Atopic dermatitis in childhood

Sue Lewis-Jones

Atopic dermatitis (eczema) is the commonest of the childhood dermatoses, accounting for 20% of all dermatological referrals with many cases continuing into adulthood. The morbidity and costs to society are high.

The atopic diseases comprise atopic dermatitis (AD), asthma and allergic rhinitis. Atopy appears to be increasing in Western society and AD now affects 15–20% of UK school children, compared with only 5% in the 1950s (Williams, 1992). The reasons for this are uncertain but are not accounted for entirely by increased recognition or self-referral.

AD is an extremely itchy inflammatory dermatosis usually starting in early infancy. This article outlines current thoughts on the clinical presentation, aetiology and management of AD in childhood.

DIAGNOSIS AND CLINICAL FEATURES

Table 1 shows the 1994 UK Working Party's criteria for the diagnosis of AD, which are simple to use, 88% sensitive and 93% specific (Williams et al, 1994). The primary symptom is itchiness leading to scratching, which causes epidermal cell damage with release of inflammatory mediators, which in turn stimulates the itchiness leading to further scratching and so on (the so-called 'scratch–itch cycle').

TABLE 1.
The UK Working Party's minimum diagnostic criteria for atopic dermatitis*

Itchiness plus three or more of the following:
History of flexural involvement (including cheeks in children under 10 years)
Personal history of atopic disease (or in first degree relative under 4 years)
Generally dry skin in last 12 months
Visible flexural eczema (or cheeks/forehead, extensor surfaces of limbs under 4 years)
Onset under the age of 2 years

From Williams et al (1994).
*93% specificity and 88% sensitivity

The majority of cases of AD start under 2 years of age, usually with a facial rash (*Figure 1*) and rash on the extensor aspects of the limbs and the upper trunk. The more typical flexural pattern appears later and results from the irritant effect of sweat (*Figure 2*). The napkin area is frequently spared because of the occlusive hydrating effect of modern napkins. Dry skin or xerosis is the earliest histological sign of eczema and the majority of patients will have dry skin from birth or soon after.

Figure 1. Atopic eczema frequently starts on the face and cheeks in early infancy. This girl demonstrates a reticulate erythema with slight scaling and poorly defined edges to the rash.

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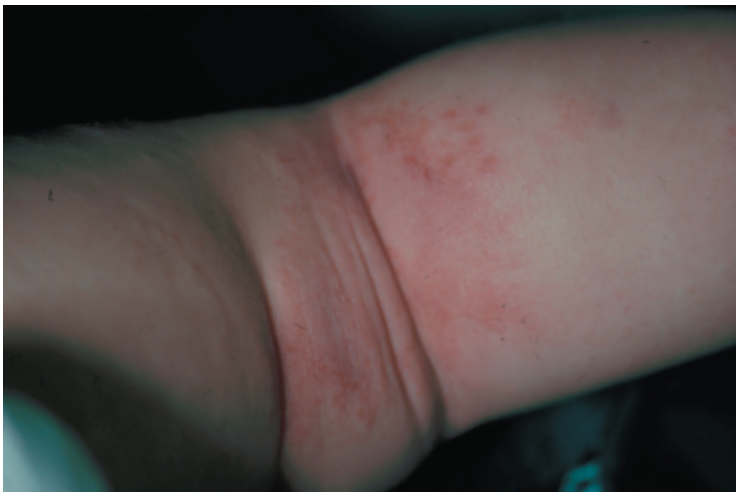

Figure 2. Flexural eczema in typical site on the popliteal fossa.

The rash is typically patchy, red (erythematous) and lesions have poorly defined edges. Very acute inflamed eczema will show erythema, oedema and often excoriations with serous weeping and crusting (*Figures 1* and *2*), whereas chronic lesions are characterized by scaling, thickening and lichenification — an exaggeration of the normal skin creases — but there is often a mixture of the two (*Figure 3*).

Dryness of the scalp is common and many infants have 'cradle cap' before developing AD. Some exhibit a seborrhoeic pattern of dermatitis with a blotchy, usually non-pruritic rash in the flexures of the groin, neck and axillae, but not all of these go on to develop AD.

The hands and feet are frequently affected and often show increased palmar and plantar markings, giving a wizened appearance (*Figure 4*). Severe hand eczema with fissuring is often associated with a worse prognosis and is a major cause of loss of work in adults.

