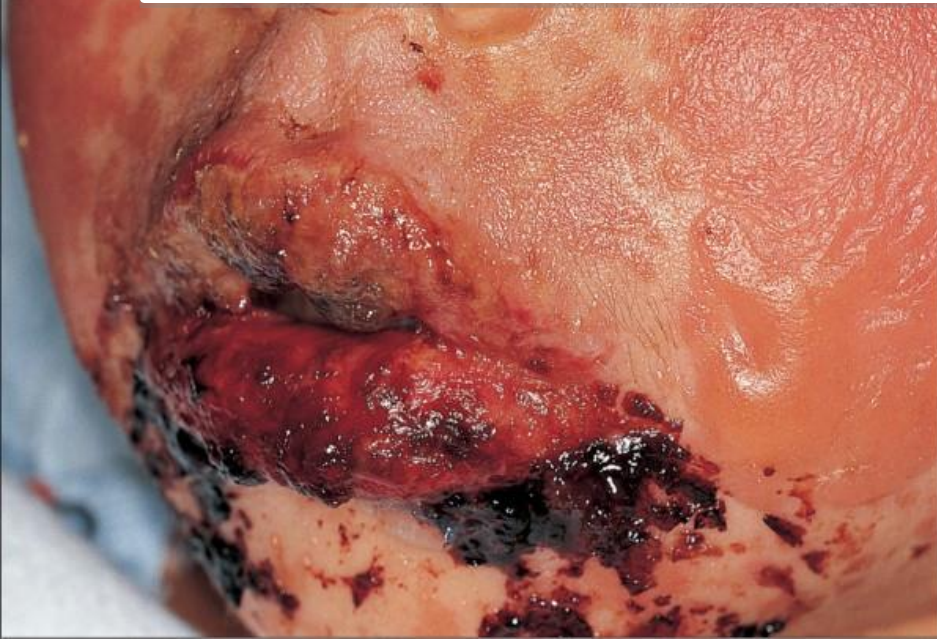
MEDICATIONS MOST FREQUENTLY ASSOCIATED WITH SJS AND TEN

Allopurinol	Lamotrigine
Aminopenicillins	Phenylbutazone*, ³
Amithiozone (thioacetazone)*, ¹	Piroxicam
Barbiturates	Sulfadiazine*, ¹
Carbamazepine	Sulfadoxine*, ¹
Chlormezanone*, ²	Sulfasalazine
Phenytoin antiepileptic	Trimethoprim-sulfamethoxazole

Infections



Mycoplasma common cause of SJS in kids



EM, SJS, TEN

- u Fever, flu-like symptoms *precede* eruption by a few days
- u SJS & TEN: Involvement of 2 or more mucosal surfaces
 - Oral mucosa and conjunctivae most common
 - May be present before rash
- u Stop drug promptly
 - Decreases mortality rate (from 26% to 5%) in drugs with short half lives



