



## Birch-Related Pollen Food Allergy Syndrome Presenting as Long-Term Fruit and Nut Avoidance in an Adolescent: Diagnostic Clarification Using Component-Resolved Diagnostics

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### ABSTRACT

Pollen Food Allergy Syndrome (PFAS), also referred to as oral allergy syndrome, is a common form of secondary IgE-mediated food allergy resulting from cross-reactivity between inhaled pollen allergens and homologous proteins present in plant-derived foods. Pollen-food allergy syndrome (PFAS) is characterized by sensitization to pollen antigens, resulting in itching, numbness, and mucosal edema of the lips and mouth within minutes of ingesting the causative food.

**Keywords :** Pollen Food Allergy Syndrome (PFAS), Component-resolved diagnostics (CRD) Rhino conjunctivitis, Anaphylaxis.

### INTRODUCTION

Pollen-food allergy syndrome (PFAS) is characterized by sensitization to pollen antigens, resulting in itching, numbness, and mucosal edema of the lips and mouth within minutes of ingesting the causative food.(1)

Occasionally, respiratory symptoms, rhinitis, and gastrointestinal involvement may occur. However, though rarely, severe cases are accompanied by systemic manifestations, such as wheezing, vomiting, skin rashes, and anaphylaxis (2).

PFAS incidence has increased recently, and it is being frequently encountered in daily medical practice. Moreover, PFAS is a subtype of oral allergy syndrome (OAS), an IgE-dependent immediate-type allergy wherein a food antigen causes oral mucosal symptoms.(3)

Pollen Food Allergy Syndrome (PFAS), also referred to as oral allergy syndrome, frequently occurs in individuals sensitized to birch pollen, particularly to the major allergen Bet v 1, which belongs to the pathogenesis-related protein-10 (PR-10) family. These proteins are widely distributed across pollens, fruits, vegetables, and certain nuts, leading to immunological cross-recognition by IgE antibodies in sensitized individuals (4).

According to the European Academy of Allergy and Clinical Immunology (EAACI) molecular allergology framework, PFAS represents a classic example of secondary food allergy caused by cross-reactive pan-allergens, in contrast to primary food allergy driven by stable storage proteins (5).

PR-10 proteins are thermolabile and rapidly degraded by digestion, which explains why clinical manifestations are typically limited to localized oral symptoms and systemic reactions are uncommon (6,7).

The development of component-resolved diagnostics (CRD) has greatly enhanced the ability to differentiate true primary food allergy from pollen-related cross-reactivity, allowing clinicians to stratify the risk of severe reactions and provide more accurate dietary counselling (8).



We present a case of birch-related PFAS in an adolescent from Northern California who had avoided fruits and nuts since childhood because of presumed risk of anaphylaxis, in whom molecular allergen diagnostics clarified the underlying mechanism and guided appropriate manage.

### Case Presentation

A 17-year-old male residing in Northern California presented for evaluation of suspected food allergy and long-standing avoidance of fruits and nuts. The patient reported that since early childhood he experienced immediate oral symptoms after ingestion of certain fruits, particularly apple and strawberry, and occasionally pineapple. These reactions consisted of itching of the tongue, itching of the hard and soft palate, mild throat discomfort, and transient oral irritation. At times these symptoms were associated with nasal itching, sneezing, rhinorrhea, and occasional ocular itching with watering of the eyes.

The symptoms typically developed within minutes of ingestion of the implicated foods and resolved spontaneously without medical treatment. Because of these reactions, the patient's parents discontinued these foods from his diet during childhood due to concerns about potential progression to severe food allergy or anaphylaxis.

The patient also reported a remote history of a possible allergic reaction to a nut during childhood, although he was unable to recall the specific nut or the details of the reaction. Since that time he had maintained a nut-free diet as a precautionary measure. Importantly, there had been no documented episodes of systemic allergic reactions, including no breathing difficulty, wheezing, stridor, hypotension, oxygen desaturation, or hospital admissions following food ingestion. None of the previous reactions required emergency treatment, nebulization therapy, or epinephrine administration. There was therefore no clinical history suggestive of anaphylaxis.

