

Hypersensitivity to sesame seed

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Allergic reaction to sesame seed was first reported and described in 1950 by Rubenstein.¹ The patient was a 31-year-old Caucasian man who, shortly after the ingestion of the confection halvah, suddenly experienced tightness in the throat, nausea, vomiting, weakness, and generalized pruritic erythema. On at least two previous occasions there had been similar reactions; clinical shock occurred with a fourth episode. Intradermal skin testing with sesame extract revealed a significant reaction, with a negative reaction in a nonsensitive patient.

Another similar case was reported by Uvitsky² in 1951. This was a 40-year-old man who, within 10 minutes after the ingestion of a piece of halvah, began to experience burning and tingling of the lips and throat, followed by angioedema of the lips, tongue, and uvula, and, lastly, wheezing respiration. Direct and passive transfer testing revealed significant skin reactivity to sesame seed antigen. Clinical symptoms were reproduced with the ingestion of sesame seed in a blind test, with suitable control.

Since that time there have been no further reports in the literature, to my knowledge. Recently, I have personally seen and evaluated three similar cases; a fourth case was brought to my attention but was not available for further diagnostic study. My observations of the unusual frequency of such a rare sensitivity reaction and of statistics which suggest an increasing consumption of sesame seed in this country have occasioned the review of previous literature and the presentation of the following three additional case reports.

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CASE REPORTS

Case 1. A 19-year-old Caucasian man ate one piece of halvah in December, 1962. Within 5 minutes he experienced paresthesia of tongue, palate, and posterior pharynx. The symptoms progressed to profound pruritis and difficulty in swallowing, generalized erythema, generalized pruritis, and wheezing. The dispensary at which he was examined documented the angioedema of the tongue and uvula, generalized erythema, and wheezing. The patient complained of marked weakness, the blood pressure was 80/40, and there was tachycardia, the pulse rate being 140 per minute. He was treated with epinephrine subcutaneously and hydrocortisone intravenously; within 3 hours there were no residual signs of the attack.

Prior to the age of 12 years he had had urticaria twice. His mother attributed this to eating peanut butter, and she eliminated it from his diet. Now, however, the patient is able to tolerate both peanut butter and peanuts without ill effect. We learned that when the patient was 6 years old urticaria had developed after ingestion of sesame seed crackers and that there had been three other instances of urticaria between the ages of 6 and 9 years after ingestion of sesame seed-containing foods which were controlled with oral antihistamines. For the 10 years from age 9 until the present, the patient has tried not to eat foods with sesame seeds. Inadvertently he sometimes ate small portions of foods which produced palatal, glossal, and posterior pharyngeal pruritis; each time, the foods contained sesame seed and the symptoms subsided soon after he took one 50 mg. Benadryl hydrochloride (diphenhydramine hydrochloride) capsule. During the recent severe reaction he had no antihistamines available. He specifically denied all other allergic symptoms and knowledge of other allergens. Family history revealed significant allergic symptoms in his father and two of four siblings.

We determined the content of the offending halvah confection to be crushed sesame seed, corn syrup, sugar, egg albumin, and cottonseed oil shortening. Intracutaneous skin testing was performed with antigens obtained from Hollister-Stier Laboratories. The reactions were negative to peanut, soybean, cottonseed, flaxseed, corn, egg albumin, ragweed, and timothy. We prepared a crude sesame seed extract by crushing 1 Gm. of sesame seed and extracting it in 10 c.c. of Coca's fluid for 48 hours and made a 1:1,000 solution from the concentrate. Scratch testing with the solution caused an intense reaction with a wheal and pseudopods. After 4 minutes we removed the antigen from the arm when he complained of marked local pruritis. Within 10 minutes there was associated palatal and pharyngeal pruritis which responded to the administration of 50 mg. of Benadryl.

Scratch testing with histamine 1:10,000 elicited marked local pruritis, wheal, and pseudopod reaction, but no associated constitutional symptoms. The crude sesame extract, in concentrations of 1:1,000 and 1:100, gave negative scratch tests in five atopic and five nonatopic patients. Passive transfer testing with the patient's serum in a nonatopic patient revealed a wheal and pseudopod reaction to the sesame seed extract, as well as a negative control.

