Advances in
Clinical and Medical Sciences
Volume 2, Issue 2

Case Report

Successful Desensitization to Peanut in a Rast Class II Child

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Abstract

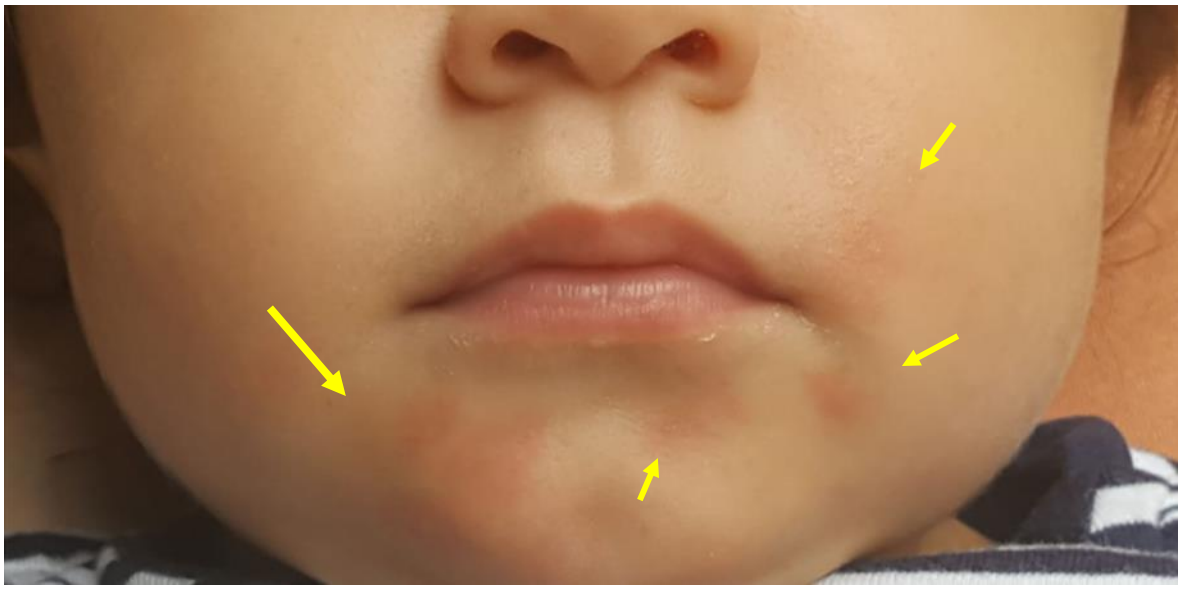
Peanut, tree nut, and seed allergies are some of the most common food allergies in both children and adults. These allergies tend to cause serious reactions and usually persist over time. The prevalence of peanut allergy is variable around the world. The highest rates are seen in Westernized countries such as the United States, the United Kingdom, Canada, and Australia, where the prevalence is approximately 1 to 2 percent [1-10]. In Paraguay we do not have statistics, but there is little reference. We present the case of a child with peanut allergy, with a class 2 peanut radioallergosorbency test (RAST) who we have desensitized with an innovative protocol for countries like ours, where the burden and costs must be taken into account. The only product approved by the US Food and Drug Administration (FDA) for OIT (oral immunotherapy) is a specific peanut allergen oral immunotherapy powder preparation that contains consistent amounts of the major peanut proteins.

Keywords: Allergy, urticaria, desensitization, immunotherapy, RAST, tolerance.

Clinical case

1 year 8 month old boy from Asunción with a history of vomiting, hives on the face and cough after eating chocolate with peanuts. We give him a quarter of the peanut kernel and after 2 minutes he expels the peanut and the child reports itching and shows his tongue, then erythematous lesions appear around the mouth (figure 1).

