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Sesame food allergy and sensitization in children: the natural history and long-term follow-up

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Sesame food allergy (SFA) in children is an increasingly recognized one in many countries. Our objective was to describe the course and natural history of SFA. Seventy-four patients sensitized to sesame were evaluated using clinical records, questionnaires, skin prick tests (SPT), *in vitro* specific immunoglobulin (sIg) E, and oral challenges (OC) and categorized into three groups: group A: patients who experienced allergic reaction after ingestion of sesame-containing food (n = 45); group B: patients evaluated for atopic dermatitis and found to be sesame SPT-positive (n = 11); group C: patients with sensitization to sesame allergen (n = 18). Group A patients were followed for an average of 6.7 yr. Analysis of our results revealed that 76% of patients in group A developed the allergy by the age of 2. The median age at onset of allergy in these patients was 1 yr. Immediate reaction to a minimal amount of sesame was characteristic. Skin was the most common site of involvement, followed by respiratory and gastrointestinal systems. Tolerance developed in only 20% of the patients. High sIgE (>0.15 IU) was demonstrated only in 75% of those in which it was examined. Sixteen patients performed oral sesame food challenge which was found positive in 88%. No correlation was found between the size of SPT and the level of *in vitro* sesame IgE antibodies, the outcome of OC results, and the development of tolerance to sesame. In conclusion, SFA tends to appear early in life, but unlike cow's milk and egg allergy, persists in 80% of the cases. Typical reactions combined with positive sesame SPT are reliable for diagnosis.

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The reported prevalence of food allergy in children is close to 8% (1) and may reach up to 19% when using less strict diagnostic criteria (2). Food allergy, however, is often transient (3, 4) and the prevalence of food allergy in adults is estimated to be much approximately 2% (5). Patients with eczema in general (6) and atopic dermatitis (AD) in particular, comprise a specific risk group in which the prevalence of food allergy reaches 37% in moderate to severe patients (7).

While earlier reports suggested that many foods are allergenic, it is now recognized that the vast majority of allergic reactions can be accounted for by a few foods (8). The offending foods include cow's milk, hen's eggs, peanuts, nuts, fish, shellfish, wheat, and soy (9). The level of risk for fatal or near fatal reactions to the ingestion of the allergenic food is also related to the specific food. Peanuts, nuts, and fish cause most of the fatal or near fatal reactions (10, 11). Recently, a questionnaire-based survey in Britain suggested that sesame was responsible for a significant number of severe reactions (12). The likelihood of an individual developing tolerance to a food is not fully predictable and it depends mainly on the specific food (1), although strict

avoidance is considered to be helpful (13). Interestingly, allergies to fish and peanuts are less likely to subside and both of these allergies tend to develop after the first year of life (1, 14).

In 1950, Rubenstein reported a very striking case of a 31-year-old male who experienced anaphylaxis on several consecutive occasions after exposure to 'halva' and other sesame-containing products. He thus added sesame to the exclusive list of allergenic foods (15). Only 14 yr later, Torsney reported 3 patients with sesame food allergy (SFA) (16) and another 30 yr lapsed until the first series of nine adult patients with sesame allergy was reported (17). Until recently, however, SFA was more of an incidental note rather than a recognized important food allergen. Interestingly, in 1996, Perkins had to use his personal experience in order to draw attention to sesame as an important allergen (18). Four years later, we presented a cohort of 33 patients with SFA (19). Others confirmed the high prevalence of SFA in Israel, particularly in children (20–22). In addition, major allergens of sesame have been identified, including a 14-kDa protein identified as the 2S albumin precursor and other putative allergens (23, 24). While the presence of sesame allergy is now widely recognized, the natural history of SFA is not known. More recently, the subject of SFA has received attention as a global problem (25). Here, we present a cohort of 74 patients sensitized to sesame with a long-term follow-up after 45 patients with sesame allergy.

