DOI: 10.1111/j.1468-3083.2012.04713.x JEADV

CASE REPORT

Successful treatment of widespread eczema and sleep disruption in a 7-month-old infant with methylprednisolone aceponate 0.1%

A. Torrelo*

Department of Pediatric Dermatology, Hospital del Niño Jesús, Madrid, Spain
*Correspondence: A. Torrelo. E-mail: atorrelo@aedv.es

Abstract

A 7-month-old infant, with no known food allergy or intolerance, presenting with eczema lesions affecting many parts of his body, was diagnosed with infantile eczema. A skin barrier defect, due to filaggrin deficiency, was suspected. Prior treatment with emollients was not effective and produced discomfort. After twice-daily application of methylprednisolone aceponate (MPA) 0.1% (Advantan®), in addition to temporary withdrawal of emollients, for 10 days. At the follow-up examination, improvements in all eczema lesions, scratching behavior and sleep quality were observed, and eczema was considered almost clear and under control. Advantan was prescribed to control new flares of eczema, and heavy emollients were recommended to treat residual dryness and damage from original lesions. No side effects of Advantan were reported. In an infant with newly diagnosed eczema, Advantan was safe and effective in alleviating lesions and scratching severity and restoring sleep.

Introduction

Infantile eczema is very common, and is estimated to affect 10–15% of all infants worldwide. Despite the frequency of the condition, eczema in infants is a heterogeneous skin disorder. It may appear under many different clinical forms, ranging from a few scattered plaques to diffuse erythroderma. It may affect any part of the skin, with flexural involvement or flexural sparing. It may take the appearance of an exudative eruption or a 'dry', scaling eczema. It may be associated with severe itch or with no itch at all. Eczema in infants may be due to many different causes. The most common is the classical 'atopic eczema' that is commonly associated with barrier defect due to filaggrin mutations. In other cases, no overt barrier defect is observed, and patients show completely normal looking skin in the areas not affected by eczema. Irritative eczema is also common in infants, especially in the perioral region and the neck. Flexural eczema in infants is more related to contact with irritant products, in toddlers with abundant fatty tissue and deep skin creases, than to atopy.

Patient history

A 7-month-old male infant presented with eczema affecting many areas of his skin, including chest, arms, forearms and legs. The lesions had appeared 3 months before, and had spread slowly. The child was otherwise healthy, and thrived normally; anamnesis did not reveal any suspected food intolerance or allergy. However, the child's sleep was disturbed, and he scratched often.

The infant had been managed with topical emollients, with no improvement, and seemed to show discomfort following emollient application.

Clinical examination

A faint erythema and minimal scaling covered most of his skin. There were areas of skin cracking, minimal fissuring with a reticular appearance and slight exudation, located mainly on his

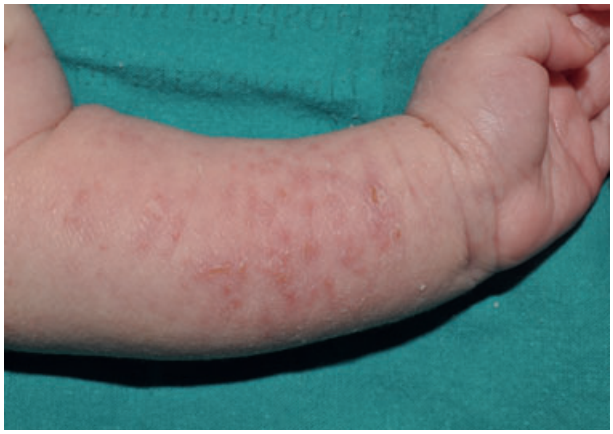

Figure 1 Erythematous scaling with cracking, fissuring and reticular appearance on the forearm of a 7-month-old infant prior to treatment.

