Perspective

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Assessment of Food Allergy Severity

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Perspective

Food allergies are potentially fatal, and there is presently no cure. Food allergy prevalence and hospitalizations for food-induced anaphylaxis have both grown in recent decades. Food allergy management is based on rigorous avoidance of the culprit allergen and the carrying of emergency medicine to treat an acute allergic response if an accidental exposure occurs. Accidental responses to popular foods such as milk, eggs, and peanuts are frequent, and many families live in continuous terror of anaphylaxis. Food allergies put limits on patients and families social life in addition to nutritional restrictions.

There are several methods for categorizing allergic responses based on severity, and when studied in depth, there may be discrepancies between them. However, there is widespread agreement that symptoms affecting the airway, respiration, circulation, and awareness are severe. Because severe allergic reactions can be fatal, it is critical to correctly identify patients at risk and provide them with emergency medication, a written treatment plan, and education on how and when to use the medication, allergen avoidance, and its practical management and implications in everyday life.

The severity of allergic reactions, on the other hand, is determined by a variety of factors, some of which are related to the allergen (e.g., matrix, dose, and processing), others to the IgE-mediated immune response (e.g., IgE diversity, IgE levels, IgE avidity, mast cells and basophils), and others to the host, namely age (young adults and teenagers are at the highest risk of fatal reactions) and allergic c (e.g. timely and effective adrenaline use).

Objective biomarkers, such as specific IgE to allergen extracts, specific IgE to individual allergens, specific IgE to allergen peptides, Basophil Activation Test (BAT), and Mast Cell Activation Test (MAT) following allergen stimulation, have been investigated in various studies to see if they could predict severity in individual patients. The evidence regarding the potential use of specific IgE levels to allergen extracts as measured by IgE levels in serum or wheal diameter on Skin Prick Test (SPT) has been contradictory, with some positive studies, i.e. studies showing an association between higher levels and severe reactions, and some negative studies, i.e. studies not showing such an association.

Some research have connected particular IgE to major allergens, such as Ara h 2 from peanut, Cor a 9 and Cor a 14 from hazelnut, and Ana o 3 from cashew nut, to the severity of allergic responses to the corresponding nut; however, not all investigations were successful in demonstrating this relationship. A significant discovery, both at the entire allergen level (e.g., Ara h 2 from peanut) and at the level of allergen peptides (which contain epitopes), is that the higher the number of allergens or peptides (i.e., epitopes) recognized by IgE, the more severe the allergic reaction. Furthermore, IgE avidity for the allergen has been linked to more severe responses. Following allergen stimulation, all of these IgE properties (quantity, specificity, avidity, and variety) are evaluated together in the BAT and MAT. Thus, BAT and MAT may be regarded as tests of IgE function, i.e., its capacity to trigger effector cell activation and degranulation, resulting in the release of mediators responsible for allergic symptoms. A higher percentage of activated basophils and mast cells have been linked to more severe symptoms. A lack of Platelet-Activating Factor (PAF) acetyl hydrolase, which destroys PAF, has also been linked to severe anaphylaxis.

There are limitations to verifying a biomarker's value in predicting the severity of allergy responses, which may explain some of the discrepancies seen between published researches. For example, the clinical spectrum of the patients studied (e.g., patients with risk factors for severe reactions are frequently excluded from challenge studies, and in some studies, non-allergic subjects or patients who have outgrown food allergy have been included), the oral food challenge protocol (e.g., doses used, interval between doses, duration, matrix, and vehicle of the challenge food), and the oral food challenge protocol (e.g., doses used, interval between doses, duration, matrix, and vehicle of the challenge. Furthermore, the severity of allergic responses can be measured outside of the setting of oral food challenges, for

example, by utilizing questionnaires with patients self-reporting or examining instances brought to the emergency room, which might add another degree of inconsistency and subjectivity. A biomarker is simply one element of a complicated allergen-specific immune response, and studies analyses are often done at a group level, which may not be easily translated to predict severity at the individual level. Furthermore, the degree of responses during challenges might fluctuate over time and may not be representative of the severity of allergy reactions in the population.

Because the data on the utility of SPT and specific IgE in predicting the severity of allergic responses has been contradictory, we tend to reassure patients in clinic that allergy test findings only inform about the chance of having an allergic reaction, not its severity. While this is partially correct, the fact that a big SPT is related with a higher chance of responding to the allergen indicates that it is also associated with a higher likelihood of a severe reaction. A big SPT and a high level of peanut-specific IgE were proven predictors of severe reactions in a recent comprehensive research that looked at the outcomes of challenges of participants in the LEAP and related trials. The BAT was found to be the greatest single biomarker for predicting the severity of allergy responses in this investigation, outperforming IgE to peanut components, particularly Ara h 2. To predict severe or life-threatening allergic responses during peanut challenges, a cutoff of 48% of CD63+ basophils exhibited 100% sensitivity, 97% specificity, 41% positive predictive value, and 100% negative predictive value. Because of these performance characteristics, all individuals with severe peanut responses had results above the threshold, but an individual patient with a result over the specified cutoff would not always have a severe reaction, which might be comforting for patients in the clinic. An SPT to peanut larger than 8 mm and an Ara h 2-specific IgE greater than 1.5 KU/L also worked well, with 100% sensitivity and somewhat lower but still good specificity. Combining multiple factors in a multivariate model improved the accuracy of identifying high-risk individuals, and nomograms were developed to simplify the use of such models in the clinical environment.

Therefore, every biomarker or combination of biomarkers must be evaluated in light of all available clinical information as well as the patient's personal, family, and societal context. Special precautions, such as increased patient education, more frequent follow-up visits, a wristband indicating the culprit allergens, and, of course, adrenaline auto-injectors as part of the emergency treatment plan, can be implemented for patients at high risk of severe food allergy responses. Such biomarkers can also be used to identify the most severe food allergic patients, for whom oral food challenges can be postponed (or done with a different protocol if necessary) and who may benefit from specific treatment, such as biological and other emerging therapeutic approaches, as well as appropriate psychosocial support, to improve their overall well-being and ability to live with a severe food allergy.