Personal practice

Dietary treatment of atopic eczema

T J DAVID

Department of Child Health, University of Manchester

Atopic eczema is merely a skin disease to doctors, but to children it can be a disfiguring handicap. Conventional treatment comprises the use of emollients, steroids, sedating antihistamines at night, antibiotics, bandaging, and the avoidance of irritant detergents or clothing.¹ Even with the most devoted application of these measures, there remain a number of children with persistent and troublesome disease. This paper examines the role for elimination diets. The subject is controversial, a legacy of the serious lack of objective data.

Evidence from clinical studies

There are no double blind studies in children with eczema comparing dietary therapy with other treatment, and given the cooperation required with diets it is difficult to see how it would be possible to apply a diet in a truly blind manner. Absence of such studies is serious, for without them it is impossible to tell whether diets are simply of temporary benefit (until the child grows out of the disease) or whether such treatment actually influences the ultimate prognosis by shortening the duration of the disease. There is also a lack of published data about the long term results of different elimination diets. A starting point are the following established facts:

(1) Double blind challenge studies have established that atopic eczema can be a feature of cows' milk protein intolerance. The incidence of eczema in patients with cows' milk protein intolerance depends on how they are selected, but lies between 10% and 40%.^{2,3}

(2) Studies of double blind food challenges have established that in selected children with atopic eczema, a number of foods in doses of 8 g or less can provoke an immediate (within two hours) itchy erythematous macular rash, accompanied by a significant rise in the plasma histamine concentration.^{4,5} There are two problems with these types of data. Firstly, implicit in these studies is the inference that these erythematous rashes would lead to the development or worsening of eczema if

further doses of challenge material were given, but this has not been proved. Challenges were given as little as five hours apart, precluding the observation of delayed onset reactions or worsening of eczema. The conventional dermatological view in Britain is that these immediate erythematous reactions are indeed common in children with atopic eczema but represent an entirely separate and unrelated problem.⁶ Secondly, a single provocation with a small dose in the laboratory differs greatly from the real life situation of continuous or repeated exposure to larger doses of foods. For example, Hill found that whereas 8 to 10 g of cows' milk powder (corresponding to 60 to 70 ml of milk) was adequate to provoke a response in some patients with cows' milk protein intolerance, other patients (with late onset symptoms and particularly eczema³) required up to 10 times this volume of milk daily for more than 48 hours before symptoms developed.⁷

(3) There are several studies in which children with atopic eczema improved on an elimination diet and were then challenged openly or double blind with single foods.⁸⁻¹⁰ The drawback to this study design is that positive double blind challenges can confirm the presence of food intolerance but do not establish how much improvement is attributable to food avoidance and how much is due to a placebo effect.

Patient selection

Elimination diets are easy to prescribe but difficult to follow. By preventing a child from eating food consumed by family or friends a diet can itself be a handicap, and the emotional consequences of such treatment have never been studied. Elimination diets are costly,¹¹ and even the most highly motivated family may be unable or unwilling to comply with the diet despite improvement in the eczema.⁹

In view of the relative complexity of diets, it makes no sense to use a diet where simple and safe topical treatment will suffice. Local experience is that a large number of children referred with eczema

have unrecognised but simply treatable problems such as bacterial infection,¹² dry ichthyotic skin,¹ or long dirty fingernails, for which dietary treatment would be irrelevant.

Skin prick tests and radioallergosorbent (RAST) tests are not helpful because of the high rate of false positive¹³ and false negative¹⁴ results. Neither test can indicate which patient will respond to a diet, or which foods should be eliminated.¹⁵

Objective data from which one could derive recommendations for patient selection are unavailable. Based on local treatment and follow up, the best indications for trial of an elimination diet are:

(1) Widespread (for example, affecting 15% or more of the skin surface area) atopic eczema unresponsive to topical treatment.