Figure 1



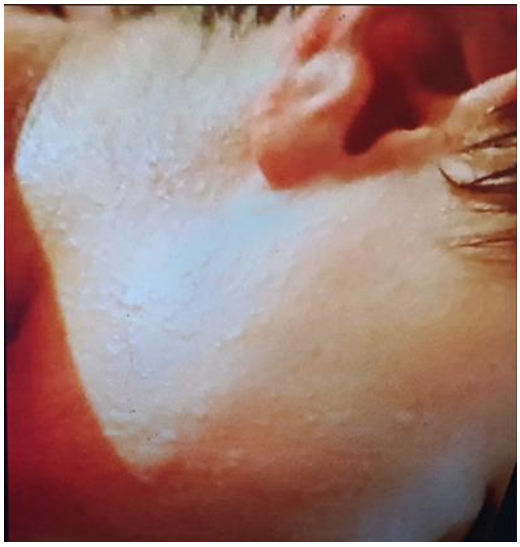
Peanut allergy is not usually common in our country, but it exists. In the figure below, 45 minutes after treatment you can see how the lesions disappear.

Figure 2

Accidentally, 6 months later he ate peanuts again and had the same symptoms. It was suggested to the family to suspend peanuts until the age of 5 years, waiting for immunological maturation.

At the age of 5 years and 4 months he accidentally ate 3 peanut cookies, they had approximately 10 peanut kernels per cookie, according to what the person who made the cookies indicated and the child did not have any reaction, he ate peanut cookies again the next day and for 7 days followed, on day 8 hives reappeared on the face, trunk and arms, which were very itchy and accompanied by a sporadic cough. (Figure 3, 4, 5 and 6)

Figure 3, 4 and 5



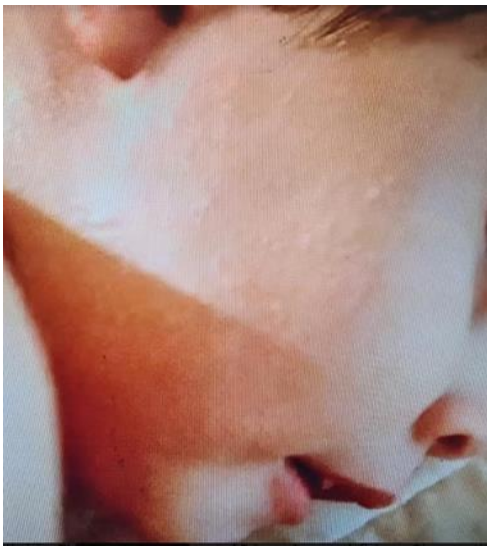
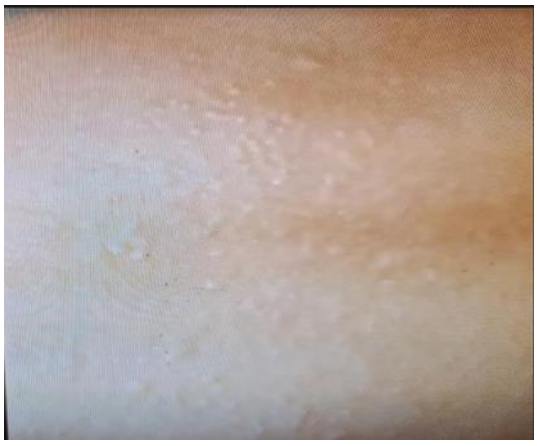


Figure 6 (child's back)



We requested total IgE and a RAST to the peanut, the results of which are in figure 7 below

