

Association of sensitization to peanut allergen components with clinical reactivity



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Background:

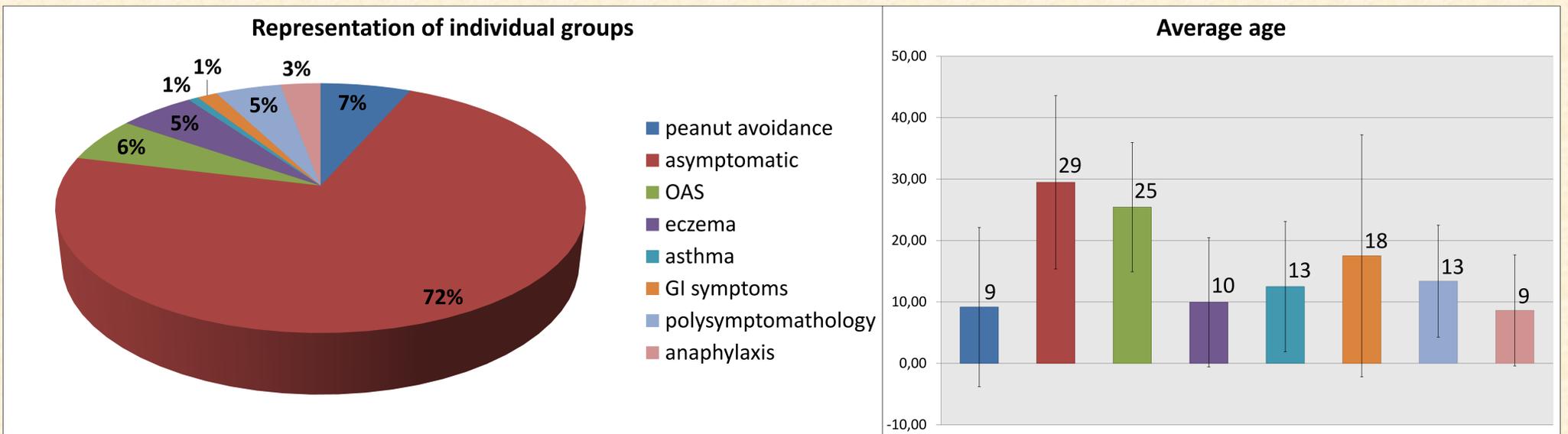
Peanut allergy is one of the most common food allergies which may cause severe reactions. However, many patients sensitized to peanuts tolerate it or develop only mild symptoms as oral allergy syndrome (OAS). Skin prick tests and specific IgE antibodies to peanut extract are not able to differentiate these patients. Presence of specific IgE antibodies to distinct peanut allergen components may draw attention to the potential risk of more severe reactions.

Objective:

Sensitization to peanut allergen components in our group of patients was analysed and related to the type of clinical reactivity.

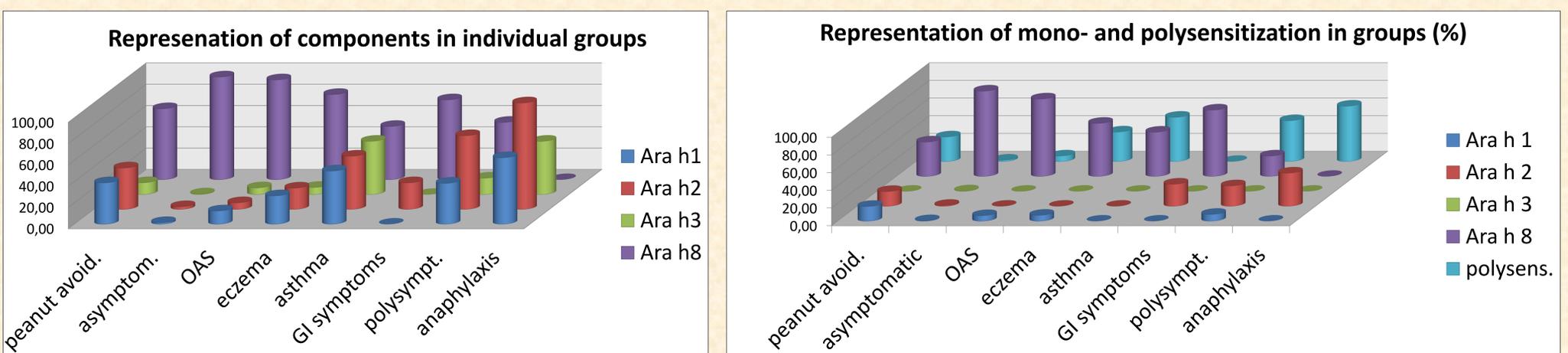
Methods:

We evaluated the group of 274 patients (avg. age 25 yrs, 130 men and 144 women) by means of retrospective analysis. Patients were sensitized to at least one of four investigated peanut allergens: Ara h 1, Ara h 2, Ara h 3 and Ara h 8. Microarray ImmunoCAP ISAC (Phadia, Uppsala, Sweden) for specific IgE detection was used. Patients were divided into 8 groups according to their dominant clinical symptoms. Frequency of sensitization to individual allergen components in each group was assessed.



