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Editorial

A dermatologist's view on allergy tests

The prevalence of atopic dermatitis (AD) and related diseases has doubled in urbanised cities over the last 20 years. It is also a common belief among Chinese about the role of food allergy in diverse skin diseases. Parents often blame allergy for their children's disease and seek causes for AD that they can cure by avoidance. Thus, there are overwhelming requests for allergy tests and their interpretations in our daily practice. Most of the allergy tests except skin patch tests are tests for IgE-mediated hypersensitivities. Our concern is how useful these tests are in patients' management. What is the role of such tests in the management of skin diseases with potential allergy aetiologies? Can such tests tell what we want to know such as significance of food allergy in atopic dermatitis (AD) and its prevention? In this issue, the article by Ho MHK et al provided us with a brief review on the application of allergy tests from paediatricians' point of view.

The term 'atopy' was first used to describe a group of diseases comprising asthma, hay fever in patients whom a family history of similar disorders was common. Many of whom also had infantile eczema. Subsequent identification of abnormal regulation of IgE production led to the grouping of these diseases as atopic in nature. Some authors use the term to describe the inherited tendency to develop IgE antibodies to specific allergens and believe that it predisposes individuals to the development asthma, allergic rhinitis and AD. Consensus regarding a precise definition for atopy is still lacking.

AD is a difficult condition to define, because it lacks a diagnostic test and shows variable clinical features. Atopic illnesses are usually defined using clinical criteria that have been determined by consensus and consideration must be given to the heterogeneous nature of these diseases. It is further complicated by the impreciseness of its association with atopy and the enigmatic nature of atopy itself. The pathogenesis of AD is not fully understood and it remains a matter of debate as to whether AD is an allergic disorder or is an inflammatory skin disease frequently associated with other forms of atopy. A review of literature suggests that there is some evidence that allergens are contributing to the pathology of AD but the extent to which they are involved in the induction of disease remains to be established.

Difference in opinion exists between dermatologists and allergists in the inclusion of allergen-specific IgE as the mandatory criterion of AD. Defining AD in this framework of atopy is not complete. It is difficult to understand AD entirely on the basis of the presence of an abnormal IgE response to environmental allergens. Firstly, approximately 30% of individuals with AD have a normal total IgE level and they lack specific sensitization against inhalant and food allergens as evidenced by negative IgE in blood and skin prick test (SPT) results. On the other hand, highly elevated IgE levels can be present without clinical manifestations of atopy and eczema. Secondly, AD is rarely attributable to a specific allergic reaction, and IgE antibodies present often

appear to be an epiphenomenon. A positive specific IgE test only indicates a level of biological sensitivity to the relevant allergen but it may persist in the absence of symptoms.

An important feature of atopic illness is the tendency to recognise ubiquitous environmental antigens such as house dust mites or certain foods as allergens and to develop specific IgE antibodies against them. Compared with other atopic diseases, AD has the highest proportion of multiple raised IgE antibodies and positive skin tests. Therefore, children with significant AD are more likely to have IgE sensitization to at least some if not many tested allergens. IgE sensitization to multiple allergens may not be associated with any symptoms upon clinical challenge and attempts to avoid all those allergens often do not improve AD. Ideally, the early identification of the chief allergens and the successful avoidance or desensitization can prevent and modify the course of the allergic diseases. Unfortunately, these effects have not yet been demonstrated in AD.

There are discrepancies in the approach of AD by paediatricians/allergists and dermatologists. Allergists tend to believe that there is often a role of allergy in patients with AD. It remains controversial regarding the role of food allergy in children with AD, dietary manipulation for the control and prevention of eczema. For instance, the co-occurrence of eczema and food allergies, such as anaphylaxis to peanut, does not necessarily mean that ingestion of the foods is exacerbating or causing the eczema as patients already avoid peanut totally anyway. Dermatologists often observe that positive SPT or radioallergosorbent test (RAST) especially to foods often shows no correlation with clinical manifestations of AD. The fact that the child is reported to react adversely to one or more foods does not necessarily mean that the child's AD will improve when that food is avoided. Even when the diagnosis of food sensitivity is confirmed by stringent food challenge, it does not prove that the improvement of AD is caused by the diet.

One of the reasons may be most patients with AD perhaps seek paediatricians' advice before dermatologist. Paediatricians tend to see infants and young children for AD while dermatologists are consulted for persistent AD in older children. The relation between AD and exposure to food allergens seems to decline sharply with age. Thus only in very young individuals, there is a place for testing with food allergens. Some of these food allergies outgrow quickly and as a result some of these patients' skin condition improve quickly in the first 2 years of life. They do not necessarily improve solely to dietary manipulation and adherence to topical treatment cannot be overemphasized. On the other hand, it remains for me to say that there exists a subgroup of young children with AD in which dietary manipulation such as removal of milk or egg from the diet together with appropriate dermatologic treatment will result in significant improvement. Without randomised controlled studies, it is impossible to tell how much of this improvement is due to a placebo effect or concurrent therapy. The shortage of randomised controlled studies of dietary treatment means that the role of elimination diets remains uncertain. There is inadequate evidence that specific food avoidance alters the natural history of AD. The disease course might still be confounded by other environmental triggers. Moreover, we do not have reliable tests for the diagnosis of food hypersensitivity to predict the outcome of dietary elimination in AD.

From dermatologists' point of view, interpretations of allergy tests for food hypersensitivity are notoriously difficult in children with AD because of a large number of

false-positive reactions. The best guide to the relevance of an allergen is a clear and consistent history of exacerbation of AD and other organ manifestations following exposure. Given the uncertain role of allergy in AD and avoidance of those sensitized allergens identified by the tests is often difficult to comply or imposing a significantly negative impact to one's nutritional status or quality of life, it is logical to apply standard conventional therapy first.

In conclusion, simple tests for the identification of allergens that can provoke AD following exposure are not available currently. Evidence of benefit from allergy testing is not conclusive and outcome of avoidance regimes are controversial. Clinicians need to understand the implication and limitation of allergy tests before subjecting patients to the expenses, procedures and subsequent tedious interventions as a result of the testing. I believe a diagnosis of allergy is based chiefly on a careful clinical history and allergy tests are at most to support the diagnosis. Indiscriminate allergy tests covering wide range of indications and allergens simply add no benefits to patients' management. I concur with authors that a way forward is the close collaboration between private practitioners and specialist service provided by secondary or tertiary referral centres in managing allergic diseases. Those conditions that are refractory to what ought to be appropriate treatment and are suspected to have an allergic component can be referred to the specialist clinic for further advices.

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