Dermatology Pearls for the Hospitalist: How to Avoid the Pitfalls

Lindy P. Fox, MD
Assistant Professor
Director, Hospital Consultation Service
Department of Dermatology
University of California, San Francisco
foxli@derm.ucsf.edu

Goals of this lecture

• Drug eruptions
  – Tell the difference between a benign and serious drug eruption
  – Know which drug(s) to stop
• Purpura
  – How to think about it

Goals of this lecture

• Herpes simplex/zoster in the hospital
  – Unusual presentations
  – Appropriate infection control
• Psoriasis
  – How to avoid precipitating a medical emergency
• The red leg
  – How to tell when it’s not cellulitis
• Pyoderma gangrenosum
  – Avoid a potential nosocomial disaster
• Common benign dermatoses in the hospital

I think it’s a drug eruption. Now what do I do?
Drug reactions:
3 things you need to know
1. Type of drug reaction
2. Statistics:
   – Which drugs are most likely to cause that type of reaction?
3. Timing:
   – How long after the drug was started did the reaction begin?

Case
- 46 year old HIV+ man
  - admitted to ICU for r/o sepsis
- Severely hypotensive → IV fluids, norepinephrine
- Sepsis? → antibiotics are started
- At home has been taking trimethoprim/sulfamethoxazole for UTI

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<th>Drug Name</th>
<th>Days</th>
<th>Rash Onset</th>
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Question: Per the drug chart, the most likely culprit is:
- A
- B
- C
- D
- E
- F
- G

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Question: Per the drug chart, the most likely culprit is:
- A
- B
- C
- D
- E
- F
- G
Drug Eruptions: Degrees of Severity

Simple
- Morbilliform drug eruption
- Minimal systemic symptoms

Complex
- Drug hypersensitivity reaction
- Stevens-Johnson syndrome (SJS)
- Toxic epidermal necrolysis (TEN)
- Systemic involvement

Morbilliform (Simple) Drug Eruption
- Begins 5-10 days after drug started
- Erythematous macules, papules
- Pruritus
- No systemic symptoms
- Risk factors: EBV, HIV infection
- Treatment:
  - D/C medication
  - diphenhydramine, topical steroids
- Resolves 7-10 days after drug stopped
  - Gets worse before gets better

Common Causes of Cutaneous Drug Eruptions
- Antibiotics
- NSAIDs
- Sulfa
- Allopurinol
- Anticonvulsants
Simple drug eruption - day 3

Simple drug eruption - day 7

Hypersensitivity Reactions

• Skin eruption associated with systemic symptoms and alteration of internal organs
• “DRESS”: Drug reaction w/ eosinophilia and systemic symptoms
• “DiHS” = Drug induced hypersensitivity syndrome
• Begins 2-6 weeks after medication started
  – time to abnormally metabolize the medication
• May be role for HHV6
• Mortality 10-25%

Hypersensitivity Reactions
Drugs

• Aromatic anticonvulsants
  – phenobarbital, carbamazepine, phenytoin
  – THESE CROSS-REACT
• Sulfonamides
• Lamotrigine
• Dapsone
• Allopurinol (HLA-B*5801)
• NSAIDs
• Other
  – Abacavir (HLA- B*5701)
  – Nevirapine (HLA-DRBI*0101)
  – Minocycline, metronidazole, azathioprine, gold salts
• Each class of drug causes a slightly different clinical picture
Hypersensitivity Reactions

Clinical features

- Rash
- Fever (precedes eruption by day or more)
- Pharyngitis
- Hepatitis
- Arthralgias
- Lymphadenopathy
- Hematologic abnormalities
  - eosinophilia
  - atypical lymphocytosis
- Other organs involved
  - myocarditis, interstitial pneumonia, interstitial nephritis, thyroiditis

