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CASE REPORT

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Oral mite anaphylaxis after pizza consumption in a child: A case report and review of the literature

Vasiliki Maria Kymioni*, Irine Karatsoli, Nikolaos Koronas, Ifigenia Karagiorga, Konstantinos Kakleas

Pediatric Allergy Unit, First University Department of Pediatrics, "Agia Sophia" Children's Hospital, National and Kapodistrian University of Athens School of Medicine, Athens, Greece

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Abstract
Introduction: House dust and storage mites are allergens associated with allergic rhinitis and asthma. Oral mite anaphylaxis manifests after ingesting contaminated flour with mites. We present the case of a 13-year-old child who developed anaphylaxis after consuming pizza.
Case presentation: A 13-year-old boy presented with respiratory distress after consuming pepperoni pizza, which he had previously tolerated. Skin prick and specific IgE test results revealed sensitization to house dust and storage mites. He passed an oral food challenge (OFC) for pepperoni pizza.
Discussion: Anaphylaxis to foods contaminated by mites (also known as "pancake syndrome") is often overlooked. In this case, the diagnosis was supported by the skin prick tests and specific IgE, although the flour used for pizza preparation could not be tested. The patient passed an OFC using ingredients from a different source.
Conclusion: Oral mite anaphylaxis is an unusual presentation that must be considered when other possible food allergy triggers have been excluded and the patient is sensitized to mites.

Introduction

Domestic mites, including house dust and storage mites, are common indoor arthropods and significant sources of allergens.[1] While house dust mites typically reside in household environments, storage mites can contaminate food products, particularly flour. In rare cases, ingestion of mite-contaminated food can lead to oral mite anaphylaxis (OMA), a severe allergic reaction also known as pancake syndrome.[2]We report the case of a 13-year-old child who developed anaphylaxis after eating pizza, consistent with OMA.

***Corresponding author**: Vasiliki Maria Kymioni, Pediatric Allergy Unit, First University Department of Pediatrics, "Agia Sophia" Children's Hospital, Thivon and Papadiamantopoulou, Athens, Greece. *Email address*: vmariakymioni@gmail.com

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Case Report

A 13-year-old boy presented with periocular edema, hoarseness, and respiratory distress 15 min after consuming pepperoni pizza. He had tolerated this food previously. Aside from pepperoni, ingredients included mustard, salt, sugar, paprika extract, and traces of egg, soya, and celery, all of which he had consumed uneventfully before. There was no intense exercise, though he reported strenuous walking. No other cofactors were identified.

The patient had allergic rhinitis and was sensitized to tree and grass pollens. Skin prick testing showed sensitization to nuts (he was consuming nuts in the form of sweets and foods), pollens, and strong reactivity to house dust mites (*Dermatophagoides pteronyssinus* and *Dermatophagoides farinae*) (Table 1). Serum IgE was positive for house dust and storage mites, including *Tyrophagus*, *Lepidoglyphus*, and *Glycyphagus* (Tables 2a and 2b), with notably high *Der p 2* and negative *Der p 1 (D. pteronyssinus*). Total IgE was 198 IU/mL. The ω-5 gliadin test was negative, arguing against wheat-dependent exercise-induced anaphylaxis. Mast cell tryptase was measured at hospital admission and at 12 h post-reaction and was found to be 5.2 ng/mL and 4.8 ng/mL, respectively (normal range: 1–11.4 ng/mL); hence, mast cell activation syndrome and mastocytosis were excluded.

He passed an oral food challenge (OFC) with pizza made from ingredients sourced elsewhere. As the original flour could not be tested, and given his pattern of sensitization and clinical history, the reaction was attributed to oral mite anaphylaxis (OMA) from mite-contaminated flour. None of the other household members who consumed the same pizza reported any symptoms, which may be attributed to the lack of sensitization to mites or variability in the allergen load per portion.

Discussion

We present the first case in Greece of a 13-year-old adolescent who developed an allergic reaction to pizza pepperoni that he was previously consuming and eventually was diagnosed with OMA, or "pancake syndrome."