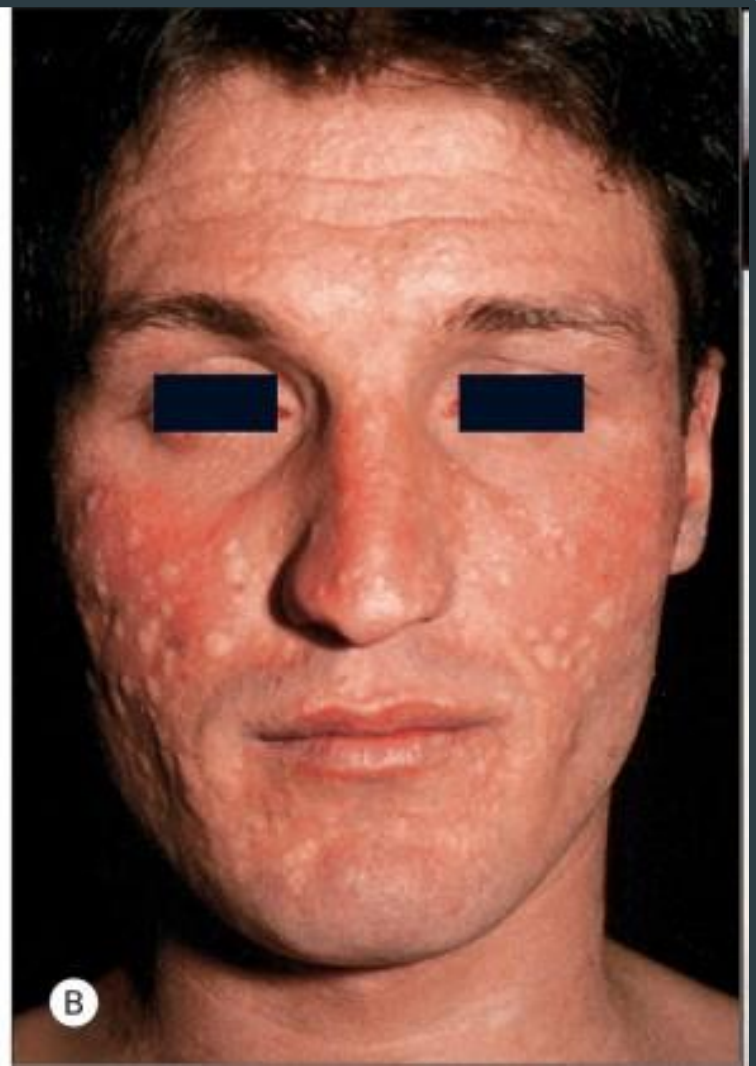
TEN



SJS/TEN

- u Treatment: burn unit
 - Supportive
 - u Fluids, nutrient, electrolyte replacement, infection control
 - IVIG:
 - u Stops keratinocyte apoptosis, blocks death receptor FAS(CD95)
 - Immunosuppressive therapy
 - u Controversial
 - u May increase morbidity/mortality
 - Systemic steroids - early short trial, rapid taper
- u Outcome Measures:
 - Age, severity of underlying disease & extent of skin loss
 - Epidermal regrowth - 3 weeks
 - Ocular scarring and vision loss, nail abnormalities, transient widespread verrucous hyperplasia, confluent SKs
- u Mortality rates: SJS 5%, TEN 30%

TEN (50% body surface area involvement)
before (A) and 3 weeks after (B) treatment with
IVIg (0.75 g/kg/day for 4 days)



Erythema Multiforme



What is the most common infectious cause of erythema multiforme?

What is the most common infectious cause of erythema multiforme?

u HerpesVirus



Infectious

Staphylococcal Infections

- Cutaneous manifestations of endocarditis
- Impetigo of Bockhart
- Sycosis Barbae
- Folliculitis
- Furunculosis
- Pyogenic Paronychia
- Botryomycosis
- Pyomyositis
- Impetigo
- Staph Scalded Skin Syndrome
- Toxic Shock Syndrome



Staphylococcal Infections

- u Skin lesions: pustules, furuncles, erosions with honey-colored crusts, bullae, erythema and desquamation, or vegetating pyodermas
- u Staph is the most common bacterial infection in children
 - u Extremely contagious
 - u Person-to-person contact
- u Endocarditis:
 - u Osler node: painful, erythematous nodule pale center located on fingertips, thenar, and hypothenar eminences
 - u Janeway lesions: non-tender, angular hemorrhagic lesions on palms

Osler nodes



- u Both Janeway lesions & Osler nodes are due to septic emboli
- u Osler nodes are painful
 - u Osler=Ouch

Janeway lesions



Community Acquired MRSA

- u First appeared in isolates of *S.aureus* in 1961
- u Risk factors:
 - u Age > 65
 - u Exposure to MRSA
 - u Prior Abx therapy
 - u Recent hospitalization or chronic illness—IV Vancomycin or Linezolid
- u If MRSA suspected:
 - u Clindamycin
 - u Trimethoprim/sulfamethoxazole (alone or with rifampin)
 - u Minocycline/Doxycycline
 - u Oral linezolid (very expensive)
 - u Empiric Vancomycin in all pts w/ severe, life-threatening infection
- u Colonized Pts of anterior nares with MRSA or with localized impetigo → mupirocin



IMPETIGO CONTAGIOSA

DDx:

- u Tinea corporis (circinate lesions)
- u Toxicodendron/rhus dermatitis
- u Varicella (small, discrete vesicles)
- u Ecthyma (crusted ulcers, not erosions)



