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ORIGINAL ARTICLE

Approach to children with IgE-mediated food allergy with a focus on oral allergy syndrome

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Purpose: To assess the prevalence of food allergy, with a focus on oral allergy syndrome (OAS) in a population of children and to investigate relevant allergen sensitization associated with these adverse reactions.
Methods: This study involved 1,660 children (aged 4 to 13 years) experiencing seasonal allergy symptoms, who were enrolled in the 2015 prospective Seongnam Atopy Project (SAP 2015) in a South Korean municipality. Parents completed a structured questionnaire to assess children with OAS, collecting information on the duration, severity, and factors related to symptoms. Skin prick tests (n=498) and blood sampling (n=464) were performed to measure allergic sensitizations, total eosinophil counts, and levels both total immunoglobulin E (IgE) and birch-specific IgE.
Results: The prevalence of OAS among the children enrolled in this study was 4.4% (95% confidence interval, 3.7%–5.1%). The most common symptom was mouth itching (n=106, 65.0%), and the primary food allergen associated with symptoms was kiwi (n=48, 29.5%). Peanut sensitization (14.8% vs. 57.1%, $P<0.001$) and sensitization to other nut products (15.6% vs. 47.6%, $P=0.001$) were linked to a higher incidence of systemic reactions.
Conclusion: The prevalence of 4.4% underscores the significant health impact of OAS, especially in children experiencing food allergy-related symptoms. Notably, common allergens, such as kiwi, and the potential for additional systemic reactions associated with this condition highlight the importance of raising awareness. (*Allergy Asthma Respir Dis 2024;12:78-84*)

Keywords: Child, Food allergy, Oral allergy syndrome

INTRODUCTION

Food allergy, which is an immune response caused by antigens included in food, manifests itself in diverse conditions. Food allergy is known to affect as many as 3%–4% of adults.[1] In children, reported estimates are higher ranging up to 7%.[1,2] The reported prevalence of food allergy is increasing in Western countries and also in Asia including Korea, probably due to rapid changes in eating habits and environments.[3] Cow's milk, hen's egg, and wheat are common allergens in young children, often outgrown, while peanut, tree nuts, and seafood allergies are more likely to continue.[2] Food allergy caused by immune reactions to food is typically IgE-mediated. The conditions range from isolated oropharyngeal symptoms ("oral allergy syndrome" [OAS]) to anaphylaxis.[4]

Contact of food with orolabial mucosa may cause immediate symptoms as itching, numbness, swelling of mouth, tongue, and lips. These localized IgE-mediated reactions are termed OAS, a type of food allergy characterized by usually mild symptoms in the oral cavity triggered by the consumption of causative foods.[5] Since OAS is closely related with set of symptoms resulting from cross reactions caused by pollen, it is also termed as pollen-food syndrome.[6] OAS is a hypersensitivity reaction to food allergens due to prior sensitization to highly homologous plant inhalant allergens,[7] such as birch and oak pollen.

Due to regional variations in food allergy prevalence and rapidly changing dietary patterns, there is a lack of data on the prevalence and primary causes of food allergy in school-aged children in Korea.[8] Moreover, limited studies have focused on OAS and sensitization patterns among Korean elementary school-aged children. The study aimed to evaluate the prevalence of food allergy, with a

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focus on OAS, in a population of children experiencing seasonal allergy symptoms. Additionally, we investigated correlations between allergen sensitizations and serum markers for diagnosing OAS.

MATERIALS AND METHODS

1. Subjects

This cross-sectional study was conducted in Korea from June through July of 2015 (Seongnam Atopy Project 2015).[9] Participants were recruited from the general pediatric population of a regional community. Children (4–12 years-old) from 3 kindergartens and 6 elementary schools in Seongnam took part in the study. We distributed structured questionnaires to the parents and the questionnaire was self-administrated. Approximately 4,111 children and their caregivers were invited to participate, and questionnaires were distributed. Participants who returned incorrectly completed questionnaires with missing values or failed to provide written consent were excluded from the analysis, resulting in 3,704 properly completed questionnaires. Subsequently, 1,660 children who reported experiencing seasonal allergy symptoms were included in the study. The study was approved by the Institutional Review Board (IRB) of the CHA Bundang Medical Center (IRB No. 2015-05-075). All parents or guardians provided written informed consent for participation.

