Cutaneous Disorders
What We’ll Discuss

- A little background
  - Types of lesions
    - Be able to describe or recognize a description
  - Treatment modalities

- Types of skin lesions as laid out in the basic standards
Primary Lesions

- **Macules**
  - Flat, nonpalpable, small area (< 2cm) of color change

- **Patch**
  - Large macule (> 2cm)

- **Papule**
  - Small (< 1.5 cm) palpable mass

- **Nodule**
  - Papule > 1.5 cm, may be solid or cystic

- **Plaque**
  - Palpable > 1.5 cm lesion with flat top
Primary Lesions

- **Vesicle**
  - Fluid filled papule < 1 cm
- **Pustule**
  - Vesicle filled with pus
- **Bulla**
  - Vesicle > 1 cm
- **Wheal**
  - Smooth pink papule caused by localized edema
Secondary Lesions

- **Scale**
  - Abnormal build up of keratinized epithelium

- **Crust**
  - Dry plasma proteins, results from inflammation
Secondary Lesions

- Fissures
  - Deep cracks in dry skin surfaces, extends into the dermis

- Erosions
  - Disruption of the surface epithelium, inflammatory; traumatic
  - Excoriation
Secondary Lesions

- **Hyperpigmentation**
  - Increase in melanin containing epidermal cells; often due to chronic disease

- **Lichenification**
  - Abnormal dense layer of keratinized epidermal cells
Treatment Basics

- If it’s dry wet it and if it’s wet dry it
  - Emollients: Moisturizers
  - Ointments: Moisturize
  - Wet dressings with tap water or NS for weeping or open areas
Systemic Corticosteroids

- Consider for:
  - Urticaria, angioedema, toxicodendron dermatitis, contact/allergic dermatitis

- Definitely for:
  - Erythema multiforme, toxic epidermal necrolysis, vasculitis
Topical Corticosteroids

- Tachyphylaxis—decreased responsiveness to drug due to enzyme mediated events

- All patients should have a 7 day free period in prolonged treatment (2 wk on 1wk off)
Topical Corticosteroids

- Potency is an indication of ability to produce vasoconstriction
  - Group 1 most potent, 7 least (consider table)
  - Fluorination increases potency but also increases risk of adverse reactions
    - Contra-indicated in pregnancy
Topical Corticosteroids

- **Vehicle:**
  - This effects absorption

- **How much to prescribe?**
  - BSA (use rule of 9’s like a burn) and multiply by 30 = single application
    - 9% surface area rash: 0.09x30=2.7; if TID application would need 9g/day
Antihistamines

- 1st generation
  - Diphenhydramine and hydroxyzine

- 2nd generation
  - cetirizine, fexofenadine, loratadine
    - Less frequent dosing
    - Less sedative effect
    - Cost more
  - Discourage topical
    - Too easy to overdose as absorption not consistent

- H-2 antagonists also may be helpful in allergic-mediated events
  - Ranitidine, famotidine
Antifungal Agents

- **Imidazole class**
  - Clotrimazole, miconazole, ketoconazole
  - Effective against yeasts and dermatophytes

- **Polyenes**
  - Nystatin, amphotericin B
    - Only effective against candida

- Treat for 2-4 weeks to prevent recurrence
Atopic Dermatitis
Atopic Dermatitis

- **Etiology/Pathophysiology**
  - Interaction of multiple factors
    - Genetics
    - Environmental
      - Improves in summer, flares in winter
      - Foods may trigger exacerbation
    - Immunologic
- **Acute and chronic types**
  - Frequent recurrences throughout life
Atopic Dermatitis

- **Acute**
  - Poorly defined erythematous patches, papules and plaques
    - Scale may be present
  - Erosions may develop due to scratching
  - Age of onset in childhood
    - 60% within first 2 months of life
Atopic Dermatitis

- Extremely pruritic
  - Develop a scratch-itch cycle that exacerbates the problem
Chronic Atopic Dermatitis