Anticonvulsant Hypersensitivity Reaction
Allopurinol Hypersensitivity

Hypersensitivity Reactions Treatment

- Stop the medication
- Avoid cross reacting medications!!!!
  - Aromatic anticonvulsants cross react (70%)
    - Phenobarbital, Phenytoin, Carbamazepine
    - Valproic acid and Keppra generally safe
- Systemic steroids (Prednisone 1.5-2mg/kg)
  - Taper slowly: 1-3 months
- Allopurinol hypersensitivity may require steroid sparing agent
  - NOT azathioprine (also metabolized by xanthine oxidase)
- Completely recover, IF the hepatitis resolves
- Check TSH monthly for 6 months
- Watch for later cardiac involvement (low EF)
Severe Bullous Reactions

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

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

- Medications
  - Sulfonamides
  - Aromatic anticonvulsants (carbamazapine [HLA-B*1502], phenobarbital, phenytoin)
  - Allopurinol (HLA-B*5801)
  - NSAIDs (esp. Oxicams)
  - Nevirapine (HLA-DRB1*0101)
  - Lamotrigine
  - Weaker link: Sertraline, Pantoprazole, Tramadol

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

<table>
<thead>
<tr>
<th>Disease</th>
<th>BSA</th>
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<tr>
<td>SJS</td>
<td>&lt; 10%</td>
</tr>
<tr>
<td>SJS/TEN overlap</td>
<td>10-30%</td>
</tr>
<tr>
<td>TEN with spots</td>
<td>&gt; 30%</td>
</tr>
<tr>
<td>TEN without spots</td>
<td>Sheets of epidermal loss &gt; 10%</td>
</tr>
</tbody>
</table>

Causes:
- Drugs
- Mycoplasma
- HSV

Erythema, bullae
Skin pain
Mucosal membranes ≥ 2

Causes:
- Drugs
Stevens-Johnson Syndrome

- **Incidence**
  - 6 cases per million per year
- **Etiology**
  - Typical drugs
    - NSAIDs, sulfonamides, anticonvulsants, allopurinol
  - Mycoplasma: up to 25% of pediatric patients with SJS
- **Mortality**
  - 5%

Stevens-Johnson Syndrome

- **Prodrome**
  - fever, respiratory symptoms, headache, vomiting, diarrhea
- **Clinical morphology:**
  - Widespread typical targets or
  - Atypical “targetoid” or bullous
    - +/- skin pain, fragility, blisters
  - Two or more mucous membranes involved
A Special Case: Phenytoin + XRT = SJS

Toxic Epidermal Necrolysis

- Incidence
  - 0.4-1.2 cases per million per year in general population
  - 1 case per thousand per year in HIV
- Etiology: almost always a medication
  - NSAIDs, sulfonamide, anticonvulsants, allopurinol
- Mortality up to 25-35%
  - Sepsis, multiorgan failure

SCORTEN

- Criteria
  1. Age > 40 yrs
  2. Presence of malignancy
  3. BUN > 27 mg/dL
  4. Glucose >252 mg/dL
  5. Pulse > 120 bpm
  6. Bicarbonate <20mEq/l
  7. BSA > 10%
- Mortality rates
  - 0-1 3.2%
  - 2 12.2%
  - 3 35.3%
  - 4 58.3%
  - ≥5 90%

Toxic Epidermal Necrolysis

- Prodrome: fever, sore throat, burning sensation in eyes X 1-3 days before skin lesions appear
- Clinical features
  - Flat atypical purpuric targets
  - Lesions become dusky, poorly demarcated, and confluent (>30% BSA)
  - Lesions often blister
  - Nikolsky sign
  - Skin is PAINFUL
  - Often have mucous membrane involvement
Toxic Epidermal Necrolysis

• Systemic involvement can occur
  – GI tract
  – Pulmonary
    • Hypoxemia without chest X-ray abnormalities
    • Bronchial epithelial sloughing
  – Liver
    • LFTs can be abnormal
  – Leukopenia common

Stevens-Johnson Syndrome (SJS)/ Toxic epidermal necrolysis (TEN)
SJS/TEN: Emergency Management