The patient reported tolerance to several other common allergenic foods, including peanuts, seafood, milk, eggs, and wheat, without any adverse reactions. His past medical history was notable for mild atopic dermatitis localized to the cubital fossae, which had been well controlled with topical therapy. Family history was significant for seasonal allergic rhinitis in his mother, who required intermittent intranasal corticosteroid therapy during pollen seasons.

In addition to the food-related symptoms, the patient described recurrent seasonal nasal and ocular symptoms occurring annually between January and March, characterized by sneezing, nasal itching, rhinorrhea, and itchy watery eyes. During this period he occasionally experienced mild episodic wheezing, although these respiratory symptoms were infrequent and never required hospitalization or maintenance inhaled therapy. Outside of this seasonal window, he remained completely asymptomatic during the rest of the year. This pattern suggested seasonal allergic rhinoconjunctivitis related to tree pollen exposure (9).

Physical examination during the clinic visit was unremarkable. No active eczema lesions were present and respiratory examination was normal. Laboratory evaluation demonstrated a total serum IgE level of 23 kU/L, which was within the normal range. Because of the coexistence of seasonal respiratory allergy symptoms and oral reactions to fruits and nuts, pollen food allergy syndrome was suspected, and component-resolved diagnostics (CRD) was performed to clarify the sensitization profile.



Tree Pollen				
Acacia (बबूल)	••••	Aca m		< 0.10
Tree of Heaven (कल्पवृक्ष)	••••	All a		< 0.10
Alder (एल्डर)	•	Aln g 1	PR-10	4.14
	•	Aln g 4	Polcalcin	< 0.10
Silver birch (सिल्वर बर्च पेड़)	•	Bet v 1	PR-10	4.30
	•	Bet v 2	Profilin	< 0.10
	•	Bet v 6	Isoflavon Reductase	< 0.10
Paper mulberry (कागज शहतूत का पेड़)	••••	Bro pa		< 0.10
Hazel pollen (हेज़ल)	••••	Cor a_pollen		0.39
	•	Cor a 1.0103	PR-10	6.61
Sugi (सुगी का पेड़)	•	Cry j 1	Pectate Lyase	< 0.10
Cypress (सरसु के पेड़)	•	Cup a 1	Pectate Lyase	< 0.10
	••••	Cup s		< 0.10
Beech (बीच)	•	Fag s 1	PR-10	2.88
Ash (ऐश)	••••	Fra e		0.13
	•	Fra e 1	Ole e 1 Family	< 0.10
Walnut pollen (अखरोट)	••••	Jug r_pollen		< 0.10
Mountain cedar (पर्वत देवदार पेड़)	••••	Jun a		< 0.10
Mulberry (शहतूत का पेड़)	••••	Mor r		< 0.10
Olive (जैतून)	•	Ole e 1	Ole e 1 Family	< 0.10

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Grass Pollen				
Bermuda grass (बरमुडा घास)	••••	Cyn d		0.10
	•	Cyn d 1	Beta-Expansin	< 0.10
Perennial Ryegrass (राई घास)	•	Lol p 1	Beta-Expansin	0.44
Bahia grass (बाहिया घास)	••••	Pas n		< 0.10
Timothy grass (टिमोथी घास)	•	Phl p 1	Beta-Expansin	0.57
	•	Phl p 2	Expansin	0.61
	•	Phl p 5.0101	Grass Group 5/6	< 0.10
	•	Phl p 6	Grass Group 5/6	< 0.10
	•	Phl p 7	Polcalcin	< 0.10
	•	Phl p 12	Profilin	< 0.10
Common reed (अम ईख घास)	••••	Phr c		< 0.10
Cultivated rye, Pollen (चाई)	••••	Sec c_pollen		< 0.10