- Lichenification
- Painful fissures
- Eye changes
  - Alopecia of lateral eyebrow
  - Increased pigmentation
  - Infraorbital fold in eyelids
    - Dennie-Morgan sign
Treatment of Atopic Dermatitis

- May see spontaneous remission in up to 40%
- Patient education about need to avoid scratching/rubbing
- Application of wet dressings
- Emollients
- Glucocorticoids
  - Topical low to mid-potency
  - Systemic only for severe, intractable exacerbations
Treatment of Atopic Dermatitis

- Oral antihistamines

- Antibiotics
  - Topical for mild infection/impetigo
  - Oral antibiotics if severe or if MRSA by culture
Contact Dermatitis
Contact Dermatitis

**Irritant type**

- Exposure to chemical or physical agent capable of irritating skin
- Most common form of occupational skin disorders
- Common etiologic agents
  - Abrasives
  - Cleaning agents
  - Plants
  - Animal enzymes
  - Dessicant powders
  - Excessive exposure to water
- May occur minutes after exposure or after delay of up to 24 hours
Irritant Contact Dermatitis

- **Distribution**
  - Isolated to areas that contacted the irritant

- **Treatment**
  - Heals within 2-6 weeks of removal of noxious agent
  - Avoid the irritant!!
  - Wet dressings (Burrow’s solution) q2-3h
  - Glucocorticoids
    - Topical Class I
    - Prednisone for 2 weeks then taper for severe cases
Allergic Contact Dermatitis

- Etiology/Pathophysiology
  - Uncommon in young children and patients > 70 years old
  - Delayed, cell-mediated hypersensitivity reaction
  - Common allergens that precipitate this disease
    - Nickel Sulfate
    - Neomycin sulfate
    - Fragrance mixes
    - Bacitracin
    - Rhus genus plants
    - Rubber compounds
Eruptions usually starts 48 hours or more after contact
- Crescendo effect—worse eruptions with repeated exposures
- May see “acute illness” syndrome with severe cases, esp poison ivy

Distribution
- Initially confined to area of contact
  - Plant dermatitis often linear
Allergic Contact Dermatitis

**Treatment**
- Remove offending agent
- Stop topical creams except for steroids
  - May cause second allergy
- Wet dressings every 2-3 hours
- Glucocorticoids
  - Topical Class I-III
  - Oral prednisone if severe or exudative
    - Start at 70mg for adults and taper over 2 weeks
Psoriasis
Psoriasis

- **Etiology/Pathophysiology**
  - Hereditary disorder
  - Trigger factors
    - Physical trauma (Koebner’s phenomenon)
    - Infections
      - Acute streptococcal infection-guttate psoriasis
    - Stress
    - Drugs
Psoriasis

- Rash Appearance
  - Classic lesions
    - Sharply marginated erythematous papule with silvery-white scale
      - Auspitz’s sign
    - Papules grow into sharply marginated plaques with lamellar scaling
    - Plaques coalesce to form polycyclic or serpiginous patterns
Psoriasis

- Distribution
  - Elbows/Knees
  - Sacral-gluteal
  - Scalp
  - Palms/soles
  - Trunk (Guttate)
Psoriasis

- Nails
  - Pitting
  - Subungual hyperkeratosis
  - “Oil spots”
    - Yellow-brown spots under the nail plate
  - Pathognomonic for psoriasis
Psoriasis

Treatment

- Should be under care of dermatologist!!!!!!!!!!!!
- Heavy duty topical steroids
- Intralesional injections of steroids
- Immunosuppressants
  - Methotrexate
  - Cyclosporine
Abscesses
Abscesses

- **Etiology/Pathophysiology**
  - Breakdown of normal protective barrier with contamination by local flora
  - 1-2% of all presenting complaints to ED

- **Scalp, trunk, extremities**
  - S. aureus most common
  - S. epidermidis and S. hominis