2. Definitions

We used a structured questionnaire for assessing children with food allergy and OAS, with diagnoses based on a detailed history. Children answering 'Yes' to symptoms such as rhinorrhea, nasal obstruction, sneezing, and other reactions to implicated foods were suspected of food allergy. Those answering 'Yes' to mouth/ear itch, swelling of lips, foreign body sensation, tightness of throat, and other symptoms were suspicious of OAS. Systemic reactions, including dyspnea, abdominal pain, nausea/vomiting, and hypotension, were also assessed.[10,11] Subjects who reported any reactions to food were also asked about the causable food involved. Open questions in the questionnaire were used to identify specific foods that they thought caused their allergy.[7] Onset and characteristics of symptoms were also reviewed. Foods were classified as fruits (kiwi, apple, pear, watermelon, peach, apricot, cherry, and prune), vegetables (celery, tomato, potato, soy, and carrot), nuts (hazelnut, walnut, almond, and pine nut), or peanuts. Patients with itching tingling, stinging, numbness or swelling of lips, tongue or throat at the site of food contact after biting chewing or swallowing plausible foods were diagnosed with OAS.

3. Hematologic profile and specific IgE measurement

We performed blood sampling to measure white blood cell count, total eosinophil count (TEC), serum-specific birch IgE, and total IgE levels (ImmunoCAP System, Thermo Fisher, Uppsala, Sweden). Skin prick tests were conducted in participants with consent to assess sensitization to allergens. Twenty-two common food and aeroallergens using extracts and control solutions from Lofarma (Milan, Italy) consisted of standard panel of common allergens including aeroallergens, house dust mite and cat were used.[10] The processes were performed on the forearm by an experienced technician according to international guidelines. Subjects were classified as atopic if a positive reaction (wheal diameter $\geq$3 mm) occurred in response to 1 or more allergens.

4. Classification of respondents

Subjects who performed skin prick tests were classified into 1 of 3 groups. The OAS+ group consisted of subjects with OAS symptoms, and with positive results to pollen allergens in skin prick tests. The OAS- group consisted of subjects without OAS symptoms, but with positive results to pollen allergens in skin prick tests. The control group consisted of subjects without related symptoms negative skin prick tests results.

5. Statistics

Statistical analysis was performed using IBM SPSS Statistics ver. 23.0 (IBM Co., Armonk, NY, USA). Differences in categorical variables were tested using the chi-square test. For normally distributed results, comparisons were analyzed by the analysis of variance, and different modalities by *post hoc* analysis. In order to investigate differences in the prevalence of OAS and tree pollen sensitization according to age, we performed the chi-square test for trend (linear by linear association). We divided the participants into 2 groups by age (children aged $\leq$9 years; and >9 years) and compared the age difference between groups according to tree pollen sensitizations. We assessed correlations of nonnormal distribution data such as specific IgE and total IgE levels by using Spearman rank correlation coefficient. Comparisons between subgroups were performed using Dunn test. The power of different immunological parameters in differentiating the OAS+ and

OAS- groups were compared using receiver operating characteristic (ROC) curves. The pROC program within R software was used to compare the areas under the curves (AUCs). A *P*-value below 0.05 was considered statistically significant.

RESULTS

1. Prevalence of OAS and its changes according to different age groups

A total of 190 participants (5.0%) responded that they had meaningful allergic symptoms after certain food consumption, and 28 (0.7%) had systemic symptoms (Fig. 1). Typical signs/symptoms of OAS were checked in 163 of 3,704 children who were finally enrolled (4.4%; 95% confidence interval [CI], 3.7–5.1). OAS increased with age, but the trend was statistically insignificant (linear to linear $P=0.05$). However, when we compared the prevalence of OAS between the 2 groups by age, the difference was significantly higher in older children: 5.2% (97 of 1,855) in children aged >9 years; and 3.6% (66/1,849) in children aged ≤9 years ($P=0.014$). We also performed skin prick tests in 498 subjects (13.5%); a total of 276 (55.4%) were sensitive to 1 or more allergens, and 99 (19.9%) were sensitive to tree allergens. Sensitization to tree pollen increased with age (linear to linear $P<0.001$). Among the 498 patients who completed the questionnaire and performed the skin prick test, we classified 23 (4.6%) as OAS+, 76 (15.3%) as OAS-, and 77 (15.5%) as controls.

