Atopic dermatitis in children: a practical approach

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Abstract

Atopic dermatitis is a common condition that takes a significant time from the daily work of general paediatricians. The principles of Child Health apply well in the management of this condition including attention to details and a consideration for the impact on the quality of life of the child and family. The clinical assessment involves an enquiry about triggers as well as details of therapy. All should be done through a multidisciplinary approach and liaison with primary care.

Regular daily topical emollients and intermittent topical corticosteroids are the cornerstone of therapy. Resistant eczema should raise the suspicion of secondary infection, usually staphylococcal or streptococcal, poor compliance and psychological factors.

Keywords atopic dermatitis; emollients; food allergy; topical calcineurin inhibitors; topical corticosteroids

Epidemiology

Atopic dermatitis (AD) is a chronic pruritic, inflammatory, skin disease that typically begins in early childhood. AD is one of the most common skin disorders in children. Prevalence in children in UK is up to 20%. Disease onset typically occurs by 6 months of age in 45%, by 1 year of age in 60% and by 5 years of age in 85% of affected infants and children. Up to 70% of children have a spontaneous remission before adolescence.

The prevalence of AD in children has increased steadily over the last three decades of 20th century. This is paralleled by increases in the prevalence of asthma, allergic rhinoconjunctivitis, eosinophilic oesophagitis and gastroenteritis.

However recent data suggests that AD and hay fever prevalence might have levelled off or decreased over the last 10 years in those aged 12 years or older, whereas it continues to increase in younger children. Migrant studies reveal that AD prevalence increases in populations that move from an area of low to high prevalence, supporting the role of environmental factors in the expression of AD.

AD, asthma and allergic rhinitis are known as the 'atopic triad'. The concept of 'atopic march' evolved from clustering of these conditions in the same individuals and families. AD is frequently the first disorder of the atopic triad. It is possible that epicutaneous sensitization to allergens predisposes to development of asthma and allergic rhinitis. In a large multicenter study, by 5 years of age, 50% of children with early AD and a strong family history of allergy had allergic airway disease or asthma compared with 12% in patients without AD or a family history of atopy.

Impact on the child and family

AD has the potential to be a major handicap with considerable personal, social and financial consequences. In children with AD, quality of life is impaired owing to pruritus, sleep disturbance, pain, irritability, restricted activities, and adverse social interactions. Severe AD may result in poor school performance, behavioural problems, low self-esteem, decreased participation in sport and other social activities. Parents of children with moderate to severe AD experience sleep disturbance, exhaustion, frustration, and worry owing to their child's disease. The family stress related to the care of children with moderate or severe atopic dermatitis is significantly greater than that of the care of children with type 1 diabetes mellitus.

Aetiology

The pathophysiology of AD is not completely understood. Complex interaction of defects in skin barrier function, host immune response, environmental factors and infectious agents in a genetically susceptible individual are thought to result in AD.

The concordance rate for AD is higher among monozygotic twins (77%) than among dizygotic twins (15%). Parental atopy, in particular AD, is significantly associated with the manifestation and severity of early AD in children.

Defective epidermal barrier function is a hallmark of AD. This results in increased trans-epidermal water loss and dry skin. It will also allow increased trans-epidermal penetration of environmental allergens and triggers inflammation. Mutations in the genes coding for filaggrin, a key protein in barrier function, might play an important part in early-onset AD and asthma. Fillagrin mutations are identified in 30% of European patients with AD. Reduced ceramide levels, genetic variation in stratum corneum tryptic enzyme and epidermal collagen may also play a role. Exogenous proteases from *Staphylococcus aureus* and house dust mites, and the use of soaps and detergents further damage the barrier function.

Defective barrier function allows penetration of high-molecular-weight allergens in pollens, house dust mite products, microbes and food. Early-onset atopic dermatitis usually emerges in the absence of detectable IgE-mediated allergic sensitization. IgE-mediated sensitization occurs several weeks or months later. Antigen-specific IgE is the major recognition structure for allergens on basophils and mast cells. Keratinocytes in atopic skin produce cytokines that signal dendritic cells to drive Th2 polarization. Inflammation in AD is biphasic: an initial TH2 phase

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precedes a chronic phase in which Th0 cells and Th1 cells are predominant.

S. aureus colonization with or without clinical signs of infection occurs in more than 90% of patients with AD and contributes to the severity of inflammation. Scratching increases the binding of *S. aureus* to skin. *S. aureus* enterotoxins increase the inflammation in AD and provoke the generation of enterotoxin-specific IgE, which correlates with the severity of disease. Enterotoxins also contribute to emergence of resistance to topical corticosteroid treatment.

The cytokines generated during inflammation in AD down regulate the natural production of antimicrobial peptides in the epidermis — cathelicidin and defensin. This results in increased susceptibility to colonization with *Staphylococcus* and the yeast *Malassezia furfur*; the latter plays an important role in head and neck eczema. Patients with atopic dermatitis are predisposed to eczema herpeticum and eczema vaccinatum because of a reduced production of cathelicidin, which has potent antiviral activity.