- **Skin near oral or nasal mucosa**
  - Streptococci
Abscesses

- Intertrigenous and perianal areas
  - Gram-negative anaerobes
    - E. coli, P. mirabilis, Klebsiella spp
- Axillae
  - P. mirabilis
- Abscess due to foreign body
  - S. aureus
- IV drug abuse
  - Mixed infections, anaerobes predominate
    - Peptostreptococcus
    - Staphylococcus and streptococcus
Treatment of Abscesses

Incision and drainage

- All that is needed for superficial and localized infection
- Antibiotics controversial
  - No EBM to support use
  - Consider if immunocompromised
  - Consider if fever
  - Consider if surrounding cellulitis
- Prophylactic antibiotics pre-procedure for those predisposed for endocarditis
Cellulitis/Lymphangitis

- **Etiology/Pathophysiology**
  - Bacterial invasion of skin
  - Elderly, immunocompromised or those with peripheral vascular disease
  - Staphylococci and streptococci most common in adults
    - H. influenzae most common in children
  - **Difficult to isolate causative agent**
    - Needle aspiration < 10% successful
    - Punch biopsy < 20% successful
Cellulitis/Lymphangitis

- **Appearance**
  - Pain, induration, warmth and erythema
  - Look for lymphangitis/lymphadenitis
    - Indicate more serious infection
  - High fever/chills suggestive of bacteremia
Treatment Cellulitis/Lymphangitis

- Outpatient
  - Simple infection in otherwise healthy adult
    - Dicloxacillin
    - Macrolides
    - Amoxicillin-clavulanate
    - MRSA regimen
  - Follow-up within 2-3 days
Treatment Cellulitis/Lymphangitis

- **Inpatient**
  - Cellulitis of head and neck
    - First generation cephalosporins
    - Penicillinase-resistant penicillins
    - Consider MRSA
  - Diabetics
    - Second or third generations cephalosporin
    - Imipenem, miopenem or trovafloxacin if severe
    - Consider MRSA
  - Evidence of bacteremia
Impetigo
Impetigo

- Etiology/Pathophysiology
  - Superficial infection caused by Group A Streptococcus or S. aureus
  - More common in young children
    - More common in warmer weather
  - If the causative agent is Streptococcal, may develop glomerulonephritis later
Impetigo

- Rash Appearance
  - Red papule that progresses to vesicle than pustule
  - Pustules breakdown and coalesce to form honey-colored crust
- Distribution of lesions
  - Face, especially around nose and mouth
  - Lower extremities
Impetigo

- **Treatment**
  - Increasing penicillin resistance
  - Dicloxacillin or cephalaxin
  - Topical mupirocin ointment effective
Secondary Syphilis
Secondary Syphilis

- **Etiology**
  - Treponema pallidum infection

- **Rash appearance**
  - 6-8 weeks after initial skin chancre resolves, see a generalized macular exanthema
    - This resolves over weeks
  - Recurrent maculopapular or papular eruptions occur and resolve over months to a year
Secondary Syphilis

- Distribution
  - Trunk
  - Head, especially hairline
  - Neck
  - Palms/soles
Secondary Syphilis

- Treatment
  - Benzathine penicillin G 2.4 million units single dose
  - Doxycycline 100mg BID for 2 weeks in penicillin allergic
Rocky Mountain Spotted Fever
Rocky Mountain Spotted Fever

- **Etiology**
  - Infection by *Rickettsia rickettsii* via tick vector

- **Rash appearance**
  - Discrete macules and maculopapules
    - Blanchable
  - Evolve into petechiae over 2-4 days
Rocky Mountain Spotted Fever

- **Distribution**
  - Begins on wrists and ankles
  - Moves centrally
  - Involves palms/soles

- **Treatment**
  - Doxycycline 100mg BID for 7 days
  - Chloramphenicol is alternative
    - Many toxicities
Meningococcemia
Meningococcemia

- Rash appearance
  - Classic:
    - Petichiae that become palpable with gray necrotic centers
  - Purpura fulminans
    - Seen in patients with shock and DIC
    - N. meningitides is most common, but not only cause