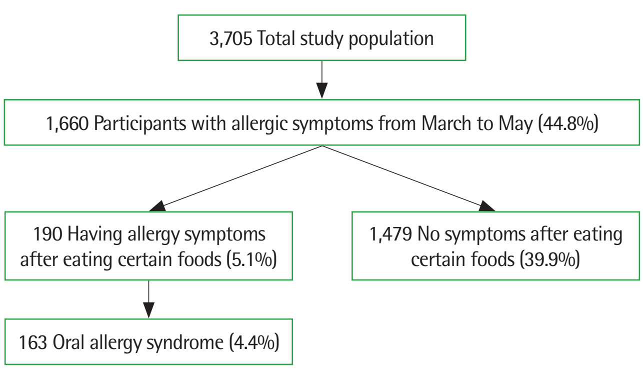

Fig. 1. Flow of participants.

2. Food sensitivity and symptoms of OAS

The mean number of food allergens related with symptoms was 1.6±1.1, and common foods causing OAS were kiwi (n=78), tomato, peach, peanut, and apple in order or frequency (Fig. 2A). The most prevalent symptom was itchy mouth (n=104), followed by foreign body sensation of throat (n=55) and swelling of the lips (n=52) after consumption of certain foods (Fig. 2B). Systemic symptoms were also observed: nausea/vomiting (n=14), dyspnea (n=7), and abdominal pain (n=5). Compared to subjects with only local reactions, those with systemic reactions were sensitized to more allergens (1.5±1.3 vs. 2.2±1.2, $P=0.031$) and had a greater number of symptoms (1.7±1.2 vs. 6.0±2.9, $P<0.001$). Compared to subjects sensitized to other allergens, those sensitized to peanuts (14.8% vs. 57.1%, $P<0.001$) and other nut products (15.6% vs. 47.6%, $P=0.001$) had a higher incidence of systemic reactions.

