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CASE REPORT

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Anaphylaxis following prick-by-prick testing with peanut

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Abstract

Anaphylaxis due to prick-by-prick testing is a rare but potentially life-threatening complication. Personnel performing testing should be able to recognize and treat anaphylaxis. Adrenaline must be available. In high-risk patients with a history of anaphylaxis, specific igE with components and a basophile activation test is desirable before skin prick testing.

KEYWORDS

allergy and immunology, acute medicine, ear nose and throat, dermatology

1 | BACKGROUND

Food allergy is an immunologically mediated hypersensitivity reaction. Food allergies are examined with skin prick tests and laboratory tests. In food allergy, many allergens are not available as standardized allergen extracts, or the extracts does not contain the relevant allergen. Also, the predictive value of skin prick testing demonstrating a true allergic reaction over isolated sensitization is higher than the predictive value of specific IgE with components.[1,2] Therefore, prick-by-prick testing with native food is a commonly used procedure to aid better diagnostic results. This, however, increases the chance of systemic reactions. Also, due to the increasing demands for standardization in the medical industry and the following decreasing availability of allergen extracts, has created a paradox need for more unstandardized prick-by-prick testing. We here present a case of anaphylaxis following prick- by-prick testing to peanuts and aid to raise awareness toward the possible occurrence of systemic reactions when performing skin prick testing.

2 | CASE REPORT

A 39-year-old man came to our clinic for examination of food allergies. He had experienced repeated anaphylaxis, presenting with asthma and urticaria, due to ingestion of peanut, cashew nut, hazelnut, banana, and fish. He underwent skin prick testing to aeroallergens with standardized extracts several years ago with no special occurrences. He had undergone successful subcutaneous allergen immunotherapy to birch and timothy 10 years ago. As a child, he had suffered from atopic dermatitis until the age of three. The patient had gastroesophageal reflux treated with 20 mg pantoprazole daily, no other regular medication or comorbidities.

At our clinic, the patient underwent prick testing with standardized prick test extracts (ALK) for cat, dog, house dust mite, birch, timothy, egg white, cow milk, wheat, and peanut. We simultaneously performed prick-by-prick testing with roasted and salted peanut, banana, and chili-coated peanuts, which the patient brought for testing.

After 10 minutes, the patient developed severe local swelling of the area where peanut prick tests had been performed. The swelling increased to an urticarial weal of 10 cm diameter, and the redness quickly spread along the lymphovascular drainage path to the axilla (Figure 1). Only after reaching the axilla, systematic symptoms occurred. After 15 minutes, he developed urticaria in the axilla that spread to the thorax and both arms. He also developed a dry cough, shortness of breath, and wheezing. Measurement of peak expiratory flow (PEF) fell to 300 L/min, and he had obstructive pulmonary sounds on auscultation. Blood pressure: 146/93 mmHg. Pulse rate: 88

FIGURE 1 The picture shows the local spreading of the allergic reaction on the left arm where the prick-by-prick test was performed. The severe local spread anticipated the systemic reaction

beats per minute. He had a saturation of 100% O2 throughout. The patient was treated with 0.5 mg adrenaline (1 mg/mL) intramuscular and five puffs a 0.2 mg of salbutamol via inhalation chamber. He quickly responded with subjective improved breathing and regress of the urticaria. He received 5 mg desloratadine orally and 20 mg of stand by prednisolone tablets. After an observation time of 2 hours, he was resituated with a blood pressure of 133/84 mm Hg, pulse rate 59, saturation 100% O2, and a peak expiratory flow of 510 L/min. Pulmonary auscultation normalized and the skin showed only a residual local swelling around the prick tests. Blood tests obtained later the same day showed total IgE 272 kU/L (<120), tryptase 6.1 mcg/L (<12), specific Ig E for peanut 89 KU/L (<0.35), Ara h8 < 0.35 kU/L (<0.35), Ara h1 23.4 kU/L (<0.35) and Ara h2 58.1 kU/L (<0.35), Ara h9 was < 0.35 kU/L. There was no positive IgE for fish, shellfish, and tropomyosin. Specific IgE for banana was <0.35 kU/L. The patient shoved a blood profile with broad sensitization toward all treenuts and almonds including components for storage proteins, but the LTP components Ara h9, Cor a 8, Jug r 3 where negative. The patient is now at good health and has follow-up appointments for further allergy evaluation at our clinic. The patient has been supplied with an adrenaline autoinjector and is instructed to avoid nut and peanut consumption.

3 | DISCUSSION

Anaphylaxis due to prick and prick-by-prick testing is known but rare. The prevalence is estimated between 0,02% and 0,12%.[3,4] Very rarely fatalities due to skin prick tests are reported. A survey by Bernstein et al[5] found one fatality due to skin prick tests over 12 years in North America. We detected the following risk factors for anaphylaxis in our patient: large local reaction to skin prick testing, previous anaphylaxis due to peanut, peanut specific IgE peanut 89 kU/L (<0, 35 kU/L), and a component sensitization pattern of storage proteins (Ara h1 and Ara h2). In peanut allergy, the molecular profile of allergy components has been seen to predict real peanut allergy in some study populations.[6] A combined sensitization pattern of Ara h1, Ara h 2, and Ara h 3 is linked with a risk of more severe reactions,[7] but studies have shown several limitations and inconsistencies, and this has been pointed out in an AAAAI/ACAAI/JCAAI food allergy position paper.[8] In retrospect, the high numbers of prick tests performed on our patient, including three simultaneous tests of peanut (including two prick-by-prick), may well have increased the risk of anaphylaxis and the number of simultaneous test will be reduced in the future. Also if the peanut IgE with components had been obtained bevor skin prick testing, the number of peanut allergens tested most likely would have been reduced, possibly avoiding the patient's anaphylaxis.