Methods

Patients

The Helsinki Review Board of the Assaf Harofeh Medical Center approved this study. The ethics committee was notified of the use of our observational data derived from the care of these patients. Seventy-six patients sensitized to sesame were recruited from the Assaf Harofeh Food Allergy Clinic and Community Allergy Clinics. Two patients were lost to follow-up and were not available for the final evaluation. The remaining 74 patients were divided into three groups. Group A: patients referred following a convincing clinical allergic reaction after ingestion of sesame (45 patients). All patients but one immediately developed rash after exposure and the other one developed respiratory symptoms. Group B: patients who were evaluated for AD and demonstrated a positive sesame prick test. In these patients, however, it was not clear whether ingestion of sesame evoked the same reaction (11 patients). Group C: patients who were evaluated for other allergic diseases and positive sesame SPT was found incidentally (18 patients). Fifteen were evaluated for food allergy other than sesame, two for utricaria, and one for asthma.

The diagnosis of SFA was confirmed by a positive history and SPT and in some of the patients by elevated specific immunoglobulin E (sIgE), and oral challenge (OC). Patients from group A were followed up with regular scheduled clinic visits for an average total of 6.7 yr after the initial event. Patients' last contact was made by questionnaires. Specific attention was paid to the following details:

1. Description of the onset of sesame exposure: age at onset; clinical presentation; quantity of sesame causing the reaction; and time from exposure to allergic response.
2. Diagnosis: Age of diagnosis and results of SPT, sIgE, and OC.
3. Follow-up: Any accidental exposure and characteristics of the reaction. Repeated SPT and OC were offered to patients.
4. Patient allergies: diseases, foods.
5. Family allergies: diseases, foods.

Patients from groups B and C continued to consume sesame and were instructed to report any reaction. As groups B and C demonstrated only sensitization to sesame they were not included in the description of the course and natural history of SFA.

Skin prick test

Skin prick tests (SPT) were performed through a standard technique with a Wyeth needle (Heinz Herenz, Hamburg, Germany). A glycerinated sesame extract (1:10; Bayer Corp., Elkhart, IN, USA), histamine, and negative controls were applied. The mean diameters of the wheal and erythema were measured after 15 min. A scoring system similar to the one utilized to study peanut allergy (13) compared the size of sesame SPT with the histamine result as follows:

0 – no wheal.
1+ – wheal less than half of the histamine diameter.
2+ – wheal equal to or greater than half of the histamine diameter.
3+ – wheal equal to histamine diameter.
4+ – wheal greater than the histamine diameter, but less than twice the histamine diameter.
5+ – wheal equal to or greater than twice the histamine diameter.

A score of 2+ or greater was considered positive. The wheal was positive in all results but one was 3 mm and above.

Specific *in vitro* immunoglobulin E

Patients' sIgE antibodies were determined using an AlaSTAT microplate allergen sIgE kinetic enzyme immunometric assay, standardized to WHO 2nd IRP 75/502 (DPC, CA, USA). Blood samples were collected by venous puncture, sera separated, and kept frozen prior to assay. The levels of sIgE more than 0.15 IU were considered positive.

Oral challenge

Open oral sesame challenge test was offered to our patients. However, only 22 agreed to perform a challenge. Ground seeds of sesame (tehina) were utilized. The initial oral dose was 0.1 g of a sesame seed salad preparation that contained 10% protein (Miki, Rishon LeZion, Israel). This dose was doubled every 15 min through 6.4 ***g*** over 90 min. The total cummulative dose was 12.7 ***g***. All reactions were recorded for type, time of onset, severity, and duration. Patients were followed for a 4-h observation period and were instructed to report any unusual symptoms after that period. Tolerance was determined when the patient reported no reaction after eating sesame.

Statistics

Data analyses were performed with SPSS 11.0 software. Differences between the groups were assessed by chi-square analysis or Fisher's exact test for discrete variables and by *t*-tests or ANOVA for continuous variables. All reported p values are two-sided.