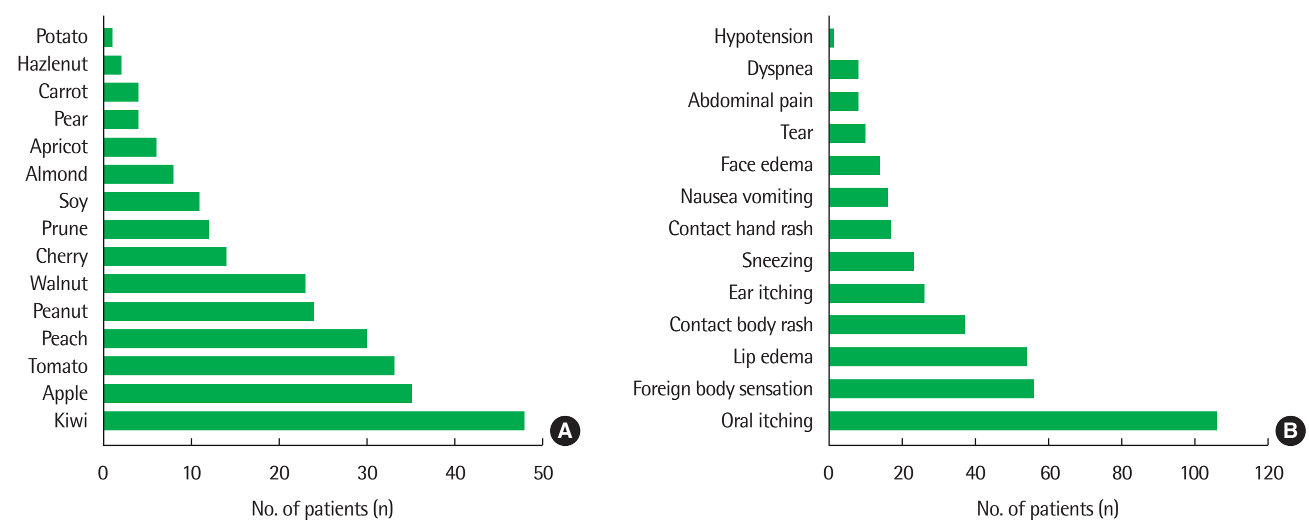

Fig. 2. (A) Number of patients who had oral allergy syndrome (OAS) in response to different plant foods. The greatest frequencies were for kiwi, tomato, and peach. (B) Symptoms of patients with OAS.

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3. Comparison among OAS+, OAS–, and controls

Clinical characteristics (based on questionnaire results) and skin prick test results of subjects in the 3 groups are presented in Table 1. The 3 groups had similar sex distribution ($P=0.077$) and body mass indices ($P=0.403$). The prevalence of co-morbid rhinoconjunctivitis and atopic dermatitis and of parental allergic rhinitis and atopic dermatitis were greater in the OAS+ group. However, the 3 groups had no significant differences in specific allergen sensitizations according to the skin prick test results.

When comparing the eosinophil count and total IgE results, OAS+ showed significantly higher levels compared to both the OAS- group (eosinophil count $P<0.001$ and total IgE results $P=0.006$) and the controls (eosinophil count $P<0.001$ and total IgE results $P<0.006$). Similar results were observed when comparing the specific IgE results, with OAS+ group showing significantly higher levels compared to both the OAS- group ($P<0.001$) and the controls ($P<0.001$) (Fig. 3).

Table 1. Demographic and clinical characteristic of the OAS+, OAS-, and control groups (N=176)

Characteristic	Control (n=77)	OAS- (n=76)	OAS+ (n=23)	*P*-value
Age (yr)	9.3±1.9	10.0±1.8*	9.8±1.6	0.042
Sex				0.077
Boys	30 (39.0)	41 (53.9)	14 (60.9)	
Girls	47 (61.0)	35 (46.1)	9 (39.1)	
BMI, *z*-score (95% CI)	-0.09 (0.95)	-0.04 (0.96)	-0.34 (0.70)	0.403
Comorbidity				
Asthma	-	4 (5.3)	2 (8.7)	0.546
Rhinoconjunctivitis	-	47 (61.8)	23 (100)†	<0.001
Atopic dermatitis	-	18 (23.7)	12 (52.2)†	0.031
Parental allergy				
Asthma	2 (2.6)	2 (2.6)	1 (4.3)	0.897
Allergic rhinitis	24 (31.2)	45 (59.2)*	19 (82.6)*	<0.001
Atopic dermatitis	5 (6.5)	10 (13.2)*	6 (26.1)*,†	0.036
Skin prick test				
Birch	-	46 (60.5)	17 (73.9)	0.242
Oak	-	52 (68.4)	14 (60.9)	0.501
Elm	-	25 (32.9)	8 (34.8)	0.866
House dust mites	-	58 (76.3)	19 (82.6)	0.525
Plant food allergen	-	13 (17.1)	8 (34.8)	0.069
Animal food allergen	-	21 (27.6)	7 (30.4)	0.794
Allergy sensitization	-	76 (100)	23 (100)	-

Values are presented as mean±standard deviation or number (%) unless otherwise indicated.
OAS, oral allergy syndrome; OAS+, OAS symptoms and positive results to pollen allergens in a skin prick test; OAS-, no OAS symptoms and positive results to pollen allergens in the skin prick test; BMI, body mass index; CI, confidence interval; HC, healthy control.
Comorbidity refers to a co-occurring illness, as defined by the International Study of Asthma and Allergies in Childhood, based on symptoms during the last 12 months.
A positive skin prick test result at least 1 of 22 allergens was defined as atopy. Plant food allergens were apple, peach, kiwi, orange, tomato, strawberry, celery, peanut, wheat, and walnut. Animal food allergens were egg, milk, cod, pork, mussel, and shrimp.
*Significantly different ($P<0.05$) from the control group. †Significantly different ($P<0.05$) from the OAS- group.

