

Mustard Allergy in Children

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The prevalence of allergies to individual foods varies with the dietary practices of different populations. A recent study performed in France and validated by standard procedures¹ ranks mustard in fourth place in children's food allergies, behind eggs, peanuts, and cow's milk. Mustard allergy is not well known. The purpose of this study was to identify children who are allergic to mustard, to assess clinical features and other associated allergies, and to define skin prick test (SPT), specific IgE, and dose response by oral food challenge.

STUDY POPULATION AND METHODS

Our study was performed by investigating 36 consecutive children with positive mustard SPT from 3600 mustard SPTs performed (1%). Fresh extracts (mustard seed powder including *Sinapis alba* and *Brassica juncea*, 1:10 w/v) were used for SPT. Mustard-specific IgE was determined by the CAP system (Pharmacia, France). The strong taste of mustard makes it impossible to perform double-blind, placebo-controlled, food challenges (DBPCFC), and the use of capsules misses the first oral contact. However, we based the diagnosis on DBPCFC. The study population consisted of 36 children, 22 boys and 14 girls (sex ratio M/F 1.5), aged 10 months to 15 years (average age, 5.5 years). The allergic group consisted of 15 children with suggestive symptoms, positive SPT and DBPCFC. The nonallergic group consisted of 21 children with positive SPT but negative DBPCFC. We compared all our patients to 22 controls without a history of food allergy. We assessed the control group for mustard SPT and mustard-specific IgE. We performed 12 labial food challenges (LFC) immediately followed by DBPCFC.

RESULTS

Our study was performed on 36 children having positive mustard SPT. The 54 initial clinical features of the studied subjects were (sometimes in association) atopic dermatitis in 28 cases (51.8%), urticaria and/or angioedema in 20 cases (37%), asthma in 5 cases (9.2%), and, for one child, laryngeal edema with oral allergy syndrome and rhino-conjunctivitis (1.8%). No anaphylaxis was observed. The allergic group consisted of 15 children with positive DBPCFC. The non-allergic group concerned 21 children with positive SPT and negative DBPCFC. In the allergic group, symptoms after mustard

ingestion started under 3 years of age for 8 children (53.3%).

Associated food allergies were found in 24 of the 36 patients (66.6%). Associated inhalant allergen sensitization was found in 24 patients in the study group. There was no significant difference for the food allergies and associated inhalant allergen sensitizations. The mean wheal diameter for mustard SPT was 8.8 mm for the allergic group and 5.3 mm for the non-allergic group ($P < 0.05$). The median concentration of mustard-specific IgE was 23.8 kU/L in the allergic group as compared to 4.9 kU/L in the non-allergic group and 0.37 kU/L in the control group ($P < 0.001$). The sensitivity of mustard-specific IgE was 80%, the specificity 66.6%, the positive predictive accuracy (PPA) 63.1%, and the negative predictive accuracy (NPA) 82.3%. The area under the Receiver Operating Characteristic curve (ROC) was 0.891 (sd 0.019). The best PPA was 80% at levels of 19.8 kU/L for mustard-specific IgE. The mustard CAP system R never reached a 95% predictive value in the study population.

We observed 3 positive LFC followed by positive DBPCFC and 2 positive LFC followed by negative DBPCFC. The sensitivity of LFC was 50%, the specificity 60%, the PPA 60%, and the NPA 50%.

The cumulative reactive dose by DBPCFC varied from 1 to 936 mg. No reactions to placebo were observed. The mean cumulative reactive dose was 153 mg. The reactions observed during the DBPCFC were urticaria (14 cases), rhino-conjunctivitis (3 cases), angioedema (1 case), oral allergy syndrome (1 case), and eczema (1 case).

DISCUSSION

The mustard plant belongs to the *Brassicaceae* family. Mustard powder is a mixture of two species: *Brassicaceae*, *Sinapis alba* L. (yellow mustard) and *Brassica juncea* L. (oriental mustard). Cross-sensitivity between *Brassicaceae* is rare.² In our study, we noted a single

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sensitization to radishes, not expressed clinically. Caballero found an association between mugwort pollen hypersensitivity and sensitivity to other edible plants, including mustard.³ In our study, we did not observe any particular association with pollen.

Mustard allergy started early in life, usually before the age of 3 years: 8 of 15 allergic children were under three years of age. Mustard is considered a hidden allergen in the usual infant diet. But the increased frequency of occurrence at early ages could be explained by the presence of mustard in baby foods for babies just being weaned. Mustard is also present in commercial foods for toddlers. Contact with mustard is frequent in infancy. Niinimäki et al. report positive SPT to mustard in three infants aged 12 to 18 months, breast-fed until the age of 11 months, who had never consumed mustard before.⁴ A sensitization *in utero* or through breast feeding is suggested. We did not observe anaphylaxis due to mustard, but a first case of anaphylaxis was described by Panconesi in 1980 after the subject had eaten a pizza.⁵ Since then, there have been several publications.^{5,9}

In our study, the size of the mustard SPT and the mustard-specific IgE concentration appeared to be significant. The mean average mustard SPT was 8.8 mm in the allergic group. The average concentration of mustard-specific IgE was 23.8 kU/L in the allergic group. The LFC does not seem to be appropriate for diagnosing mustard allergy. DBPCFC is the gold standard for food allergy diagnosis. It is necessary, however, to define an inert vehicle that masks the taste of mustard if true DBPCFC are to be performed. Protein extracts will probably need to be used, as reported for peanuts.¹⁰ The dose response has never been reported for mustard as far as we know. It was not very high in our patients, an estimated mean of 153 mg. Panconesi and Wildstrom have shown that the amounts of mustard ingested were very small in the cases they observed. In two of the five cases, mustard came from the accidental contamination of the food by cooking utensils.^{5,6}

CONCLUSION

Allergic reactions to mustard start early in life, usually under 3 years of age. Frequent contact with mustard in

infancy could be explained by the presence of mustard in baby foods. As for peanuts, sensitization certainly takes place *in utero* or through breast-feeding. Clinical symptoms were not severe in children. The problem with DBPCFC is the question of which vehicle to use to mask the mustard and avoid its irritating character. Protein extracts would probably be needed to do DBPCFC. Dose response is low. These data suggest that screening tests for food allergies should be changed to systematically include mustard, even before the age of 3 years. The evolution of this food allergy needs to be clarified by further studies. The treatment goal for food allergy is the elimination of the offending food. Having an accurate label on a food product is imperative.

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