

Exercise-induced anaphylactic reaction to hazelnut

Martín Muñoz F, López Cazaña JM, Villas F, Contreras JF, Díaz JM, Ojeda JA. Exercise-induced anaphylactic reaction to hazelnut. *Allergy* 1994; 49: 314–316. © Munksgaard 1994.

F. Martín Muñoz, J. M. López Cazaña, F. Villas, J. F. Contreras, J. M. Díaz, J. A. Ojeda

Allergy Service, Hospital "La Paz", Madrid, Spain

Reproducible, exercise-induced anaphylactic reactions occur in some patients only after certain foods have been eaten before exercise. We describe a patient in whom hazelnuts were a triggering factor for exercise-induced anaphylaxis. Skin tests and RAST were positive for nuts and grass and weed pollen. The exercise challenge test after hazelnut ingestion was positive. Food hypersensitivity should be investigated in all cases of exercise-induced anaphylaxis.

Key words: anaphylaxis; exercise; food.

Dr Flora Martín Muñoz
Allergy Service
Hospital "La Paz"
Madrid 28029
Spain

Accepted for publication 11 December 1992

Exercise-induced anaphylaxis (EIA) is now recognized as a distinct form of physical allergy. The syndrome is characterized by symptoms such as cardiovascular collapse and upper respiratory tract obstruction in association with flushing or urticaria, a rise in plasma histamine, and pathologic findings of mast cell degranulation (11–14). Predisposing events in the form of ingestion of either a specific food, such as celery (6, 14), shellfish (9, 12), or wheat flour (1), or any food (9) have been reported in a few patients, and a familial tendency is suggested in some reports (4). Anaphylaxis suggests mediator release, but the mechanism of release is unknown. An IgE mechanism is suggested in atopic patients (6).

We report the first case in which hazelnuts were the triggering factor in reproducible EIA.

Case report

Since the age of 10 years, a 13-year-old girl had suffered numerous attacks of urticaria, respiratory difficulty, "lump in the throat", and hoarseness after exercise, with no seasonal character. She had eaten nuts and seeds (almonds, peanuts, walnuts, chestnuts, sunflower seeds, and hazelnuts) and meals of all kinds without adverse reaction, and she had tolerated acetylsalicylic acid (ASA). She had no personal or family history of atopy.

Physical examination while the patient was asymptomatic was normal. Routine hematology studies, measurements of whole complement activity

(CH 50) and C3 and C4 complement proteins, immunoglobulin classes, and blood biochemistry were within normal range. Spirometric measurements before and after exercise were within normal values. Prick tests with a battery of the usual inhalants and foods were positive for the mixture of grass and weed pollen and nuts and seeds (almond, hazelnut, walnut, sunflower seed, chestnut, and peanut). We used saline solution (0.9%) as a negative control and histamine HCL at 1/100 w/v as a positive control. Readings were made after 15 min and were considered to be positive if the largest diameter of the papule obtained was greater than the positive control.

The total IgE (IMX, Abbot Diagnostics, USA) level was 731 kU/l. Specific IgE (Phadezym RAST, Pharmacia Diagnostics, Sweden) determination revealed RAST class 4 for hazelnut and sunflower seed; class 3 for peanut and mugwort; class 2 for ryegrass, chestnut, and walnut; class 1 for almond; and class 0 for shellfish and eggs.

The exercise challenge was performed according to the protocol of Bruce et al. (2). The patient ran on a treadmill with increasing speed and inclination until her heart rate reached a certain level, as determined by sex, age, and athletic history.

Spirometric measurements were performed with a Vitalograph® spirometer. The exercise challenge test was done in two phases: without prior food intake, and 30 min after ingestion of 30 g of walnuts, peanuts, almonds, sunflower seeds, and hazelnuts on different days. The oral challenge tests were per-

formed as placebo-controlled, single-blind tests with metabisulfites (MBS) and ASA. Four progressively increased doses of MBS were administered (10, 25, 50, and 100 mg) in lemon juice with a 30-min interval between doses. The peak expiratory flow (PEF) was recorded immediately before each dose and 30 min after taking 100 mg. The oral challenge tests with ASA were performed with 25, 50, 125, 250, and 500 mg on different days. The PEF was recorded immediately before each dose and 30, 60, 90, and 120 min after ingestion. The provocation tests were performed after obtaining informed consent from the patient and her family.

The ingestion of almonds, peanuts, sunflower seeds, walnuts, and hazelnuts without exercise induced no symptoms; but 3 h after eating hazelnuts, when she returned home, she ran vigorously for 3 min and approximately 5 min later, she noticed pruritus, urticaria, respiratory difficulty with wheezing, and hoarseness. She returned quickly to our hospital, where we treated her. About 60 min after ingesting 15 g of chestnuts, she developed urticaria and wheezing. Both attacks were controlled with epinephrine corticosteroids and antihistamines.

The exercise challenge without prior ingestion of foods, and 30 min after ingesting almonds, peanuts, or sunflower seeds, was normal. After she ate walnuts, the exercise test was positive; lung function revealed obstruction: FEV₁ decreased from 2.51 to 1.77 l (−30%), and MEF 25–75 decreased from 3.22 to 1.39 l (−55%) without any other symptom. She recovered after therapy with inhaled salbutamol. After she ate hazelnuts, no immediate obstructive response occurred 5 min after exercise, but 45 min after exercise, the patient experienced respiratory difficulty, exhibiting wheezing, dysphonia, and generalized urticaria. Lung function was measured at this time and revealed obstruction: FEV₁ decreased from 2.51 to 1.75 l (−30%) and MEF 25–75 decreased from 3.07 to 1.53 l (−49%). There was no fall in blood pressure. The attack was controlled with epinephrine, corticosteroids, and antihistamines.

