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Can food allergy trigger acute pancreatitis? Exploring an unusual etiology

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Abstract
Background: Food allergy (FA) is an immune-mediated hypersensitivity reaction to specific dietary antigens. While FA typically manifests with skin, respiratory, or gastrointestinal symptoms, its role in acute pancreatitis remains unclear. Acute pancreatitis is a potentially life-threatening inflammatory condition with well-known etiologies; however, in up to 30% of cases, no identifiable cause is found, resulting in a diagnosis of idiopathic acute pancreatitis (IAP).
Objective: This prospective study aimed to investigate the potential association between FA and IAP in adult patients.
Material and Methods: Forty-nine adult patients diagnosed with IAP were evaluated. During the acute episode, serum tryptase, total immunoglobulin E (IgE), and eosinophil levels were measured. Eight weeks post-recovery, skin prick tests (SPT) and serum-specific IgE (sIgE) tests were performed for common and suspected food allergens. Patients with positive results underwent further testing, including prick-to-prick testing.
Results: Among 49 patients, eight (16.3%) demonstrated sensitization to food allergens, such as cow's milk, wheat, red meat, banana, plum, and tomatoes. These patients had significantly higher total IgE levels (P = 0.001). No significant differences were found in attack severity or tryptase levels between FA and non-FA groups. One patient experienced recurrence after re-exposure to the identified allergen. SPT alone failed to detect sensitization in several cases, whereas sIgE and prick-to-prick tests improved diagnostic accuracy.
Conclusion: This is the first prospective study to suggest a potential link between FA and IAP. In cases of unexplained pancreatitis, FA should be considered in the differential diagnosis. Multidisciplinary collaboration between gastroenterologists and allergists is recommended for accurate diagnosis and management.

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Introduction

Food allergy (FA) is defined as the immune system developing adverse hypersensitivity reactions to harmless dietary antigens.[1] Food-related allergic reactions typically occur within minutes to a few hours after exposure to food allergens. Symptoms can affect multiple organ systems, primarily the skin, respiratory system, cardiovascular system, and gastrointestinal system.[1,2] The symptoms can range from mild to severe and may lead to anaphylaxis, a life-threatening allergic reaction.

Acute pancreatitis is an inflammatory disease of the pancreas that causes serious morbidity and mortality. There are many causes of acute pancreatitis. The most common causes include obstruction to the bile duct by a stone, alcohol use, dyslipidemias, and genetic factors.[3-5] Despite advances in diagnostic technologies, etiology remains uncertain in approximately 10–30% of patients, and these patients are diagnosed with idiopathic acute pancreatitis (IAP).[6] A diagnostic algorithm for IAP is proposed in the literature, but it must be noted that FA is not mentioned at any stage of the algorithm.[7] Accurate identification of the etiology of pancreatitis and better diagnostic follow-up could reduce the burden on health systems and improve quality of life. In this study, we aimed to investigate the possible presence of FA in etiology of patients diagnosed with IAP who were hospitalized in gastroenterology department of our tertiary hospital.

Materials and Methods

This is a prospective study conducted with adult IAP patients at the gastroenterology clinic of SBU Gülhane Training and Research Hospital. The inclusion criteria were being older than 18 years and having no identifiable etiology of acute pancreatitis. Initially, 60 patients were included in the study. However, 11 patients were excluded because they did not return for follow-up. The study was completed with 49 patients. This human study was approved by Ethics Committee of the Ankara Gulhane Training and Research Hospital, with protocol code of the research being 2022/63, and conducted in accordance with the Declaration of Helsinki. All adult participants provided written informed consent to participate in this study.

Acute pancreatitis was diagnosed and classified based on the revised Atlanta criteria. Diagnosis requires at least two of following: typical abdominal pain, serum pancreatic enzymes were at least three times the upper limit of normal, and characteristic imaging findings.[1,8] Severity was categorized as mild (no organ failure or complications), moderate (transient organ failure or local/systemic complications), or severe (persistent organ failure), the latter associated with high morbidity and mortality.[8] All patients in our study were diagnosed and graded accordingly. No invasive procedures were performed, and no pathological samples were obtained. During the attack phase, none of the patients presented with potential triggers, such as physical exercise or infections.

