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REVIEW ARTICLE

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Highlights and recent developments in food and drug allergy, and anaphylaxis in EAACI Journals (2017)

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Abstract

This review highlights research advances and important achievements in food allergy, anaphylaxis, and drug allergy that were published in the Journals of the European Academy of Allergy and Clinical Immunology (EAACI) in 2017. Food allergy and anaphylaxis research have continued to rapidly accelerate, with increasing numbers of outstanding developments in 2017. We saw new studies on the mechanisms, diagnosis, prevention of food allergy, and novel food allergens. Drug hypersensitivity, as well as hereditary angioedema, has been highlighted in the present review as the focus of recent developments. The EAACI owns three journals: Allergy, Pediatric Allergy and Immunology (PAI), and Clinical and Translational Allergy (CTA). One of the major goals of the EAACI is to support health promotion in which prevention of allergy and asthma plays a critical role and to disseminate the knowledge of allergy to all stakeholders including the EAACI junior members. This paper summarizes the achievements of 2017 in anaphylaxis, and food and drug allergy.

KEYWORDS

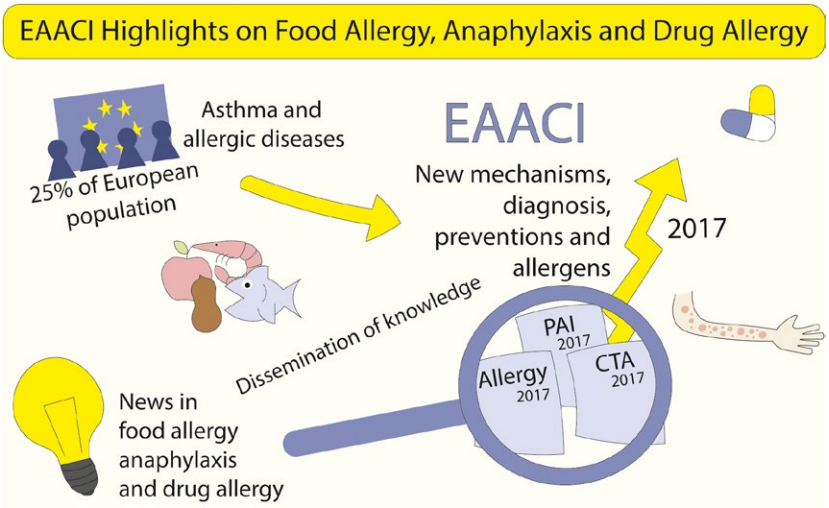
anaphylaxis, drug allergy, food allergy, hypersensitivity

1 | INTRODUCTION

The European Academy of Allergy and Clinical Immunology (EAACI) has three official journals: Allergy, Pediatric Allergy and Immunology (PAI), and Clinical and Translational Allergy (CTA). One of the major goals of the EAACI is to support health promotion in which prevention and control of allergy play a critical role and to disseminate the knowledge of allergy to all stakeholders including the EAACI junior members.

The EAACI Journals have reported on the prediction and primary and secondary prevention of allergic diseases and asthma, and food allergy in 2016.[1,2] This paper summarizes the achievements of 2017 in anaphylaxis, and food and drug allergy.

Abbreviations: Ana o3, *Anacardium occidentale* allergen 3; CD, cluster differentiation; EAACI, European Academy of Allergy and Clinical Immunology; FAQLQ-PF, Food Allergy QoL-Parent Form; ICD, International Classification of Diseases; PR-10, pathogenesis-related protein family 10; Pru p3, *Prunus persica* allergen 3; rChi A, recombinant chitinase A.



2 | ANAPHYLAXIS

Most published studies on anaphylaxis are retrospective or register-based. Data on subsequent diagnostic workup are sparse. A total of 226 patients seen with suspected anaphylaxis at the emergency care setting had a subsequent diagnostic workup at the Allergy Center.[3] The diagnosis was confirmed in 54% of patients. The most common elicitor was drugs (41.1%) followed by venom (27.4%) and foods (20.6%). Atopic diseases were significantly associated only with food-induced anaphylaxis. Cofactors were present in 58.1% and were significantly associated with severe anaphylaxis.

