

ethylenediamine derivatives. A high frequency of sensitization to 1,2-diaminopropane is plausible due to the greater penetration capacity of its carrier, the nonionic, highly lipophilic *N,N'*-disalicylidene-1,2-diaminopropane, as compared to the (at physiologic pH) protonated ethylenediamine.

Sensitivity to ethylenediamine is also important in asthmatic patients treated with aminophylline, since this preparation is a complex formed of theophylline and ethylenediamine. Patients sensitive to ethylenediamine have been reported to manifest systemic reactions when treated with such preparations (4).

Key words: adhesive plaster; contact dermatitis; *N,N'*-disalicylidene-1,2-diaminopropane; ethylenediamine.

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Non-IgE-mediated anaphylaxis to sesame

SESAME (*Sesamum indicum*, Pedaliaceae family) is grown extensively for its seeds throughout the tropics and subtropics in the Middle East, the USA, China, Asia, and Latin America. Sesame is currently used for the production of whole seeds, extracted oil, and seed paste (tahini). Common foods containing sesame seeds are vegetarian burgers, Oriental food, bread, cakes, pastries, toppings, spice mixtures, salad dressings, and diet food. The presumed low antigenicity of sesame oil has led to its use in the pharmaceutical and cosmetic industries as a vehicle for parenteral and local therapeutics. This increasing consumption of ses-

ame might explain the growing frequency of reported cases of immediate-type systemic reactions after ingestion and/or inhalation of sesame seeds and/or oil in recent years (1-8).

■ We present a patient with recurrent anaphylactic reactions after ingestion of sesame, although no evidence of an IgE-mediated mechanism was found. A 45-year-old woman had facial erythema, flushing, generalized pruritus, and conjunctivitis after she had eaten crackers. Similar symptoms occurred several times after ingestion of sesame oil in different preparations. Within minutes after ingestion of sesame-dressed chicken, she had flushing, generalized itching, nausea, vomiting, and dizziness. She had no personal or familial history of allergic and atopic disorders. Routine skin prick and intracutaneous tests were all negative for a standard series of common inhalant allergens and for an extensive panel of food allergens. Prick and scratch tests with poppy seeds, carob-bean flour, german wheat, flaxseed, pine nuts, mustard seeds, and sunflower seeds were negative. Prick and scratch tests performed with native sesame oil (cold-pressed and heat-extracted) and raw, peeled, crushed, and unpeeled sesame seeds did not show any skin reaction. The total serum IgE level was 13.1 kU/l. Specific IgE antibodies could be detected neither against sesame seeds nor against a panel of common inhalant allergens (sx1) (Pharmacia CAP System, Uppsala, Sweden). A double-blind, placebo-controlled food challenge (DBPCFC) was carried out in the outpatient clinic with full facilities for resuscitation. The challenge was performed with crushed sesame seeds (10 g) and sesame oil (10 g) masked in bread. The placebo bread was baked with hazelnut (15 g) and hazelnut oil (10 g). No adverse reaction was observed to the oral intake of hazelnut-containing bread. Two hours after provocation, no raised value of tryptase in serum (Pharmacia CAP System) or *N*-methyl-histamine in urine was found. Ten minutes after ingestion of two mouthfuls of sesame-containing bread, the patient developed generalized flushing, itching, and conjunctival symptoms. The symptoms disappeared within 20 min after treatment with intravenous antihistamine and steroids. The urine analysis (2 h after exposure) demonstrated a markedly elevated level of *N*-methyl-histamine (10 920 µg/g creatinine, normal range 70-160 µg/g creatinine). No elevated level of serum tryptase was detected (11 µg/l, normal range 5.6-13.5 µg/l). The later scratch test with the above-mentioned bread remained negative. Serologic analysis 8 weeks after the DBPCFC did not show any sesame seed-specific IgE antibodies.

■ Sesame seeds or oil is still a rare, but extremely potent cause of food allergy (6–8). We present a case of presumed monovalent sesame allergy with negative skin tests and without detectable specific IgE antibodies. Recent studies have shown the poor sensitivity of detecting sesame seed-specific IgE antibodies (7) in sesame-allergic patients. The objective symptoms and the significant release of histamine demonstrated an activation of basophil leukocytes and/or skin mast cells. In our case, the level of serum tryptase – as a specific indicator of human mast-cell involvement – was not elevated. This observation may correspond with previous studies which demonstrate variable levels of mast-cell tryptase after controlled challenges. More likely are single activation of basophil leukocytes and no human mast-cell involvement. It has been suggested that potential allergens in sesame are sensitive to heat (1). For our DBPCFC, sesame oil and seeds were hidden in baked bread. Thus, there are relevant allergens in sesame which are heat-resistant. It is also possible that through the procedure of heating, neoallergenic determinants, not present in unheated, native sesame, are created. The fact that the skin tests were negative suggests that the anaphylactic reaction was not IgE-mediated (non-immune-mediated food intolerance). Another hypothesis is that through digestion new and unknown allergenic epitopes are formed. A review of the literature shows that further investigations are needed to clarify the relevant allergenic components in sesame seed and sesame oil.

■ In conclusion, we report the potential seriousness of a missed diagnosis of sesame allergy. At present, although detailed allergologic history and examinations are performed, the DBPCFC remains the “gold standard” in diagnosing sesame allergy (9).

Key words: anaphylactic reaction; double-blind, placebo-controlled food challenge; *N*-methyl-histamine; sesame oil; sesame seeds; tryptase.

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Gastrointestinal and skin symptoms caused by sesame with negative skin tests, no measurable specific IgE response, and positive DBPCFC.

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Evaluation of nasal cytology: a comparison between methods

THE nose is the ideal site to evaluate the allergic inflammatory mucosal response since it is readily accessible and may be repeatedly provoked. Nasal biopsy would provide ideal samples to study the inflammatory response (1); however, since it is a traumatic technique, it is hardly suitable for repeated observations. Therefore, different methods of cell collection have been proposed which allow repeated, nontraumatic samplings in the same subject (2–6). This study aimed to compare washing and scraping collections for inflammatory cell content and quality of preparations.

Samples were collected from 16 volunteers, eight atopic (out of season) and eight nonatopic, participating in a different study protocol for which they underwent a series of nasal challenges (7) with methacholine (5 mg), histamine (0.01 mg), and seasonal allergen (1300 AU) during a period of 8 days. Since this study aimed just to compare the two techniques, no relation to the challenges has been considered. Secretions from lavage were collected and centrifuged, and the pellets cytospun onto microscope slides. Scrapings were performed in the same nostril, immediately after lavage, at the middle third of the inferior turbinate with a Rhino-

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