- Distribution
  - Trunk/Extremities
  - Palms/soles
  - Mucous membranes

- Treatment
  - Antibiotics ASAP
    - Ceftriaxone
  - Add vancomycin if any suspicion of streptococcal infection
Toxic Shock Syndrome

- Infection with toxin producing Staphylococcus aureus
  - Tampon use
  - Nasal packing
  - Body art and piercings
  - Nonsurgical skin lesions

- Causes circulatory shock
Toxic Shock Syndrome

- Rash:
  - Nonpruritic, blanching erythroderma
    - Resolves in 3-5 days
    - Desquamation hands/feet in 5-15 days
  - Conjunctival and mucosal hyperemia
  - Petechiae
  - Alopecia
  - Fingernail loss
Toxic Shock Syndrome

- Aggressive management of shock
  - Fluids
  - Pressors
- Seek out focus of infection
- Antimicrobial agents
  - Do not alter outcome, does decrease recurrence rate
  - Methylprednisolone
    - Improvement in some cases
  - IV immunoglobulin
    - Improvement in some cases
Candida

- **Etiology/Pathophysiology**
  - Most commonly due to Candida albicans
  - Antibiotic therapy frequent trigger
    - Increases candidal load
  - Other hosts factors that increase susceptibility
    - Immunocompromise
    - Diabetes mellitus
    - Obesity
    - Hyperhydrosis
    - Systemic/topical glucocorticoids
Candida

- Occluded areas such as skin folds and under diapers, between fingers/toes

- Pustules on erythematous base that become confluent
  - Sharply demarcated
  - Satellite lesions
Candida

- **Treatment**
  - Keep area dry
  - Topical antifungals
    - Consider steroid preps short-term to speed resolution of symptoms
  - Oral agents if resistant to above
    - Fluconazole
    - Itraconazole
    - Ketoconazole
Tinea Pedis

- Contact with spore walking in bare feet
- Erythema, scaling and maceration
  - May see bullae
- May spread to other areas of body
- Interdigital or moccasin type distribution
- May see superinfection

Treatment:
- If macerated, Burrow’s wet dressings
- Topical antifungals
- Oral antifungals for
  - Moccasin type
  - Nail involvement
Tinea Cruris

- Usually associated with tinea pedis
- Large, scaling, well-demarcated red-brown plaques with central clearing
- May see papules or pustules at margins
- Rarely involves scrotum or penis

Treatment
- Topical antifungal agents
- Consider oral if also have tinea pedis or follicular component
Tinea Corporis

- Autoinoculation from tinea pedis or capitis
- Contact with contaminated animals or soil
- Scaling, sharply-margined plaques
  - Enlarge peripherally and clear centrally
    - Annular configuration

- Treatment generally with topical agents
Tinea Capitis

- Person-person, animal-person transmission via fomites
  - May see epidemics
- Hair breaks off just above scalp
- Kerion
  - Inflammatory tinea capitis
  - Boggy, purulent inflamed nodules and plaques
  - Painful
  - Hair falls out rather than breaking off
- Wood’s lamp not always helpful
  - T. tonsurans is # 1 cause in US and does NOT fluoresce
Tinea Capitis

Treatment

- Griseofulvin still considered drug of choice, but some evidence that other oral agents may work as well
- Prednisone 1mg/kg/day for 2 weeks for painful kerion
- Systemic antibiotics if suspect superinfection
Pityriasis (tinea) Versicolor
Pityriasis (tinea) Versicolor

- Superficial overgrowth of hyphal form of Malassezia furfur
  - Aka P ovale or P orbiculare
- Well-demarcated scaling patches, round or oval in shape
  - Variable pigmentation
  - Usually on trunk
- Can confirm diagnosis with KOH prep
- Treatment
  - Selenium sulfide or ketoconazole shampoo
  - Azole creams
  - Oral antifungals not FDA approved for this
Parasitic Infections

- Pediculosis
- Scabies
Scabies
Scabies

- **Etiology/Pathophysiology**
  - Infestation with *Sarcoptes scabei*
  - Person to person transmission through close contact
  - Extremely pruritic!