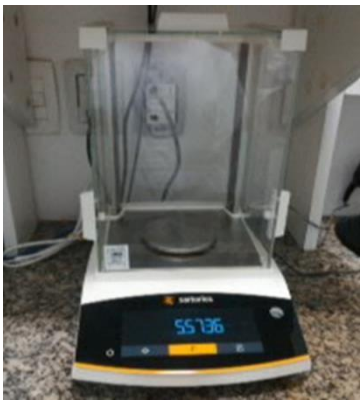
Figure 7

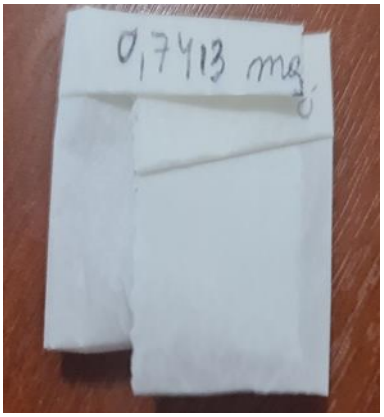
ANALYSIS	RESULTS	REFERENCES
IMMUNOLOGY		
IGE (Immunoglobulin E) Method: Enzime linked florescente assay	98 UI/ml	Up to 52 UL/ml
RAST Maní Method: Chemioluminescence	Class (II): 2.14 KUL	Class 0: less than 0.35 KUL Class l: 0,36 to 0,71 Class ll: 0,72 to 3,59 Class lll: 3.6 to 17,5 Class lV: 17.6 to 50 Class V: 50,1 to 100 Class Vl: greater than 100

METHOD

We weighed a peanut kernel with a Sartorius scale, the result was that 1 (one) peanut kernel weighed 0.7413 mg. (figure 9)
Figure 9 Sartorius balance, with which the peanuts were weighed, 1 (one) peanut weight 0.7413 mg

Figure 9





At 7 years of age, desensitization begins (after signing the informed consent by his mother, which appears in the annexes) to peanuts, knowing that the typical threshold dose to trigger objective symptoms is equivalent to one to three peanut kernels [12]. Photo: Initial peanut desensitization dose (circled). Figure 10

Figure 10

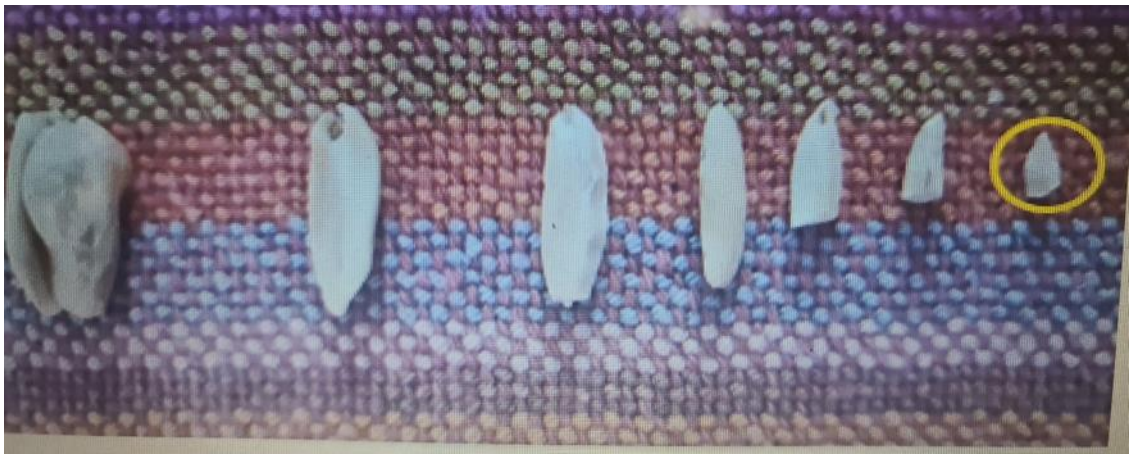
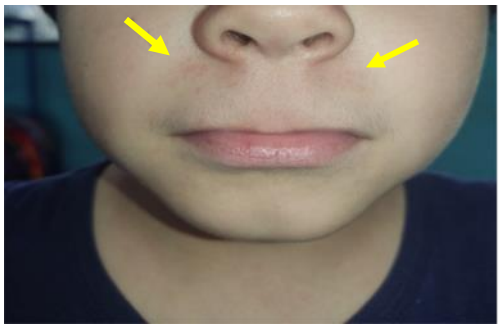
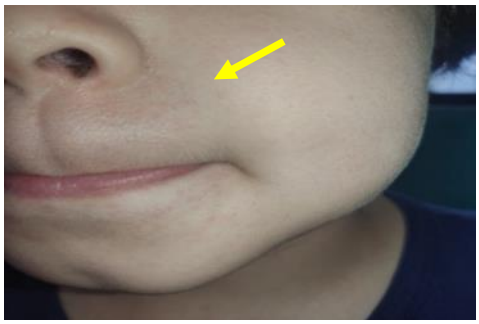


