Atopic dermatitis

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Eczema / dermatitis is an inflammatory skin reaction
Dermatitis

Atopic

Phototoxic

Contact

Photoallergic

Seborrhoeic

Irritant
Atopic dermatitis

- AD: Specific form of dermatitis with strong association with atopy
- Eczema: “To boil over”: Chronic disease with recurrent exacerbations
- Common in developed countries (15 – 20 %) less common in developing countries (0 – 5 %)
- Specific morphology, distribution and evolution
Aetiopathogenesis

- Genetics
- Atopy
- Environmental factors
- Skin barrier disruption
Outside-inside / Inside-outside

• **Outside – inside hypothesis**
  – Xerosis and abnormal permeability of skin barrier drives eczema with secondary sensitisation

• **Inside – outside hypothesis**
  – Inflammatory responses to irritants and allergens drives eczema with secondary barrier disruption:
The brick wall model of the skin barrier
Diagnosis

• Typical lesion morphology
• Typical distribution
• Typical onset and evolution
• Chronic, relapsing course

• Pruritis
• Atopy
Lesion morphology

<table>
<thead>
<tr>
<th>Acute</th>
<th>Vesicular</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Erythroderma</td>
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<tr>
<td>Subacute</td>
<td>Erythema</td>
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<tr>
<td></td>
<td>Scaling</td>
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<tr>
<td></td>
<td>Crusting</td>
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<tr>
<td>Chronic</td>
<td>Crusting</td>
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<td></td>
<td>Lichenification</td>
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Distribution and evolution

Infantile  
- Face involved, nappy area spared
- Acute type lesions
- Koebner phenomenon: linear changes where scratching has occurred
- Knee involvement from crawling
## Distribution

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Description</th>
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| Infantile | Face involved, nappy area spared  
             Acute type lesions  
             Koebner phenomenon: linear changes where scratching has occurred  
             Knee involvement from crawling |
| Childhood | Acute and chronic lesions  
             Flexoral  
             Sides of neck  
             Discoid patches  
             Lichenification |
| Adulthood | As above  
             Areas of sweating  
             Vulva, nipples  
             Follicular accentuation |
Pruritis

Hallmark of AD: “An itch that rashes”.

Subconscious scratching

Excoriation, secondary infection

Irritability, restlessness, poor sleep, poor school performance

Inflammatory changes and lichenification

Old (sedating) antihistamines good for treatment of AD itch.
Patient education

• Aetiology and pathology
• An explanation of the chronic and relapsing nature of the disease
• An explanation that all therapies are able only to treat the condition but not cure it.
• A discussion about the scientific basis of alternative therapies
• Honest discussion about medication side effects.
Patient education

- Avoidance of irritants and individual allergens
- Skin hygiene and care.
- Itch prevention (avoiding hot bedrooms, cutting of nails, avoidance of woollen and other rough, scratchy clothing, avoidance of overdressing, avoidance of soaps and adequate moisturisation)
Treatment strategy

- According to severity
- Education and information
- Written care plans
- Stepped approach to management
- Addressing the barrier defect
- Treating the inflammation
- Treating sequelae
The Three Steps of Atopic Eczema Management

Step 1: Complete Emollient Therapy

Step 2: Identification and avoidance of allergens/triggers

Step 3: Treatment of flare with topical CS and CI

Please & not Assemble

education
Phases and treatment of Atopic Dermatitis

- **Acute phases (flare-ups):** Topical steroids

- **Interval (non-acute) phases:** Emollient treatments
Every day treatment

• Avoid triggers and aggravators
• Use bath oils and wash with soap substitute: cleansing oils or creams
• “Moisturize” – use a good emollient at least twice a day
• Clothing: non scratch clothes, gentle soaps, no washing powders
Emollients

• Oil based emollients are effective for restoring skin barrier function in AD.
• Creams and lotions may be less effective than ointments due to the lower proportion of oil.
• Different preparations are suitable to use at different times
• Emollients should always be used even if the skin is clear
Every day treatment

• Education of caregivers is key
• Early use of steroids
• Adequate steroid use
• Non-steroid therapies: Topical calcineurin antagonists
  – tacrolimus (Protopic)
  – pimecrolimus (Elidel)
TCIs

• Skin selective inhibitors of inflammatory cytokine production by T-cells

• Less potent than steroids but few side effects
  – may cause transient “burning”
  – minimal percutaneous absorption
  – no adverse systemic effects
  – no potential for skin atrophy
  – no increased risk of skin infections
  – well tolerated especially in delicate skin areas like the face and neck folds
Flaring treatment