We fed the patient large quantities of corn syrup, egg white, and cottonseed oil for 3 successive days with no adverse reaction. We withheld feeding sesame seed to him in view of the seriousness of the previous reaction and the fact that a mild constitutional reaction to scratch testing was sufficient evidence of the clinical problem.

We soaked a cotton pledget with sesame oil and placed it in contact with the buccal mucous membrane for 20 minutes without noticeable reaction. We fed the patient sesame, cottonseed, peanut, and corn oils in a blind fashion on separate days. Again, no symptoms resulted.

Case 2. This 64-year-old Caucasian man ate several sesame seed crackers while drinking cocktails. Within minutes glossal, palatal, and posterior pharyngeal pruritus developed, progressing to difficulty in swallowing and associated with generalized erythema and pruritis. He became lightheaded and felt nauseated. He was brought to the emergency room where generalized erythema, angioedema of the tongue and uvula, blood pressure of 60/40, and pulse of 132 per minute were found. He was treated with epinephrine subcutaneously and Benadryl parenterally, and within 3 hours he was asymptomatic. The physical examination was then normal.

The patient said that twice in the past, shortly after he had eaten sesame seed rolls, mild palatal and glossal pruritis and scattered urticarial lesions occurred. With the onset of

these symptoms he had taken a Chlor-Trimeton tablet, which he felt modified any progression of the symptoms. On the night of the severe reaction, however, he had no antihistamines on hand. He related that for the past 10 years, while living in the eastern United States, he had symptoms compatible with seasonal allergic rhinitis which he controlled easily with oral antihistamines. The ingestion of watermelon and cantaloupe causes mild oral pruritis only, and the extent of this reaction is so limited that he eats melon deliberately. On specific questioning he denied any other allergic manifestations or allergens.

He denied any familial allergic predisposition. Scratch testing with a 1:1,000 crude sesame seed extract produced a large wheal and pseudopod reaction. Intracutaneous skin testing showed significant positive reactions to ragweed, maple, ash, and sycamore, and there were negative reactions to molds and many foods in common use. Passive transfer with the patient's serum in a nonatopic individual revealed a wheal and pseudopod reaction to the sesame seed extract, with a negative control.

Sesame oil was soaked on a cotton pledget and placed in contact with the buccal mucous membrane, without noticeable reaction. We fed sesame oil to the patient in a fasting state, and no clinical symptoms resulted. It was not felt that we should feed the patient sesame seed because of the severity of his previous reaction to it. In fact, the patient refused to accept the feeding.

Case 3. This 40-year-old Caucasian woman said that from her teens until about age 23 she had had adverse reactions when she ate sesame seed or coconut. Her reactions to both foods were alike in type and in severity and were progressive in character over these years. At the beginning of an attack, oral, palatal, and posterior pharyngeal paresthesia and pruritis occurred. The reaction would begin shortly after eating the food and would persist for 20 to 30 minutes. Over the years, in addition, there were scattered urticarial lesions of short duration. When she was 22 years old, within 5 minutes after the ingestion of sesame seed, she had oral, palatal, and glossal paresthesia and pruritis, difficulty in swallowing, generalized erythema and pruritis, marked swelling of the palmar surfaces bilaterally, difficulty and discomfort in bending her fingers, wheezing respirations, and nausea and vertigo without syncope. She was given several injections; since her reactions had been so severe and had not lessened within 2 hours, she was admitted to a hospital for 12 hours. On discharge, she was asymptomatic.

Over the past 17 years she was meticulous in avoiding sesame seed and coconut, and on no occasion had she had any other similar symptoms until April, 1964. At this time she was served rolls topped with sesame seeds. She removed the seeds; however, after the first few bites she noted the onset of oral and pharyngeal paresthesia and pruritis. Within 5 minutes the symptoms progressed to generalized erythema, pruritis, and wheezing respirations. She immediately took two 4 mg. Chlor-Trimeton (chlorpheniramine maleate) tablets; because she was asymptomatic within 30 minutes and observed that the severity did not equal the previous reaction as she recalled it, she felt that this drug had arrested the progression of her symptoms.