Most of the OMAs reported in the literature come from tropical or subtropical climates.[3] The main reason is that increased heat and humidity in these climates favor the proliferation of mites in the food. Only a few reports of OMA stem from other regions, such as Porto Alegre, Brazil, and the United States.[3] Very recently, reports have emerged from temperate climates, such as the Netherlands and Ireland, highlighting the impact of climate change and global warming in the Northern Hemisphere.[4,5] Canavan et al. have stated that in Ireland, when the reaction occurred, the environmental temperature was 23°C, which is higher than the average.[5] In our case, the average temperature during the spring of 2023, when the reaction occurred, was 25°C.[6] Additionally, contamination could have ensued due to inappropriate storage outside the fridge at ambient temperature. Hence, similar cases are expected to occur soon in more countries with temperate climates, and medical professionals should be aware of this condition.

In our case, the culprit food was pizza dough prepared using wheat flour. The literature also supports this, whereas patients with similar symptoms had consumed foods prepared with wheat flour, including pancakes, sponge cakes, bread, pasta, and white sauce.[7] Other possible culprits include beignets and okonomiyaki (bonito and mackerel covered with flour).[8,9] Cheese, ham, chorizo, and salami are unusual foods that are less likely to be contaminated with mites.

Our patient was found to be sensitized to *Dermatophagoides pteronyssinus* and *Dermatophagoides farinae*, as well as to storage mites (*Acarus siro, Glycyphagus domesticus*, *Lepidoglyphus destructor, and Tyrophagus putrescentiae*). This finding aligns with other studies that report common OMA triggers, such as domestic species (*D. pteronyssinus, D. farinae, and Blomia tropicalis*), as well as storage mites (*Suidasia medanensis, Aleuroglyphus ovatus, Lepidoglyphus destructor, Tyrophagus putrescentiae, Thyreophagus entomophagus, and Blomia freemani)*.[3] The OMA cases from Ireland were attributed to *T. entomophagus*.[5]

The patient we present was found to be sensitized to *Der p 2* but not to *Der p 1*. This is consistent with the pathogenesis of OMA, since cooked or baked foods can trigger symptoms, and *Der p 2* is a heat-labile antigen.[10,11] Group 2 allergens have also been described as major allergens from storage mites: *Lep d 2* (*L. destructor*), *Gly d 2* (*G. domesticus*), and *Tyr p 2* (*T. putrescentiae*). There is cross-reactivity between Group 2 allergens of storage and house dust mites, with reported sequence identities of 36% between *Lep d 2* and *Der p 2*, 41% between *Tyr p 2* and

Table 1 Skin prick test results.

Allergen	Wheal diameter (mm)	Allergen	Wheal diameter (mm)
Histamine	8	*Dermatophagoides farinae*	15
Negative	0	Hazelnut	5
Pine tree	5	Almond	3
Olive tree	8	Walnut	8
Mugwort	0	Peanut	0
Ragweed	0	Cashew	5
Grass	5	Pistachio	3
Dermatophagoides pteronyssinus	17	White flour	0

Table 2 Specific IgE RAST allergens (CAP method).

Allergen	Concentration (IU/mL)
rDer p2 House dust mite	5
rDer p1 House dust mite	<0
Peanut	1.59
Hazelnut	1.18
Almond	0.94
Dermatophagoides pteronyssinus	3.38
Dermatophagoides farinae	6.41
Allergen	Concentration (IU/mL)
rPru p 3 LTP, peach	0.85
rTri a 19 ω-5 Gliadin, wheat	0.02
Tyrophagus putrescentiae	1.54
Lepidoglyphus destructor	0.92
Glycyphagus domesticus	0.69
Acarus siro	1.87
Banana	0.91
Peach	1.03

Der p 2, and approximately 80% among Group 2 allergens from storage mites.[11]

Our patient's peach *Pru p 3* (*Prunus persica*)–specific IgE level was 0.85 kUA/L, which falls just above the manufacturer's threshold for sensitization (≥0.35 kUA/L). From the clinical history, the patient was eating fruits and vegetables without any reaction. Additionally, he would consume foods and sweets that contained nuts, such as chocolate, baklava, hazelnut spread, and walnut pie, without any problem. Furthermore, *Pru p 3*–specific IgE levels are generally insufficient to support a diagnosis of nonspecific lipid transfer protein (nsLTP) allergy, in the absence of a suggestive clinical history. Higher *Pru p 3* titers have been associated with increased risk of severe reactions and cross-reactivity, whereas low levels typically indicate subclinical sensitization.[12] Furthermore, the *Pru p 3*: total IgE ratio in our patient was 0.0043, based on a total IgE of 198 IU/mL. According to Olivieri et al., a ratio ≥ 0.0073 provides optimal discrimination between true nsLTP allergy and asymptomatic sensitization, with an AUC (Area Under the Curve) of 0.89.[13] As our patient ratio falls well below this threshold, the findings do not support a diagnosis of nsLTP allergy. The absolute *Pru p* 3 level also falls far below the median (6.24 kUA/L) observed in nsLTP allergic patients in the same study. These findings collectively suggest that our case does not exhibit nsLTP allergy.[13]