TABLE 1: PEANUT DESENSITIZATION SCHEME

DAY	Weight in MILLIGRAMS of peanut kernel	Protein content per peanut kernel
1	0.14826 1/5 grain	
2	0.18532 ¼ grain	
3	0.24711 1/3 grain	
4	0.37065 ½ grain	
5	0.74131 (1 grain)	161 to 325 mg per grain average 223mg
6	0.8895	
7	0.9984	
8	0.9901	
9	1,4826 (2 grain)	
10	1.6308	
11	1,6679	
12	1.7297	
13	2,2239 (3 grain)	
14	3 grain y ½	
15	4 grain	
16	4 grain 1/2	
17	5 grain	
18	10 grain	
19	15 grain	
20	20 grain	

The patient tolerates the first 8 doses and on day 9 with 1 peanut kernel (approximately 223 mg of peanut protein) he presents pain in the epigastrium, erythematous reactions around of the nose, and itching of the tongue, (photo)





He does not receive treatment, the itching disappears after 20 minutes and the lesions begin to fade after three hours.
The previous dose is repeated the next day, he only reports slight itching of the tongue, the ascending doses are continued without any medication, the mild itching of the tongue immediately after ingestion of the peanut continues until the ingestion of three whole peanut kernels, dose with which the itching of the tongue disappears.
We provoked the patient until we reached 20 peanut kernels per day, without any reaction.
We reached the fourth month without symptoms, we suspended the peanuts for 1 month, and we gave him peanuts again after the month, checking the good tolerance of it, without any symptoms.

Pathogenesis

Nine major and minor allergenic proteins have been identified in peanut (Arachis hypogaea), designated Ara h 1 to 9, which are responsible for IgE-mediated reactions.
[13, 14]. The dominant allergens in most populations are Ara h 1 to 3, which are seed storage proteins vicilin, conglutin, and glycinin, respectively. Ara h 4, 6 and 7 are also seed storage proteins. Ara h 4 is a nearly identical isoform of Ara h 3. Ara h 6 is highly homologous to Ara h 2, and Ara h 7 is also a conglutin.

Ara h 5, 8 and 9 are proteins associated with pollen and food allergy syndrome (oral allergy syndrome). Ara h 5 is a profilin, Ara h 8 is a homolog of Bet v 1 (a birch allergen) [13], and Ara h 9 is a non-specific lipid transfer protein similar to Pru p 3 (a peach allergen).) [13,14]

In the United States and Europe, 44 to 77 percent of patients with peanut allergy have specific IgE for Ara h 1 and 2, and 25 to 77 percent have specific IgE for Ara h 3 [15, 16]. In contrast, IgE specific to Ara h 8 or 9 is more common in individuals with concomitant sensitization to birch or peach pollen, especially in northern and southern Europe, respectively [12-13]. The presence of IgE antibodies to Ara h 2 is most closely associated with systemic reactions to peanut [17].

Peanut allergy can develop through primary sensitization to the food itself or through secondary sensitization through sensitization to cross-reactive allergens (for example, birch pollen). [13]

Risk Factors for Peanut Allergy

Risk factors for the development of peanut allergy include severe atopic dermatitis and/or chicken egg allergy in young infants [18]. Peanut allergy is also associated with the use of skin care products containing raw peanut oil in young children with a history of atopic dermatitis [19] and the extent of peanut consumption at home [20].

Family factors: Initial data from observational studies suggested that younger siblings of children with peanut allergy were at increased risk of developing peanut allergy [21,22]. However, data from subsequent studies indicate that this finding is in part due to late introduction in this population [23].

Genetic factors: In a case-control study, loss-of-function mutations in filaggrin were associated with peanut allergy with a positive oral food challenge test (odds ratio [OR] 5.3) [24] . These findings were replicated in a different population (OR 1.9) in the same study.

