

Anaphylaxis to sunflower seed

Anafilaxia à semente de girassol

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ABSTRACT

Sunflower seed (*Helianthus annuus*) sensitisation occurs to storage proteins (2S albumins) and to lipid transfer proteins (LTPs), but it rarely induces anaphylaxis. We report the case of a 32-year-old female with allergic rhinoconjunctivitis and three anaphylactic reactions within minutes after eating sunflower seed. The first episodes occurred after eating sunflower seeds as a snack, and the last after eating chips fried in sunflower oil. Skin-prick-tests were positive to sunflower seed, specific IgE was 79 kU/L and ImmunoCAP ISAC[®] revealed sensitisation to cross-reactive LTPs (nPru p 3, nArt v3, rPar j 2). Sunflower seed extract inhibited (60%) IgE binding to nArt v3 and nPru p 3, with no inhibition to the other allergens identified. Epinephrine autoinjector device was prescribed and strict avoidance of sunflower seed and foods cooked in sunflower oil was advised.

Keywords: anaphylaxis, food allergy, lipid transfer proteins, sunflower oil, sunflower seed.

RESUMO

A sensibilização à semente de girassol (*Helianthus annuus*) tem sido descrita a proteínas de reserva (albuminas 2S) e a proteínas de transferência de lípidos (LTPs), mas reacções anafiláticas associadas à ingestão são raras. Reportamos o caso de uma mulher de 32 anos, com rinoconjuntivite alérgica e três episódios de anafilaxia minutos após ingestão de semente de girassol. Os primeiros episódios ocorreram após ingestão de sementes secas e o último após ingestão de batatas fritas confeccionadas em óleo de girassol. Realizou testes cutâneos por picada positivos para semente de girassol, com IgE específica de 79 kU/L e ImmunoCAP ISAC[®] positivo para LTPs (nPru p 3, nArt v 3, rPar j 2). O extracto de semente de girassol inibiu 60% da IgE de ligação a nArt v 3 e a nPru p 3, sem inibição dos outros alérgenos identificados. Actualmente em evicção rigorosa de semente de girassol, sendo portadora de dispositivo auto-injector de adrenalina.

Palavras-chave: Alergia alimentar, anafilaxia, óleo de girassol, proteínas de transferência de lípidos, semente de girassol.

INTRODUCTION

Food allergy to seeds and nuts is increasing in Portugal, both in terms of prevalence and severity. Sunflower seed (*Helianthus annuus*), despite being frequently consumed in the form of oil, margarine, bread products and seeds, as an aperitif or in salads, rarely induces anaphylaxis¹⁻⁵. Sensitisation may occur to storage proteins (2S albumins) and to lipid transfer proteins (LTPs). Sunflower seed is known to have an LTP (Hel a 3) with a molecular weight of ~9 kDa⁵. There are different 8 albumins in the 2S fraction of sunflower seed which have also been identified as potential allergens in sesame seed, walnut, peanut and mustard⁶.

CLINICAL CASE

A 32-year-old female patient with persistent moderate-severe allergic rhinoconjunctivitis since her twenties, sensitised to artemisia, grass, olive, pellitory, plantain, chenopod and salsola pollens and cat dander. She was seen by an allergist in 2010 due to the occurrence of three immediate-onset (5-10 minutes) anaphylactic reactions (the first in 2007) upon ingestion of sunflower seed. These episodes were characterised by periorbital angioedema, oropharyngeal pruritus, rhinitis, cough, stridor and wheeze which regressed on administration of oral antihistamine and corticosteroids.

