ARQUIVOS DE ASMA, ALERGIA E IMUNOLOGIA
© 2023 ASBAI

The forbidden fruit, a case of oral allergy

O fruto proibido, um caso de alergia oral

Inês Falcão[1], Leonor Cunha[1]

ABSTRACT

In adolescents and adults, 30% to 60% of food allergies are associated with pollen allergy and are included in the pollen-food syndrome (PFS). This syndrome is characterized by allergic symptoms elicited by the ingestion of fresh fruits or vegetables in patients with seasonal allergic rhinitis/rhinoconjunctivitis. The authors present the clinical case of an adolescent who, after primary sensitization to grass and olive tree pollens, subsequently manifested by cross-reactivity symptoms of oral allergy with the ingestion of fresh fruit. After diagnostic workup with the Immuno-Solid-phase Allergen Chip (ISAC) assay, profilins were identified as the proteins responsible for the cross-reactivity.

Keywords: Syndrome, pollen, fruits, adolescent, profilin.

RESUMO

Trinta a 60% das alergias alimentares em adolescentes e adultos são associadas à alergia ao pólen e estão incluídas na síndrome pólen-frutas (SPF). Esta síndrome é caracterizada por sintomas alérgicos provocados pela ingestão de frutas ou vegetais frescos em pacientes com rinite/rinoconjuntivite alérgica sazonal. Os autores apresentam o caso clínico de um adolescente que após sensibilização primária através de pólens de gramíneas e oliveira manifestou posteriormente, por reatividade cruzada, sintomas de alergia oral com a ingestão de frutas frescas. Após recurso ao método de diagnóstico *Immuno-Solid-Phase Allergen Chip* (ISAC) verificou-se que as profilinas foram as proteínas responsáveis pela reatividade cruzada.

Descritores: Síndrome, pólen, frutas, adolescente, profilina.

Introduction

Allergic rhinitis (AR) is common in Brazil, with a mean prevalence of 29.6% among adolescents and 25.7% among children according to the International Study on Asthma and Allergies in Childhood.[1] Another study demonstrated that the incidence of seasonal allergic rhinitis (SAR) is 5% in children aged 4 years, 8.5% in children aged 6-7 years, and 19% in preadolescents.[2]

Thirty to 60% of food allergies in adolescents and adults are associated with pollen allergy and are included in the pollen-food allergy syndrome (PFAS).[3] PFAS prevalence is significantly higher in Northern Europe because of birch pollen allergy. Osterballe et al.[4] estimated that 40% to 50% of patients with birch allergy had PFAS.[3]

PFAS is characterized by allergic symptoms that appear after the ingestion of fresh fruits or vegetables in patients with SAR/rhinoconjunctivitis.[5] It is more common in adolescents and young adults and is more frequently characterized by the appearance of symptoms in the oral cavity after immediate contact with the food, including oral pruritus, angioedema of the lips, tongue, palate or oropharynx, laryngeal tightness, and paresthesia. This group of typical symptoms has been named oral allergy syndrome (OAS).[3] However, in more severe cases (3%), there

1. Centro Hospitalar e Universitário do Porto, Serviço de Imunoalergologia - Porto, Porto, Portugal.

Submitted Mar 11 2022, accepted Jul 03 2023.

Arq Asma Alerg Imunol. 2023;7(1):127-9.

http://dx.doi.org/10.5935/2526-5393.20230016-en

may be systemic involvement with gastrointestinal, cardiovascular, and respiratory tract symptoms. Thus, the terms PFAS and OAS should not be used interchangeably.[6]

This phenomenon occurs when pollen proteins cross-react with homologous food proteins. That is, proteins found in pollens and fruits and vegetables have a similar amino acid sequence that can promote specific IgE cross-reactivity, causing allergic symptoms. These proteins are called panallergens. Pathogenesis-related protein type 10 (PR-10), nonspecific lipid transfer proteins (LTPs), and profilins are the main panallergens responsible for PFAS and the best characterized to date.[3,7] The type of protein involved in PFAS is one of the factors that can influence symptom severity.[3]

The estimated prevalence of PFAS in patients with pollen allergy is 47% to 70%.[8] Clinical history is the cornerstone of PFAS diagnosis, which can be challenging, as it is not always easy to differentiate between primary sensitization, cross-reactivity, and co-sensitization.[9] The molecules that cause PFAS are usually labile and degraded by heat and digestive enzymes.[10]

Profilins are proteins that cause sensitization to tree and grass pollen and are mostly found in fruits and vegetables of the *Rosaceae* family, such as apple, peach, pear, banana, mango, tomato, carrot, among others.[11] Patients sensitized to this group of proteins typically have mild reactions, such as OAS, and can tolerate the fruits if they are cooked.[10]

Clinical case

We present the case of a 14-year-old boy diagnosed with AR to grass and olive tree pollen at age 9 by an allergist/immunologist, who later developed PFAS. The patient initially reported mild oral itching after eating apples and pears, with worsening symptoms over the years, namely angioedema of the lips and tongue after eating bananas, watermelons, melons, and peaches. He denied any association with cofactors such as taking antibiotics, nonsteroidal anti-inflammatory drugs, proton pump inhibitors, physical exercise, alcohol consumption, or others. He also denied experiencing symptoms when eating other foods and had no relevant family history of allergic diseases.