- **Rash Appearance**
  - Burrows are pathognomonic
Scabies

- Distribution of lesions
  - Hands and feet
    - Burrows in web spaces of fingers
  - Flexural surfaces of elbows and knees
  - Umbilicus
  - Groin and genitals
  - Usually spares face except in infants
Scabies

- **Treatment**
  - Permethrin and lindane are equally effective
  - Leave on overnight and rinse off in AM
  - Lindane neurotoxic - contraindicated in
    - Infants
    - Children
    - Pregnant females
- **Oral Ivermectine**
Herpes Simplex
Herpes Simplex

Etiology/Pathophysiology

- Transmission via contact of infected secretions with mucous membranes or open skin

- HSV 1 causes oral or on occasion genital lesions
  - May cause gingivostomatitis in children < 5 years of age
  - Recur in 60-90%

- HSV2 is primarily spread sexually
  - Small threat of intrauterine transmission—should start oral acyclovir in pregnant patients

- Herpetic whitlow
  - Either HSV 1 or HSV 2
Herpes Simplex

- **Rash Appearance**
  - Small, thin walled vesicles clustered on an erythematous base

- Lesions are generally painful

- **Finger involvement:**
  - Vesicles tend to coalesce and may look pustular
  - Do NOT I+D!
Herpes Simplex

- **Treatment**
  - **Oral HSV**
    - Acyclovir may decrease duration if begun within first 72 hours
    - Oral acyclovir useful in recurrent outbreaks
    - Topical penciclovir shortens duration of symptoms
    - Consider prophylactic use of acyclovir for severe or frequent recurrences
  - **Genital**
    - Oral antiviral agents
Herpes Zoster
Herpes Zoster

- Etiology/Pathophysiology
  - Reactivation of varicella zoster virus
    - Human herpes virus 3

- At risk for dissemination
  - Lymphoma
  - Leukemia
  - Diabetes
  - Other immunocompromise
Herpes Zoster

- Rash Appearance
  - Clusters of vesicles on erythematous base
  - Dermatomal distribution
  - Usually do not cross midline
  - Vesicles rupture and crust as in chicken pox
Herpes Zoster

- Distribution of lesions
  - Thoracic and lumbar dermatomes most common
  - May involve cranial nerves
  - **If tip of nose** involved look for corneal involvement
    - Hutchinson sign
    - Ophthalmology consult if corneal lesions
  - **If ear canal** involved may see facial paralysis
    - Ramsay Hunt Syndrome
Herpes Zoster

- **Treatment**
  - Oral antivirals have been shown to decrease the duration of post-herpetic neuralgia
    - Best if started within first 72 hours of disease
  - Corticosteroids may improve quality of life indices in elderly
    - 21 day taper beginning at 60mg/day
  - Systemic analgesia
    - Amitryptiline or carbamazepine may help
    - IV acyclovir is required for disseminated disease
Molluscum Contagiosum
Molluscum Contagiosum

- **Etiology/Pathophysiology**
  - Molluscum contagiosum virus
    - A poxvirus
    - Skin to skin contact for transmission
  - Self-limited epidermal infection
  - May see hundreds of lesions in patients with HIV

- **Rash Appearance**
  - Skin-colored papules
    - Often [umbilicated](#)
Molluscum Contagiosum

- **Distribution of lesions**
  - Any site of exposed skin
    - Genital if sexually transmitted
    - Face in HIV positive patients

- **Treatment**
  - Usually resolves spontaneously within 6 months
  - Does not improve with HAART therapy in HIV positive patients
  - 5% imiquimod cream
  - Surgical removal
    - Curettage, cryotherapy, cautery
Rubella-”German Measles”

- **Salmon pink macules** and papules that start on forehead and spread inferiorly during first day
- Exanthem fades completely by third day
- **Forchheimer’s sign**
  - Petichiae on soft palate
Measles