During acute pancreatitis, complete blood count and serum tryptase levels were assessed. A skin prick test (SPT) could not be performed during this period, but serum-specific immunoglobulin E (sIgE) level was assessed for both aeroallergens and foods, including those consumed prior to the onset of symptoms. After 8 weeks of attack, all patients underwent SPT with common food allergens (cow's milk, egg white/yolk, wheat, soy, peanut, tree nuts, and fish) and suspected culprit foods. Given potential for cross-reactivity, SPT with aeroallergens (tree, grass, weed pollens, mold, house dust mites [HDM]) was also performed.[9]

Results

The median age of patients was 47 years. The youngest patient was aged 18 years, and the oldest was aged 83 years. Of patients, 44.9% (n = 22) were males and 55.1% (n = 27) were females. The mean body mass index (BMI) was calculated as 27.77, and no patients were found to be obese.

During the attack; median eosinophil level was 0.1 (min: 0, max: 0.3); median total IgE level was 40.8 (min: 0.1, max: 1758); mean serum tryptase level was 4.01 ± 1.87, and no patients had elevated tryptase levels (Table 1).

A family history of allergy was present in 26% (n = 13) of patients. Significant food allergen positivity was detected in eight patients following testing for foods consumed prior to pancreatitis episode. Sensitivities included cow's milk (n = 2), wheat flour (n = 2), red meat (n = 1), red meat + wheat flour + tomato (n = 1), banana (n = 1), and plum (n = 1), confirmed by prick-to-prick test after recurrent attacks (Table 2). Among these eight patients, two had a history of other allergic diseases and two had a family history of atopy (defined as drug allergy, FA, and allergic rhinitis or asthma).

Comparison between patients with and without FA showed that FA group was older and had higher eosinophil and tryptase levels, although these differences were not statistically significant. Amylase and lipase levels were also comparable between groups. However, total IgE levels were significantly elevated in FA group (P = 0.001) (Table 3).

Of the total 49 patients, 46 had mild, 2 had moderate, and 1 had severe acute pancreatitis. In subgroup with FA (n = 8), 7 patients had mild and 1 had moderate pancreatitis. A comparison of attack severity between patients with and without FA revealed no statistically significant difference (P = 0.35) (Table 3).

Table 1 Demographic and laboratory features of patients.

Features	Value
Patients (total No.)	49
Gender (male/female)	22/27
Age, median (min-max)	47 (18-83)
Body Mass Index (kg/m^2), median	27, 77
Personal history of allergy, N	8
Family history of allergy, N	13
Eosinophil median (min-max)	0.1 (0.00-0.30)
Tryptase (mean ± SD)	4.01 ± 1.87
Total IgE median (min-max)	40.8 (0.1-1758)

Table 2 Summarized details of cases of positive food allergen-induced acute pancreatitis.

	Age/ gender	Food consumed	Time between food consumption and reaction (hour)	Family history of allergies	Comorbid diseases	Eosinophil value	Specific IgE (sIgE) value	Skin prick test results	Total IgE (IU/mL)	Tryptase
Patient 1	35/ male	Cow's milk	1	No	-	0.1	Cow's milk sIgE: 0.59 ku/L casein sIgE: 0.61 ku/L	Cow's milk prick: negative	578	2.88
Patient 2	66/ male	Beef wheat	2	No	Drug allergy	0	Wheat flour sIgE: 0.39 ku/L beef sIgE: negative	Wheat flour prick: positive	313	7.29
Patient 3	58/ male	Beef	4	No	-	0.1	Beef sIgE: 0.56 ku/L	Beef prick: negative Cow's milk prick: negative	510	6.91
Patient 4	65/ male	Beef egg tomato wheat	3	No	Hypertension Coronary artery disease	0.1	Beef sIgE: 0.47 ku/L Wheat flour sIgE: 0.45 ku/L Tomato sIgE: 0.93 ku/L Egg sIgE: negative	Could not be applied	1758	4.13
Patient 5	23/ male	Cow's milk	1	Yes	-	0.3	Cow's milk sIgE: 0.53 ku/L	Grass mix: positive	161	3.9
Patient 6	21/ male	Chicken meat	3	Yes	Allergic rhinitis asthma	0.1	Banana sIgE: 1.88 ku/L	Could not be applied	644	2.85
Patient 7	47/ female	Beef egg tomato wheat (pizza)	4	No	Hypertension Hypothyroidism	0.2	Beef sIgE: 0.61 ku/L+ egg sIgE: negative wheat flour sIgE: negative	Could not be applied	386	3.17
Patient 8	61/ female	Plum	2	No	Hypertension psoriasis	0.2	-	Mix inhalant prick test: negative Plum prick-to-prick test: positive	1129	7.4

Note: sIgE values above 0.35 inches were considered positive.

