Management of children and adult patients with atopic dermatitis

From pathogenic insights to therapy

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Impaired skin barrier and Th2 inflammation

1 Defective epidermal barrier

2 Environmental triggers

3 Skin inflammation

4 Pruritus

Environmental triggers:
- Defective epidermal barrier

CD4, CD8:
- IL-4
- IL-5
- IL-13
- IFN-γ

DC, IgE:
- Y

B cells, IgE:
- Y

Eosinophils:

Mast cells:
1 Restoring the skin barrier

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

• Vitamin D3
  – binds to vit.D responsive element on cathelicidin promotor
    – production of cathelicidin ↑
  – 14 AE vs. placebo; cholecalciferol 4000IU/d, 21 days
  – Increase of cathelicidin expression in AE skin
  – Cave: topical vitamin D might induce TSLP

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

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 in allergic patients

• Corticosteroids
  – Short term therapy
3 Anti-inflammatory therapy

- **Topical:**
  - Corticosteroids
  - Calcineurin inhibitors

- **Systemic:**
  - UV Therapy
  - Cyclosporine, corticosteroids, azathioprine, MMF, MTX
  - Alitretinoin
  - Biologics

- **Proactive therapy**
  - Corticosteroids
  - Calcineurin inhibitors
4 Anti-pruritic therapy

• Emollients
  + polidocanol
  - Wet wraps
• Effective anti-inflammatory therapy
• Antihistamines
• Cold packs
• Psychological interventions, relaxation
• Educational programmes
  – for patients and parents