Figure 4. Severe hand dermatitis exhibiting hyperlinearity (increased palmar markings), lichenification and fissuring. This is frequently a poor prognostic sign for eczema.

Figure 3. Severe eczema with lichenification, excoriation and fissuring.

Twenty two minor features are associated with AD (Hanifin and Rajka, 1980). These include earlobe fissuring, keratosis pilaris, periocular darkening, keratoconus, exaggeration of the infraorbital creases (Morgan–Dennie–Brown fold), perioral pallor, cold extremities and delayed blanching of the skin on minor trauma.

PROGNOSIS

The majority of mild cases of AD can be expected to clear during childhood, although the overall clearance is only about 50% (Williams and Wüthrich, 2000). Of those with severe AD 80% will continue to suffer throughout life, but there is usually some improvement in severity. Approximately 75% of severe cases will develop allergic rhinitis and 50% will develop asthma (Linna et al, 1992).

INFECTION AND AD

Serous crusts are an ideal culture medium for bacteria, particularly *Staphylococcus aureus*, which is found in 90% of lesional skin in AD. Impetiginized areas have golden crusts, marked inflammation and sometimes pustules (*Figure 5*). Treatment with antibiotics leads to clinical improvement and it has been postulated that exotoxins produced by *Staph. aureus* are capable of acting as 'super antigens', bypassing the normal antigen-presenting pathways and triggering the release of inflamma-

tory cytokines (McFadden et al, 1993). However, long-term antibiotic treatment, particularly with topical antibiotics, leads to the development of resistant organisms and should be avoided in the absence of significant 'clinical' infection.

Eczema herpeticum

Widespread infection with herpes simplex virus is a serious clinical problem peculiar to atopics (*Figure 6*). It is surprisingly uncommon considering that 20% of the population suffer from recurrent herpes simplex. This may be a reflection of an idiosyncratic immune response. Hospitalization is usually required, with prompt treatment with systemic aciclovir or valciclovir, along with an ophthalmology opinion for ocular involvement.

AETIOLOGY AND EPIDEMIOLOGY

There is a strong genetic component to the development of AD (Schultz Larsen, 1993), but it is thought likely that environmental factors are equally important in its development, in particular a 'Western lifestyle' (Diepgen, 2000). There appears to be considerable variation in prevalence between different societies and social groups. There is a high level of AD in immigrants to the West from third world countries. AD is more common in smaller families and those in social class 1 families (Strachen, 1989).

Strachen (1989) postulated that an increase in hygiene and a reduction in exposure to childhood infection might alter the immune response, predisposing to atopy. Aeroallergens such as house dust mite, pollens, animal danders, cigarette smoke, exhaust fumes and industrial pollutants are all considered to be possible contributors to the development of AD (Diepgen, 2000), and measures to reduce exposure are an important, if problematic part of management. Many atopics are intolerant of skin irritants so that soaps, detergents, biological washing powders and fabric conditioners should be avoided.

Both allergic and irritant contact dermatitis may occur in AD. Potential allergens include perfumes, cosmetics and topical medicaments, including steroids and antibiotics. In cases unresponsive to treatment, or when previously quiescent AD recurs, patch testing should be performed.

Climatic changes also affect AD. Heat causes sweating and flexural pruritus. Low humidity increases dryness but high humidity increases the risk of secondary infection. However, sun exposure may improve AD because of its immunosuppressive effect.

IMMUNOPATHOGENESIS

Altered reactivity to a number of environmental allergens is an important feature of atopy, and the immune dysfunction of AD is complex and not fully understood (Archer, 2000). Increased levels of immunoglobulin (Ig) E are found in most atopics. Archer (2000) has postulated that specific allergens, through activation of the T cells, stimulate the production of IgE. Binding of IgE to mast cells causes degranulation and the release of inflammatory cytokines.

In cases where there is concern that a particular allergen, such as egg, may be an important contributory cause then levels of specific IgE to that allergen can be measured. However, it can be difficult to interpret the results without some experience — low levels of specific antibody in an individual with a high total IgE may just reflect an increased background 'over-reactivity' resulting from previous exposure to the antigen.

Figure 5. Impetiginized eczema above right eye.

Figure 6. Eczema herpeticum in the napkin area. Mother had a herpetic whitlow on the finger.

High specific IgE levels or a moderately raised level in a child with normal total IgE are more likely to be relevant.

