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ORAL ALLERGY SYNDROME AND DENTAL ALLERGOLOGY

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SUMMARY:

The reactivity to food proteins is specific, but in certain patients cross reactions are observed between food proteins of plant or animal origin and non-food ones. The most common clinical manifestation of this cross reactivity is oral allergy syndrome (OAS). It develops mainly in patients with pollen allergy after fresh fruit and vegetables consumption. The symptoms are oro-pharyngeal pruritus, papules and vesicles which resemble mouth ulcers on the labial mucosa and labial, palatal and lingual swelling. There may also be a sensation of pharyngeal swelling. The symptoms appear very quickly and may be followed by urticaria or contiguous facial erythema. [1] The progress of this syndrome is due to homology of structural proteins in foods of plant origin and in pollens.

Key words: oral allergy syndrome, atopic allergens, food allergy

INTRODUCTION:

Every single foodstuff can cause hypersensitivity reaction. Major food allergens are water-soluble thermostable glycoproteins that are hardly destructed by acids and proteases and have molecular weight of 10-60 kDa. Researches show that several foods have higher allergenic potential and induce more than 90% of allergic reactions in humans. In small children these foodstuffs are milk, eggs, soy bean, peanuts and wheat, and in adults – peanuts, tree nuts, fish and seafood.[2, 3, 4]

The classification of Gell and Coombs is traditionally used for the description of different allergic reactions to foods although these reactions are more complex. The most detailed studies are for food allergy reactions as a manifestation of type I hypersensitivity (IgE-mediated immune response). In cases of insufficiently developed oral tolerance or break-through of its action, increased production of IgE is observed in certain individuals. These IgE antibodies bind to high-affinity receptor FceRI in mast cells and basophils and low-affinity receptor FceRII in macrophages, monocytes, lymphocytes, eosinophils and platelets. Binding of antigens to IgE already bound by the FceRI on mast cells causes cross-linking of the bound IgE and the aggregation of the underlying FceRI, leading to the degranulation and the release of mediators from the cells (histamine, prostaglandins and leukotrienes). The mediators cause vasodilation, smooth muscles contraction and mucus secretion that determine the specific symptoms of immediate hypersensitivity. [2] Basophils, upon the cross-linking of their surface IgE by antigens, release type 2 cytokines like interleukin-4 (IL-4) and interleukin-13 (IL-13) and other inflammatory mediators, inducing the late phase of IgE-mediated reaction.

IgE-mediated hypersensitivity to foodstuffs causes numerous symptoms - anaphylaxis, urticaria, angioedema, oral and gastro-intestinal symptoms, etc. The oral allergy syndrome is of great interest for us in this study as a manifestation of cross reactivity between food proteins of plant or animal origin and non-food ones. It develops mainly in patients with pollen allergy after fresh fruit and vegetables consumption. For example, the pollen of Betulaceae family plants (birch, alder, hazel) contains proteins (Bet v1, Bet v2, etc.) that are also found in apples, raw potatoes, carrots, celery, hazelnuts. [5] Similar homology of structural proteins is a base for cross reactivity between proteins in mugwort pollen (Art v1) and in celery, apple, peanuts and kiwi; between ragweed pollen and water melon, melon and banana. [6] Such homology is also found between proteins of latex and banana, avocado, kiwi, chestnut and papaya. [7]

The purpose of this research is to investigate the manifestations of food allergy in the oral cavity; to clarify the role of food allergens in the development of oral allergy syndrome and to study its relation with cross reactivity to major atopic allergens.

MATERIALS AND METHODS:

The target group comprises of patients with oral complaints. They were taken allergic history first - with

questions about existing allergic disease, pollen allergy, atopy and food allergy. They were also asked about the foodstuffs that caused hypersensitivity to them, about the induced symptoms and the time these symptoms appeared. Special attention was paid to oral allergy syndrome - patients had to describe the manifestations in their specific cases.

Then different skin tests were carried out - prick tests with food and atopic allergens and patch tests with dental allergens.

We used the following food and atopic allergens - pork, egg white, tomato, potato, carrot, hazelnut, chicken, milk chocolate, peanut, soy bean, Dermatophagoides pteronyssinus, mugwort, ragweed, birch, Alternaria. Positive reaction was accomplished with Histamine HCl solution. The results were read on the 20-th minute and evaluated according to the size of the papule (Table 1).