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Impetigo Contagiosa

- u Presentation: 2mm erythematous papule develops into vesicles and bullae. Upon rupture a straw colored seropurulent discharge dries to form yellow, friable crust.
- u Etiology: *S. Aureus* > *S. Pyogenes*.
- u Lesions located on exposed parts of body.
- u Group A Strep can cause AGN
 - u Children <6 yrs old
 - u 2% to 5% of infections
 - u Serotypes 49, 55, 57, 60 strain M2 most associated
 - u Good prognosis in children

Toxic Shock Syndrome

u Presentation:

- u Acute, febrile, multi-system disease
- u S. Aureus :
 - u Cervical mucosa historically in early 1980's
 - u Also seen with: wounds, catheters, nasal packing. Mortality 12 %
- u Group A Strep :
 - u Necrotizing fasciitis. Mortality 30%

Toxic Shock Syndrome (TSS)

- u *S. aureus* exotoxin (TSST-1) isolated in 90% of cases
- u *Strep M* types 1 and 3 (80% produce exotoxin A)
- u Acute, febrile, multi-system illness characterized by:
 - u Myalgias, n/v/d, HA, pharyngitis
 - u Rapid progression to shock
 - u Diffuse *scarlatiniform* exanthem starts on trunk & spreads centripetally
 - u Erythema + edema of palms, soles, and mucous membranes
 - u Beau's lines in nails after recovery + Telogen Effluvium

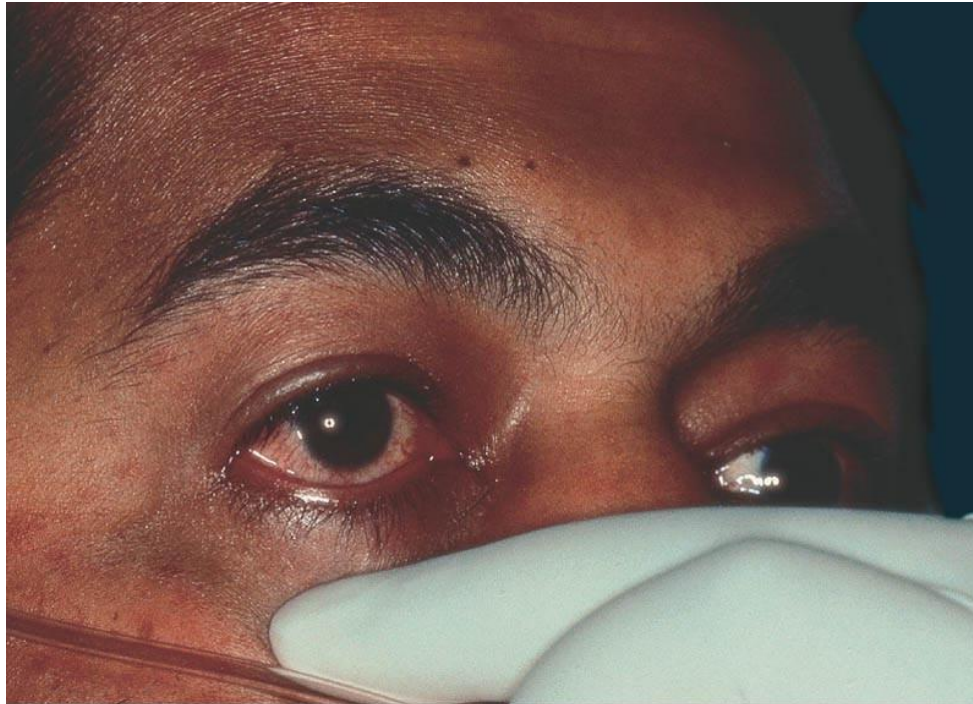


Toxic Shock Syndrome (TSS)

- u CDC diagnostic guidelines...
- u Diffuse macular erythrodermic rash
- u Bulbar conjunctival hyperemia and palmar erythema
- u Temp 38.9 or higher
- u 3 or more organ systems (GI, renal, hepatic, heme, CNS)
- u Desquamation of palms, soles 1-2 weeks after onset (vs SSSS)
- u Negative RMSF, leptospira, rubeola titers
- u Negative blood, urine, CSF cultures
- u Hypotension