(2) Patient aged under 12 months, partly because elimination diets are simpler to administer and control in infancy,⁹ and also because it is a general observation that results are better in this age group.¹⁵

(3) Patient with symptoms in other systems, particularly persistent loose stools, or other atopic disorders such as asthma. It is far less common for food intolerance to cause a single symptom (for example, eczema) than multiple symptoms (for example, eczema, asthma, loose stools, and vomiting). In one study where selected children with atopic eczema received double blind food challenges, gastrointestinal symptoms (vomiting, loose stools, abdominal pain) occurred in 52% of positive challenges, and respiratory symptoms (asthma, stridor, sneezing) occurred in 32% of positive challenges.⁴

(4) Severe eczema in exclusively breast fed infants. In one study, in six of 37 breast fed infants, eczema improved when the mother avoided cows' milk protein and egg and relapsed when these were reintroduced.¹⁶ It was impossible to predict which baby would respond to maternal dietary exclusion, and it is reasonable to try maternal avoidance of cows' milk in an infant with eczema who is being exclusively breast fed. Other foods can provoke eczema in this way, but their detection depends on parental suspicion followed by avoidance and challenge.

(5) Parents keen to try a diet. Where eczema is widespread and unresponsive to simple treatment, and where the parents are keen to try food elimination, it makes sense to try a diet with proper control and supervision rather than driving the family into the hands of an unorthodox therapist.¹⁷

Elimination diet procedure

The fundamental strategy is to try an elimination

diet for a defined period of time. Six weeks is long enough to ensure that any improvement will not have been missed, while also allowing for the inevitable day to day fluctuations in disease activity. At the end of this period the patient is assessed by the same person who saw the patient at the beginning of the diet, and the diet abandoned if there has not been much improvement. Parents (and children) are sometimes so keen that a diet should succeed that they perceive improvement where none exists, and they need the help of an objective observer. Otherwise there is a risk that a useless diet will be continued indefinitely.

If the diet has clearly helped, and the parents and child wish to continue, then the next step is to try to identify food triggers by the reintroduction of foods singly at the rate of one new food every five to seven days. Foods which are tolerated are left in the diet. The presence of food intolerance is suggested by worsening of the disease after introduction of a food, followed by improvement when the food is stopped. Failure of the disease to improve when a new food is stopped suggests an alternative cause for the worsening. The identification of food triggers is far from easy because of confounding variables such as the normal daily fluctuation in disease activity, intercurrent infections (which may worsen or improve eczema), exposure to other triggers (for example, pets), or teething. There is a small risk of anaphylaxis occurring with food reintroductions, particularly with cows' milk and with patients on elemental diets.¹⁸

Supervision by a dietician is required, for without it there is a risk that the diet will fail because specific food items have not been fully excluded. Elimination diets carry the risk of nutritional inadequacy,¹⁹ even where a cows' milk substitute has been prescribed, and is another reason for dietetic support.²⁰

Local experience is that in atopic eczema the incidence of allergy to household pets and house dust mites is at least as common, if not more so, than food intolerance. It is logical to consider attempts to avoid these in conjunction with a diet, as a diet may fail in the face of continued exposure to pets or dust mites. As with foods, tests cannot predict the outcome of environmental allergen avoidance.

Types of diet

(1) HALF HEARTED ATTEMPTS TO 'HAVE A GO'

The very small quantity of food which can provoke an adverse reaction means that the 'try cutting down his milk' type of tinkering with the diet is most unlikely to succeed. The advantage of a carefully conducted diet is that even if it fails at least the

parents will be satisfied in the knowledge that it was tried properly.

(2) COMPLETE AVOIDANCE OF KNOWN TRIGGERS

A trial of rigorous avoidance of known or suspected triggers is a logical first step. It is common to see a child with a clear history of intolerance to a food, but where the food is being incompletely avoided. An example would be a child with a history suggesting cows' milk protein intolerance who is avoiding cows' milk but consuming products which contain whey or casein, or who is receiving goats' milk. (The appreciable antigenic similarity between cows' and goats' milk proteins²¹ suggests that goats' milk is unlikely to be tolerated in most children with genuine cows' milk protein intolerance.)

