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CLINICAL TOLERANCE TO PEANUT DESPITE SENSITIZATION TO ARA H 1 AND ARA H 2: A SUPERVISED ORAL FOOD CHALLENGE–BASED CASE REPORT

Microbiology

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ABSTRACT

Peanut (Arachis hypogaea, Ara h) allergy is a potentially severe form of food allergy affecting an estimated 1% of adults and up to 3% of children in developed countries. The prevalence of peanut allergy has been increasing in the last few decades.Arachis hypogaea 2 (Ara h 2)-specific IgE is to date the best serologic marker to diagnose peanut allergy. Ara h 2 and Ara h 6 share sequence and structural similarities, and the majority of patients with peanut allergy are sensitized to Ara h 2.

KEYWORDS

Peanut allergy (PA) ,Component-resolved diagnostics (CRD), Oral tolerance induction (OTI), Skin Prick Testing,

INTRODUCTION

Component-resolved diagnostics (CRD) have improved the diagnostic precision of peanut allergy by identifying sensitization to storage proteins such as Ara h 1 and Ara h 2, which are traditionally associated with systemic reactions and persistent allergy (1)

Sensitization to Ara h 2 has been proposed as a promising biological marker for the severity of peanut allergy and may reduce the need for oral food challenges. (2)

Component-resolved diagnostics allow differentiation between primary peanut allergy and cross-reactive sensitization (3)

Ara h1 is a vicilin-type seed protein and a major peanut allergen. Co-sensitization to Ara h1, Ara h2, and Ara h3 is associated with increased risk of systemic reactions. (4)

Peanut allergy (PA) is not only one of the most common allergies in children but also one of the most important causes of life-threatening anaphylaxis events (5)

To date, no curative treatment for food allergy is available, except for oral tolerance induction (OTI), which is feasible for only a few children, and oral immunotherapy (OIT), which is not always accessible in every country. The principal therapy is allergen avoidance with rescue medication such as intramuscular epinephrine in case of inadvertent consumption. (6)

Because food allergy has a significant impact on patients' lives and unnecessary dietary restrictions can be harmful, an accurate diagnosis is of utmost importance (7)

While the prevalence of peanut sensitisation varies between 2.3% and 10.1% in Europe, only 0.3%–0.9% of these patients are clinically allergic, implying that testing is needed to distinguish these patients.(8)

The oral food challenge (OFC) remains the gold standard for food allergy diagnosis. (9)

Even if patients have strong evidence of PA (a clear association between peanut consumption and allergic manifestations along with sensitisation), it is generally mandatory to assess the allergen-reactive dose to design an OTI protocol. However, this test remains time-consuming, expensive, stressful, and sometimes difficult to interpret, especially in young children. (10)

In this context, reliable biological markers of clinical allergic reaction were investigated. Thus, the detection of allergen-specific IgE (sIgE) and molecular allergens such as Ara h 2 was shown to discriminate between patients with PA and patients sensitised to whole peanut extract but not clinically responsive to peanut. (11)

More recently, the basophil activation test (BAT) has emerged as an interesting in vitro functional assay that goes even further than just exploring sensitisation. It measures the expression, by flow cytometry, of surface activation markers such as CD63, a particular marker for basophil degranulation. (12)

Peanut allergy is one of the most common causes of food-induced anaphylaxis in children and often persists into adulthood (13)

Sensitization to storage proteins, particularly Ara h 2, has been shown to correlate strongly with clinical peanut allergy and reaction severity (14)

Nevertheless, EAACI guidelines emphasize that IgE sensitization alone cannot confirm or exclude clinical allergy and that oral food challenge remains the reference standard (15)

CASE PRESENTATION

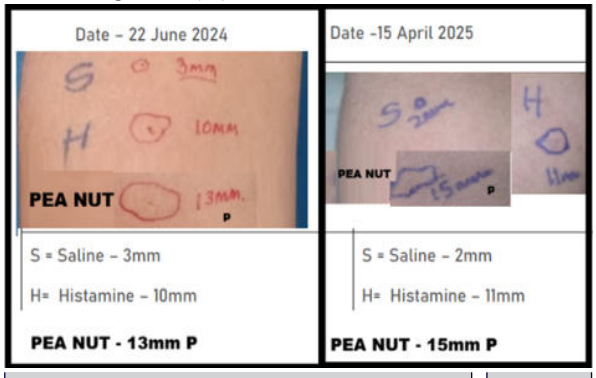
Clinical History

An 11-year-old boy was referred for evaluation of suspected peanut and tree nut allergy. He had a background of childhood atopy with dry skin suggestive of atopic dermatitis and had previously outgrown milk and egg allergy. There was a positive family history of atopic disease.