Toxic Shock Syndrome (TSS)



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Toxic Shock Syndrome (TSS)

- u Initially isolated from cervical mucosa in menstruating women, but recently from wounds, catheters, diaphragms, nasal packing
- u Mortality of non-menstrual cases higher (12%) compared with menstrual (5%)
- u Rapidly progressive type...
 - u Usually secondary to group A or group B strep (strep M types 1 and 3, 80% produce exotoxin A)
 - u Similarities to staph TSS, except rapidly progressive, soft-tissue destruction... necrotizing fasciitis; case fatality rate of 30%

Toxic Shock Syndrome (TSS)

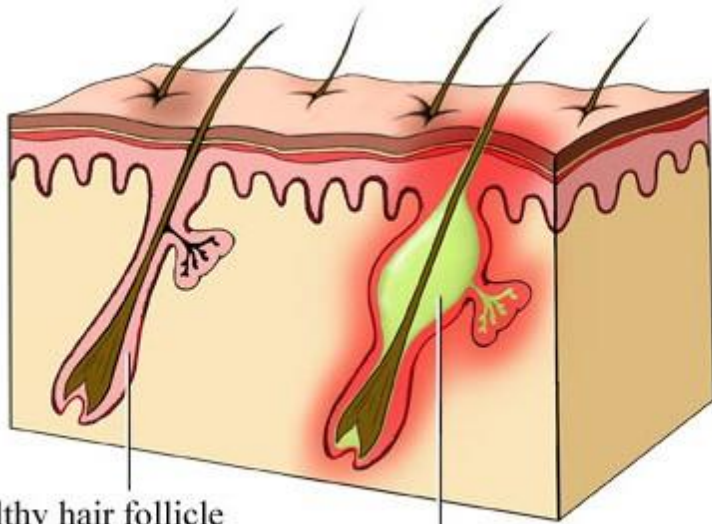
TSS (Staph)

- u TSST-1 toxin
- u Staph enterotoxins
- u Superantigens that promote TNF- α , IL-1, IL-6
- u Clinical:
 - u Perineal erythema, desquamation, strawberry tongue
 - u Tampons (5% mortality rate)
 - u Nonmenstrual cases (surgical packing, catheters, meshes, abscesses); higher mortality (12%)

TSS (Strep)

- u Group A strep (M types 1,3)
- u SPE = pyrogenic
- u Exotoxins A,B,C = superantigens
- u Clinical:
 - u Preceded by **soft tissue infection** (localized extremity pain) 80% of time
 - u High mortality

Folliculitis



- u Superficial (follicle ostium) or deep infection of the hair follicle
 - u Common in AIDS, frequent cause of pruritus
 - u Can occur on eyelashes, pubis (sexual contact, STD), thighs
- u *S. aureus* most common infectious cause
- u *Pseudomonas* assoc. with swimming pools + jacuzzis
 - u Alkaline water & low chlorine content
 - u Bathing suit distribution
- u Other Gram-negatives (*Klebsiella*, *E. coli*, *Enterobacter*, *Proteus*) = implicated in pts on long-term abx therapy for Tx of acne/rosacea

Scarlet Fever

- u Group A β hemolytic strep produces exotoxin
- u Diffuse erythematous exanthem marked by erythematous, (tiny 1-2mm) papular eruption, with a rough, sandpaper quality
- u Occurs during course of strep pharyngitis, 24-48 hrs after onset of pharyngeal symptoms
 - u Also headache, malaise, chills, anorexia, nausea, high fevers
- u Check ASO titer, throat swab
- u Strawberry tongue, with enlarged, exudative tonsils
- u MC between 1-10 years of age