Table 1. Positive results in skin-prick-tests, serum specific IgE and the ImmunoCAP ISAC[®]

| Skin-prick-tests (mm) | Specific IgE (kU/L) | ImmunoCAP ISAC [®] (ISU) |
|--|---|---|
| <i>Olea europaea</i> : 18x7 | <i>Olea europaea</i> : 17,9 | nOle e 1: 23 |
| Poaceae: 11x6 | <i>Dactylis glomerata</i> : 22,7 <i>Festuca elatior</i> : 18,1 <i>Lolium perenne</i> : 18,2 <i>Phleum pratense</i> : 17,4 <i>Poa pratensis</i> : 22,1 | nCyn d 1: 7,8 rPhl p 1: 2,2 nPhl p 4: 0,5 |
| <i>Artemisia vulgaris</i> : 13x6 | <i>Artemisia vulgaris</i> : 49,7 | nArt v 1: 16 nArt v 3: 0,8 |
| <i>Parietaria judaica</i> : 7x5 | <i>Parietaria judaica</i> : 14,6 | rPar j 2: 37 |
| <i>Salsola kali</i> : 6x5 | n.t. | nSal k 1: 10 |
| <i>Chenopodium album</i> : 9x4 | n.t. | – |
| <i>Plantago lanceolata</i> : 3x3 | n.t. | – |
| Cat: 5x4 | – | rFel d 1: 0,8 |
| Sunflower seed: 9x7 Pistachio: 4x3 Pine nut: 5x3 Mustard: 5x4 | Sunflower seed: 79 Pistachio: 1.41 n.t. n.t. | nPru p 3: 3,2 |

ISU: ISAC[®] Standardized Units; n.t.: not tested.

In the skin-prick-tests a wheal with mean diameter ≥ 3 mm was considered a positive cut-off.

In the serum specific IgE levels $\geq 0,35$ kU/L were considered a positive cut-off.

In the ImmunoCAP ISAC[®] levels $\geq 0,3$ ISU were considered a positive cut-off.

The first episodes onset after ingestion of dried seeds, more precisely, sunflower seeds, and the third after eating chips fried in sunflower oil. The patient also complained of oropharyngeal pruritus on ingestion of pistachio nuts. She was able to ingest potato with no adverse reactions and denied complaints with other nuts, sesame seed and peanut. She did not eat mustard as she didn't like the flavour.

In the allergology and clinical immunology appointment, skin-prick-tests (using extracts from Bial-Aristegui[®], Bilbao, Spain) were performed. These were positive to sunflower seed (9x7mm), mustard (5x4mm), pistachio nut (4x3mm) and pine nut (5x3mm), and negative to other nuts, sesame seed and peanut. Serum total immunoglobulin E (IgE) was 158 UI/mL and specific IgE to sunflower seed was 79 kU/L (Phadia – Thermo Fisher Scientific[®], Uppsala, Sweden). ImmunoCAP ISAC[®] (Immuno Solid-phase Allergen Chip) was performed and revealed a predominantly LTP sensitisation profile (nPru p 3, nArt v3 and rPar j 2), shown in Table I.

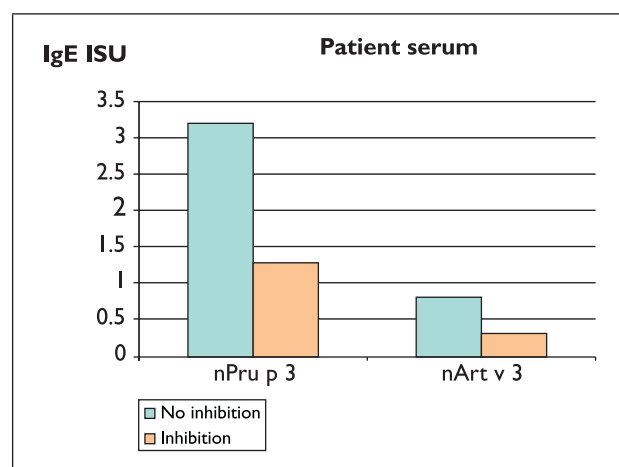


Figure 1. Result of the ISAC[®] inhibition study to sunflower seed extract with patient's serum: inhibition of nPru p 3 of 3.2 to 1.28 ISU (60%) and inhibition of nArt v 3 of 0.8 to 0.3 ISU (62.5%)