The child had **no prior history of anaphylaxis, wheeze, syncope, or emergency epinephrine use.** Parents reported occasional minimal accidental peanut exposure without objective symptoms. Because of increasing sensitization markers, further evaluation was undertaken.

Skin Prick Testing

Serial SPT demonstrated increasing peanut sensitization, with wheal size increasing from 13 mm to 15 mm over a 10-month period, accompanied by pseudopodal extensions. Histamine and saline controls were appropriate. Pseudopods were interpreted as indicating active sensitization but not predictive of reaction severity, consistent with EAACI guidance (16)



Component-Resolved Diagnostics

Legumes

Peanut (मूंगफली)	Ara h 1	7/8S Globulin	0.60
	Ara h 2	2S Albumin	2.77
	Ara h 3	11S Globulin	< 0.10
	Ara h 6	2S Albumin	0.12
	Ara h 8	PR-10	1.79
	Ara h 9	nsLTP	< 0.10
	Ara h 15	Oleosin	< 0.10

Serum CRD showed sensitization to:

- **Ara h 1:** 0.60 kUA/L
- **Ara h 2:** 2.77 kUA/L
- **Ara h 8:** 1.79 kUA/L

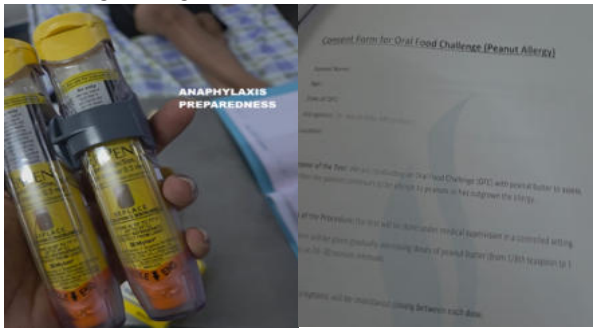
Sensitization to Ara h 1 and Ara h 2 suggested primary peanut sensitization,while Ara h 8 positivity indicated possible pollen-related cross-reactivity (17)

However, IgE levels alone were considered insufficient to predict clinical reactivity (18)

Oral Food Challenge

Indication

OFC was performed to determine true clinical reactivity in a sensitized child without a convincing reaction history, in accordance with EAACI diagnostic algorithm.



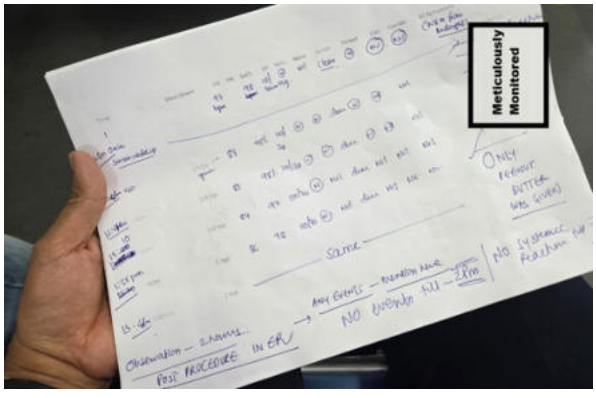
Pre-Challenge Assessment

Antihistamines were withheld for five days, and peanut was avoided for 2–4 weeks prior to challenge . Baseline clinical examination and vital parameters were normal. Written informed consent was obtained from the parents.

Challenge Protocol

A graded open OFC using peanut butter was performed in an emergency-equipped setting, following EAACI recommendations

Incremental doses were administered up to a cumulative dose equivalent to approximately 10 g of roasted peanut (20)



Outcome

The child tolerated all administered doses without respiratory, gastrointestinal, cardiovascular, or generalized cutaneous symptoms (21)

A transient localized swelling beneath the upper lip was observed, without progression or systemic involvement .

The patient was observed for 2.5 hours following the final dose . No delayed or biphasic reaction was noted, and the patient was discharged the following day in stable condition (22)

Follow-up

The patient remains under close follow-up, with parents instructed to monitor and record symptoms daily . At subsequent follow-up, no allergic symptoms have been reported, and peanut ingestion has not resulted in adverse reactions (23)

DISCUSSION

Ara h 2 is considered the most specific marker for clinically relevant peanut allergy, yet this case demonstrates that sensitization does not inevitably imply clinical reactivity. (24)

Possible explanations include moderate Ara h 2 levels, co-sensitization to Ara h 8, and absence of prior objective reactions [20]. EAACI guidelines clearly state that no IgE cutoff can replace OFC in diagnostically ambiguous cases [16].

CONCLUSION

Peanut allergy is a prominent IgE-mediated food allergy that has garnered substantial clinical attention owing to the severity of reactions and the increasing prevalence and rates of anaphylaxis. Peanut allergy affects about 25% of children with food allergy in the United States. Although many food allergies are transient with age, peanut allergy appears to be particularly persistent.

