Practical tips in the treatment of eczema. Part II

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Highlights / Hoogtepunte

- Complications of topical corticosteroid use and the treatments thereof.
- Tips on treating pruritis.
- Second line therapy of eczema.
- Komplikasies van lokale kortikosteroïedbehandeling en die behandeling daarvan.
- Wenke vir die behandeling van pruritis.
- Tweede linie middels vir ekseem.

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INTRODUCTION

This is the fourth article in a series on eczema. The first 2 articles were on the diagnosis of contact eczema and atopic eczema. The previous article discussed practical aspects of the different treatment modalities. This article discusses the side effects of topical steroids and how to treat them, tips on treating pruritis as well as other second line therapies.

SIDE EFFECTS OF TOPICAL STEROIDS

The unwanted effects of topical steroids are directly related to their potencies.

1. Epidermal effects include:

a. Epidermal thinning – which is associated with a decrease in epidermal kinetic activity.
b. Decrease thickness of keratinocyte layer.
c. Flattening of the dermo-epidermal convolutions.
d. Melanocyte inhibition, a vitiligo-like condition, has been described. This occurs more commonly with steroids under occlusion or intralesional steroids.

2. Dermal effects

Reduced collagen synthesis as well as ground substance result in the formation of striae and easy rupture on trauma due to poor support of vasculature.

3. Vascular Effects

Fixed Vasodilatation

Corticosteroids at first produce vasoconstriction of the superficial small vessels, followed by a phase of rebound vasodilatation, which in later stages is fixed.

Rebound phenomenon

As vasoconstriction wears off, the small vessels overdilate allowing oedema, enhanced inflammation and sometimes pustulation.

4. Side effects of systemic absorbtion

- Inhibition of the pituitary-adrenal axis by excessive application of moderately potent or stronger steroids is well documented.

  Recommended weekly dosage is less than 50 g of superpotent steroids, and less than 100 g of potent steroids. In addition, prolonged usage at this level is best avoided.
- Cushingoid features may be seen in infants inappropriately treated.
- Stunting of growth in a child treated with long-term fluorinated steroids has been observed, but the weaker steroids are safe in children.
- Posterior subcapsular cataracts and glaucoma are other hazards.
- Allergic contact dermatitis to the steroid molecule may also occur.
- Iatrogenic clinical syndromes due to topical corticosteroids:

  a. Perioral dermatitis.
  b. Tinea incognito – the clinical signs of fungal infection are obscured due to reduction of inflammation.
  c. Impetigo incognito – topical steroids may impair the host's immunological response against the infection.
  d. Infantile gluteal granuloma – violaceous red nodules occur in infants wearing diapers due to an alteration of host response to candida.
  e. Pustular psoriasis – where topical steroids are used to treat chronic plaque psoriasis where the rebound phenomenon following cessation of treatment leads to the onset of an acute pustular stage.
  f. Steroid rosacea can follow long term corticosteroid application, and when used for longer than 1 month, may exacerbate pre-existing acne, induce steroid acne or cause reversible hypopigmentation.

The most common localised side effect, though, is skin atrophy – frequently on the flexural areas – and results in telangiectasia, striae, purpura and skin fragility.

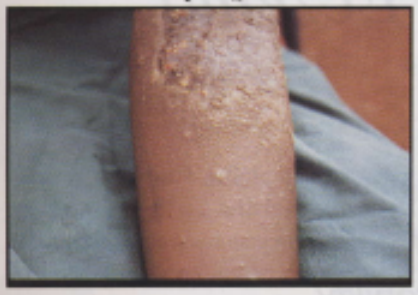
5. Superinfections

Bacterial

Colonisation with *Staphylococcus*

aureus is almost always present in patients with atopic dermatitis. Clinically this may manifest as pustulation, purulent exudation, purulent crust formation and oozing. Whithout these obvious signs of impetiginisation, colonisation may still be present and have a role in the maintenance of inflammation. It has been suggested that staphylococcal exotoxins may induce IgE and thus have a role in IgE mediated inflammation in atopic dermatitis.

Figure 1: Atopic eczema with superimposed pyogenic infection – impetiginisation



Antimicrobial and antiseptic therapy.

In cases of clear pustulation a course of systemic antibiotics may be given. Often s. aureus is penicillin resistant and flucloxacillin or erythromycin is the agent of choice. Mixed infection with streptococci is common and flucloxacillin is a second choice in those cases.

When secondary impetiginisation is recurrent, antiseptic therapy should be considered. The use of betadine iodide and chlorhexidine in soap is one approach.

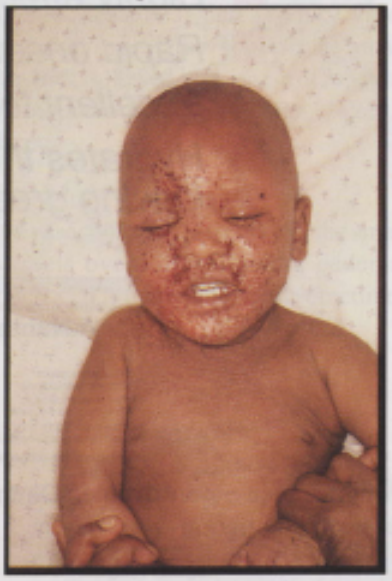
Combinations of steroid-antimicrobial agents have not in the past been readily accepted by dermatologists because the broad spectrum of therapeutic activity encourages laxity in diagnosis. The flexural areas, for which combination treatment is often advocated, provide conditions in which the likelihood of adverse effects is increased. The combination treatments may be used in eczema with proven secondary infection but many dermatologists still prefer to give an antibiotic systematically.