	●	Ole e 9	β-1,3-Glucanase	< 0.10	
Date palm (खजूरा का पेड़)	●	Pho d 2	Profilin	< 0.10	
London plane tree (लंदन प्लेन पेड़)	●	Pla a 1	Plant Invertase	< 0.10	
	●	Pla a 2	Polygalacturonase	< 0.10	
	●	Pla a 3	nsLTP	< 0.10	
Cottonwood (कॉटनवुड)	●●●	Pop n		< 0.10	
Elm (एल्म)	●●●	Ulm c		< 0.10	
<b>Weed Pollen</b>					
Common Pigweed (चौलाई का पेड़)	●●●	Ama r		< 0.10	
Ragweed (रैगवीड)	●●●	Amb a		< 0.10	
	●	Amb a 1	Pectate Lyase	< 0.10	
	●	Amb a 4	Plant Defensin	< 0.10	
Mugwort (मगबौट)	●●●	Art v		< 0.10	
	●	Art v 1	Plant Defensin	< 0.10	
	●	Art v 3	nsLTP	< 0.10	
Hemp (गांजा)	●●●	Can s		< 0.10	
	●	Can s 3	nsLTP	< 0.10	
Lamb's quarter (बथुआ)	●●●	Che a		< 0.10	
	●	Che a 1	Ole e 1 Family	< 0.10	
Annual mercury (एनुअल मर्क्युरी)	●	Mer a 1	Profilin	< 0.10	
Wall pellitory (दीवार पेलिटरी)	●●●	Par j		< 0.10	
	●	Par j 2	nsLTP	< 0.10	
Ribwort (इसबगोल)	●●●	Pla l		< 0.10	
	●	Pla l 1	Ole e 1 Family	< 0.10	
Russian thistle (रूसी थीसल पेड़)	●●●	Sal k		< 0.10	
	●	Sal k 1	Pectin Methylesterase	< 0.10	
Nettle (बिछुआ का पेड़)	●●●	Urt d		< 0.10	

Molecular allergen testing demonstrated sensitization to major grass pollen allergens, including Lol p 1 (0.44 kUA/L), Phl p 1 (0.57 kUA/L), and Phl p 2 (0.61 kUA/L), indicating low-level sensitization to grass pollen allergens that may contribute to seasonal allergic rhinitis during spring or early summer.

More prominently, the patient demonstrated strong sensitization to PR-10 tree pollen allergens, including Aln g 1 (4.14 kUA/L) from alder pollen and Bet v 1 (4.30 kUA/L) from birch pollen, as well as cross-reactive homologues such as Cor a 1.0103 (6.61 kUA/L) from hazel pollen and Fag s 1 (2.88 kUA/L) from beech pollen. This molecular sensitization pattern is characteristic of birch-related tree pollen allergy, in which primary sensitization to Bet v 1 leads to cross-reactivity with homologous PR-10 proteins in related tree pollens (10).



Fruits				
Kiwi (कीवी)	●	Act d 1	Cysteine protease	< 0.10
	●	Act d 2	TLP	< 0.10
	●	Act d 5	Kiwellin	< 0.10
	●	Act d 10	nsLTP	< 0.10
Papaya (पपीता)	●●●●	Car p		< 0.10
Orange (संतरा)	●●●●	Cit s		< 0.10
खरबूज	●	Cuc m 2	Profilin	< 0.10
Fig (अंजीर)	●●●●	Fic c		< 0.10
Strawberry स्ट्रॉबेरी	●	Fra a 1+3	PR-10+LTP	1.18
Apple (सेब)	●	Mal d 1	PR-10	0.60
	●	Mal d 2	TLP	< 0.10
	●	Mal d 3	nsLTP	< 0.10
Mango (अम)	●●●●	Man i		< 0.10
Banana (केला)	●●●●	Mus a		< 0.10
Avocado (एवोकाडो)	●●●●	Pers a		< 0.10
Cherry (चेरी)	●●●●	Pru av		< 0.10
Peach (आड़ू)	●	Pru p 3	nsLTP	< 0.10
Pear (नाशपाती)	●●●●	Pyr c		< 0.10
Blueberry (ब्लूबेरी/नीलबदरी)	●●●●	Vac m		< 0.10
Grapes (अंगूर)	●	Vit v 1	nsLTP	< 0.10

