Today's Treatment

Diseases of the Skin

Infantile Eczema

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The development of eczema in an infant is often the first manifestation of atopy, which may also be characterized by asthma, urticaria, and allergic rhinitis. We know that individuals inherit a tendency to allergic disorders and a child with infantile eczema will usually have a family history of some of these allergic manifestations. Little is known, however, about the mechanisms involved, but recently an immunoglobulin IgE has been identified, which is present in increased amount in atopic individuals. The serum levels do not always parallel the activity of the disease and the precise role of IgE has yet to be determined. Probably IgE becomes attached to the surface of mast cells and sensitizes them so that, when they are exposed to a specific allergen, the sensitized mast cells readily release histamine and other vasoactive amines. Possibly in the highly sensitive individual it is the rate of production of IgE that is important and if much of it is cell-bound then the serum level might not necessarily be grossly raised.

In the allergic forms of asthma this mechanism is probably very important and specific allergens which react with antibodies in the IgE fraction have been identified. Clinically this is possibly the basis of prick testing with specific allergens, which results in urticarial wheals in sensitive people. Specific allergens have not been identified in eczema and skin testing is of no help. In atopic eczema there is also a pharmacological genetic abnormality which results in a reduction of physiological antagonism of the vasoactive substances at the adrenergic-receptor sites. This is probably the explanation for the white dermographism or abnormal blanch reaction which occurs on stroking the normal skin of atopic subjects.

Evolution

The clinical features of the eczema reaction have been described in an earlier article. Atopic eczema is diagnosed on the history and the pattern of development of the eczema. The first signs do not usually appear until three to four months of age and at this stage the characteristic localization is absent. The eruption often appears on the face, and over the cheeks and brow (where it rubs against the cot), and the eczema may also appear on other areas where there is friction, such as the trunk or the outer aspect of the limbs. Usually from about 12-18 months the areas of eczema become localized round wrists, ankles, antecubital fossae, and behind the knees. Though in severe cases the eczema may be more widespread, this pattern persists in atopic people into adult life.

In most children the severe features have settled by the age of 10 years, but, lichenification of the skin often persists in the folds of the elbows and behind the knees—where tendency to exacerbation may remain. Probably less than half the children with eczema may develop asthma or hay fever at some time: in general those with severe atopic eczema are more likely to develop asthma, but there is little evidence that the two conditions alternate—as has sometimes been suggested. Nevertheless, childhood allergic asthma has a good ultimate prognosis and usually clears after puberty.

Parents' Co-operation

In treating infantile eczema the doctor must explain the nature of the condition and its prognosis to the mother and, if possible, both parents. Many parents are extremely anxious, and often exhausted by a hyperactive child, who never stops scratching, and whose skin is disfigured by eczema. If their fears can be somewhat allayed and they can be taught how to manage the child and his skin, more will be achieved than by the most potent topical steroid. This often takes much time and patience, but the situation must be avoided where the parent is constantly looking for a cure or a new ointment which will completely clear the child's skin.

At present we can suppress the eczema reaction, but in an atopic individual there will be times when the skin will flare up and the parent must be prepared to cope with these distressing exacerbations as promptly and effectively as possible to prevent the vicious circle of scratching and eczematization becoming re-established. They should be told that during teething, intercurrent infections, when there is stress or change in environment—that is, starting school or moving house—additional care may be required. The mother must not become over-protective and, depending on the state of the child's skin at any given time, all activities can be permitted. Only if there are severe and repeated exacerbations or an attack of asthma provoked, for example, after swimming or horse riding should these be forbidden. Later in life it is wise to advise children who have persistent atopic features to avoid work in high-risk occupations. Girls should not become hairdressers, where constant exposure to wet conditions leads almost invariably to severe exacerbations of eczema on the hands, and boys should avoid engineering, coal mining, and other dirty occupations.

Management

General advice about the skin may be necessary. Many children are very uncomfortable after bathing and the daily baths may have to be reduced. Medicated soap should not be used except when there is overt infection and the minimum amount of a plain soap, such as simple soap, should be used routinely. Children

with severe eczema can wash with emulsifying ointment *B.P.* instead of soap. About 10% of atopic children tend to have the dominant type of ichthyosis which gives rise to dry scaling with follicular hyperkeratosis mainly on the back and extensor aspect of the arms and legs. The skin is made more comfortable by rubbing emulsifying ointment *B.P.* in to the dry areas after bathing.

