DRUG ERUPTIONS and OTHER DISORDERS

Lloyd J. Cleaver D.O., F.A.O.C.D, F.A.A.D.
Professor of Dermatology ATSU-Kirksville College of Osteopathic Medicine
INTERNAL MEDICINE BOARD REVIEW COURSE
Disclosures

- No Relevant Financial Relationships
DRUG ERUPTIONS
Drug Reactions
3 things you need to know

1. Type of drug reaction
2. Statistics
   ► What drugs are most likely to cause that type of reaction?
3. Timing
   ► How long after the drug was started did the reaction begin?
Clinical Pearls

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

- Skin is one of the most common targets for drug reactions
- Antibiotics and anticonvulsants are most common
  - 1-5% of patients
- 2% of all drug eruptions are “serious”
  - TEN, DRESS
- More common in adult females and boys < 3 y/o
- 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**
   - Eye drops, suppositories, implants, injections, patches, vitamin and health supplements, friend’s medications
3. **ALWAYS consider medications as possible cause** (no matter how atypical)
<table>
<thead>
<tr>
<th>Clinical presentation</th>
<th>Percentage that are drug-induced (%)</th>
<th>Time interval</th>
<th>Mortality (%)</th>
<th>Selected responsible drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exanthematous eruption</td>
<td>Child: 10–20 Adult: 50–70</td>
<td>4–14 days</td>
<td>0</td>
<td>Aminopenicillins Sulfonamides Cephalosporins Anticonvulsants Allopurinol</td>
</tr>
<tr>
<td>Urticaria</td>
<td>&lt;10 30</td>
<td>Min-hours Min-hours</td>
<td>0 5</td>
<td>Penicillins Cephalosporins NSAIDs Monoclonal Abs Contrast media</td>
</tr>
<tr>
<td>Anaphylaxis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fixed drug eruption</td>
<td>100</td>
<td>First exposure: 1–2 weeks Re-exposure: &lt;48 hours, usually within 24 hours</td>
<td>0</td>
<td>TMP-SMX NSAIDs Tetracyclines Pseudoephedrine</td>
</tr>
<tr>
<td>Acute generalized exanthematous pustulosis (AGEP)</td>
<td>70-90</td>
<td>&lt;4 days</td>
<td>1-2</td>
<td>β-Lactam antibiotics Macrolides Calcium channel block.</td>
</tr>
<tr>
<td>Drug reaction with eosinophilia and systemic symptoms (DRESS)</td>
<td>70-90</td>
<td>15-40 days</td>
<td>5-10</td>
<td>Anticonvulsants Sulfonamides Allopurinol Minocycline Lamotrigine</td>
</tr>
<tr>
<td>Stevens–Johnson syndrome (SJS)</td>
<td>70-90</td>
<td>7-21 days</td>
<td>5 30</td>
<td>Sulfa Anticonvulsants NSAIDS Allopurinol</td>
</tr>
</tbody>
</table>
Note confluence of lesions on trunk.
Morbilliform reaction to ampicillin-amoxicillin
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

- Usually intermittent drugs:
  - NSAIDs
  - Sulfonamides (TMP) → majority of genital fixed drug eruptions
  - Barbiturates, TCNs, phenolphthalein, acetaminophen, cetirizine, celecoxib, dextromethorphan, hydroxyzine, lamotrigine, phenylpropanolamine, erythromycin, herbs
HELP!

I am Red and Scaly From Head to Toe!
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...

- **ROS:** Photophobia & dysphagia/odynophagia
- **Recently Rx** an antibiotic for a “large boil on her leg”
- **PMHx:** seizure disorder
- **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

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

- **Definitions**
  - SJS = less than 10% BSA
  - SJS/TEN overlap = 10-30% BSA
  - TEN = greater than 30% BSA
## Associated Medications SJS/TEN

<table>
<thead>
<tr>
<th>Medications Most Frequently Associated with SJS and TEN</th>
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<tbody>
<tr>
<td>Allopurinol</td>
</tr>
<tr>
<td>Aminopenicillins</td>
</tr>
<tr>
<td>Amithiozone (thioacetazone)*,1</td>
</tr>
<tr>
<td>Barbiturates</td>
</tr>
<tr>
<td>Carbamazepine</td>
</tr>
<tr>
<td>Chlormezanone*</td>
</tr>
<tr>
<td>Phenytoin antiepileptic</td>
</tr>
<tr>
<td>Lamotrigine</td>
</tr>
<tr>
<td>Phenylbutazone*</td>
</tr>
<tr>
<td>Piroxicam</td>
</tr>
<tr>
<td>Sulfadiazine*</td>
</tr>
<tr>
<td>Sulfadoxine*</td>
</tr>
<tr>
<td>Sulfasalazine</td>
</tr>
<tr>
<td>Trimethoprim-sulfamethoxazole</td>
</tr>
</tbody>
</table>
Infections

Mycoplasma common cause of SJS in kids
EM, SJS, TEN

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

- Treatment: burn unit
  - Supportive
    - Fluids, nutrient, electrolyte replacement, infection control
  - IVIG:
    - Stops keratinocyte apoptosis, blocks death receptor FAS (CD95)
  - Immunosuppressive therapy
    - Controversial
    - May increase morbidity/mortality
  - Systemic steroids - early short trial, rapid taper

- 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

- 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?

