Dermatology Pearls for the Hospitalist: How to Avoid the Pitfalls

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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 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
Question:
Per the drug chart, the most likely culprit is:

<table>
<thead>
<tr>
<th></th>
<th>Day -&gt;</th>
<th>-8</th>
<th>-7</th>
<th>-6</th>
<th>-5</th>
<th>-4</th>
<th>-3</th>
<th>-2</th>
<th>-1</th>
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<td>G</td>
<td>trimethoprim/sulfamethoxazole</td>
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</tr>
</tbody>
</table>

Rash onset: Admit day
Drug Eruptions: Degrees of Severity

<table>
<thead>
<tr>
<th>Simple</th>
<th>Complex</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morbilliform drug eruption</td>
<td>Drug hypersensitivity reaction</td>
</tr>
<tr>
<td>Minimal systemic symptoms</td>
<td>Stevens-Johnson syndrome (SJS)</td>
</tr>
<tr>
<td></td>
<td>Toxic epidermal necrolysis (TEN)</td>
</tr>
</tbody>
</table>

Systemic involvement
Potentially life threatening

Common Causes of Cutaneous Drug Eruptions

- Antibiotics
- NSAIDs
- Sulfa
- Allopurinol
- Anticonvulsants
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

Simple drug eruption- day 1
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-DRB1*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 pneumonitis, 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) tapering dose over 1-3 months
- Allopurinol hypersensitivity may require other immunosuppressive therapy
  - E.g. Cellcept
  - NOT azathioprine (also metabolized by xanthine oxidase)
- Completely recover, IF the hepatitis resolves
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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<tbody>
<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>

### Atypical targets
- Mucosal membranes ≥ 2

### Causes:
- Drugs
- Mycoplasma
- HSV

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

<table>
<thead>
<tr>
<th>SJS</th>
<th>TEN</th>
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</thead>
<tbody>
<tr>
<td>Erythema, bullae</td>
<td>Causes: Drugs</td>
</tr>
<tr>
<td>Skin pain</td>
<td></td>
</tr>
<tr>
<td>Mucosal membranes ≥ 2</td>
<td></td>
</tr>
<tr>
<td>Causes:</td>
<td></td>
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<tr>
<td>Drugs</td>
<td>Mycoplasma</td>
</tr>
<tr>
<td>HSV</td>
<td></td>
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Stevens-Johnson Syndrome

• Incidence
  – 6 cases per million per year

• Etiology
  – Typical drugs
    • NSAIDs, sulfonamide, 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
Stevens-Johnson Syndrome (SJS)

Stevens-Johnson Syndrome (SJS)
A Special Case: Phenytoin + XRT = SJS

Stevens-Johnson Syndrome (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

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**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)

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
  – 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
  - SJS: high dose corticosteroids -1.5-2 mg/kg prednisone (no RCT)
  - TEN: IVIG 0.5-1g/kg/d x 4d

Pathogenesis of TEN

<table>
<thead>
<tr>
<th>Normal skin</th>
<th>TEN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Express Fas (CD95)</td>
<td>Induction of Fas L → Fas: Fas L binding induces widespread apoptosis of keratinocytes</td>
</tr>
</tbody>
</table>

Cell Death
IVIG (intravenous immunoglobulin) as a treatment for TEN

Human IVIG has antibodies against Fas L

IVIG blocks Fas mediated apoptosis \textit{in vitro}
&
Arrests development of TEN \textit{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**

*Semin Cutan Med Surg 2006. 25:91-3
^Allergology Int 2006. 55: 9
**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**
  - B-lactam
  - Macrolides
  - Cephalosporins
  - Quinolones
  - Tetracyclines
  - Other
    - Bactrim
    - Metronidazole
    - Vancomycin

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

- **Other**
  - Allopurinol
  - Calcium channel blockers
  - Carbamazepine
  - ACE inhibitors
  - Furosemide
  - Thalidomide
  - Nifedipine
  - 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

Drug-Induced Linear IgA Disease

• 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
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
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)

Anticoagulant + Trauma

Traumatic purpura in patient on warfarin mimicking warfarin skin necrosis
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

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

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

Septic Vasculitis (Bacterial)

- Meningococcemia
- Gonococcemia
- E. coli
- Klebsiella
- Staphylococcus
- Pseudomonas
- Rickettsia rickettsii (Rocky mountain spotted fever)
- Francisella tularensis
- Acute bacterial endocarditis
  - Osler nodes, Janeway lesions
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 (several 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
Vascular- infiltration in vessel wall

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

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

- Calciphylaxis
- Oxalosis

Hyperinfection Strongyloidiasis
Hyperinfection Strongyloidiasis

BAL Specimen - *Strongyloides stercoralis* filariform larvae

Polyarteritis Nodosa

Image courtesy of Timothy Berger, MD
Cutaneous Polyarteritis Nodosa

Calciphylaxis (early)
Calciphylaxis (late)