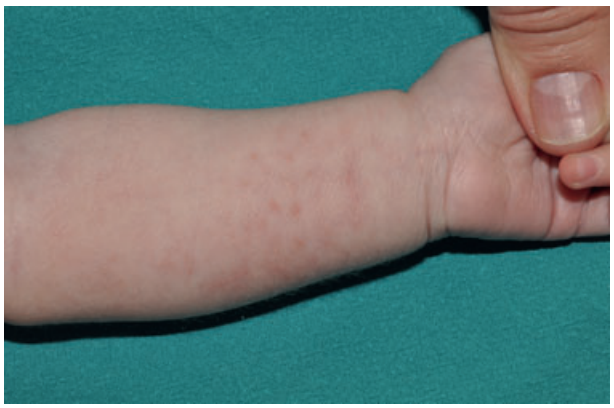

Figure 2 Notable improvement in eczema lesions on the forearm following 10 days of treatment with MPA 0.1% emulsion.

forearms and legs. The palms showed minimal erythema with increased skin linearity (Fig. 1).

Diagnosis

Infantile eczema, with suspected skin barrier defect due to filaggrin insufficiency.

Treatment and follow-up

Emollients were temporarily withdrawn. Methylprednisolone aceponate (MPA) 0.1% (Advantan®, Intendis, Berlin, Germany) emulsion was applied twice daily for 10 days on most of the body surface, more intensely on his forearms and legs. Bathing was permitted with soothing cleansers. A follow-up visit was scheduled for 10 days later.

Disease course

At follow-up, a notable improvement was observed in all eczema lesions. However, a faint scaling and 'dry', chapping skin, remained (Fig. 2). Heavy emollient application was recommended. MPA 0.1% (Advantan) was prescribed for early control of eczema, to be applied as soon as cracking or fissuring appeared. After 10 days of treatment, improvements in sleep quality and scratching behaviour were reported. The disease was considered almost clear, and under control, although new attacks of eczema were expected. No side-effects from MPA were observed.

Discussion

Infants with intense barrier defect and suspected to have filaggrin deficiency usually have early-onset infantile eczema, which may be difficult to control and lead to irritable scratching, sleep or feeding problems in the short term. These children are at risk of developing allergy and asthma later in life. In these cases, early treatment of eczema, and skin barrier restoration with emollients are key for good control of the skin disease and of the progression to other 'atopic' diseases, in the so-called 'atopic march'. Filaggrin deficiency leads to increased entry of potential irritants and allergens into the bloodstream, as well as to eczema, and eczema itself causes further skin barrier disruption and filaggrin deficiency.[1–6] Therefore, early control of infantile eczema is likely to have a great positive impact on a child's quality of life and on the outcome of 'atopic' disease. In the case of this 7-month-old infant, treatment with a latest-generation topical corticosteroid (MPA 0.1%) resulted in clearing of widespread eczematic lesions, along with observed improvements in sleep quality and scratching behaviour, after 10 days of therapy. Ongoing emollient use is important for alleviating residual dryness in individuals with a suspected barrier/filaggrin deficit.

Conflicts of interest

AT has declared no conflicts of interest.

References

1 Irvine AD, McLean WH, Leung DY. Filaggrin mutations associated with skin and allergic diseases. *N Engl J Med* 2011; **365**: 1315–1327.
2 Simon D, Kernland Lang K. Atopic dermatitis: from new pathogenic insights toward a barrier-restoring and anti-inflammatory therapy. *Curr Opin Pediatr* 2011; **23**: 647–652.
3 Brown SJ, Asai Y, Cordell HJ *et al.* Loss-of-function variants in the filaggrin gene are a significant risk factor for peanut allergy. *J Allergy Clin Immunol* 2011; **127**: 661–667.
4 O'Regan GM, Irvine AD. The role of filaggrin in the atopic diathesis. *Clin Exp Allergy* 2010; **40**: 965–972.
5 O'Regan GM, Sandilands A, McLean WH *et al.* Filaggrin in atopic dermatitis. *J Allergy Clin Immunol* 2009; **124**: R2–R6.
6 Palmer CN, Irvine AD, Terron-Kwiatkowski A *et al.* Common loss-of-function variants of the epidermal barrier protein filaggrin are a major predisposing factor for atopic dermatitis. *Nat Genet* 2006; **38**: 441–446.