ATOPIC DERMATITIS

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CONFLICT OF INTEREST

• Abbvie – Principal investigator for clinical research and speaker

OBJECTIVES

Differentiate severity of atopic dermatitis and appropriate treatment

Identify initial treatment strategies and therapies

Recognize the signs and symptoms of atopic dermatitis
THE ITCH THAT RASHES.....

- Chronic inflammatory pruritic dermatosis
- Immunologic etiology
- Environmental factors
- Associated diagnoses
- Itch is a hallmark
- Demonstrates physical signs
- Demonstrates socioeconomic effects
- Differentiate from other inflammatory skin conditions
- Challenge to treat
  - Multifactorial
  - Classic treatments
  - Emerging treatments

INTRODUCTION

- Chronic, inflammatory disease
  - Characterized by pruritis & chronic course of exacerbations/remissions
  - Most common inflammatory disease
  - Major health concern in many countries
- Atopic March (Triad + 1)
  - Food allergy, asthma, allergic rhinitis
- Onset
  - Usually infancy, occasionally adulthood
  - 50% in first year of life
  - Vast majority first 5 years
    - Adult AD: consider when dermatitis has characteristic distribution and when other ddx has been excluded
    - ACD: photodermatitis, CCL

EPIDEMIOLOGY

- Prevalence
  - Most high-income & some low-income countries
  - 30% developed countries
  - Vastly 10% in many other countries
  - Cumulative 30% across globe

- Are we too clean?
  - 2-3 fold increased prevalence over past several decades
  - Controversial hygiene hypothesis: decreased exposure to infectious agents in early childhood increases susceptibility to AD
  - Iceland has a very high rate (27%) yet no dust mites, few trees, and no pet ownership

- Onset
  - 50%-60% in first year of life
  - 90%-95% by 5 years
• Food allergy → predilection for infants & young children
• Asthma → older children
• Rhinaconjunctivitis → predominates in adolescents

PATHOGENESIS

• Complex
  - Interaction of epidermal barrier dysfunction, immune dysregulation, environment
  - Filaggrin (FLG) mutations
    - Normally binds to keratin fibers in epithelial cells → alterations cause defective epidermal barrier
    - Leads to transepidermal water loss and xerosis → allowing penetration of allergens/irritants

• Genetic Basis
  - 80% identical twins show concordance
  - More than ¼ offspring of AD mothers develop AD in 1st 3mo of life
    - > ½ offspring by 2yr
  - 79% risk if both parents are atopic
# Atopic Dermatitis

## Clinical Criteria
- **Essential** – pruritus (all stages)
- **Plus ≥ three of the following**
  - History of xerosis (dry skin)
  - Personal history of allergic rhinitis or asthma (atopic triad)
  - Onset < 2 yo
  - History of skin crease involvement
    - Antecubital, popliteal, ankle, neck, periorbital
  - Visible flexural dermatitis

## Clinical Features
- Based on type
  - Acute form
    - Erythema, edema, vesicles, oozing, crusting
  - Subacute and chronic forms
    - Lichenification, papules, nodules, excoriations
  - Further classified into...
    - Early-onset type (Pediatric)
    - Late-onset type (Adult)
    - Senile-onset type (Senile)

## Pediatric AD
- **Infantile** (birth to 6 months)
  - Acute presentation and clinical features
  - Favor face, scalp, and extensor surfaces
  - 50% or more present in 1st year of life (usually after 2 months)
- **Childhood** (2+ yo to 12 yo)
  - Usually more chronic in nature, though acute flares may occur
  - Favor flexures
  - Diffuse xerosis becomes more prominent

## Age Related Changes
- **Adolescent/adult AD**
  - Age > 12 yo
  - Lichenified plaques 
  - Prominent involvement of flexures, face, neck (retroauricular), upper arms, back, and acral sites
  - AD beginning during childhood is a/w more severe, treatment-resistant disease
  - May manifest as isolated prurigo nodularis, hand or eyelid dermatitis
- **Senile AD**
  - Age > 60 yrs
  - Marked xerosis rather than typical AD lesions
### ASSOCIATED FEATURES