White: early



Red: 4-5th day



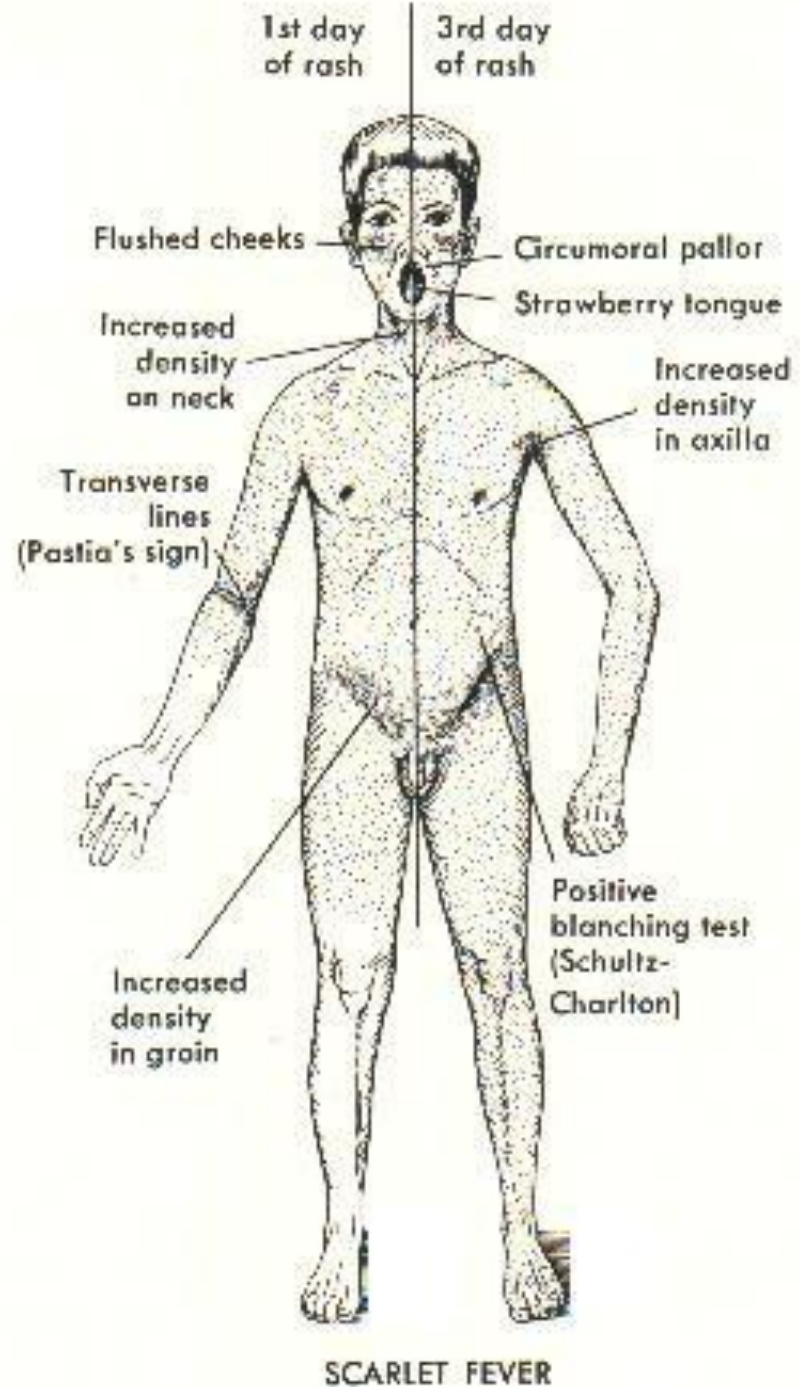
Strawberry tongue, with enlarged, exudative tonsils

Scarlet Fever



Rash with circumoral pallor

Scarlet Fever



Erythema Marginatum

- u Two skin signs among diagnostic criteria for rheumatic fever
 - u Erythema marginatum (early)
 - u Subcutaneous nodules (late)
 - u (carditis, chorea, polyarthritis)
- u Spreading patchy erythema spreads peripherally, polycyclic; evanescent lasting hours to days
- u Lesions asymptomatic



Staphylococcal Scalded Skin Syndrome

Ritter's disease, Pemphigus neonatorum

- u Primarily children < 6y/o
- u Characterized by red, blistering skin 2° a staph infection from distant foci
- u Localized toxigenic strain of *S. aureus*
 - u Naso-oro-pharynx or conjunctiva
 - u Tender, flaccid, **sterile bullae (culture negative)**
 - u *Not at the DEJ as in TEN which exhibits 'full thickness necrosis'*