Results

Patient characteristics

Patient characteristics are depicted in Table 1. The median age at onset of sesame allergy in group A was 1 yr (average 2.6 yr). Seventy-six percent of these patients developed sesame allergy by the age of 2. Patients in group A were referred after a reaction to a sesame-containing product. These included 29 who had ingested tehina (66%), 10 who had eaten halva (23%), and 5 who had eaten other sesame-containing products (11%). Tehina and halva were fed to infants as additives to vegetables because of their high source of calories as early as a few months of life. All patients in group B had AD with a positive SPT to sesame. Eight patients of this group were referred for evaluation of AD, two for egg allergy, and one for fish allergy. Eight out of 11 of these patients (73%) had other positive food SPT including eggs (7), peanuts (4), milk (3), fish (3), nuts (3), soy (2), rice (2), and wheat, orange, melon, tomato, mango, carrot and banana (group B). These patients did not have a history of immediate response to sesame and they were not evaluated systematically for the various allergies but the parents were instructed to avoid the suspected foods. Positive sesame SPT was found in Group C as an incidental finding: 15 of these patients (83%) were referred because of other food allergies (eight to milk, four to eggs, two to nuts, and one to each of peanuts, soy, and banana); two patients were evaluated because of urticaria; and one for asthma. As most of the patients in group C were referred because of food allergy, it was not surprising to find a significantly higher rate of allergy to other foods in this group compared with groups A and B.

Table 1. Patient characteristics

	Group A	Group B	Group C
Number of patients	45	11	18
Males (%)	53	82	72
Age at onset (yr)			
Average/median	2.6/1.0	3.1/1.2	3.8/1.2
Range	0.3–18.5	0.3–16	0.3–18.3
Age at diagnosis (yr)			
Average/median	4.5/1.6	5.6/1.8	3.7/1.2
Range	0.3–29	0.5–32	0.3–18.3
Current age (yr)			
Average/median	11.7/8.3	9.1/7.3	8.0/5.7
Range	2.2–54.2	3.0–35	3.1–20.6
History of other allergic diseases (%)	33 (73)	10 (91)	12 (67)
History of other food allergy (%)	31 (69)	8 (73)	18 (100)*
Family history of allergic diseases (%)	25/41 (59)	5/10 (50)	9/15 (60)

*All patients in group C were referred to because of food allergy.

The clinical symptoms of sesame allergy in group A are presented in Table 2. Almost all patients had skin involvement (98%) followed by respiratory (58%) and gastrointestinal (33%) symptoms. Anaphylactic shock was observed in two patients. Immediate reaction to a small amount of sesame (by history) characterized sesame allergy (Table 3).

Diagnosis

Diagnosis of sesame allergy was based on medical history and a positive SPT. SPT was positive in all 72 patients where it was performed. Sesame-sIgE antibodies were measured in 21

Table 2. Clinical symptoms of patients with sesame food allergy (number = 45) (group A)

System	Number	Percent
Skin	44	98
Erythema	30	67
Angioedema	25	56
Pruritus	23	51
Tears	21	47
Urticaria	18	40
Respiratory	26	58
Rhinorrhea	17	38
Wheezing and dyspnea	16	36
Cough	9	20
Nasal itching	5	11
Stuffy nose	4	9
Gastrointestinal symptoms	15	33
Vomiting	11	24
Nausea	6	13
Diarrhea	2	4
Abdominal pain	1	2
Irritability	21	47
Shock	2	4

Table 3. Characterization of reaction to sesame in group A

Reaction time less than 10 min	38 patients (88%)
Reaction time 10–30 min	3 patients (7%)
Reaction time up to 2 h	2 patients (5%)
Response to small amount (by history)	30 patients (70%)
Response to more than small amount	13 patients (30%)
Positive SPT	44/44 patients (100%)
Elevated sIgE	9/12 patients (75%)
Positive food challenge	14/16 patients (88%)

Data is missing for two patients.
SPT, skin prick test; sIgE, specific immunoglobulin E.

patients, but were found elevated in only two-thirds of them. OC was performed in 21 patients and was positive in 14 (67%). All positive challenges were in patients from group A. In group A, OC was performed in 16 patients and confirmed the diagnosis in 14 patients (88%) (Table 3). In the two patients where the challenge was negative, the interpretation was that the patients may had developed tolerance, as in both cases the age at onset of sesame allergy was 8 months and the challenges were carried out about 1 yr later. The single patient from group B who underwent OC could tolerate sesame. In group C, four patients underwent OC which were negative. However, another patient from this group was accidentally exposed to sesame and experienced anaphylactic reaction.

The level of sesame-sIgE was measured in 12 patients from group A and was elevated in nine (Table 3). The range of positive sIgE was 0.36–88 IU. Noticeably, a 5.5-year-old girl had anaphylactic shock upon exposure to sesame and a strongly positive SPT, but negative sIgE antibodies. All four patients with elevated sIgE to sesame who underwent OC did not tolerate sesame.