This case highlights that Ara h 1 and Ara h 2 positivity does not uniformly predict clinical peanut allergy .(25)

Specific IgE to Ara h 2 and Ara h 6 is the best serologic markers to diagnose peanut allergy.(26)

Supervised OFC remains indispensable for accurate diagnosis and prevention of unnecessary dietary restriction.

Ethical Statement

Written informed consent was obtained from the patient's parents. No patient photographs are included, as the child experienced discomfort during the procedure. The patient continues under close clinical follow-up.

REFERENCES

1. Sicherer SH, Sampson HA. Food allergy: epidemiology, pathogenesis, diagnosis, and treatment. *J Allergy Clin Immunol*. 2014.
2. Krogulska A, Wood RA. Peanut allergy diagnosis: moving from basic to more elegant testing. *Pediatr Allergy Immunol*. 2020; 31: 346-357.
3. Neuman-Sunshine DL, Eckman JA, Keet CA, et al. The natural history of persistent peanut allergy. *Ann Allergy Asthma Immunol*. 2012; 108: 326-331.e3.
4. Flokstra-de Blok BMJ, Dubois AE, Vlieg-Boerstra BJ, et al. Health-related quality of life of food allergic patients: comparison with the general population and other diseases. *Allergy*. 2010; 65: 238-244.
5. Lyons SA, Clausen M, Knulst AC, et al. Prevalence of food sensitization and food allergy in children across Europe. *J Allergy Clin Immunol Pract*. 2020; 8: 2736-2746.e9.
6. Perry TT, Matsui EC, Conover-Walker MK, Wood RA. Risk of oral food challenges. *J Allergy Clin Immunol*. 2004; 114: 1164-1168.
7. Nowak-Wegrzyn A, Assa'ad AH, Bahna SL, et al. Work Group report: oral food challenge testing. *J Allergy Clin Immunol*. 2009; 123: S365-S383.
8. Ebo DG, Sainte-Laudy J, Bridts CH, et al. Flow-assisted allergy diagnosis: current applications and future perspectives. *Allergy*. 2006; 61: 1028-1039.
9. Santos AF, Douiri A, Bécares N, et al. The basophil activation test differentiates between allergy and tolerance in peanut-sensitized children. *J Allergy Clin Immunol*. 2014; 134: 645-652.
10. Santos AF, du Toit G, Douiri A, et al. Distinct parameters of the basophil activation test reflect the severity and threshold of allergic reactions to peanut. *J Allergy Clin Immunol*. 2015; 135: 179-186.
11. Duan L, Celik A, Hoang JA, et al. Basophil activation test shows high accuracy in the diagnosis of peanut and tree nut allergy: The Markers of Nut Allergy Study. *Allergy*. 2021; 76: 1800-1812.
12. Santos AF, Lack G. Basophil activation test: food challenge in a test tube or specialist research tool? *Clin Transl Allergy*. 2016; 6: 10.
13. Venter C, et al. Diagnosis and management of food allergy. *Allergy*. 2021.
14. Sampson HA. Peanut allergy. *N Engl J Med*. 2002.
15. Fleischer DM, et al. Oral food challenge as gold standard. *J Allergy Clin Immunol Pract*. 2020.
16. Dreborg S. Skin prick testing in food allergy. *Allergy*. 2015.
17. Dang TD, et al. Ara h 2 as a predictor of peanut allergy. *J Allergy Clin Immunol*. 2012.
18. Matricardi PM, et al. Component-resolved diagnosis in food allergy. *Allergy*. 2016.
19. Thomsen SF. Genetics of atopy. *Allergy*. 2015.
20. Bird JA, et al. Standardized OFC dosing. *J Allergy Clin Immunol Pract*. 2017.
21. Niggemann B, et al. Practical aspects of food challenges. *Allergy*. 2012.
22. Simons FE, et al. Observation after food challenge. *Allergy*. 2011.
23. Sicherer SH. Limitations of specific IgE testing. *J Allergy Clin Immunol*. 2011.
24. Mittag D, et al. PR-10 proteins and cross-reactivity. *Allergy*. 2004.
25. Nicolaou N, et al. Sensitization vs clinical allergy. Clin Exp Allergy. 2010. Oliver Hemmings
26. a,b,c, George Du Toit a,b,d, Suzana Radulovic a,b,d, Gideon Lack a,b,c,d, Alexandra F Santos, Ara h 2 is the dominant peanut allergen despite similarities with Ara h 6, J Allergy Clin Immunol,2020 Sep;146(3):621–630.e5. doi: 10.1016/j.jaci.2020.03.026