MEDICAL AND SOCIAL COST

AD accounts for up to 20% of dermatological referrals and about 40–50% of a paediatric dermatologist's time (unpublished data, S Lewis-Jones, 2001). Although the financial cost to society is not known a study in infants with AD in a general practice in the Lothian area suggests a projected figure of £465 million per annum, of which one-third is borne by the family (Herd et al, 1996).

PSYCHOSOCIAL ASPECTS

The misery of AD cannot be understated. Quality of life studies have shown that AD causes considerable physical and psychological morbidity in children (Lewis-Jones and Finlay, 1995), infants (Lewis-Jones et al, 2001), and affects their families (Lawson et al, 1998). Severe pruritus leads to sleep loss in at least 80–90% of infants, and also affects parents and siblings (Reid and Lewis-Jones, 1995).

Manual hobbies and sports, especially swimming, can be uncomfortable and difficult. Social aspects such as having a pet, holidays and staying overnight with friends may be restricted because of treatment or contact with potential allergens. Many children report feeling 'different' and suffer embarrassment, social isolation, peer rejection, teasing and bullying (Lewis-Jones and Finlay, 1995). This leads to lack of confidence and sometimes school phobia. Performance may be affected by loss of schooling, sleeplessness, sedating medication and loss of concentration from itching.

MANAGEMENT OF AD IN CHILDHOOD

It should be possible to manage the majority of mild cases of AD in a primary care setting, particularly in conjunction with specially trained nurses. More severe cases may require help from a specialist unit. Treating severe childhood AD successfully requires a 'team approach', which can include a paediatric dermatologist or paediatrician with dermatology training, specialist nurses, dieticians and preferably access to paediatric clinical psychologists. Some dermatology units now have nurse-led clinics, telephone helplines, treatment centres and liaison nurses working in the community. Demonstration of treatment techniques and education of parents and children are the most important methods of ensuring successful therapy.

General measures

Keep children cool, use cotton clothes (not nylon or wool) and synthetic bedding. Avoid biological washing powder, fabric conditioners, soaps, detergents, bubble baths, perfumed products and other irritants. Keep the house dust levels low with frequent vacuuming (including mattresses) and damp dusting. Gortex covers reduce house dust mite levels in the bed; however, they have no benefit elsewhere and are expensive. Special vacuums are also not of proven benefit (Tan et al, 1996).

Avoid contact with pets where possible. If this is difficult, at least keep the pets away from living areas since fabric upholstery retains animal dander for months.

Try distraction rather than confrontation to reduce scratching and use cotton scratch mitts or gloves at night.

Topical therapies

Emollients are the most important treatment for AD and should be used copiously, frequently and in all cases — up to about 500 g every 1–2 weeks. This can significantly reduce the severity of AD, the need for topical steroids in 40% of cases and the strength of steroid required (Cork et al, 1999). However, to be effective it is essential to give visual demonstrations with verbal and written instructions in a care plan. Back this up at subsequent visits by ascertaining that the parents have understood and are continuing to use the treatment correctly. Generally speaking use creams with a high water content to cool hot inflamed eczema, and ointments that are very greasy for dry skin. Emollients can be used in the bath and as soap substitutes.

Topical steroids

Topical steroids are still the most effective therapy for AD and if used carefully should not cause problems, provided that potent preparations are avoided except for short periods in severe cases. Overuse, particularly with potent steroids, can cause thinning of the skin, which is reversible in the early stages and more of a problem in elderly skin than in children, but can lead to irreversible striae. Systemic absorption with the risk of hypothalamic–pituitary–adrenal axis (HPA) suppression is more likely to occur with potent steroids used over a wide surface area for prolonged periods. Patel et al (1995) found no evidence for HPA axis suppression in 14 prepubertal children, even after a median 6.5 years use of mild to moderately potent steroids in children with severe AD of greater than 50% body surface area.

Patients often quote steroid phobia as the reason for their lack of use (Charman et al, 1999), and the failure of many doctors, nurses or other medical workers to be properly informed about steroids causes unnecessary worry for patients.

Minimizing the risks of topical steroids: It is essential that doctors, parents and patients are familiar with the potency of steroids prescribed (there is a useful list in the Monthly Index of Medical Specialties, 2001). Give a written care plan and steroid information leaflet such as that available from the National Eczema Society (see *Useful addresses*).

