Atopic Dermatitis

Medical Student Rotation Lecture Series
Department of Dermatology
University of Southern California
What is Eczema?

• “Eczema” and “dermatitis”
  • Synonyms, descriptive rather than a diagnosis
  • Refer to a broad range of conditions that begin as spongiosis and may progress to a lichenified stage

• Types of Eczema
  – Atopic Dermatitis
  – Contact Dermatitis
  – Seborrheic Dermatitis
  – Stasis Dermatitis
Atopic Dermatitis

- Chronic, pruritic inflammatory disease associated with a personal or family history of atopy

- Affected families may manifest any combination of the **Atopic Triad**
  - Asthma
  - Allergic rhinitis
  - Atopic dermatitis

- If one parent is atopic, >50% of their children will develop allergic symptoms by 2 years old

**Important to ask about personal and family history of atopy for any patient presenting with a rash**
Epidemiology

• The most common inflammatory skin condition in pediatrics

• Chronic course with periods of remission and flares

• Common: 20% of children and 2-10% of adults

• Begins by age 6 months in 45%, by 1 year in 60%, and by 5 years in 85% of affected patients. Tends to improve with age, but not all patients “outgrow”.

• AD is the first manifestation in the “atopic march” (50-80% will develop asthma or AR)
Pathogenesis

• Multifactorial
• The following factors are thought to play a role:
  – Genetics
  – Skin barrier dysfunction
  – Impaired immune response
  – Environment

• **Th2 immune response in AD as compared to psoriasis which involves Th1 and Th17 pathways**
Clinical Presentation

• “The Itch that rashes”
  – Itching often precedes the appearance of lesions
  – Perpetuating cycle: more scratching -> more itching -> more scratching
  – Pruritus **significantly** affects quality of life
  – One of the goals of treatment is to break this cycle

• Lesions typically begin as erythematous papules that coalesce to form erythematous plaques with associated weeping, crusting, or scale
Clinical Presentation

• Stages of AD
  – Infantile (2 months to 2 years)
  – Childhood (2 to 12 years)
  – Adolescent/Adult (>12 years old)
Infantile AD

• Erythematous ill-defined plaques with overlying scale, oozing, crusting

• Areas of involvement:
  – Cheeks, forehead, scalp
  – Extensor surfaces
  – Trunk
Infantile AD
Childhood AD

• Lesions are less exudative and become lichenified

• Areas of involvement:
  – Flexural areas: antecubital & popliteal fossae
  – Head (especially periorificial)
  – Neck
  – Wrists, Hands, Ankles, Feet
Childhood AD
Adolescent/Adult AD

• Similar to childhood form, with chronic lichenified plaques over flexural regions

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  – Wrists, Hands, Ankles, Feet

**Hands are commonly affected in adolescents and adults and may be the sole manifestation.**
Adult AD
• Things to consider in adults with new onset or difficult to control eczematous rash:
  – Scabies
  – Contact Dermatitis
  – Drug Eruption
  – Photodermatitis
  – HIV
  – Mycosis Fungoides
Associated Findings

• Xerosis
• Dennie morgan lines
• Allergic salute
• Allergic shinners
• Hertoghe sign
• Hyperlinear palms and soles
• Lichen simplex chronicus
• Keratosis pilaris
• Pityriasis Alba
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• Bacteria
  – Staph colonization and secondary infections are common in atopic patients
  – Can causes flares
  – Ask about: flares, painful lesions, fevers, chills
  – Look for tender areas of skin with overlying oozing, yellow crusting, fissuring, erosions, pustules, impetiginization
Infections - Viral

• **Eczema herpeticum**
  – Rapid dissemination of a herpes simplex virus infection over eczematous skin in AD patients
  – Presents with eruption of vesicles -> monomorphc punched out erosions with crusting
  – Widespread with predilection for head, neck, trunk
  – Complications
    • Herpetic keratoconjunctivitis
    • Herpetic meningoencephalitis
    • Superinfection with staph or strep
Infections - Viral

• Molluscum contagiosum
  – Caused by pox virus
  – Presents with flesh colored to pearly umbilicated papules
  – Patients with AD are predisposed to development of widespread molluscum contagiosum
Management

Goals of Management:
1) Repair skin barrier
2) Decrease inflammation
3) Treat/prevent infection
4) Control pruritus
5) Avoid triggers
1) Repair Skin Barrier

Gentle skin care instructions

• **Bathe** with warm water. No scrubbing or washcloths. Pat dry with towel.