Twelve years before, during the last trimester of her second pregnancy, she had had bronchial asthma. This persisted for 2 weeks post partum, but to the time of this admission, this condition has not recurred. Family history revealed that her mother had urticaria.

Scratch testing to crude sesame seed extract 1:1,000 produced a significant wheal, and the 1:100 dilution produced a wheal with pseudopods. Intradermal skin testing produced significant reactions to chocolate, pecan, walnut, house dust, and hormodendrum. Negative reactions were obtained to ragweed, timothy, maple, alternaria, and fusarium.

Passive transfer in a nonatopic person revealed a significant reaction to crude sesame extract and a negative control. Sesame oil was placed in contact with the buccal mucous membranes by means of a cotton pledget, without noticeable reaction.

The patient refused to consider the ingestion of either sesame seed or oil, on the basis that she needed no confirmation of her clinical history.

DISCUSSION

Sesame indicum and its congener, *S. orientale*, are members of the *Pedaliaceae* family and are herbaceous plants native to the tropics of both the Old and the

New World. In addition to the cultivated *S. indicum*, there are at least eighteen wild species of the genus *Sesamum*.³ The plant is an annual. It is variously known as benne, til, simsim, and ajonjoli. It has a grooved, capsular, two-celled fruit which contains many oily seeds. For centuries the plant has been grown as a crop in Asia and Africa, with China and India being the chief producers. World production of sesame seed in 1962, excluding mainland China, is estimated provisionally at 1.25 million short tons, a 5 per cent increase above the 1955 to 1959 average output.⁴ In the United States, sesame seed has been produced commercially only since 1950, and in 1957 about 9 million pounds⁵ were harvested. The production of sesame seed in North America in 1962 was 166,000 short tons, which represents an approximate 17 per cent increase above the average output from 1955 to 1959. Heretofore, the unpredictability of seed ripening time and difficulty in machine harvesting had restricted the planting of this crop. The recent development of improved nonshattering varieties of sesame, which are more disease resistant and which have better seed quality and greater adaptability to mechanized harvesting, has increased the total yearly crop in this country. Despite this fact, imports of sesame seed into the United States from 1959 to 1962 have increased from 13 to 20 million pounds per year.⁶

Historically, we find sesame seed being used as currency during the sixth century B.C.; the Greek historian Herodotus (circa 485 to 428 B.C.) recorded that the culturally advanced Babylonians used sesame oil to the exclusion of all others.

At the present time, annual world production of sesame oil approaches that of olive oil. Sesame oil is for the most part expeller-extracted⁷ and can be obtained in both refined and unrefined forms. One method of preparation is simply that of applying heat to the unhulled seed and then putting it through high pressure cylinders. The crude oil is purified by alkali refining, filtration, and steam deodorization. It appears inconceivable that any protein allergen could retain its activity after such processing.⁸ Normally, sesame seed oil content ranges in per cent from approximately 45 to 63, with an average of 54.⁹ This high oil yield enhances the value of the sesame seed over that of any other edible oilseed.

The marketable sesame products are the whole seed, the extracted oil, and the residual chaff as meal. The expressed oil is used as salad or cooking oil, in shortening, margarine, cosmetics, perfume, and in some pyrethrum insecticide sprays as a synergist. The oil has served the pharmaceutical industry for many years as a vehicle for parenteral injection because it is less antigenic than cottonseed or peanut oil.¹⁰ The sesame meal residual from the pressing operation is a good cattle and poultry feed. In addition, experiments have been and are being conducted to reduce sesame meal into an almost pure protein product to be used in fortifying food products,⁷ and a patent has been granted for such a sesame seed flour.¹¹

The whole seed is used as an ingredient of, or in topping for, bread, cakes, pastries, and candies. The ground seed has a nutlike flavor, similar to that of almond, and frequently is used as an almond substitute. Foods containing sesame seed are as varied as are the countries which produced the plant. A sesame

seed paste called taheeni, tahini, tachini, or tahin, depending on the country of origin, may be purchased in cans from many food specialty stores. This paste is used by the Chinese in making sesame paste noodles, a dish called Gee See La Pae and another dish called Chinese Chrysanthemum Pot in this country. In Turkey the paste is used to make helva, a dessert which is called halva or halvah in other parts of the world.