A fingertip unit weighs 0.5 g and will cover the surface area of two hands (*Figure 7*) (Long and Finlay, 1991). This is an extremely useful and simple measure to demonstrate. Keep a careful record of the amount and potency of steroids used and monitor growth using height and weight charts. Try to use steroids intermittently and maintain control with emollients. Short-term (1–5 days) use of potent steroids early in an acute flare will often control the AD quickly and reduce the total amount of steroid use overall. Do not use anything stronger than hydrocortisone on the face. Recommend regular optician screening for long-term use to avoid the extremely rare complications of cataract and glaucoma. If a child is not responding to simple measures with mild to moderately potent steroids, then consider referral to a dermatologist.

Bandages and wet wraps

Cotton bandages, mitts and gloves, and 'all-in-one' sleep suits are helpful in preventing scratching and allowing healing. Dry bandages can be used or zinc paste-impregnated bandages such as Steripaste (SSL International, Knutsford, Cheshire). Tar or ichthammol bandages are useful for chronic lichenified eczema but should be avoided in infants.

Wet wrapping is a technique whereby dampened stretchy cotton bandages are placed over a layer of emollients, with or without steroids, and a dry layer of bandages placed on top. Many types of bandages can be used for this procedure but the most widely used technique favours Tubifast (SSL International, Knutsford, Cheshire) (Bridgman, 1995).

Wet wrapping is extremely useful for difficult, severe cases but should not be applied to overtly infected areas. It undoubtedly improves dryness and eczema rapidly, improves sleep and mood, and can reduce the amount of steroid used. However, it is expensive, time consuming and increases the risk of steroid absorption, so care should be taken and long-term use with steroids minimized. Older children often find wet wraps embarrassing, cold and constrictive. Good randomized studies on wet wrap use are currently lacking.

Antiseptics and antibiotics

Systemic treatment with flucloxacillin, or erythromycin in cases of penicillin allergy, is essential for widespread impetiginized eczema, and antiseptics such as potassium permanganate or Sterzac (SSL International, Knutsford, Cheshire) can be added to the bath. Short-term steroid antibiotic combinations are invaluable, but long-term treatment may cause development of resistant organisms.

DIET AND AD

Food allergy and intolerance is most likely to occur in infants under 3 years and the majority will lose their reactivity after this age, except for certain cases of nut allergy. Most children with mild AD do not require and do not appear to respond to dietary manipulation. Moreover unsupervized diets can lead to malnutrition including rickets.

It has been suggested that about 10% of infants can benefit from dietary interventions. Eggs and milk are the commonest allergens implicated and the best to try first (Lever et al, 1998). A detailed clinical and dietary history should be taken, looking for adverse reactions such as immediate contact urticaria, redness and swelling of the skin, lips or tongue, and

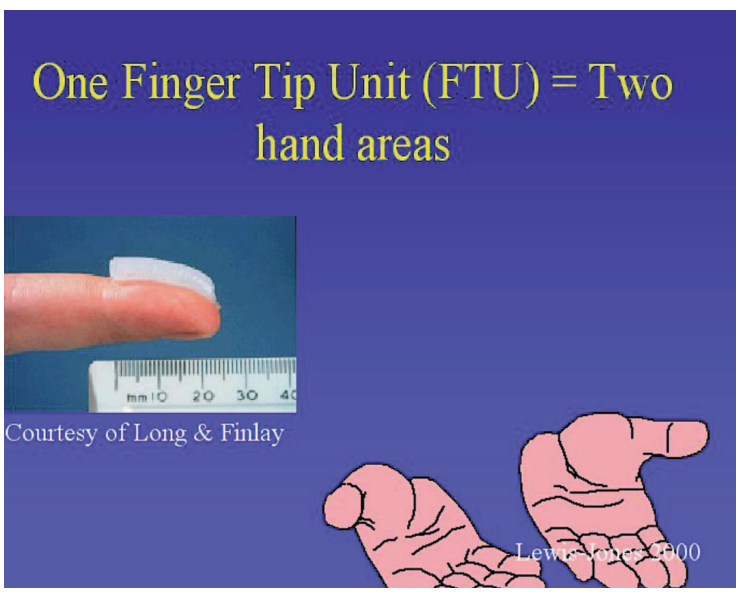

Figure 7. A fingertip unit weighs approximately half a gram and covers the surface area of two hands.

itchiness. More severe reactions include nausea, vomiting, diarrhoea, wheezing or difficulty in breathing, and circulatory collapse and anaphylaxis.