<table>
<thead>
<tr>
<th>Condition</th>
<th>Associated Feature</th>
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<tbody>
<tr>
<td>Icthyosis vulgaris</td>
<td>Allergic salute</td>
</tr>
<tr>
<td>Keratosis pilaris</td>
<td>Pityriasis alba</td>
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<tr>
<td>Palmoplantar hyperlinearity</td>
<td>Keratoconus</td>
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<tr>
<td>Dennie-Morgan lines</td>
<td>Eczema herpeticum</td>
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<td>White dermatographism</td>
<td>Circumoral pallor</td>
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<tr>
<td>Periorbital darkening</td>
<td>Herlghke sign</td>
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**Pityriasis alba**

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**Keratosis Pilaris**
REGIONAL VARIANTS OF ATOPIC DERMATITIS

- **Ear**
  - Erythema/scaling/fissuring under earlobe and retroauricular region
- **Eyelid**
  - Lichenification of periorbital skin
  - MC related to ACD
- **Lips**
  - Cheilitis sicca → “Chapped Lips”
  - Lip-lickers dermatitis
  - Cinnamon, dyes, sunscreens, etc.
- **Nipple**
  - Runners

REGIONAL VARIANTS OF ATOPIC DERMATITIS

- **Diaper**
  - Aka Napkin derm
  - Related to urine/feces ICD
- **Hand**
  - **Intrinsic**
    - Atopic, psoriasis, dyshidrosis, hyperkeratosis
  - **Extrinsic**
    - ICD, ACD, water exposure
    - **Infectious**
      - Tinea, S. aureus

Susceptibility to Infection

- More than 90% of chronic lesions contain S. aureus (Impetigo)
- S. aureus produces δ-toxin → mast cell degranulation and Th2 inflammation
- Treatment:
  - Skin baths and reduction of nasal carriage (Mupirocin)
  - Capsaicin creases controlling infection triggered AD
- Frequent infections → chronic suppressive oral ABX therapy
  - Cephalosporins, trimethoprim/sulfamethoxazole, clinda, doxycycline
- HSV - Eczema herpeticum
  - Predominantly HSV-1
- VZV - Eczema vaccinatum
  - Widespread vaccinia infection → complication of varicella vaccine
- Molluscum contagiosum
Eczema
Herpeticum = Eczema + HSV

Laboratory Testing
- IgE not typically helpful
- Allergy Testing
  - RAST testing or Prick test may be warranted
- Food Allergy Testing
  - Consider in children severe/refractory AD
  - Eggs, milk, peanuts, soy, and wheat MC culprits
  - Food allergy most commonly causes a type I immediate hypersensitivity reaction
  - 10%-15% of children with severe AD have coexistent food allergies
- Consider testing for allergens
  - Dust mites, pollen, animal dander, & fungi in teens/adults w/ severe or refractory AD on exposed skin

Prevention - high-risk children
- What we know...
  - Soy formulas do not appear to reduce risk
  - Prolonged exclusive breastfeeding beyond 3–4 mo is not protective
  - Maternal allergen avoidance during pregnancy does not reduce risk
  - Including house dust
    - Prebiotics not currently recommended; some studies suggest reducing AD
  - Aggressive emollient therapy to repair genetic or acquired epidermal barrier defect

Food allergy
- Role of food allergy in AD complicated
  - 35% of children with moderate-severe AD have food allergy
  - 85% of children with AD have elevated IgE to food or inhalant allergens, making diagnosis of food allergy with serum or prick tests alone inadvisable
- Testing
  - First optimize AD
  - Do not prescribe PO antihistamines
  - Food restrictions can cause malnutrition
  - Only consider in <5 yo if failed standard care

Food prick test type 1
- High sensitivity, moderate specificity
PREVENTATIVE TREATMENT

- Education and support
  - Written “action plans”/scheduling “stepwise approach”
  - Ointments and water-in-oil creams more occlusive & less burning than oil-in-water creams and lotions
  - Perfumes, talc, bath and body works and herbal extracts should be avoided
  - Emollients
    - CeraVe, Cetaphil, Aveeno, Vanicream, GoldBond, Eucerin, Aquaphor, Stiefel Neutrogena Norwegian Formula
    - Short, lukewarm baths w/ minimal soap
    - 1/3 cup bleach to full tub of water once to twice weekly
    - Bleach baths, especially if history of skin infection – > STAPH
    - 1/2 cup bleach to full tub of warm water once to twice weekly
  - Wet dressings +/- topical steroids (“Wet Wraps”)
  - Avoid fragrance filled products