Retiform Purpura
DDX

- Vascular
- Intravascular
  - Thrombotic
  - Embolic
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

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
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)
Thromboangiitis obliterans

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
Cryoglobulinemia

Herpes Viruses in the Hospital
Herpes Pearls in the Hospital Diagnostic Tests

- **Direct fluorescent antibody (DFA)**
  - Detects both HSV and VZV

- **Viral culture**
  - HSV grows on culture, VZV does not

- **Skin biopsy**
  - Shows virologic changes, but cannot tell HSV from VZV histologically without PCR

NG tube and ET tube “pressure ulcers” are often HSV
HSV in the Immunocompromised Host

- Atypical course
  - Chronic enlarging ulcers
  - Multiple sites
  - Cutaneous dissemination
- Atypical morphology
  - Ulcerodestructive
  - Pustular
  - Exophytic
  - “Verrucous” (usually VZV)

- 38 yo M with AIDS (CD4 4) admitted for cough
- 7 months of painful lesion on right D2 after manicure
- Treated with doxycycline, cephalexin, fluconazole
Case

• 81 yo female bedridden patient admitted for urosepsis
• PMH: bullous pemphigoid on prednisone 5 mg, azathioprine 100 mg
• Called to help manage bullous pemphigoid
Chronic HSV in the Bedridden, Immunosuppressed Patient

Disseminated HSV
Herpes Zoster - Pearls
Herpes Zoster

• Hutchinson’s sign
  – Vesicles on the nasal tip or side suggest nasociliary nerve branch involvement
• Call ophthalmology

Herpes Zoster

• Ramsay Hunt syndrome
  – Vesicles in distribution of the nervus intermedius (external auditory canal, pinna, soft palate, anterior 2/3 of tongue)
  – Associated with vertigo, ipsilateral hearing loss, tinnitus, facial paresis
• Call ENT
Disseminated zoster

• Definition
  – ≥ 20 lesions outside of 2 contiguous dermatomes
• At risk group
  – Immunosuppressed, elderly
• Viscera can be affected
• Treatment
  – Acyclovir 10-12 mg/kg IV q8hr
  – Until lesions are completely healed over (or clear!)
• Contact and respiratory isolation

Herpes Zoster – modes of transmission

• Herpes zoster virus transmitted from person to person by
  1. Direct contact with lesions of varicella OR zoster
  2. Airborne spread from respiratory secretions
  3. Airborne spread from aerosolization of virus from skin lesions
    • Varicella
    • Disseminated zoster
    • Localized zoster (rare)

Herpes Zoster and Isolation-Current Guidelines

• Varicella, disseminated HZ, localized HZ in immunocompromised persons
  – Infected patients
    • Contact and airborne precautions
    • Staff must have prior history of varicella or vaccination
    • Patients isolated in room until lesions crusted or resolved
  – Infected staff
    • Excluded from work until lesions crusted or faded

Am J Infect Control 2007; 35:S65-164

Herpes Zoster and Isolation-Current Guidelines

• Localized HZ in immunocompetent
  – Infected patients
    • Contact precautions
    • Cover lesions (isolation in rooms not required)
  – Infected staff
    • Cover lesions and should be removed from direct patient care of patients with high risk of severe complications from varicella for the duration of rash

Am J Infect Control 2007; 35:S65-164
Herpes Zoster – How infectious is it really?

• Transmission of nosocomial varicella (3 cases: 1 HCW, 2 residents) from an index case with herpes zoster in a single long-term-care facility
  – J Infectious Diseases 2008; 197:646-53

• Varicella-zoster virus DNA found in the saliva of patients with herpes zoster
  – Possible aerosolization of virus from respiratory tract in patients with localized HZ!
  – J Infectious Diseases 2008; 197:654-7

Herpes Zoster– do we need to change current practice?

• HZ lesions are infectious, even when covered
• Virus may aerosolize from the skin or the respiratory tract of patients with HZ
  – Isolation and respiratory precautions in all patients with HZ?

The red leg: Cellulitis and its (common) mimics

- Cellulitis/erysipelas
- Stasis dermatitis
- Contact dermatitis

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
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

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

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

Avoid topical antibiotics to leg ulcers
- Metronidazole OK (prevents odor)
### The Red Leg: Key features of the physical exam:

<table>
<thead>
<tr>
<th></th>
<th>Fever</th>
<th>Pain</th>
<th>Warmth</th>
<th>Bilateral</th>
<th>Streaking</th>
<th>Lymphadenopathy</th>
<th>Elevated WBC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cellulitis</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Almost never</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Consider another diagnosis</td>
<td>No</td>
<td>+/-</td>
<td>+/-</td>
<td>often</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

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
The flesh eating leg ulcer.

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
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)
  – Colonoscopy (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