Late reactions cause a worsening of the eczema. Affected infants usually show a widespread blotchy, urticated pattern of eczema and a marked pruritus, often out of proportion to the clinical picture and unresponsive to topical therapy. If a food is suspected then total and specific IgE should be checked. Some specialist centres can offer prick testing but this should only be done where resuscitation procedures are available. An exclusion diet should only be undertaken with the supervision of a dietician trained in paediatrics. The gold standard for testing is a double-blind placebo-controlled food challenge, but this is available in very few specialist centres and is expensive and time consuming to perform.

BREASTFEEDING AND AD

Prolonged and exclusive breastfeeding for at least 9 months is thought to reduce the development and severity of AD. Maternal exclusion diets are not considered appropriate and can lead to underweight babies (Kramer, 1998). However, it would seem sensible to avoid non-essential food, such as peanuts, during pregnancy if severe allergy to that food has already occurred in an older sibling.

SYSTEMIC TREATMENTS

Sedative antihistamines are of some benefit for short-term relief of night-time itchiness and sleep disturbance but can lead to daytime drowsiness, affecting school performance. Non-sedating antihistamines are occasionally of benefit in older children. Antihistamine use should be avoided in children under the age of 6 months.

Evening primrose oil (EPO) has not been proven to improve AD even though there is a deficiency of gamma-linolenic acid (GLA) in AD; however, EPO only contains 8.9% GLA. Meta-analysis of placebo-controlled studies involving EPO showed no clearcut improvement apart from a reduction in itchiness (Morse et al, 1989).

Chinese herbal remedies can be effective but should be prescribed only by a reputable practitioner trained in their use as they are potentially hepatoxic, and 3-monthly liver function tests are recommended. Similarly, exercise caution with the use of topical Chinese creams — a recent study found that 60% contained 'hidden' potent topical steroids (Keane et al, 1999).

IMMUNOSUPPRESSIVE THERAPIES

Severe childhood eczema unresponsive to standard therapies may require systemic treatment, but the benefits must outweigh the risks and it is recommended that treatment is undertaken in units with specialist expertise. Long-term immunosuppressive therapy carries the theoretical risk of carcinogenicity. Systemic steroids are best avoided when treating childhood AD, especially in those under 2 years of age and during the pubertal growth spurt. Other immunosuppressive agents used include cyclosporin and azathioprine.

Phototherapy and photochemotherapy are useful in the short term but prolonged treatment should be avoided in children because of the long-term risk of skin cancer.

CONCLUSION

AD is the most common of all childhood skin diseases and accounts for much psychosocial morbidity and financial cost. Successful treatment of childhood AD depends on good teamwork between the child, his/her parents and the 'medical carers'. The latter may include a general practitioner, a specialist nurse, or possibly a pharmacist, since many patients self-medicate first. Severe cases or those unresponsive to therapy will require the help of a dermatologist.

Useful addresses

The National Eczema Society
Hill House
Highgate Hill
London N19 5NA
Tel: 020 7281 3553
Fax: 020 7281 6395

The Skin Care Campaign
Hill House
Highgate Hill
London N19 5NA
Tel: 020 7281 3553
Fax: 020 7281 6395

The most important ingredient for success is education, both verbal and visual, with a clearly written care plan. Backup with telephone helplines, good clinical records and re-evaluation of treatment techniques will increase success and ultimately reduce costs by providing efficient, cost-effective services. Early recognition and good management will lead to more effective treatment and may help to prevent chronic disabling disease. HM

Conflict of interest: Dr Lewis-Jones is an advisor for desloratidine, a new antihistamine from Schering Plough.

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KEY POINTS

- The prevalence of atopic dermatitis is increasing in Western society and currently affects 15–20% of infants in the UK.
- Itchiness is the primary symptom, leading to scratching, which in turn produces lesions.
- Demonstration of treatment techniques and education of parents and children is essential for ensuring successful therapy.
- Emollients are the most important treatment for atopic dermatitis and should be used copiously and frequently — up to 500 g every 1–2 weeks.
- Increased use of emollients decreases the quantity and strength of steroid required.
- Food allergy is mainly confined to infants under 2 years of age and egg and milk are the most likely allergens.
- Exposure to airborne allergens such as house dust mite, pollen and pet dander should be kept to a minimum.
- Potent topical steroids should only be used for short periods of 1–5 days in children — refer to a dermatologist if frequent potent steroids usage is required.
- Do not use long-term topical antibiotics.