- Herpes Virus
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

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

- Both Janeway lesions & Osler nodes are due to septic emboli
- Osler nodes are painful
  - Osler=Ouch
Janeway lesions
Community Acquired MRSA

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

DDx:

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

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

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

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

Red: 4-5\textsuperscript{th} day

Strawberry tongue, with enlarged, exudative tonsils
Scarlet Fever

Rash with circumoral pallor
Scarlet Fever

- Flushed cheeks
- Circumoral pallor
- Strawberry tongue
- Increased density on neck
- Increased density in axilla
- Transverse lines (Pastia's sign)
- Increased density in groin
- Positive blanching test (Schultz-Charlton)
Staphylococcal Scalded Skin Syndrome
*Ritter’s disease, Pemphigus neonatorum*

- Primarily children < 6 y/o
- Characterized by red, blistering skin 2° of a staph infection from distant foci
- Localized toxigenic strain of *S. aureus*
  - Naso-oropharynx or conjunctiva
  - Tender, flaccid, sterile bullae *(culture negative)*

*Not at the DEJ as in TEN which exhibits ‘full thickness necrosis’*
Staphylococcal Scalded Skin Syndrome

*Ritter’s disease, Pemphigus neonatorum*

- Prodrome of malaise, fever, irritability, sore throat, & severe tenderness of the skin → Purulent rhinorrhea or conjunctivitis

- **Erythema 1st appears on head**, then generalized in 48 hours
  - Skin develops wrinkled appearance due to flaccid bullae in superficial epidermis → *spares palms, soles, mucous membranes*
  - Perioral crusting, mild facial edema

- Scaling & desquamation continue for 3-5 days w/ re-epithelialization in 10-14 days
  - Mortality rates: 3% in children, over 50% in adults, 100% in adults w/underlying disease
Staphylococcal Scalded Skin Syndrome

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

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

St. Anthony’s Fire, Ignis sacer

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

- Complications:
  - Septicemia, deep cellulitis, necrotizing fasciitis

- Differential diagnosis:
  - Contact derm (plants, drugs, dyes) although not assoc. w/ f/c, pain
  - Lupus erythematosus butterfly pattern

- Treatment:
  - PCN, erythromycin at least 10 days
  - Ice compresses
  - Inpatient and IV Abx
Cellulitis

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

- Cellulitis is almost always unilateral, if bilateral, think stasis dermatitis
- Complications (rare in immunocompetent hosts)
  - Gangrene
  - Metastatic abscesses
  - Septicemia
- Initial therapy cover *staph* and *strep*:
  - 1st gen ceph or pcn’ase resistant pcn
  - Suspect MRSA if unresponsive
Don’t be Fooled
Pseudomonas aeruginosa

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

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

- **Hot tub folliculitis**
  - 1-4 days after exposure
  - Maintain chlorination of water 7.2-7.4
  - Apocrine areas (breast, axilla)
  - *risk for malignant external otitis (facial nerve palsy in 30%)*
  - Folliculitis self-limiting (7-14 days)
  - Cipro for systemic symptoms
Meningococcemia

- *N. meningitidis*
  - gram-negative diplococcus
- Virulence related to polysaccharide capsule (gonorrhea does not have)
- Endotoxin → inflammation
- Serogroups A, B, C, W135, X, Y and Z
  - Vaccines cover A, C, Y, W-135
- Transmitted from person to person via respiratory secretions
- Complement deficiencies components C5 to C9
  - Properdin or immunoglobulin deficiency, asplenia, and HIV infection
Meningococcemia
Meningococcemia

- *N. meningitidis*
  - gram-negative diplococcus
- Endotoxin $\rightarrow$ inflammation
- Transmitted from person to person via respiratory secretions
- Complement deficiencies components C5 to C9
  - Properdin or immunoglobulin deficiency, *asplenia*, and HIV infection
- Flu-like s/s that RAPIDLY progress
- Acute: Fever, chills, hypotension, meningitis, 50-60% have petechiae (trunk, ext)
- *Angular infarcts with erythematous rim and gun-metal gray interior*
- *Waterhouse-Friderichsen Syndrome*” (adrenal hemorrhage/ infarct) may occur 2* hypotension
  - Nasal carriage in 5-10%
  - Chronic form, very rare
Case

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

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

- Labs
  - 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

- Hospital admission
  - Further evaluation and treatment
Pathology Report

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

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

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

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

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

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

- Vitamin K Deficiency
- Vitamin B3 Deficiency
- Vitamin C Deficiency
- Zinc Deficiency
- 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

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

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

- **Functions of Zinc:**
  - Wound healing
  - Immune/reproductive/neuropsychiatric function

- **Inc. zinc requirements:**
  - infections, post-surgery, pregnancy, cancer

- **Largely dependent on food intake**
  - Nuts, whole grains, green leafy vegetables, shellfish, human milk

- **Presents most commonly in infancy**
  - *Premature*
    - suboptimal absorption, high zinc requirements, inadequate body stores
  - Breast milk generally provides adequate zinc
    - Occurs at the time of weaning from breast milk to cow’s milk
Zinc Deficiency

Genetic

“acrodermatitis enteropathica”

zinc transporter mutation

Acquired

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

- Triad: *Dermatitis, diarrhea* and *alopecia*
  - **Dermatitis:**
    - Acral and periorificial distribution
    - Patchy, red, dry scaling with exudation and crusting
    - Angular chelitis and stomatitis; drooling
  - **Diarrhea:** *suspect in infant with chronic diaper rash & diarrhea*
  - **Alopecia:** generalized
- **Additional Sx:**
  - Growth retardation
  - Impaired wound healing
  - CNS findings
  - Emotional lability & irritability
Zinc Deficiency

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

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

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

**Clinical Manifestations**

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

Questions?

lcleaver@atsu.edu
Cleaver Dermatology
Cleaverderm.com
660-627-7546