

A REVIEW OF THE MANAGEMENT OF CHILDHOOD ATOPIC ECZEMA AT THE PRIMARY HEALTHCARE LEVEL

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SUMMARY

Atopic eczema (AE) is a chronic and often debilitating skin disorder with a high prevalence in young children. This article briefly highlights management strategies that can be used at a primary healthcare level to control AE severity and improve quality of life.

This article has been peer reviewed.

INTRODUCTION

Atopic eczema (AE), also known as 'atopic dermatitis', is a chronic inflammatory skin disease characterised by cutaneous hyperactivity when exposed to certain environmental triggers and involves genetic and allergic factors that, in turn, influence the immunological system.¹ AE may be defined as an itchy, relapsing, flexural skin rash on the limbs of older children and the extensor surfaces on the limbs of younger children. The rash occurred within the previous 12 months and was classified as severe eczema when associated with one or more disturbed nights per week.²

The 2009 International Study of Asthma and Allergies in Childhood³ found that prevalence rates differed between countries. The conclusion reached was that AE had reached a plateau in countries such as the United Kingdom and New Zealand.⁴ Prevalence of more than 15% was found in Africa, Latin America and Oceania.

The prevalence of AE continues to increase in low-income countries such as those in Latin America or South East Asia. South Africa has also shown an increase in AE prevalence since 1995.²

AE PREVENTION STRATEGIES

Although AE appears unavoidable in those who are predisposed to the condition, many interventions are believed to delay the onset of AE and reduce its overall prevalence.⁵

Primary prevention strategies are intended to prevent the disease from developing by avoiding the risk factors that

are known to contribute to AE development.⁵ Some of these strategies are:

- avoiding exposure to microbes;
- exposing children to dogs during the first year of life;
- breast feeding at least until six months and avoiding formula milk;
- avoiding the use of broad-spectrum antibiotics, as far as possible, and opting for narrow-spectrum antibiotics, if really necessary;
- making a conscious effort to avoid allergens and irritants.⁵

Secondary prevention methods aim to reduce the intensity of AE outbreaks and prevent further disease progression. These include:^{5,6,7,8}

- effective control of AE outbreaks and ensuring minimal inflammation to avoid deterioration of the skin barrier;
- restoration of the skin barrier function through continuous application of indicated emollients in a liberal fashion; and
- the use of topical corticosteroids or tacrolimus as maintenance therapy.

Preventing skin barrier dysfunction is currently receiving a great deal of attention and clinicians must be aware of the preventative measures being researched.

MANAGEMENT OF ATOPIC ECZEMA

AVOIDANCE OF ALLERGENS

Since patients with AE are more prone to disease flare-ups when exposed to an aggravating factor, the complete avoidance of any compound or allergen is strongly advised. Aggravating factors must be identified and removed or, if

impossible, be limited within the child's surroundings.⁹

PARENT EDUCATION

Parent education has been found to be effective in reducing the clinical severity of AE.¹⁰ In a primary care setting where newer drug therapy guidelines cannot be enforced – often due to a lack of resources – educating parents about the management of AE in children is essential and should be regarded as a first step in AE therapy.¹⁰

EMOLLIENT THERAPY

Since AE skin is characterised by dryness and impaired skin barrier function, microfissures often develop in the skin.¹¹ These microfissures allow various pathogens to penetrate the skin surface easily. Keeping the skin hydrated with ointments is of utmost importance and forms one of the cornerstones of the treatment of AE.¹² Many soaps, perfumes and detergents have also been associated with increased AE flare-ups;⁹ emollient soap substitutes, such as emulsifying ointment, should be used instead of soap and other detergents when bathing.¹³ The use of emollients in AE:

- helps to restore skin barrier function (therefore decreasing the likelihood of infection-inducing organisms from entering the skin);
- reduces pruritic symptoms;
- increases skin hydration by replacing naturally occurring skin surface oils; and
- increases the efficacy of topical corticosteroids.^{9,13,14,15}

No relevant clinical evidence currently exists that advocates the existence of a superior method of applying emollients.¹³ Good practice points for the application of emollients do, however, suggest that they should be applied:

- liberally and frequently (at least two to four times daily);
- during and after bathing;
- in the direction of hair growth (avoiding accumulation at hair bases that otherwise may lead to folliculitis).

