



A Case Report of Allergy to Peach's Lipid Transfer Protein

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case study

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ABSTRACT

Peach belongs to the Rosaceae family. One of the particularities of this allergy is the difference in clinical reactivity according to geography. We report a 4-year 8-month-old boy with well-controlled asthma and no symptoms of allergic rhinitis, that at the age of 1 year, 15 minutes after the ingestion of a fruit compote, presented urticaria with respiratory discomfort. At the age of 3 years, following ingestion of fruit salad, the child presented a few minutes later, a similar episode but of greater intensity. At the age of 4, following contact with a fruit basket, the child immediately presented a generalized reaction more severe and intense than the previous ones. Investigation of allergies to aerial allergens was negative. Skin tests for cooked and raw peach were positive for the peel and pulp. Specific-IgE was positive for Pru p 3 (5.7 KIU/l) and negative for Pru p 1. LTPs are particularly stable and resistant to proteolysis and heat. Management consisted of avoidance of peach, education of the child and family, and prescription of an emergency kit including self-injectable adrenaline. Our observation highlights the strong association between Pru p 3: peach's Lipid Transfer Protein (LTP) positivity and the severity of allergy symptoms. We consider this case as a primary sensitization to peach's LTP.

Keywords: Lipid transfer protein ; peach; Pru p 3.

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1. INTRODUCTION

Peach allergy seems to be increasing. The major allergen is a lipid transfer protein quite common in the plant kingdom and is involved in the defense of plants against external aggressions. Our work aims to study the epidemiological, allergenic, clinical, and therapeutic characteristics of peach allergy.

2. CASE REPORT

We report an observation collected in the pediatric department of the children's hospital of Rabat Morocco. The male patient was aged 4 years and 8 months when first seen at our consultation for management of urticaria. At the age of 1 year, it was related to the appearance of acute dyspnea with urticaria 15 minutes after ingestion of a fruit compote. The patient then presented a respiratory difficulty treated by antihistamines and steroids; however, the parents could not associate any cause. At the age of 3 years, he presented: 2nd episode of dyspnea with generalized urticaria in January a few minutes after eating a fruit salad. The child received this time B2 mimetic, antihistamine, and corticosteroid therapy, with good evolution.

At the age of 4 years: the patient presented laryngeal dyspnea with diffuse urticaria, just 5 minutes after the parents returned from the supermarket, the parents having deposited a basket of fruit next to the child. The last two allergic episodes were more severe than the first. Clinical examination was unremarkable : eupneic child no auscultation rales no skin signs, no delay in weight and height. The patient also had controlled asthma under fluticasone 250 µg/day. The family story of atopy revealed the mother with allergic conjunctivitis, a brother, and a sister with psoriasis. The patient further ingested without symptoms banana, oranges, apricot, cherries, apples, and melon. After investigation, we noticed that peach was present at the fruit salad and in the fruit basket, so, considering the clinical history, the medical orientation was an exclusion diet for peaches which resulted in absence of symptoms. Regarding the laboratory workup: Total IgE: 15.60 IU/ml; Specific IgE to *Dermatophagoides pteronyssinus* < 0.10 kUI/L; Specific IgE to *Dermatophagoides farinae* < 0.10 kUI/L. Prick test to cooked and raw peach came back positive to pulp and skin. An oral challenge test was not performed due to the severe reactions. Considering the clinical

symptomatology and the positive prick test to cooked and raw peach, we diagnosed peach allergy. To determine the severity profile we researched specific-IgE to *Pru p 3* (5,7 kUI/L) and specific-IgE to *Pru p 1* (<0.1 kUI/L). Treatment is essentially preventive, consisting of avoidance of peaches, patient education, and prescription of an emergency kit including self-injectable adrenaline.