It is important to manage anaphylaxis in primary care.[4] The difficulty in recognizing anaphylaxis is due, in part, to the variability of diagnostic criteria, which in turn leads to a delay in the administration of appropriate treatment, thus increasing the risk of death. The use of validated clinical criteria can facilitate the diagnosis of anaphylaxis. Intramuscular epinephrine (adrenaline) is the medication of choice for the emergency treatment of anaphylaxis. Long-term management of anaphylaxis should include avoidance of triggers, following confirmation by an allergology workup. Special attention should also be paid to cofactors, as these may easily confound the cause of the anaphylaxis. Patients experiencing anaphylaxis should administer epinephrine as soon as possible. Education (including the use of the Internet and social media), written personalized emergency action plans, and self-injectable epinephrine have proven useful for the treatment of further anaphylaxis episodes.

The experience from an inner-city emergency department on anaphylaxis treatment was studied at the emergency department of the St. Pierre Hospital in Brussels.[5] About 0.04% (100/230 878) of all emergency visits in adults presented with anaphylaxis. The majority (64%) of patients were treated according to the EAACI guidelines for the management of anaphylaxis, but only a small minority (9%) received the recommended adrenaline auto-injector for self-administration at discharge.

Successful treatment of anaphylaxis in the community relies on early and correct use of epinephrine auto-injectors. In the Netherlands, pharmacists supply epinephrine auto-injectors to patients and have a crucial role in instructing patients in how and when to use epinephrine auto-injectors. In the Netherlands, food-allergic patients at high risk of anaphylaxis who receive their epinephrine auto-injector from a community pharmacy are often not instructed on how to use an epinephrine auto-injector or receive incorrect instructions.[6] Pharmacists show considerable gaps in knowledge about food allergy and its management. But gaps in anaphylaxis diagnosis and management exist also for physicians as a French study reports.[7]

Clinical practice guidelines are important tools to promote evidence-based clinical care, but not all countries have the capacity or infrastructure to develop these in-house. The EAACI has recently developed guidelines for the prevention, diagnosis, and management of food allergy and the management of anaphylaxis. In order to inform dissemination, adaptation, and implementation of plans, a study attempted to identify countries that have/do not have national guidelines for food allergy and anaphylaxis.[8] Overall, 5 of 193 (3%) countries had at least one guideline for food allergy or anaphylaxis. One (1%) country had a national guideline for the prevention of food allergy, three (2%) countries had a guideline for the diagnosis of food allergy, and three (2%) countries had a guideline for the management of food allergy. Three (2%) countries had an anaphylaxis guideline. There is an overwhelming majority of countries missing any national clinical practice guidelines for food allergy or anaphylaxis.

Anaphylaxis was difficult to be coded under the International Classification of Diseases (ICD)-10. In the ICD-11, the new "Allergic and hypersensitivity conditions" section is under the "Disorders of the Immune system" chapter, and a recent Brazilian retrospective analysis showed the value of the new codification.[9] This study is the first example of how the new "Allergic and hypersensitivity conditions" section of ICD-11 can improve the quality of official vital statistics data and the visibility of an important public health concern. Field testing the new anaphylaxis' classification for the ICD-11 revision confirmed its accuracy in a hospital setting.[10]

3 | MECHANISMS AND DIAGNOSIS OF FOOD ALLERGY

An unusual case of allergy to wheat and milk protein was described in a child with de novo KAT6A mutation.[11]

The performance of oral food challenges, the gold standard diagnosis for food allergy, was harmonized over the last years. However, documentation and interpretation of challenge results, particularly in research settings, are not sufficiently standardized to allow valid comparisons between studies. A new framework for the documentation and interpretation of oral food challenges in population-based and clinical research was performed by follow-up of the EuroPrevall/iFAAM birth cohort.[12] A proposed toolbox sets a standard for improved documentation and harmonized interpretation of double-blind placebo-controlled food challenges has been developed to reduce the influence of subjective judgment of supervising physicians. Severe anaphylaxis requiring intensive care during oral food challenge is not always due to peanuts.[13]

