1. ? Diagnosis
2. Diagnosis
3. ? Diagnosis
4. Diagnosis
5. ? Diagnosis
THE CHALLENGES OF DERMATITIS

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Dermatologist
DERMATITIS

- ENDOGENOUS
- EXOGENOUS
- BOTH
ENDOGENOUS

- ATOPIC DERMATITIS
- SEBORRHOEIC DERMATITIS
- STASIS/VARICOSE DERMATITIS
- NUMMULAR (DISCOID) DERMATITIS
EXOGENOUS DERMATITIS

- CONTACT DERMATITIS
  - IRRITANT
  - ALLERGIC

- PHOTOREACTIVITY DERMATITIS
BOTH

- HAND DERMATITIS
- ASTEATOTIC DERMATITIS
ATOPIC DERMATITIS
(ATOPIC ECZEMA)

A Wholistic approach

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ATOPIC DERMATITIS

- Does not have a single cause
- Monotherapy does not exist!!
- Aetiology and exacerbations are multifactorial in origin
ATOPIC DERMATITIS

- Is not an allergy
- People with atopic dermatitis have an increased incidence of allergies
ATOPIC DERMATITIS

- Patients are frequently very uncomfortable
- Often aggressive treatment is required
ATOPIC DERMATITIS

- Genetically determined
- 70+% have a FH of atopy
- 10 – 20% of <5’s have ADs
- Increasingly prevalent in industrialised temperate countries.
- Occurs in first year in 85%, 95% before 5 yrs
ATOPIC DERMATITIS

- >80% US children → second decade

- % of children with mod severe Ads increases with age, esp after 3 yrs
Comorbidities

- incl. depression, anxiety, ADD/ADHD

- keratoconus, cataracts/glaucoma

- incr’d accidental/traumatic injury (?sleep deficit)
ATOPIC DERMATITIS

- Spontaneous improvement occurs
- Many carry the disorder into adulthood
- Underlies 80% of occupational dermatoses
CAUSES OF ATOPIC DERMATITIS

- Skin structurally different
- Skin ‘biochemically’ different
- Immunological changes.
CAUSES OF ATOPIC DERMATITIS

- Greatest risk factor for ADs is FLG mutations
STRUCTURAL CHANGES

- Impaired skin barrier function - fillagrins
- Xeroderma
  - Keratosis pilaris
  - AD ichthyosis (ichthyosis vulgaris)
  - Pityriasis alba
  - Denny-Morgan folds
‘BIOCHEMICAL CHANGES’

- White dermographism
IMMUNOLOGICAL CHANGES

- Up to 80% have increased IgE
- Some decrease in cell mediated immunity
- Fundamental imbalance between the response of TH1 and TH2 lymphocytes
IMMUNOLOGICAL CHANGES

- Onset of acute disease is associated with Th2/IL22 cytokine activation

- Chronic lesions show intensification of TH1, Th2, and IL22 activation

- (NBUVB & CyA decr. Th2 & IL22 cytokine expression)
PRACTICAL CAUSES AND AGGRAVATING FACTORS

- Xeroderma
- Irritants
- Climatic changes
- Infection
- Food and other allergies
IRRITANTS

- ‘Usual’ irritants
- Soaps, soap powders, detergents.
- Chlorine in swimming pools
- Fibres
- Friction
CLIMATE CHANGES

- Hot or cold temperatures
- Sudden changes in temperature
- Low humidity
INFECTION

- Staph. Aureus most important
- Uninfected atopic skin has increased number of Staph.
- May act as a ‘superantigen’ activating lymphocytes
STAPH AUREUS

- 80 – 90% harbour S. aureus in active sites
  - poor barrier
  - decr’d antimicrobial peptides
STAPH AUREUS

- Decrease in normal flora in flares & inc’d S. aureus
- S aureus increases Th2 % IL22 response
- Decrease in S. aureus $\rightarrow$ decrease in severity of ADs
STAPH AUREUS

- Containers may be contaminated

- 65% parents carry same strains of S. Aureus as their children with ADs

- Pets may have same microbes, incl. MRSA
FOOD AND OTHER ALLERGIES

- Literature on role of food allergens is extensive, controversial, & contradictory
- Majority have +ve skin prick tests or RAST/EAST tests to foods
- Very few appear to be relevant
- Small % aggravated by certain foods
- Most common: cow’s milk, eggs, soy, peanuts, wheat, & fish + tree nuts, shellfish & sesame
- Maternal dietary restriction not effective unless diagnosis of food allergy already established

- No strong evidence of protective effect of 3/12 exclusive breast feeding wrt food allergy

- Introduction of solids from 4 – 6 months may allow immune system to become tolerant and prevent food allergy

- Early exposure to peanut, wheat, egg, and cow’s milk may be protective

- Delayed initial exposure to cereals until after 6 months may increase risk of developing wheat allergy
FOOD ALLERGIES

- No reason to delay introduction of potentially allergenic foods in 4 – 6 month age group, including those with siblings with food allergies.

- Introduce a new food every 2 – 3 days
FOOD AND OTHER ALLERGIES

- Ask parents!

