

Anaphylaxis with The Manifestation of Convulsions

* Aikaterini Salavoura¹, Nikoleta Lalioti¹, Panagiota Sotiropoulou¹, Dimitrios Chatzis¹

¹ Department of Allergy, Inflammation and Asthma, 1st Pediatric Clinic University of Athens, Greece.

Abstract

Febrile episodes accompanied by convulsions are a common pediatric condition. We present the clinical history and laboratory evaluation of a 2.5-year-old boy with a concomitant food allergy to cashew who experienced convulsions during a febrile episode after consuming cashews. Differential diagnosis was challenging, as was identifying of the culprit food. A food challenge confirmed an allergy to cashew tree nuts.

Key Words: Anaphylaxis, Central Nervous System, CNS manifestations, Febrile convulsions.

* Please cite this article as: Salavoura K, Lalioti N, Sotiropoulou P, Chatzis D. Anaphylaxis with The Manifestation of Convulsions. J Ped Perspect 2025; 13 (7): 19587-19592. DOI: **10.22038/jpp.2025.87964.5546**

*Corresponding Author:

Aikaterini Salavoura, Georgiou Papandreou 45, Goudi; Tel: +30-6977592484 ; E-mail: salavourakaterina@gmail.com

1- INTRODUCTION

Febrile convulsions are a common benign pediatric condition. Simple febrile convulsions are characterized by high fever, convulsions appearing on the first day of a feverish episode, short duration and a benign course. Complex febrile seizures, on the other hand, could appear at any time during the fever period and usually last longer, requiring some form of intervention. Complex febrile seizures predispose children to epilepsy, especially if there is a relevant family history.

The name 'anaphylaxis' comes from the Greek words "άνά" (meaning contrary) and "φύλαξις" (meaning protection) and was established by Charles Richet in 1913 when he was awarded the Nobel Prize. The description from the European academy of allergy and clinical immunology (EACCI) group is that the disease is a sudden, multisystem, direct reaction of hypersensitivity that is IgE or non-IgE mediated, leading to the massive release of vasoactive substances from mastocytes and basophils. The diagnosis is based on clinical criteria, as there are no laboratory tests to confirm the diagnosis and measurement of tryptase is only supportive of the diagnosis (1).

2- CASE PRESENTATION

A 2.5-year-old boy was hospitalized due an episode of convulsions accompanying fever that has started three days prior. The child had an atopic predisposition from his mother, but his individual history was uncomplicated. He had previously consumed walnuts, peanuts, and hazelnuts without adverse reactions, but he had never been exposed to cashews or pistachios. During a febrile infection, the child consumed some walnuts and five minutes later, a diffuse urticaria rash appeared on his whole body except for his face. At the same time, the child became irritable, complained of pain in his mouth and teeth. During an

examination by his pediatrician half an hour later, the child lost consciousness, developed an episode of tonic-clonic convulsions, and fell asleep. The pediatrician gave him an antihistamine and referred the child to secondary care. The child remained symptomatic and experienced dizziness for approximately one hour. He was given an injection of epinephrine, intravenous corticosteroids and intravenous fluids upon arrival at the secondary care facility and was then transferred to a central hospital. After the injection of adrenaline, the child completely recovered and upon arrival at the central pediatric hospital, he was well. Laboratory investigation in the hospital revealed slightly elevated leukocytes (WBC: 13150/μl, neutrophils: 91%, lymphocytes: 5.2%, Hb: 10.3g/dl, Hct: 31%, PLT: 379000/μl) and increased C-reactive protein (CRP): 64.4mg/l. Lumbar puncture was normal, and cultures of cerebrospinal fluid (CSF) and blood were also normal. Tryptase levels were not measured. Despite the negative laboratory findings, the child received IV a third generation cephalosporin.

A full allergic investigation was performed. The results of specific IgE evaluation and Prick to Prick tests (PZP on the skin) are presented in tables 1 and 2, respectively. Since the results were inconclusive, a component resolved diagnostic investigation was performed, showing rPru p 3 LTP (=0), rPru p 4 (=0), rJug r 1 (=0), rJug r 3 (=0), and rAna o 3=2.59(class 2). Jug r4, Jug r6, or Jug r2 were not studied.

The clinical history was complex and posed significant diagnostic challenges. A febrile convulsion was the most probable diagnosis, although the episode occurred on the third day of fever. However, it was a coincidence that the episode appeared a few minutes after the consumption of a culprit food. A concurrent infection was excluded by lumbar puncture. Sepsis was

ruled out from the blood count examination and negative blood cultures.

The time of symptoms appearance, the skin manifestations, and the rapid recovery after the epinephrine injection made an anaphylactic episode with manifestations from the central nervous system (CNS) quite possible. Unfortunately, serum tryptase was not measured to detect anaphylaxis (Figure 1).

