

SESAME SEED ALLERGY IN CHILDREN

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SUMMARY:

Background: Sesame seed allergy is becoming more common in childhood. The aim of this study is to define the clinical signs and the results of allergological work-up of this food allergy as well as the demographical data in children. Sesame seed allergy outcome is unknown.

Materials and methods: 14 children were recruited from 3 allergy centers in France. The diagnosis of food allergy was based on a convincing clinical history and positive skin prick tests and/or an elevated sesame specific IgE. Food challenge test was done when results of history and allergological work-up were conflicting. A reintroduction test was done when a child seemed to outgrow his (or her) food allergy.

Results: The median age at the beginning of sesame seed allergy was 5 years (range from 5 months to 16 years old). All patients reacted immediately after sesame seed consumption and presented as a first manifestation: edema (9 cases, 48%), urticaria (5, 27%), and one of each of the following symptoms (vomiting, rhinitis, conjunctivitis, asthma and anaphylactic shock). One patient had recurrent anaphylactic shocks and another an anaphylactic shock after subsequent sesame seed exposure; these 2 patients were asthmatic. The median of the wheal size was 5 mm (range 3 to 15 mm). The commercial sesame seed extract was less sensitive than the native seed. The median of sesame seed IgE was 5.58 kUA/L (range 0.35 to 100 kUA/L). The follow up lasted from a few months to 6 years. Three patients outgrew their food allergy. All of these patients showed a previous drop of sesame seed IgE and skin prick-tests became negative.

Conclusion: Sesame seed allergy is not very different than other food allergy. We reported the spontaneous outgrowing of sesame seed allergy without being able to define the predictive criteria for a good outcome.

INTRODUCTION

Sesame allergies have been existing for a long time and it seems to be common in certain countries. In Israel, this food allergy is the 3rd most prevalent of food allergies (1). Its appearance in Europe is more recent (2) but is spreading more and more. The observed cases have been primarily in adults, only one study is exclusively focused in children (3). We analyzed the clinical and allergological data of 14 sesame allergic children.

MATERIALS AND METHODS

Study population

In a retrospective study, 14 children were recruited in the pediatric allergy centers in Toulouse, Boulogne

and Paris. They were included on the basis of a clinical history compatible with sesame seed allergy. All the children had skin prick-tests (SPTs) and/or specific IgE antibody measurements. A food challenge test was performed in the case of discrepancy between the clinical history and the allergological tests. In order to appreciate the outcome, a reintroduction test was done each time there were arguments in favor of the disappearance of their food allergy.

Methods

■ **Clinical history:** a "convincing" clinical history is an isolated food intake that leads to anaphylactic manifestations calling for medical attention within a 3-year period (4). In this case, a food challenge test is unnecessary to confirm the diagnosis.

■ **Cutaneous tests:** patients 1 to 12 were administered SPTs with commercial sesame seed extract (CSSE) (Allerbio, Varennes en Argonne, France) and patients 13 and 14 with another commercial extract (Stallergènes, Antony, France). Native sesame seed extract (NSSE) was also used for patients 1 to 4 and

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was administered by prick-prick method of Dreborg & Foucard (5). Other SPTs were carried out depending on the clinical history. A positive reaction was defined as wheal with a mean diameter 3 mm or more than the negative control.

■ **Specific sesame seed antibodies:** the measurement specific IgE antibodies was done by the CAP system method (Pharmacia, St Quentin en Yvelines, France) for 11 patients.

■ **Food challenge test:** a food challenge test was done when there was a discrepancy between the clinical history and the allergological tests. The oral challenge test consisted in swallowing progressively increasing doses of crushed sesame seed mixed with apple compote. A test was positive when it leads to signs (urticaria, edema, asthma, rhinitis, vomiting, abdominal pain or anaphylactic shock) less than 2 hours after intake. As previously described (6), the labial challenge consisted in depositing ground sesame seed on the outer aspect of the lower lip for 10 seconds to 2 minutes depending on the risk of systemic reaction. The reading is done after 30 minutes. A test is considered positive when it leads to an urticaria of the cheeks or the chin or more severe signs such as asthma or anaphylactic shock (6). Labial food challenge was performed for patient 1 who presented an anaphylactic shock at sesame re-exposure and had a negative sesame SPTs and a very high specific IgE antibodies. Oral food challenge was done for patients 2 and 5 to assess their food allergy and for patients # 5 and # 11 to appreciate the outcome.