Depending on the stage the topical treatment of the eczema reaction would be as outlined in the article by Dr. H. Baker (*B.M.J.*, 1 December, p. 544). Since atopic eczema is likely to recur a steroid as dilute as possible should be used. On the face in particular fluorinated corticosteroids should be avoided and 0.5% hydrocortisone cream or ointment will provide adequate control without running the risk of producing severe atrophy or telangiectasia. On areas of chronic eczema affecting the limbs dressings are often very effective and a convenient method of doing this is for the mother to apply a piece of Ichthopaste bandage to the affected area and then cover this with a crepe bandage. Alternatively, the mother can make a spread dressing with 1% ichthammol paste on calico or a piece of linen. This type of dressing, without a corticosteroid, is also very effective in treating the painful fissures which may develop on the fingers and toes of atopic children of school age.

If the skin is not broken but grossly thickened then considerable improvement may follow the use of a Coltapaste bandage or a rub of 1% tar paste at night. If tar preparations produce any inflammation they should be immediately withdrawn. To reduce pruritus, which is a major symptom, many children will get relief by an antihistamine at night such as trimeprazine. This can be given as an elixir in young children.

SUDDEN DETERIORATION

Sudden deterioration in atopic eczema, particularly if topical corticosteroids are in use, may herald the onset of secondary infection. This may produce frank pustulation or impetiginous lesions, but the clinical signs of infection may not be so obvious. A swab should be taken and it will usually be necessary to stop all local steroids and substitute a suitable antibiotic ointment or cream such as soframycin or gentamicin. If the infection occurs in very young children or if there is any sign of lymphangitis or cellulitis then systemic therapy will also be necessary and in severe infections cloxacillin or flucloxacillin would be used. When there is recurrent chronic folliculitis a steroid antibiotic or bactericidal combination may be necessary, such as Gentacin-H or Vioform-Hydrocortisone cream. These should not be continued indefinitely because of the danger of sensitization. When there are repeated infective episodes the child, and in some cases other members of the household, should have nasal swabs to exclude staphylococcal carriage. While recurrent infection is a severe problem in a few cases it is remarkable how many atopics with broken skin are free from skin sepsis.

Most cases of infantile eczema can be managed at home but, if the eczema is very extensive, a period of intensive hospital treatment produces a dramatic remission. This may give the patient a fresh start but because the atopic tendency persists a suitable home regimen must be instituted. In young children systemic corticosteroids should be avoided and in older indi-

viduals their use can be justified only in the most severe cases because of the long-term side effects and the fact that after giving systemic corticosteroids the patient's eczema becomes more resistant to normal therapy and withdrawal becomes almost impossible.

VIRUS INFECTION

Infection of the skin with a virus causes two rare but very severe and life-threatening complications in the atopic person. If a herpes simplex infection becomes disseminated it may give rise to what is sometimes called Kaposi's varicelliform eruption. This may be accompanied by a severe systemic upset and in some cases encephalitis. In treating this condition the prompt administration of hyperimmune gamma globulin obtained from the Blood Transfusion Service limits the infection, and intravenous idoxuridine may improve the chances of survival in encephalitis.^{1 2}

Smallpox vaccination is contraindicated in atopic individuals but they may be inadvertently vaccinated on occasions or accidentally vaccinated by contact with others. This can give rise to eczema vaccinatum. The prognosis in this condition can be improved by the administration of antivaccinia immunoglobulin, also obtained through the Blood Transfusion Service.³ Given prophylactically this hyperimmune globulin probably diminishes the severe complications of vaccinia in an atopic person and, though this is not yet a routine procedure, vaccination could be done with vaccinia immunoglobulin given simultaneously if there was a high risk of smallpox. Possibly also methisazone has a place in the treatment of the complications of vaccinia.

Prevention

As more is known about the role of IgE it may be possible to prevent the eczema reaction developing. Disodium cromoglycate (which is effective in blocking histamine release in the lungs if given prophylactically) has had a major effect in controlling allergic asthma. Unfortunately this substance has no effect on the skin because it is not absorbed.

It has been suggested⁴ that atopic infants are IgA-deficient early in life and this may lead to over-stimulation of their IgE system before it is fully developed. Thus possibly the infants' exposure to foreign proteins should be limited in the early months of life. Practicably this might be very difficult but would provide an argument for breastfeeding atopic infants and delaying mixed feeding until after 6 months. Nevertheless, in practice there is little evidence that diet has any effect in eczema.

References

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