• Stop all unnecessary medications
  – The major predictor of survival and severity of disease
• Ophthalmology consult
• Check for Mycoplasma: 25% of SJS in pediatric patients
• Treat like a burn patient
  – Monitor fluid and electrolyte status (but don’t overhydrate)
  – Nutritional support
  – Warm environment
  – Respiratory care
• Death (up to 25% of patients with more than 30% skin loss, age dependent)

SJS/TEN: Treatment

• Topical
  – Protect exposed skin, prevent secondary infection
  – Aquaphor and Vaseline gauze
• Systemic: controversial
  – No role for empiric antibiotics
    • Surveillance cultures
    • Treat secondary infection (septicemia)
  – Consider antivirals, treat Mycoplasma if present
  – SJS: high dose corticosteroids -1.5-2 mg/kg prednisone (no RCT)
  – TEN: IVIG 1g/kg/d x 4d

Pathogenesis of TEN

Normal skin
Express Fas (CD95)

TEN
Induction of Fas L → Fas: Fas L binding induces widespread apoptosis of keratinocytes

IVIG (intravenous immunoglobulin) as a treatment for TEN

Human IVIG has antibodies against Fas L
IVIGblocks Fas mediated apoptosis in vitro & Arrests development of TEN in vivo
TEN Treated With IVIG

Start IVIG 48 hrs later: no bullae

IVIG for TEN
Dose and Response

- Recommended dose: 0.5-1.0g/kg/d over 3-5 days
- Arrest in disease progression in 24-48 hours
- Complete re-epithelialization within 4-10 days
- Decreases mortality?*
  - Decreases to 6-12% in some studies
  - Other studies report increased mortality
- 7 of 9 studies (non-controlled clinical studies with ≥ 10 pts)
  - Overall mortality benefit of IVIG in doses > 2g/kg*
- Risk factors for failing to respond to IVIG**
  - Delayed use of IVIG (≥ day 10), lower dose (2g/kg total), underlying chronic diseases, higher BSA involved (>65%), older age
- Also batch-to-batch variation in anti-Fas activity

*Semín Cutan Med Surg 2006. 25:91-3  
*Allergology Int 2006. 55: 9-16  
**Arch Derm 2003. 139:26-32

Miscellaneous Drug Eruptions You Should Know About

- Acute generalized exanthematous pustulosis
- Linear IgA bullous dermatosis

Acute Generalized Exanthematous Pustulosis = Pustular Drug Eruption

- Sudden onset (2.5-5d after med started)
- 17% patients have previous history of psoriasis
- Memory T cells produce neutrophil promoting cytokines: IL-3, IL-8 and GM-CSF
- Pinpoint subcorneal pustules on scarlatiniform erythema
- Denudation in intertriginous areas
- Fever, eosinophilia (30%), neutrophilia (90%)
- Completely resolves if offending medication discontinued in ≤ 15 days (I think much sooner)
### Acute Generalized Exanthematous Pustulosis = Pustular Drug Eruption

- **EuroSCAR (97 cases of AGEP, 1009 controls):**
  - Macrolides
  - Ampicillin/amoxicillin
  - Quinolones
  - (hydroxy)chloroquine
  - Sulphonamides
  - Terbinafine
  - Diltiazem
  - No infections found
  - Not associated with personal or family history of psoriasis


### Acute Generalized Exanthematous Pustulosis = Pustular Drug Eruption

- **Antibiotics**
  - β-lactam
  - Macrolides
  - Cephalosporins
  - Quinolones
  - Tetracyclines
  - Other
    - Bactrim
    - Metronidazole
    - Vancomycin

- **Antifungals**
  - Griseofulvin
  - Itraconazole
  - Terbinafine
  - Nystatin

- **Other**
  - Allopurinol
  - Calcium channel blockers
  - Carbamazepine
  - ACE inhibitors
  - Furosemide
  - Thalidomide
  - PUVA
Drug-Induced Linear IgA Disease