Skin care products: A British study found that the development of peanut allergy was associated with the use of skin care products containing peanut protein in crude oil form in children with atopic dermatitis, particularly those with active eczematous rashes [25].

Timing of first exposure: The timing of a food introduction likely influences the development of allergy versus tolerance. Peanut allergy has more than doubled in young children in countries where it was recommended to delay the introduction of peanuts until at least three years of age [1,4]. Additionally, the rate of peanut allergy is lower in countries where peanuts are introduced at a younger age [6].

Clinical Features: Clinical manifestations of IgE-mediated reactions

DERMATOLOGICAL: pruritus, redness, urticaria/angioedema, diaphoresis.
EYES: conjunctival injection, lacrimation, periorbital edema, pruritus.
RESPIRATORY TRACT: nose/oropharynx (sneezing, rhinorrhea, nasal congestion, oral pruritus, metallic taste), upper respiratory tract (hoarseness, stridor, choking sensation, laryngeal edema), lower respiratory tract (dyspnea, tachypnea, wheezing, cough, cyanosis)
CARDIOVASCULAR: conduction disturbances, tachycardia, bradycardia (if severe), arrhythmias, hypotension, cardiac arrest.
GASTROINTESTINAL: nausea/vomiting, abdominal cramps, bloating, diarrhea.
NEUROLOGICAL: Sensation of impending doom, syncope, dizziness, seizures.

Threshold Dose: The typical threshold dose to trigger objective symptoms is equivalent to one to three peanut kernels [12]. Peanut kernels vary in size, with peanut protein content ranging from 161 to 325 mg per peanut kernel (average 223 mg). Peanut flour is partially defatted and is typically 50 percent protein (12 percent fat). Patients with severe reactions typically have lower threshold doses of peanut protein than patients with mild symptoms [27]. The dose predicted to cause a reaction in 5 percent of patients (ED 05) with peanut allergy is 1.5 mg of peanut protein. However, in a study of 378 children with peanut allergy of any severity, only eight patients (2.1 percent) challenged with 1.5 mg of peanut protein had an objective reaction that met predetermined reaction criteria [28]. No association was observed between reacting to this threshold dose and peanut skin prick test responses or Ara h 2-specific IgE levels. The lowest reported level of peanut triggering an IgE-mediated reaction in both children and in adults it is 0.05 mg of peanut protein [27,29].

FACTORS ASSOCIATED WITH HIGHER LIKENESS OF PASSING A CHALLENGE INCLUDE:

Lower levels of food-specific IgE or smaller SPT hives (cutoff values vary from food to food and may be lower for young children)
Decreasing trends over time in the size of food-specific IgE or SPT wheals, even if levels remain relatively high
Absence of interval history of symptoms triggered by accidental exposure
Less failed oral food challenge and failure at a higher dose

Diagnosis

The clinical history is very important in guiding the diagnostic evaluation in a patient with suspected peanut allergy; testing is not recommended if the clinical history suggests that the probability of an allergy is low [30].

Foods suspected of causing an IgE-mediated reaction can be evaluated with skin tests or food-specific IgE levels (e.g., ImmunoCAP), which indicate whether or not the patient has IgE antibodies against the suspected food. The larger the mean diameter of the skin test papule or the higher the number on the immunoassay, the greater the likelihood that the food being tested is the cause of the allergic reaction. An oral food challenge should be performed to establish the diagnosis if these tests are negative in the face of a convincing history.

Treatment of Peanut Allergy

Avoidance
Family education: It is essential to educate yourself and be aware of the foods that contain the specific allergen.

Emergency action plan: in the event of a serious allergic reaction, a plan must be in place
emergency action prescribed by your doctor, preferably an allergist, and provide detailed instructions on how to use self-injectable epinephrine (adrenaline) and when to seek emergency medical attention.

Medications to treat symptoms: Oral antihistamines may be prescribed to treat mild to moderate symptoms (itching, hives, or nasal congestion).