- Continue with the every day treatment
- Steroid ointments
- Wet wraps or dressings
- Cool compresses
- Antibiotics
Use of topical steroids

• Benefits outweigh the risks
• Potency tailored to severity
• Mild potency for face and neck
• Exclude secondary infection
• Not for prolonged use on normal skin
• Wean off to lowest possible strength
Treatment of Acute flare

**Trunk and extremities**:
- Moderate potency steroid (0.1 % Betamethasone : Lenovate)
- Taper steroids → emollient or tar

**Face**:
- Mild potency steroid (1% Hydrocortisone : Procutan)
- Emollient

**Scalp**
- Betadine shampoo or cream
- 10 % steroid scalp lotion
- Tar shampoo or olive oil

**Antipruritics (sedatives)**
- Hydroxyzine (Aterax) 2mg/kg/day.
- Promethazine (Phenergan) 0.5mg/kg
Treatment of Secondary Infection

**Bacterial**: B-haem strep or Staph aureus

Swab if chronic

Empiric treatment with Flucloxacillin or erythromycin for 10 days
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**Eczema herpeticum** : Herpes Simplex
Vesicular eruption with umbilicated center in atopic individual.
Symptomatic treatment
Acyclovir
Second line treatment for AE

• Wet dressings
• Bath oils
• Anti-staph approach
• Bleach baths
• Admission to hospital
Wet Dressings

- Reduce the itch
- Cooling and soothing
- Moisturise the skin
- Protect the skin
- Promote sleep
Third line treatment for AE

- Refer to specialist
- Dietary manipulation
- UV light
- Ciclosporin
- Methotrexate
- Azathioprine
- Oral Steroids
Contents

• Introduction
• Aetiopathogenesis
• Diagnosis
• Severity
• Education
• Treatment stages

• Everyday
• Flares
  2\textsuperscript{nd}/3\textsuperscript{rd} line
  • Topical calcineurin antagonists
• Food and AD
• Prognosis
Calcineurin inhibitors

- Calcineurin inhibitors are cell-selective inhibitors of inflammatory cytokine synthesis / release:
  - selectively target T cells and mast cells
  - no effect on keratinocytes, fibroblasts, endothelial cells, Langerhans’ cells
  - no induction of skin atrophy
  - permeate much less through skin than corticosteroids

- However: Less effective
Calcineurin inhibitors

- Useful on very delicate areas eg eyelids
- May be used as maintenance therapy
- Should always be combined with adequate emollient therapy
- Not as effective as topical steroids for treatment of flares
Eczema and foods

• The relationship between atopic dermatitis and food allergy is complex and not always causal
• 60-80% of children with severe AD are sensitised to foods
• True allergy: Children 35-40% vs Adults 2%
• Much lower proportion in primary care
• More likely if: Moderate or severe, associated other symptoms, not responsive to treatment, early onset
• Both food-specific IgE-mediated and cellular mechanisms
Eczema and foods

• Egg most common
• Foods can lead to immediate non-eczematous reactions
  – Immediate non ecematous reactions can themselves aggravate eczema (scratching)
• Foods can lead to intermediate flares of eczema
• Specific avoidance of foods can lead to longer term improvement of eczema
Spectrum of reactions

DBPCFC to milk, egg, wheat and soy in children. Median age 2 years with AD and suspected FA.

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Spectrum of reactions in primary care?

Assuming a 5% food allergy rate

Pure speculation journal
Eczema and foods

- Test before exclusion: Severe cases, early onset, associated acute reactions
- No role for indiscriminate or long term exclusion of important foods
- Needs good history + food diary
- Avoidance guided by tests or very rarely exclusion diet with rechallenge
- Dietician in team. Supplement vitamins, calcium.
Eczema and foods

- Avoidance guided by RAST, SPT + APT
- Removal $\rightarrow$ resolution; reintroduction $\rightarrow$ exacerbation
- May require elemental diet + slow reintroduction ... always under a dietician for short periods
- Never exclude > 6 weeks without rechallenge
- Challenges may need to include responses for delayed type reactions + objective measures eg SCORAD
Prognosis

• Tendency to improve at 3 to 5 years
• Three stages of distribution of rash
• 75% remission by 10 – 14 years
• Complete remission at 4th to 5th decade