(3) COWS' MILK PROTEIN AND EGG AVOIDANCE

Trials of this treatment have given conflicting results.^{15 22} Local experience is that less than 20% of patients benefit, and the main place for this diet is in infancy.

(4) ELIMINATION OF COMMON POSSIBLE TRIGGERS

The patient avoids foods for which there is a history of intolerance, plus approximately 10 common food triggers such as cows' milk, egg, wheat, fish, legumes (pea, bean, soya, lentil), tomato, nuts, berries and currants, citrus fruit, and food additives (azo dyes, benzoates, and sulphites). Local experience is that less than 20% experience a useful clinical benefit.

(5) THE FEW FOOD DIET

This consists of exclusion of all foods except for five or six items.²³ Such diets comprise a meat (usually lamb or turkey), three vegetables (for example, potato, rice, and carrot or a brassica—cauliflower, cabbage, broccoli, or sprouts), a fruit (usually pear) and possibly a breakfast cereal (for example, Rice Crispies). Local experience is that in children under the age of 5 years with 25% or more skin surface affected, approximately a quarter can be expected to usefully improve on the diet.

(6) ELEMENTAL DIET

The application of an inpatient regimen of four to six weeks of a so called elemental diet (for example, Vivonex, Norwich Eaton Laboratories) is the ultimate test of whether food intolerance is relevant or not, but until more data are available this approach must be regarded as experimental. Based on unpublished local experience, the drawbacks comprise the lack of a guarantee of success, family disruption associated with two to three month's hospitalisation, and sometimes loose stools (due to hyperosmolarity

of the diet), severe weight loss, and profound hypoalbuminaemia.

Conclusions

Like many chronic diseases, there is a strong placebo effect of any new treatment applied with enthusiasm, particularly if the parents are convinced it will help. Long term follow up of patients seen locally and elsewhere suggests, however, that probably no more than 10% of children with atopic eczema severe enough to warrant referral to hospital are likely to derive lasting benefit from the use of an elimination diet.¹⁵ As diets are difficult and potentially hazardous, and are possibly best employed in conjunction with pet and dust mite avoidance, it is logical to apply simpler conventional treatment first. Despite the somewhat disappointing overall results of diets, a number of patients with severe disease experience dramatic and lasting benefit from specific food avoidance, and the detection of these children by the application of empirically based diets is an important part of the management of eczema.

References

- David TJ. The practical management of atopic eczema in childhood. *Maternal and Child Health* 1986;**11**:60–4.
- Goldman AS, Anderson DW, Sellers WA, Saperstein S, Kniker WT, Halpern SR. Milk allergy. I. Oral challenge with milk and isolated milk proteins in allergic children. *Pediatrics* 1963;**32**: 425–43.
- Hill DJ, Ford RPK, Shelton MJ, Hosking CS. A study of 100 infants and young children with cows' milk allergy. *Clin Rev Allergy* 1984;**2**:125–42.
- Sampson HA, McCaskill CC. Food hypersensitivity and atopic dermatitis: evaluation of 113 patients. *J Pediatr* 1985;**107**: 669–75.
- Sampson HA, Jolic PA. Increased plasma histamine concentration after food challenges in children with atopic dermatitis. *N Engl J Med* 1984;**311**:372–6.
- Allen R. Role of diet in treating atopic eczema: dietary manipulation has no value. *Br Med J* 1988;**297**:1459–60.
- Hill DJ, Ball G, Hosking CS. Clinical manifestations of cows' milk allergy in childhood. I. Associations with in-vitro cellular immune responses. *Clin Allergy* 1988;**18**:469–79.
- Hill DJ, Lynch BC. Elemental diet in the management of severe eczema in childhood. *Clin Allergy* 1982;**12**:313–5.
- Hathaway MJ, Warner JO. Compliance problems in the dietary management of eczema. *Arch Dis Child* 1983;**58**:463–4.
- Van Asperen PP, Lewis M, Rogers M, Kemp AS, Thompson S. Experience with an elimination diet in children with atopic dermatitis. *Clin Allergy* 1983;**13**:479–85.
- Macdonald A, Forsythe WL. The cost of nutrition and diet therapy for low-income families. *Hum Nutr Appl Nutr* 1986;**40A**:87–96.
- David TJ, Cambridge GC. Bacterial infection and atopic eczema. *Arch Dis Child* 1986;**61**:20–3.
- Sampson HA, Alberg R. Comparison of results of skin tests, RAST, and double-blind, placebo-controlled food challenges in children with atopic dermatitis. *J Allergy Clin Immunol* 1984;**74**: 26–33.
- Hill DJ, Duke AM, Hosking CS, Hudson IL. Clinical manifesta-