Table 1: Evaluation of prick test results.

Reaction	Size of papule and erythema (mm)
Negative (-)	De≤3 mm
Weakly positive (+)	Dp=3-5 mm; De>Dp
Moderately positive (++)	Dp=5-10 mm; De>Dp
Strongly positive (+++)	Dp=10-15 mm; De>Dp
Extreme positive (++++)	Dp>15 mm; De>Dp

We chose 10 dental allergens (from the series of Chemotechnique Diagnostics) for patch testing – MMA, TEGDMA, BIS-GMA, Potassium dichromate, Mercury, Cobalt(II) chloride, Goldsodiumthiosulfate, Nicklesulfate, Eugenol, Epoxy resin. The results were read on the 72-nd hour and evaluated according to the recommendations of the International Contact Dermatitis Research Group (Table 2).

Table 2: Evaluation of patch test results.

-	**Negative reaction**
?	Doubtful reaction
+	Weak reaction (non-vesicular)
++	Strong reaction (oedematous or vesicular)
+++	Extreme reaction (ulcerative or bullous)
IR	Irritant reaction

Specific IgE assays were used to confirm the IgE-dependent sensitation found in skin testing.

The statistic processing of received data was accomplished with SPSS 15.0 software program and the following methods - descriptive analysis, analysis of variance, cross tabulation, chi-squared test and Fisher's exact test.

RESULTS:

19 patients are examined completely till now – 4 of them male and 15 female. The female patients significantly dominate over the male ones – 78,9% against 21,1% - as the level of significance p=0,012. The age of the patients ranges between 17 and 75 years and the average age is 40,8±19 years.

16 of patients (84,2%) declare that they suffer from allergic disease, so they dominate in the group and the level of significance is great (p=0,003). 63,2% of patients report for symptoms, localized in the oral cavity, e.g. oral allergy syndrome. In patients with allergic disease, pollen or food allergy the relative part of these with OAS is bigger, compared to the part without OAS. But, the difference is statistically significant for the group with allergic disease only (p=0,036). (Fig. 1)

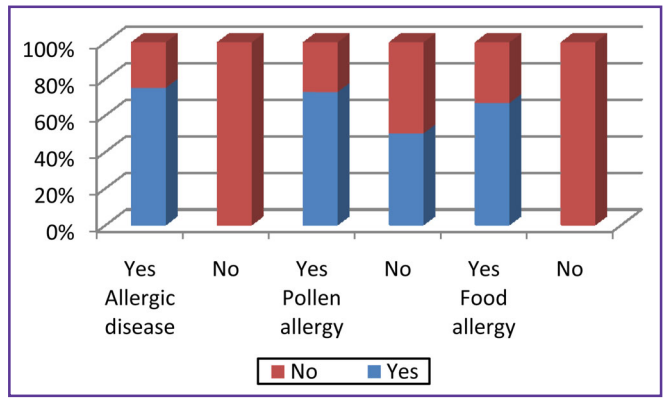

Fig. 1: Distribution of patients with OAS according to history of allergy.

18 of patients (94,7%) report for food allergy manifestations. In 22,2% of them the symptoms were in the childhood only; in 27,8% of cases the symptoms started in the childhood and continue till nowadays and in the rest 50% the symptoms appeared in the present.

The most common manifestations of food allergy are OAS, rash, facial erythema and facial swelling (all symptoms are given in Fig. 2). Most frequently these symptoms are caused by consumption of fruits, eggs and nuts (all etiological factors are given in Fig. 3).

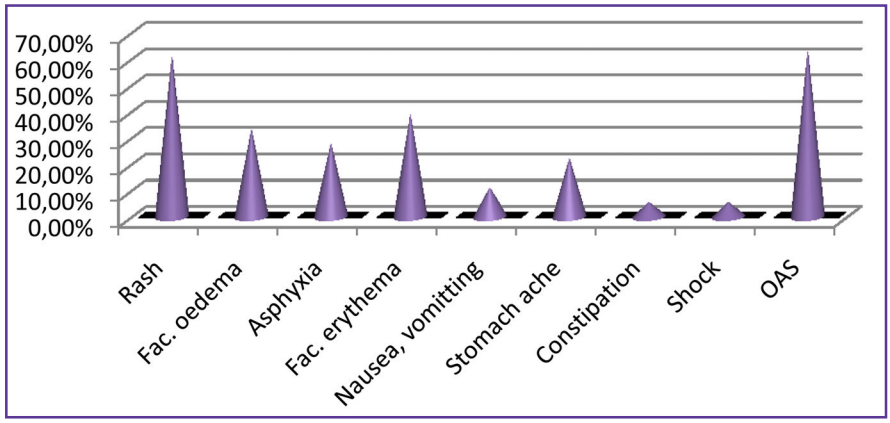

Fig. 2: Symptoms of food allergy.

