

*Allergy & Respiratory Diseases, Department of Internal Medicine
 Padiglione Maragliano
 L.go R. Benzi 10
 16132 Genoa
 Italy
 Tel: + 39 10 3538908
 Fax: + 39 10 3538904
 E-mail: passalacqua@unige.it

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Food-exercise-induced anaphylaxis in a boy successfully desensitized to cow milk

L. Caminiti, G. Passalacqua*, D. Vita, P. Ruggeri, G. Barberio, G. B. Pajno

Key words: anaphylaxis; cow milk allergy; desensitization; exercise.

It is well known that exercise can induce systemic reactions, even anaphylactic shock, in some patients suffering from food allergy. Typically, exercise-induced anaphylaxis occurs on exercise immediately after the ingestion of a specific food. Nonetheless, it can occur also in the postprandial state without the certain identification of a specific causative food. In addition, the occurrence of anaphylaxis when the responsible food is ingested immediately after the exercise has been described (1). Food-dependent exercise-induced anaphylaxis has been variously associated with cereals, seafood, peanuts, tree nuts, eggs, milk and vegetables. We describe herein a severe anaphylactic reaction after exercise and milk ingestion in a child who previously underwent a successful desensitization protocol for cow milk (CM) allergy.

The patient was a 9-year-old boy who was diagnosed as having CM allergy at the age of 8 months. The clinical presentations of his CM allergy were urticaria, angioedema and wheezing. The diagnosis had been supported by skin prick test, CAP-RAST assay, and double blind, placebo-controlled food challenge (DBPCFC). Skin test was carried out on the anterior forearm surface with the prick by prick technique using undiluted CM. A negative control (saline solution) and a positive control (histamine 10 mg/ml) were also used (2). The patient showed an 8-mm-positive prick by prick test. The RAST assay (CAP system; Pharmacia, Uppsala, Sweden) gave a

Exercise after milk ingestion provoked anaphylaxis in a boy with induced tolerance to milk.

value of 4.80 KU/l for CM-specific IgE (normal value <0.35 KaU/l). The DBPCFC with CM and soy formula as placebo, performed at the clinic with full facilities for resuscitation, confirmed the diagnosis of allergy to CM. After the diagnosis was established, the patient began strict allergen avoidance and underwent a new food challenge every subsequent year, in order to verify if a remission had occurred. In fact, CM allergy usually remits or attenuates after 3 years of age. In our case, because of the persistence of symptoms confirmed by a stably positive food challenge, we decided to attempt a desensitization (tolerance induction), with slowly increasing doses of CM. The desensitization lasted 180 days, starting with two drops of cow's milk diluted 1:25 and progressively increasing, until a dose of 200 ml of milk (3) could be tolerated.

The protocol of oral desensitization was completed with limited side effects including: localized urticaria, stomach-ache and oral itching. These side effects were mild and self-resolving and required no treatment; but one episode of urticaria was easily controlled with oral antihistamines. At the end of the desensitization, the oral food challenge was negative and the boy could drink regularly about 300 ml of milk daily for breakfast. On one occasion, the boy participated in a football match just 30 min after having breakfast with the usual dose of milk. At the beginning of the second run, he suddenly developed generalized urticaria, angioedema, wheezing and dyspnoea. He was immediately treated on the field by the sports physician with intramuscular adrenaline, diphenhydramine and beta-metasone. He was then admitted at the EC unit, where he gradually recovered. Two months later, he was admitted to the emergency department with a similar major episode, which occurred again during strenuous exercise following ingestion of cow's milk.

After these episodes, we ascertained that the patient could freely drink milk, without subsequent physical exercise, and that physical exercise (full football match) without previous ingestion of milk did not provoke symptoms. The parents were then informed about the possible association of milk ingestion and exercise and were advised to get the boy

to avoid milk at least 3 h before any exercise (4).

The body of evidence concerning 'specific oral induction tolerance' for food allergy in humans is rather poor and it is not known how much effective and persistent this tolerance is (5). We report herein, for the first time, a case of successful induction of tolerance to a food with the subsequent appearance of severe exercise-induced symptoms. Thus, we notice, and advice, that achievement of induced tolerance to milk may be only partial and that the tolerance can be overcome by a relevant nonspecific stimulus such as strenuous exercise. In addition, the possibility of a 'recall food allergy' should be suspected after exercise (6), which follows the ingestion of food(s) previously tolerated.

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Department of Internal Medicine
Padiglione Maragliano
L.go R. Benzi 10
16132 Genoa
Italy
Tel: + 39 10 3538908
Fax: + 39 10 3538904
E-mail: passalacqua@unige.it

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Rhinitis because of toothpaste and other menthol-containing products

M. Andersson*, M. Hindsén

Key words: allergy, asthma, menthol, rhinitis.

Menthol (2-isopropyl-5-methyl cyclohexanol) is a cyclic alcohol widely used as constituents in several foods, cosmetic products, soaps and toothpastes. It has been reported that menthol can induce asthma and urticaria.

We present a case of a 44-year-old man, with a history of aspirin intolerance, rhinitis and mild asthma. Each time he brushed his teeth, pronounced rhinitis occurred. Profound nasal symptoms also occurred after shampooing, and after intake of certain sweets such as 'Fishermans friend'. The patient himself suspected that menthol could be the cause of his symptoms.

A clinical examination revealed a normal upper respiratory tract. To investigate whether nasal symptoms could be reproduced, a tooth brushing provocation was undertaken twice. Once after regular (once daily) use of antihistamines (Desloratadine®; Schering-Plough AB, Stockholm, Sweden) and intranasal steroids (Budesonide®; AstraZeneca AB, Södertälje, Sweden), and once without any treatment, 2 weeks apart. Symptoms were scored at baseline, before, 10 min

after tooth brushing with a menthol-containing toothpaste, according to a standardized protocol (1). Forced expiratory volume in 1 s was assessed by a Spirometer. Immediate and pronounced nasal symptoms occurred within 10 min after tooth brushing without any regular treatment, but no symptoms were observed when on treatment. Forced expiratory volume in 1 s decreased 10 min after tooth brushing on both occasions from 3.63 l/min to 3.0 l/min (on treatment) and from 3.18 l/min to 2.72 l/min without treatment.

Positive-contact urticarial reactions were seen when applying menthol 2% petrolatum (2) (Chemotechnique Diagnostics, Malmö, Sweden), peppermint oil 5% in petrolatum (Lever oy, Turku, Finland), toothpastes Colgate® 50% (Colgate-Palmolive, New York, NY, USA) in aqua and Aqua fresh® 50% (Glaxo-SmithKline, Research Triangle Park, NC, USA) in aqua and a chewing-gum (extra strong mint) – these different suspected compounds – to normal skin for 20 min on the volar aspects of the patient's arms. We also tested with a positive and a negative control. These tests were performed on two occasions with the same results.

There are few reports about menthol-containing products like toothpaste, which can cause asthma (3, 4), but this is, as far as we know, the first report of rhinitis symptoms caused by products containing menthol. The most likely explanation seems to be a true IgE-mediated reaction where small molecules of menthol are inhaled and which cross-link with mast cells in the airway mucosa. This is supported by the positive open application tests to several of the products that the patients reacted to and also the positive nasal provocation test. Obviously, the treatment with antihistamines and intranasal steroids offered protection. The best way to be symptom-free, however, is to exclude products that cause symptoms. The patient is now using menthol-free products and his symptoms have abated. The patient also had a history of aspirin intolerance which might have increased his response (5).

*Department of Otorhinolaryngology
Lund University Hospital
Se-221 85