The culprit food was not conveniently identified. Specific IgE measurements revealed a multisensitized individual in foods that he had consumed previously (Table 1). Multiple sensitizations to tree nuts and legumes in combination with stress due to the fever suggested a possible Lipid Transfer Protein (LTP) sensitization. However, further evaluation with blood tests, Prick to Prick tests and molecular component measurements excluded this possibility.

Walnut sensitization was confirmed neither by Prick to Prick testing, molecular

investigation nor by a challenge test. Challenge to walnut was negative and the child was able to consume (0,2gr /0,7gr / 2gr/ 7gr/ 20gr of walnut gradually). On the contrary, Prick to Prick sensitization and molecular results was significant for the cashew tree nut and pistachio, suggesting a possible contamination of walnut that the child consumed (Table 2). A more detailed history unraveled that both cashew and walnut were packed in the same box. A food challenge to cashew proved that the culprit food for the anaphylaxis was the cashew tree nut with reaction in the first dose.

The child was treated with antibiotics in the first place. However, after the allergic workout a strict avoidance of the consumption of cashew and pistachio was suggested. An auto injected epinephrine subscription was recommended and advice regarding the use of autoinjected adrenaline and an anaphylaxis plan was given to the parents.

Table-1. Specific IgE to a variety of foods.

| Food | IgE (KU/L, ImmunoCap method) | Class |
|------------------|------------------------------|-------|
| Egg white (F1) | 1.93 | III |
| Egg yolk (F75) | 0.879 | II |
| Milk(F2) | 1.21 | II |
| Mackerel (F3) | 3.81 | III |
| Peach (F95) | 0.183 | 0/1 |
| Walnut (F256) | <0.10 | 0 |
| Peckan (F201) | <0.10 | 0 |
| Hazelnut (F17) | 0.472 | II |
| Peanut (F18) | 0.488 | II |
| Cashew (F202) | 3.22 | III |
| Pistachio (F203) | 3.66 | III |
| Almond (F20) | 0.277 | I |

Table-2. Prick to Prick (PZP) to our child revealed sensitization to cashew and pea.

| Food | Diameter (mm) | | |
|-----------|---------------|----------|-----|
| Histamine | 5 (mm) | Almond | 0 |
| Pistachio | 5 | Peanut | 9 |
| Cashew | 9,5 | Pea | 4.5 |
| Pistachio | | Birch | 0 |
| Walnut | 0 | Peach | 0 |
| Hazelnut | 0 | Profilin | 0 |

3-DISCUSSION

Our case is considered unusual in terms of its clinical presentation and the complexity of reaching a diagnosis. While a febrile convulsion could be the most likely diagnosis, the time of presentation and the prolonged recovery raise concerns about a complex febrile seizure episode. The age of presentation is relevant to febrile convulsions, but family history was not suggestive of an epilepsy background. Febrile convulsions due to viral infections are infrequently accompanied by rashes, but the rashes persist and they do not disappear with adrenaline. The possibility of a febrile convulsion episode and concurrent food reaction cannot be excluded as well. Overall, the coincidence of the appearance of urticaria, teeth pain, and convulsions after the consumption of walnut raises many questions regarding the possibility of a common febrile episode.

Anaphylaxis presenting with CNS manifestations is a rare occurrence. The high titers of sensitization to cashew and the appearance of symptoms immediately after the first dose during the provocation test suggest that the possibility of an acute reaction to the food is most probable. Symptoms from the CNS during an anaphylactic episode are the result of hypoxia of the CNS due to the manifestations of the allergic shock (2).

The evaluation of the patient's food allergy is also considered a model for a step-by-step wise approach to food allergy diagnosis. Prick-to-Prick testing is considered as reliable as measurements of IgE with the ImmunoCap method. However, in our patient, there was a discrepancy between the results of these two methods. Ambiguous results of skin tests and specific IgE measurements can be clarified by currently available molecular

techniques. However, a food challenge test remains the cornerstone of diagnosis in food allergies (3). In our case, measurement of IgE in the blood detected sensitizations that did not correlate with the clinical history, potentially leading to inappropriate food restrictions. In our case, restricting of all nuts and also nuts that he had already consumed would avoid contamination. The challenge process clarified that the responsible allergen for the reaction was cashew (4). Due to cross reactivity with pistachio, both of these nuts were excluded from the patient's diet, and appropriate prevention measures for subsequent episodes as well as an epinephrine autoinjector were provided.

Although Lipid Protein allergy was not confirmed in our patient, allergists in Greece should always consider this diagnosis in patients showing multiple sensitizations to different groups of food. Non Specific LTP are proteins that transfer lipids in vitro and are related to the protection of the seeds of plants from disadvantageous conditions such as drought. These proteins protect against bacteria and fungi that destroy seeds and plants. They are plentiful within fruits, vegetables, and tree nuts. Their accumulation in the seed is related to the geographical area of the crop and the weather patterns. Patients sensitized to these proteins are usually older than our patient and they present with a variety of symptoms from oral allergy syndrome to anaphylaxis. Tooth pain in our patient could be suggestive of an LTP sensitization. Allergic reactions to LTP proteins are frequently characterized by the presence of co-factors such as exercise or stress, as in our case during a febrile episode (5). The prevalence of this syndrome is thought to increase in Mediterranean countries due to the high consumption of fruits and vegetables (6).