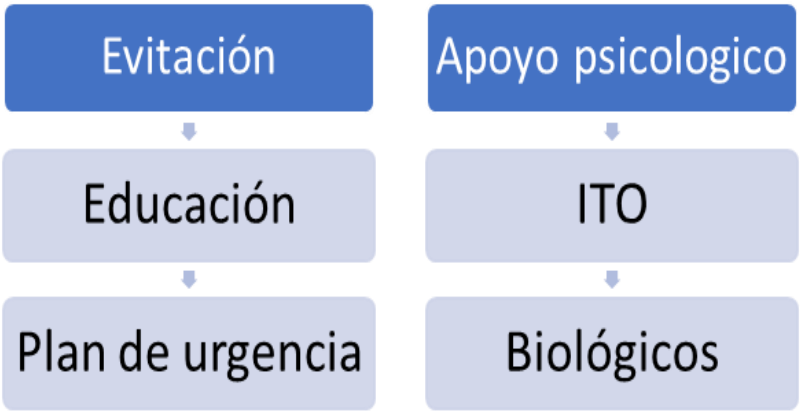
Psychological support: living with a food allergy can have a significant emotional impact. It is important to seek psychological support and advice to control stress and anxiety.

Oral Immunotherapy: (ITO)Oral immunotherapy (a type of desensitization) is available for peanut, In 2020, the US Food and Drug Administration (FDA) approved an allergen-specific formulation of oral immunotherapy powder of peanuts with the provision of continuous monitoring through a Risk Evaluation and Mitigation Strategy (REMS) that includes requirements to ensure safe use and minimize the risk of anaphylaxis.

The ultimate goal of therapeutic approaches for food allergy is to induce permanent tolerance to the food, in which allergic reactions do not recur upon re-exposure after a period of abstinence.

Biotherapeutic agents: Especially monoclonal antibodies that can block some important pathophysiological pathway. In this way, the use of anti-IgE monoclonal antibodies has been investigated, or those directed against type 2 cytokines: anti IL-4R, anti IL-13, anti IL-5, anti IL-5R, even against alarmins: anti IL-33 , anti IL-25 or anti TSLP, towards different manifestations of food allergy. [31,32]. Whose clinical use is not yet authorized.

PEANUT ALLERGY TREATMENT



Avoidance, Education, Emergency plan, psychological support, Oral immunotherapy, Biological

DEFINITIONS OF CLINICAL DESENSITIZATION, SUSTAINED UNRESPONSE, ORAL TOLERANCE

Desensitization	It is defined as an increase in the threshold of reaction to a food allergen in a supervised challenge test during active treatment. It is uncertain whether desensitization is equivalent to protection against reactions due to accidental ingestion. Desensitization is usually achieved after months of treatment and, importantly, only continues during treatment.
Lack of sustained response (SR)	It is defined as the lack of clinical reaction to a food allergen after stopping active therapy for a period of time. RS is thought to require some level of continuous exposure to the allergen to maintain the quiescent state.
Oral tolerance	It is defined as a complete lack of clinical reactivity to an ingested food allergen, usually as a natural phenomenon. This state of clinical tolerance is not believed to depend on continued exposure to the food allergen.

Conclusion

Peanut allergy can be severe

Peanut allergy may rarely recur in patients who have undergone an oral food challenge [33]. The first reported cases of recurrence occurred in children who did not incorporate peanuts into their regular diet after passing the challenge.

We recommend consuming normal portions of the food on a regular basis after passing a food challenge test. We also advise patients to maintain an emergency plan for at least one year, until they have demonstrated that the food is tolerated in the usual diet in normal amounts.

Patients should be educated on the proper management of accidental exposures.

The lesson of this case is that patients who have a low level of specific IgE (RAST) to peanuts, class (l) or class (ll), who did not have anaphylaxis and with factors associated with a greater possibility of passing a challenge, They may be safe candidates for desensitization, especially in countries like ours where the economic possibility of performing oral immunotherapy is not feasible.