- RAST or skin prick tests often more useful if negative
FOOD ALLERGIES

- Only 50% with +ve skin prick test have a positive allergy challenge test.
SKIN PRICK TESTS

- > 8mm wheal $\rightarrow$ 95% chance of allergy
- 3 – 8 mm wheal $\rightarrow$ 50% chance of allergy
- <3 mm wheal – likely to be tolerant
Does ADs increase the risk of food allergy?

- Risk of challenge proven food allergy in the first year of life
  - no ADs – 4.1%
  - Ads – 20.9%

- Egg 6X, peanut 11X, sesame 40X
? Fillagrin decr. plays a role in development of food allergy, ? Increases risk of food sensitisation
FOOD ALLERGY

- Early onset and more severe ADs → 5X risk of development of food allergy
FOOD AND OTHER ALLERGIES

- House dust mite
- Grasses, pollens, etc.
PATTERN - INFANCY

- Xeroderma
- Face and scalp often first involved
- Trunk and limbs, especially flexures
- Nappy rash
- Lichenification seldom seen
- May be exudative
- Infection
PATTERN - CHILDHOOD

- Xeroderma
- Often exudative
- Flexural
- Periorbital
- ‘Lip-lick’ dermatitis
- Lichenification frequent
- Excoriation & crusting common
- Infection
PATTERN - ADULTHOOD

- Xeroderma
- Lichenification
- Face and neck frequently involved
- Hand dermatitis
- Erythroderma
COMPLICATIONS

- Family dynamics!!
- Eczema herpeticum
- Eczema coxsackium (CSK A6)
- Molluscum contagiosum
- Occupational dermatoses
Wizard of ID

Are there any special restrictions on these?

Prescriptions

You can't eat, drink, smoke or make love.

Prescriptions

Take 'em yourself.
TREATMENT

- Time consuming
- ‘Consumer’ resistance
- Unhelpful advice
- The latest ‘magic cure’
- Steroid (& antibiotic) phobia
- Avoid poor dietary nutrition
TREATMENT

- Aggressive treatment early to show ‘something can be done’
- Explanation re the nature of the disease
- A plan for the various phases of ADs
TREATMENT – AVOID IRRITANTS

- Soaps
- Soap powders
- Clothing fibres
- Extremes of temperature
- Low humidity environments
TREATMENT - XERODERMA

- Emollients
- Emollients
- Emollients
- Emollients
EMOLLIENTS

- Alpha Keri, BK, DP, Hydroderm lotions
- Glyc. 10% in Aqueous or sorbolene cream
- (Urea 10% creams)
- QV, Cetaphil, Aveeno, etc. creams
- Lipobase, healthE Fatty Cream
- Lipobase Repair
- WSP, Emuls. Oint
- Bath oils
TREATMENT - ANTIHISTAMINES

- It is the sedation rather than the antihistamine action which is helpful at times. Use short term

- If frequent wheal and flare reactions

- (Ketotifen)
TREATMENT - ANTIBIOTICS

- Cover Staph aureus ( +/- beta haem strep)
- Required if exudative and crusting
- Occasionally needed medium to long term
- Give parents a script or repeats to have ‘on hand’
- Bleach baths
BLEACH BATHS

- 2 X per week

- 1cc per litre (1/2 cup per full bath)

- Na hypochlorite gel for shower

- Is Na hypochlorite anti-inflammatory as well as anti-bacterial?
TREATMENT – TOPICAL CORTICOSTEROIDS

- Extremely effective in treating and controlling ADs
- Very safe if used appropriately
- Use mod. potent or potent short term
- Use mildly potent for maintenance if required
- Safe to use in infected dermatitis (bacterial) if infection also being treated
TREATMENT – TOPICAL CORTICOSTEROIDS

- Dermol & Diprosone OV
- Betnovate, Nerisone, Diprosone, Elocon, Advantan
- Locoid
- Aristocort, Eumovate
- Hydrocortisone 1%
TREATMENT – TOPICAL CORTICOSTEROIDS

- Aggressive therapy, especially early on

- Proactive treatment as maintenance, esp early on, 2 – 3 X per week leads to overall decrease in TCS use.
TREATMENT – TOPICAL CORTICOSTEROIDS

- The base is important!
- Ointments better then creams
TREATMENT – IMMUNOMODULATORS

- Topical – tacrolimus
  - pimecrolimus
- Oral – cyclosporin A
  - methotrexate
  - azathioprine
  - mycophenolate
METHOTREXATE

- Effective to very effective in 75% of 31 ADs paed patients
- Median duration of use 14/12
TREATMENT – DIET

- Avoid foods known to flare or aggravate patient’s ADs
- Dietician’s advice
TREATMENT – OTHERS

- Tar preparations
- Evening primrose or flax seed oil
- Probiotics
- Chinese herbs
- Narrowband UVB
- Immunosuppressants – azathioprine, methotrexate, cyclosporin, mycophenolate
TREATMENT – OTHERS

- Biologics – dupilumab – anti IL4 receptor antagonist
- Oral or topical PDE4 inhibitors to suppress immune stimulation