4. Diagnostic power of different parameters

We generated ROC curves to assess the relationship between

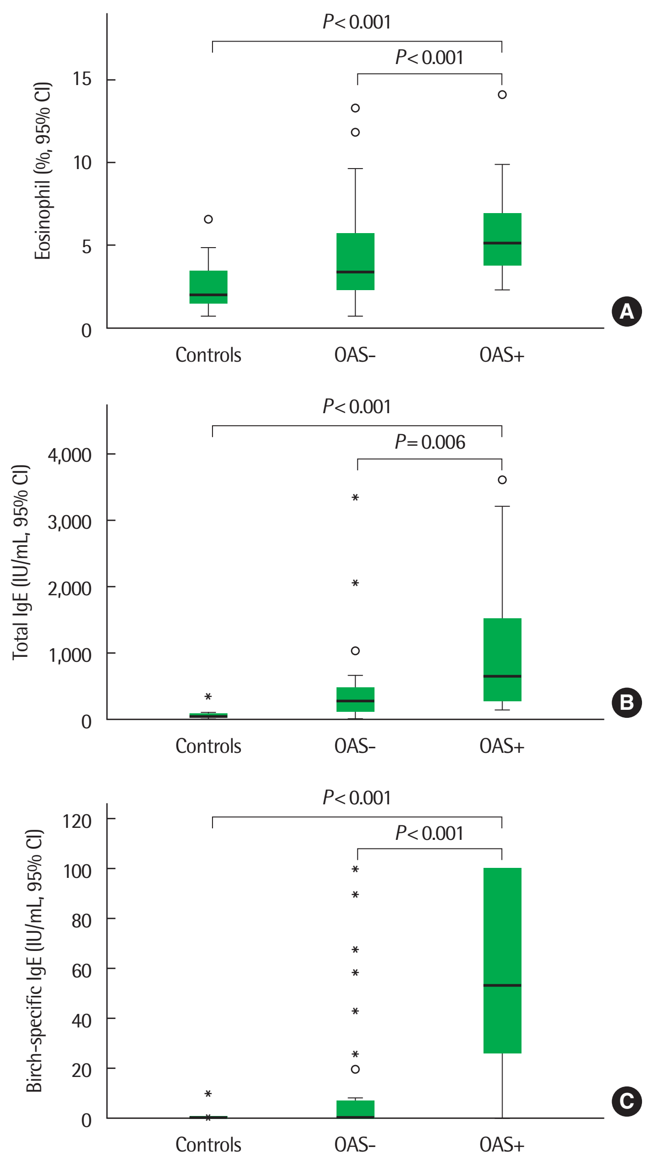

Fig. 3. Comparison of serum biomarker levels from controls, OAS-, and OAS+ subjects. The horizontal bar represents the median and the box length represents the interquartile range. (A) Eosinophil counts. (B) Total IgE. (C) Birch-specific IgE. Outliers are identified by o (1.5–3.0×the interquartile range [IQR]), extreme values by * (>3×IQR). CI, confidence interval; OAS, oral allergy syndrome.

