

Atopic dermatitis

Clive B Archer

Abstract

Atopic dermatitis (AD) and atopic eczema (AE) are interchangeable names for a condition that affects 15–30% of children and up to 10% of adults. Filaggrin plays a key role in epidermal barrier function; null mutations in the filaggrin gene are associated with AD, with resultant barrier dysfunction. AD affects many aspects of family life. Patient and parent education is an important aspect of its management. Emollients applied frequently, even when the eczema is better, help prevent exacerbations. Appropriate use of topical corticosteroids, at various strengths depending on body site and patient age, is still a mainstay of treatment. Topical calcineurin inhibitors can be useful immunomodulators, particularly in children. Intermittent use of sedative antihistamines and oral or topical antibiotics may be required. Garments and bandages can help to decrease scratching, especially in children. In a secondary care setting, it is often more effective to gain control of the disease using a combination of treatments and then maintain control once achieved. Second-line treatments with narrow-band ultraviolet B or immunosuppressive drugs can be required for chronic severe AD. Clinical trials using biological agents, particularly dupilumab (a human monoclonal antibody that inhibits the actions of interleukin-4 and -13) have been encouraging.

Keywords Atopic dermatitis; atopic eczema; emollients; filaggrin; quality of life; second-line treatments; topical calcineurin inhibitors; topical corticosteroids

Introduction

This article discusses atopic dermatitis (AD) in terms of its impact on patients' lives and how to diagnose and treat it.

AD is the most common childhood inflammatory skin condition and currently affects 15–30% of children and 2–10% of adults. Atopy, the triad of dermatitis (eczema), asthma and allergic rhinitis, is increasing worldwide, and the prevalence of AD has doubled or tripled in industrialized countries over the past three decades.¹ Approximately 30% of children with AD develop asthma, and 35% develop allergic rhinitis.^{2,3}

AD frequently starts in infancy (45% of cases beginning in the first 6 months of life), and 70% of children are affected before the age of 5 years. Approximately 60% of children grow out of their AD by adolescence, although up to 50% have further episodes as an adult. Onset of AD in adulthood is also recognized and accounts for about 10% of cases seen in secondary care. The aetiology of AD is complex and involves both genetic and

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Key points

- Atopic dermatitis (AD) is a genetic condition related to null mutations in the filaggrin gene. This results in skin barrier dysfunction, allowing increased exposure to irritants and allergens
- AD is common and usually (but not always) settles with the onset of adulthood
- Daily emollients should be applied all over to help combat dryness and exacerbations of AD
- NICE guidelines recommend 250–500 g per week of a patient's or parent's preferred emollient
- Appropriate strength topical corticosteroids should be used on/off as necessary
- Topical corticosteroids should be applied only to active areas of AD
- For mild AD in neonates and infants, mild or moderate-potency topical corticosteroids applied to the affected skin are usually highly effective
- For moderate/severe AD in an infant or child, moderate or super-potent topical corticosteroids may be needed to control the disease
- Food allergy testing should be offered if there are concerns, and a normal diet should be encouraged in the absence of evidence of specific food allergies

environmental factors. Twin and family studies have shown that genetic factors are important in the predisposition to AD. AD and other atopic disorders show clustering in families, and children whose parents have AD show a higher risk of developing eczema than the children of parents with hay fever or asthma. The discovery that null mutations in the filaggrin gene are associated with AD has proved a significant breakthrough in understanding the genetic basis of AD.⁴ Filaggrin plays a key role in epidermal barrier function, and the resultant barrier dysfunction can allow increased exposure to irritants and allergens, resulting in hyperreactivity to environmental triggers and the induction of immunoglobulin E autoantibodies.

Studies in migrant populations have shown the importance of environmental factors, as children moving from Jamaica to London are twice as likely to have AD.⁵ A higher incidence of AD is associated with urban and industrial settings, higher socioeconomic status and smaller family size.

Impact on quality of life

Having a child with AD can be challenging and can affect many aspects of family life. Eczematous skin is dry and itchy, leading to scratching, particularly at night. Studies have shown that more

than 60% of children with AD have a disturbed sleep pattern. Infants with moderate to severe AD can have poor weight gain, and problems with play and social interaction. The impairment of quality of life caused by childhood eczema has been shown to be the same or more than in other common childhood disorders such as diabetes mellitus and asthma.

