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CLINICAL AND MOLECULAR FEATURES OF POLLEN-FOOD ALLERGY SYNDROME: DIAGNOSTIC CHALLENGES AND PERSONALIZED MANAGEMENT

Department of Phthisiatry and Pulmonology,
Microbiology, Virology, and Immunology
Assistant **Tashmatova G.A.**
Andijan State Medical Institute.
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Abstract Background: Pollen-food allergy syndrome (PFAS) is an IgE-mediated allergic condition caused by immunological cross-reactivity between inhaled pollen allergens and structurally similar proteins present in plant-derived foods. The clinical presentation ranges from mild oral symptoms to potentially severe systemic allergic reactions.

Objective: To analyze the molecular mechanisms, clinical manifestations and contemporary diagnostic approaches to pollen-food allergy syndrome, with particular emphasis on component-resolved diagnostics and individualized patient management.

Materials and Methods: A narrative review of scientific literature was conducted using publications concerning pollen sensitization, cross-reactive food allergens, clinical manifestations and diagnostic approaches. Particular attention was given to allergen families, including PR-10 proteins, profilins and nonspecific lipid transfer proteins (nsLTPs), as well as recent international recommendations.

Results: PFAS is primarily associated with sensitization to airborne pollen followed by immunological cross-reactivity with homologous food proteins. PR-10 proteins and profilins are generally associated with predominantly localized oral manifestations, whereas stable allergen families, particularly nsLTPs, may be associated with systemic reactions. Clinical assessment combined with skin prick testing, prick-to-prick testing, serum-specific IgE and molecular allergen analysis improves diagnostic accuracy. Recognition of individual sensitization profiles is important for assessing reaction severity and preventing unnecessary dietary restrictions.

Conclusion: PFAS requires an individualized diagnostic and therapeutic approach. Integration of clinical history with molecular allergology may improve identification of clinically relevant allergens, facilitate risk assessment and optimize dietary recommendations.

Keywords: pollen-food allergy syndrome, cross-reactivity, pollen sensitization, PR-10 proteins, profilin, lipid transfer proteins, molecular allergology, component-resolved diagnostics.

Introduction

Pollen-food allergy syndrome is an increasingly recognized clinical condition among individuals with seasonal allergic rhinitis and sensitization to environmental pollen allergens. It develops when the immune system recognizes structurally similar proteins present in pollen and certain plant-derived foods.Unlike primary food allergy, in which sensitization usually occurs through the gastrointestinal tract or skin, PFAS generally develops following primary sensitization to inhaled pollen allergens. Subsequent consumption of foods containing homologous proteins triggers an immediate IgE-mediated allergic response[1].

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The most characteristic manifestations include itching and tingling of the lips, tongue, palate and pharyngeal mucosa. These symptoms typically develop within minutes after consuming raw fruits, vegetables, nuts or legumes.Although the majority of reactions are localized, some patients may develop urticaria, angioedema, gastrointestinal symptoms, respiratory manifestations and, rarely, anaphylaxis.The clinical significance of PFAS is related to the molecular characteristics of the involved allergens. Differences in protein stability, resistance to heat treatment and susceptibility to gastrointestinal digestion influence the severity and distribution of allergic manifestations.

Recent developments in molecular allergology have improved understanding of these mechanisms and provided opportunities for more precise diagnosis.The objective of this review is to examine the clinical and molecular characteristics of PFAS and discuss modern diagnostic and management strategies.

Materials and Methods

This article presents a narrative review of published scientific literature concerning pollen-food allergy syndrome.The analysis was based on the supplied manuscript by G.A. Tashmatova and additional international publications addressing the immunological mechanisms, epidemiological characteristics, molecular allergen profiles and clinical management of PFAS.The following aspects were examined:Mechanisms of pollen-food cross-reactivity.Clinical significance of major allergen protein families.Regional and environmental factors influencing sensitization.Clinical manifestations and assessment of reaction severity.Skin testing and serum-specific IgE determination.Component-resolved diagnostics.Individualized dietary management and prevention of systemic reactions.Recent international recommendations on the diagnosis and management of IgE-mediated food allergy were also considered.