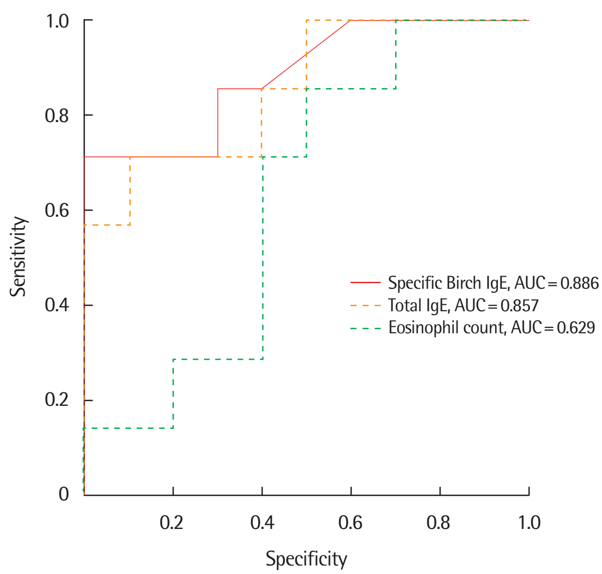

Fig. 4. Receiver operating characteristic curve analyses of the sensitivity and specificity of specific Birch IgE, total IgE, and eosinophil count for the diagnosis of OAS. IgE, immunoglobulin E; OAS, oral allergy syndrome; AUC, area under the curve.

eosinophil counts, total IgE, and birch-specific IgE concerning the diagnosis of OAS. The AUC was calculated to determine the sensitivity and specificity of total IgE for this purpose (Fig. 4). Subsequently, we conducted ROC analysis to evaluate the significance of different serum parameters. The AUCs were highest for birch-specific IgE (0.886; 95% CI, 0.567–0.954) and lowest for TEC (0.629; 95% CI, 0.492–0.897).

DISCUSSION

In this analysis of data, we investigated the prevalence of allergy symptoms after certain food consumption and OAS, and estimated common manifestations. Common food triggers that might be linked with these conditions were also investigated. The prevalence of food allergy symptoms in our study was 5.0% (n = 163), and OAS was 4.4% (n = 28). The most common food allergy symptoms were oral itching, foreign body sensation, and lip edema in order of frequency which was in accordance with previous national surveys.[12] The major allergens responsible were kiwi, followed by peach, and pear.

In a recent review, rates of food allergy were estimated as 5% in children and 3%–4% in adults.[13] The rate of IgE-mediated food allergy has been reported to be higher in children compared to that of adults as in demonstrated by a national cohort study performed in United States.[14] Similar patterns were also reported in studies of Asia, although epidemiological surveys are relatively limited.[15] In a survey conducted in China, prevalence of IgE-mediated food allergy nearly doubled in children less than 24 months over the past decade.[16] Data on food allergy and OAS in Korean children are scarce, given the regional variations in food allergy and the increasing prevalence over the past 2 to 3 decades.[17] However, due to the regional variability in pollen dispersal and the significant association between OAS and pollen sensitization, it is probable that findings from other countries may not accurately represent the situation in our nation. Our increased awareness of these trends prompted an investigation into the incidence, manifestations, and common causes of food allergy in Korean children. Despite geographic variations that might result in lower pollen sensitization and different dietary habits regarding highly allergenic foods in Korea, we found that the incidence of food allergy and OAS in Korean children is comparable to that in Western children.[18-20]

The prevalence of OAS in Europe is 2% to 4.2%,[21] and estimated at 5% among children.[22] A previous study reported that 24% to 48% of children with seasonal allergic rhinitis have experienced symptoms of OAS.[6] In surveys conducted in America and Europe investigating children, the prevalence of OAS was 1%–5%.[23,24] Reported prevalence of OAS according to studies performed in East Asia was 1.7% to 10.2%.[25,26] Differences in sensitization among populations to pollen may affect the prevalence of OAS, and sensitization patterns differ according to regions, age, and dietary exposures.

Subjects with food allergy and OAS underwent assessment using a detailed questionnaire. For those reporting a food allergy or OAS, skin prick testing was conducted to confirm sensitization to the suspected trigger food, addressing discrepancies between self-reports and diagnoses.[27] While a double-blind oral food challenge stands as the standard test for diagnosing the cause of food allergy, it necessitates a hospital environment. A skin prick test was employed as an alternative given the nature of the study. Despite the potential risk of anaphylaxis, skin prick testing is recognized as an effective tool for diagnosing IgE-mediated food allergy.[27]

Interestingly, we found that the most common cause related with allergy symptoms after certain food consumption was kiwi. This may be explained by cross reactions caused by Act d 8 (PR10) and Act d 9 (profilins), which are component allergens shared by both kiwi and birch. Possibility that the major allergen of kiwi may be Act D 8 in Korea children exists, which is a finding similar to

East European children, but not Act d1.[28] However, given the fact that kiwi is the most common cause in temperate zone countries such as Korea where the major allergen offending OAS is not elucidated and symptoms caused by cross-reactivity is not frequently observed, this may be controversial.

