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POLLEN-FOOD ALLERGY PATIENTS EXPERIENCE

Toshmatova G.T.

Assistant Andijan State Medical Institute.

Department of Phthisiatry and Pulmonology, Microbiology, Virology, and Immunology

Abstract. In sensitized individuals, when a particular pollen allergen interacts with a related food allergen, it triggers pollen-food allergy syndrome (PFAS). While oral symptoms are the most common manifestation, some individuals may experience additional symptoms such as nasal congestion, skin reactions, respiratory issues, or even life-threatening anaphylactic shock. The development of pollen-food allergy syndrome (PFAS) involves mast cells in the oral mucosa, which are sensitized to specific pollen antigens bound to IgE. These mast cells then cross-react with food antigens, triggering a localized type I allergic response when ingesting foods containing protein antigens (panallergens) that share structural similarities with pollen antigens. The prevalence of PFAS is influenced by regional variations in pollen type and abundance. Due to the widespread presence of various pollen allergens, such as alder and grass, linked to this condition, research on per- and polyfluoroalkyl substances (PFAS) is a global endeavor. This review examines the latest findings on the pathophysiology, epidemiology, and clinical manifestations of PFAS. We also highlight crucial research gaps that must be addressed to improve patient outcomes for those affected by PFAS.

Key words: Pollen-food allergy, ethiology, pathogenesis, cross-reaction, clinical types

Sensitivity to pollen antigens is the characteristic of pollen-food allergy syndrome (PFAS), which results in edema of the lips and mouth mucosa, numbness, and itching minutes after eating the offending food. Rarely, respiratory symptoms, rhinitis, and gastrointestinal problems may appear. However, severe cases are rarely accompanied by systemic symptoms such as anaphylaxis, skin rashes, vomiting, and wheezing. PFAS have become more common in recent years, and physicians frequently encounter them in the course of their job. Moreover, oral allergy syndrome (OAS), an immediate-type allergy that is IgE-dependent and results in symptoms for the oral mucosa in reaction to a dietary antigen, includes PFAS as a subtype. Amlot et al. first proposed the notion of OAS in 1987, pointing out that oral symptoms that appear soon after eating foods that test positive for prick tests can be a sign of more serious symptoms.
As a result, while not all PFAS symptoms are OAS symptoms, the majority are. The Japanese Guidelines for Food Allergy 2020 divide food allergies into four clinical types: neonatal and infantile gastrointestinal allergies, special type food allergies (such as food-dependent exercise-induced anaphylaxis [FDEIA] and oral allergy syndrome [OAS]), and immediate-type food allergies (such as urticaria and anaphylaxis). 1. In people with immediate-type food allergies, the gastrointestinal tract becomes sensitized to proteins that are difficult to digest. Because the triggering and sensitizing antigens are the same, ingesting the sensitizing allergen results in allergy symptoms. The patient becomes sensitive to allergens inhaled, such as pollen antigens, in the case of PFAS, a special type of food allergy, and similar molecules in plant-based foods react with these allergens to cause the allergy.

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When people who are sensitive to certain pollen allergens come into touch with the matching specific food antigens, they develop symptoms of PFAS. [2] These reactions depend on how similar the primary allergen components are to one another. The pathophysiology of PFAS is often mediated by pan-allergens, which are commonly present in a broad range of foods and pollens. Plant survival depends on basic proteins known as pan-allergens; as a result of the genes encoding these proteins being conserved over time, a variety of plants have developed cross-reactivity and allergic reactions. Pathogen-related (PR) protein-10/Bet v 1-related proteins, lipid transfer proteins (LTPs), profilin, and other pan-allergens are examples of antigen families. The majority of PFAS symptoms are caused by type I allergic reactions that are exclusive to the oral mucosa. Patients experience swelling and itching of the tongue, lips, oral mucosa, and pharyngeal mucosa within an hour of consuming the trigger food. The symptoms that PFAS can cause include those related to the nose (nasal obstruction, rhinorrhea, sneezing), eyes (tearing, conjunctival hyperemia, itching), skin (facial swelling, itching, generalized rash), digestive system (abdominal pain, vomiting, diarrhea), respiratory system (hoarseness, wheezing, dyspnea), and, in rare cases, anaphylaxis. [2] Symptoms usually appear when raw food is consumed. Since soy milk, a processed soybean product, is often not heat treated for more than 30 minutes during processing, consuming it raw can result in anaphylaxis. This preserves Gly m4's antigenicity. Bean sprouts, a vegetable made from sprouted soybeans, should also be handled carefully because few cooking methods call for heat treatment for more than 30 minutes. Systemic symptoms are influenced by the allergens' stability in the associated diet and pollen. There are many different allergen components in food, and each one has a different tolerance for heat and digestion.

In a similar vein, Pur p 3 in peaches is LTP and likely to cause systemic symptoms, but Pur p 1 in peaches is PR-10 and unlikely to do so. Furthermore, GRP, which is also very stable like LTP, is Pur p 7 in peaches. 30, 32LTP is present in a number of pollens, such as mugwort (Art v 3) and ragweed (Amb a 6). Sensitization to specific pollen-derived LTPs and GRPs may cause cross-reactivity among homologous proteins of food origin and increase symptom severity.