The reaction threshold was examined from 352 children undergoing open food challenges to hen's egg or cow's milk, either fresh or extensively heated into a muffin. There was no significant shift in dose-distribution curves due to the baking process, implying that existing threshold data for these allergens can be applied to allergen risk management, even when these allergens are heat-processed into baked foods.[14]

4 | FOOD ALLERGENS

The advent of molecular allergology revolutionized the approach to the allergic patient in particular to understand cross-reactivities.[15] Molecular allergology and the observation that not every patient has the same allergic profile, even when reacting to the same allergenic source, have originated the concept "one size does not fit all." Sensitization to microarrayed species-specific plant components precedes that of cross-reacting allergens.[16] Sensitization to PR-10 proteins is indicative of distinctive sensitization patterns in adults with a suspected food allergy.[17] Sensitization to PR-10 food proteins could occur without concomitant sensitization to common PR-10 from pollen in a subset of subjects. Less commonly recognized PR-10 proteins appear to be an indication of polysensitization. Negative Act d 8 testing indicates systemic kiwifruit allergy among kiwifruit-sensitized children.[18] IgE levels >2 KU/L to Ana o3 improved prediction of a positive oral food challenge after cashew consumption in comparison with whole cashew extract, but the results are not straightforward and the importance of history taking remains.[19] Prediction of cashew nut allergy in sensitized children for a positive double-blind placebo-controlled food challenge based on gender, specific IgE to Ana o3, and skin prick tests is high.[20] Pru p 3, a marker allergen for lipid transfer protein sensitization, is also a relevant food allergen present in Central Europe.[21] There is a low percentage of clinically relevant pistachio nut and mango co-sensitization in cashew nut-sensitized children.[22] Peanut allergy in children is often associated with allergies to tree nuts and/or legumes. A total of 317 children with peanut allergy evaluated at the Allergy Unit of the Saint Vincent Hospital of Lille were tested. By cluster analysis, three phenotypes of peanut allergy were identified differentiated by their symptoms, threshold level of peanut sensitivity, and differences in cross-allergy to tree nuts and other legumes.[23]

Maize chitinase A is a tough allergenic molecule that was purified from *Pichia pastoris* and tested for its allergenicity.[24] rChiA is a valuable molecule for further studies on structure-allergenicity relationships and as a tool for diagnosing allergies.

Kiwifruit allergy is not a problem in kiwifruit-cultivating regions. A cross-sectional study recruited 20 800 of the randomly selected 6- to 18-year-old urban schoolchildren from Rize city in the eastern Black Sea region of Turkey.[25] Kiwifruit allergy prevalence was low in a city where it is cultivated and highly consumed.

In Korean children, peanut, walnut, and pine nut are the most common food allergens inducing anaphylaxis, and anaphylaxis could occur at remarkably low levels of specific IgE.[26]

Several hydrolyzed cow's milk formulas are available for the avoidance of allergic reactions in cow's milk-allergic children and for prevention of allergy development in high-risk infants. However, infant milk formulas differ regarding their allergenic activity and induction of T-cell and cytokine responses.[27] Partially hydrolyzed whey formula intolerance can be observed in cow's milk-allergic patients.[28] In the Ulm Birth Cohort Study, a new statistical approach shows no association between maternal milk fatty acid composition and childhood wheeze or asthma up to 13 years of age.[29] Although most cow's milk-allergic children outgrow their allergy, the mechanism of the natural development of tolerance remains poorly understood. Differences between IgE and IgG4 binding intensity to cow's milk peptides decreased when the patients became tolerant.[30] This study suggests that the overlap between IgE and IgG4 might be important in natural tolerance acquisition.