- Rash generally starts on 4th febrile day
- Erythematous macules and papules on face
  - Start at hairline and behind ears and spread centrifugally
  - Reach feet by day 3
  - Involves palms and soles
Measles

- **Koplik’s spots** *(pathognomonic)*
  - Appear before exanthema
  - Clusters of blue-white spots on erythematous base on buccal mucosa opposite premolars
Hand, Foot and Mouth Disease

- Coxsackie viruses and other enteroviruses
- Highly contagious
- Painful ulcerative oral lesions
- Macules and papules that evolve to vesicles
  - Palms and soles
  - Fingers and toes
  - Buttock
Erythema Infectiosum

- Human parvovirus B19
- Edematous, confluent plaques on malar face
  - “Slapped cheek” appearance
- Nonfacial lesions appear later
  - Erythematous macules and papules that become confluent
    - Lacy or reticular appearance
    - Extensor surfaces of extremities, trunk and neck
Pityriasis rosea
Pityriasis rosea

- Suspect Human herpes virus 7
- Rash Appearance
  - Herald patch occurs in 80%
- Distribution of lesions
  - “Christmas tree” distribution
  - Follows lines of cleavage on trunk
- Treatment
  - Oral antihistamines
  - Consider topical or oral glucocorticoids
  - May improve with UVB phototherapy or sunlight
Purpura and Petichiae

- Causes
  - Trauma
    - Forceful vomiting
    - Prolonged tourniquet
  - Infectious vasculitis
  - Meningiococcemia (see above)
Purpura and Petichiae

- Platelet dysfunction or thrombocytopenia
  - Immune
    - Collagen vascular disorders (SLE)
    - Leukemia
    - Lymphoma
    - Postinfectious states, e.g., rubella, rubeola
    - Post-transfusional
    - Idiopathic
Purpura and Petichiae

- Hemolytic-Uremic Syndrome
- Dilution (transfusion)
- Drug induced
  - Coating of platelet by drug for an antigenic complex
    - Digoxin, quinine, quinidine, heparin; aspirin, and phenytoin
Purpura and Petichiae

- Henoch-Schonlein purpura
  - Small vessel vasculitis
  - Purpura, arthritis, abdominal pain and hematuria
  - Pinkish maculopapules that evolve into palpable petichiae and purpura
    - Propensity for lower trunk, buttock and legs
  - Role of the ED physician is to make the diagnosis
Urticaria
Urticaria

- Etiology/Pathophysiology
  - IgE mediated
    - Type I anaphylactic hypersensitivity
  - Often due to food/drugs
    - Complement mediated
  - Serum sickness
    - Autoimmune
  - Environmental allergens
    - May see anaphylaxis
  - Physical causes
    - Cold, water, solar, pressure
Urticaria

- Rash Appearance
  - Wheals
    - Well demarcated edematous papules and plaques
  - Angioedema
    - Edema extends into subcutaneous tissue
    - Less well defined
- Distribution
  - At site of contact/trauma or generalized
Urticaria

- Treatment
  - Remove offending agents/triggers
  - H1 blockers
    - Hydroxazine, loratidine, diphenhydramine
  - H2 blockers
    - Cimetidine
  - Mast cell stabilizers
  - Glucocorticoids
Mongolian Spot
Mongolian Spot

- Usually in Asian population
- Most commonly lumbosacral
- Treatment
  - None
- May be mistaken for bruise/abuse
Lichen Planus
Lichen Planus

- **Etiology/Pathophysiology**
  - Idiopathic, but cell-mediated immunity plays a role

- **The 4 “p”s**
  - Papular, Polygonal, Purple, Pruritic

- **Distribution of lesions**
  - Wrists, Lumbar, Shins, Scalp, Glans of penis, Mouth

- **Treatment**
  - Topical or systemic glucocorticoids
  - Cyclosporine
  - Systemic retinoids
Erythema Multiforme
Erythema Multiforme/ Etiology

- Continuum from EM minor to EM major to Stevens-Johnson Syndrome
- Incidence highest in young adults (20-40 y/o)
- Males twice as often as females
- More common in spring and fall
Stevens Johnson Syndrome

