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Opinion Article

Pollen Food Allergy Syndrome in Japanese Children and Adolescents: Risk Factors and Pollen Sensitisation

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DESCRIPTION

Pollen Food Allergy Syndrome (PFAS), which is categorised as a Class 2 food allergy, is brought on by cross reactivity with pollen antigens. Bet v 1 homologs, commonly referred to as pathogenesis-related Protein type 10 (PR-10), and profilin proteins are the main allergens of PFAS. The oral cavity is the only place where PFAS symptoms, also known as Oral Allergy Syndrome (OAS), manifest since they are frequently broken down by heat or enzymatic processing. According to recent reports, systemic symptoms are brought on by the cross-reactivity of pan-allergens like nonspecific lipid transfer protein, Lipid Transfer Protein (TLP), and Gibberellin Regulated Protein (GRP). Moreover, profilin and PR-10 have also been linked to systemic symptoms.

While the majority of PFAS patients suffer OAS symptoms, up to 3% of patients can develop systemic symptoms without oral symptoms, and 1.7% can develop anaphylactic symptoms. Birch tree pollen is the sole source of sensitization for Bet v 1 homologs (birch and alder, among others). In contrast, profilin sensitises a range of pollens, including those from birch trees, grasses (such as timothy and orchard grass), and weeds in the Asteraceae family (e.g., ragweed and mugwort). Because of the kind of pollen and its impact on the amount of pollen in the air, it is known that the prevalence of PFAS varies by area. The reported prevalence is 4.7%-48% in kids with allergic disorders. In Japan, Japanese cedar pollen is the primary cause of seasonal allergy rhinitis.

Among pollen-sensitive Japanese children and adolescents, this study is the first to investigate the risk of producing PFAS and the relationship between pollen-specific IgE levels and PFAS development. The prevalence of PFAS was 20.5% among kids and teenagers with any type of pollen sensitization, 8.5% among those aged 3-6, 20% among those aged 7-12, and 36.3% among those aged 13-18. The incidence of PFAS in pollen-sensitive preschoolers in Japan has never been documented before. Age and alder sensitization were risk factors for the development of PFAS. It has been shown that higher pollen-specific IgE levels promote the production of PFAS.

Among patients sensitised to only Japanese cedar and those sensitized to Japanese cedar, alder, ragweed, and orchard grass, the OR for the prevalence of PFAS was determined to be 36.83 (95% Cl 8.93-151.83, P 0:001). The criterion for PFAS positive was 2.54 UA/mL (ROC curve 0.79, sensitivity 78.9%, and specificity 70.9%); alder was most strongly related with the formation of PFAS. Birch is a significant source of PFAS, however in Japan, it only grows in northern regions like Hokkaido. Alder, a tree that belongs to the same family as birches, is common in regions south of Honshu.

In Japan, the correlation coefficient between birch and alder is very high, demonstrating that both birch and alder pollen specific IgE antibodies are produced. In this investigation, alder-specific IgE was quantified. The main contributors to pollinosis in Japan are Japanese cedar, ragweed (Asteraceae), and orchard grass (Poaceae), which are all found throughout the country. Except for Aln g 1, which is more closely related to PFAS since it is related to PR-10, alder-specific IgE levels were assessed.

PFAS are developed in preschoolers, and 8.5% of those with any pollen sensitization had PFAS. In this study group of Japanese children and adolescents with any pollen sensitization, PFAS risk factors included older age, a diagnosis of allergic rhinitis, alder sensitization, greater pollen-specific IgE levels, and increasing frequency of pollen sensitizations. IgE levels particular to alders can forecast the emergence of PFAS.

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