REFERENCES

1. Sicherer SH, Muñoz-Furlong A, Sampson HA. Prevalencia de alergia al maní y a los frutos secos en los Estados Unidos determinada mediante una encuesta telefónica aleatoria: un estudio de seguimiento de 5 años. J Allergy Clin Immunol 2003; 112:1203.
2. Grundy J, Matthews S, Bateman B, et al. Aumento de la prevalencia de alergia al maní en niños: datos de dos cohortes secuenciales. J Allergy Clin Immunol 2002; 110:784.
3. Kagan RS, Joseph L, Dufresne C, et al. Prevalencia de la alergia al maní en niños de escuelas primarias en Montreal, Canadá. J Allergy Clin Immunol 2003; 112:1223.
4. Hourihane JO, Aiken R, Briggs R, et al. El impacto de las recomendaciones del gobierno a las madres embarazadas sobre la no ingesta de maní en la prevalencia de la alergia al maní en los niños del Reino Unido al ingresar a la escuela. J Allergy Clin Immunol 2007; 119:1197.
5. Ben-Shoshan M, Kagan RS, Alizadehfar R, et al. ¿Está aumentando la prevalencia de la alergia al maní? Un estudio de seguimiento de 5 años en niños de Montreal. J Allergy Clin Immunol 2009; 123:783.
6. Du Toit G, Katz Y, Sasieni P, et al. El consumo temprano de maní en la infancia se asocia con una baja prevalencia de alergia al maní. J Allergy Clin Immunol 2008; 122:984.
7. Kljakovic M, Gatenby P, Hawkins C, et al. Prevalencia y tratamiento informados por los padres de la alergia al maní y a los frutos secos en niños en edad escolar en el Territorio de la Capital Australiana. J Paediatr Child Health 2009; 45:98.
8. Mullins RJ, Dear KB, Tang ML. Características de la alergia al maní en la infancia en el Territorio de la Capital Australiana, 1995 a 2007. J Allergy Clin Immunol 2009; 123:689.
9. Sicherer SH, Muñoz-Furlong A, Godbold JH, Sampson HA. Prevalencia en Estados Unidos de alergia autoinformada al maní, los frutos secos y el sésamo: seguimiento de 11 años. J Allergy Clin Immunol 2010; 125:1322.
10. Ben-Shoshan M, Harrington DW, Soller L, et al. Un estudio poblacional sobre la prevalencia de la alergia al maní, los frutos secos, el pescado, los mariscos y el sésamo en Canadá. J Allergy Clin Immunol 2010; 125:1327.
11. Vickery BP, Berglund JP, Burk CM, et al. La inmunoterapia oral temprana en niños en edad preescolar alérgicos al maní es segura y altamente efectiva. J Allergy Clin Immunol 2017; 139:173.
12. Hourihane JO, Allen KJ, Shreffler WG, et al. Estudio del umbral de alérgenos del maní (PATS): nuevo estudio de provocación alimentaria oral de dosis única para validar la inducción de dosis en niños con alergia al maní. J Allergy Clin Immunol 2017; 139:1583.
13. Krause S, Reese G, Randow S, et al. Proteína de transferencia de lípidos (Ara h 9) como un nuevo alérgeno de maní relevante para una población alérgica mediterránea. J Allergy Clin Immunol 2009; 124:771.
14. Asarnoj A, Ostblom E, Ahlstedt S, et al. Síntomas notificados relacionados con el maní entre los 4 y los 8 años de edad en niños sensibilizados al polen de maní y abedul: resultados de la cohorte de nacimientos de BAMSE. Allergy 2010; 65:213.