- Immune-mediated subepidermal blistering disease
  - Antigen: 97 kDa of BPAG2 (BP180)
  - DIF: band-like (linear) IgA deposition at DEJ

- Clinical features
  - Subepidermal blisters accentuated in flexural areas
  - Morphology: herpetiform or rosette-like

- Can be caused by medications
  - Vancomycin most common

**Common causes**
- Vancomycin
- Penicillins
- Cephalosporins
- Captopril

**Others**
- Amiodarone
- Sulfamethoxazole
- Diclofenac
- Furosemide
- Glyburide
- GCSF
- IFN
- Lithium
- Phenytoin
- Piroxicam
- Rifampin
Oh No! The Patient Has Purpura!
Purpura

- Clinical morphology guides the differential diagnosis
- When fever is present, usually due to systemic inflammatory process or infection

Purpura Definitions

- Purpura = extravasated red blood cells
  - Hemorrhage is an integral part of the lesion and not secondary to inflammation
- Nonpalpable purpura
  - Petechiae - pinpoint spots
  - Macular purpura - larger than pinpoint
- Palpable purpura
  - Palpability implies inflammation damaging vessel
- Retiform purpura
  - Purpura in netlike pattern

Morphology of Purpura

- Petechiae
- Macular purpura
- Palpable purpura
- Retiform purpura
Petechiae

- Platelet Related
- Non-platelet Related

Petechiae- Platelet Related

- Thrombocytopenia
  - Idiopathic thrombocytopenic purpura
  - Leukemia/bone marrow failure
  - Heparin induced thrombocytopenia
  - Thrombotic thrombocytopenic purpura
  - Hemolytic uremic syndrome
  - Disseminated intravascular coagulation (DIC)
  - Drug induced
  - Cirrhosis

- Abnormal platelet function
  - Congenital/hereditary
  - ASA, NSAIDs
  - Thrombocytosis
  - Renal insufficiency

Petechiae- Non-platelet Related

- Valsalva (retching, childbirth)
- Trauma
- Scurvy
- Actinic damage
- Amyloid
- Steroid (topical or systemic) induced atrophy
- Fragility syndromes- Ehlers-Danlos
- Hypergammaglobulinemic purpura of Waldenström
- Infection- early Rocky Mountain Spotted Fever
- Early leukocytoclastic vasculitis

Scurvy

Images courtesy of Timothy Berger, MD
Morphology of Purpura

- Petechiae
- **Macular purpura**
- Palpable purpura
- Retiform purpura

Macular Purpura - Differential Diagnosis

- Thrombocytopenia + infection/inflammation/trauma
- Abnormal platelet function + infection/inflammation/trauma
- Infection
- **Anticoagulant + trauma**
  - DIC
  - Renal or hepatic dysfunction
  - Anticoagulant medications
  - Vitamin K deficiency
- Poor dermal support + trauma
  - Actinic damage
  - Amyloid
  - Steroid-induced atrophy
  - Fragility syndromes: Ehlers-Danlos
  - Trauma
  - Scurvy
- Other
  - Leukocytoclastic vasculitis
  - Hypergammaglobulinemic purpura of Waldenström
  - Emboli (fat, cholesterol)

Thrombocytopenia + Trauma

Linear purpura (=vibex) on upper arm due to blood pressure cuff in thrombocytopenic patient

Steroid induced atrophy, actinic damage, trauma (pneumatic compression device)
Traumatic purpura in patient on warfarin mimicking warfarin skin necrosis

**Anticoagulant + Trauma**

Hypergammaglobulinemic Purpura of Waldenström

- Female, episodic showers of "stinging" macular or palpable purpura
- Biopsy may show leukocytoclastic vasculitis
- Polyclonal hypergammaglobulinemia
- Association with Sjögren Syndrome, SLE, HCV, cryoglobulinemia