■ **Evolution:** we followed these patients from a few months to 6 years. Some patients had a second measurement of sesame seed IgE antibodies. A reintroduction test was carried out each time there were arguments supporting that the food allergy had disappeared.

RESULTS

Patients' data are summarized in table 1. The mean age at the beginning of sesame allergy was 5 years (range from 10 months to 16 years). Five patients were under 2 years old. The sex ratio is 2 boys for 1 girl. Nine children (66%) have a family history of allergies. Eleven children had associated atopic manifestations. Atopic eczema/dermatitis syndrome was the leading sign of atopy (6 cases, 43%) followed by asthma (5 cases, 36%) and rhinitis (3 cases, 21%).

All our patients had a clinical reaction after exposure to sesame seed. The first manifestation of sesame seed allergy was edema (9 cases, 64%), urticaria (4

cases, 28%), and one case of each of the following symptoms: vomiting, rhinitis, conjunctivitis, asthma, and anaphylactic shock. It must be emphasized that an anaphylactic shock is possible as a first reaction (patient 4) or after exposure to sesame seed even though the first contact led only to laryngeal edema (patient 1). The anaphylactic shock affected only mono-sensitized (patient 4) or pauci-sensitized (patient 1) patients; they had asthma as an isolated atopic manifestation. The clinical reaction of all the patients occurred after intentional intake of sesame, except for a breast-fed infant. This child presented a facial edema at 5 months old during breastfeeding; her mother had eaten large amounts of sesame seed beforehand. This infant was subject to urticaria at 12 months, after eating sesame bread.

Nine patients out of 14 (64%) had multiple sensitization. The sensitization distribution was as follow; nuts or shell fruits (14 times), leguminous (6), milk products (2), hen's egg (3), aeroallergens (3), and wheat (1 child). The nuts found are: hazelnuts (4 patients), cashews (4), pine nuts (2), pistachios (3), almonds (2), and walnuts (2). The leguminous are: peanuts (3), pine nuts (2) and lupine (1).

With the exception of patients 1 through 4, who were positive to prick-test with NSSE and negative to the test with the CSSE, the other patients were all positive with the CSSE. The median wheal size was 5 mm (range 3 to 13 mm). The IgE was measured for 11 patients, 3 were tested negative. The median of sesame seed IgE antibodies was 5.58 kUA/L (range 0.35 to 100kUA/L).

Follow up ranges between a few months and 6 years. Signs at least as severe as those following the exposure still follow sesame seed consumption. Three patients outgrew their food allergy. These 3 patients showed a negative skin prick-tests results and a drop in sesame seed antibodies. Spontaneous loss of the allergy was confirmed by a reintroduction test.

DISCUSSION

According to Rancé et al., sesame seed allergy is the 15th most prevalent pediatric food allergy in France and involves 0.6% of a population of 703 children with food allergies (7). Nevertheless, sesame is the 7th leading cause of severe food allergies among all age groups in France (8). In Israel, sesame seed allergy prevalence is 0.18% of a general population of infants under 2 years old and 0.42% of a cohort of atopic Australian children (9). Sesame sensitization prevalence is estimated at 19% in Australia and contrasts with the rarity of the allergy prevalence in the same population (9).