15. Koppelman SJ, Knol EF, Vlooswijk RA, et al. Alérgeno del maní Ara h 3: aislamiento del maní y caracterización bioquímica. Allergy 2003; 58:1144.
16. Shreffler WG, Beyer K, Chu TH, et al. Inmunoensayo de microarrays: asociación de la historia clínica, la función de IgE in vitro y la heterogeneidad de los epítopos alergénicos del maní. J Allergy Clin Immunol 2004; 113:776.
17. Ballmer-Weber BK, Lidholm J, Fernández-Rivas M, et al. Los patrones de reconocimiento de IgE en la alergia al maní dependen de la edad: perspectivas del estudio EuroPrevall. Allergy 2015; 70:391.
18. Nicolaou N, Murray C, Belgrave D, et al. Cuantificación de IgE específica para extracto de maní entero y componentes del maní en la predicción de alergia al maní. J Allergy Clin Immunol 2011; 127:684.
19. Du Toit G, Roberts G, Sayre PH, et al. Ensayo aleatorizado sobre el consumo de maní en lactantes con riesgo de alergia al maní. N Engl J Med 2015; 372:803.
20. Lack G, Fox D, Northstone K, et al. Factores asociados con el desarrollo de la alergia al maní en la infancia. N Engl J Med 2003; 348:977.
21. Brough HA, Santos AF, Makinson K, et al. La proteína de maní presente en el polvo doméstico está relacionada con el consumo de maní en el hogar y es biológicamente activa. J Allergy Clin Immunol 2013; 132:630.
22. Sicherer SH, Furlong TJ, Maes HH, et al. Genética de la alergia al maní: un estudio en gemelos. J Allergy Clin Immunol 2000; 106:53.
23. Tsai HJ, Kumar R, Pongracic J, et al. Agregación familiar de alergia alimentaria y sensibilización a alérgenos alimentarios: un estudio basado en la familia. Clin Exp Allergy 2009; 39:101.
24. Lavine E, Clarke A, Joseph L, et al. Evitación del maní y diagnóstico de alergia al maní en hermanos de niños alérgicos al maní. Clin Exp Allergy 2015; 45:249.
25. Brown SJ, Asai Y, Cordell HJ, et al. Las variantes con pérdida de función en el gen de la filagrina son un factor de riesgo significativo para la alergia al maní. J Allergy Clin Immunol 2011; 127:661.
26. Lack G, Fox D, Northstone K, et al. Factores asociados con el desarrollo de la alergia al maní en la infancia. N Engl J Med 2003; 348:977.
27. Wensing M, Penninks AH, Hefle SL, et al. Distribución de dosis umbral individuales que provocan reacciones alérgicas en una población con alergia al maní. J Allergy Clin Immunol 2002; 110:915.
28. Hourihane JO, Allen KJ, Shreffler WG, et al. Estudio del umbral de alérgenos del maní (PATS): nuevo estudio de provocación alimentaria oral de dosis única para validar la inducción de dosis en niños con alergia al maní. J Allergy Clin Immunol 2017; 139:1583.
29. Ballmer-Weber BK, Fernandez-Rivas M, Beyer K, et al. ¿Cuánto es demasiado? Distribuciones de dosis umbral para 5 alérgenos alimentarios. J Allergy Clin Immunol 2015; 135:964.

30. Greenhawt M, Shaker M, Wang J, et al. Diagnóstico de alergia al maní: actualización de parámetros de práctica de 2020, revisión sistemática y análisis GRADE. J Allergy Clin Immunol 2020; 146:1302.
31. Hirano I, Dellon ES, Hamilton JD, et al. Efficacy of dupilumab in a phase 2 randomized trial of adults with active eosinophilic esophagitis. Gastroenterology 2020; 158 (1): 111-122.e110. Khodoun MV, Tomar S, Tocker JE, Wang YH, et al. Prevention of
32. food allergy development and suppression of established food allergy by neutralization of thymic stromal lymphopoietin, IL- 25, and IL-33. J Allergy Clin Immunol 2018; 141 (1): 171-179.e171.
33. van Erp FC, Boot J, Knulst AC, et al. Fracaso en la reintroducción después de desafíos negativos conmaní en niños. Pediatr Allergy Immunol 2014; 25:580.

Citation: Silvio Espinola, Adv Clin Med Sci, “Successful Desensitization to Peanut in a Rast Class II Child”. 2024; 2(2): 107

Received Date: September 06, 2024; Published Date: September 12, 2024

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