In Japan, persistent eczema is related to the development of high-affinity, but not low-affinity, IgE against ovomucoid.[31]

Fruit and vegetable intake is not associated with asthma or chronic rhino-sinusitis in European adults.[32]

5 | PREVENTION AND CONTROL OF FOOD ALLERGY

Better recognition, diagnosis, and management of non-IgE-mediated cow's milk allergy in infancy were proposed in the iMAP guideline, an international interpretation of the MAP (Milk Allergy in Primary Care). Its interpretation was considered in two papers.[33,34]

Food-induced anaphylaxis to a known food allergen in children often occurs despite adult supervision.[35]

Allergen immunotherapy for IgE-mediated food allergy is still a matter of debate and was approached in a systematic review and meta-analysis.[36]

Combining anti-IgE with oral immunotherapy leads to a significant decrease in frequency and severity of allergic reactions[36,37] but is discussed controversially.[38]

Individual healthcare plans for allergic children at school represent an important management strategy for food allergy. They were implemented in schools in France in 2003 to improve the management of allergic children. Lessons from a 2015-2016 school year survey were published.[39] Anaphylaxis remains rare in the school setting, and food allergy is often a suspected cause of reaction. Staff training should be improved.

Risk of anaphylaxis and implications for social activities affect patients' quality of life and psychological well-being. We previously found that young patients reported higher levels of alexithymia (difficulty in recognizing and expressing emotions). Recognizing the specific role of affect regulation in health behaviors may constitute an important step in supporting patients to explore more adaptive strategies.[40] Clinicians should be aware of the implications of insecure attachment for health and illness. They should support patients in limiting social impairment finding a balance between safety and psychologic well-being.[41] Changes in patient quality of life during oral immunotherapy for food allergy have been studied.[42] The FAQLQ-PF improved in some but deteriorated in others during immunotherapy. Patients with impaired quality of life at baseline improved significantly despite the treatment burden. Some patients with better quality of life at baseline might deteriorate during treatment.

6 | DRUG ALLERGY

The diagnosis of ß-lactam allergy is still an important topic. A systematic review and meta-analysis[43] aimed to identify whether inpatient penicillin allergy testing affected clinical outcomes during hospitalization. Twenty-four studies met the inclusion criteria. Inpatient penicillin allergy testing is safe and effective in ruling out penicillin allergy. The rate of negative tests is comparable to outpatient and perioperative data. Patients with a documented penicillin allergy who require penicillin should be tested during hospitalization given its benefit for individual patient outcomes and antibiotic stewardship. Using cutaneous microdialysis in penicillin-allergic patients, positive intracutaneous test induced by penicillin is mediated by histamine and other mediators.[44] The optimal step doses for drug provocation tests to prove ß-lactam hypersensitivity was determined in 182 positive tests.[45] Although the diagnosis of ß-lactam allergy is well-established, a review attempted to find whether oral challenge without skin tests in children with non-severe beta-lactam hypersensitivity could be used.[46] More data are however needed to change the paradigm and make recommendations in pediatric guidelines. Delayed-type beta-lactam hypersensitivity develops in a subset of patients. The cellular immunological processes underlying the drug-specific response are known. However, antigen-specific B lymphocytes and T lymphocytes are activated in piperacillin-hypersensitive patients.[47] In primary health care, suspected penicillin allergy is associated with increased antibiotic use, including second choice antibiotics, and more healthcare use.[48]

Non-steroidal anti-inflammatory agents and aspirin are another common cause of drug reactions. Clinical presentation of non-steroidal anti-inflammatory drugs exacerbated respiratory disease (NERD) is heterogeneous. A cluster analysis of 302 Korean patients with respiratory symptoms found four distinct subtypes with different clinical/biochemical findings and asthma exacerbations.[49] Nonsteroidal anti-inflammatory drugs commonly induce urticaria/angioedema. Loss of hypersensitivity has been reported for IgE-mediated reactions. However, it has not been assessed in non-immunological reactions such as urticaria/angioedema. A 72-month follow-up of 38 patients showed that most became tolerant.[50] Tolerance occurred in patients with less severe initial reaction. The EAACI Drug Interest Group on Challenge performed a multicentric study in 10 centers (310 subjects). In patients with stable ischemic heart disease and histories of non-severe hypersensitivity reactions to non-steroidal anti-inflammatory agents and aspirin, an aspirin challenge is advisable. Patients with an acute coronary syndrome and histories of hypersensitivity reactions to aspirin, especially following doses lower than 100 mg, should directly undergo desensitization.[51]