Patient	Sexe	Age at onset (in years)	Past medical history	Causative food	Signs and symptoms at first exposure	SPT (mm) with commercial extracts	SPT (mm) with native sesame seed	Specific IgE antibodies (kU/L)	Other sensitisations	Food challenge (labial / oral)	Signs and symptoms at subsequent exposures
1	M	9	Asthma	Tehina	LE, Lx	0	ND	> 100	Hazelnut, pistachio	LFC : level 3	As at re-exposure
2	M	8	None	« Sesame crackers »	U, FE	0	3	< 0.35	None	OFC : U with 66mg of sesame seed	
3	F	5	Asthma	Sesame bread	AS	0	3	< 0.35	Almond	ND	Recurrent AS
4	M	16	Asthma	Sesame seed	U	0	6	< 0.35	None	ND	Asymptomatic inadvertent intake, negative SPT and specific IgE at 0.86
5	F	2.5	Asthma, eczema, rhinitis	Tehina, halwah	LE	5	ND	6.15	None	ND	specific IgE at 0.86 (3 years later)
6	M	4	Eczema	Halwah	LE	6	ND	ND	None	ND	
7	F	1.5	Eczema	Halwah	U	ND	ND	5.8	Hazelnut, peanut, egg, wheat, goat milk	ND	
8	M	0.91	Eczema	Tehina	A, FE, Eye	3	ND	5.58	Chick pea, birch pollens	ND	Specific IgE < .35, negative re-introduction test (2.5 years later)
9	F	0.41	Asthma, eczema, rhinitis	Maternal milk	LE	7	ND	> 100	Egg, chicken meat, cashew nut, pistachio, peanut,, pine nut, grass	ND	U and FE after inadvertent intake (6 months later)
10	M	2	Eczema	Halwah candy	Eye, FE, U	8	ND	6.8	Cashew nut, pistachio, grass, pollens	ND	Specific IgE < .35 and negative (OFC 3 years later)
11	F	7	Asthma, rhinitis	Sesame bread	U, Eye	3	ND	ND	Cashew nut	ND	
12	M	0.83	None	Sesame bread	Lx, Eye	3	ND	21.2	Egg, peanut, pistachio	ND	
13	M	6	Eczema	« Sesame crackers »	U, FE, Eye, vom	5	ND	ND	Peanut, lupine, walnut, pine nut, hazelnut, almond, cashew nut	ND	
14	M	4	Eczema	Chinese food	Lx, Eye	4	ND	ND	Peanut, egg, house dust mite	ND	

M : male ; F : female ; E : edema ; L : labial ; Lx : laryngeal ; Eye : eyelid ; F : facial ; U : urticaria ; AS : anaphylactic shock ; vom : vomiting ; RC : rhino-conjunctivitis

Table 1: Demographic and clinical data of 14 children with sesame allergy.

The average age of our patients was 5.8 years old. This is older than the 10 month old mean in the series reported by Levy (3). Personal atopy prevalence was 85.7% or 2.5 times more common than the Israeli children allergic to sesame whose personal atopy is 35%. Family history of atopy was 3 times more common in our patients than in those of Dalal who reported only 25% of sesame allergic infants randomly chosen (1).

The sensitization age of our patients is unknown, but a breastfed infant reacted at 5 months. Various authors (2, 9, 10) have reported the early sensitization to sesame explained by early diet exposure (1, 9). Multi-sensitization concerned 60% of children. Nuts and peanuts were the most dominant. These results are in accordance with those of Australian's authors (9). Only Levy and Danon's patients, who were very young, showed associated sensitization to cow's milk and hen's egg (3). This common sensitization to nuts and peanut is linked to homologies between sesame seed allergens and various food allergens. Ses i 1, a major sesame seed allergen (accepted nomenclature), shares many homologies with Ber e 1 from Brazil nut, Ric c 1 from ricin seed and an unidentified sunflower seed allergen (10, 11). Ses i 2 (nomenclature to be approved) shares many homologies with Ses e 1, Ber e 1, Jug r 1 from walnut and Ara n 2 from peanut (11). Ses e 3 (nomenclature to be approved) shares many homologies with Jug r 4 and Ara n 1 (11). These structural homologies could play an important role in associated multi-sensitivity to sesame and the large difference between common sensitization to sesame seed and real allergy, which is quite rare.