Table 3 Comparison of patients with and without food allergies.

	Food allergy positive	Food allergy negative	P value
Age, median (min-max)	47 (21-66)	46 (18-83)	0.415
Eosinophil, mean± SD	0.128 ± 0.09	0.10 ± 0.08	0.414
Tryptase, mean ± SD	4.44 ± 1.88	3.94 ± 1.88	0.51
Total IgE, median (min-max)	510 (161-1758)	36.4 (0,1-1498)	0.001
Amylase, median (min-max)	1093 (131-1960)	712 (38-3713)	0.841
Lipase, median (min-max)	1071 (230-3912)	1467 (15-7611)	0.502
Atlanta Severity score			0.35
Mild (N, %)	39 (95.1%)	7 (87.5%)	
Moderate (N, %)	1 (2.4%)	1 (12.5%)	
Severe (N, %)	1 (2.4%)	-	

All patients were managed with standard treatment protocols for acute pancreatitis, including fluid resuscitation, pain control, and pancreatic rest. No additional treatment was required, and no complications developed during follow-up in any patient.

Discussion

To our knowledge, this is the first prospective study suggesting a possible link between IAP and FA. The key finding is the need to consider FA in the etiology of IAP.

Acute pancreatitis is an inflammatory disease of pancreas. Although many causes have been identified to date, its etiology and pathogenesis are still under investigation. In adults, the most common cause is obstruction to main biliary-pancreatic duct because of gallstones.[5]

Food Allergy is a hypersensitivity reaction triggered by immunological mechanisms following the ingestion of a specific food. Although it is difficult to determine its exact prevalence, its incidence has increased in recent years, affecting approximately 10.8% of adults.[10,11] FA symptoms vary widely, most often involving the skin and mucosa, but may also affect the respiratory, gastrointestinal, and cardiovascular systems.[2] After allergen exposure, symptoms may be single-system or multi-system, including anaphylaxis. Our study highlights clinical heterogeneity with some patients presenting only isolated acute abdominal symptoms, lacking involvement of other systems, especially cutaneous manifestations.

There are case reports in the literature showing that acute pancreatitis was provoked after consumption of mustard, milk, eggs, bananas, codfish, and kiwi fruit.[12-19] Similarly, in our study, we identified significantly positive results in SPT and sIgE tests performed with foods consumed prior to pancreatitis episodes in eight patients (Table 2). The identified food allergens aligned with those commonly reported in the adult population.

Of the eight patients diagnosed with FA, one had a personal history of drug allergy, one had a family history of atopic disease, and one had both personal and familial history of atopic disease (including drug allergy, allergic rhinitis, and asthma). These findings aligned with the literature and supported that, as with other atopic conditions, a personal or family history of atopy is among the strongest risk factors of FA.[10] A pediatric study showed that individuals with a family history of any allergic disease had a 40% higher risk of developing FA, which increased to 80% when two family members were affected.[20] A 2018 review reported a 48-year-old patient who developed acute pancreatitis after kiwi consumption. Initially attributed to alcohol, the second episode after 2 weeks of kiwi intake led to a positive prick test, confirming kiwi sensitivity. The patient also had allergic rhinitis, asthma, and drug allergy.[1]

Based on our current knowledge, mechanism of food-induced acute pancreatitis is still unclear. Although tomography imaging shows normal pancreas in most patients, endoscopic examinations reveal edema in the intestinal wall and duodenal inflammation.[14-16,18] A 2005 study proposed ampullary obstruction, bile reflux, and premature zymogen activation as possible mechanisms of food-induced pancreatitis. In that case, endoscopy showed inflammation of the ampulla of Vater, and pathology revealed widespread mast cell infiltration.[16] A similar mechanism was suggested in a case of pancreatitis linked to milk allergy.[13] Based on these findings, we proposed that, similar to Kounis syndrome, allergenic food consumption could cause edema of the ampulla of Vater, leading to obstruction in bile flow and premature activation of pancreatic enzymes, resulting in pancreatitis. FA should be considered a potential trigger in IAP.