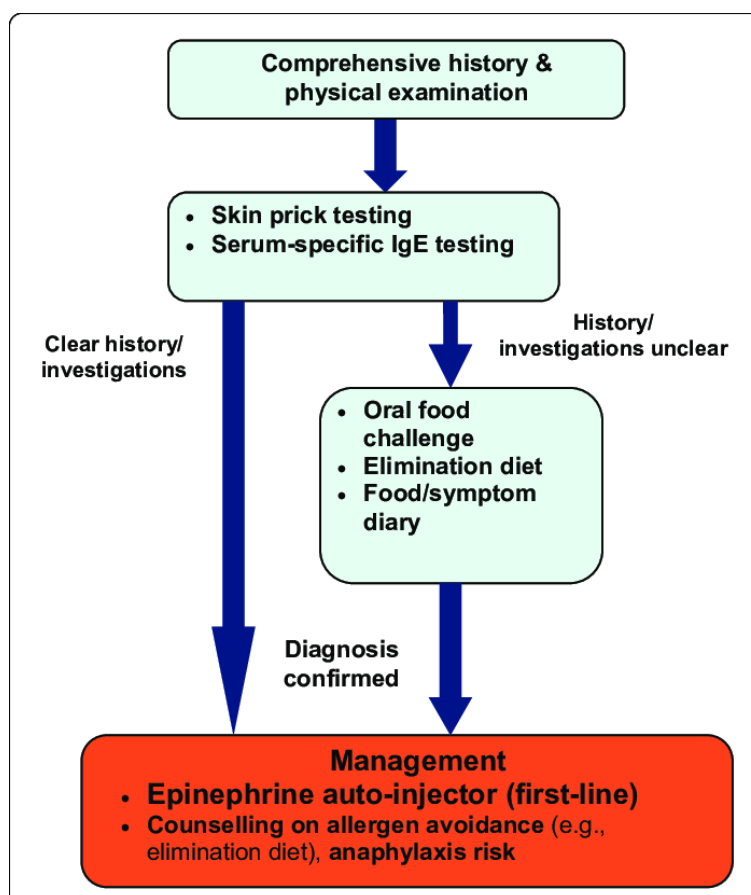


Figure-1: Simplified algorithm for the diagnosis and management of food allergy (4).

4- CONCLUSION

A rare case is presented of a febrile episode accompanied by urticaria, tooth pain, convulsions and significant sensitization to cashew. In this case, the coincidence of appearance of urticaria and convulsions after eating cashew would make the possibility of an anaphylaxis episode most probable.

Tree nut allergy is described in 1.2% of the population in Europe (EACCI). Allergy to cashew is a common tree nut allergy due to the recent increase in consumption. Reactions to foods vary from mild to severe including anaphylaxis. Symptoms of CNS due to anaphylaxis are a rare complication. Detecting the culprit food for reactions can be challenging in some cases. In this case contamination of walnuts with cashew was misinterpreted

by the parents. Thus, clinical history and laboratory tests could lead to a misdiagnosis. In all cases the gold standard to confirm a diagnosis of food allergy is oral challenge processes.

5- REFERENCES

1. Muraro A, Werfel T, Hoffmann-Sommergruber K, Roberts G, Beyer K, Bindslev-Jensen C, et al. EAACI food allergy and anaphylaxis guidelines: diagnosis and management of food allergy. *Allergy*. 2014 Aug;69(8):1008-25.
2. Lieberman P, Nicklas RA, Randolph C, Oppenheimer J, Bernstein D, Bernstein J, et al. Anaphylaxis—a practice parameter update 2015. *Annals of Allergy, Asthma & Immunology*. 2015 Nov 1;115(5):341-84.
3. Andorf S, Borres MP, Block W, Tupa D, Bollyky JB, Sampath V, et al.

Association of clinical reactivity with sensitization to allergen components in multifood-allergic children. *The journal of allergy and clinical immunology: in practice*. 2017 Sep 1;5(5):1325-34.

4. Wasserman S, Watson W. Food allergy. *Allergy, Asthma & Clinical Immunology*. 2011 Nov 10;7(Suppl 1):S7.

5. Van Winkle RC, Chang C. The biochemical basis and clinical evidence of

food allergy due to lipid transfer proteins: a comprehensive review. *Clinical reviews in allergy & immunology*. 2014 Jun;46(3):211-24.

6. Asero R, Pravettoni V. Anaphylaxis to plant-foods and pollen allergens in patients with lipid transfer protein syndrome. *Current opinion in allergy and clinical immunology*. 2013 Aug 1;13(4):379-85.