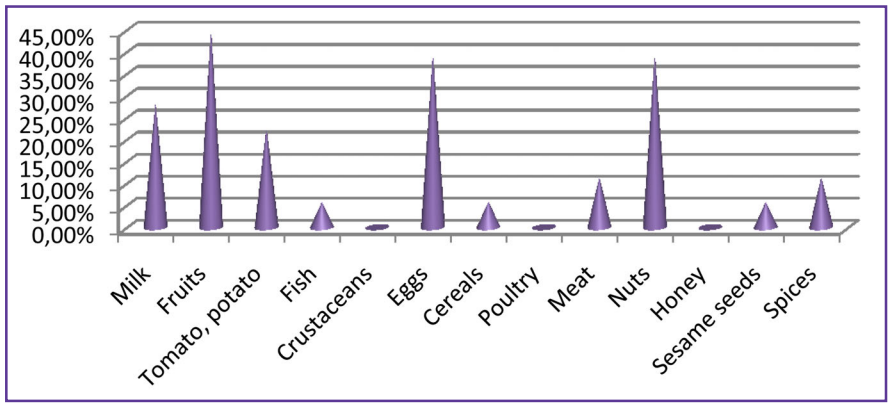

Fig. 3: Food allergy etiology.

19 patients are patch-tested - 3 of them male and 16 female (15,8% against 84,2%). The average age of these patients is 38,59±17,68 years.

78,95% of the patients show sensibilization to one or more dental allergens (Table 3). Strong positive reactions (++) are read in 3 patients with sensibilization to Ni, BIS-GMA and Hg, and very strong positive reactions (+++) - in 3 patients with sensibilization to Ni. Comparison of data displays high coincidence between positive tests to dental allergens and history of allergic disease in investigated patients. These results are presented in Table 4.

MMA	TEGDMA	BIS-GMA	Cr	Hg	Co	Au	Ni	Eugenol	Epoxy resin
3 (15,8%)	1 (5,3%)	1 (5,3%)	6 (31,6%)	3 (15,8%)	7 (36,8%)	4 (21,1%)	7 (36,8%)	3 (15,8%)	2 (10,5%)

Table 3: Frequency of positive patch tests to dental allergens.

	Allergy (n=16)	Atopy (n=4)	PA (n=10)	OAS (n=12)	FA (n=16)
Patients with positive patch test	75,0%	75,0%	70,0%	66,7%	75,0%

Table 4: Frequency of positive patch tests in patients with history of allergy.

32 patients are prick-tested with food and atopic allergens. The most frequent strong positive reactions are to D. pteronyssinus, birch and hazelnut (Table 5).

Table 5: Frequency of positive prick tests to food and atopic allergens.

Allergen (tested patients)	Positive reaction (+++)
Hazelnut (n=25)	8,0%
Milk chocolate (n=24)	4,2%
Soy bean (n=29)	3,4%
D. pteronyssinus (n=32)	15,6%
Grasses (n=18)	5,6%
Birch (n=14)	14,3%

DISCUSSION:

OAS is a manifestation of type II food allergy, that's why the average age of investigated patients is so high and the majority of these patients exhibit food allergy symptoms just in the present.

According to data, we've collected till now, the most common symptoms of OAS are oral pruritus, mucosal erythema and lingual swelling. In cases with pruritus only, we have no evidence for immunological origin of the reaction.

68,8% of investigated patients with food allergy develop OAS - the fact is based probably on the requirements for the target group of the research.

OAS could be a consequence of pollen-food syndrome - in our research 68,8% of investigated patients have also pollen allergy.

Although 78,95% of investigated patients show sensibilization to one or more dental allergens, this is just a pilot study and the information is insufficient to draw a conclusion. This refers also to the results of the prick tests with food and atopic allergens.

CONCLUSION:

The proper diagnosis of oral manifestations of food allergy and the differentiation from other oral symptoms will optimize the treatment and prevention approach.

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