Psychosocial aspect

- Currently recommended
- Ask general questions about itch, sleep, impact on daily activity, persistence of disease
- Assess associated signs/symptoms
  - Rhinitis/conjunctivitis, asthma, food allergy, sleep disturbance, depression, & other neuropsychiatric conditions
- Incorporate into treatment plan when appropriate

Prognosis

- AD tends to clear in most children by puberty
- Historically 75% resolve by adolescence
  - However, new study suggests that only 50% remit by early adulthood
- If persists beyond childhood – tends to be chronic
MEDICAL TREATMENT

- Topical corticosteroids are mainstay
- May experience rebound flares after short courses of systemic corticosteroids
- Sedative antihistamines as adjunctive treatment for itch
  - First-generation antihistamines (Hydroxyzine) are safe for use in infants/toddlers
- Treat secondary infections
  - AD, skin/erosions, gram-negative peptidoglycan and a compromised barrier → infections
- Treatment ladder
  - 1st: Topical treatments (steroids, calcineurin inhibitors)
  - 2nd: Light therapy (nbUVB > bbUVB, UVA1, and PUVA)
  - 3rd: Systemic meds (corticosteroids, cyclosporine, methotrexate, and etanercept) depending on severity
**TREATMENT CONTINUED**

- Once flares controlled → use non-steroidal for maintenance
  - Topical calcineurin inhibitors
    - Tacrolimus (Protopic)
    - Pimecrolimus (Elidel)
  - Topical PDE4 inhibitors
    - Crisaborole (Eucrisa)
- Moisturize, moisturize, moisturize!

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**Oral Therapies**
- **Steroids**
  - Avoided due to rebound flares
  - Tapered course if used
- **Cyclosporine**
  - Rapid improvement of skin and pruritus
  - Nephrotoxicity, HTN
  - Start 3 mg/kg/day, → reduce to minimal effective maintenance dose
  - Usually ~2 mg/kg/day
- **Azathioprine**
  - 2–3.5 mg/kg/day if normal TPMT activity, 0.5–1 mg/kg/day if low
- **Mycophenolate mofetil**
  - 1–2 g/day, 20–50 mg/kg/day in adults
- **Methotrexate**
  - 7.5–25 mg/week
- **Sedating antihistamines**
  - Hydroxyzine, diphenhydramine, doxepin
  - Reduces sleep scratching

**Biologics**
- **Omalizumab**
  - Monoclonal ab that inhibits binding of IgE to FcεRI
  - Mixed results → most say no help for AD
- **Rituximab**
  - Anti-CD20 monoclonal ab
- **Dupilumab (Dupixent)**
  - Injectable monoclonal ab that inhibits IL-4 and IL-13 cytokines

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**General Management – Severe & Systemic Involvement**

- **Sedating antihistamines**
  - Hydroxyzine, diphenhydramine, doxepin
  - Reduces sleep scratching

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**Eczema Action Plan**

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**TREATMENT RECOMMENDATIONS**

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**Inclusion Exclusion**

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**References**

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TOPICAL CORTICOSTEROIDS

- It is safe to alternate potent and low to medium strengths for flares
  - Clobetasol 0.05% oint./Betamethasone dipropionate 0.05% oint. BID x 2 weeks
  - Alternate with Triamcinolone 0.1% cream/ointment BID x 2 weeks per flare
- OR
  - Clobetasol 0.05% oint./Betamethasone dipropionate 0.05% oint. BID x 1 week, then twice weekly prn
- SE
  - Steroid induced atrophy & acne/rosacea
  - "Stretch marks"
  - Avoid medium to high potency steroids on face, axillae, under breasts and groin
  - Fungal infection (Tinea incognito)
  - "Giada using mixed steroid and anti-fungal (Lotrisone) & Betamethasone 0.05% + 1%

See Medscape or Dermatol Nurs 2006 by Jannetti Publications, Inc. for chart listing by Class; Generic Name; and Formulation

CLOBETASOL 0.05% CREAM AND TRIAMCINOLONE 0.1% CREAM
Steroid Induced Acne

5 years with a class 5 topical steroid

10 Days Later after D/Cing

Steroid induced atrophy
CAN I GET A REFILL?