Some patients showed particular diagnosis problems. Patients 1 to 4 showed negative SPTs with CSSE and positive SPTs with NSSE when it had performed (patients 2 to 4). This weak sensitivity of CSSE was taken into account recently by the manufacturer who improved the SPTs (12). The improvement of the CSSE led to positive SPT in the patients tested later (patients 6 to 13). Many studies suggested that sesame seed allergens involved in this food allergy vary among countries. Pastorello and al identified Ses i 1 which seemed to play an important in Italian patients (10) while Beyer and al (11) showed that Ses i 2 may play a similar role in American patients among various other sesame seed allergens. Recently, Wehner (13) and al identified an 14 kD 2S albumin precursor in white sesame of "Ethiopian" origin which played an important role in Israeli patients. This 14 kD 2S albumin wasn't identified by Kollop-Sarda and al (14) as an major allergens in French patients. The contrasting specific IgE results showed by patients 1 to 4 may depend on the geographical origins of the sesame seed tested and the involvement of different epitopes in these patients.

The seriousness of the sesame allergy has already been emphasized; 6 anaphylactic shocks have been reported in children in an epidemiological study (1). Anaphylactic shock revealed the sesame seed allergy (patient 4) or appeared after re-exposure following an initial episode of local edema (patient 1). The concept of systemic reaction revealing sesame seed allergy is documented for teenagers (15, 16). Severe reactions were noted in sesame mono-sensitized patients (15, 16, 17). These increasingly serious manifestations can reach anaphylactic shock and reappear during a food challenge test (16). However, anaphylactic shock didn't occur during the food challenge performed during the reintroduction test whatever it was, labial or oral. Adding the various cases of anaphylactic shock caused by sesame consumption as reported in the literature to the number of published sesame allergic patients suggests that anaphylactic shock appears in 27% of sesame seed allergic patients (1-3, 7, 8, 10, 11, 13-37).

There wasn't a reported case of sesame oil allergy in our population. Torsney (22) and Uvitsky (25) highlighted negative sesame oil prick-tests for sesame allergic patients. Nevertheless, other authors have reported one sesame oil allergy case and a positive food challenge test with 3 ml of sesame oil (2). More often sesame oil contained in cosmetics induced cutaneous symptoms (32, 33, 34).

Very often, sesame seed allergy diagnosis is based on "convincing clinical histories". The "convincing clinical history" is a clinical manifestation (urticaria, edema, angio-edema, rhinitis, asthma or anaphylactic shock) occurring in less than 2 hours following the food intake. The diagnosis of food allergy was confirmed by SPTs and specific IgE antibodies measurements. Native extract gave proof of sensitivity when the commercial extract sometimes rendered false negative results. However using sesame seed of different colors altered the wheal size in the same patient (37). The variety of sesame seeds and the large number of isoallergens present in sesame could explain the weak performance of some commercial extracts and perhaps some of the native extracts if only one type of sesame seed is used (37). Unfortunately, a serum immuno-analysis wasn't done to identify the relevant allergens in this study. Reactions to sesame seed upon re-exposure were characterized by occurrence of anaphylactic shock (16, 17). This evolution justifies the complete avoidance of sesame in the diet and the prescription of an emergency kit and short acting beta-2 agonist if needed. Three of our patients outgrew their food allergy, they showed sesame negative skin prick-tests and specific IgE antibodies. These three patients were

"cured" when they were still younger than the age average of sesame seed onset. Two of our patients had asthma and only one is mono-sensitized to sesame. The symptomatology of the newly tolerant patient was dominated by immediate cutaneous reactions and multi-sensitizations. Recently, Dalal and al (38) reported the spontaneous resolution of sesame seed allergy within 2 years in 3 Israeli infants. These children were characterized clinically by cutaneous or gastro-intestinal symptoms and few sensitizations.