- tions of cows' milk allergy in childhood. II. The diagnostic value of skin tests and RAST. *Clin Allergy* 1988;**18**:481-90.
- ¹⁵ Atherton DJ. Diet and atopic eczema. *Clin Allergy* 1988;**18**:215-28.
- ¹⁶ Cant AJ, Bailes JA, Marsden RA, Hewitt D. Effect of maternal dietary exclusion on breast fed infants with eczema: two controlled studies. *Br Med J* 1986;**293**:231-3.
- ¹⁷ David TJ. Unhelpful recent developments in the diagnosis and treatment of allergy and food intolerance in children. In: Dobbing J, ed. *Food intolerance*. London:Baillière Tindall, 1987:185-214.
- ¹⁸ David TJ. Anaphylactic shock during elimination diets for severe atopic eczema. *Arch Dis Child* 1984;**59**:983-6.
- ¹⁹ David TJ, Waddington E, Stanton RHJ. Nutritional hazards of elimination diets in children with atopic eczema. *Arch Dis Child* 1984;**59**:323-5.
- ²⁰ Devlin J, Stanton RHJ, David TJ. Calcium intake and cows' milk free diets. *Arch Dis Child* 1989;**64**:1183-4.
- ²¹ Crawford LV, Grogan FT. Allergenicity of cows' milk proteins. IV. Relationship of goats' milk proteins as studied by serum-agar precipitation. *J Pediatr* 1961;**59**:347-50.
- ²² Neild VS, Marsden RA, Bailes JA, Bland JM. Egg and milk exclusion diets in atopic eczema. *Br J Dermatol* 1986;**114**:117-23.
- ²³ Thomas B, ed. *Atopic eczema (infantile eczema, dermatitis). Manual of dietetic practice*. Oxford: Blackwell, 1988:527-31.

Correspondence to Dr TJ David, Booth Hall Children's Hospital, Charlestown Road, Blackley, Manchester M9 2AA.

Dr Clement A Smith

Recently, at the age of 87, Dr Clement Smith died peacefully at his home in Cambridge, Massachusetts. In the 1940s and 50s it was he more than anyone else who set the scientific tone for the new speciality of neonatal paediatrics. He made important contributions to the study of hyaline membrane disease relating to abnormalities in lung mechanics and to the associated metabolic disturbance attributable to tissue breakdown. It was, however, as a reviewer and critical commentator that he made his most important contributions. In particular his book, *Physiology of the Newborn Infant*, first published in 1945, served as a powerful advocate of the scientific approach to this subject. Among the 50 or so fellows who studied under Clem in the course of his research career, many came from European countries, including the United Kingdom. In this way he planted the seeds of his scientific curiosity in many places far removed geographically and spiritually from Boston.

Born and raised in Ann Arbor, he retained the pleasing style of a grown up Tom Sawyer. Mark Twain was certainly one of his many literary heroes. His early education included a master's degree in English Literature and he never lost his relish for the pleasures of reading and writing. His long career at Harvard Medical School began with a residency at Children's in 1931 and ended only with his last attendance at grand rounds in December 1988. From 1962-73 he was Editor of the important journal, *Pediatrics*; and in 1983 he published a history of The Boston Children's Hospital. Many of his pupils will remember with pleasure the good tempered wit, the modest courtesy, and the generous hospitality of this old fashioned American gentleman. His influence on the development of neonatal paediatrics will continue for many years.

L B STRANG
University College London