STEROID ATROPHY

- Prescribed clobetasol 0.05% ointment by provider for dry skin
- Used over 10 years daily on arms
- Lives in nursing home and the prescription renewed over and over
- ALWAYS give orders of when to stop and start medication, whether in the office, nursing home, or hospital

CLOTRIMAZOLE/BETAMETHASONE FOR OVER 3 YEARS

5 days after stopping
TAKE HOME POINTS

- Use clotrimazole/betamethasone on appropriate areas
- DO NOT give more than one tube
- Use appropriate steroids or steroid sparing agents for face or intertriginous area
- Do not refill rx without seeing the patient, or ensuring it is appropriate

TINEA INCOGNITO
(STEROID + FUNGAL INFECTION)

LATEST AD/ECZEMA TREATMENTS
CRISABOROLE (EUCRISA)

- PDE4 (Phosphodiesterase 4) Inhibitor \(\rightarrow\) overactive in inflamed eczema
- Approved 2 years of age and up with mild to moderate AD
- Topical ointment, apply BID for maintenance
- Non-steroidal, safe all over body
- SE: ACD, burning/stinging with application during acute flares
  - Stinging is usually improved after subsequent applications
  - Can be significant and patients may discontinue
  - Counseling pt and family is essential

DUPILUMAB (DUPIXENT)

- Injectable biologic for moderate to severe adult atopic dermatitis
  - An IgG4 monoclonal Ab which binds IL-4 & IL-13 receptors
  - Results in suppressed Th2 mediated inflammation
- 300mg injection q 2 weeks
  - FDA Approved 18 yrs and older; ongoing studies for younger
  - Approved for asthma for 12 yrs and older
- SE: hypersensitivity, conjunctivitis, keratitis

ON THE HORIZON

- Janus Kinase (JAK) Inhibitors
  - Interferes with JAK-STAT signal pathway
  - Oral medication
  - Tofacitinib (Xeljanz, Jakvinus) targets JAK1/3
  - FDA approved rheumatoid arthritis in RA, psoriatic arthritis, atopic dermatitis, ulcerative colitis
  - Ruxolitinib (Jakafi) targets JAK1/2
  - FDA approved polycythemia vera and myelofibrosis in adults
  - JAK Inhibitors under investigation for autoimmune diseases
  - RA, psoriatic/pсориatic arthritis, atopic dermatitis, ulcerative colitis
- At least 19 different medications in research pipeline targeting atopic dermatitis including biologics and topical medications

See Bolognia 4th for illustration of Mechanism of Action of Janus Kinase (JAK) Inhibitors.
ECZEMATOUS DERMATOSES

Eczema is “the itch that rashes”

Overview
• 3 stages
  • Acute, Subacute, Chronic
  • Eczema is Greek “to boil”
• Etiology
  • Inflammation due to contact with specific allergens, chemicals, or other acute inflammatory processes
• Treatment
  • Same as AD

ECZEMA

Overview
• 3 stages
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Stages
• Acute
  • Intense pruritus, fluid-filled vesicles, swollen plaques
• Subacute
  • Less wet and weepy, scaling, indistinct border, +/- pruritus
• Chronic
  • Thick, lichenified “washboard” skin
  • Moderate to intense pruritus
ASTEATOTIC DERMATITIS (ECZEMA CRAQUELÉ)

- Xerotic skin w/ fine cracking
- Typically >60 yo; worse in winter
- Resembles "cracked porcelain" → hence eczema craquelé
- Erythema and scale +/- oozing, and crusting
- Pruritic, favors lower legs
- Elderly
- Natural moisturizing factor → ↓ water binding capacity → when humidity is low in winter → xerosis → asthetatic derm

Treatment
- Emollients to prevent
- Topical corticosteroids and TCIs for flares

DRY-RIVER BED APPEARANCE

DYSHIDROTIC ECZEMA

- Presents as a symmetric vesicular hand and foot dermatitis
- Moderate to severe pruritus precedes the appearance of vesicles on the palms and sides of fingers ( tapioca like lesions)
- Pain is chief complaint
- Vesicles slowly resolve over 3-4 weeks
- Etiology
  - Allergic contact (68%)
  - Idiopathic (20%)
  - Fungal (12%)
- Treatment
  - Topical steroids, wet compresses, oral antibiotics, methotrexate, PUVA therapy
**HAND ECZEMA**

- 10% prevalence with 4% of population having active flare
  - MC occupational dermatitis (80%) → often long lasting, relapsing course
  - Wet work = skin in liquids or gloves > 2 hrs/day OR washing > 20x/day is risk factor
  - Spike in 20-29 y/o females from child care & house cleaning
- Genetic risk factors unknown
  - AD patients with filaggrin mutation may be at higher risk/severity
- Psychosocial
  - Depression, reduced quality of life
  - Long sick-leave periods, sick pension, occupational change

