Successful Oral Desensitization in Sesame Allergy in an Adult Woman

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IgE-mediated sesame allergy affects 0.1%-2.8% of the population depending on genetic and environmental factors [1]. Affected patients present a wide spectrum of clinical manifestations that span from wheals to anaphylaxis [2]. In most instances, this allergy is persistent and rarely resolves spontaneously [3]. Currently, most patients with sesame allergy are instructed to avoid contact with foods containing the food allergen. However, studies have shown that contact with as little as 0.13 mg of sesame protein can trigger a reaction [4], thus implying that food containing trace amounts of the antigen must be avoided. The development of methods aimed at desensitizing patients to sesame protein could provide nutritional safety for persons at risk.

A search based on the terms sesame, allergy, desensitization, and treatment revealed only 1 study in which the authors used multiple allergen desensitization to control the clinical manifestations of sesame allergy [5]. The diagnosis of sesame allergy was based on double-blind placebo-controlled challenge, which was positive in 24% of patients. The desensitization protocol was divided into 3 phases: an initial escalation day, home dosing, and a maintenance phase. Up to 5 distinct antigens were administered orally beginning with an individual dose of 0.1 mg, which was increased to a maximum of 5.0 mg on the initial escalation day. During the home-dosing phase, patients took a biweekly dose, increasing from 6.0 mg up to 4.0 g. Finally, during the maintenance phase, patients were instructed to take 4.0 g of each food allergen continuously, with visits at varied intervals, depending on the clinical outcomes obtained during desensitization. The authors reported a change in the blood levels of sesame-specific IgE and IgG4. No specific comments regarding the clinical outcomes of the patients with sesame allergy were provided, although the authors reported a 40% reduction in the percentage of adverse events per dose in peanut allergy during the initial escalation day to 6% during the maintenance phase and that a similar trend was observed for the other antigens, including sesame. There are no published protocols on sesame allergy.

We report a case of oral desensitization to sesame. The protocol was approved by the Medical Research Ethics Committee of the University of Campinas, Campinas, Brazil (#89404518.0.0000.5404).

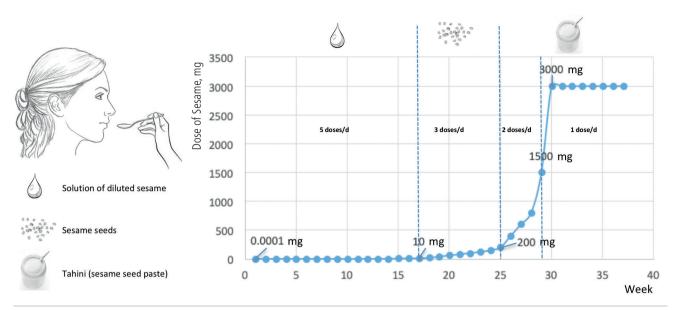


Figure. Graphic representation of the sesame desensitization protocol. In the first phase (day 0-week 16), the patient was given diluted crude white sesame extract in escalating doses beginning with 0.0001 mg up to 10 mg, 5 doses/d (every 4 h, during waking hours). Doses were increased on a weekly basis, under medical supervision. In the second phase, the patient was given 3 doses/d of crude white sesame seeds for 8 weeks, beginning with 10 mg and escalating on a weekly basis up to 150 mg, under medical supervision. In the third phase, tahini was given twice a day, and doses were increased twice a week, beginning with 200 mg and increasing to 800 mg. In the fourth phase, 1500 mg tahini was given once a day, for 1 week and then 3000 mg once a day for 4 weeks. The patient then underwent sesame-rich food challenge, which was negative. Thereafter, the patient moved to the maintenance phase, with 3000 mg of tahini once a day, continuously.

The patient was a 48-year-old white woman who reported anaphylaxis or skin-restricted reactions beginning at age 27 years following the consumption of sesame-rich foods and even after the consumption of trace amounts of the antigen. She required repeated administration of epinephrine for recurrent episodes of anaphylaxis (7 episodes over a 20-year period). On admission, blood sesame-specific IgE was negative, consistent with data reported elsewhere [6]. However, prick-to-prick testing revealed positive responses to crude and toasted white and black sesame and also to tahini, a condiment made of toasted ground hulled sesame. We subsequently prepared a solution containing 10 mg/mL of crude white sesame extract and performed a dose-response prick-test, which yielded a positive result in a dilution of up to 1:100. The following oral desensitization protocol was then applied. First, the patient was given 2 doses of 0.1 mL of a solution containing 0.1 µg of crude white sesame extract with a 30-minute interval between doses, and a clinical evaluation was performed over 2 hours. Next, the patient was given 3 additional doses of the same extract with a 4-hour interval to complete 5 doses/d. There were no clinical manifestations, and the protocol continued for 6 additional days with 5 doses/d with the same amount of sesame extract. The dose was escalated weekly for 15 weeks. The patient was given 5 doses/d of the following amounts of the extract: 0.2 µg, $0.5 \,\mu g$, $1.0 \,\mu g$, $2.0 \,\mu g$, $5.0 \,\mu g$, $10 \,\mu g$, $20 \,\mu g$, $50 \,\mu g$, $100 \,\mu g$, $200 \,\mu g$ μg , 500 μg , 1.0 mg, 2.0 mg, 5.0 mg, and 10 mg. Then, she was given 3 doses per day of 10 mg of crude white sesame seeds; this regimen was maintained for 8 weeks, with escalation to reach a dose of 150 mg. Desensitization continued with tahini, beginning with 2 doses/d and 200 mg/dose increasing

2-fold/wk up to 800 mg/dose. Finally, the patient was given 1 dose/d of tahini beginning with 1.5 g/dose in the first week and 3.0 g/dose in the second week. The dose of 3.0 g/day was maintained for 4 weeks, and the patient underwent a sesamerich food challenge test, which was negative. Moreover, skin prick testing performed with a solution containing 10 mg/mL of sesame extract was negative in all dilutions from 1:10 000 to 1:1. Desensitization was considered successful, and the maintenance phase was based on a dose of 3.0 g of tahini once a day. The patient can now freely consume foods containing sesame with no clinical manifestations. She was recommended to maintain the daily consumption of sesame indefinitely and keep an epinephrine autoinjector to hand.

The skin prick test was always performed in parallel with a positive control (histamine) and a negative control (dilution buffer). Wheals >3.0 mm were considered positive for as long as the negative control presented no wheals. During some steps of the desensitization protocol, the patient presented minor symptoms including itching and wheals, which were controlled using antihistamines.

In conclusion, this is the first report of successful oral desensitization to sesame. Only 1 previous report has examined simultaneous desensitization to a number of distinct food antigens [5]. As we report a case, we cannot propose a definitive protocol, although we believe the approach we applied may provide the basis for further investigation in the field. We acknowledge that the protocol is complex and not easily applied in very active people or children and that not providing the actual amount of sesame protein used in the protocol is a limitation.

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Conflicts of Interest

The authors declare that they have no conflicts of interest.

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