It is however important to remember that specific IgE components can only demonstrate sensitization and must be combined with further testing and the patient's history to demonstrate clinical allergy. This challenge of interpreting laboratory results in allergy is well demonstrated in our patient as he had also experienced anaphylaxis after ingestion of banana and several fish species (in absence of nut exposure), and he has no positive specific IgE toward these allergens. If a skin prick test is impracticable, a combination of molecular diagnostic and basophile activation test should be obtained, and if negative, an oral provocation test can be considered.

Ara h 2 is considered the main component for diagnosing real peanut allergy, especially when the level of Ara h 2 is high compared to the level of total IgE. In Norway, many patients have mild peanut reactions despite high total IgE to peanut due to monosensitization to Ara h 8 (a PR-10 protein). This is a result of high birch exposure with sensitization to the cross reacting protein Bet v 1. LTP (panallergen) sensitization is rare in Norway, but is a known challenge in the Mediterranean region and a risk factor for severe reactions.

The use of molecular cutoffs differs between study populations in age and geographic regions, and this must be considered when using molecular diagnosis in regular practice. Also, this is still a costly test and only few Norwegian hospitals perform molecular allergy testing. As skin prick testing is per now seen as superior in predicting clinical allergy, this

is normally the first procedure performed. However in patients with anaphylaxis after food ingestion, measuring base line tryptase, specific IgE and components of the suspected culprit is desirable bevor skin prick testing. Per now, we do not use molecular diagnosis as a single decisive tool, but as one instrument out of several to evaluate the severity of an allergy, to predict potential for cross reactions and for advising patients on managing their reactions.

In retrospect, the high numbers of prick tests performed including three simultaneous tests of peanut (including two prick-by-prick) may well have increased the risk of anaphylaxis. As the number of available commercial allergen extracts has fallen decreased due to higher demands of standardization and the following economic considerations,[9] prick-by-prick testing is becoming more utilized. Hence, the number of prick test reactions could be rising, and knowledge of the potential for systemic allergic reactions anaphylaxis and its treatment is essential. Anaphylaxis is a severe, possible life-threatening hypersensitivity reaction. Our patient received anaphylaxis treatment in line with international guidelines[10] with prompt intramuscular administration of 0,5 mL (1 mg/mL) adrenaline. Adrenaline is the drug of choice in anaphylaxis treatment, and antihistamines, steroids, salbutamol, and other medications are additive treatments to be considered after adrenaline administration. A repeated challenge in the treatment of anaphylaxis is the late recognition of anaphylaxis and the low adherence to the guidelines concerning adrenaline administration, and the importance of having adrenaline available while testing cannot be stressed enough.[11]

4 | LEARNING POINTS

- Even though anaphylaxis due to skin prick test including prick-by-prick testing is rare, it can occur at any time during testing.
- This case is an important reminder of the risk of anaphylaxis due to skin prick testing, and the need to be prepared to treat anaphylaxis everywhere skin prick tests are performed.
- In patients with a story of anaphylaxis to a suspected culprit, obtaining baseline tryptase, specific IgE with components, is desirable bevor considering skin prick testing.
- Molecular diagnostics can aid decision-making bevor skin prick testing the patient.
- Anaphylaxis is treated with administration of intramuscular adrenaline.
- We suggest increased awareness toward patients with large and quickly developing skin prick test.

CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTION

Marie Alnæs: is the single author on the case report. All work involved on the report was performed by Marie Alnæs.

CONSENT STATEMENT

The patient has given his written consent to publication of the case.

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REFERENCES

1. Heinzerling L, Mari A, Bergmann K-C, et al. The skin prick test – European standards. *Clin Transl Allergy*. 2013;2013(3):3.
2. Larenas-Linnemann D, Luna-Pech JA, Mösges R. Debates in allergy medicine: allergy skin testing cannot be replaced by molecular diagnosis in the near future. *World Allergy Organ J*. 2017;10:32.
3. Liccardi G, D'Amato G, Walter Canonica G, et al. Systemic reactions from skin testing: literature review. *J Invest Allergol Clin Immunol*. 2006;16(2):75-78.
4. Norrman G, Falth-Magnusson K. Adverse reactions to skin prick testing in children: prevalence and possible risk factors. *Pediatr. Allergy Immunol*. 2009;20:273-278.
5. Bernstein DI, Wanner Mark, Borish L, et al. Twelve-year survey of fatal reactions to allergen injections and skin testing: 1990–2001. *J Allergy Clin Immunol*. 2004;113(6):1129-1136.
6. Lieberman P, Glaumann S, Batelson S, et al. The utility of peanut components in the diagnosis of IgE-mediated peanut allergy among distinct populations. *J Allergy Clin Immunol Pract*. 2013;1:75-82.
7. van Erp FC, van Erp FC, Klemans RJB, Meijer Y, van der Ent CK, Knulst AC. Using component-resolved diagnostics in the management of peanut-allergic patients. *Curr Treat Options Allergy*. 2016;2016(3):169-180.
8. Sampson HA, Aceves S, Bock SA, et al. Food allergy: a practice parameter update. *J Allergy Clin Immunol*. 2014;134(2014):1016-1025.
9. Heinzerling L, Mari A, Bergmann KC, et al. The skin prick test - European standards. *Clin Transl Allergy*. 2013;3(1):3.
10. Simons FER, Ardusso LRF, Bilò MB, Cardona V, et al. International consensus on (ICON) anaphylaxis. *World Allergy Organ J*. 2014;7(1):9.
11. Ribeiro M, Herberto JC. Diagnosis and treatment of anaphylaxis: there is an urgent needs to implement the use of guidelines, *Einstein (Sao Paulo)*. 2017;15(4):500-506.

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