**Treatment**

- Avoidance, counseling, barriers, creams, topical steroids
  - Topical steroids under occlusion (non-latex gloves) overnight for 3 nights → great for flares!!
- Wash hands as infrequently as possible
  - Ideally, soap should be avoided, wash hands in lukewarm water
- Avoid direct contact with household cleaners and detergents
  - Wear rubber gloves when irritants are encountered
  - White cotton gloves can be worn under rubber gloves

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**NUMMULAR ECZEMA**

- Unknown pathogenesis
  - Round, coin-shaped “nummular” pink plaques
  - MC on extremities
  - Very pruritic
  - Acute (eczematous) or chronic (lichenified) appearance
- Associated factors
  - External irritants, venous HTN, infection, atopy, xerosis
  - Secondary Staph infection common
- Treatment
  - Mid to high potency topical steroids
  - TCs, and phototherapy: good skin care w/ emollients
STASIS DERMATITIS

- Incompetent valves of lower extremities
  - Venous HTN → capillary leak → extravasation of fluid, plasma proteins, RBCs → Pitting edema and hemosiderin deposits over distal third of leg
  - Scaling, inflammation, and pruritus or tenderness
  - Skin changes often begin on medial ankle

- A/w lipodermatosclerosis (stasis panniculitis)
  - "Inverted wine bottle" appearance w/ light circumscribed edematous areas over distal third of calf from chronic inflammation → adherent skin/subcutaneous tissue/fascia

- Treatment
  - Manage venous HTN w/ compression stockings (1st line) & elevation
  - For dermatitic component: emollients/topical steroids
  - Commonly misdiagnosed as bilateral leg cellulitis

AUTOSENSITIZATION
AKA "ID REACTION"

- Secondary eczematous lesions develop in sites distant from primary exposure site
  - Usually ACD +/- stasis dermatitis
  - ~60% w/ contact dermatitis and stasis dermatitis develop id reactions
  - Can also occur in tinea pedis

- Disseminated lesions appear days to weeks after primary lesion
  - Eczema tends to be ill-defined and symmetric, often occurring in analogous anatomical sites
  - Palms, soles, extremities

- Pathogenesis unknown but possibly related to...
  - Hematogenous dissemination of allergens
  - Sensitization threshold in distant skin sites after primary inflammation
  - Circulating activated memory T-cells

- Treatment
  - Topical steroids, PO antihistamines, treat underlying causes

CONTACT DERMATITIS
### CONTACT DERMATITIS

#### Epidemiology

- **Irritant (ICD, 80%) > allergic (ACD, 20%)**
- Occupations most affected:
  - Manufacturing/mining (UK)
  - Agricultural workers (USA)
- ICD is the most common form of occupational skin disease.
- Causes:
  - Soaps
  - Wet work
  - Petroleum products
  - Cutting oils
  - Coolants
- Seen in: Petrochemical, rubber, plastic, metal, and automotive industries.
- Most common causes of ACD:
  - Nickel (worldwide)
  - Poison ivy (USA)

#### Pathogenesis

- **Irritant Contact Derm (ICD)**
  - Direct damage of keratinocytes
  - Not immune-mediated
  - No previous sensitization required
- **Allergic Contact Derm (ACD)**
  - Immune-mediated, delayed-type (type IV) hypersensitivity, initial sensitization to allergen is required
  - Reexposure → T-cell mediated release of cytokines/chemotactic factors → eczema within 48 hrs

#### Clinical features – ACD

- **Acute**
  - Erythema, edema, papules, oozing, vesiculation
  - Sharp demarcation between normal and involved skin
- **Subacute**
  - Acanthosis, ↑crusting/scaling, ↓vesiculation
- **Chronic**
  - Marked lichenification, fissuring/scaling, no vesicles, less well-defined than acute

#### Clinical features – ICD

- Clinical presentation variable
  - Burning may be worse than itch
  - Hands #1, face #2
- **Acute ICD**
  - Vesiculation/necrosis, well-demarcated
- **Chronic ICD**
  - Dryness, scaling, lichenification, fissuring, poorly defined
- **Airborne ICD**
  - Resembles photoallergic reaction, but involves upper eyelids, philtrum, and submental region
- **Phytophotodermatitis**
  - Will discuss shortly