Emollients are sensitive to bacterial contamination. Emollient dispensers should be used where possible and when they are stored in jars, patients should use a

clean spatula or spoon to remove the emollient in order to minimise bacterial contamination of the leftover emollient within the jar.¹³ The application of emollients is therefore one of the parent education aspects that is very important in the primary treatment of AE.

The use of water-based lotions in order to hydrate the skin is strongly discouraged. The chronic use of aqueous cream (BP formulation) is associated with an increased desquamatory and inflammatory protease activity. Also, changes in corneocyte maturity and size possibly indicate accelerated skin turnover when aqueous cream BP is chronically applied. A similar study found that aqueous cream BP caused a decrease in stratum corneum thickness and an increase in baseline trans-epidermal water loss. These detrimental effects on the skin barrier function are attributed to the surfactant used in aqueous cream BP, sodium lauryl sulphate – a known irritant that adds insult to injury to those suffering from AE.¹⁷

TOPICAL CORTICOSTEROIDS (TCS)

The use of TCS in AE has been extensively researched and large systematic reviews found positive patient therapy outcomes with the use of TCS compared to placebos.¹⁸

Topical corticosteroids are divided into four different classes according to their anti-inflammatory potency. These classes are: mild, moderately potent, potent and very potent.¹³ The potency of each corticosteroid is a function of its molecular structure and not the concentration used.¹³ Therefore, the selection of the corticosteroid for the treatment of AE must be based on the potency of the corticosteroid.¹⁹ The vehicle in which the corticosteroid is dispersed is also of importance. Ointments are most preferable, followed by creams and, lastly, lotions.¹⁹ Table II describes the corticosteroids with the products available, including their topical potencies with classifications and duration of action.^{20,21}

High-potency steroids should not be used for longer than three weeks when there is a severe AE flare, and their use should be restricted to thickened AE lesions only. There is no benefit to applying TCS more than once

TABLE I: TABLE WITH TYPES OF EMOLLIENT IN SOUTH AFRICA^{13,16}

TYPE	EXAMPLES	DESCRIPTION
Emollient creams	Cream E45®, Pure cream®, Nutraplus®, Eulactol Cream®, Cetomacrogol Cream	Creams are designed as left-on products for the skin and soak into the skin faster than ointments. They can easily be applied during the day, unlike ointments.
Emollient ointments	50% white soft paraffin in liquid paraffin, Epaderm®, Emulsifying ointment, Epizone E®, Eucerin®, Epimax®, SBR®, Vaseline®, HEB Simplex®	These products are more effective than creams in keeping the skin hydrated. The drawback is that their greasy properties cause discomfort. Nightly applications are recommended in order to avoid discomfort.
Emollient soap substitutes	Cetraben®, Oilatum®, Cetaphil®, Eucerin® bath and shower oil, Emulsifying ointment	These products contain emollient ingredients with very mild emulsifiers. They are used instead of soap when bathing. These products do not lather like soap.
Bath additives	E45® Emollient Bath Oil®, Oilatum®, Emulsifying ointment	These products are added to bath water, into which they disperse. The emollient ingredients tend to stick to the skin and form a thin film over the skin, which moisturises the skin.

TABLE II: TOPICAL CORTICOSTEROID PREPARATIONS AVAILABLE IN SOUTH AFRICA^{20,21}

DRUG (ONLY TOPICALLY ACTIVE)	TRADE NAMES IN RSA:	TOPICAL POTENCY (ANTI-INFLAMMATORY)	DURATION OF ACTION	POTENCY CLASSIFICATION
Hydrocortisone acetate (cortisol)	Stopitch®, Mylocort®, Dylocort®, Skincalm®, Biocort®, Vari-Hydrocortisone®	1	Short to medium	Mildly potent
Methylprednisolone	Advantan®	5	Short to medium	Moderately Potent
Betamethasone	Persivate®, Betnovate®, Lenovate®, Repivate®, Vari-Betamethasone®	10	Long acting	Potent
Momethasone	Elocon®	10	Long acting	Potent
Clobetasol	Dermovate®, Dovate®, Xenovate®	>10	Long acting	Very Potent

daily during acute AE exacerbations, except when there is inadequate response to once-daily applications of TCS.¹⁹ The application frequency may then be increased to twice daily.^{13,15,22} Twice-weekly application of TCS in adjunct to emollient-based maintenance therapy following stabilisation of acute AE flare showed a significant reduction in the AE relapse rate.¹³ Emollients reduce the amount of TCS needed during therapy.^{14,15} Parents should be educated to apply only the amount of topical corticosteroids needed to adequately cover the areas of inflamed skin.¹⁴