We conducted an analysis to investigate the correlation between symptoms of OAS and eosinophil counts, total IgE, and birch-specific IgE. The findings suggest that both total IgE and birch-specific IgE offer robust confirmation of the clinical diagnosis of OAS. This observation may be attributed to the interaction of birch-specific IgE with the PR-10 protein in the primary trigger for OAS which are mostly fruit or vegetables.[26] These pan-allergens, characterized by highly reactive antigens, play a significant role in the progression of OAS pathology due to their substantial structural similarity with molecular structures. Further studies are warranted to confirm the current findings and considering primary sensitizations separately may be necessary.

There are limitations to this study that require caution in interpreting the results. The children in this study were enrolled from elementary schools located in a limited region, and therefore, the findings might not fully represent the pediatric population. Additionally, serum-specific IgE testing was exclusively conducted for birch due to financial constraints. It is important to acknowledge the possibility of potential cross-reactivity and consider that other allergens, not estimated in this study, may be the primary antigens in this condition.

In conclusion, prevalence of OAS was 4.4% in our population of Korean children, and kiwi was the most common cause of OAS. Our analysis of different IgEs indicated that IgE against birch pollen is effective in the diagnosis of OAS. Our study provides epidemiologic information that allergenic food consumption may trigger adverse allergic reaction affecting quality of children's life. Causable foods may be often served in meals in daycare centers or schools which may unexpectedly ingested by children. Therefore, the possibility of adverse reaction caused by foods and the notorious causes should be noticed in perspective of public health.

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REFERENCES

1. Wang J. Management of the patient with multiple food allergies. Curr Allergy Asthma Rep 2010;10:271-7.
2. Jeong K, Lee S. Natural course of IgE-mediated food allergy in children. Clin Exp Pediatr 2023;66:504-11.
3. Lee SC, Kim SR, Park KH, Lee JH, Park JW. Clinical features and culprit food allergens of Korean adult food allergy patients: a cross-sectional single-institute study. Allergy Asthma Immunol Res 2019;11:723-35.
4. Santos AF, Lack G. Food allergy and anaphylaxis in pediatrics: update 2010-2012. Pediatr Allergy Immunol 2012;23:698-706.
5. Turnbull JL, Adams HN, Gorard DA. Review article: the diagnosis and management of food allergy and food intolerances. Aliment Pharmacol Ther 2015;41:3-25.
6. Ludman S, Jafari-Mamaghani M, Ebling R, Fox AT, Lack G, Du Toit G. Pollen food syndrome amongst children with seasonal allergic rhinitis attending allergy clinic. Pediatr Allergy Immunol 2016;27:134-40.
7. Price A, Ramachandran S, Smith GP, Stevenson ML, Pomeranz MK, Cohen DE. Oral allergy syndrome (pollen-food allergy syndrome). Dermatitis 2015;26:78-88.
8. Jeon YH. Pollen-food allergy syndrome in children. Clin Exp Pediatr 2020;63:463-8.
9. Ha EK, Kim JH, Lee SW, Jee HM, Shin YH, Baek HS, et al. Atopic dermatitis: correlation of severity with allergic sensitization and eosinophilia. Allergy Asthma Proc 2020;41:428-35.
10. Lee SJ, Ha EK, Jee HM, Lee KS, Lee SW, Kim MA, et al. Prevalence and risk factors of urticaria with a focus on chronic urticaria in children. Allergy Asthma Immunol Res 2017;9:212-9.
11. Geroldinger-Simic M, Zelniker T, Aberer W, Ebner C, Egger C, Greiderer A, et al. Birch pollen-related food allergy: clinical aspects and the role of allergen-specific IgE and IgG4 antibodies. J Allergy Clin Immunol 2011;127:616-22.e1.
12. Kim MA, Kim DK, Yang HJ, Yoo Y, Ahn Y, Park HS, et al. Pollen-food allergy syndrome in Korean pollinosis patients: a nationwide survey. Allergy Asthma Immunol Res 2018;10:648-61.
13. Sicherer SH, Sampson HA. Food allergy: a review and update on epidemiology, pathogenesis, diagnosis, prevention, and management. J Allergy Clin Immunol 2018;141:41-58.
14. Liu AH, Jaramillo R, Sicherer SH, Wood RA, Bock SA, Burks AW, et al. National prevalence and risk factors for food allergy and relationship to asthma: results from the national health and nutrition examination survey 2005-2006. J Allergy Clin Immunol 2010;126:798-806.e13.
15. Lee AJ, Thalayasingam M, Lee BW. Food allergy in Asia: how does it compare? Asia Pac Allergy 2013;3:3-14.
16. Ben-Shoshan M, Turnbull E, Clarke A. Food allergy: temporal trends and determinants. Curr Allergy Asthma Rep 2012;12:346-72.
17. Song KB, Park MJ, Choi EJ, Jung S, Yoon J, Cho HJ, et al. Food allergy in early childhood increases the risk of oral allergy syndrome in schoolchildren: a birth cohort study. Pediatr Allergy Immunol 2022;33:e13786.