Staphylococcal Scalded Skin Syndrome

Ritter's disease, Pemphigus neonatorum

- u Prodrome of malaise, fever, irritability, sore throat, & severe tenderness of the skin → Purulent rhinorrhea or conjunctivitis
- u Erythema 1st appears on head, then generalized in 48 hours
 - u Skin develops wrinkled appearance due to flaccid bullae in superficial epidermis → spares palms, soles, mucous membranes
 - u Perioral crusting, mild facial edema
- u Scaling & desquamation continue for 3-5 days w/ re-epithelialization in 10-14 days
 - u Mortality rates: 3% in children, over 50% in adults, 100% in adults w/underlying disease

Staphylococcal Scalded Skin Syndrome

u **Diagnosis:**

- u Cultures from intact bullae are negative (sterile bullae)
- u Culture instead from conjunctiva, nasopharynx, perineum, or pyogenic foci on the skin

u **Treatment:**

- u Inpatient IV penicillinase-resistant antibiotic agents
 - u Nafcillin
 - u If penicillin-allergic → macrolides or aminoglycosides
- u Supportive care such as fluid and electrolyte replacement and local wound care

Comparison of TEN and SSSS

	TEN	SSSS
Cause	Usu. Drug induced	Toxin-producing <i>S. aureus</i> infection
Age	Adults	Infants and young kids
Histology	D-E separation ; dermis w/ variable inflammatory infiltrate	Granular layer split in epidermis; dermis lacks inflammatory infiltrate
Distribution of rash	Areas of sparing	Generalized w/ flexural accentuation
Mucous Membranes	Involved; erosions	Uninvolved
Nikolsky's sign	In some areas; difficult to elicit	Present in seemingly uninvolved skin
Face	Vermilion lip redness; edema, erosions	Perioral crusting and radial fissuring with mild facial swelling
Tx	Standard burn Tx; IVIg, CSt. (controversial)	Abx (B-lactamase resistant) and supportive care

Erysipelas

St. Anthony's Fire, Ignis sacer

- u **Group A *B*-hemolytic *strep***
- u **Group B *strep* in newborns**
- u **Acute infection of dermis & superficial dermal lymphatics**
 - u Local redness, heat, swelling
 - u Raised, indurated border that spreads
 - u Legs and face MC sites (scalp barrier to extension)
- u **Prodromal constitutional Sx:**
 - Lymphadenopathy
 - Leukocytosis > 20,000



Erysipelas

- u Complications:
 - u Septicemia, deep cellulitis, necrotizing fasciitis
- u Differential diagnosis:
 - u Contact dermatitis (plants, drugs, dyes) although not associated with fever/chills, pain
 - u Lupus erythematosus butterfly pattern
- u Treatment:
 - u PCN, erythromycin at least 10 days
 - u Ice compresses
 - u Inpatient and IV Abx



Cellulitis

- u Deep dermal & SQ infection of mainly *Staph. aureus* or *Strep. pyogenes*
- u Suppurative inflammation usually following a wound
 - u MC port-of-entry is due to T. pedis
 - u Purulent and necrotic material will drain
 - u Local erythema, tenderness, malaise
- u Erythema becomes intense rapidly and spreads → Streaks of lymphangitis
- u Risk factors:
 - u DM, Alcoholism, lymphedema, IVDA, PVD
 - u Damage to lymphatic system (vein stripping)



Cellulitis

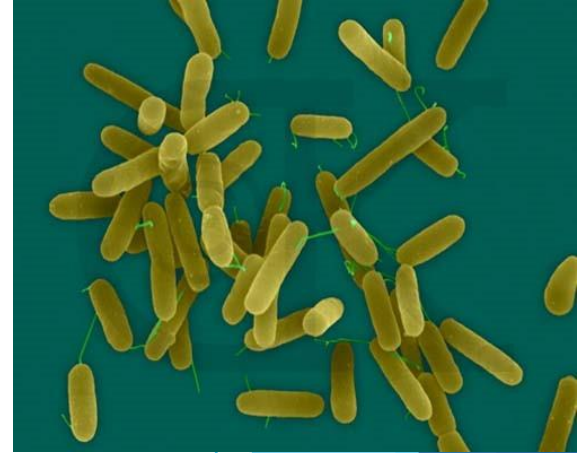
- u Cellulitis is almost always unilateral, if bilateral, think stasis dermatitis
- u Complications (rare in immunocompetent hosts)
 - u Gangrene
 - u Metastatic abscesses
 - u Septicemia
- u Initial therapy **cover *staph* and *strep***:
 - u 1st gen ceph or pcn' ase resistant pcn
 - u Suspect MRSA if unresponsive