Size of SPT and relationship to disease

The average scoring size of positive SPT was 4.4+ and the range was 2+ to 5+. There was no significant difference in the scoring size of SPT between the three groups. No correlation was found between the size of SPT to the level of sIgE, the outcome of OC results, and the development of tolerance to sesame (Table 4).

Development of tolerance

The average period of follow-up for group A was 6.7 yr (median 6.4 yr, range 1.8–14 yr). During this period, nine patients developed tolerance (20%). The characteristics of the patients who developed tolerance and those with persistent SFA are presented in Table 4. The median active disease period which represents the duration from first reaction to tolerance was 2.3 yr (average 3.7 yr, range 0.8–11 yr). Eight out of nine patients who developed tolerance were less than 6.5 yr old. The median age at tolerance was 4.4 yr (average 6.8 yr). Clinical scoring and severity of symptoms were found not to be predictive for the development of tolerance. The age at onset, scoring size of SPT, history of allergy to other foods, and other concomitant allergic diseases in the patients and families were also found to be non-predictive (Table 4). On the other hand, behavioral changes, such as irritability and crying, revealed by exposure to sesame

Table 4. The characteristics of patients who develop tolerance and those with persistent allergy in Group A

	Tolerance (n = 9)	Persistent allergy (n = 36)
Age (year) at initial event (average/median)	3.1/1.0	2.2/1.0
Age (year) at diagnosis (average/median)	3.6/1.5	3.8/1.7
Active disease period (average/median)	3.7/2.3	9.2/6.6
History of other food allergies (%)	7/9 (78)	24/36 (67)
History of other allergic diseases (%)	5/9 (56)	26/36 (72)
History of food allergy in family (%)	2/9 (22)	7/36 (19)
History of allergic diseases in family (%)	6/9 (67)	18/36 (50)
Emergency department visit (%)	2 (22)	9 (25)
Wheezing and dyspnea (%)	2 (22)	14 (39)
Shock (%)	0	2 (6)
Irritability (%)	1	20 (56)*
Average scoring of size of skin prick test	4.2	4.4
Average of systems clinically involved**	1.8	1.8

*$p = 0.004$.
**Skin, gastrointestinal, and respiratory systems.

were found to be significantly higher in patients with persistent sesame allergy ($p = 0.004$). The odds ratio of persistent sesame allergy was estimated using logistic regression to be 11.16 for irritability ($p = 0.02$), and 2.56 for other allergic diseases in patients ($p = 0.08$).

Discussion

We present here a large cohort of patients with SFA, describing the age of onset, presentation, diagnosis, and a prolonged follow-up. The clinical spectrum of sesame sensitization ranged from an incidental positive SPT performed for unrelated conditions up to a full-blown allergic reaction. The clinical presentation of SFA was similar to the clinical presentation of other food allergies. Skin involvement was observed in almost all cases followed by the respiratory symptoms including rhinorrhea, wheezing, dyspnea, and cough. Although skin involvement in food allergy is rather common the high prevalence of rash (44/45 patients) in reactions to sesame is exceptional. The gastrointestinal system was also a common site of reaction with vomiting and nausea, but was less frequently involved than skin or respiratory systems. Interestingly, behavioral changes, such as irritability and crying, were also frequent symptoms in SFA. Furthermore, these symptoms were found to be a high risk factor for persistence of clinical allergy. Anaphylactic shock was uncommon in this cohort. In contrast to Kanny's report that five out of nine patients with SFA presented with anaphylaxis (26), in our series, life-threatening anaphylactic shock was observed in only two patients. Additionally, unlike a previous report, not all children in our series with SFA had AD (20).

Most patients displayed the allergy early in life, prior to their second birthday. This finding is in agreement with previous reports from Israel (20–22) but in contrast to reports from Europe and the United States (25). One potential explanation for this result is the earlier exposure to sesame (tehina) in the diets of infants in Israel, which is often given because of its high caloric content.

