

Food Allergy in Atopic Eczema

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Food allergies are commonly suspected in young children with atopic eczema, though among dermatologists, there have been varying opinions regarding the importance of food allergy in atopic eczema. Food allergy reactions can be divided into rapid-onset and late-onset reactions which may occur up to twenty four hours after exposure. Rapid onset reactions are further divided into contact reactions and systemic reactions. Contact reactions commonly present as urticaria at the point of contact with the provocative food. Other less common forms of contact reactions include oro-pharyngeal angioedema, in which there is swelling of the oropharynx and lips with difficulty in breathing and is often mistaken for anaphylaxis. Abdominal pain and vomiting may also occur as a contact reaction within the gastro-intestinal tract, with egg being the most common allergen implicated in these situations. Systemic reactions most often occur as generalised urticaria and nasal congestion. Occasionally, wheeze and anaphylaxis may occur. It is important to distinguish between contact allergy and pseudo-allergic reactions in which the reaction is due to irritation. Salty or acidic foods such as tomatoes, citrus fruits and crisps are commonly responsible for pseudo-allergic reactions.

There have been conflicting results on the efficacy of dietary treatment in atopic eczema. Most of these studies were open, uncontrolled studies which did not include the late response in the assessment of the patient. Also, the amounts of food used for testing were very much less than that normally taken and may not have been an accurate reflection of the actual eating habit of the patient. The late response is not uncommon. In a study on cow's milk allergy in atopic eczema, 19 out of the 30 patients studied did not develop an allergic response till after 24 hours, while only 11 developed a

reaction within six hours. Foods often suspected of leading to an exacerbation of eczema include cow's milk, peanut, soy, fish, egg, and wheat. There has been much evidence to suggest that an exclusion diet is beneficial. In one placebo-controlled, crossover trial of an egg and cow's milk exclusion diet, 14 of the 20 children who completed the trial improved with the exclusion diet, while only one child improved on the control diet. Although in many studies, skin testing and RAST were used to test for suspected foods, in the above study, there was no correlation between the response to an exclusion diet and the presence or absence of positive skin tests or RAST to milk or egg. In another study, suspected foods were excluded and gradually re-introduced every four weeks till a maintenance diet was defined. Thirty-five per cent of the children tested, who were under eight years old with atopic eczema responded to exclusion of milk and egg. In contrast, in other studies, there was no difference in skin condition between patients who initially responded to an exclusion diet and those who did not when evaluated at one year. In addition, many studies have not been able to reproduce the exacerbation of eczema by the suspected foods. This may be because the response to an allergen depends on the circumstances during exposure. This is illustrated by a recently described condition, **food-dependent exercise-induced anaphylaxis**. In this condition, there is no allergic response when the patient is exposed to either the provocative food or exercise alone, but when exposed together, anaphylaxis occurs.

There are several problems associated with an exclusion diet, including a limited response rate and an unknown duration of benefit. Reliable tests to identify provocative foods are not available. With overzealous exclusion of foods, there is a risk of inadequate nutrition especially in young children in whom food allergies are more likely to be suspected. There is also a risk of inducing obsessive eating habits. In cases where the patient is relatively tolerant of the skin condition, an exclusion diet may actually worsen the quality of life. In addition, patients may have already excluded certain foods and the benefit from further exclusion of foods may be limited. This is particularly relevant in Hong

Kong where belief in dietary restriction for illness is common.

In cases where other treatments have failed and the history is suggestive of food allergy, an exclusion diet may be tried. In the assessment of the patient, it is important to identify foods which lead to rapid onset reactions to avoid anaphylaxis. Skin tests or RAST tests may be able to provide further information on suspected food allergies. Dietary restrictions which are already in force are also identified. If the provocative foods cannot be reliably identified, an empirical diet excluding cow milk, egg, artificial colours and preservatives may be tried. The patient is assessed after four to eight weeks. If an exclusion diet has improved the skin condition, it is continued for up to one year. After one year, reintroduction of the provocative food may be attempted as the patient may have developed tolerance during this period. As a precaution, the provocative food may be applied topically to test for contact allergy before ingestion is tried. It is often assumed that patients are permanently intolerant of the provocative food but in many cases tolerance develops following a period of

avoidance, especially in young children. There has been evidence that in children with a family history of atopic eczema, avoidance or delaying exposure to the provocative foods such as cow's milk may prevent or delay the onset of eczema. An exclusion diet may therefore be more beneficial in these cases.

In conclusion, although food allergy is often suspected in young children with atopic eczema, it is difficult to assess as the allergic response is not always reproducible. An exclusion diet may be relevant in those patients in whom the history is suggestive of food allergy or where there is a family history.

Learning points:

Food allergies are often suspected in young children with atopic eczema, but many factors need to be considered before an exclusion diet is introduced. The efficacy of an exclusion diet is often difficult to assess as allergic responses are not always reproducible. Tolerance to provocative food may develop after a period of avoidance.