Maintenance therapy with TCS in AE is difficult, due to the fact that constant use of TCS has been associated with many localised adverse effects. These include skin thinning, bruising, perioral dermatitis, folliculitis, pruritus and an increased fungal infection risk.¹⁸ Some of the reported systemic effects due to long-term TCS use include hypothalamic-pituitary-adrenal (HPA) axis suppression, growth retardation and other adrenal abnormalities. As a result, clinicians have tried to limit the use of TCS in children.¹² The adverse effects of TCS are linked to the following aspects: the concentration applied, the amount applied, the frequency of TCS application and the length of TCS application.¹⁹ Owing to the possible adverse effects, it is advised that AE patients receiving TCS therapy should be reviewed at three to six monthly intervals.^{18,19} Education of the parents at the primary healthcare level regarding the correct use of TCS is very important.

TOPICAL CALCINEURIN INHIBITORS (TCI)

Topical calcineurin inhibitors belong to the immune-modulant drug class and have established efficacy in treating AE, although they are not as effective in managing AE as TCS.¹³ TCI treatment should be considered only if TCS treatment is contra-indicated or causing adverse effects. The TCIs available in South Africa include tacrolimus and pimecrolimus.²¹

SYSTEMIC IMMUNOSUPPRESSANT THERAPIES

Azathioprine, methotrexate and cyclosporine A are used in chronic, severe cases of AE and can be prescribed

and monitored only by specialists.²³ This treatment option therefore does not form part of the primary healthcare treatment of AE.

ANTIHISTAMINES

There has been some controversy regarding the use of antihistamines in AE.¹³ The role of antihistamines in AE in managing pruritic and inflammatory effects make it appear as a logical choice for at least partially managing AE, but there is little data to support the use of antihistamines in AE.^{24,25} The use of antihistamines in AE is mostly related to the sedative properties of the first-generation antihistamines. There is not enough evidence found in a systematic review done in 2013 that oral H₁ antihistamine as monotherapy has an effect on atopic eczema,²⁶ but it could be used as an add-on therapy due to its sedative effects.^{13,19,26}

TOPICAL ANTIHISTAMINE USE

Topical antihistamine use has delivered neutral results with regard to AE improvement and is generally not recommended because of the excipients within the topical formulation and the possibility that these excipients may exacerbate AE.²⁷

STAPHYLOCOCCUS AUREUS (SA) MANAGEMENT IN AE:

The majority of individuals diagnosed with AE are colonised with SA, which is known to exacerbate AE flares. A means of managing SA colonisation and preventing acute flares due to SA is sought after by many researchers.²⁸ No advantage has been shown with the use of oral antibiotics, topical antibiotics, topical antibiotic-corticosteroid combinations, fabrics with antibacterial properties, antibacterial soaps, topical antiseptics, antibacterial bath additives and silver impregnated textiles.²⁸ This outcome was concluded to be due to the fact that many of these methods reduced the bacterial load, but that the SA load returns within two months.²⁸ Larger randomised control studies are needed to investigate the treatment potential within many of these methods.

CONCLUSION

AE is a complicated skin disorder that can lead to a

significant decrease in a patient's quality of life. AE originates due to various risk factors. Treatment with TCS forms the mainstay of therapy. Adjuvant use of emollients increases the effectiveness of TCS and decreases the amount of TCS needed. Chronic application of emollients is recommended to prevent deterioration of skin-barrier function. The choice of TCS and emollient therapy, when treating AE, will have a direct effect on whether therapeutic outcomes are achieved or not. Choosing the TCS and emollient, as well as parent education on the correct use

of these treatments, is imperative at the primary healthcare level. Other forms of therapy to manage AE should be considered only if TCS and emollient therapy are not sufficient to control acute AE flares. But AE often needs referral from the primary healthcare level to other levels of care.

DECLARATION OF CONFLICT OF INTEREST

The authors declare no conflict of interest.

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