The gold standard to confirm food allergy is a double-blind placebo-controlled food challenge (DBPCFC) (8). We made the diagnosis of SFA primarily on extensive clinical history and positive SPT (average size 4.4+ in relation to histamine). Other investigators have also argued that if extensive medical history is obtained and supported by a positive SPT (27) or sIgE quantification, less than half of the patients need challenge (28). In the evaluation of a large series of food allergies, a DBPCFC, which is time-consuming, hard to perform in children, and not without risk, is not common (14, 29, 30). Furthermore, asthma attack was found more frequently in sesame OC (42%) than in milk, egg, and peanut challenges (31). Nevertheless, food challenges were offered to all patients, but less than half of the eligible patients elected to undergo the procedure. Among those who did, the vast majority were proven to have SFA, by a positive challenge.

In this cohort, SPT was found more reliable than sIgE in confirming allergy, in agreement with the previous reports (27, 29, 32). While SPT was positive in all patients who were examined, sesame sIgE antibody measurements were less sensitive, and missed one out of four allergic patients. This rate of sensitivity of sIgE, while higher than in a previous study (30) (perhaps due to improved reagents), still is significantly less sensitive than the SPT. The dissociation between the skin and serum markers of sIgE may be because the high affinity of sIgE to its receptors on mast cells and basophils favor the tissue pool until tissue saturation is achieved, relatively depleting sIgE circulating antibody levels (27). Since summarizing this cohort we have found several additional patients with extensive history and positive SPT without elevated sIgE for sesame. Therefore, until a better system is available, *in vitro* sIgE antibodies for sesame cannot be used to predict clinical reactivity as can be performed for egg, cow's milk, peanut, and fish allergies up to a 95% predictive value (33).

Perhaps associated with the poor specificity of the sIgE to allergy, it was noted that there was no correlation between the size of SPT to sIgE and OC results. The same results were reported by Clark and Ewan for nuts allergy (29). This is in contrast to the work of Hill et al. (27) who used 100% level of SPT size to spare DBPCFC for cow's milk, egg, and peanut allergies. Positive SPT to sesame can be an incidental laboratory finding and part of the patients may not have clinical food allergy, as with other foods and in agreement with a previous report (22).

The data obtained here indicate that most patients with SFA obtain it early in life and about 80% will continue to suffer from SFA. It appears that SFA belongs to the group of foods such as fish (34) and peanuts (14) that have a low rate of development of tolerance. This is in contrast to cow's milk protein and hen's egg protein, to which a high percentage of children develop tolerance (1, 34). However, unlike fish allergy (35), SFA tends to appear earlier in life (20). The

pattern of age of diagnosis and rate of development of tolerance is very similar to peanuts (14). Almost all the patients in the present study who developed tolerance did so within 6 yr. We noted only one patient who developed tolerance later. From our data, there was no evidence that the younger the age of acquisition of the allergy, the higher the chance for the development of tolerance, at least in the age group of above 1 yr.

Based on these results, and the extent of follow-up in this study (average 6.7 yr, median 6.4 yr), we can estimate that the sesame allergy tolerance rate is only 20%. The follow-up period in this study is longer than that of other studies of food allergy tolerance. In a recent review of the natural history of food allergies (1), the data from six series on cow's milk allergy was based on follow-up periods between 6 months and 5 yr. The median follow-up in one series on egg allergy was 17 months (36) and the follow-up duration in a series on food allergies in children with AD was 2 yr (37). Bock followed up children with adverse reaction to foods until their third birthday (3). Several additional series similarly followed patients with food allergies for up to 5 yr (4, 14, 38, 39).

With the rise in the use of sesame in the westernized diet, physicians need to have an increased recognition and awareness of its potential allergic side effects (40). In addition, the potential for cross-reactivity to other nuts must be explored. For instance, oleosins were recently described to be a major allergen in sesame seeds (41), hazelnuts (42), and peanuts (43). However, in our set of patients we did not notice a significant cross-reactivity with either peanut or hazelnut. This highlights the need for further study and identification of sesame's clinically relevant allergens (23, 24).

In summary, SFA is a very common food allergy in Israel (19, 22), tends to appear early in life, and persists to adulthood in most cases. A comprehensive clinical history combined with an SPT establishes the diagnosis while sIgE is not yet a reliable measure for the exclusion of SFA.

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