CONCLUSION

Sesame allergy is rare in Europe. The clinical diagnosis is usually easy but the low sensitivity of the commercial extracts often leads to the use of native extracts for prick-tests. For reducing the number of false negative skin prick-tests in sesame allergic patients, it must be helpful to use seed of different colors as they alter the weal size. In further studies, it may be helpful to report the origin and color of sesame seed consumed by sesame allergic patients and used by the allergist to assess the diagnosis. We have reported in 3 out of 14 patients a secondary tolerance to sesame without being able to define the predictive criteria that would lead to this favorable evolution. Even our results are reassuring, prospective studies are necessary to confirm them and define which patients are prone to a spontaneous sesame seed tolerance.

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PRESS RELEASE

Summer weather increases symptoms for asthma patients The AAAAI stresses the importance of proper asthma management

MILWAUKEE - Every season has its own special hazards for people with asthma, and summer is no exception. Ozone alerts, forest fire smoke and wind blown tree and plant pollens can trigger asthma attacks. Even people with mild asthma can experience asthma attacks severe enough to send them to the emergency department. More than 20 million Americans have asthma, and it is important for people with all levels of asthma severity to recognize their symptoms and know what to do if they experience an asthma exacerbation, according to the American Academy of Allergy, Asthma and Immunology (AAAAI).

"Since asthma is a chronic disease, it requires continuous management - not just when it gets bad," said Carlos Camargo, MD, a fellow of the AAAAI and an emergency physician at Massachusetts General Hospital in Boston. "Recent studies have shown that many of the inflammatory changes of moderate and severe asthma are also present in mild asthma.

"People with asthma should monitor their symptoms, understand what triggers their asthma and work closely with their doctors."

Asthma is a chronic lung disease characterized by the inflammation of the airways. For people who have asthma, the air flowing in and out of their lungs may be blocked by muscle swelling and squeezing.

If you have mild asthma, it means you generally have asthma symptoms (wheezing, coughing, and shortness of breath) less than two times a week. Regardless of your asthma classification, a severe exacerbation can send you to the hospital:

There were 1.8 million asthma-related visits to emergency departments in 2000 (more than 728,000 of these involved children under 18).

There were 465,000 asthma-related hospitalizations in 2000 (214,000 of these involved children under 18).

To ensure your asthma doesn't result in a visit to the emergency department, the AAAAI recommends taking the following precautions:

Avoid asthma triggers, including specific allergens such as house dust mites, cockroach allergen, indoor mold, animal dander and irritants such as cigarette smoke.

Manage asthma with proper medications. People with asthma have inflamed airways that can become even more inflamed after exposure to various triggers. The main purpose of asthma medications is to reduce this inflammation.

For exercise-induced asthma, spend time warming up before starting strenuous activity and gradually cool down afterward. avoiding activity during a respiratory tract infection, and avoiding exertion in extremely cold weather may help prevent an asthma attack.

Be aware of changes in the external environment, such as temperature changes, increased ozone levels and increased pollen counts.

Work with an allergist/immunologist to develop an effective management and treatment plan.

To make sure their asthma is under control, people with mild asthma may need to work with an allergist/immunologist, a physician specially trained to manage and treat allergies and asthma. Any patient who has had a single life-threatening asthma exacerbation should see an asthma specialist.

Together, you and your allergist/immunologist can work to ensure that asthma does not interfere with your optimal quality of life. Studies have shown that asthma specialty care results in a 27% reduction in emergency hospital care for asthma. An allergist/immunologist will discuss your patient history and talk about ways to prevent and control symptoms.

To find an allergist/immunologist in your area or to learn more about asthma, call the AAAAI Physician Referral and Information Line at 1-800-822-2762 or visit the AAAAI Web site at www.aaaai.org. The American Academy of Allergy, Asthma & Immunology is the largest professional medical specialty organization in the United States, representing allergists, asthma specialists, clinical immunologists, allied health professionals, and others with a special interest in the research and treatment of allergic disease. Established in 1943, the AAAAI has nearly 6,000 members in the United States, Canada and 60 other countries.

EDITOR'S NOTE: To speak with an asthma expert or for more information, contact John Gardner, Media Relations Manager, at (414) 272-6071 or jgardner@aaaai.org. Visit the AAAAI Web site, www.aaaai.org, for the most comprehensive information related to allergic disease.