In the literature, most patients with OMA are adolescents and young adults with a previous history of allergic rhinitis and/or asthma who develop between 10 and 240 symptoms after food consumption.[3,14] Our patient was an adolescent and was also suffering from allergic rhinitis. Cases of OMA have also been described in children.[15] Although OMA can manifest with symptoms from any system, the most prominent clinical presentation typically includes facial swelling and upper and lower airway symptoms, such as cough, hoarse voice, rhinitis, dyspnea, and wheezing, as seen in our patient. In the case of the 10-year-old patient, the main symptoms were from the gastrointestinal system and included abdominal pain, nausea, vomiting, and diarrhea.[15] Two deaths have been reported in the literature in adults with comorbidities.[7,16] One report also associates anaphylaxis after consuming flour contaminated with mites with exercise.[17] In one case from the Netherlands, both the father (48 years old) and the daughter (18 years old) reacted after consumption of a beignet contaminated with mites, and both were found to be sensitized.[4] OMA has also been described after inhalation of cooking vapors from a commercial pancake mix contaminated with the house dust mite *D. farinae*.[18]

The diagnosis of OMA is based on the clinical history, particularly the consumption of contaminated food and the development of symptoms. Some risk factors are associated with the presence of OMA, such as a history of atopic disease, mite allergy, aspirin, and nonsteroidal anti-inflammatory drug hypersensitivity, ingestion of foods contaminated with mites, wheat flour, and ingestion of more than 1 mg of mite allergen (>500 mites per gram of flour).[3]

Established criteria, including typical symptoms, support the diagnosis of OMA after consuming wheat-based foods, a history of atopy (e.g., rhinitis, asthma, eczema), positive skin tests to mite allergens, negative tests to commercial wheat extracts, tolerance to uncontaminated wheat products, and ideally, detection of mites or mite allergens in the suspect flour. In this case, the diagnosis was based on the temporal link to pizza ingestion, mite sensitization, a negative wheat skin prick test, and the successful reintroduction of wheat-based foods. Unfortunately, the original food store declined to provide a sample of the flour, preventing direct confirmation of contamination. It remains essential to exclude genuine wheat allergy and wheat-dependent, exercise-induced anaphylaxis, both of which were deemed unlikely given the negative ω-5 gliadin test and absence of intense exertion.

Prevention is the mainstay of treatment for OMA. Storing flour in sealed containers at low temperatures, preferably in the refrigerator, reduces mite contamination. Additional measures include the use of air purifiers, regular cleaning, and the application of acaricides.[3,19]

While sublingual immunotherapy is effective for respiratory mite allergies, its role in OMA is unclear. We noted improvement with subcutaneous immunotherapy, though symptom resolution followed avoidance strategies, limiting conclusions.[20] Further studies are needed to assess the role of immunotherapy in OMA.

A key limitation of this case is the lack of direct testing of the original flour for mite contamination with direct microscopy or ELISA, since the company has refused to provide our team with part of the contaminated dough. Nevertheless, the diagnosis remains clinically plausible based on the patient's sensitization, symptom timing, and tolerance to other ingredients. Similar cases have been reported in the literature, where diagnosis was based solely on clinical criteria.[3,7]

Conclusion

Oral mite anaphylaxis (OMA) is a potentially severe allergic reaction triggered by ingestion of food prepared with mite-contaminated flour. It is often underdiagnosed,

especially in temperate climates with lower awareness. Clinical presentations vary from mild symptoms to full anaphylaxis. Proper storage of flour—particularly in sealed containers at low temperatures—is crucial to minimize the risk of recurrence. Maintaining a high index of suspicion and clinical vigilance is essential for the timely recognition and management of these conditions.

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Author's Contribution

All authors contributed equally to this article.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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