Hypersensitivity reactions to intravenous iron preparations are well known. With newer preparations, reactions are rare. However, severe reactions may still occur. The mechanisms remain currently unclear. Thirty-one patients with mild to severe reactions were evaluated. Skin prick tests and basophil activation tests were negative in all patients. Eighteen controlled re-administration in 15 patients were performed. Twelve patients tolerated the procedure, including three with a previous grade IV hypersensitivity reaction. Two developed urticaria, and one developed urticaria and dyspnea.[52]

Herbal medicines are largely used in children. Although acute hypersensitivity reactions are generally considered to be rare, more information was needed on the frequency and type of these reactions, especially in children. The WHO global individual case safety report database VigiBase™ in children was used retrospectively between 1968 and 2014. VigiBase™ contained 2646 ICSRs with 14 860 distinct adverse reactions reported in association with herbal medicine in children. Among those, 79 cases with 107 allergy-like reactions were considered, some being anaphylactic shocks.[53]

Immediate moxifloxacin hypersensitivity is still incompletely understood. It may involve mechanisms difficult to capture by traditional CD63-/CD203c-based basophil activation test. Deciphering the complexity of quinolone drug reaction seems important.[54] Alphagal is a possible target of IgE-mediated reactivity to antivenom.[55]

There are similarities and differences between Europe and North America in the approach to the diagnosis of drug hypersensitivity reactions.[56] Although over the years both European and US experts have published statements on general procedures for evaluating drug hypersensitivity reactions, a substantial discordance in their daily management exists. In this review, we highlight both the differences and the similarities between Europe and the United States While a general consensus exists on the importance of skin tests, concordance between Americans and Europeans exists solely regarding their use in immediate reactions and the fact that a confirmation of a presumptive diagnosis by drug provocation tests is often

the only reliable way to establish a diagnosis. Finally, great heterogeneity exists in the application of in vitro tests, which require further study to be well validated.

As a strong inducer of IgE antibodies to substituted ammonium ion epitopes, pholcodine was a postulated cause of allergic anaphylaxis to neuromuscular blocking agents. Three years after the withdrawal of pholcodine in Norway, a significant reduction in IgE sensitization and anaphylaxis reporting was seen. Six years without pholcodine showed that Norwegians are significantly less IgE-sensitized and clinically more tolerant to neuromuscular blocking agents.[57] This important public health measure showed that some drastic actions can be very effective.

7 | HEREDITARY ANGIOEDEMA

The consensus documents published to date on hereditary angioedema with C1 inhibitor deficiency (C1-INH-HAE) have focused on adult patients. Many of the previous recommendations have not been adapted to pediatric patients. An international consensus on the diagnosis and management of pediatric patients with hereditary angioedema with C1 inhibitor deficiency[58] was devised during the 9th C1 Inhibitor Deficiency Workshop in Budapest, 2015. It gave recommendations for early diagnosis in infants and children, information cards held by the patients, and treatment.

The health-related quality of life among children with hereditary angioedema with C1 inhibitor deficiency is shown by a study recruiting children from Israel and Hungary.[59]

Hereditary angioedema with normal C1 inhibitor and specific mutations in the F12 gene (HAE-FXII) exists, and recommendations for treatment were given from a personal experience in 72 patients.[60]

Idiopathic non-histaminergic acquired angioedema (InH-AAE) is a rare disease for which there are no available laboratory parameters to clearly define the disorder. A literature review was performed, and omalizumab was found to induce a complete remission in the 20 patients treated.[61]

8 | POLITICAL AGENDA

Allergic diseases and asthma represent over 25% of the European population and cause a very high burden. Strategies for early diagnosis, prevention, and control need to be anchored on a strong political agenda to implement the results of the research into practice. Two important political activities at the EU Parliament were reported in the journals: A European Summit on the Prevention and Self-Management of Chronic Respiratory Diseases (March 29, 2017)[62] and a European symposium on the awareness of allergy for the promotional campaign (April 26, 2016-April 28, 2016).[63]

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