#### Laboratory testing

- Patch testing will confirm ACD
  - Patches applied to upper back
  - Removed at 48 h (day 2) and read = 1st reading
  - Second reading day 3–7 (usually)
  - **TRUE test**
  - 3 panels of 12 allergens each
  - www.trueset.com

#### Treatment

- **Gold standard - education and avoidance**
  - For ICD, many cases resolve spontaneously due to "hardening" phenomenon
  - After acute ACD exposure (i.e., poison ivy)
    - Whole area/body should be first washed with water ± 15 min. — then soap can be considered
    - Within 3 hrs, half of oleoresin is absorbed, effects cannot be reversed
    - Systemic antihistamines over 3 weeks are very effective
    - Potent topical corticosteroids
CONTACT DERMATITIS

Specific contactants - ACD
- Metals
  - Nickel (MC worldwide)
  - Chromium (shoe), cobalt (jewelry), mercurial (dental)
- Latex, rubbers, neoprene
- Adhesives
- Vinal polys – common cause of sensitivity ACD
- Preservatives (Quaternium-15)
- Fragrances (Balsam of Peru, Essential oils)
- Topical antibiotics (Neomycin in Neosporin)

Specific contactants - iCD
- Fiberglass
- Tx = talcum powder
- Bodily fluids – saliva, urine, feces
- Tx = barrier – zinc oxide paste
- Alkali burns more severe than acids
- Plants
  - Nettle, pineapples, daffodils, hot peppers, poison ivy, buttercups, garlic, parsley, parsnips, celery, lemon, lime (bartenders), grapefruit

Nickel ACD
CONTACT DERMATITIS

Rhus Dermatitis
- Anacardiaceae family, Toxicodendron spp.
  - Allergen: urushiol (an oleoresin)
  - Contained in leaves, stems, and roots
  - Sensitizing ingredient: pentadecylcatechol
- Poison ivy/poison oak/poison sumac
  - Direct contact (plant/fingers) → linear/streaky erythematous vesicles/bullae
  - Indirect contact (pet/burning plant) → diffuse
- Black lacquer/spot dermatitis
  - Sap from Toxicodendron species turns black w/ oxidation in stratum corneum

"Leaves of 3, let it be!"
CONTACT DERMATITIS

Phytophotodermatitis (ICD)
- Non-immunologic – it can happen to anyone
- Need UVA light plus topical or oral photosensitizer
- Furocoumarins (psoralens) are MC photosensitizers
- Limes, celery and rue (herb-of-grace) are the most common causes
- Photodistribution
- Can appear in bizarre cutaneous patterns of inflammation
- Brushing against a plant
- Streaks from celery/lime juice
- Common in bartenders and when on tropical vacations

PSORIASIS
- Chronic inflammatory immunologic condition
- Erythematous scaly plaques
- May be itchy, but generally not as itchy as AD
- Elbows, knees, sacral, but can be anywhere
- May have associated arthritis, nail changes
- Usually do not see in infants or less than age of 1

PITYRIASIS ROSEA
- Mild inflammatory exanthem characterized by salmon-colored papular and macular lesions, often covered with a scale
- Moderate pruritus may be present
- Herald patch – initial lesion, larger than succeeding lesions
- Affects trunk in Christmas tree pattern
- Related to human herpes virus 6 & 7
SEBORRHEIC DERMATITIS

- Very common
- 2-5% of population
- Chronic, superficial, inflammatory disease
- Locations
  - Scalp
  - Eyebrows
  - Eyelids
  - Nasolabial creases
  - Sternal area
  - Ears
- Scaling on an erythematous base
- Yellow, greasy appearance
- Pruritus
- Infants – Cradle Cap

MYCOSIS FUNGOIDES

- Cutaneous T-cell lymphoma (CTCL)
- May resemble eczema for years
  - Often misdiagnosed as eczema
  - May require multiple biopsies over multiple years to diagnose
- Usually begins as erythematous patches, and may progress to scaly plaques and then to nodules & tumors

TINEA CORPORIS

- Inflammatory dermatosis resulting from a dermatophyte – fungus
- Erythematous scaly plaque with advancing border
  - Almost always scale
- Steroids will make worse – fungus food
  - Consider tinea anytime a topical steroid worsens a dermatitis
REFERENCES