Additives, preservatives, fragrances, stabilizers and antioxidants are components of many topical preparations and adverse reactions to any of these should be considered in a patient whose dermatosis exacerbates during therapy. True allergy to the steroid itself may also occur.

Eczema Herpeticum (Kaposi's varicelliform eruption)

Infection with herpes simplex virus is another effect of the loss of effective local immunity in atopic dermatitis. Excema herpeticum is such a manifestation of cutaneous dissemination of herpes lesions. It may occur in other conditions, but is mainly associated with disseminated atopic dermatitis. In most cases it starts on the face with rapid dissemination in a more or less symmetrical distribution.

Figure 2: Atopic eczema in a child complicated by infection with herpes simplex virus – eczema herpeticum



Treatment with acyclovir should be given as early as possible. It will inhibit further vesicle formation and the patient will feel better in a couple of hours. In limited cases 200 mg 5 times daily for 5 days is advised. If intravenous acyclovir is given, a dosage of 5 mg/kg every 8 h or 250 mg/m^2 body surface area for 5 days is advised.

Pityrosporum Orbiculare

Finally, it has been suggested that the yeast P. orbiculare superimposed an atopic dermatitis may also be important in the maintenance or exacerbation of eczematous lesions. Atopic patients may produce P. orbiculare specific IgE and ketoconazole treatment may lead to improvement of the eczema and even decrease of IgE levels. The precise role of anti-yeast therapy in the management of atopic dermatitis, however, remains to be investigated.

Other first line therapy

Intralesional Steroids

A few recalcitrant dermatoses eg. nodular prurigo and lichen simplex, may respond to injection of steroid into the lesion. Triamcinolone is often used but dermal atrophy and leukoderma may occur. Blindness has been reported following intralesional injection of the eyebrow skin.

Ichtammol and tar

Preparations containing ichtammol and coal tar may be helpful as a maintenance treatmant in patients with lichenification, preferably a 1-10% coal tar solution in an appropriate vehicle, rather than crude coal tar. Localised areas can be treated with bandages impregnated with these agents applied overnight.

TIPS ON TREATING PRURITUS

We still do not understand which mediators elicit the intense itch in atopic dermatitis although most today suspect interleukin-2 as being important, which is a cytokine released from activated T-lymphocytes. Treatments that inhibit activated T-lymphocytes will always reduce itch, and cyclosporin, tacrolimus and ascomycin, which specifically inhibit the release of interleukin-2, do so.

Histamine does not play a major part, although urticarial symptoms can be of some importance, if illicited via specific type I allergies. Antihistamines can be tried in adults, but their use in children is more questionable as they have fewer type I allergies. Sedative antihistamines may be helpful in improving sleep. Non-sedating antihistamines have hardly any effect on patients with atopic dermatitis.

On children under 1 year of age, antihistamines have been implicated in sudden infant death syndrome. Therefore it is not advised to use antihistamines in these youngsters. In pregnancy antihistamines may be given especially the older compounds such as promethazine.

A simple remedy to relieve itch is by cooling the skin. This means that parents should not dress their atopic child in very warm clothes and avoid high temperatures especially in the room where the child is sleeping. Use topical steroids or anti-inflammatory treatment should preferably be used in the afternoon or early evening, while emollients should be used in the morning. Clothes should be cotton and not wool. Removing the label in the neck area may help.

SECOND LINE TREATMENT

The majority of patients will respond to first-line treatment. Patients who fail to respond should be reviewed to check compliancy exclude antibiotic resistant infection, or herpex simplex infection. Only there after can second line treatment be considered.

1. Intensive topical treatment

The strength of steroid treatment can be increased for a short period as out-patient. If this is ineffective, inpatient treatment will often control severe exacerbations.

2. Wet wrap technique

For controlling severe atopic dermatitis in the young children (See part 1).

3. Occlusion

The incorporation of a corticosteroid such as flurandrenolone into the adhesive of a plastic tape with high occlusiveness enhances of the potency of the steroid by encouraging hydration of the stratum corneum. Whole body occlusion was formerly widely used, but adverse effects were so common, it fell into disrepute.

4. Allergy management

There is a great demand from patients to perform allergy testing, however allergy testing should only be done in children with severe eczema. Remember that very few children have an allergy to cow's milk or other foods, and a significant number of atopic dermatitis patients do not have allergies.

5. Diet

The only diet that has an affect on eczema is breast feeding, which will delay and even reduce the number of affected children. Diets without cow's milk and egg are much debated and convincing evidence-based studies are lacking. Also, avoidance of sugar does not improve disease activity.

Very recently prophylactic administration of lactobacillus Cg to mothers and newborn infants in high risk families was observed to reduce the clinical expression of atopic dermatitis during the first 2 years of life by almost 50%.

Further studies are needed to see if the use of such probiotics can so dramatically reduce A.D. development.

6. House dust mites

The house dust mite carries a proteolytic effect and could thus be irritating to the skin, augmenting the inflammation of atopic dermatitis.

CONCLUSION

Atopic dermatitis is not an easy disease to treat because concepts of its aetiology are too simple, and definitely not only "allergic". Your contact with parents is very important to avoid lack of compliance. Parents need time and regular follow up to secure long term treatment plans. A.D. can easily end up being a nuisance, but it should be viewed as a challenge – not only for patients / parents, but also for doctors.

In a next issue the third line therapy and new treatments will be discussed.❐

Photographs: Courtesy Prof WK Jacyk.

Please refer to the CPD Questionnaire on page 51.