- SJS has mortality of 10% in spite of aggressive therapy
  - Secondary infection is most common cause of death
  - May also see fluid/electrolyte imbalance
Erythema Multiforme

- Hypersensitivity reaction
  - May see recurrence with repeated exposures
  - 75% of children with EM due to Herpes simplex infection
  - Triggers (non-identified in 50%)
    - Infectious
      - Mycoplasma
      - Herpes simplex
    - Drugs
      - Anticonvulsants
      - Antibiotics
    - Malignancy
  - Malaise, fever, myalgias, arthralgias
  - Diffuse pruritis or skin burning may be present before rash
Erythema Multiforme

- **Rash Appearance**
  - Variable (multiforme)
  - Maculopapular that evolve to **target (iris) lesions** over 24h
    - Central area may become cyanotic, purpuric or vesicular
  - May see urticarial plaques
  - May develop painful and pruritic deep vesiculbulous lesions
  - Especially on mucous membranes

- **Distribution of lesions**
  - Symmetrically, dorsal hands and feet
  - Extensor surfaces of extremities
  - Mucous membranes
  - Ocular in 10% of EM and 75% of SJS
Erythema Multiforme/Treatment

- Outpatient for EM
- Admit if (consider burn unit)
  - Extensive disease
  - Systemic toxicity
  - Mucous membrane involvement
- Systemic steroids
  - Symptomatic relief but no proven benefit in duration or outcome
- Systemic analgesics
- Antihistamines
- Diphenhydramine and viscous lidocaine rinse for stomatitis
- Burow’s solution for blisters
- Consider prophylactic acyclovir to decrease recurrence of HSV
Toxic Epidermal Necrolysis

- **Etiology/Pathophysiology**
  - Some consider this to be a continuation of EM and SJS
  - No predilection of sex
  - Most common trigger is drugs
    - Sulfa and PCN
    - Anticonvulsants
    - Oxicam NSAIDS
  - Malignancy
  - HIV
Toxic Epidermal Necrolysis

- Warm erythema starting at eyes, nose, mouth and genitalia then generalizes
  - Becomes tender and confluent
- Develop flaccid, ill-defined bullae
  - + Nikolsky sign
- Epidermis is shed in large sheets
- May see ocular involvement
- May see diffuse involvement of mucous membranes
  - GI tract
  - Respiratory tract
Toxic Epidermal Necrolysis

- **Treatment**
  - Admission to a burn unit
  - Correction of fluid and electrolyte abnormalities
    - One of the leading causes of morbidity and mortality
  - Prevent infection
  - May require intubation due to sloughing of respiratory epithelium
Erythema Nodusum
Erythema Nodusum--Etiologies

- Infection
- Sulfonamides
- Oral contraceptive pills
- Penicillin
- Bromides
- Vaccines
- Sarcoidosis
- Inflammatory bowel disease
- Pregnancy
- Behcet Syndrome
- Leukemia and lymphoma
- Idiopathic
Erythema Nodosum

- Rash Appearance/ Etiology
  - Inflammation of subcutaneous fat
    - Tender, warm, ill-defined erythematous nodules
- Distribution of lesions
  - Pretibial area of lower extremities
- Treatment
  - Treat underlying cause
  - Bed rest, elevation
  - NSAIDS
Pemphigous/Pemphigoid

- Starts in mouth often
- Autoimmune
- +Nikolsky sign
- Most common in 50-60 year old
  - Can see in neonates
- Treatment with steroids

- Mucous membranes rarely involved
- Autoimmune
- -Nikolsky
- Age 50-70
- Treatment similar
Scalded Skin Syndrome

- **Etiology/Pathophysiology**
  - Staphylococcus aureus infection
    - NOT clinically apparent
    - Exotoxin is the culprit
      - Exfoliatin
  - Causes acantholysis and intraepidermal cleavage of skin