Results and Discussion

Immunological mechanisms of pollen-food cross-reactivity. The pathogenesis of PFAS is based on an IgE-mediated immune response involving cross-reactive allergens.During primary sensitization, inhaled pollen proteins stimulate the production of allergen-specific IgE antibodies. These antibodies bind to high-affinity FcεRI receptors on mast cells and basophils.When a sensitized individual subsequently consumes plant-derived foods containing structurally similar proteins, these allergens may bind and cross-link surface-bound IgE antibodies.This interaction activates mast cells and leads to the release of histamine, leukotrienes, prostaglandins and other inflammatory mediators.The resulting clinical manifestations depend on the allergen involved, its molecular stability, the amount consumed and individual patient characteristics.

An important feature of PFAS is that different molecular allergen families produce different clinical patterns.Major molecular allergen families [3]Several plant protein families are particularly important in the development of pollen-food cross-reactivity.PR-10 proteinsPR-10 proteins are structurally related to Bet v 1, the major allergen of birch pollen.Cross-reactive proteins have been identified in various plant foods, including apples, hazelnuts, cherries, peaches and soybeans.These proteins are generally sensitive to heat treatment and gastrointestinal digestion. Consequently, patients frequently experience symptoms after consuming raw foods while tolerating adequately cooked or processed products[2-4].Typical clinical manifestations include oral itching, lip swelling and mild pharyngeal discomfort.Profilins are highly conserved proteins found in many plants and pollens. Their widespread distribution explains the possibility of cross-reactivity between different pollen species and a broad range of fruits and vegetables.

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Profilin sensitization may be associated with oral allergy symptoms following the consumption of melon, watermelon, banana, tomato and other plant-derived foods.Profilins are generally heat-labile, and reactions are often limited to the oral cavity.Nonspecific lipid transfer proteins

Nonspecific lipid transfer proteins represent an important allergen family because of their relative resistance to heat and digestive enzymes[5].These proteins occur in peaches, apples, nuts and other plant foods.Unlike reactions predominantly associated with PR-10 proteins, nsLTP-mediated allergy may produce generalized urticaria, angioedema, gastrointestinal symptoms and anaphylaxis.The presence of nsLTP sensitization should therefore be considered during clinical assessment of patients reporting systemic reactions to plant foods.

Other allergen families

Additional proteins, including gibberellin-regulated proteins (GRPs), may contribute to cross-reactive allergic reactions.Their molecular stability and distribution in different plant foods may influence clinical manifestations and the probability of systemic reactions.Relationship between pollen sensitization and food allergyThe relationship between inhaled pollen allergens and food-derived proteins is influenced by molecular similarity and geographical distribution of pollen species[9].Birch pollen is a well-recognized sensitizing source in many European regions. Its major allergen, Bet v 1, shares structural similarities with PR-10 proteins found in apples, hazelnuts and other plant foods.Alder pollen may also contribute to cross-reactive sensitization because its major allergen, Aln g 1, demonstrates substantial homology with Bet v 1.Grass pollen sensitization may be associated with reactions to several plant foods through profilin-related cross-reactivity.Mugwort pollen is another clinically important sensitizing source, particularly in relation to certain vegetables, spices and nsLTP-containing foods.Regional differences in pollen exposure, climate, vegetation and seasonal distribution contribute to variations in PFAS prevalence and clinical presentation.These observations highlight the importance of considering local environmental allergen exposure when evaluating patients with suspected PFAS Clinical manifestations and severity assessment[8] The clinical presentation of PFAS is heterogeneous.

The most characteristic manifestation is oral allergy syndrome, which develops shortly after ingestion of the responsible food. Common symptoms include:

Itching and tingling of the lips.

Swelling of the tongue and oral mucosa.

Itching of the palate and pharynx.

Mild lip and facial edema.

In some patients, symptoms extend beyond the oral cavity.