The patient tolerated up to 100 mg of MBS without symptoms or decrease in PEF values.

About 120 min after ingesting 500 mg of ASA, she developed wheals on her face, wheezing, and decrease of the PEF value from 3 to 2.2 l/s (−27%). The attack was controlled with epinephrine corticosteroids and antihistamines. No new attacks of EIA occurred after the introduction of an appropriate nuts-avoidance diet and avoidance of ASA and other nonsteroidal anti-inflammatory agents.

The patient and her family did not consent to her undergoing a new challenge, to confirm the diagnosis.

Discussion

Certain adverse reactions to physical stimuli are termed “physical allergy” because the symptoms appear to be caused by mediators released from mast cells. Both EIA and cholinergic urticaria are physical allergies that can be precipitated by exercise (10). Kidd et al. (6) have distinguished two subsets of EIA: nonspecific food-dependent EIA and specific food-dependent EIA. The original description of shellfish ingestion before exercise causing anaphylaxis was reported by Maulitz et al. (8); subsequently, foods such as celery (6, 15), shellfish (12), and wheat flour (1) have been reported. Another precipitating factor is drug intake. Sheffer & Austen (11) and Dohi et al. (3) reported patients who experienced anaphylactic symptoms when they took aspirin before eating and exercise. Moreover, a familial tendency has been reported in some studies. Longley & Panush (7) reported familial EIA linked to the A3-B3-DR3 haplotype.

Our patient now has specific IgE against several nuts and seeds: almond, peanut, sunflower seed, walnut, hazelnut, and chestnut. She tolerates almond, peanut, and sunflower seed in all circumstances. She also tolerates walnuts and hazelnuts, but she experiences adverse reactions when she exercises afterwards. The ability of the patient to tolerate the ingestion of these nuts will obviate the need for her to undergo the double-blind, placebo-controlled food challenge to which she and her family were opposed. Walnut causes her exercise-induced asthma, and she has EIA after eating hazelnut. Moreover, she reports intolerance of chestnut.

Without prior ingestion of walnut or hazelnut, our patient is able to exercise without adverse reactions.

Finally, she develops urticaria and asthma after taking ASA without exercise afterwards.

There have been only a few reports on the pathophysiology of EIA. Increases in plasma histamine levels in EIA patients after exercise provocation testing have been reported by several authors (3, 5). Sheffer et al. (13) reported degranulation and morphologic changes in skin mast cells after exercise in patients with EIA. The detailed mechanisms involved in EIA are still unknown. Nonetheless, the irregularity of the attacks in the susceptible population strongly suggests that there are additional variables in every person experiencing the exercise-induced syndrome.

The primary factor in the prevention of this problem is its recognition. Our patient did not have seasonal manifestations, and her food and pollen hypersensitivity was unknown, as was her ASA intolerance.

We conclude that food hypersensitivity should be investigated in all patients with EIA.

References

1. ARMENTIA A, MARTÍN-SANTOS JM, BLANCO M, CARRETERO L, PUYO M, BARBER D. Exercise-induced anaphylactic reaction to grain flours. *Ann Allergy* 1990; 65: 149–51.
2. BRUCE RA, BLACKMON JR, JONES JW, STRAIT G. Exercise testing in adult normal subjects and cardiac patients. *Pediatrics* 1963; 32: 742–56.
3. DOHI M, SUKO M, SUGIYAMA H, et al. Food dependent, exercise-induced anaphylaxis: a study on 11 Japanese cases. *J Allergy Clin Immunol* 1990; 87: 34–40.
4. GRANT JA, FARNAM J, LORD RA, et al. Familial exercise-induced anaphylaxis. *Ann Allergy* 1985; 54: 35–8.
5. KATSUNUMA T, IIKURA Y, NAGAKURA T, et al. Exercise-induced anaphylaxis: improvement after removal of amalgam in dental caries. *Ann Allergy* 1990; 64: 472–5.
6. KIDD JM, COHEN SH, SOSSMAN AJ, FINK JN. Food-dependent exercise induced anaphylaxis [Abstract]. *J Allergy Clin Immunol* 1982; 69: 103.
7. LONGLEY S, PANUSH RS. Familial exercise-induced anaphylaxis. *Ann Allergy* 1987; 58: 257–9.
8. MAULITZ RM, PRATT DS, SCHOCKET AL. Exercise-induced anaphylactic reaction to shellfish. *J Allergy Clin Immunol* 1979; 63: 433–4.
9. MCNEIL D, STRAUSS RH. Exercise-induced anaphylaxis related to food intake. *Ann Allergy* 1988; 61: 440.
10. NOVEY HS, FAIRSHER RD, SALNESS K, SIMON RA, CURD JG. Postprandial exercise-induced anaphylaxis. *J Allergy Clin Immunol* 1983; 71: 498–504.
11. SHEFFER AL, AUSTEN KF. Exercise-induced anaphylaxis. *J Allergy Clin Immunol* 1980; 66: 106–11.
12. SHEFFER AL, AUSTEN KF. Exercise-induced anaphylaxis. *J Allergy Clin Immunol* 1984; 73: 699–703.
13. SHEFFER AL, SOTER NA, MCFADDEN ER JR, AUSTEN KF. Exercise-induced anaphylaxis: a distinct form of physical allergy. *J Allergy Clin Immunol* 1983; 71: 311–16.
14. SHEFFER AL, TONG AKF, MURPHY GF, et al. Exercise-induced anaphylaxis: a serious form of physical allergy associated with mast cell degranulation. *J Allergy Clin Immunol* 1985; 75: 479–84.
15. SILVERSTEIN SR, FROMMER DA, DOBOZIN B, ROSEN P. Celery-dependent exercise-induced anaphylaxis. *J Emerg Med* 1986; 4: 195.

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.