

the hairdressers' shop. Thus, she habitually walked through the shop while other customers were having their hair tinted or bleached. The patient and her family had no history of atopic rhinitis or asthma.

Skin prick testing of a screening series and of indoor allergens revealed no sensitizations. Total IgE was 114 kU/l, a screening for inhalative allergens showed IgE of <0.35 kU/l (CAP class 0), and a screening test for chemical allergens also showed IgE of <0.35 kU/l (CAP class 0). Skin prick testing of a bleaching agent (Blondor Pulver) used in the hairdressers' shop gave a positive reaction (+++). Further

investigation revealed **Sensitization to ammonium persulfate led to eczema, rhinitis, and asthma in a hairdresser.** Therefore, skin prick testing of

a hairdressers' panel used in patch testing was performed, revealing a positive reaction (++) to APS 2.5%. This reaction could be confirmed by negative testing of APS 2.5% in 10 nonatopic and 10 atopic volunteers. No specific IgE (<0.35 kU/l) to APS could be detected in an external laboratory with a commercially unavailable RAST.

In contrast to type IV sensitizations, which are frequent in hairdressers, type I sensitizations to hair-care products are rare. Besides APS, permanent wave solutions (5), conditioners (6), hair spray (7), and dyes such as henna (8, 9), senna (10), and basic blue 99 (11) have been described as causative.

The mechanism underlying asthma induced by APS is disputed. Asthma due to a type I sensitization, including specific IgE for APS, has been described (12). Besides specific allergy, APS seems to be able to induce bronchial hyperreactivity, at least in a rabbit model (13). In our patient, no specific IgE could be detected, possibly because of the lack of a standardized method for the detection of specific IgE to the hapten APS. Nevertheless, positive skin prick testing, in contrast to negative testing, in a series of volunteers

indicates specific sensitization. The good correlation of skin prick testing with APS, history of asthma, and lung function has been shown in a series of industry workers with exposure to APS (14).

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Oral rush desensitization to milk

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Key words: cow's milk allergy; oral rush desensitization.

● Food hypersensitivities are common in children less than 3 years old. Allergic or intolerance reactions are mostly caused by vegetables, cereals, bird's eggs, and cow's milk. In predisposed children, allergy to cow's milk is often the first manifestation of food allergy (1). By the age of 3, most children, especially those with intolerance reactions, outgrow this sensitivity. About one-third of patients with IgE-mediated type I reactions are at an increased risk of retaining their sensitivity. The treatment of choice is the complete avoidance of cow's milk proteins (2). Because of the widespread use of cow's milk proteins in food, this approach is often difficult. Diet failures with anaphylactoid complications are not uncommon. Therefore, new therapeutic regimens are desirable.

We report on a 12-year-old girl with persistent IgE-mediated cow's milk protein allergy. After the introduction of cow's milk to

her diet, she developed generalized urticaria and Quincke's edema of the lips and eyelashes. A milk-free diet stopped her symptoms, but they recurred to a variable extent after diet failure with products containing cow's milk proteins.

Skin prick tests were performed with diluted fresh homogenized and pasteurized milk containing 3.5% fat. Starting with a dilution of 1:10 000 (milk/water), the skin threshold was identified at a dilution of 1:100 (milk/water). Specific IgE against cow's milk protein (4.26 kU/l), α -lactalbumin (4.71 kU/l), and casein (3.97 kU/l), as well as specific IgE against cooked milk (2.82 kU/l), whey (2.23 kU/l), and milk powder (0.5 kU/l) was found.

Because it is difficult to avoid cow's milk proteins, we performed oral desensitization with cow's milk for 5 days under clinical conditions, starting with 1 ml of cow's milk 1/100, doubling the dose every second hour, with 4–6 doses per day, until reaching 32 ml 1/100, and then repeating the same with dilution 1/10. Undiluted cow's milk was administered in a similar manner, continuing with 64, 100, and 200 ml and then daily intake of fresh cow's milk. The patient tolerated the procedure well. She was advised to continue drinking milk daily. Six months later, the patient was still free of symptoms.

Oral desensitization with food was introduced early in the therapy of food allergies. Schemes for cow's milk protein desensitization were normally performed over many weeks (3). We suggest a 5-day oral rush desensitization under clinical conditions. Our case has proven that this treatment is a fast and safe procedure to induce tolerance of cow's milk.

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Unexpected anaphylaxis to cashew nut

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Key words: anaphylaxis; cashew nut; food allergy; pistachios.

● DETECTION of culpable food(s) in patients with suspected food anaphylaxis can be difficult since both open and blind challenges might be impossible or unethical. This is a topical issue, considering allergen cross-reactivity between foods and inhalant allergens, the knowledge of which is of paramount importance in avoidance of dangerous foods. Allergy to the cashew nut and other plants belonging to the Anacardiaceae family (1) is well known.

Belonging to the Terebinthales order, the Anacardiaceae family includes edible species from the genera *Pistacia*, *Mangifera*, and *Anacardium* (Table 1).

Pistacia vera, i.e., the pistachio nut, is a tree native to the Middle East, but it is cultivated also in Italy, mainly in Sicily. The pistachio is a green, elongated nut, used in pastry, ice cream, and sauces, or as an appetizer.

The *Mangifera* genus includes the mango (*Mangifera indica* or *M. laurina*), a tropical fruit with an orange core and a sour taste.

Anacardium occidentale, commonly known as the cashew nut, is eaten as a snack with drinks.

Other, nonedible Anacardiaceae species products (such as balsamic resins from *Terebinthus* plants and Chios turpentine) are used in natural medicine for their expectorant properties; other plants of this family are cultivated for ornamental (*lentiscus*, *Pistachio chinensis*, *scotanus*, false pepper) and industrial (tannin from *Sommachus*; glossy black varnish from *Toxicodendron*; a very strong wood called quebracho from *Schinopsis*) purposes.

We describe the case of a patient allergic to pistachio who had

anaphylaxis due to cashew nuts. A 42-year-old white man was admitted to our emergency room complaining of

A patient allergic to pistachio nuts developed anaphylaxis after eating cashew nuts.

mouth and lip itching, slurred voice, dyspnea, and vomiting a few minutes after eating some cashew nuts. Prompt treatment with steroids, antihistamines, and epinephrine relieved the patient's symptoms, leading to a complete recovery in about 24 h.

After some weeks, the patient underwent a careful allergy examination at our allergy clinic. History disclosed three previous episodes of adverse reactions to pistachio. At the age of 30 years, when eating chocolate and hazelnut candy, the patient reported suffering strong mouth itching followed by vomiting.

Three years later, the patient complained of the same symptoms while eating pasta with a sauce based on garlic, basil, and pine nuts and attributed the symptoms to pine nuts and basil. After a few months, mouth itching and vomiting occurred again when the patient ate pistachio nuts.

The patient inquired into the ingredients of the provoking foodstuffs and found that the candy contained pistachio extract and that pistachio nuts had been used for the pasta sauce. The patient then held pistachios