AD can cause psychological difficulties for all the family. Caring for a child with eczema is time-consuming and costly. The financial burden includes direct costs such as the purchase of special bedding or washing machines, and hidden costs such as loss of parental financial income. These have the greatest impact on low-income families. There are many other consequent family restrictions.

The Children's Dermatology Life Quality Index was the first validated specialty-specific dermatology scale developed for children aged 4–16 years, and over the past 10 years it has been shown to be a useful, reliable, repeatable tool showing good sensitivity to clinical change. Other tools include the Infant's Dermatitis Quality of Life Index and the Dermatitis Family Impact score.

Diagnosis

AD is characterized by poorly defined erythema with oedema, vesicles and weeping in the acute stages, and skin thickening (lichenification) in the chronic stage. AD commonly starts on babies' cheeks, which can become red and sore. As the condition progresses, it can affect the common flexural sites such as behind the knees (Figure 1) and in the elbow creases (Figure 2), as well as the neck folds. A common time for AD to flare is when babies are weaned. At this time, children delight in exploring new foods and tend to smear them around their face. Many foods are irritants, and this, in combination with dribbling, can cause severe facial and neck flares, particularly in the fat neck folds of toddlers.

Hands are another site to check, as many preschool activities involve playing with irritant substances. Around this time, scratching may have become a habitual activity. Common sites that are easy to scratch are the wrists and ankles, whereas



Figure 1 Atopic dermatitis of the popliteal fossae, with lichenification and post-inflammatory hyperpigmentation. (Courtesy of St John's Institute of Dermatology, London).

occluded and more protected sites such as the nappy area, abdomen and back are often clearer.

It is now known that the epidermal barrier dysfunction affects all skin, so it is important to examine non-eczematous skin, which can feel dry. Hyperlinear palms are found in some cases of atopic eczema and in the ichthyoses; interestingly, these are associated with filaggrin gene mutations.

The validated UK Working Party criteria for the diagnosis of AD have been modified from the consensus criteria of Hanifin and Rajka. To have a diagnosis of AD, an individual must have an itchy skin condition in the last 6 months, plus three or more of the following:

- onset before 2 years of age (not applicable if the child is <4 years of age)
- a history of flexural involvement
- a history of generally dry skin
- a history of other atopic disease (or a history in a first-degree relative for a child <4 years of age)
- visible flexural dermatitis.

Differential diagnosis

In infants, seborrhoeic dermatitis should be considered; this can present with cradle cap and salmon-coloured scaly plaques in the nappy area.

Discoïd eczema can prove more of a diagnostic challenge and tends to be seen in older children. Patches can be found on the trunk and limbs, and can be confused with psoriasis. Differentiating features are the more clearly demarcated plaques and silvery scale of psoriasis, and that discoïd eczema tends to be itchy.

Other conditions that can be confused with eczema are keratosis pilaris, which is frequently associated with AD. This disorder of keratinization of the hair follicles is usually found on the outer aspects of the upper arms and thighs, but can also be found on the cheeks; it results in a rough texture and appearance. Although it is usually asymptomatic and not itchy, it can cause distress because of its appearance.

Other differential diagnoses to consider are the ichthyoses. Ichthyosis vulgaris commonly coexists with eczema. A common condition, it affects 1 in 250 people. Fine, light grey scales and dry skin develop in early childhood, but there is no inflammation. Rarer conditions such as X-linked dominant ichthyosis can present with larger scales and the 'dirty neck' sign.

Treatment of atopic dermatitis

As with many chronic skin conditions, the key to managing AD is parent, patient and family education, using a multidisciplinary approach. The first interview with a child suffering from eczema and their family is an opportunity to gain their confidence and understand parental concerns. Parents often feel confused having seen a multitude of health practitioners, and it is important to give clear explanations about the disease, the disturbed barrier function and how and why treatments are used.

Treatment demonstrations showing application amounts and which sites to apply them to, with tips for application, can help to avoid over- and undertreatment and improve parental confidence. Information about avoidance of irritants and allergens such as soaps, detergents, house dust mite and pollen is useful.

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