Simit is a form of Turkish pretzel which contains sesame seed, as do some celery pretzels, sesame wafers, crackers, and pretzels in this country. Tartatour bi taheeni is an eggplant and sesame paste. Hoomis bi tahini is a sesame paste with chickpeas. Both may be served as an hors d'oeuvre dip for crackers and breadstuffs. Baba Ghanouj is an eggplant dish containing sesame seed. Benne seed cakes are a traditional dish in some areas of the southeastern United States. Mexican pablano with mole sauce likewise contains sesame seed. All of these foods can be obtained in Washington, D. C., and are probably available in gourmet restaurants throughout the United States.

Sesame seed is considered to be an extremely potent allergen and a severe allergic reaction to it should present an easily recognized pattern to the alert clinician. The clinical picture usually presented is one of oral and glossal pruritis, generalized erythema, angioedema of the uvula, clinical shock, and wheezing. The treatment of choice in such a patient is epinephrine subcutaneously, as is administered in instances of any anaphylactoid reaction, and supplemented if necessary by other supportive measures such as antihistamines.

It should be noted that in all the reported sesame seed-sensitive patients, there had been an apparent progression of the clinical reaction with repeated antigen challenge. Several of the patients felt that the prompt ingestion of oral antihistamines at the onset of symptoms either aborted or suppressed the clinical symptoms. For both of these reasons it is important to recognize sesame seed sensitivity without delay so that the patient may (1) eliminate the causative agent, and (2) use suitable medication in the event of a dietary indiscretion.

The most effective form of therapy of any allergic disease is prevention through education. In the case of sesame seed hypersensitivity, the elimination of sesame seed from the diet should not be difficult if the patient is apprised of the general food groups which contain it. Likewise, he should be educated to read prepared food labels so that he may forestall the unwitting ingestion of sesame seed. The following is a list of producers and some of their crackers and confections which contain sesame seed.

F.F.V. Brand. Vega Thins, Wheat Thins.

JOYVA Corporation. Halvah (pastry bars).

National Biscuit Co. (NABISCO). Cracker Chatter, Sesame Thins, Sociables, Triangle Thins.

Burrry's Division of Quaker Oats. Oval Thins, Euphrates Wheat Wafers.

Sesacraft, Inc., Sesa Treat Products. Sesame Chips, Sesame Sticks, Sesa-Treat.

S.S. Pierce Co. Overland Cocktail Tidbits, Sesame Niblets, Sesame Seed Loaf.

Sunshine Biscuits. Sesame Cheese.

There is no evidence, on review of all the reported cases, that sesame oil has

ever been responsible for the production of clinical symptoms. In two cases reported here, the patients were fed sesame oil without adverse effect. This correlates well with the classic investigative works of Spies, Bernton, and Stevens,^{12, 13} Figley,¹⁴ and Layton and associates¹⁵ who have shown that many edible vegetable oils are not allergenic. It should be pointed out that serious constitutional reactions and deaths^{16, 17} have been reported from intradermal testing with water-soluble seed extracts and that scratch testing should be performed prior to the deliberated consideration of intradermal skin testing.

In light of the threefold factors, the increasing total world production of sesame seed, the stepped up North American production and the quantity of United States imports, the incidence of hypersensitivity reactions may well increase proportionately. My experience would appear to support this assumption. Also, if, as previously stated, the experiments being conducted to reduce sesame meal into an almost pure protein product for fortifying food products are successful, the number of hypersensitivity reactions may increase greatly.

SUMMARY

Severe reactions due to hypersensitivity to sesame seed are characterized by oral and palatal pruritis, generalized erythema, wheezing, and clinical shock. Such reactions are usually preceded by milder ones, which, if promptly and correctly diagnosed, may prevent clinical progression of symptoms by elimination of the offending food from the diet. Sesame oil ingestion has not produced any similar clinical reactions, to our knowledge. The total consumption of sesame seed in the United States is increasing, a fact which suggests that the incidence of sesame seed hypersensitivity will probably increase in like proportion.

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