The number of patients with allergic rhinitis is increasing worldwide. In Europe, climate change has led to an increase in the amount and duration of birch pollen dispersal, increasing the prevalence of birch pollen sensitization.[4] In Japan, allergic rhinitis prevalence was 29.8%, 39.4%, and 49.2% in 1998, 2008, and 2019, respectively, and it continues to increase, with nearly half (49.5%) of children aged 10–19 years developing hay fever in 2019. Although cedar pollen is the most predominant pollen antigen in Japan, non-cedar pollen allergies are also on the rise, contributing to an increased PFAS prevalence. In Japan, the birch's range is restricted to all of Hokkaido and the higher-elevation areas north of central Japan. Therefore, several patients with PFAS associated with birch pollinosis are found in Hokkaido. Notably, PFAS also occurs in areas where birch pollen dispersal is rarely observed. In a previous survey of 6824 outpatients with PFAS in Fukui Prefecture, where birch pollen is rarely dispersed, we found that PFAS prevalence was 10.8%, with *Cucurbitaceae* and *Rosaceae* foods. Serum antigen-specific IgE antibody positivity was significantly higher in the group of patients with birch exposure (PFAS group vs. controls, 31.7% vs. 8.6%), alder (17.7% vs. 2.0%) and timothy grass (31.7% vs. 19.9%). Furthermore, the main allergenic components of each pollen, Bet v1 (birch, 29.3% vs. 9.3%), Aln g1 (alder, 26.5% vs. 7.3%) and Phl p1 (timothy grass, 24.1% vs. 13.9%) were also significantly

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higher in patients with PFAS. This suggests that alder and timothy grass pollens are established causative pollens in Fukui Prefecture.[4] The alder belongs to the same family as birch and is common in deciduous forests widely distributed throughout Japan from Hokkaido to northern Kyushu. Bet v 1, the major allergen of birch pollen, and Aln g 1, the major allergen of alder pollen, have more than 80% protein homology; thus, PFAS is observed upon alder pollen sensitization even in areas without birch pollen. Cedar pollen is associated with tomatoes. However, other pollinosis, such as alder pollinosis, often overlap with cedar pollinosis, and because tomatoes have pan-allergens, such as Sola l 4 (PR-10) and Sola l 1 (profilin), PFAS may be caused by pollens other than cedar.[5] In the United States, Gupta *et al.* reported a prevalence of food allergy of 10.8% in a survey of over 40,000 U.S. adults. In a multicenter study at 9 sites in southern Europe, 167 (20.5%) of 815 patients with seasonal allergic rhinitis aged 10–60 years had PFAS, with prevalence varying significantly among centers, from 7.5% to 41.4%.Thus, PFAS prevalence varies widely owing to regional differences in pollen sensitization patterns, influenced by geographical conditions, such as pollen type and quantity. n PFAS, a history of hypersensitivity symptoms in the oral and pharyngeal mucosa upon ingestion of certain foods is essential in making a diagnosis. In several cases, PFAS is diagnosable from the clinical course. A high index of suspicion is needed if complaints are caused by fruits (such as apples, melons, and kiwis), legumes (such as soybeans and bean sprouts), and vegetables (such as carrots and celery). Typically, symptoms appear only with raw foods and foods not thoroughly cooked through, within minutes after consumption, and are limited to oral lesions.[6] Although additional testing is not always required in patients with seasonal allergic rhinitis who report characteristic symptoms to typical raw food triggers, it is important to confirm sensitization to the specific pollen associated with the causative food. To definitively identify the causative food, a positive prick test result or detection of food-specific IgE in the serum is used. Regarding the prick test, commercial food extracts are not suitable as prick test antigens because the antigen is altered. The prick-to-prick test is performed by introducing a prick test needle into unprocessed food and then directly into the patient's skin.[7] If the prick test result is positive for raw fruits and vegetables but negative for cooked fruits and vegetables, PFAS can be diagnosed. The prick test provides rapid results with high sensitivity and specificity.[8] For several allergens, skin tests have been considered to have higher diagnostic sensitivity and specificity than serum-specific IgE tests.[8] However, because PFAS induces symptoms through cross-reactivity between pollen and food antigens, a negative result may be obtained if the causative antigen is present in trace amounts. The diagnosis can usually be made with a detailed clinical history, serum-specific IgE, and prick test. However, an oral challenge test may be performed if the history and diagnostic tests are inconclusive, or the patient's diet is unnecessarily restricted. Caution should be exercised in patients with a history of severe anaphylactic reactions to exceedingly small amounts of allergen exposure. PFAS is caused by cross-reactivity between pollen and food antigens and can occur in children and adults of all ages. An understanding that symptoms are mostly confined to the oral mucosa, but occasionally, respiratory, gastrointestinal, skin, or anaphylactic manifestations can be induced is important for its management. The most crucial factor in diagnosing PFAS is having a thorough medical history; skin prick tests and serum-specific IgE are sufficient tools. In theory, eating items that cause oral symptoms should be avoided in order to prevent developing PFAS. However, because pan-allergens create PFAS, once the condition starts, the number of foods that elicit allergy reactions increases significantly, and it may be difficult to eliminate the antigen. The etiology of PFAS is made worse by the different allergenic ingredients present in meals and pollen

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antigens. Therefore, having a thorough grasp of PFAS is crucial for both diagnosing and counseling patients.

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