3. DISCUSSION

The peach (*Prunus persica*) belongs to the Rosaceae family (apples, cherries, pears, apricots, raspberries, strawberries, hazelnuts, and almonds [1] .There is a difference in clinical reactivity based on geography. Sensitization to the fruit may be primary (as seen more commonly in the Mediterranean basin) or secondary (as seen predominantly in northern Europe). In Spain, peach is the main fruit responsible for food allergy (5.1%) [2] . In Israel, peach is the leading food allergy with 75% of positive oral challenge tests in children over 10 years old [3]. In Northern Europe, sensitization to Rosaceae requires prior sensitization to birch pollen (apple-birch syndrome) [4]. On the other hand, in the Mediterranean region, a severe systemic syndrome due to allergy to LTP is found. In Morocco, the main peach production areas are Meknes, Ifrane, Khenifra, Boulmane, Gharb, and Beni-Mellal. In the case of primary sensitization to the fruit, without the pollen allergy, the allergens involved are defense proteins of the LTP family. Clinically, the symptoms can range from oral syndrome to anaphylaxis with a systemic syndrome of nausea, vomiting, diarrhea, generalized urticaria, and even asthma [5].The oral allergy syndrome is characterized by oropharyngeal symptoms appearing less than a few minutes after ingestion of plant foods, often in a patient allergic to pollens, which is not the case for our patient. Clinically, children with a high atopic background are often the most likely to develop severe food allergies. Co-factors influence the reproducibility and severity of reactions. Exercise or physical exertion, taking antacids also increases intestinal permeability, the speed of passage of allergens and their quantity arriving in the systemic circulation can cause severe reactions. Therefore, their potential involvement must always be considered. Skin tests are performed with native foods or commercial extracts and are read after 20 minutes with a sensitivity of 95% [6]. A skin test with fresh fruits and vegetables is more sensitive for diagnosis than the use of

commercial food [7]. Skin tests should be performed with the pulp and peel of the fruit because they have a different allergenic profile. As for the oral challenge test, it remains difficult to perform. A recently proposed value of 2.69 KUI/L has been reported for recombinant *Pru p 3* specific-IgE antibody values that identify peach allergic patients with a higher risk of severe symptoms after peach ingestion [8]. The main allergens of the peaches are *Pru p 1* (PR10 family); *Pru p 3* (LTP family); *Pru p 4* (profilin family) and *Pru p 7* (peamaclein). *Pru p 1* is heat-sensitive plant defense proteins known to be mostly limited to produce oral allergy syndrome [8]. Profilins have been identified as minor allergens and would explain in part the possibility of cross-reactions with grass pollens. In 171 patients with a confirmed allergy to birch, 6% had a cross-reaction to peach [9]. Lipid transfer proteins are found in a great number of plants. They can cause severe allergic reactions due to their protein structure and heat-resistant nature [10] During the LTP allergy syndrome, the sensitization is mainly done by food or by the respiratory route. The LPT is involved in the defense of plants against external aggression. The skin of peaches contains seven times more of it than the pulp and is therefore much more allergenic [11]. The allergenicity of LTPs depends on the existence of IgE-binding epitopes distributed on the surface of these proteins. There are three epitopes of LTPs that are well exposed on the surface of allergens. Epitope 2 (E 2 *Pru p 3*), which has the same sequence in all rosaceous LTPs, is responsible for many cross-reactions with cherry and apple [12]. However, allergenicity is only slightly reduced after peeling and heating. Indeed, LTPs have a helical helix stabilized by 4 desulfurized bridges which allows an extreme resistance to proteolysis at extreme temperatures and PH modification and ultrafiltration [13]. French researchers have observed that organic fruits produce a higher amount of defense proteins of the LTP family because the fruits are grown without pesticides, which increases their production of defense proteins to protect themselves from diseases. Therefore, eating organic will not protect people with fruit allergies. *Pru p 3* plays a role in sensitization to other LTPs belonging to the Rosaceae family [10]. About 4.5% of peach extract positive patients were negative for *Pru p 3*, *Pru p 4*, and *Pru p 1*. This result was probably determined by sensitization to other molecules contained in peach such as Peamaclein [10]. *Pru p 7* or peamaclein is an antimicrobial peptide of the gibberellin regulated

protein family. They are found both in the skin and in the pulp of the fruit and are involved in the defense of plants and fruits [10]. Peamaclein is a relatively small molecule compared to *Pru p 3* which has a higher molecular weight. It is heat resistant and resists acid digestion, not digested by trypsin. Sensitization to Peamaclein is clinically associated with severe symptoms [10]. There are 3 molecular profiles of peach: Patients with allergy to *Pru p 3* alone (without sensitization do *Pru p 1* and *Pru p 4*) often have severe reactions up to anaphylaxis. Patients with allergies to *Pru p 3* and *Pru p 1* usually are protected against severe systemic reactions. Allergy to *Pru p 1* alone usually renders only local reactions [10].

4. CONCLUSION

The case reported corroborates the association between the specific-IgE *Pru p 3* quantification and the severity of allergy symptoms. This observation highlights not only the importance of a well-done anamnesis in food allergy but also the importance of determining the specific-IgE antigenic profile to establish the future risk of sensitization and development of allergy.

CONSENT

As per international standards, informed and written parental consent was collected and preserved by the authors.

ETHICAL APPROVAL

As per international standards, written ethical permission was collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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