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POLLEN ALLERGY AT THE TABLE: CLINICAL PROFILE OF A PATIENT WITH ALLERGIC RHINITIS AND POLLEN-FOOD ALLERGY SYNDROME

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Abstract

Background: Allergic rhinitis (AR) is highly prevalent worldwide and is rarely an isolated condition. Sensitization to inhalant pollen allergens can lead to unexpected clinical consequences beyond the respiratory tract, one of which is pollen-food allergy syndrome (PFAS), also known as oral allergy syndrome. Patients with AR sensitized to birch, ragweed, or grass pollen frequently develop hypersensitivity reactions to a wide range of plant-derived foods.

Objective: This review aims to summarize current knowledge on the epidemiology, pathogenesis, clinical manifestations, diagnosis, and management of patients with comorbid allergic rhinitis and pollen-food allergy syndrome.

Methods: A narrative review of the literature was conducted, focusing on recent advances in molecular allergology and clinical management of PFAS.

Results: PFAS occurs in 23–76% of patients with AR, depending on geographic region and pollen sensitization profile. The underlying mechanism is cross-reactivity between heat-labile pollen proteins (e.g., Bet v 1, profilins) and homologous proteins in fresh fruits, vegetables, and nuts. Clinically, PFAS typically presents as oral allergy syndrome (itching, burning, and angioedema of the lips, tongue, and palate) occurring within minutes of consuming raw trigger foods. Importantly, most cross-reactive allergens are heat-labile, allowing patients to tolerate cooked or processed forms of the same foods. Although symptoms are usually mild and confined to the oropharynx, systemic reactions, including anaphylaxis, occur in 1.7–8.7% of cases. Diagnosis is based on a thorough history, skin prick testing, specific IgE measurement, and component-resolved diagnostics to differentiate labile from stable allergens. Management includes control of underlying AR with intranasal corticosteroids and antihistamines, dietary avoidance of raw trigger foods, patient education on recognizing systemic symptoms, and, in selected cases, allergen immunotherapy (AIT) directed against the primary pollen sensitization.

Conclusion: PFAS is a common but underdiagnosed comorbidity of allergic rhinitis. Clinicians should routinely screen AR patients for food-induced oral symptoms. A molecular approach to diagnosis enables accurate risk stratification and personalized dietary advice. Allergen immunotherapy for pollen AR may reduce PFAS symptoms, although further studies are needed.

Keywords: Allergic rhinitis; pollen-food allergy syndrome; oral allergy syndrome; Bet v 1; cross-reactivity; pollen; food allergy; component-resolved diagnostics

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1. Introduction

Allergic rhinitis (AR) is one of the most common allergic diseases worldwide, affecting up to 40% of the population. Despite being often perceived as a trivial condition, AR significantly impairs quality of life, sleep, and work performance [1]. However, the clinical picture of AR frequently extends beyond nasal symptoms (sneezing, rhinorrhea, nasal congestion, and itching). Many patients, particularly those sensitized to tree and grass pollens, experience unusual reactions after consuming fresh fruits, vegetables, or nuts, manifested by itching and swelling in the oral cavity.

This condition, known as pollen-food allergy syndrome (PFAS) or oral allergy syndrome (OAS), is not a primary food allergy but rather a cross-reactive phenomenon. The immune system mistakenly recognizes certain food proteins as the previously encountered pollen allergens [2,3]. Understanding the mechanisms of this cross-reactivity is essential for effective diagnosis and treatment, as the management of PFAS differs fundamentally from that of primary food allergy.

In this article, we discuss why a patient with allergic rhinitis may develop intolerance to apples, peaches, celery, or soy, and how to establish an optimal management strategy.

2. Epidemiology and Clinical Significance

The prevalence of PFAS depends directly on geography and the spectrum of airborne pollen allergens. In regions where birch pollen predominates (Northern and Central Europe, parts of North America, Russia), up to 70% of sensitized patients may experience symptoms of cross-reactive food allergy [2,4]. According to published data, 23–76% of patients with AR across different countries report symptoms of allergy to at least one plant-derived food product [5].

For a long time, PFAS was considered a condition of adults. However, recent studies indicate that it is also quite common in pediatric practice, especially in children with seasonal allergic rhinitis [6]. Notably, more than half of patients with PFAS have intolerance to more than two types of foods, creating significant dietary restrictions [7].

3. Pathogenesis: The "Deceived" Immune System

The key mechanism of PFAS is cross-reactivity. A patient with AR produces IgE antibodies against specific pollen proteins (e.g., the major birch allergen Bet v 1). Many plant-derived foods contain homologous proteins whose amino acid sequences match pollen proteins by 70–85% [3,8].

When a raw apple or peach is consumed, the immune system recognizes Mal d 1 or Pru av 1 as the "disguise" of the pollen allergen. Mast cells "armed" with IgE in the oral and pharyngeal mucosa immediately release histamine, causing a localized allergic reaction [9].

Major groups of panallergens:

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1. PR-10 proteins (Bet v 1 homologs): The most clinically significant group. Characteristic of birch and related trees (alder, hornbeam). They cause reactions to apples, cherries, apricots, peaches, pears, celery, carrots, soy, and peanuts [2,10].

2. Profilins (Bet v 2 homologs): Highly conserved proteins present in all plants. They cause milder reactions to citrus fruits, tomatoes, melons, and bananas [3].

3. Non-specific lipid transfer proteins (nsLTP): More common in southern regions (Mediterranean area), associated with sensitization to mugwort and ragweed pollen. They cause reactions to peaches, corn, lettuce, and grapes. Unlike PR-10 proteins, LTPs are heat-stable and resistant to digestion, carrying a higher risk of systemic reactions [4,11].