Skin prick tests were performed with histamine 7 mm and saline solution 0 mm and were positive for apple 7 mm, pear 6 mm, banana 5 mm, melon 4.5 mm, and peach 6 mm extracts. The prick by prick test was positive for apple, banana, melon, and pear and peach peel and pulp extracts.

Because the patient was polysensitized and was under multiple dietary restrictions, an ImmunoCAP™ ISAC assay was performed to elucidate the sensitization profile and characterize the patient's primary sensitization. The ISAC assay revealed high sensitivity to grass pollen due to species-specific allergens (Phl p 1, p 2, p 5, p 6, p 11) and high positivity for cross-reactive components, namely profilins (Phl p 12, Bet v 2, Hev b 8, Mer a 1). Because the patient was only sensitized to profilins, an oral food challenge with cooked apple was conducted, which was negative.

The patient was prescribed oral antihistamines and intranasal corticosteroids for use during the pollen season. In case of accidental ingestion of any of the previously mentioned foods, the patient was also prescribed emergency drugs including antihistamines, systemic corticosteroids, and an epinephrine auto-injector for anaphylaxis. He was advised to avoid eating pears, bananas, watermelons, peaches, and melons.

Conclusion

In this clinical case, primary sensitization occurred via the respiratory route through grass and olive tree pollen. The patient later developed OAS to fresh fruit as a result of cross-reactivity due to sensitization to profilins. Since these proteins are unstable when digested by pepsin and are degraded by heat, we expected the patient to tolerate cooked fruit, as shown in the oral food challenge with cooked apple.[10]

Performing the ISAC assay was crucial for ruling out the involvement of proteins such as PR-10 and LTPs, which are associated with more severe reactions. This allowed us to describe the profile of the polysensitized patient and a achieve a possible prognosis of reaction severity.

In clinical practice, if a patient has sensitization to grass and/or tree pollen, it should always be investigated whether there is a history of symptoms related to fruit or vegetable ingestion. Patients should be informed about the characteristics and distribution of the allergens to which they are sensitized.

References

1. Ibiapina CC, Sarinho ESC, Camargos PAM, Andrade CR, Cruz Filho AAS. Rinite alérgica: aspectos epidemiológicos, diagnósticos e terapêuticos. J bras pneumol. 2008;34(4). doi: https://doi.org/10.1590/S1806-37132008000400008 .
2. Ludman S, Jafari-Mamaghani M, Ebling R, Fox AT, Lack G, Du Toit G. Pollen food syndrome amongst children with seasonal allergic rhinitis attending allergy clinic. Pediatr Allergy Immunol. 2016;27(2):134-40.
3. Poncet P, Sénéchal H, Charpin D. Update on pollen-food allergy syndrome. Expert Rev Clin Immunol. 2020 Jun;16(6):561-78. doi: 10.1080/1744666X.2020.1774366.
4. Osterballe M, Hansen TK, Mortz CG, Høst A, Bindslev-Jensen C. The prevalence of food hypersensitivity in an unselected population of children and adults. Pediatr Allergy Immunol. 2005;16(7):567-73. doi: 10.1111/j.1399-3038.2005.00251.x.
5. Werfel T, Asero R, Ballmer-Weber BK, Beyer K, Enrique E, Knulst AC, et al. Position paper of the EAACI: food allergy due to immunological cross-reactions with common inhalant allergens. Allergy. 2015 Sep;70(9):1079-90. doi: 10.1111/all.12666.
6. Ma S, Sicherer SH, Nowak-Wegrzyn A. A survey on the management of pollen-food allergy syndrome in allergy practices. J Allergy Clin Immunol. 2003;112(4):784-8. doi: 10.1016/s0091-6749(03)02008-6.
7. Radauer C, Bublin M, Wagner S, Mari A, Breiteneder H. Allergens are distributed into few protein families and possess a restricted number of biochemical functions. J Allergy Clin Immunol. 2008;121(4):847-52.e7. doi: 10.1016/j.jaci.2008.01.025.
8. Ta V, Scott D, Chin W, Wineinger N, Kelso J, White A. Differential skin test reactivity to pollens in pollen food allergy syndrome versus allergic rhinitis. Allergy and Asthma Proceedings. 2015;36:379-85.
9. Skypala IJ, Calderon MA, Leeds AR, Emery P, Till SJ, Durham SR. Development and validation of a structured questionnaire for the diagnosis of oral allergy syndrome in subjects with seasonal allergic rhinitis during the UK birch pollen season. Clin Exp Allergy. 2011;41(7):1001-11. doi: 10.1111/j.1365-2222.2011.03759.x.
10. Jankiewicz A, Aulepp H, Baltes W, Bögl KW, Dehne LI, Zuberbier T, et al. Allergic sensitization to native and heated celery root in pollen-sensitive patients investigated by skin test and IgE binding. Int Arch Allergy Immunol. 1996;111(3):268-78. doi: 10.1159/000237377.
11. Hauser M, Roulias A, Ferreira F, Egger M. Panallergens and their impact on the allergic patient. Allergy Asthma Clin Immunol. 2010;6(1):1. doi: 10.1186/1710-1492-6-1.

No conflicts of interest declared concerning the publication of this article.

Corresponding author:
Inês Falcão
E-mail: inesffalcao@hotmail.com