Image courtesy of Paul Schneiderman, MD

**Morphology of Purpura**

- Petechiae
- Macular purpura
- **Palpable purpura**
- Retiform purpura

**Palpable Purpura**

**Etiology**

- Idiopathic (45-55%)
- Infection (15-20%)
- Inflammatory diseases (15-20%)
- Medications (10-15%)
- Malignancy (<5%)
Palpable Purpura

- **Immune complex vasculitis**
  - Idiopathic, infection, drug, malignancy
  - IgA vasculitis, Henoch-Schönlein purpura
  - Urticarial vasculitis
  - Hypergammaglobulinemic purpura of Waldenström
  - Bowel-bypass syndrome
  - Mixed cryoglobulinemia
  - Connective tissue disease associated

- **Pauci-immune complex vasculitis**
  - ANCA-associated
  - Microscopic polyangiitis
  - Wegener granulomatosis
  - Churg-Strauss
  - Cocaine (p-ANCA +)
  - Sweet’s syndrome

- **Other**
  - Leukemic vasculitis


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Palpable Purpura “PLUS”

- Size of vessels is a clinical clue to underlying etiology
- Medium-sized vessel involvement leads to dermal/subcutaneous nodules, ulcerations, and/or retiform purpura
- Differential diagnosis
  - Septic vasculitis
  - ANCA-associated vasculitis
  - Mixed cryoglobulinemia
  - Connective tissue disease associated
  - Leukemic vasculitis
  - Polyarteritis nodosa (very rare)
  - More than one process occurring simultaneously

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Septic Vasculitis (Bacterial)

- Meningococcemia
- Gonococcemia
- E. coli
- Klebsiella
- Staphylococcus
- Pseudomonas
- Rickettsia rickettsii (Rocky mountain spotted fever)
- Francisella tularensis
- Acute bacterial endocarditis
  - Osler’s nodes, Janeway lesions

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Meningococcemia (acute)

Image courtesy of Peter Heald, MD
Mixed Cryoglobulinemia

Morphology of Purpura

- Petechiae
- Macular purpura
- Palpable purpura
- Retiform purpura

Retiform Purpura

- Due to vessel occlusion
- Range in size- small (mm) to large (cm)
- Prominent early erythema most likely represents an infectious or inflammatory etiology while lack of erythema suggests microvascular occlusion*
- Fever also more common with infectious and inflammatory causes


Retiform Purpura DDX

- Intravascular
- Vascular
- Thrombotic
- Embolic
Retiform Purpura

Vascular infiltration in vessel wall

- Infectious
  - Bacterial
    - Meningococemia
    - Gonococcemia
    - Staphylococcus
    - E. coli
    - Klebsiella
    - Pseudomonas
  - Fungal
    - Mucor/Rhizopus
    - Aspergillus
    - Candida
    - Fusarium
  - Other
    - Strongyloidiasis
    - Lycia (leprosy)

- Vasculitis
  - IgA vasculitis
  - Connective tissue disease vasculitis
  - Mixed cryoglobulinemia
  - Polyarteritis nodosa
  - Microscopic polyangiitis
  - Wegener’s granulomatosis
  - Churg-Strauss syndrome
  - Calciniphylaxis
  - Oxalosis

Hyperinfection Strongyloidiasis

BAL Specimen - Strongyloides stercoralis filariform larvae

Hyperinfection Strongyloidiasis
Polyarteritis Nodosa

Cutaneous Polyarteritis Nodosa

Cocaine associated PR3+ vasculitis

Calciphylaxis (early)
Calciphylaxis (late)

Retiform Purpura

Emboli

- Clinical
  - Few lesions
  - Small vessel occlusion
  - Acral/distal
  - Post procedure

- Emboli- DDX
  - Cholesterol
  - Cardiac
    - Marantic endocarditis
    - Septic endocarditis
    - Libman-Sachs endocarditis
    - Atrial myxoma
  - Air
  - Fat
    - upper extrem> lower extrem