The belief that "serum tryptase levels always rise in food-induced anaphylaxis" is incorrect. Food-induced anaphylaxis is primarily a clinical diagnosis, not a laboratory-based finding. Tryptase levels may support the diagnosis in uncertain cases; however, a negative result does not exclude the diagnosis.[11] One possible explanation is that intestinal mucosal mast cells, which are implicated in food-induced anaphylaxis, contain lower levels of tryptase, compared to cutaneous mast cells, leading to reduced tryptase release into the circulation.[21] In our study, tryptase levels measured during pancreatitis were not elevated. However, the absence of elevated tryptase does not exclude the involvement of type I hypersensitivity reactions, such as anaphylaxis, in the pathogenesis of the attacks. Furthermore, case reports in the literature do not provide consistent data regarding tryptase levels in similar cases.

The prevalence of FA varies with age, with most reactions occurring in early childhood.[2] The IgE-mediated allergies to cow's milk, egg, soy, and wheat often resolve from childhood into adulthood, whereas allergies to shellfish, fish, peanuts, and tree nuts tend to persist and rarely resolve.[22] The new-onset allergies to cow's milk and wheat flour observed in adulthood in our study are noteworthy, as such patients are often misdiagnosed with conditions such as indigestion or food intolerance (e.g., lactose intolerance) by different specialties. Therefore, the possibility of FA should not be overlooked in adults. It is of great importance to accurately differentiate FA from other conditions in patients who report adverse reactions following food consumption. This is particularly critical in determining the need for long-term dietary restrictions. Another notable finding in our study was that patients diagnosed with red meat allergy did not report a prior history of tick exposure. It is well established that there is a potential link between tick bites and red meat allergy because of cross-reactivity with the alpha-gal epitope. Accordingly, we informed patients with red meat allergy about the possibility of previous or future tick exposure.

The correct diagnosis of FA is very important in reducing the risk of potentially life-threatening allergic reactions.[23] Unfortunately, there is still no proven radical treatment for FA. Since even consuming very small amounts of certain foods can cause severe reactions, the only effective preventive strategy is the strict avoidance of allergenic food. Accordingly, we advised eight patients included in our study to eliminate all foods from their diets to which they were sensitized. Our all patients except one complied with our recommendation. One patient, who had previously experienced an episode of acute pancreatitis following plum consumption, ingested plums again a few months later and experienced the second episode of acute pancreatitis within 6 months. Following this second episode, plum consumption was strictly prohibited. All patients were monitored for approximately 1 year, during which no allergic complaints were observed.

In our study, SPT were negative in patients diagnosed with FA, but sIgE tests were positive. This finding highlights the limitations of diagnostic tests. In particular, in FA, the inadequacy of a single diagnostic tool indicates that test results must be evaluated in conjunction with clinical history. Indeed, the European Academy of Allergy and Clinical Immunology (EAACI) emphasizes in its Food Allergy Diagnosis Guidelines that SPT and sIgE tests are highly sensitive but have low specificity, meaning that clinically insignificant positive results are possible. Furthermore, although the oral food provocation test is considered the gold standard, it is stated that it cannot be applied to every patient because of practical limitations.[24] The literature shows conflicting results regarding the sensitivity and specificity of SPT and sIgE tests in the diagnosis of FA. For example, in study done by Sabit et al., it was stated that SPT performed for milk protein had high sensitivity but limited specificity, compared to commercial allergen extracts.[25] Patti et al. reported that prick-to-prick test provided higher specificity than SPT using commercial walnut extracts in the diagnosis of walnut allergy.[26] Chua et al. reported that SPT results provided better predictive value than blood tests in the diagnosis of peanut allergy.[27] The conflicting findings that emerged in these studies show that the accuracy of tests used in the diagnosis of FA may vary from patient to patient.

In our study also, it was once again demonstrated that a negative SPT alone couldn't be considered sufficient to rule out FA. Therefore, in order to confirm the diagnosis, it is essential to evaluate the results in conjunction with sIgE and/or prick-to-prick testing. The prick-to-prick test provides valuable information, especially when performed with fresh foods and in cases where commercial extracts are insufficient. This test prevents false-negative results by increasing sensitivity. In our case, the prick-to-prick test performed with fresh plum yielded a positive result, which played a decisive role in the diagnostic process. In summary, for accurate diagnosis of FA, the combined use of SPT, sIgE measurements, prick-to-prick tests, and oral food challenge tests significantly improves the reliability and accuracy of the diagnostic process.

The primary limitation of our study is the small number of patients. Additionally, owing to the severity of the reactions experienced, oral food challenge tests could not be performed, based on a mutual decision made by both clinical team and patients, to avoid potential risks. Further research involving larger patient cohorts is necessary to better understand the molecular mechanisms and clinical squeal of FA-induced acute pancreatitis.