Food allergen component testing revealed PR-10-related sensitization to fruits, including Fra a 1+3 (1.18 kUA/L) from strawberry and Mal d 1 (0.60 kUA/L) from apple, both of which are homologous to Bet v 1. In addition, testing demonstrated Cor a 1.0401 (1.12 kUA/L) from hazelnut, representing the PR-10 allergen component responsible for pollen-related hazelnut reactions. Importantly, there was no evidence of sensitization to hazelnut storage proteins such as Cor a 9 or Cor a 14, which are associated with primary hazelnut allergy and increased risk of systemic reactions (11).

Taken together, the molecular allergen profile demonstrated primary sensitization to PR-10 tree pollen allergens with secondary cross-reactivity to homologous fruit and nut proteins, consistent with birch-related pollen food allergy syndrome. The clinical history of localized oral symptoms without systemic involvement further supported this diagnosis.



Nuts				
Cashew (काजू)		Ana o		< 0.10
		Ana o 2	11S Globulin	< 0.10
		Ana o 3	2S Albumin	< 0.10
Brazil nut (ब्राज़ील अखरोट)		Ber e		< 0.10
		Ber e 1	2S Albumin	< 0.10
Pecan (एक प्रकार का अखरोट)		Car i		< 0.10
Hazelnut (पिंजल फल)		Cor a 1.0401	PR-10	1.12
		Cor a 8	nsLTP	< 0.10
		Cor a 9	11S Globulin	< 0.10
		Cor a 11	7/8S Globulin	< 0.10
		Cor a 14	2S Albumin	< 0.10
Walnut (अखरोट)		Jug r 1	2S Albumin	< 0.10
		Jug r 2	7/8S Globulin	< 0.10
		Jug r 3	nsLTP	< 0.10
		Jug r 4	11S Globulin	< 0.10
		Jug r 6	7/8S Globulin	< 0.10
Macadamia (मैकाडामिया)		Mac i 2S Albumin	2S Albumin	< 0.10
		Mac inte		< 0.10
Pistachio (पिस्ता)		Pis v 1	2S Albumin	< 0.10
		Pis v 2	11S Globulin subunit	< 0.10
		Pis v 3	7/8S Globulin	< 0.10
Almond (बादाम)		Pru du		< 0.10

The patient and family were counselled regarding the pathophysiology of PFAS, including the fact that PR-10 allergens are heat-labile and easily degraded during digestion, which explains why symptoms are typically limited to the oral mucosa and why cooked forms of many fruits are often tolerated (6).

The relatively low risk of severe systemic reactions associated with PR-10 sensitization was explained, and the patient was informed that lifelong strict avoidance of fruits and nuts was not medically necessary. Gradual reintroduction of tolerated foods, particularly in cooked or processed forms, was discussed as a potential strategy.

Management of the patient's seasonal allergic rhinoconjunctivitis was recommended in accordance with guideline-based therapy, including intranasal corticosteroids and oral antihistamines during the pollen season (9).

Because the patient demonstrated clinically relevant sensitization to birch-related tree pollens with seasonal respiratory symptoms, the option of allergen immunotherapy targeting tree pollen allergens was discussed as a potential disease-modifying treatment. Evidence suggests that pollen immunotherapy may improve pollen-related respiratory symptoms and may reduce associated PFAS symptoms in some patients (12).

The patient was also educated regarding recognition of symptoms suggestive of anaphylaxis, although the overall risk was considered low based on the molecular allergen profile and clinical history.