Retiform Purpura DDX

Vascular

Intravascular

Thrombotic

Embolic

Emboli- Aortic Thrombus
Emboli- Endocarditis

Image courtesy of Peter Heald, MD

Emboli- infected LV thrombus

Retiform Purpura
DDX

Vascular

Intravascular

Thrombotic

Embolic

Retiform Purpura
Thrombotic

- Abnormal coagulation
- Thrombotic vasculopathy
- Platelet Plugging
- Cold-related
- Red cell occlusion
Retiform Purpura
Thrombotic- Abnormal Coagulation

- Classic hypercoagulable states
  - Protein C, S deficiency
  - Antiphospholipid antibody syndrome
- Coumadin necrosis
  - Protein C deficiency/dysfunction
- DIC/Purpura fulminans

Antiphospholipid Antibody Syndrome

Coumadin Necrosis

Protein C Consumption
DIC Image courtesy of Peter Heald, MD

Purpura Fulminans (DIC)

Retiform Purpura
Thrombotic- Thrombotic Vasculopathy

- Livedoid vasculopathy
- Sneddon’s syndrome
- Malignant atrophic papulosis (Degos’ disease)
- Thromboangiitis obliterans (Buerger’s disease)

Livedoid Vasculopathy, ACLA+, R/O Sneddon's syndrome

Retiform Purpura
Thrombotic−Platelet Plugging
- Heparin induced thrombocytopenia/ heparin necrosis
- Thrombotic thrombocytopenic purpura- Hemolytic uremic syndrome
  - Microangiopathy
- Paroxysmal nocturnal hemoglobinuria
- Thrombocytosis
  - Essential thrombocythemia
  - Polycythemia vera
- Hyperviscosity

Heparin Induced Thrombocytopenia

Retiform Purpura
Thrombotic−Other
- Cold-related
  - Cryoglobulinemia (Type I)
  - Cryofibrinogenemia
  - Cold agglutinins
- Red cell occlusion
  - Sickle cell disease
  - Severe hemolytic anemia
Cellulitis

- Infection of the dermis
- Gp A beta hemolytic strep and Staph aureus
- Rapidly spreading
- Erythematous, tender plaque, not fluctuant
- Patient often toxic
- WBC, LAD, streaking
- Rarely bilateral
- Treat tinea pedis

The red leg:
Cellulitis and its (common) mimics

- Cellulitis/erysipelas
- Stasis dermatitis
- Contact dermatitis

Cryoglobulinemia

Image courtesy of Peter Heald, MD
Stasis Dermatitis

- Often bilateral, L>R
- Itchy and/or painful
- Red, hot, swollen leg
- No fever, elevated WBC, LAD, streaking
- Look for: varicosities, edema, venous ulceration, hemosiderin deposition
- Superimposed contact dermatitis common
Contact Dermatitis

• Common causes
  – Applied antibiotics (Neomycin, Bacitracin)
  – Topical anesthetics (benzocaine)
  – Other (Vitamin E, topical benadryl)
• Avoid topical antibiotics to leg ulcers
  – Metronidazole OK (prevents odor)

• Itch (no pain)
• Patient is non-toxic
• Erythema and edema can be severe
• Look for sharp cutoff
• Treat with topical steroids

The Red Leg:
Key features of the physical exam:

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<th>Fever</th>
<th>Pain</th>
<th>Warmth</th>
<th>Bilateral</th>
<th>Streaking</th>
<th>Lymphadenopathy</th>
<th>Elevated WBC</th>
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<tr>
<td>Cellulitis</td>
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<td>Yes</td>
<td>Yes</td>
<td>Almost</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Consider another diagnosis</td>
<td>No</td>
<td>+/-</td>
<td>+/-</td>
<td>often</td>
<td>No</td>
<td>No</td>
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Contact Dermatitis

• Common causes
  – Applied antibiotics (Neomycin, Bacitracin)
  – Topical anesthetics (benzocaine)
  – Other (Vitamin E, topical benadryl)

• Avoid topical antibiotics to leg ulcers
  – Metronidazole OK (prevents odor)
When psoriasis is a life-threatening disease.