Respiratory manifestations: nasal congestion, rhinorrhea, sneezing, coughing, wheezing and dyspnea.Cutaneous manifestations: urticaria, generalized itching and angioedema.Gastrointestinal manifestations: abdominal discomfort, nausea, vomiting and diarrhea.Systemic reactions: generalized allergic reactions and, rarely, anaphylaxis.The clinical severity is influenced by the molecular characteristics of the responsible allergen and additional factors, including the quantity of food consumed, exercise, alcohol intake and other individual cofactors.A history of respiratory symptoms or asthma should receive particular attention because uncontrolled asthma may complicate the management of allergic reactions. Modern diagnostic approaches

The diagnosis of PFAS requires a comprehensive evaluation of the patient's clinical history and immunological sensitization profile.Clinical historyA detailed history should establish:

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The type of pollen associated with seasonal allergic symptoms.The specific food responsible for the reaction.The time interval between food consumption and symptom development.Whether symptoms occur after raw, cooked or processed food.The severity and duration of previous reactions.The presence of additional risk factors and cofactors.A characteristic history of oral symptoms following raw plant-food consumption in a pollen-sensitized individual may strongly suggest PFAS.Skin prick testing Skin prick testing is commonly used to evaluate IgE-mediated sensitization.However, commercial extracts of certain fruits and vegetables may contain insufficient quantities of unstable allergens.For this reason, the prick-to-prick technique using fresh food may provide additional diagnostic information.Comparison of reactions to raw and appropriately processed food may help identify sensitivity to heat-labile proteins.

Serum-specific IgE Determination of serum-specific IgE against suspected pollen and food allergens provides additional evidence of sensitization.However, positive specific IgE results indicate immunological sensitization and do not independently establish clinically relevant food allergy.Results must always be interpreted in the context of the patient's clinical history. Component-resolved diagnostics Component-resolved diagnostics represent an important development in modern allergy practice.Unlike conventional testing with whole-allergen extracts, molecular diagnostics identify IgE antibodies directed against individual allergenic proteins.

This approach may help distinguish between sensitization to PR-10 proteins, profilins and nsLTPs.

For example, detection of Bet v 1-related sensitization may support a birch-associated cross-reactive pattern, whereas sensitization to nsLTP components may indicate a different clinical risk profile[7-9].Molecular testing can therefore contribute to:

Identification of the primary sensitizing allergen.

Differentiation between cross-reactive and potentially primary food allergy.

Assessment of the possibility of systemic reactions.

Selection of appropriate dietary recommendations.

Reduction of unnecessary food avoidance.

Nevertheless, molecular testing should complement rather than replace clinical evaluation.

Individualized management and prevention The principal management strategy for PFAS involves identifying clinically relevant trigger foods and providing individualized dietary recommendations[5].Patients with reactions limited to raw foods may tolerate appropriately cooked or processed forms, particularly when the responsible allergens are heat-labile.However, tolerance must be assessed individually. Cooking does not reliably eliminate the allergenicity of heat-stable proteins such as many nsLTPs. Unnecessary elimination of multiple fruits and vegetables should be avoided because excessive dietary restrictions may reduce nutritional diversity and quality of life.Patients should receive information about recognizing early allergic symptoms and avoiding known clinically relevant triggers.Individuals with a history of systemic reactions or anaphylaxis require an individualized emergency management plan. When indicated, prescription of an epinephrine auto-injector and education regarding its use are essential.Oral food challenges may be considered when the diagnosis remains uncertain, but they should be performed only under appropriate medical supervision[6]. Clinical significance and future research directions

Despite increased recognition of PFAS, several important clinical questions remain unresolved.

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The relationship between individual molecular sensitization profiles and the severity of clinical manifestations requires further investigation.Additional research is needed to determine how geographical differences in pollen exposure, environmental changes and seasonal allergen concentrations influence the development of PFAS.Another important area involves the development of standardized diagnostic algorithms that integrate clinical history, molecular testing and individual risk assessment.Future studies may also investigate the role of environmental factors, immune regulation and allergen-specific immunotherapy in patients with pollen-associated food reactions.

Conclusion

Pollen-food allergy syndrome is a clinically heterogeneous IgE-mediated condition resulting from immunological cross-reactivity between airborne pollen allergens and homologous proteins present in plant-derived foods.

The clinical presentation is determined by the molecular characteristics of the involved allergens. PR-10 proteins and profilins are frequently associated with localized oral manifestations, whereas stable allergen families, particularly nsLTPs, may be associated with systemic allergic reactions.

A combination of detailed clinical history, skin prick testing, serum-specific IgE determination and component-resolved diagnostics provides a more comprehensive approach to diagnosis.

Individualized dietary recommendations, identification of patients at risk of severe reactions and appropriate emergency planning are essential components of clinical management.

Further research into molecular sensitization patterns and regional environmental factors may contribute to improved prevention and personalized treatment of PFAS.

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