- **At risk for this disease:**
  - Infants and small children
  - Immunosuppressed
  - Renal insufficiency
Scalded Skin Syndrome

- Appearance of Rash and Rash Distribution
  - Diffuse, tender erythroderma
  - Prominent perioral, periorbital, grain as well as skin creases
  - Sandpaper texture
  - Spares the mucous membranes

- Exfoliative phase
  - Second day of illness
  - Skin peels at area of minor trauma
  - Large flaccid vesicles form and shed in sheets
  - Skin underneath resembles scalded skin
Scalded Skin Syndrome

- Desquamation
  - Days 3-5
- Normal skin in 7-10 days
- Treatment
  - Fluid resuscitation
  - Correction of electrolyte abnormalities
  - Correction of fluid deficits
  - Identify and treat infection
    - Oxacillin or Vancomycin
Kaposi Sarcoma
Kaposi Sarcoma

- **Etiology/Pathophysiology**
  - Multisystem vascular neoplasm
    - Reactive cellular proliferation rather than malignancy
    - May be related to HHV 8
    - May involve any organ

- **“Classic” KS**
  - Elderly males of Eastern European heritage
  - Usually involves the legs

- **HIV associated KS**
  - Rapid progression and extensive systemic involvement
  - More common in homosexual men with HIV
    - 18% at this time--Had been nearly 50% early in epidemic
Kaposi Sarcoma

- Appearance of Lesions
  - Begins as ecchymotic macule
  - Forms a plaque, nodule or tumor
    - Violaceous, red, or tan that becomes purple-brown
  - Lesions may be wide-spread but often spare the trunk
  - Oral lesions most commonly involve hard palate
Melanoma
Melanoma

- **Etiology/Pathophysiology**
  - Most malignant tumor of skin
  - In the US the lifetime risk of developing invasive melanoma 1 in 75 as of 2002
- **Early detection is key!!**

- **Increased melanoma risk**
  - M: Moles (atypical nevus) > 5
  - M Moles(common) > 50
  - R: Red hair and freckling
  - I: Inability to tan
  - S: Sunburn (especially before age 14)
  - K: Kindred: family history of melanoma
Melanoma

- A- Asymmetry of shape
- B- Border is irregular
- C- Color is not uniform
- D- Diameter is large (> tip of pencil eraser)
- E- Elevation and enlargement
The Internal Medicine Stuff

- **Acanthosis nigricans**
  - Obesity
  - Insulin resistance
  - Adenocarcinoma of the GI tract

- **Amyloidosis**
  - Usually seen with blood dyscrasias
    - Most common is myeloma
    - Easy bruisability and skin discoloration noted most around eyes
IM with a Little EM thrown in...

- *Discoid Lupus Erythematosus*
- Red to violaceous papules and plaques
  - Sharply demarcated
  - Central clearing with peripheral scale
- Distribution
  - Face
  - Scalp (with alopecia)
  - Trunk or extremities
    - Usually symmetrical
- Treat initially with topical steroids
**Erythema chronicum migrans**

- Associated with Stage I Lyme disease
- B. burgdorferi via tick vector
- Begins as erythematous macule that spreads outward with central clearing
  - Target or Bulls-eye lesion
- Treatment
  - Oral doxycycline or amoxicillin or cefuroxime axetil
Neurofibromatosis
- Genetic disorder of skin and neural tissue

Café au lait spots
- Smooth, light brown macules and patches that are seen within first three years of life

Neurofibromas
- Soft, skin colored pedunculated papules
- Seen in late childhood-adolescents
- May have a few to hundreds of lesions
The Last of the Internal Medicine Exam

- **Tuberous Sclerosis**
  - Autosomal dominant genetic disorder with triad of:
    - Ash leaf spots
    - Mental retardation
    - Epilepsy

- Ash leaf spots
  - Present at birth to first 2 years of life

- **Shagreen patch**

- Angiofibromas
  - Pink or red papules or nodules on face
“Years wrinkle the skin, but to give up enthusiasm wrinkles the soul.”

Samuel Ullman