Case Report

A case report of mono-sensitization to peanut component Ara h6

Maria G. Bartellino*, Lisa Barisciano

Department of Pediatrics, Goryeb Children’s Hospital at Morristown Medical Center, Morristown, New Jersey, USA

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*Correspondence:
Dr. Maria G. Bartellino,
E-mail: Maria.Bartellino@atlantichealth.org

ABSTRACT

Peanut is the most common food allergen in the US, affecting 1-2% of the population and studies show that it is still on the rise. Component testing has offered better insight into the likelihood of reactivity with exposure. Extensive literature shows Arachis hypogaea (Ara h) 2 as being the most clinically significant component identified to correlate with reactivity with exposure to the peanut protein; however, there is minimal research on the reactivity of Ara h6. This case report describes a patient with a clinical reaction to peanut as a toddler and subsequent positivity on annual skin testing with commercial peanut extract, likely confounded by positive birch with advancing age. Immuno CAP testing revealed a negative Ara h2 and positive Ara h6, describing mono-sensitization to Ara h 6 and high probability of clinical reactivity. The importance of this case is to raise awareness of other highly allergenic components in patients with peanut allergy.

Keywords: Arachis hypogaea, Component testing, Peanut allergy

INTRODUCTION

Peanut is the most common allergen in the US, affecting 1-2% of the population and studies show that it is still on the rise, likely due to dietary habits and increase in pollen exposures compared to other countries.1,2

The spectrum of peanut allergies ranges from mild pollen-induced cross-reactivity to life-threatening anaphylaxis. For some, the threshold for reaction can be as minimum as 0.2 mg of peanut protein.3 With the addition of component blood testing, authors have been able to better characterize the likelihood of reaction with exposure to peanut.

More than 13 peanut major and minor allergenic proteins in peanuts have been identified by the allergen nomenclature subcommittee of the International Union of immunological societies. Of these 13, there are several that are most clinically relevant in causing IgE-mediated reactions. Arachis hypogaea (Ara h) 5, 8, and 9 are associated with oral allergy syndrome. Ara h 8 in particular is chemically related to birch pollen.4,5 It confers pollen-induced cross-reactivity and is rarely associated with anaphylaxis. Ara h 1, 2, 3, 4, 6, and 7 are seed storage proteins and are therefore more allergenic than other components.5,6 Ara h2 and h6 are both 2S albumins and have been shown to be most clinically significant in exhibiting the highest probability of reactivity with exposure to peanut.6,7,8 Interestingly, 90% of those with peanut allergy in the US have specific IgE to Ara h 2 component.

Literature has shown a strong association between the severity of reaction with the increase in sensitization to both Ara h2 and Ara h6. Review of studies is significant for patients with either mono-sensitization to Ara h 2 or sensitization to both Ara h 2 and h 6.9,10 There is little research, however, describing patients with mono-sensitization to Ara h6.
The objective of the case report is to describe a patient with sensitization to Ara h 6 in the setting of elevated component to birch pollen.

CASE REPORT

Patient is an 8-year-old female with history of mild intermittent asthma presenting for annual follow-up for food allergy and allergic rhinoconjunctivitis.

At 14 months-old, our patient developed ocular edema and facial urticaria which progressed to diffuse urticaria following a smear of peanut butter on her face. There was no coughing, wheezing nor vomiting associated with the exposure. Skin prick testing with a commercial peanut extract was positive with a wheal of 10x18 mm and flare of 45 mm. Positive control had appropriate reactivity, with a wheal of 5 mm and flare of 25 mm (the negative control was not reactive). Initial immunoCAP testing performed at 2 years old demonstrated a peanut level of <0.35 kU/L.

Table 1 shows the outlines subsequent test results and skin prick testing at the patient’s annual follow-up visits. At 4 years-old, our patient began to develop clinical seasonal allergy symptoms.

<table>
<thead>
<tr>
<th>Age</th>
<th>Peanut extract wheal/flare (mm)</th>
<th>Immuno CAP</th>
<th>Birch wheal/flare or ImmunoCAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>14 months old</td>
<td>10x18/45</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2 years old</td>
<td></td>
<td>Peanut &lt;0.35</td>
<td>-</td>
</tr>
<tr>
<td>3 years old</td>
<td>25x35/45</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4 years old</td>
<td>25 x 45/50</td>
<td>-</td>
<td>10x15/30 - with clinical seasonal symptoms</td>
</tr>
<tr>
<td>5 years old</td>
<td>10x22/40</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6 years old</td>
<td>10 x 20/40</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7 years old</td>
<td>12 x 18/30</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>8 years old</td>
<td>20/45</td>
<td>Roasted peanut prick prick 10 x 30 wheal</td>
<td>Total peanut 1.93 Ara h1,2,3,9 &lt;0.1 Ara h8 21.6 Ara h6 0.95 Birch &gt;100</td>
</tr>
</tbody>
</table>

Skin prick testing to commercial peanut extract revealed a wheal of 25 x 45 mm and flare of 50 mm, which was larger than previous. Environmental skin prick testing was significant for reactivity to birch extract (10x22 mm wheal; 30 mm flare). At 8 years-old, skin prick testing revealed reactivity not only to commercial peanut extract but also roasted. ImmunoCAP testing was obtained revealing elevated peanut level (1.93 kU/L), elevated Ara h 6 (0.95 kU/L), and an Ara h 8 (21.6) with birch (>100 kU/L). Ara h 1, 2, 3, and 9 were all negative. It is important to note that our patient had maintained strict avoidance of peanut with no accidental exposure to peanut after her initial reaction.

DISCUSSION

This case report demonstrates a patient with a history of a clinical reaction to peanut as a toddler. During her lifetime, she has not had any accidental exposures to peanut and, therefore, no further known reactions. Annual skin testing repeatedly demonstrated sensitization, despite initial undetectable peanut specific IgE on ImmunoCAP testing. Subsequent total peanut specific IgE in early childhood increased while still undetectable for Ara h 1, 2, and 3. The positivity seen in the total peanut was likely exacerbated by the presence of significant birch reactivity, which has been described in literature.4 Due to the confounding positivity of birch and a negative Ara h 2, an oral peanut challenge would have been considered for this patient. However, the very large size of her peanut skin test (particularly to roasted peanut which denatures the birch related protein) along with her sensitization to Ara h 6, the decision was made to continue strict avoidance of peanut as there is a very high probability of a clinical reaction with exposure.11,12

Advances in allergic component testing have offered guidance in understanding reactivity potential in patients with peanut allergy. Extensive research is available describing patients with a high probability of reactivity to peanut showing either mono-sensitization to Ara h 2 or sensitization to both Ara h 2 and h 6, however there is little research describing mono-sensitization to Ara h 6 alone.9,10 Due to its similarity to Ara h 2 in that they are both 2S albumins, Ara h 6, however there is little research describing mono-sensitization to Ara h 6 alone.9,10 Due to its similarity to Ara h 2 in that they are both 2S albumins, Ara h 6 is also a component that has a high probability of reactivity with exposure. It is important to note that the Ara h 6 component is not found on routine component testing. Therefore, without clinical...
suspicion, this highly allergenic component may be overlooked as an important marker of a peanut allergy.

CONCLUSION

Among all the allergens, including tree nuts, peanut is the most common. Mono-sensitization to Ara h 2 has been linked to increased probability of reactivity, however this case shows that mono-sensitization to Ara h 6 also can lead to significant reactions. Although it is not commonly found on routine component testing, it is important to note the allergenic potential of Ara h 6.

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REFERENCES