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The Management of the Problem Atopic Child in 1988

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Are there really any changes in the management of the problem atopic child in the last twenty to thirty years.

Though there are arguments even among expert dermatologists about the correct method of use of the topical corticosteroids in the atopic child, when one analyses these apparent differences critically and talks to individuals, the differences are more those of the published word than the practical acts. Some authors state categorically that all they use in childhood eczema is 1% hydrocortisone preparations. Analysis of their clinical practice does not, however, support the accuracy of their remarks. My cynical view is that many of us are afraid to put forward our real views for fear that they will be incorrectly interpreted by primary care physicians and that damage to our patients may result. The aggressive and rational use of topical corticosteroids remains the greatest advance in the management of eczema in the last three decades and it is my belief that we should now be educating all those concerned in the management of these children in the correct method of handling potent topical steroids in even young children. The misery caused to patient and family by uncontrolled eczema is far and away more serious than possible steroid side effects. We are all carried away by the anecdotal reports of serious side effects and even death caused by the inappropriate use of large quantities of potent topical steroids in children and, it is salutory to note that the worst case reports have been in psoriasis and not in eczema. There is also little doubt now that topical steroids do not stunt childrens growth unless used in gross excess or in insufficient dosage to control the stunting disease (atopic eczema). Many children cross growth percentile lines after effective control of their eczema, often with quite high potency topical corticosteroids.

It is now widely recognised that the use of gradually reducing concentrations of topical corticosteroids give rise to the longest remissions and the best control. Sudden changes from high to low potency steroids must be avoided at all costs. In my personal view I believe that education of the parents in correct use of varying potency of topical corticosteroids is relatively easy and worthwhile. All my patients are in possession of topical corticosteroids of widely varying potencies with detailed instruction on their correct usage. The use of diary cards and the return of all empty or part empty tubes to be weighed should also be a part of the routine management of the severe patients.

The role of topical and systemic antibiotics is another field which needs to be discussed. Aly in 1981 (1) first suggested that long term Erythromycin was of value in controlling flaring eczema, but many bacteriologists view this with anxiety as there is an obvious risk of the development of resistant organisms. I have recently become interested in this aspect and have developed a so called sandwich treatment. This involves the application of a topical antibacterial followed a few minutes later by a topical corticosteroid and then a layer of an emollient. This technique has been used on 22 patients so far with very gratifying but totally uncontrolled results. Three of the patients so treated had been given systemic corticosteroids by other physicians before we saw them. One child, the first, had spent 18 of the previous 24 months off school, 11 of them in hospital. She has lost no time off school in the last three years, but rapidly deteriorates if the topical antibiotic is stopped. What I completely fail to understand is why this technique works better than the use of a straight topical steroid antibiotic combination. Other workers are, I know, looking more scientifically at the basis for these sort of clinical results and most of us feel that flares of eczema result from superficial staphylococcal colonisation or subclinical infection. The topical antibacterial we have used is pseudomonic acid. There were three reasons for selecting this particular agent. Firstly the fact that it will never be used systemically, secondly its wide spectrum activity against staphylococci, and thirdly the fact that resistant organisms are very rare. Indeed this antibacterial can eradicate multiresistant methycillin resistant staphylococci.

I next turn to the most emotive and controversial aspect of the management of the atopic child, diet. That food allergy or intolerance occurs in atopic children is beyond argument. Many of our patients identify the responsible food either by refusal or obvious

exacerbation of their eczema, but scientific well controlled information is very hard to find. There are well controlled trials of egg and milk free diets in young infants and I believe all will agree that the use of such a diet for a minimum of 8 weeks in an infant with bad atopic eczema is a well worthwhile exercise. The proportion of children who benefit from such therapy varies widely in the literature from 10 to 65%. One problem seems to me to be that when children are placed in a controlled study of dietary measures, there is a very large placebo effect. The proponents of total exclusion/elemental diets are now more wary than in the past, especially after episodes of anaphylaxis following the introduction of new foods. Another of the very curious features is the ability of the older atopic to ingest foods which caused problems at an earlier age with apparently no reaction.

An even greater problem is raised by food additives in the form of colourants and preservatives. Many children apparently improve when additive and colourant free diets are suggested, but again it is very difficult to do controlled studies, and challenges with the suspected materials are often inconclusive and may be dangerous. That there are some children whose eczema improves with such dietary manipulation is undoubted, but as yet there is no accurate way of determining those children who will respond. Scratch tests, RAST tests and IgG4 antibody levels either alone or in combination still do not give a clear indication of the likely antigens.