Results:

- 1) Patients who avoid eating peanuts (18 persons, avg. age 9 yrs): 17% sensitized only to Ara h 1, 17% only to Ara h 2, 38% only to Ara h 8, 28% polysensitized.
- 2) Patients with no symptoms after peanut exposure (198 persons, avg. age 30 yrs): 96% sensitized only to Ara h 8, 1% to Ara h 1, 1% to Ara h 2, 1% to Ara h 3 and 1% polysensitized.
- 3) Patients with oral allergic syndrome (16 persons, avg. age 25 yrs): 88% sensitized only to Ara h 8, 6% to Ara h 1 and 10% polysensitized.
- 4) Patients with worsening of atopic eczema after peanut exposure (15 persons, avg. age 10 yrs): 60% sensitized only to Ara h 8, 7% only to Ara h 1, 12% to Ara h 1 and Ara h 2, remaining 21% to Ara h 8 and one of the left components.
- 5) Asthma attack after peanut exposure (2 persons, avg. age 13 yrs): 50% sensitized only to Ara h 8, 50% polysensitized.
- 6) Patients with gastrointestinal symptoms after peanut exposure (4 persons, avg. age 18 yrs): 25% sensitized only to Ara h 2, 75% to Ara h 8.
- 7) Patients with polysymptomatology after peanut exposure (13 persons, avg. age 13 yrs): 8% sensitized to Ara h 1, 23% sensitized to Ara h 2, 23% to Ara h 8 and 46% polysensitized.
- 8) Patients with anaphylaxis (8 persons, avg. age 9 yrs): all of these patients were sensitized to Ara h 2, of which 12% in addition to Ara h 1, 50% in addition to Ara h 1 and 3. No sensitization to Ara h 8 was seen in this group.



Discussion:

Severe symptoms of peanut allergy were observed more frequently in children. Oral allergy syndrome was present both in children and adults. Asymptomatic patients were mostly adults and 99% of them were co-sensitized to birch pollen (Bet v 1), so that cross-reactivity in the frame of PR-10 allergen family is the reason for their Ara h 8 sensitization. The same reason for sensitization is present in a part of patients avoiding peanuts. The sensitization to other allergen components may be due to traces of peanut allergen contained in various foods or other cross-sensitizations. Ara h 2 seems to be the most significant component in relation to severe clinical symptoms in our study. Polysensitized patients are threatened by the most severe allergic reaction. The importance of Ara h 2 has been suggested in studies from other Central and Northern European countries. However, peanut allergy has a different molecular basis in different geographic areas, probably because of different pollen exposures and dietary customs.

Conclusion:

We confirmed the findings suggesting that allergy to seed storage proteins (especially Ara h 2) and polysensitization is frequently responsible for the severe allergy symptoms. The sensitization to Ara h 2 or polysensitization may be used as an important predictor of an acute allergic reaction to peanut. Sensitization to Ara h 8 is frequently associated with mild symptoms as OAS and may be present quite frequently also in asymptomatic patients.

References: 1) Nicolaou N, Murray C, Belgrave D, Poorafshar M, Simpson A, Custovic A. Quantification of sIgE to whole peanut extract and peanut components in prediction of peanut allergy. *Journal of Allergy and Clinical Immunology* 2011;127:684-685. 2) Vereda A, van Hage M, Ahlstedt S, Ibanez MD, Cuesta-Herranz J, van Odijk J, Wickman M, Sampson HA. Peanut allergy: Clinical and immunologic differences among patients from 3 different geographic regions. *Journal of Allergy and Clinical Immunology* 2011;127:603-607. 3) Vadas P, Wai Y, Burks W, Perelman B. Detection of Peanut Allergens in Breast Milk of Lactating Women. *The Journal of the American Medical Association* 2001;285:1746-1748. 4) Wesley Burks. Peanut allergy. *Lancet* 2008;371:1538-46. 5) Eller E, Bindsley-Jensen C. Clinical value of component-resolved diagnostics in peanut-allergic patient. *Allergy* 2012; DOI: 10.1111/all.12075

The authors of this presentation declares no conflicts of interest.