The underlying mechanisms for pruritus, the most important symptom of AD, are not known. Antihistamines are not always effective in relieving the pruritus. This argues against a prominent role for histamine in causing AD related pruritus. Neuropeptides, proteases, kinins and cytokines may play an important role in inducing pruritus.

AD is more prevalent in urban, nuclear families compared to rural areas. The 'Hygiene hypothesis' postulates that declining family size, improvement in personal amenities and higher standards of the personal cleanliness have reduced the opportunity for infections in young children and increased susceptibility to AD.

Clinical features

During the first few months of life, AD typically affects face and scalp. Intensely pruritic erythematous papules affect cheeks and forehead (Figure 1). Periorbital and perioral areas are relatively spared. The lesions show significant oedema, leading to oozing and crusting unrelated to secondary infection. Flare-up of lesions around mouth is common with teething and initiation of solid foods. This is probably due to irritation caused by saliva and foods. It is important to keep in mind contact urticaria to food when the lesions are predominantly around mouth. Pruritus and dry scaling of scalp are common (Figure 2). The scalp lesions may represent overlap with seborrheic dermatitis.

The lesions may remain localized to the face or extend to the trunk and extensor aspects of the extremities. By 8—10 months of age the extensor surface of the arms and legs are involved, perhaps because of the friction associated with crawling (Figure 3). The lesions on trunk and extremities are often symmetric, scattered ill-defined erythematous patches. AD typically spares diaper area. This is because of combination of increased hydration in the diaper area, protection from triggers by the diaper, and inaccessibility to scratching and rubbing.

Involvement of the ante-cubital and popliteal fossae, hands, feet, ankles, wrist periorbital area, perioral area and neck is more common in older children and adolescents (Figure 4). However these sites might also be affected in infants and young children. Older children are less likely to have the exudative lesions of infancy and instead exhibit more lichenified papules and plaques representing more chronic disease (Figure 5). Though flexural areas are commonly involved, some children show an "inverse" pattern with primarily involvement of extensor areas.

In children 1 year of age or older, coin shaped sharply defined erythematous scaly plaques (nummular lesion — Figure 6) might accompany the more typical dry scaling erythematous patches of AD. It is important not to confuse them with Tinea corporis. Nummular lesions tend to be more recalcitrant to topical therapy and are frequently secondarily infected. In Africaribbean children the lesions of AD are often more papular (follicular AD).

Generalized dry skin (xerosis) is common. Pruritus is frequently severe, leading to sleep disturbances. Lymphadenopathy in the

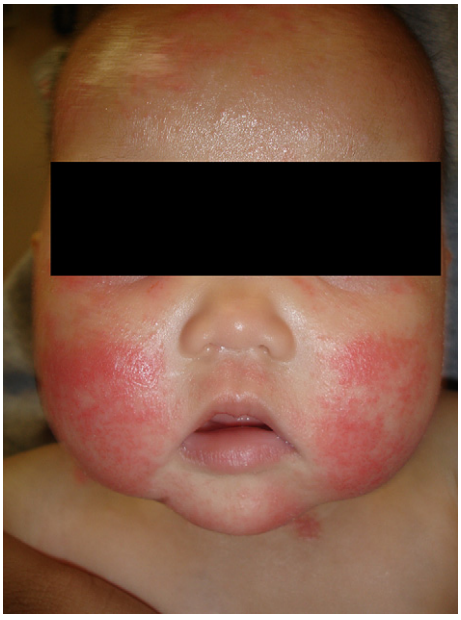

Figure 1 Erythematous papules and plaques with background oedema typical of infantile atopic dermatitis.

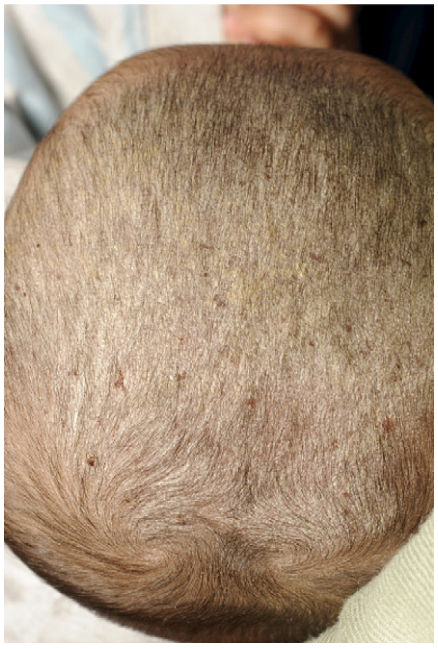

Figure 2 Dry scaly lesions on the scalp. May overlap with seborrheic dermatitis.