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Editorial

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Food Hypersensitivity and Allergic Diseases: A New Threat in India

Much of what happens in the industrialized countries of North America and Europe comes to dominate the Indian scene decades later. This is true of social norms, fashion, architecture, films, and diseases of affluence such as hypertension, heart disease and diabetes. To this list, we can add allergic diseases. The dark specter of allergic disorders is very much with us now and is likely to grow geometrically in the coming years. Recent data from India supports this apprehension(1).

The "allergic march" refers to a sequence of events that is seen from fetal life to adulthood, and consists of sensitization to foods, gastrointestinal symptoms, eczema, wheezing and asthma, and hay fever(2). In many individuals, this sequence occurs almost as a preprogrammed event but in some persons, one or more syndromes may be skipped. It also follows that the prevention of the initial step of sensitization to foods and other potential allergens may well reduce the incidence of other symptoms and signs(3,4).

In this selective review, some of the cardinal clinical features of food allergy and atopic disease are reviewed and emphasis is placed on the distinct possibility of prevention of allergic disorders by intervention in early life.

Immunologic processes and genetic factors

There are four distinct immunologic mechanisms that underlie food hyper-sensitivity(2). More than one process can be at work simultaneously. This is important to realize because distinct clinical and laboratory tests are required to diagnose each of the pathogenetic mechanisms. Type I refers to IgE-mediated immediate hypersensitivity that can manifest as urticaria and anaphylaxis. Type II is mediated through antibody fixed on the surface of a cell, and may manifest as anemia, leukopenia and thrombocytopenia. Type III is mediated by antigen-antibody-complement complexes and its clinical features may include nephropathy and gastroenteropathy. Type IV is mediated by T cells and one clinical outcome may be contact dermatitis.

Many HLA types and clusters of genes have been associated with predisposition to allergic disease. No single genetic locus is linked to a disease entity. There is also some suggestion that maternal influence may be more important in some cases and paternal one in others. Obviously much more needs to be learnt in this field.

A link between genetics and immunology might lie in the pattern of TH1 : TH2 balance that could be an important pathogenetic factor. It is now known that individuals with allergic disease have a greater predilection for TH2-linked cytokine production; for example interleukin-4 and interleukin-6. On the other hand, individuals with no allergic symptoms generally have a greater production of TH1-linked cytokines, such as interleukin-2 and interferon-gamma. In this context, a less sanitary environment and exposure to common childhood diseases would help in reducing the risk of allergy in late childhood, as observed to some extent in less privileged populations. However, the magnitude of this effect and its translation into any practical recommendations remains to be worked upon. There is preliminary information to suggest that selected micronutrients in certain doses may have the potential to alter TH1:TH2 balance(1).

Clinical Features

Virtually any organ of the body may be affected by allergic disease. Thus skin manifestations may include skin rash, urticaria, angioedema and purpura. Gastrointestinal symptoms may include colic, diarrhea, malabsorption and blood in stools. Wheezing, asthma and rhinitis may occur. Other unusual and rare clinical features include migraine, hyperactivity, tics, reduced attention span and anaphylaxis.

Infants at Risk

Parental history positive for any of the atopic manifestations increases the risk of an allergic disorder in the offspring. If both parents are affected, and particularly if both have the same clinical syndrome such as asthma or eczema, the risk of its occurring in the child is much higher. The approximate risk is 40% with one parent affected, 60% with both parents affected, and 85% if both parents have the same clinical syndrome.

In addition, some laboratory tests might help, for example a high serum IgE in the first week of life, reduced number of gamma-delta CD8+ T lymphocytes, reduced mannan-binding protein and altered cord blood alpha-linolenic acid levels. Preliminary data suggest that a high interleukin-4: interferon-gamma ratio is indicative of high risk.

Management

A common sense approach that includes reduced exposure to offending foods and other allergens will keep the symptoms and signs under reasonable control. Identification of offending agents can be achieved by extensive clinical history, skin tests, limited number of blood tests and clinical challenges. Infants with severe allergy to milk can be given extensively hydrolyzed formulas (5,6). Soy formulas are also useful but one runs the risk of subsequent development of allergy to soy in as many 45% of patients.

Antihistaminics and local steroid creams are required for control of itching and cutaneous lesions. Local steroid sprays are useful for rhinitis and asthma. In patients with history of anaphylaxis, it is mandatory to provide them with self-administered adrenaline. Children as young as 4 years can be taught to inject themselves with adrenaline when the need warrants it. Two doses should be kept handy and a second dose administered if the symptoms have not abated within 15 minutes. Even a single injection of adrenaline will avert fatality.

Prevention

Exclusive breast feeding for four months is associated with reduced occurrence of eczema and gastrointestinal manifestations(7-10). Some studies suggest that a long-term reduction in the incidence of asthma and rhinitis might also occur(11). The benefits are greater if the mother's diet during lactation is controlled(8,9,12). Highly allergenic foods such as dairy products, egg, peanut and soy should be eliminated.

In those not breast-fed or when supplemental formula is required, a partial whey-hydrolyzate formula is the best choice in terms of effectiveness, taste and cost(11, 13-15). Extensively hydrolyzed formulas are also effective; however, they are expensive and do not taste good. There are also some theoretical considerations that weigh in favour of partial rather than extensively hydrolyzed formulas for prevention(15). Delayed introduction of solids and reduced exposure to tobacco smoke and dust mites is helpful. It is also necessary to explore new approaches for restoring optimum immune responses(16), in particular the balance between TH1 and TH2 balance.

Final comments

The prevalence of allergic diseases has increased several-fold in most industrialized countries of the world

in the last 25 years. India is following that path. The incidence of allergic disorders, particularly asthma, has increased considerably. It would be prudent to consider active measures for the prevention of food hypersensitivity and allergic disease.

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