Conclusion

There are only a limited number of case reports in the literature suggesting that food allergens can trigger acute pancreatitis. Our study is the first prospective study conducted on this topic. Although acute pancreatitis because of FA is a rare condition, a better understanding of this association may help clinicians to have earlier diagnosis and appropriate management. In this context, it is important to consider FA in the differential diagnosis of patients presenting with pancreatitis, especially in cases where no clear etiology is identified. A multidisciplinary approach involving both allergists and gastroenterologists may be beneficial for identifying potential allergens and facilitating early diagnosis. The possibility of food-induced pancreatitis should not be overlooked, and it is strongly recommended that patients with IAP must be evaluated by an allergist.

Mandatory Disclosure on Use of Artificial Intelligence

The authors declare that no AI-assisted tools were used in the preparation of this manuscript. All references have been manually verified for accuracy and relevance.

Author Contributions

Balaban YA: study design; data collection, analysis, and interpretation; patient follow-up; writing the article, preparing the draft, and revising it; Inan MI: data collection, analysis, and interpretation; article revision; Kalkan F: data collection; article draft preparation; Sonmez

E: data collection; article draft preparation; Buyukturan G: patient follow-up, data collection; article draft preparation; Demirel F: Data collection; preparation of the article draft; Selcuk A: Data interpretation; revision of the article; Yesillik S: Data interpretation; revision of the article; Sakin YS: analysis and interpretation of data and revision of the article; Kartal O: study design; analysis and interpretation of data and revision of the article; All authors have given their final approval of the version and accept responsibility for all aspects of the work.

Conflict of Interest

The authors declared no potential conflict of interest with respect to research, authorship, and/or publication of this article.