4. Clinical Manifestations: From Mild Discomfort to Systemic Reactions

The classic presentation of PFAS is oral allergy syndrome, which develops within minutes (rarely 1–2 hours) after consuming the trigger food in its raw form [12].

Typical symptoms:

- Itching, burning, and tingling of the lips, tongue, and soft palate
- Angioedema of the lips and oral mucosa
- Sensation of "scratching" in the throat, less commonly itching in the ears
- In some cases, rhinorrhea or sneezing as an exacerbation of AR symptoms [5,13]

Key features and risks:

Heat lability: Most cross-reactive allergens are heat-labile. Patients can safely eat baked apples or cooked carrots but cannot tolerate these foods raw. Heating denatures PR-10 and profilin proteins [2,14].

Seasonality: PFAS symptoms worsen during the flowering season of the causative plant (spring for birch, summer–autumn for weeds) because the immune system is in a state of hyperreactivity at the peak of pollen exposure [15].

Systemic reactions: Although most cases are limited to the oropharynx, generalization can occur. Urticaria, bronchospasm, and even anaphylactic shock have been described in the literature, with a frequency of 1.7–8.7% among PFAS patients [4,16]. Nuts, soy, and celery are particularly dangerous in this regard, especially when associated with sensitization to stable allergens like nsLTP or seed storage proteins.

5. Diagnosis: What to Look For?

Diagnosis of PFAS is based on a combination of allergy history and specific tests. Unfortunately, the condition often remains undiagnosed because patients do not always associate mild oral itching after an apple with spring hay fever [17].

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Clinical rule of thumb:

Every patient diagnosed with allergic rhinitis (especially seasonal) should be asked: *"Do you experience itching or swelling of your lips or mouth after eating fresh fruits or vegetables?"*

Laboratory confirmation:

Skin prick testing (SPT) and specific IgE: May show sensitization to pollen and food products. However, there is a risk of false-positive results due to cross-reactivity without clinical relevance. The use of fresh foods (prick-by-prick) increases diagnostic accuracy [2,18].

Component-resolved diagnostics (CRD): The gold standard. CRD allows differentiation between sensitization to heat-labile (PR-10, profilin) and heat-stable (nsLTP, seed storage proteins) allergens, which is critically important for predicting the risk of severe reactions [3,11].

Oral food challenges: Performed with caution in a hospital setting due to the risk of systemic reactions. The use of raw foods is mandatory, as cooked foods may yield false-negative results [19].

6. Management

Therapy for a patient with comorbid AR and PFAS involves several levels of intervention.

6.1. Control of Allergic Rhinitis (Basic Therapy)

Adequate treatment of AR reduces the overall allergic background. First-line drugs include intranasal glucocorticosteroids and second-generation oral antihistamines [1]. Reducing systemic inflammation may also decrease the severity of PFAS symptoms.

6.2. Dietary Strategy

Avoidance in raw form: Patients should exclude raw trigger fruits and vegetables. However, complete elimination of all potentially cross-reactive foods is usually not necessary and may lead to unnecessary nutritional restrictions [20].

Thermal processing: Baking, boiling, or steaming "dangerous" foods is recommended, as heating denatures PR-10 and profilin proteins [2,14].

Peeling: Allergen concentration in the peel can be up to 7 times higher than in the pulp. Peeling an apple may sometimes avoid a reaction [21].

Tolerance testing: Under medical guidance, patients can safely determine which forms of a food (e.g., apple sauce, baked apple, peeled apple) are tolerated.

6.3. Allergen Immunotherapy (AIT)

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Allergen-specific immunotherapy with pollen extracts (e.g., birch) is the only disease-modifying treatment for AR. There is accumulating evidence that AIT may reduce sensitivity to cross-reactive food allergens, although this effect is variable and requires further investigation [22,23]. A recent meta-analysis suggested that AIT for pollen-induced AR can reduce PFAS symptoms in a subset of patients, but response is not universal [24]. Nevertheless, treating the underlying cause (AR) should be the priority.

6.4. Emergency Management

Patients must be educated to recognize warning signs of systemic progression (throat tightness, difficulty breathing, hoarseness, dizziness, generalized urticaria). They should carry second-generation antihistamines and, if there is a history of systemic reaction, an epinephrine auto-injector [16,25].

7. Special Considerations

7.1. Geographic Variability

In Northern Europe and North America, birch pollen (Bet v 1) is the dominant sensitizer, and PFAS typically presents as mild oral symptoms to Rosaceae fruits (apple, pear, peach) and Apiaceae vegetables (carrot, celery). In Southern Europe, nsLTP sensitization (Pru p 3 from peach) predominates, leading to more severe, systemic reactions to a wider range of plant foods, including those that are heat-stable [4,11]. In Central Europe, grass pollen (Phl p 1, Phl p 5, and profilin) is a common cause of PFAS, typically with mild symptoms to tomato, melon, and citrus [3].

7.2. Pediatric Population

In children, PFAS should be suspected when seasonal AR is accompanied by complaints of mouth itching during fruit consumption. Unlike primary food allergy, PFAS rarely causes anaphylaxis in children, but severe reactions have been reported [6,26].

8. Conclusion

Allergic rhinitis and pollen-food allergy syndrome are links in the same chain. The presence of AR significantly increases the risk of reactions to plant-based foods, requiring active screening for PFAS by clinicians. Understanding the molecular mechanisms of cross-reactivity allows not only accurate diagnosis but also the development of a safe diet that does not deprive the patient of essential nutrients (thanks to thermal processing). Given the high prevalence of birch sensitization across Europe, North America, and parts of Asia, every practicing allergist should be aware of the "fruit and vegetable masks" of pollen allergy. Control of nasal symptoms together with food literacy is the key to a high quality of life for these patients.

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