Don't be Fooled



Pseudomonas aeruginosa



- u Obligate aerobe, Gram(-) bacillus
- u Can produce blue pigment (pyocyanin) or yellow-green pigment (fluorescein)
- u Produces exotoxin A (role unclear)
- u Widely distributed: water, soil, plant life, animal carriers, dust, sewage
- u Intertriginous areas and moist areas most prone to infection
- u GI reservoir of infection in ICU patients
- u Disease spectrum: paronychia, folliculitis (w/ Abx tx for acne), toe web space maceration, ecthyma gangrenosum, burn superinfections

Pseudomonas aeruginosa

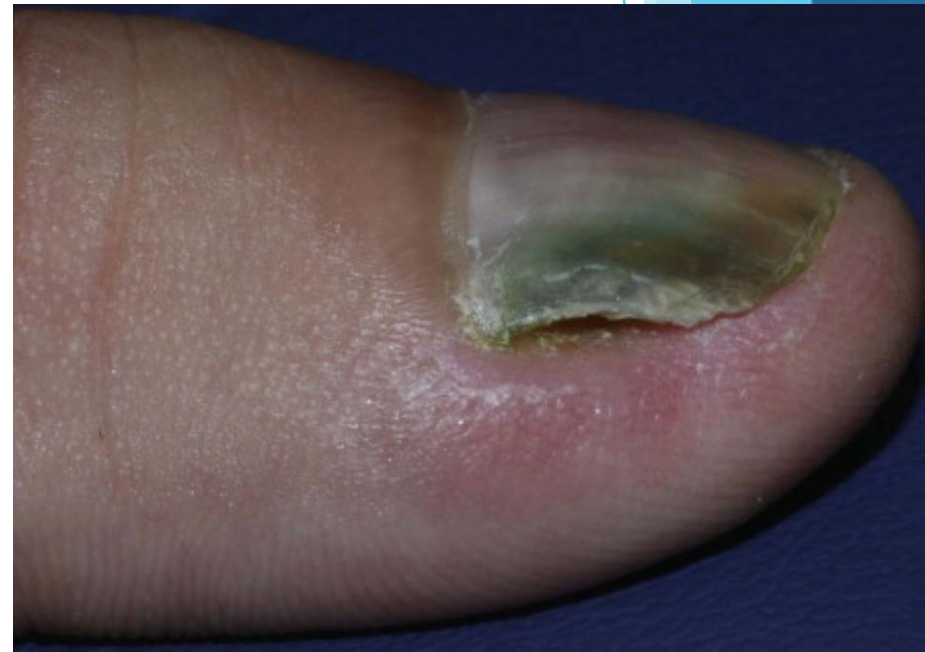
u Ecthyma gangrenosum

- u Bacteremia with skin conditions
- u Debilitated patients (leukemia, burns, chronic granulomatous diz, Ca, neutropenia)
- u Healthy infants after Abx therapy + macerated diaper area
- u Starts as a vesicle → hemorrhagic pustule → necrotic ulcers
- u **MUST** assume **pseudomonal sepsis**
- u DDX: pyoderma gangrenosum, necrotizing vasculitis, and cryoglobulinemia
- u Tx: double coverage
 - u Amioglycosiede + piperacillin
 - u +/- GM-CSF



Pseudomonas aeruginosa

- u Green nail syndrome
 - u Greenish discoloration in areas of onycholysis is due to pigment production:
 - u Pyocyanin: blue
 - u Flourescein: yellow/green
 - u Pyomelinin: black
 - u Seen in people who **chronically** have their **hands in water**
 - u DDX: subungual hematoma, melanocytic nevus, melanoma, Aspirogillus infection
 - u Benzoyl peroxide; 1% acetic acid soaks, debridement



Gram-negative toe web infection



- u Toe-webinfection

- u **Pseudomonas**

- u Occurs in chronically moist areas i.e. wet feet

- u Tx - dry area, vinegar soaks, ciprofloxacin