The role of inhaled allergens has long been recognised in respiratory allergy but has been overlooked or ignored in cutaneous disease. Control of the allergens or the instigation of desensitising regimens have been an essential part of therapy for decades in respiratory allergy. Recent work suggests that this may play a significant and, indeed, a very important part in the management of atopic eczema. It is now certain that heavy exposure to the house dust mite or its P1 antigen may lead to significant flares of the disease. House dust mite is ubiquitous in the developed world and is increasing in incidence in the Third World and even in tented civilisations. Pollens whilst varying in specific type are again worldwide. Hewitt (2) was the first to report that house dust mite and other inhaled antigens might be important in the aetiology of eczema. At about the same time Kleinhams (3) showed a positive relationship between positive scratch tests and positive RASTs to house dust mite in atopic eczematous children, the percentage of positive scratch tests being higher than positive RASTs, but as no mention is made of the level of IgE, it is difficult to interpret some of his data, though other authors have shown a close relationship between positive scratch tests and positive RASTs in children with very high IgE levels. In the early 80's Mitchell (4) amongst others, reawakened interest in the aetiological importance of the house dust mite in atopic eczema. Several workers, using house dust mite P1 antigen, produced positive patch tests on abraded skin of adult atopics. This evidence of type 4 immune reactions to this antigen only occurred in those with positive scratch tests, high levels of IgE and positive RASTs to house dust mite. These workers confirmed by skin biopsy that the cellular infiltrate was suggestive of the type 4 immune response, thus suggesting that some of the flares in eczema might be due to contact with house dust mite and by inference with some other airborne allergens (5). Other workers have found the results difficult to repeat, but this was the first real evidence that house dust mite could be of real importance. Some remain worried by the fact that these results were obtained on abraded skin, but such abrasion occurs in the atopic especially the child who scratches assiduously. Cell mediated immunity to house dust mite was also demonstrated in 11 out of 16 atopic subjects based on increased DNA synthesis after culture of the patients lymphocytes with house dust mite P1 antigen (6). They were unable to show that this reaction was mediated by IgE and others have suggested that mediation may be via IgG4 antibodies. T cells specifically sensitive to house dust mite P1 antigen were found in the majority of patients with Type 1 hypersensitivity to house dust mite P1 antigen.

Constant and persistant re-exposure to house dust mite and other inhaled antigens may lead to failure by the atopic patient or his physician to recognise their importance, thus giving rise to great difficulty in assessing the results of house dust or pollen control methods so often used successfully in respiratory allergy. There are three possible methods of attempting to deal with house dust mite. Avoidance regimens often helpful in respiratory allergy, hyposensitisation, or the removal of the house dust mite from the patient's environment.

In an open study in general practice, Chait et al. (7) reported improvement in the skin of atopic eczema patients with a house dust mite avoidance regimen. In 1985 Zachariae et al. (8) reported on hyposensitisation regimes over several years in adult atopic eczema patients. Whilst they demonstrated a worthwhile de-

crease in symptoms and signs, they felt that the level of reactions made the results generally disappointing. Others are actively studying desensitisation regimens. Rowntree et al. (9) have developed a double antibody antigen binding assay for house dust mite and other inhaled antigens. They believe this to be very specific and to suggest that both type 1 and type 4 immune reactions are responsible for the atopics reaction to airborne antigens. It is thus in recent years that interest has been revitalised in the role of ingested and inhaled allergens in atopic eczema and there seems little doubt that these aspects have been ignored. It seems clear that there is a vital part to be played by house dust mite control measures in atopic eczema patients, especially in childhood. There is good evidence that such measures are of value in respiratory allergy but as yet only anecdotal evidence in eczema, with the exception of the work of Zachariae. With the availability of Natamycin as a method of controlling the food chain of the house dust mite, a new and exciting chapter was opened.

I considered that those children with an early morning flare of their eczema, following bedtime exposure to house dust mite, could be those where this technique would be helpful. We therefore selected children with an early morning flare, high IgE, positive RAST and scratch tests to house dust mite (some with associated respiratory allergy). We monitored response by topical steroid consumption in grammes per week and clinical state as assessed by physician, child and parent. Twelve children have been treated in an open uncontrolled trial.

Three remained unchanged, three showed moderate improvement (of these two had respiratory allergy which was noted to improve) and six showed really significant improvement with reduction of topical steroid usage by over 50%. The study was undertaken over a period of six months.

I believe further work is required to establish the importance of the house dust mite in eczema, but feel very strongly that case selection must be very careful and the technique only used where there is good clinical or immunological suggestion that house dust mite is playing a part.

We now come to what might be described as the experimental therapies, some have been tried and discarded; azathioprine, levamizole, transfer factor, H2 antagonists, phospodiesterase inhibitors, photochemotherapy (we shall hear more of this in a few minutes, its place is clear in adults and now perhaps in childhood). Phototherapy with both UVA and UVB is an exciting new area.

Oral sodium cromoglycate therapy has been the subject of several trials with conflicting results. This is a useful and safe form of therapy that may help some children. The newer steroids with some differential between topical and systemic effects are another exciting development. Then we have to consider the linolenic acid story on which there is as yet conflicting evidence, many trials of gamma linolenic acid producing conflicting reports though Burton believes that overall the effect of gamma linolenic acid is favourable.

Finally, as a clinical scientist I am becoming more and more aware that in dealing with the atopic child with eczema we must try many of the safer new ideas rather than waiting for controlled trials which in this most perplexing disease may give information which clouds the issue for the individual patient. The disease remains as Sulzberger said, one which perplexes both patient and physician alike.

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