In the face of these results, we decided to perform an ISAC[®] inhibition study to extract of sunflower seed. A water soluble extract was prepared from sunflower seed mixed to a 1:1 dilution with patient's serum. After centrifugation, the supernatant obtained was incubated with the ISAC[®] allergen microarray. As control, serum from a patient allergic to Der p 1, was used, treated the same way. The sunflower seed extract inhibited (60%) (Figure 1) IgE binding to LTPs (nPru p 3 and nArt v 3); there were no inhibition to the remaining allergens identified in the ImmunoCAP ISAC[®] (nCyn d 1, rPhl p 1, nPhl p 4, nOle 1, nArt v 1, nSal k 1, rPar j 2 and rFel d 1) seen. Equally, no inhibition to the control serum occurred.

The patient is currently under strict avoidance of sunflower seed, including to food cooked in sunflower seed oil, and to nuts. She was prescribed a 0.3mg epinephrine autoinjector device for intramuscular administration.

DISCUSSION

Allergy to sunflower seed has been reported in individuals who own birds fed with sunflower seeds^{3,7}, but cases such as we describe here, of a severe IgE-mediated food allergy, are rare. This case is also remarkable since the patient had anaphylaxis to sunflower seed oil. Allergy to sunflower seed oil has been reported^{8,9}, but is considered an uncommon event as the majority of patients with allergy to this seed tolerate the small amount of the allergenic protein contained in sunflower seed oil^{2,4}. That fact can be explained by the patient's sensitisation to LTPs.

LTPs are a class of ubiquitous allergens specific to vegetable-origin foodstuffs such as fruits and cereals¹⁰. Sensitisation to LTPs is clinically significant as these are

stable, heat-resistant and peptic-acid-resistant panallergens with well-preserved protein domains which give rise to a high degree of cross-reactivity among botanically non-related foodstuffs¹⁰. These characteristics explain that sensitisation to these proteins may be the cause of the anaphylaxis on ingestion of sunflower seed oil cooked at high temperatures, as in this case we describe here.

Symptoms of allergic rhinitis on exposure to pollens occurred prior than the food allergy, something in line with the hypothesis raised that we were dealing with a case of pollen allergy with cross-reactivity to LTPs, afterwards confirmed through the ISAC[®] inhibition study. Although rPar j 2 is also an LTP, no binding to this allergen was observed. This is due to the fact that this protein belongs to another subfamily of LTPs unrelated to the LTPs associated to *Rosacea* fruits.

Equally so, the 2S albumin family is an important class of allergenic proteins common in seeds. Their presence in almost all edible seeds must be factored in, not only due to the high rate of clinical reactions in sensitised people but also due to the real possibility of cross-reactivity between different proteins of the same class¹⁰. They have also been identified as having allergenic potential in sesame seed, walnut, peanut and mustard⁶. This cross-reactivity was high in the case we present here, reflected by the sensitisation to pistachio, pine nut, and mustard, which had clinical repercussion in the case of pistachio. This justifies the choice made to recommend avoidance of nuts.

An anaphylactic reaction triggered by the LTP of sunflower seed was recently described in the literature⁵. The increasing consumption of dried sunflower seeds and their widespread use in snacks, salads and bread products, plus the fact that allergy to sunflower seed might be associated to sensitisation to aeroallergens common in Europe such as artemisia pollen⁵, explains the recent increase in the rate of reported cases.

In addition, the severity of the clinical reaction we report and the high likelihood of ingesting sunflower seed as a hidden allergen in snacks and in food cooked in sunflower oil justifies the publication of this case study: this allergy should be considered during the work-up in cases when the foodstuff triggering the allergy is not clear.

A definite and fast diagnosis allows for the timely recommendation to practice strict avoidance of not only sunflower seed and food cooked in sunflower oil, but also of foodstuffs with cross-reactivity, thus minimising the possibility of recurrence of a severe anaphylactic reaction.

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