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References

1. Manohar M, Verma AK, Upparahalli Venkateshaiah S, Goyal H, Mishra A. Food-ınduced acute pancreatitis. Dig Dis Sci. 2017 Dec;62(12):3287-97. https://doi.org/10.1007/s10620-017-4817-2
2. Boden SR, Wesley Burks A. Anaphylaxis: A history with emphasis on food allergy. Immunol Rev. 2011 Jul;242(1):247-57. https://doi.org/10.1111/j.1600-065X.2011.01028.x
3. Gukovskaya AS, Gukovsky I, Algül H, Habtezion A. Autophagy, ınflammation, and ımmune dysfunction in the pathogenesis of pancreatitis. Gastroenterology. 2017 Nov;153(5):1212-26. https://doi.org/10.1053/j.gastro.2017.08.071
4. Mayerle J, Sendler M, Hegyi E, Beyer G, Lerch MM, Sahin-Tóth M. Genetics, cell biology, and pathophysiology of pancreatitis. Gastroenterology. 2019 May;156(7):1951-68.e1. https://doi.org/10.1053/j.gastro.2018.11.081
5. Wang GJ, Gao CF, Wei D, Wang C, Ding SQ. Acute pancreatitis: Etiology and common pathogenesis. World J Gastroenterol. 2009 Mar;15(12):1427-30. https://doi.org/10.3748/wjg.15.1427
6. Mazza S, Elvo B, Conti CB, Drago A, Verga MC, Soro S, et al. Endoscopic ultrasound diagnostic gain over computed tomography and magnetic resonance cholangiopancreatography in defining etiology of idiopathic acute pancreatitis. World J Gastrointest Endosc. 2022 Jun;14(6):376-386. https://doi.org/10.4253/wjge.v14.i6.376
7. Aronen A, Guilabert L, Hadi A, Kiudelis V, Panaitescu A, Wlodarczyk B, et al. Idiopathic acute pancreatitis (IAP)—A review of the literature and algorithm proposed for the diagnostic work-up of IAP. Transl Gastroenterol Hepatol. 2024 Sep 13;9:71. https://doi.org/10.21037/tgh-23-125
8. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, et al. Classification of Acute Pancreatitis--2012: Revision of the Atlanta classification and definitions by international consensus. Gut. 2013 Jan;62(1):102-11. https://doi.org/10.1136/gutjnl-2012-302779
9. Werfel T, Asero R, Ballmer-Weber BK, Beyer K, Enrique E, Knulst AC, et al. Position paper of the EAACI: Food allergy due to immunological cross-reactions with common inhalant allergens. Allergy. 2015 Sep;70(9):1079-90. https://doi.org/10.1111/all.12666
10. Mendonca CE, Andreae DA. Food allergy. Prim Care. 2023 Jun;50(2):205-20. https://doi.org/10.1016/j.pop.2023.01.002
11. Anagnostou A. Addressing common misconceptions in food allergy: A review. Children (Basel). 2021 Jun;8(6):497. https://doi.org/10.3390/children8060497
12. Carrilllo T, Agustín M, Gusmán TM, Vilardell VU, Goiri IM. Pancreatitis aguda como complicación de anaphilaxia por mostaza. Rev Esp Alergol Immunol Clin. 1987;2:388-90.
13. de Diego Lorenzo A, Robles Fornieles J, Herrero López T, Cos Arregui E. Acute pancreatitis associated with milk allergy. Int J Pancreatol. 1992 Dec;12(3):319-21. https://doi.org/10.1007/BF02924372
14. Suzuki S, Homma T, Kurokawa M, Matsukura S, Adachi M, Wakabayashi K, et al. Eosinophilic gastroenteritis due to cow's milk allergy presenting with acute pancreatitis. Int Arch Allergy Immunol. 2012;158(Suppl 1):75-82. https://doi.org/10.1159/000337782
15. Tse KY, Christiansen SC. Eosinophilic gastroenteritis due to egg allergy presenting as acute pancreatitis. Allergy Rhinol (Providence). 2015 Jan;6(1):80-1. https://doi.org/10.2500/ar.2015.6.0105
16. Inamura H, Kashiwase Y, Morioka J, Kurosawa M. Acute pancreatitis possibly caused by allergy to bananas. J Investig Allergol Clin Immunol. 2005;15(3):222-4.
17. Pellegrino K, D'Urbano LE, Artesani MC, Riccardi C, Mancini S, Bella S, et al. Severe reaction in a child with asymptomatic codfish allergy: Food challenge reactivating recurrent pancreatitis. Ital J Pediatr. 2012 May;38:16. https://doi.org/10.1186/1824-7288-38-16
18. Gastaminza G, Bernaola G, Camino ME. Acute pancreatitis caused by allergy to kiwi fruit. Allergy. 1998 Nov;53(11):1104-5. https://doi.org/10.1111/j.1398-9995.1998.tb03824.x
19. Akgul Balaban Y, Inan M.I, Yesillik S, Kartal O. Unexpected culprit of acute pancreatitis—Adult cow's milk allergy: A case report. Revue Française d'Allergologie. 2024 July;64(4):104103. https://doi.org/10.1016/j.reval.2024.104103
20. Savage J, Johns CB. Food allergy: Epidemiology and natural history. Immunol Allergy Clin North Am. 2015 Feb;35(1):45-59. https://doi.org/10.1016/j.iac.2014.09.004
21. Schwartz LB, Irani AM, Roller K, Castells MC, Schechter NM. Quantitation of histamine, tryptase, and chymase in dispersed human T and TC mast cells. J Immunol. 1987 Apr;138(8):2611-5. https://doi.org/10.4049/jimmunol.138.8.2611
22. Anvari S, Miller J, Yeh CY, Davis CM. IgE-mediated food allergy. Clin Rev Allergy Immunol. 2019 Oct;57(2):244-60. https://doi.org/10.1007/s12016-018-8710-3
23. Peters RL, Krawiec M, Koplin JJ, Santos AF. Update on food allergy. Pediatr Allergy Immunol. 2021 May;32(4):647-57. https://doi.org/10.1111/pai.13443
24. Riggioni C, Ricci C, Moya B, Wong D, van Goor E, Bartha I, et al. Systematic review and meta-analyses on the accuracy of diagnostic tests for IgE-mediated food allergy. Allergy. 2024 Feb;79(2):324-52. https://doi.org/10.1111/all.15939
25. Sabit B, Ozdemir O, Kose E. Comparison of commercial allergen extract with various milk products of skin prick tests in diagnosing cow's milk protein allergy. Allergy Asthma Proc. 2023 May;44(3):193-9. https://doi.org/10.2500/aap.2023.44.230012
26. Patti ML, De Rose C, Brancato F, Gambacorta A, Miceli Sopo S. Sensitivity of prick test with walnut commercial extracts and of prick by prick with raw walnut compared with open food challenge in walnut allergy. Acta Biomed. 2021 Apr;92(S1):e2021067.
27. Chua GT, Chong PC, Au EY, Cheong KN, Wong WH, Chan EY, et al. Skin prick testing a better predictor than blood testing for the diagnosis of peanut allergy in Chinese children. Asian Pac J Allergy Immunol. 2021 Dec;39(4):241-8.