## Discussion

This case illustrates several important aspects of pollen food allergy syndrome and the clinical value of molecular allergy diagnostics. PFAS is now recognized as one of the most common forms of food allergy in adolescents and adults with pollen sensitization, particularly in individuals sensitized to birch pollen and related tree pollens (4,7).



Sensitization to Bet v 1, the major birch pollen allergen, can lead to IgE cross-reactivity with structurally similar PR-10 proteins present in numerous fruits and nuts, including apple, hazelnut, strawberry, peach, carrot, and celery (6,10).

A defining characteristic of PR-10 allergens is their instability to heat and digestion, which explains why reactions are usually limited to oral itching, tingling, or mild throat irritation shortly after ingestion of raw foods. These symptoms typically resolve quickly and rarely progress to systemic reactions (6).

This contrasts with primary food allergy caused by storage proteins, which are resistant to heat and digestion and therefore more likely to cause systemic reactions or anaphylaxis (8).

A key diagnostic challenge in clinical practice is distinguishing pollen-related cross-reactivity from primary food allergy, as traditional extract-based allergy tests cannot identify the specific allergenic molecules responsible for sensitization. Component-resolved diagnostics allow precise identification of the allergen components involved and can therefore provide important information regarding clinical risk assessment (5,8).

In the context of hazelnut allergy, for example, sensitization to Cor a 1 is typically associated with birch pollen cross-reactivity and mild oral symptoms, whereas sensitization to Cor a 9 or Cor a 14, which are stable seed storage proteins, is associated with true hazelnut allergy and higher risk of systemic reactions (11).

In this patient, the presence of isolated Cor a 1 sensitization without storage protein sensitization confirmed that the hazelnut reaction pattern was consistent with PFAS rather than primary nut allergy.

Another important aspect of this case is the impact of misinterpretation of mild oral allergy symptoms as severe food allergy, which led to long-term avoidance of multiple foods. Unnecessary dietary restrictions can have significant consequences, including reduced dietary diversity, nutritional imbalance, and impaired quality of life, particularly in adolescents (7).

Accurate diagnosis and counselling are therefore essential to prevent excessive dietary limitations.

The patient's seasonal rhinoconjunctivitis occurring during late winter and early spring corresponds to the typical pollination period of tree pollens such as alder and birch, which are known to trigger allergic rhinitis in sensitized individuals (6). The molecular allergen profile in this case supports the concept that pollen sensitization was the primary driver of the patient's allergic disease, with secondary food reactions resulting from cross-reactive PR-10 allergens.

Finally, this case highlights the clinical importance of integrating detailed clinical history, seasonal symptom patterns, and molecular diagnostic testing in the evaluation of suspected food allergy. Such an approach enables clinicians to differentiate between primary and secondary food allergies, provide accurate risk assessment, and guide appropriate management strategies.

#### **ICD-10 Diagnostic Codes**

Condition	ICD-10 Code
Allergic rhinitis due to pollen	J30.1
Other allergic rhinitis	J30.9
Food allergy	T78.1
Oral allergy syndrome / PFAS	T78.1XXA
Atopic dermatitis	L20.9
Mild intermittent asthma	J45.20

#### **conclusion**

Pollen food allergy syndrome is caused by cross-reactivity of pollen and food antigens and can occur in all persons. Symptoms are mostly confined to the oral mucosa, but occasionally, respiratory, gastrointestinal, skin, or anaphylactic manifestations can be induced. The most important aspect for PFAS diagnosis, with skin prick tests and serum-specific IgE are adequately helpful. To avoid developing PFAS, consumption of foods that induce oral symptoms must be avoided. PFAS is caused by pan-allergens. Once the disease develops, the number of foods that induce allergic symptoms increases drastically, and antigen elimination may not be easily achievable. In several cases, heating the causative food makes it tolerable for ingestion, but caution should be exercised in the case of PFAS caused by soy milk, which is mainly composed of Gly m 4 (soybeans) and heat-resistant LTPs and GRPs.



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