18. Sekerkova A, Polackova M. Detection of bet v1, bet v2 and bet v4 specific ige antibodies in the sera of children and adult patients allergic to birch pollen: evaluation of different ige reactivity profiles depending on age and local sensitization. Int Arch Allergy Immunol 2011;154:278-85.
19. Kim J, Hahm MI, Lee SY, Kim WK, Chae Y, Park YM, et al. Sensitization to aeroallergens in Korean children: a population-based study in 2010. J Korean Med Sci 2011;26:1165-72.
20. Moverare R, Westritschnig K, Svensson M, Hayek B, Bende M, Pauli G, et al. Different Ige reactivity profiles in birch pollen-sensitive patients from six european populations revealed by recombinant allergens: an imprint of local sensitization. Int Arch Allergy Immunol 2002;128:325-35.
21. Skypala IJ, Bull S, Deegan K, Gruffydd-Jones K, Holmes S, Small I, et al. The prevalence of pfs and prevalence and characteristics of reported food allergy; a survey of uk adults aged 18-75 incorporating a validated pfs diagnostic questionnaire. Clin Exp Allergy 2013;43:928-40.
22. Brown CE, Katelaris CH. The prevalence of the oral allergy syndrome and pollen-food syndrome in an atopic paediatric population in southwest sydney. J Paediatr Child Health 2014;50:795-800.
23. Ma S, Sicherer SH, Nowak-Wegrzyn A. A survey on the management of pollen-food allergy syndrome in allergy practices. J Allergy Clin Immunol 2003;112:784-8.
24. Rance F, Kanny G, Dutau G, Moneret-Vautrin DA. Food hypersensitivity in children: clinical aspects and distribution of allergens. Pediatr Allergy Immunol 1999;10:33-8.
25. Kim JH, Kim SH, Park HW, Cho SH, Chang YS. Oral allergy syndrome in birch pollen-sensitized patients from a Korean university hospital. J Korean Med Sci 2018;33:e218.
26. Osawa Y, Ito Y, Takahashi N, Sugimoto C, Kohno Y, Mori S, et al. Epidemiological study of oral allergy syndrome in birch pollen dispersal-free regions. Allergol Int 2020;69:246-52.
27. Longo G, Berti I, Burks AW, Krauss B, Barbi E. IgE-mediated food allergy in children. Lancet 2013;382:1656-64.
28. Le TM, Bublin M, Breiteneder H, Fernandez-Rivas M, Asero R, Ballmer-Weber B, et al. Kiwifruit allergy across europe: clinical manifestation and IgE recognition patterns to kiwifruit allergens. J Allergy Clin Immunol 2013;131:164-71.