• **“Soak and seal”** – immediate application of meds/emollient after bathing helps increase hydration and penetration of meds

• **Gentle soap** (ie. Dove white bar soap)
  – Soap only needed in seborrheic areas (axillae, anogenital, face/scalp)
  – Use soap on other parts of the body only if visibly dirty
  – Examples of drying soaps: Dial, Irish Spring, Ivory

• **Fragrance-free, thick moisturizer.**
  – Greasier = better! (fewer preservatives, don’t sting/burn on application, more occlusive to help with restoring barrier)
  – Ex: aquaphor or vaseline ointment, Eucerin or Cetaphil cream
  – Ceramide-based topicals may help replace lipid barrier
2) Decrease Inflammation

• 1st line: topical steroids
  – Ointments preferred in infants and young children
  – Strength and duration of treatment determined by site of involvement and severity of disease
    • General rule: Low potency on face/diaper area (hydrocortisone 1% or 2.5% ointment)

• 2nd line: calcineurin-inhibitor topicals – topical tacrolimus
  – Used for maintenance; during a flare can cause a burning sensation when applied so poorly tolerated
<table>
<thead>
<tr>
<th>Potency</th>
<th>Class</th>
<th>Examples</th>
</tr>
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<tbody>
<tr>
<td>Super high</td>
<td>I</td>
<td>Clobetasol propionate 0.05% Betamethasone 0.05% augmented ointment</td>
</tr>
<tr>
<td>High</td>
<td>II</td>
<td>Fluocinonide 0.05% Betamethasone 0.05% ointment</td>
</tr>
<tr>
<td>Medium</td>
<td>III-V</td>
<td>Triamcinolone acetonide 0.1% ointment Triamcinolone acetonide 0.1% cream</td>
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<tr>
<td>Low</td>
<td>VI – VII</td>
<td>Fluocinolone 0.01% oil Desonide 0.05% Hydrocortisone 1% or 2.5%</td>
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• Remember to look at the **class** not the percentage
  • ie. clobetasol 0.05% is stronger than hydrocortisone 2.5%
• A topical steroid in ointment form is stronger than its counterpart cream as ointments are more occlusive
3) Treat infection and decrease colonization

• Topical antibiotics: mupirocin

• **Dilute sodium hypochlorite soaks** (bleach baths):
  – ¼ cup household bleach in bathtub half full – at least 2x per week to decrease colonization and maintain control of disease

• Oral antibiotics
  – Keflex is typically first line unless history of MRSA
4) Control pruritus

• **Sedating** H1 antihistamines – helpful due to sedation:
  – Examples: Hydroxyzine, diphenhydramine, doxepin

• Non-sedating antihistamines not helpful in controlling pruritus

• Wet wraps
Wet wraps

- Wet bandages or clothing wrapped over emollients and/or topical steroid creams
- Helps to control flares through cooling, moisturizing, and enhancing steroid absorption

General instructions:
- Soak in a bath then apply emollient and/or steroid creams
- Apply moist bandages or pajamas followed by dry bandages or pajamas
- Child sleeps in these and they are removed in the morning
5) Avoid triggers

- Environmental allergens:
  - Eg. Dust mites, pollen, animal dander
- Environmental triggers:
  - Heat
  - Sweat
  - Wool
  - Stress
  - Overbathing
  - External irritants: rubbing alcohol, lemon juice, alkaline soaps, vigorous rubbing/scrubbing
- Food allergens – see next slide

**Ask all patients with about their bathing habits**
Food allergies and AD

• The role of allergy in AD remains controversial
  – Many patients with AD have sensitization to food and environmental allergens; however sensitization is not proof of a clinically relevant allergy

• Food allergy as a cause of, or exacerbating factor for, AD is uncommon
  – Identification of true food allergies should be reserved for refractory AD in children in whom suspicion for food allergy is high
  – Consider in patients with severe, recalcitrant AD or if other symptoms of food allergy are present (ie. vomiting, diarrhea, FTT)
Example treatment regimen

• Gentle skin care, bathing guidelines, emollients, avoidance of triggers

• Topical steroids:
  – TAC 0.1% ointment BID Monday to Friday OR 2 weeks on 1 week off to trunk/arms/legs
  – Desonide 0.05% ointment BID to face/neck/diaper area/skin creases

• Sedating antihistamine: ie. hydroxyzine

• Control of infections/colonization:
  – Bleach baths for maintenance
  – Topical mupirocin
  – Oral antibiotics for secondary infections
Severe or recalcitrant AD

• Systemic steroids
  – Short prednisone taper: <3 weeks preferred
  – IM Kenalog: 40mg into upper outer buttock (used primarily in adults)

• Light therapy with nbUVB

• Immunosuppressants
  – Cyclosporine: used for rapid control and then bridge to another agent
  – Azathioprine (Imuran)
  – Mycophenolate mofetil (Cellcept)
  – Methotrexate
Severe or recalcitrant AD

- Dupilumab (Dupixent)
  - Monoclonal antibody to the IL-4 receptor alpha subunit of the receptor complexes for IL-4 and IL-13 -> inhibits Th2 response that drives AD
  - Approved for 6 years and older
  - Overall well-tolerated
  - Adverse effects
    - Conjunctivitis/Keratitis
    - Hypersensitivity reaction <1%
Take home points

• AD is a chronic, pruritic, inflammatory skin disease

• It is one of the most common skin disorders in developed countries, affecting about 20% of children and 2-10% of adults

• Part of the atopic triad (AD, asthma, allergic rhinitis).

• Management goals include:
  1) Repair skin barrier
  2) Decrease inflammation
  3) Treat/prevent infection
  4) Control pruritus
  5) Avoid triggers
• Luu M. Neonatal and Pediatric Dermatology Part II.