Case

- 55 yr old male
- COPD, HTN, non-small cell lung cancer and mild psoriasis
- Presents with low grade fever, shaking chills, and diffuse erythema (erythroderma)
- Meds:
  - ACE inhibitor x 3 months
  - 1 week of pulsed prednisone with rapid taper for COPD flare

Pustular Psoriasis

- Often occurs when known psoriatics are given systemic steroids
- When the steroids are tapered, the psoriasis flares, often with pustules
- Can be life threatening
  - High cardiac output state
  - Electrolyte imbalance
  - Respiratory distress
  - Temperature dysregulation
Psoriasis Aggravators

- Medications
  - Systemic steroids
  - Beta blockers
  - Lithium
  - Hydroxychloroquine
- Strep infections
  - Guttate psoriasis in children
- Trauma
- Sunburn
- Severe life stress
- HIV
  - Up to 6% of AIDS patients develop psoriasis
- Alcohol for some
- Smoking for some

Case

- 67M underwent an elective saphenous vein phlebectomy for asymptomatic varicosities
- 4d post op, he develops erythema around the wound.
- Ulceration continues to expand despite multiple debridements and broad spectrum antibiotics.
- Wound cultures are negative
- 3 weeks later, he is transferred to UCSF and a dermatology consultation is called
- Tmax 104, WBC 22

The flesh eating leg ulcer.
Pyoderma Gangrenosum

- Rapidly progressive (days) ulcerative process
- Begins as a small pustule which breaks down forming an ulcer
- Undermined violaceous border
- Expands by small peripheral satellite ulcerations which merge with the central larger ulcer
- Occur anywhere on body
- Triggered by trauma (pathergy) (surgical debridement, attempts to graft)
Pyoderma Gangrenosum

• 50% have no underlying cause
• Associations (50%):
  – Inflammatory bowel disease (1.5%-5% of IBD patients get PG)
  – Rheumatoid arthritis
  – Seronegative arthritis
  – Hematologic abnormalities (AML)

Pyoderma Gangrenosum

• Workup
  – Skin biopsy for H&E and culture
  – Rheumatoid factor
  – SPEP/UPEP
  – ANCA (ulcers of Wegener granulomatosis can mimic PG)
  – Colonscopy (r/o IBD)
  – Peripheral smear, Bone marrow biopsy (r/o AML)
Pyoderma Gangrenosum

Treatment

• AVOID DEBRIDEMENT
• Refer to dermatology
• Treatment of underlying disease may not help PG
  – Topical therapy:
    • Superpotent steroids
    • Topical tacrolimus
  – Systemic therapy:
    • Systemic steroids
    • Cyclosporine or Tacrolimus
    • Cellcept
    • Thalidomide
    • TNF-blockers (Remicade)

Common Benign Dermatoses in the Hospital

• Miliaria crystallina
• Grovers Disease

Miliaria

• Miliaria refers to sweat duct occlusion
• Common in situations that induce sweating- warm environments, febrile illness, drugs, etc
• Occurs at different levels in the skin
• Miliaria
  – Crystallina- intra or sub stratum corneum
  – Rubra- malpighian layer (intraepidermal)
  – Profunda- rupture if intradermal duct and inflammation
**Miliaria Crystallina**

http://dermatlas.med.jhmi.edu/derm/index

**Grovers Disease (transient acantholytic dermatosis)**

- Sudden eruption of papules, papulovesicles; often crusted
- Mid chest and back
- Itchy
- Middle aged to older men
- Etiology unknown- heat, sweating
- Risk factors: hospitalized, febrile, sun damage
- Transient
- Treatment: topical steroids (triamcinolone 0.1% cream); get patient to move around