Management of children and adult patients with atopic dermatitis

Impaired skin barrier and Th2 inflammation

1. Defective epidermal barrier
2. Environmental triggers
3. Skin inflammation
4. Pruritus

- CD4, CD8
- IL-4, IL-5, IL-13
- IFN-γ
- DC
- Eosinophils
- Mast cells
- B cells
- IgE
1 Restoring the skin barrier

• Emollients, ointments
  – Enriched with moisturizers, lipids, ceramides
  – Wet wraps

• Vitamin D3: controversial
  – increase of cathelicidin expression in AD skin
  – Cave: topical vitamin D might induce TSLP

• Calcineurin inhibitors
  – In vitro, pimecrolimus enhances production of cathelicidin, HBD2, HBD3 by keratinocytes

• Avoid long-term therapy with topical corticosteroids

2 Avoidance of triggers

• Staph.aureus
  – Antimicrobial intervention: disinfectants, bleach baths, silver, silk clothes
  – Cave: topical antibiotics
  – vaccinations

• Soap, detergents: use syndets, oil baths

• House dust mites
  – Reduce exposure, specific immunotherapy for allergic patients

• Emotional stress
3 Anti-inflammatory therapy

• Topical:
  – Corticosteroids
  – Calcineurin inhibitors

• Systemic:
  – UV Therapy
  – Cyclosporine, corticosteroids, azathioprine, MMF, MTX
  – Alitretinoin
  – Biologics

• Proactive therapy
  – Topical corticosteroids, calcineurin inhibitors (2x per week)
4 Anti-pruritic therapy

- Emollients
  + polidocanol (local anesthetics)
  - Wet wraps
- Effective anti-inflammatory therapy
- Antihistamines
  - Controversial
  - Non-sedating
- Cold packs
- Psychological interventions, relaxation
Educational programs

- To increase compliance
- To increase knowledge on disease pathomechanisms and treatment
- For patients and parents
- Interdisciplinary courses: dermatologist, allergist, psychologist, nutritionist, nurse, physiotherapist (5 x 2 hours)
- Education, practical demonstrations of therapy, in AD clinic
Epidermal Role in AD Development

• Corneocyte defect—FLG, LEKTI, CDSN
• Irritants, proteases, pH change, microbes, allergens
• Keratinocytes → TSLP
• Influence on immune cells (Th2, IgE)
• Perturbs respiratory epithelium

(Zhang 2009; Demehri 2009, Irvine 2011)
AD & Atopy: New Directions in Prevention and Therapy


• Household peanut consumption as a risk factor for the development of peanut allergy, *Fox, et al. JACI, 123:417-23, 2009*

• Early allergen feeding: *Can early introduction of egg prevent egg allergy in infants?* *Koplin JJ, et al. JACI 126:807-13, 2010*
Preventing AD

• Primary prevention
  – Recognize babies genetically at risk; e.g. Ichthyosis/xerosis, family atopy
  – Alert parents to watch for irritant dermatitis
  – Treat irritant problems aggressively

• Secondary prevention—when AD is already evident
  – Clear with effective, remittive, topical therapy
  – Maintain remission with biw TCS, qd TCI, emollients
Hypothesis: Correcting Early Skin Barrier Dysfunction May Prevent or Delay the Development of Atopic Dermatitis

- A pilot study of emollient therapy for primary 43.4% developed eczema in the control group
  - 21.8% in the emollient group
- Incidence of eczema at 6 months (ITT)
  - OR 0.33, P < .0001

Why not recommend early emollients in atopic families?
Faulty Management Choices for AD

- Conservative TCS dosing
- Start/Stop instead of Tapering
- Diluting TCS with emollients or topical antibiotics
- Failing to pre-hydrate
- Prolonged antibiotic therapy
- Diversions from optimal therapy
  - Antihistamines
  - Allergy obsessions
Antihistamines and Childhood AD

• An indirect therapeutic aid in easing allergic comorbidities
  – Allergic rhinoconjunctivitis
• Enhancing sleep and reducing night awakenings due to itch
• A negative feature is diversion from effective topical antiinflammatory therapy
• No studies in 70 years that show an anti-pruritic effect